Chapter 2

The Etiology of Addiction
A Contemporary Biopsychosocial Approach

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A successful addiction model must synthesize pharmacological, experiential, cultural, situational, and personality components in a fluid and seamless description of addictive motivation. It must account for why a drug is more addictive in one society than another, addictive for one individual and not another, and addictive for the same individual at one time and not another. The model must make sense out of the essentially similar behavior that takes place with all compulsive involvements. In addition, the model must adequately describe the cycle of increasing yet dysfunctional reliance on an involvement until the involvement overwhelms other reinforcements available to the individual.

Introduction

The goal of the current chapter is to review contemporary perspectives on the etiology, or the causes, of addictive disorders. As illustrated by the epigraph above, this is no small task because of the complexity of these conditions and because the study of addiction is the focus of multiple disciplines using highly divergent perspectives. Furthermore, these different perspectives have not generated a single accepted account for why a person develops an addiction, but a number of empirically-grounded theoretical approaches that broadly fall into three domains—biological determinants, psychological determinants, and social determinants. These are collectively referred to as the biopsychosocial model of addiction, and the chapter will successively address these three domains, starting with neurobiological and genetic models, subsequently reviewing psychological theories, and then surveying social and societal influences. Finally, the chapter concludes with reflections on the progress and future priorities in understanding the causes of addiction.

Given the wide scope of this chapter, the emphasis will be on breadth over depth, and on theory over individual empirical studies. A fully comprehensive account of the etiology of addiction in each of these areas is beyond the scope of the chapter and incompatible with the clinical orientation of this volume. This raises the question of what the appropriate role of theory should be in the treatment of addiction. Scientific theories represent the abstracted relations among a wide array of empirical observations and, optimally, theory and treatment represent two sides of the same coin, the former describing the develop-
opment of the condition and the latter seeking to reverse-engineer the acquired dysfunction. Thus, a premise of the chapter is that a foundation in the causal models of addiction provides a scientifically-minded clinician with a framework for approaching treatment. Theories don’t provide a simple answer to why a given patient developed their presenting problem, but go deeper than symptoms to articulate the important processes and mechanisms that are putatively operative. Moreover, as the theories discussed below are grounded in empirical observation, theoretically-informed treatment is one key aspect of evidence-based treatment.

**Biological Models of Addiction**

**Neurobiological Models of Addiction**

Major progress has been made in understanding the effects of addictive drugs in the brain, leading to a number of influential neurobiological models. One of the earliest theories that shaped neurobiological perspectives was the *psychostimulant theory of addiction* [2], which identified a neurobiological common denominator across drugs of addictive potential via increases in dopamine release in the medial forebrain bundle, a neuronal tract within the mesolimbic dopamine pathway. Dopaminergic activation in this region was putatively responsible for subjective reward and for motivating behavior for survival and reproduction [3, 4]. Thus, mesocortical dopaminergic activity was theorized to be the common basis for the pleasure associated with drug effects and addiction potential across diverse pharmacological compounds. In terms of etiology, the principal mechanism was the sheer magnitude of dopaminergic stimulation produced compared to natural reinforcers. In contrast, subsequent theories specifically focused both on how drugs affected the brain acutely and how repeated administration gave rise to long-standing or potentially permanent brain changes, termed neuroadaptations, that cemented high levels of drug motivation.

Among more recent models, one of the most influential is the *incentive sensitization theory* [5, 6]. Again, incentive sensitization suggests that activation of mesolimbic dopamine substrates is critical to the development of the motivational and appetitive properties; however, incentive sensitization shifted the focus away from drug reward and parsed the role of dopamine more finely. Specifically, rather than mediating either the hedonic impact of the reward (i.e., its pleasure, how much it is liked) or rewarding learning, dopamine was hypothesized to subserve the incentive salience of the reward (i.e., its motivational value, how much it is wanted). Over time, via neuroadaptative changes that result from acute overstimulation of dopamine neurotransmission, sensitization of the incentive salience attribution is hypothesized to take place via associative (Pavlovian) conditioning, creating a chronic state of wanting (also called craving). Furthermore, the processes of wanting and liking are hypothesized to be dissociable, meaning that an individual sensitizes to the motivational
salience of the drug without necessarily increasing how much the person likes the drug. The liking process in turn is thought to be subserved by endogenous opioids in the ventral tegmental area. In terms of behavioral consequences, incentive sensitization is hypothesized to give rise to attentional bias toward drug stimuli and high levels of craving [7].

