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ALCOHOL USE AND HEALTH

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Alcohol consumption has been an important part of human behavior since recorded history and has important social and cultural significance. Unfortunately, alcohol is also a substance with strong abuse liability. Excessive alcohol consumption, or ‘problem drinking,’ can result in significant negative medical, social, interpersonal, academic, and public health consequences, including the development of alcohol use disorders (AUDs). This chapter briefly reviews the behavioral pharmacology and biochemistry of alcohol, as well as the risks and benefits of alcohol consumption. Next, the chapter highlights key biopsychosocial models of the patterns, predictors, and consequences of alcohol consumption. Finally, the chapter briefly reviews the most recent clinical intervention efforts to reduce alcohol consumption.

Behavioral Pharmacology and Neurobiology of Alcohol

Ethyl alcohol (referred to herein as ‘alcohol’) is an organic compound with the molecular formula CH₃CH₂OH. Alcohol is a psychoactive substance created from fermentation of sugars exposed to microorganisms. The process yields solutions that have a maximum concentration of 10–15% alcohol, which can be further augmented by distillation. Alcoholic beverages are thus available in a variety of concentrations, most notably, beer (~5% alcohol), malt beverages (~7% alcohol), wine (~12% alcohol), and distilled spirits (e.g., whiskey, rum, gin, vodka; ~40–50% alcohol).

Pharmacokinetics

Alcohol is typically ingested orally, absorbed through the stomach epithelium, and distributed to distal organs, including the brain, into the water content of the body (‘total body water’). Although some first-pass metabolism occurs in the stomach, primarily metabolism of alcohol occurs in the liver. Key liver enzymes involved are alcohol dehydrogenase (ADH) and the cytochrome P450–2E1 enzyme (CPY2E1), which convert ethanol into the highly toxic carcinogen acetaldehyde, and aldehyde dehydrogenase (ALDH), which subsequently converts acetaldehyde into acetate. Acetate is then easily metabolized into carbon dioxide and water, facilitating elimination from the body (Heit et al., 2013).

Pharmacodynamics

Although alcohol is classified as a central nervous system depressant (National Center for Biotechnology Information, 2017), it has long been known that alcohol has biphasic effects, including both
stimulation and sedation (Hendler, Ramchandani, Gilman, & Hommer, 2013). Stimulating effects include mood elevation, social lubrication, gregariousness, impaired judgment, and anxiety reduction. Sedating effects include a number of cognitive and motor disturbances, including lethargy, respiratory depression, and loss of consciousness.

The degrees to which stimulation and sedation are experienced can be both dose- and time-dependent. Stimulating effects of alcohol are typically experienced at lower doses, whereas sedating effects are more prominent at higher doses (King, de Wit, McNamara, & Cao, 2011). Similarly, within a given drinking episode, stimulating effects predominate relatively early in the drinking trajectory while sedative effects predominate later in the episode (Newlin & Thomson, 1990). Variability in pharmacokinetics and pharmacodynamics may play an important role in the development of problem drinking, as discussed later.

**Neurobiology**

Both acute and chronic alcohol consumption have been shown to affect numerous neurotransmitters, although the interplay between these systems in the presence of alcohol is complex (Gilpin & Koob, 2008). Alcohol’s primary neurobiological effects are in the γ-aminobutyric acid (GABA) system, a key inhibitory neurotransmitter. Both cell culture and in-vivo studies have demonstrated that alcohol exerts its effects by the phosphorylation of surface proteins on GABA receptors, potentiating GABA activity (Roberto & Varodayan, 2017). Increases in GABA-related inhibitory signaling may be one mechanism underlying the sedative effects of alcohol consumption (Kumar, Fleming, & Morrow, 2004).