An alternative formulation, the cellular learning model of addiction, makes the case that addiction should be more broadly considered a disorder of learning and memory [8, 9]. From this perspective, sensitization of dopamine neurotransmission is one part of the neurobiology of addiction, but the subsequent downstream learning processes are even more important. Specifically, this account proposes that potent psychoactive drug effects contribute to synaptic plasticity that leads to long-term increases in the salience of drug rewards and, by comparison, decreases in the salience of alternative rewards [8, 9]. This is hypothesized to take place by way of drug-induced remodeling of neuronal dendrites, axons, and synapses, either via up- or down-regulation of gene expression or expression-based effects that lead to morphological synaptic changes. Supporting this thesis, addictive drugs have been robustly found to induce alterations in gene expression associated with synaptic plasticity, including inducing ΔFosB, a relatively long-lasting transcription factor that increases sensitivity to the rewarding and locomotor stimulant effects of addictive drugs [10, 11]. Thus, the highly potent psychoactive effects of addictive drugs are theorized to become deeply instantiated in the brain via potent effects on circuitry for learning new events and remembering important ones from the past.

Other neurobiological theories contrast with the preceding models that emphasize the drug acquiring very high rewarding salience. One dominant line of inquiry has pursued addiction as a series of transitions from voluntary use to habitual use to, ultimately, compulsive use of alcohol and other drugs [12, 13]. The neurobiological substrates responsible for these changes are theorized to be a transition from processing in the ventral striatum, which is responsible for subjectively rewarding drug effects subserving goal directed behavior, to the dorsal striatum, which is responsible for motor and habit learning. This neuroadaptive change is theorized to be a transition from deliberative action-outcome instrumental learning to reflexive stimulus-response learning, such that drug-seeking ultimately becomes increasingly automatic and outside of voluntary control. A substantial body of preclinical research supports this shift [14]. The ventral-to-dorsal striatum account is not necessarily incompatible with incentive sensitization, but it certainly emphasizes automaticity in behavior over increasing subjective drug motivation.

The allostatic model of addiction [15–17] shares a parallel with the ventral-to-dorsal striatum model to the extent that it emphasizes stages in addiction characterizing the initial heavy use of alcohol and drugs for its rewarding properties followed by chronic and uncontrolled use that is no longer driven by reward-seeking. Specifically, addiction is theorized to progress through three stages: (1) binge use/intoxication; (2) withdrawal/negative affect; and (3) preoccupation/anticipation (craving). These stages map on to progressive
neuroadaptive changes in the striatum, extended amygdala, hippocampus, and orbitofrontal cortex. Furthermore, the development of addiction is characterized as an aberrant homeostatic, or allostatic, process that involves changes in reward and stress circuits following persistent exposure to addictive drugs. This model is one of the few that explicitly integrates the neurobiology of the acute rewarding effects of drugs with mechanisms related to negative reinforcement associated with withdrawal and stress (e.g., corticotrophin-releasing factor, neuropeptide Y). One of the primary advantages of this model is that it integrates a wide array of findings on molecular, cellular, and neuronal changes that are associated with the pathophysiology of addiction. However, evidence from this model is drawn primarily from studies of alcohol, as opposed to other drugs of abuse.

Finally, although most of the preceding models have emphasized neuroadaptive changes in subcortical circuitry, it is worth noting that there are also contemporary neurobiological models that focus on deficits and acquired changes in prefrontal cortex functioning. In particular, neuroimaging studies and preclinical models have revealed dysregulation in subunits of the prefrontal cortex responsible for inhibitory control and reactivity to stimuli signaling drug availability [18–20]. Thus, addiction can be understood as resulting from both pathological adaptations within motivational systems and higher-level prefrontal systems.

Genetic Influences on Addiction

A limitation to most of the preceding models is that they typically describe addiction as a general process, not in relation to individual risk. In reality, addiction develops in only a minority of individuals who experiment with addictive drugs [21] and understanding which individuals are most vulnerable has important implications for both prevention and treatment. In biological models of addiction, the question of individual vulnerability largely pertains to genetic influences and genetic variation conferring etiological risk is well established. For example, there is an extensive literature using twin and adoption designs to ascertain the aggregate heritability of addictive disorders, generally suggesting 40–60% heritability [22, 23]. More recently, substantial heritability has also been identified using genomic complex trait analysis, a novel technique that generates estimates using variation in common single nucleotide polymorphisms (SNPs) across the genome [24, 25].

These studies make it clear that genetic variation is an important influence on the development of addiction, but the mechanisms by which this influence is conferred have been elusive. Candidate gene studies have generated mixed findings and atheoretical genome-wide association studies have generally not identified significant loci. However, two notable exceptions are robust evidence that variation in a locus responsible for alcohol pharmacokinetics is a protective factor against alcohol use disorder, and that variation putatively related to nicotine pharmacodynamics is a risk factor for nicotine dependence. In the first case, the ALHD2 gene is responsible for aldehyde dehydrogenase activity, a key enzyme for breaking down acetaldehyde resulting from alcohol metabolism, and the
A allele of an SNP (rs671) within ALDH2 results in substantially lower enzymatic activity. As a result, if A allele carriers drink alcohol, they experience an acetaldehyde buildup and a number of unpleasant symptoms, including flushing, nausea, headache, and tachycardia. The A allele is relatively common in Asian populations and effectively makes carriers “allergic” to alcohol, exerting a powerful protective effect against alcohol use disorder [26]. In the second case, a number of large-scale studies have convincingly implicated variants on chromosome 15 with nicotine dependence. This region contains the α5-α3-β4 nicotinic receptor gene cluster, and nicotinic cholinergic receptors are key sites of action for nicotine. In particular, a locus in the α5 nicotinic receptor subunit gene (CHRNA5), rs16969968, has been associated with significantly increased risk for developing nicotine dependence and smoking-related diseases, such as lung cancer and chronic obstructive pulmonary disease [27].

These findings represent two success stories in understanding addiction genetics, illustrating the ways that genetic variation may influence the pharmacokinetics and pharmacodynamics of drug effects to influence addiction risk. However, it is also clear that major gaps in knowledge remain and that progress in addiction genetics has been slower than anticipated, even after the development of sophisticated genome-wide techniques. In general, there is little evidence for one major “addiction gene” or a small number of highly influential loci. Instead, the current perspective is that it is likely that hundreds or thousands of variants contribute small magnitude effects to affect risk.

Difficulty in identifying genetic influences on addiction may also be a function of the heterogeneity of the clinical phenotype, given the many permutation symptoms that may be present. To address this, there is increasing interest in identifying narrower, more discrete behavioral phenotypes that are putatively more closely related to specific neurobiological processes and genetic variation in particular [28, 29]. These characteristics are also called intermediate phenotypes or endophenotypes, and are predicted both to increase power to detect specific genes underlying the risk for a given disorder and to inform mechanisms of risk or protection.