Paradoxically, alcohol also exerts its effects on inhibitory GABA neurons in the mesolimbic dopamine pathway, resulting in inhibitory influences on the system responsible for the integration of reward signals. Complicating the picture, however, alcohol also stimulates the release of hypothalamic opioid peptides (e.g., β-endorphin) that have inhibitory effects on GABA, potentiating mesolimbic dopamine activity. Similarly, alcohol blocks excitatory glutamate signals from triggering GABA neurons in the dopamine pathway, leading to a net increase in dopamine transmission (Gilpin & Koob, 2008). The development of AUDs may thus involve the dysregulation of the mesolimbic dopamine system, significantly increasing the motivational salience of the desired substance, increasing urges to consume the drug, and increasing vigilance and attention toward stimuli and events that trigger substance availability (Berridge and Robinson (1998). This ‘incentive-sensitization’ theory is one important example of a theory that integrates complex biological and psychological processes contributing to alcohol use, as discussed later.

**Consequences of Alcohol Use**

**Risks**

Acute alcohol intoxication has been blamed for violence, sexual assault, motor vehicle accidents, academic failure, blackouts, and numerous other negative personal and societal consequences. Following episodes of alcohol consumption, drinkers experience hangovers, disturbed sleep patterns, and other aversive withdrawal symptoms (e.g., autonomic hyperactivity, agitation, nausea, anxiety), all of which can contribute to the negative sequelae of drinking (e.g., loss of work, relationship conflict) (National Institute on Alcohol Abuse and Alcoholism, 2017).

As with most psychoactive drugs, regular consumers of alcohol are at increased risk of developing tolerance (diminished drug effects with repeated use), withdrawal, and powerful alcohol cravings. According to DSM-V, AUDs are diagnosed when individuals exhibit significant impairment and
distress associated with chronic alcohol use and alcohol-seeking thoughts and behaviors, coupled with the presence of tolerance and withdrawal (American Psychiatric Association, 2013). Although AUDs may be a particularly severe consequence of alcohol use, DSM-V catalogs twenty different clinical diagnoses associated with acute and chronic alcohol consumption, highlighting the widespread risks associated with alcohol use.

Chronic drinking has also been associated with numerous physical health risks, including liver disease, cancer, fetal alcohol syndrome, cardiomyopathy, malnutrition, dementia, and diffuse neurotoxicity, to name a few. Risk of developing AUDs is highest among young adults aged 18–29, with a prevalence estimate of 16.2%. Rates in the U.S. are higher among men than women, and among Native Americans than members of other racial and ethnic groups (American Psychiatric Association, 2013).

**Benefits**

Moderate alcohol consumption (e.g., no more than one drink/day for women or two drinks/day for men) may have specific health benefits, including decreased risk of coronary artery disease, stroke, dementia, and diabetes (Reynolds et al., 2003; Standridge, Zylstra, & Adams, 2004). Recent analyses, however, have highlighted methodological shortcomings in this literature, including selection biases in many studies (Chikritzhs, Fillmore, & Stockwell, 2009). In consideration of the significant risks of alcohol use, the Dietary Guidelines for Americans (U.S. Department of Health of Human Services, 2015) concludes that alcohol’s potentially beneficial effects do not justify the initiation of alcohol consumption.

**Theories of Alcohol Use**

**Overview**

Early records of problem drinking date back to tavern regulations in the Code of Hammurabi (approximately 2240 B.C.E.). Until relatively recently, a ‘moral model’ dominated beliefs about drinking. Bolstered by the temperance movement in the 19th century and the writings of influential physician Benjamin Rush, the moral model of alcoholism focused on weakness of character and lifestyle choices that would lead to the immoral conduct of drinking (Edwards, 2012). Because drinking was considered a moral failing, few resources were allocated toward prevention and treatment.

Jellinek (1960) advanced a ‘disease model,’ noting the loss of control over drinking typically observed among users, as well as failure to remain abstinent over repeated attempts to quit. The disease model posits that AUDs are genetically determined, chronic, and progressive, and should only be treated by promoting abstinence. The disease model was further popularized by groups such as Alcoholics Anonymous, ultimately leading to the labeling of AUDs as a disease by the medical and public health communities. The propagation of the disease model was instrumental in increasing access to medical treatments, changing policy to encourage insurance companies to cover treatments, and increased funding for biomedical research related to drinking. Nevertheless, the disease model has been criticized as being overly simplistic, focusing on a biologically driven inability to control the development and progression of AUDs. In addition, the disease model does not account for the involvement of key psychological and sociocultural factors that contribute to drinking. Finally, considerable evidence has called some of the key principles of the disease model into question. For example, studies have found that loss of control among drinkers is not universal, but rather drinkers make poor drinking decisions based on a maladaptive appraisal of the pros and cons of consumption (Mello & Mendelson, 1972). In addition, many drinkers ‘mature out’ of AUDs (Davies, 1962),
arguing against their chronic and progressive nature. More recent models, therefore, have characterized AUDs as the product of maladaptive cognitions, behaviors, and decision making.