**Psychological Models of Addiction**

**A Reinforcement-Based Approach**

One of the earliest psychological theories of addictive behavior that is still actively pursued to this day is an operant learning approach. With foundations in early learning theory [30, 31], this approach theorizes that substance use is fundamentally a form of instrumental learning, meaning the behavior is primarily determined by its consequences and, more specifically, the reinforcing properties of the drug [32–34]. This comprises both positive reinforcement (i.e., effects provided by the drug that strengthen motivation), such as stimulation, social enhancement, orosensory, or gustatory properties; and negative reinforcement (i.e., states removed by the drug that strengthen motivation), such as the
alleviation of anxiety, depression, other psychiatric symptoms, or withdrawal symptoms. Importantly, these different forms of positive and negative reinforcement are not mutually exclusive, operating concurrently, and in the context of punishing drug consequences and the presence (or absence) of alternative reinforcers. In broad strokes, this approach proposes that a drug’s positively and negatively reinforcing properties, its punishing properties, the opportunity for alternative reinforcers, and the timing of the aforementioned jointly determine the reinforcing value of the drug, the final common pathway to use. These processes are theorized to be the proximal mechanisms by which other known risk factors (e.g., genetic and environmental vulnerabilities) contribute to substance use, and individual differences in each domain are responsible for differences in vulnerability across individuals.

Considerable evidence supports this approach, starting with data from early residential studies revealing that drug consumption could be studied experimentally and fundamentally conformed behavioral principles [35, 36]. Subsequently, human laboratory studies convincingly demonstrated that drug consumption conformed to key predictions from operant theory in terms of sensitivity to increases in response cost and the presence of alternative reinforcers [37–39].

More recently, a reinforcement-based model of addiction has been extended using behavioral economics, which integrates psychological and economic principles to understand decision-making and consumption behavior. This is a natural extension, following from recognition that operant behavior in complex environments with multiple options and different costs and benefits is essentially a behavioral microeconomy and that decision-making is a critical final common pathway to consumption behavior. Integrating economic concepts into addiction research also provides powerful tools for quantifying reinforcing value. One form of behavioral economic decision-making that has been extensively examined in relation to addiction is preference for smaller immediate rewards compared to larger delayed rewards. This is considered a behavioral economic index of impulsivity and is discussed below with other measures of impulsivity. In addition, purchase tasks that assess estimated drug consumption at escalating levels of price have been used to efficiently measure the reinforcing value of drugs, which is significantly associated with substance misuse and has been found to predict treatment response. An alternative measure characterizes substance-related reinforcement compared to non-drug alternative reinforcement, a measure of disproportionate reliance on drug-related reinforcement, and has also been linked to level of drug involvement [40–44]. Finally, a reinforcement-based approach has given rise to treatments that either seek to develop mutually exclusive alternative reinforcers to compete with drug use or directly reinforce elements of treatment, which are among some of the best supported treatments [45, 46].

Variability in Acute Drug Effects

A related perspective emphasizes on the importance of variation in the drug’s subjective effects as a determinant of use and misuse. This has most extensively
been investigated in relation to alcohol, but clearly has relevance to other drugs also. Early theories of alcohol effects predicted that individuals primarily drink alcohol because of its ability to reduce tension, the so-called tension reduction hypothesis. However, the evidence that a direct, consistent effect of alcohol is to alleviate tension is weak [47, 48]. Subsequently, it has become clear that alcohol’s direct effects are best understood as having both stimulant and sedative properties, with the former predominating during the ascending limb of the blood–alcohol curve and the latter predominating during the descending limb [49, 50]. In addition, attenuated response to alcohol has been identified as a risk factor for lifelong alcohol misuse [51, 52], and a recent meta-analysis revealed consistent evidence that the risk factor of having a positive family history of alcohol use disorder is conferred by attenuated alcohol effects [53]. However, it is notable that other studies have prospectively linked augmented stimulant effects to greater alcohol problems while greater levels of sedative effects are protective against the development of an alcohol use disorder [54, 55], thus suggesting that important aspects of this relationship remain insufficiently understood.