‘Maladaptive behavior’ models recognize the roles of biological, psychological, and social processes that may contribute to drinking decisions. Not unlike other maladaptive behavior, AUDs are conceptualized as one of any number of behaviors that can be explained using classic learning principles. Biological, psychological, and social factors provide context for AUDs which develop from a long series of learned maladaptive behaviors across time. Such models have led to a much clearer understanding of AUD risk.

**Biological Models**

Most biological models of alcohol use highlight the well-known finding that AUDs run in families. Early studies of ‘children of alcoholics’ reported higher risks of AUDs among individuals with family histories of problem drinking (Monteiro & Schuckit, 1988). These studies, however, did not disentangle genetic and environmental sources of variability. Twin and adoption studies provided the first solid evidence for genetic contributions to AUD risk (Alterman & Tarter, 1983). A recent meta-analysis estimated that approximately 50% of the variability in AUDs is attributable to genetics (Verhulst, Neale, & Kendler, 2015).

Evidence for the role of specific genes related to alcohol use was elusive until the 1990s, when polymerase chain reaction and similar technologies to rapidly identify single nucleotide polymorphisms (SNPs) became widely available [see Tyndale (2003) for a review]. Studies have consistently found that genes in the alcohol metabolic pathway (i.e., polymorphisms in genes coding for ADH, ALDH, and CYP2E1) are associated with AUD risk (Long et al., 1998; Okamoto, Murawaki, Yuasa, & Kawasaki, 2001).

Some (Long et al., 1998), but not all (Hill, Zezza, Wipprecht, Xu, & Neiswanger, 1999), studies, found associations between AUDs and SNPs in the dopamine neurotransmission pathway. Similarly, Blum et al. (1990) reported associations between the gene that encodes the D2 dopamine receptor and AUDs. Attempts to replicate these findings, however, have been mixed, with many reporting null results [e.g., Gelernter et al. (1991)]. Similar mixed findings have been observed with genes coding for other proteins in the dopamine, GABA, glutamate, serotonin, and opioid systems (Tyndale, 2003).

The advent of cost-effective haplotype and high-throughput genome-wide analyses brought great promise, only to continue to yield mixed results (Hart & Kranzler, 2015).

Other biological considerations in the development of AUDs include sex and race differences in alcohol metabolism. Women have lower levels of total body water (Mirand & Welte, 1994), and are, as a group, more sensitive to the effects of a given dose of alcohol (Brady & Randall, 1999). Drinkers with Northeast Asian ancestry are more likely to exhibit aversive ‘flush’ responses to alcohol, characterized by uncomfortable blushing on the face and upper body. Genetic studies have revealed that this response is mediated by genes that code for the ADH and ALDH enzymes and is predictive of lower AUD risk (Wall, Luczak, & Hiller-Sturmholz, 2016).

There are a number of key limitations to genetic models of AUDs. First, research has largely relied on cross-sectional or retrospective case-control designs. Second, studies used widely diverse definitions of AUDs and related phenotypes, making comparisons of results challenging, if not impossible. Perhaps most importantly, most studies yielded very small effects and often suffered from high Type I error rates due to analyses of hundreds of candidate SNPs, leading to repeated failures to replicate. Another reason for mixed findings is the recognition that the SNPs that have been studied are typically ‘high frequency-low penetrance’ polymorphisms. These SNPs, while frequently occurring in the population, cannot by themselves have substantial influences on complex behavioral phenotypes like AUDs. Genetic influences likely interact with myriad complex social and environmental
influences, as well as with many other genes contemporaneously. The single-gene approach thus oversimplifies a complex, diverse, and nuanced phenotype. Also notably absent in these models is a clear psychological or behavioral mechanism underlying the relationship between genetics and AUDs. Thus, the purely biological models fail to address this key question: What is it about having a specific genotype that somehow motivates alcohol consumption and leads to AUDs?