Cognitive Processes

The preceding theories reflect proximal properties of substances, but cognitive models emphasize the intervening role of mental or information processing mechanisms. One dominant cognitive model emphasizes the importance of expectancies in determining addictive behavior. Expectancies refer to cognitive templates that reflect the memorial residues from previous experiences and exist to anticipate experiences and facilitate behavior. Expectancies reflect bidirectional relationships in which experiences stamp imprints into the brain’s memory systems, and these imprints preemptively generate responses, effectively creating self-fulfilling behavioral prophecies. Expectancies are believed to be partially responsible for placebo effects, to medications in general [56] and to addictive drugs [57]. Furthermore, expectancy inventories on expectancies reveal the multifarious beliefs that individuals hold about drug effects [58]. For example, a wide variety of alcohol expectancies have been characterized, including global positive effects, sexual enhancement, social facilitation, assertiveness, relaxation/tension reduction, and interpersonal power [58], and expectancies have been significantly associated with substance use cross-sectionally and longitudinally [59–61]. Importantly, expectancies do not necessarily reflect direct pharmacological actions of alcohol so much as the individual’s aggregated construal of alcohol’s effects, resulting from the complex intersection of pharmacology, accurate and inaccurate attributions in ambiguous social and interpersonal contexts, and the background context sociocultural messages, norms, and advertising. For example, as noted above, alcohol may not have direct anxiolytic effects, but a person may attribute tension reduction properties to drinking beer because it is consumed as part of an after-work routine or because the brand markets it in that capacity.
Related cognitive determinants are motives for substance use, the pattern of reasons that a person reports for why he or she uses the drug. Like expectancies, motives are typically assessed using self-report assessments and validated measures have revealed distinct patterns of motives. For example, most drugs are used for social, enhancement, and coping motives [62–65]. However, differences are also present across drugs. For example, pain management is important for opioid users [64, 66]; sensory expansion is a distinct motivational domain for marijuana [65]; and social conformity represents a subfactor for young adult drinkers [67]. Facets of motivation have been robustly associated with levels of substance use and clinical severity, with coping motives exhibiting particularly robust associations [64, 67]. A larger array of motives have been identified for smoking, 13 in total (see Chapter 5). Of these, tolerance, craving, loss of control, and automaticity have been identified as the primary dependence motives, and are most robustly associated with nicotine dependence [68].

Within a cognitive framework, expectancies and motives can be thought of as explicit reflective cognitive processes, or declarative “top-down” processes in which the individual reports introspectively available cognitions about the drug. An important complement to those mechanisms are implicit automatic cognitive processes, or unconscious “bottom-up” processes that reflect the salience and weighting of drugs within a person’s cognitive network. Implicit cognition can be measured in a variety of different ways, but the common theme is using behavioral tasks that embed drug-related information and use behavioral performance, often interference, to reveal how salient drug information is within the person’s cognitive network. Level of cognitive bias on these measures has been significantly associated with level of substance misuse [69] and has also been found to be predictive of treatment response [70, 71]. Indeed, implicit cognition has given rise to novel adjunctive retraining treatments to degrade these acquired associations [72, 73]. Implicit and explicit measures of cognition are weakly associated, with some shared variance but both independently predicting substance involvement [74].

**Personality Factors**

The notion of an “addictive personality” has also elicited considerable interest as a psychological determinant of addiction, but has also been controversial [75], and there is weak evidence for any singular pattern of personality characteristics that is commonly present in addiction [76]. On the other hand, there is evidence that some normative personality traits are consistently associated with addictive behavior, including positive links with neuroticism and negative links with conscientiousness and agreeableness [77–80]. However, the most robust link between characterological traits and addiction is present for associations with measures of impulsivity, broadly defined as capacity for self-control of arising impulses. Importantly, impulsivity is measured in a variety of different ways and it is increasingly understood to be a multidimensional psychological trait. Self-reported impulsive personality traits on questionnaires reveal a number of
different facets. For example, the UPPS-P impulsive behavior scale comprises five subscales, including positive and negative urgency (i.e., proneness to act out during positive and negative mood states), premeditation (lack of) (i.e., level of deliberation or forethought), perseverance (lack of) (i.e., level of persistence or follow-through), and sensation seeking (i.e., preference for stimulating, exciting, or novel experiences). Of these, all of the traits have been linked to substance use, but positive and negative urgency are particularly related to clinical severity [81]. A second multidimensional measure of self-reported impulsive personality traits is the Barratt Impulsiveness Scale, which has also been robustly linked to addictive disorders and other externalizing behavior [82]. Beyond self-report, behavioral tasks can be used to measure orientation to immediate versus delayed reward (also referred to as delay discounting or delay of gratification), and capacity to inhibit prepotent motor responses (also referred to as response inhibition). In both cases, higher levels of impulsive responding have been linked to addictive disorders [83, 84]. However, it is notable that although the associations within the three domains of personality traits, delay discounting, and response inhibition are generally moderate to large, correlations across domains are generally small to negligible [85–87], suggesting they are distinct from one another.

**Developmental Psychopathology**

The last important psychological perspective is that of developmental psychopathology, an approach that seeks to understand psychiatric conditions as maladaptive deviations from normative human development. This perspective broadens the etiological lens to recognize influences prior to active drug use, such as prenatal influences and adverse childhood events [88, 89], and across the lifespan. In particular, a critical developmental window in the development of addiction is from adolescence to young adulthood, approximately 13–25. This is a broad window, but within it the vast majority of individuals will initiate their first exposures to addictive drugs and sizable proportions will progress to regular use and clinically significant misuse. For example, alcohol consumption peaks during emerging adulthood and is the most significant source of morbidity and mortality for this cohort [90, 91]. Furthermore, in the later phase of that time window, many individuals will naturally reduce consumption or stop using altogether, referred to as “maturing out” of drug use. In this way, it is not dissimilar to other forms of experimentation and role exploration that are present in adolescence, behaviors that are believed to be evolutionarily adaptive for developing autonomy, social status, and mate selection [92]. However, drug use during adolescence and young adulthood can also interfere with important developmental goals, such as educational attainment, career development, long-term relationships, and having a family [93–95], setting the stage for potentially lifelong problems. Notably, successful maturing out of substance use has been found to be a function of role transitions in terms of work, marriage, and parenthood [96–98]. Thus, there appear to be developmentally limited and
lifetime persistent forms of substance use and substance use disorder [99], subtypes that are not unlike other externalizing behavior [100]. Taken together, converging data suggest that this developmental window is similar to an ethological “critical period,” setting the stage for healthy and unhealthy substance use across the lifespan [101–103]. Furthermore, the preceding psychological mechanisms can also be understood within a developmental framework, with changes in expectancies, motives, and facets of impulsivity also predicting healthy and unhealthy changes in substance use during adolescence and young adulthood [104–108].

A final note pertaining to developmental psychopathology is that the perspective has been substantially enhanced by a deeper understanding of neurocognitive development. For example, the development of prefrontal cortex is gradual and protracted across adolescence and into young adulthood [109, 110]. Unfortunately, as a result, the developing brain appears to be more susceptible to neurotoxic effects of substance use [111, 112]. Furthermore, at least in preclinical models, adolescents appear to be more sensitive to reinforcing drug effects and less sensitive to the punishing effects [113]. Thus, adolescence and young adulthood represent a developmental window characterized by a surge in substance use during a period of neurocognitive vulnerability, with potential ramifications across the life span.

Social Models of Addiction

Social Networks

The importance of social factors in addiction is readily apparent from the observation that substance use is very commonly a social activity and the proverb that “birds of a feather flock together.” Furthermore, there is a large empirical literature supporting this perspective. For example, social enhancement features prominently in measures of expectancies and motives [58, 62–65] and estimated substance use among close social affiliates is highly correlated with personal use [114, 115]. The importance of social influences can also be seen in clinical research. For example, in large randomized controlled trials, changes in the alcohol-related composition of the important individuals in a person’s life have been found to predict treatment response, irrespective of experimental condition [116, 117]. Positive changes in social networks have been found to be mechanisms of the positive effects of Alcoholics Anonymous [118]. Furthermore, an intervention specifically developed to create a more positive social network has been shown to significantly increase behavioral and attitudinal support for not drinking and to significantly decrease drinking itself [119, 120].