Reinforcement Models

To address possible behavioral mechanisms underlying AUDs, we turn to theories highlighting individual differences in the subjective effects of alcohol. Conger (1956) advanced the ‘tension-reduction hypothesis,’ proposing that individuals consume alcohol because of its favorable effects, especially anxiety reduction (anxiolysis). Similar models, including the ‘stress-response dampening’ model (Levenson, Sher, Grossman, Newman, & Newlin, 1980) center around alcohol’s favorable subjective effects (e.g., stimulation, sedation) as motivators of consumption. Interestingly, more recent cognitive adaptations have suggested that alcohol’s anxiolytic effects may be mediated by impairments in attention, which may in turn reduce drinkers’ capacity to focus on sources of anxiety in their lives. The ‘attention-allocation’ model (Josephs & Steele, 1990) posits that, during intoxication, anxiety decreases when involved in a competing task (e.g., sorting cards) but increases when no other tasks are available (hence the ‘crying over one’s beer’ phenomenon). Josephs and Steele hypothesize that drinking focuses drinkers’ limited attention to the most salient environmental stimulus in the moment (‘alcohol myopia’), which can have either anxiolytic or anxiogenic effects, depending on what other attentional demands are present.

Individual differences in alcohol’s subjective effects have been hypothesized to predict risk of AUDs. Most notably, Schuckit et al. (2011) proposed that low levels of response (LR) to alcohol are associated with increased risk of AUDs. They suggest that insensitivity to alcohol’s subjective pharmacodynamic effects motivates LR drinkers to consume more alcohol to achieve a satisfactory level of intoxication. Consistent with a genetic explanation for this phenomenon, individuals with family histories of AUDs, as well as carriers of variants in ADH and CYP2E1 genes, exhibit LR (Webb et al., 2011).

Newlin and Thomson (1990) proposed a more nuanced version of the LR model. Their Differentiator Model (DM) proposes that risk is associated with heightened sensitivity to the pleasant, stimulating effects on the ascending limb of the blood alcohol curve, but dampened sensitivity (i.e., LR) to the putatively aversive sedating effects on the descending limb. Differentiated response to the biphasic effects of alcohol is hypothesized to have a ‘double whammy’ effect. Evidence in support of the DM is mixed [e.g., Erblich and Earleywine (2003); King et al. (2011)]. A meta-analysis by Quinn and Fromme (2011) concluded that there is strong evidence for heightened sensitivity to the stimulating effects of alcohol among individuals at increased risk for AUDs, but evidence is less compelling with respect to the dampening of sedative effects.

Closely related to reinforcement models are approaches highlighting personality traits associated with dysregulation of reinforcement, especially impulsivity. Impulsivity is a multifaceted trait characterized by acting with urgency, overvaluation of immediate gratification, poor behavioral inhibition, and sensation seeking. Theoretical conceptualizations of impulsivity suggest that impulsive individuals exaggerate the expected reinforcing properties of alcohol consumption (Lejuez et al., 2010). Interestingly, although genetic studies have attempted to identify polymorphisms associated with impulsivity among individuals at risk of AUDs, results have been inconsistent (Derringer et al., 2015; Pfeifer et al., 2015). Recent work has highlighted the importance of more precisely defining impulsivity, as not all aspects of impulsivity are predictive of AUDs. For example, there are numerous self-report indices that assess related, yet distinct, constructs, including the Impulsive Sensation Seeking Scale of
the Zuckerman–Kuhlman Personality Questionnaire (Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993), the Sensation Seeking Scale (Zuckerman, Eysenck, & Eysenck, 1978), the Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995), and, most recently, the UPPS-P scale (Whiteside & Lynam, 2001), which measures five key dimensions of impulsivity: positive urgency, negative urgency, perseverance, premeditation, and sensation seeking. Recent work suggests that positive and negative urgency are particularly strong predictors (Cyders, 2013). Urgency refers to the tendency to act rashly without thinking, and positive and negative urgency reflect the manifestation of this tendency when experiencing positive and negative affect, respectively. Notably, although positive and negative urgency have been characterized as facets of impulsivity, they also reflect momentary affective states, yielding richer indices of affect-driven impulse control, rather than impulsivity per se.