The critical influence of a person’s social ecology has been even more clearly revealed via social network analysis (SNA), a family of methodologies for quantitatively characterizing the structure of relationships among people [121–123]. There are broadly two SNA approaches, egocentric and sociocentric. Egocentric SNA refers to a person’s self-reported social network (i.e., the
network from the perspective of that individual, referred to as the “ego”). Sociocentric SNA refers to the objective social network (i.e., each person rates their relationship with each other person, such that the network is a latent property of cross-ratings). The advantage of egocentric SNA is that it provides the ego’s perspective on the important people in their lives, whereas the advantage of sociocentric SNA is that it characterizes an objective network of individuals. A number of studies have examined social network dynamics relating to addictive behavior and have generated a number of important insights. For example, in early adolescents, individuals who are central to their social networks are more likely to use alcohol [124] and have been found to have more influence on their friends’ alcohol use [125]. In addition, there is evidence for what are referred to as selection dynamics (e.g., drinkers seeking out other drinkers) and influence dynamics (i.e., the presence of drinkers in a network inducing more drinking), and these dynamics vary across adolescence [126–130]. Similarly, in adults, drinkers have been found to cluster together and social network characteristics predict changes in drinking over time [131–133], with parallel findings for other addictive disorders [115, 124, 129, 134].

Collectively, a social network perspective proposes that individuals self-select into networks of social relationships that are populated with people exhibiting similar levels of substance use (or lack thereof). For individuals with addiction, these networks are theorized to have a self-perpetuating influence on their members over time, including impeding behavior change in treatment. Thus, for some individuals, treatment may not just require abstaining or reducing alcohol, tobacco, cocaine, or heroin, but giving up important interpersonal relationships too. Here again, there is a maladaptive cycle in which social network influences recursively maintain the addictive behavior.

**Classes of Social Influence and Mechanisms**

It is important to recognize that not all members in social networks are of equal importance and the level of influence varies across the life span. In the critical period of adolescence and young adulthood, parental influences and peer influences are particularly powerful. A number of different parental influences have been identified. Arguably, the most important influence is parental substance use [135, 136], which can model the behavior, communicate perceived approval, and increase availability. In addition, parenting style is an important factor. Authoritative parenting is protective against substance use [137, 138], but the reverse is true for harsh parenting and parental hostility [139–141]. As parent–child connectedness and parental support are also negatively related to substance use [137, 138], it appears that both structure and warmth are important protective influences. Peer influences on substance use can be divided into three broad domains: overt offers, reflecting direct requests to use; modeling, reflecting passive social influence by familiar or unfamiliar peers; and social norms, reflecting the overestimation of typical behavior within a cohort [142]. All three domains are influential to varying extents [143–146]. In addition, in the case of
social norms, social media campaigns have been undertaken to modify widespread overestimates, albeit with mixed evidence of efficacy [147].

Although parental and peer influences are most relevant to adolescents and young adults, dyadic influences, or significant others, are a potent social influence throughout adulthood. This is particularly an issue because individuals who use substances are more likely to be in a relationship together [148, 149], referred to as assortative mating, leading to dual-addiction couples. Like parental substance use, substance use among significant others provides a form of modeling, communicates approval, and provides access to substances [150]. However, addiction in couples is also associated with additional adverse patterns and consequences, such as intimate partner violence and poor parenting [150]. Thus, addiction among both members of a dyad represents a particularly deep embedding of the condition within a social network.

**Sociocultural Influences**

Finally, social influences on addiction include higher-order factors within society and culture, such as religion, economic conditions, and public policy. Religion is highly influential in overall population levels of substance use [151, 152] and, in terms of public policy, levels of taxation have major impacts on tobacco and alcohol consumption [153, 154]. Related to taxation, there is robust evidence that minimum pricing for alcohol reduces consumption [155, 156] and reduces negative consequences from drinking [157]. Other regulatory public policy influences include legal age of consumption, private versus state monopoly markets, law enforcement, density of outlets, and the availability of drink specials/“happy hours” [158]. Of course, economic and policy influences largely only pertain to legal addictive drugs or gambling because illicit drugs are unregulated. However, access to evidence-based prevention and treatments, and costs of care, are also important sociocultural factors that affect treatment for all forms of addiction [159–161]. In each of the preceding cases, these represent ways that a geographic area can have a favorable or unfavorable sociocultural climate toward the development and treatment of addiction.

**Conclusions**

If the goal of scientific theory is “to carve nature at its joints,” then by extension, in clinical science, the goal of treatment is to intervene upon the dysfunction that is present in each of the resulting parts. A contemporary biopsychosocial approach carves addiction into three major sections and then further subdivides in a number of different ways. What emerges across these multifarious accounts is that there is no simple or singular answer to the question of why people develop addiction. Contemporary neurobiological theories of addiction offer incisive insights into addiction, emphasizing that psychoactive drugs use evolutionarily novel levels of stimulation to subvert, or even hijack, ancient brain systems that are responsible for adaptive motivation, learning, and executive control.
Psychologically, elevations in the reinforcing value of drug effects, maladaptive explicit and implicit cognitive processing, and deficits in self-regulatory capacities all contribute to persistent drug use, influences that are superimposed upon a developmental backdrop. Finally, social factors play a critical role, from family members and friends to extended social networks and a person’s broader sociocultural context. A common theme in these accounts is the presence of recursive etiological processes, or feedforward processes that, once initiated and sufficiently engaged, are theorized to become self-sustaining and exacerbating. In other words, across theoretical accounts, there is convergence that addiction is a disorder of “vicious cycles,” or patterns of maladaptive overconsumption that over time become increasingly difficult to change.

The array of perspectives reveals both strengths and weaknesses in the science of addiction. The contemporary approach provides a rich multidimensional perspective, spanning levels of analysis and addressing the complexity of the condition. However, rather than reflecting true synthesis across levels of analysis, a biopsychosocial approach still predominantly reflects discrete perspectives within each of these three domains. Furthermore, theoretical perspectives typically do not extend across disciplinary boundaries. Biological, psychological, and social approaches tend to be siloed away from each other, especially as methodological and disciplinary differences get larger. For example, there are no links between preclinical animal models, human developmental psychopathology, and tax policy. In this way, the field is akin to the parable about “the blind men and the elephant”—researchers in many different areas of the field are correctly identifying important aspects of a large complex problem, but no holistic theoretical viewpoint provides an overall framework.

Importantly, however, a merging of perspectives is increasingly taking place. Neuroimaging is increasingly permitting insights from preclinical models to be investigated directly in human participants affected by addiction. Genetic variables are being woven into psychological and social frameworks, and reciprocally behavioral and social measures are serving as novel phenotypes for genetic dissection or as moderators of genetic influences. Novel medications are targeting promising neural pathways from preclinical research and providing innovative mechanisms of action. These will be the advances that permit more comprehensive accounts of addiction to be developed, ones that more satisfactorily rise to the challenge of the epigraph at the chapter’s start. Furthermore, even in its current incarnation, the contemporary biopsychosocial approach nonetheless provides a wealth of etiological processes and mechanisms for clinicians to consider in treating patients, making it an indispensable perspective in evidence-based treatment of addiction.

**Note**

1. Beyond a binary distinction, it is worth noting that a wide variety of theoretically and empirically derived addiction subtypes or trajectory profiles have been identified, but that a comprehensive review of these denominations is beyond the scope of the chapter.
References