Researchers have also developed numerous behavioral indices of impulsive decision making and action. Performance on a delay discounting task is one measure that has received a great deal of attention. This task requires individuals to rate their preferences for a graded series of immediate monetary rewards versus larger rewards at varying time delays. Impulsive individuals more steeply discount delayed rewards, and therefore have a steeper ‘discounting coefficient.’ Related to alcohol use, the discounting coefficient represents increased reinforcing value of immediate reward (e.g., drinking right now) relative to potentially greater future reward (e.g., long-term physical, psychological, and social functioning). Higher delay discounting predicts numerous alcohol use outcomes (Gray & MacKillop, 2015). Other behavioral measures of impulsivity include motor inhibition tasks, including the Stop Signal, Go No-Go, and Flanker Tasks, with which individuals are tested for their ability to inhibit motor responses.

In a recent review, Weafer and de Wit (2014) concluded that there are numerous nuanced sex differences in the measures of impulsivity. In addition, Weafer, De Arcangelis, and de Wit (2015) found that heavy drinking women exhibited poorer motor inhibition than men, but did not differ on delay discounting. Cyders (2013) on the other hand, found that UPPS-P indices did not differentially predict alcohol-use outcomes by sex. In one recent attempt to better characterize the construct of impulsivity, MacKillop et al. (2016) concluded that ‘impulsive choice,’ ‘impulsive action,’ and ‘impulsive personality traits’ were three key, yet independent, facets of impulsivity that should be considered separately when developing models of AUD risk.

In 2006, Murphy and MacKillop (2006) developed a novel measure of the reinforcing value of alcohol. Based on behavioral economic principals, their Alcohol Purchase Task (APT) assesses how much alcohol individuals would hypothetically consume at a series of price points (e.g., $0/drink to $20/drink). The resulting data yield numerous indices of alcohol demand, including intensity (consumption when free), elasticity (slope of decreased consumption as a function of price), and breakpoint (price point at which consumption reaches zero). A number of studies have now found that more impulsive drinkers display higher intensity, lower elasticity (i.e., less sensitive to increases in price), and higher breakpoint, lending support to the contention that impulsivity may contribute to risk of AUDs by increasing the reinforcing value of alcohol (Murphy et al., 2015).

Encompassing reinforcement models of AUD include consideration of genetic factors, measures of alcohol’s subjective effects, indices of impulsivity, as well as measures of alcohol’s reinforcing value. Missing from the reinforcement models, however, is the etiology of motivation to consume alcohol in the first place, a factor that is addressed by conditioning models.

**Conditioning Models**

Although principles of operant conditioning are implicit in the reinforcement models, any theory of AUDs remains incomplete without an appreciation for the role of classical conditioning in the motivation to drink. Clinical reports, as well as considerable empirical data, point to the fact that
both external (e.g., alcohol-related stimuli in the environment) and internal (e.g., stress, negative and positive affect) cues can trigger powerful urges to drink. Early conceptualizations of substance use from a conditioning framework come from Wikler (1948), who proposed that drug-use environments serve as conditioned stimuli that induce withdrawal-like responses. Other theorists (S. Siegel, Hinson, Krank, & McCully, 1982; Stewart, de Wit, & Eikelboom, 1984) have elaborated on the conditioning model, raising the possibility that drug-related conditioned stimuli can induce drug-like effects, drug-opposite effects, compensatory responses or some combination of these. The net behavioral effect is an increase in motivation to use, often termed ‘craving.’ Craving is a difficult to define construct (Kozlowski & Wilkinson, 1987), but by most accounts, broadly reflects underlying urges and increased motivation to seek out and consume drugs.

Conditioning models propose that over repeated trials of alcohol use, a broad array of both external and internal stimuli become consistently associated with drinking, resulting in ubiquitous cues that trigger powerful alcohol cravings. These ‘cue-induced cravings’ have been modeled under laboratory conditions, in which drinkers are experimentally exposed to alcohol-related paraphernalia (e.g., smell of a preferred alcoholic beverage) or mild affect-inductions and asked to self-report cravings.

Not surprisingly, substantial individual differences have been observed in the magnitude of cue-(and/or affect/stress-) induced cravings. Conditioning models predict that individuals who experience stronger cue-induced cravings would be at increased risk of AUDs. These findings are consistent with the incentive-sensitization model (Robinson & Berbridge, 1993) described earlier. Repeated exposure to alcohol creates habituation to hedonic effects of the drug, but also sensitizes the drug-seeking and ‘wanting’ (i.e., craving) mechanisms through conditioning. Interestingly, this model suggests that the sensitization of conditioned cravings is mediated by dopamine activity, a prediction consistent with several candidate gene studies finding that dopamine-related polymorphisms were predictive of potentiated cue- and stress-induced cravings (Erblich, Lerman, Self, Diaz, & Bovbjerg, 2004, 2005).

Further complicating matters, the effects of cue-induced cravings on drug use outcomes have been mixed (Perkins, 2009; Shiffman et al., 2015). One reason for the mixed findings is the possibility that laboratory modeling of cue-induced craving lacks the external validity of cravings experienced during moments of affective valence, or when encountering ‘real-world’ triggers (e.g., passing by a favorite bar). In addition, longitudinal studies of the relationship between cue-induced cravings and drug use outcomes are uncommon. Some exceptions, however, have suggested that both the experience of cue-induced cravings under laboratory conditions and in the natural environment are predictive of drug use outcomes (Ferguson & Shiffman, 2014; A. Siegel, Korbman, & Erblich, 2017). More data are needed to further interrogate the predictive utility of conditioning models. Furthermore, conditioning models do not account for potentially important cognitive processes that may moderate stimulus-response relations.

Cognitive Models

Cognitive models of AUD risk eschew the simplistic stimulus-response relationships posited under the reinforcement and conditioning models and highlight how information-processing and expectancies modulate stimulus-response relationships. Bandura’s (1977) classic conceptualization of social-learning theory, focusing on principles such as reciprocal determinism and self-regulation as reflective processes, yields a richer model that integrates cognitive processing into drinking decisions. Early research focused on outcome expectancies as a driver of consumption. A classic study (Marlatt, Demming, & Reid, 1973) revealed that consumption was driven, not by the pharmacologic characteristics of alcohol, but rather by expectancies of drug effects. Numerous follow-up studies supported
these findings, and provided strong evidence against the loss-of-control-dominated disease model of AUDs, in favor of the modern, more nuanced maladaptive behavior conceptualization.

More fundamental information-processing models have gained considerable support recently. For example, Field and Cox (2008) highlighted the importance of implicit cognitive biases toward alcohol-related stimuli that prevail among heavy drinkers. A number of studies have found that heavier drinkers take longer to color-name alcohol-related words compared to neutral words on modified ‘alcohol-Stroop’ tasks (Hallgren & McCrady, 2013). Similarly, studies employing visual dot-probe tasks pairing probes with alcohol-related and neutral photos revealed that heavier drinkers exhibit biased attention toward alcohol-related stimuli, reacting more quickly to alcohol-congruent visual probes and slower to alcohol-incongruent probes. Finally, studies using implicit association tasks have shown that more favorable associations with alcohol are predictive of drinking outcomes (Luehring-Jones et al., 2016). Interplay between cognitive and conditioning models is also common. For example, Field, Mogg, Mann, Bennett, and Bradley (2013) suggest that biased processing of alcohol-related stimuli may increase focus on alcohol cues, resulting in increased cue-induced cravings. Emerging evidence supports such interplay (Manchery, Yarmush, Luehring-Jones, & Erblich, 2017). Taken together, these studies suggest that both elaborative ‘top-down,’ as well as more basic ‘bottom-up’ cognitive processes figure prominently in understanding AUD risk.

**Family Models**

Family models of AUDs focus on empirical evidence that child maltreatment, chaotic family environments, and genetics can play significant roles in the development of problem drinking. A recent meta-analysis concluded that adverse childhood experiences are major risk factors for numerous negative health and psychological outcomes, including problem drinking (Hughes et al., 2017). Similarly, low family socioeconomic status, poor parental modeling, inconsistent drinking rules, and high levels of family conflict, have all been shown to be predictors of adolescent alcohol use (Ryan, Jorm, & Lubman, 2010).

A highly influential approach in contemporary clinical practice is the family disease model. In this model, the entire family unit is considered suffering from an AUD. Family members are manifestations of the disease, assuming synergistic roles as ‘enablers,’ ‘lost children,’ ‘heroes,’ ‘scapegoats,’ and ‘mascots,’ some of which suffer from ‘co-dependence’ or other ancillary diagnoses. Although these approaches benefit from great intuitive appeal and have widespread acceptance among practitioners, there is limited evidence to support the existence of these constructs, and there is no systematic evidence to support the family disease model as a meaningful approach to understanding the initiation, maintenance, or treatment of AUDs (McCrady, Owens, Borders, & Brovko, 2014).

**Sociocultural Models**

Sociocultural models of AUDs focus on the role of structural and normative factors that contribute to drinking. Alcohol is heavily advertised in the U.S., with consumers portrayed as fun, attractive, and sophisticated. Consistent messaging about social and cultural benefits of alcohol consumption encourages drinking behavior (Berey, Loparco, Leeman, & Grube, 2017). On the other hand, many western European countries continue to have cultural norms encouraging moderate alcohol consumption in the context of meals (Kalinowski & Humphreys, 2016), resulting in more circumscribed and less risky alcohol use. Similarly, religious systems can guide the use of alcohol in a structured fashion, discouraging excessive consumption (Castaldelli-Maia & Bhugra, 2014).

Sociocultural models also focus on the importance of the built environment as a contributor to drinking behavior. As one example, Renalds, Smith, and Hale (2010) concluded that neighborhoods that are less ‘walkable’ and have less public space for leisure activities are more likely to have residents...
with AUDs. Along similar lines, the number of liquor stores in a neighborhood predicts problem drinking (Foster, Hooper, Knuiman, Trapp, & Wood, 2017), although the direction of causality in this relationship remains unclear. Enforcement of liquor laws predicts reduced drinking in urban areas (Jackson, Denny, & Ameratunga, 2014). Interestingly, however, key sociodemographic neighborhood factors, including residential mobility, crime, employment, and income have not consistently been associated with risk of problem drinking (Karriker-Jaffe et al., 2012). Finally, as indicated earlier, AUDs are more common in Native Americans than in other cultural groups; poor access to preventive services may be an important contributing factor to this disparity (Vaeth, Wang-Schweig, & Caetano, 2017). Problem drinking continues to be a pervasive problem across social and cultural boundaries.

An Integrated Biopsychosocial Model

The models described earlier have been instrumental in characterizing the way in which we think about risk of AUDs. The diverse, yet complementary approaches have propelled the field to progress from archaic moralistic and rigid disease models to a more nuanced conceptualization of an AUD as a highly complex maladaptive behavior, with influences from multifarious interrelated sources. Each of the models discussed is necessary, but by no means sufficient, to paint a complete picture of risk for problem drinking and AUDs. A more complete, albeit complex, theory of alcohol use must take all of these models into account. One useful framework for such an endeavor is an interactionist approach that highlights the interplay between dispositional factors and situational factors in understanding behavior [see Murtha, Kanfer, and Ackerman (1996) for an example]. In this framework, biological models, as well as family and sociocultural models, reflect broad dispositional contexts that create an underlying vulnerability to problem drinking. In contrast, reinforcement, conditioning, and cognitive models largely focus on situational factors, namely, how and why drinkers are motivated to consume, how they interact with alcohol, how they react to consumption, and how these interactions influence information processing (and vice versa). A unified interactionist theory of alcohol use is proposed here (see Figure 10.1), in which the complementary interplay between situational determinants of alcohol use (reinforcement, conditioning, and cognitive factors) interact with broad, contextual, ‘dispositional’ vulnerabilities, including genetics, impulsivity, built environment, and embedded familial and cultural structures in which individuals live. To be sure, the integrated model is complex, but it also places due emphasis on the diverse and interrelated biospsychosocial contributions to the understanding of alcohol use.

Interventions for Problem Drinking

For decades, the disease model of AUDs dominated the thinking associated with developing treatments. Perhaps the most famous regimen is Alcoholics Anonymous (AA). Bolstered by the rise of the disease model, AA focuses on the loss-of-control principle, and employs progressive steps with group support to encourage people to conceptualize their struggle as a lifelong disease process over which they have no control. Evidence for the efficacy of AA is decidedly mixed (Humphreys, Blodgett, & Wagner, 2014; Kelly, 2017). A Cochrane review (Ferri, Amato, & Davoli, 2006) concluded that there was a lack of unequivocal evidence for the efficacy of AA as a treatment modality for AUDs.

Common pharmacological interventions for AUDs include three FDA-approved medications: disulfiram, naltrexone, and acamprosate. Disulfiram inhibits ALDH, resulting in increased levels of toxic acetaldehyde in the body following alcohol consumption, resulting in aversive consequences. A systematic review (Jorgensen, Pedersen, & Tonnesen, 2011) concluded that although disulfiram is associated with short-term reductions in alcohol use, studies of long-term outcomes are lacking. Naltrexone, an opioid antagonist, and acamprosate, a GABA agonist, block the reinforcing effects of
drinking. Evidence for the efficacy of these drugs is promising (Rosner, Hackl-Herrwerth, Leucht, Lehert, et al., 2010; Rosner, Hackl-Herrwerth, Leucht, Vecchi, et al., 2010). Unfortunately, adherence to these medical treatments are problematic (Lohit, Kulkarni, & Galgali, 2016).

Psychological interventions for problem drinking are numerous. Driven by conditioning models, cue-exposure therapy was touted as promising intervention aimed at systematically counter-conditioning links between alcohol cues and craving responses. Unfortunately, evidence for its efficacy in reducing drinking, or even craving, is not compelling (Mellentin et al., 2017). Some (Tobena et al., 1993) have suggested that cue-exposures that occur in-vivo, rather than in a clinician’s office, would be more useful in reducing real-world cravings and alcohol use; data are needed to further evaluate this possibility.

Motivational interviewing (MI) is a well-known intervention that is founded in cognitive models of AUDs. MI helps drinkers resolve ambivalent feelings about modifying behavior and increasing motivation to quit. Research on the efficacy of MI is generally favorable but long-term maintenance of change remains challenging (DiClemente, Corno, Graydon, Wiprovnick, & Knoblach, 2017). Attention bias modification training (ABMT) is a computer-based intervention that trains drinkers to focus their attention away from alcohol-related stimuli. Although evidence for efficacy is mixed (Field & Cox, 2008; Luehring-Jones, Louis, Dennis-Tiwary, & Erblich, 2017), more research is needed to evaluate the utility of ABMT, either as a standalone treatment, or as an adjuvant to other pharmacological or psychological interventions (e.g., MI).
Finally, family models have informed the use of family therapies, including multidimensional family therapy (MDFT). MDFT is geared toward adolescent substance use and takes a holistic approach, including marshaling family support, and contemporaneously addressing other life stressors, such as academic and social challenges, delinquency, and family conflict. Evidence for the efficacy of MDFT in reducing alcohol consumption is promising (Liddle, Dakof, Turner, Henderson, & Greenbaum, 2008). Other family approaches employ the family disease model. As indicated earlier, however, although this treatment enjoys great popularity among both clinicians and patients, evidence for its utility is absent. Overall, numerous treatments for problem drinking are available, but evidence for efficacy and long-term adherence continue to present important challenges for clinicians.

Conclusions

This chapter outlined many considerations in current research on AUDs. AUDs continue to present a major public health problem in the U.S. and abroad. Increased research on the patterns, predictors, and consequences of alcohol use have led to the abandonment of moralistic and disease models of AUDs, toward a more complex and nuanced maladaptive behavior conceptualization that incorporates biological, psychological, and sociocultural factors that contribute to decisions to consume alcohol. A unified interactionist model that highlights the interplay between dispositional and situational factors will yield a richer and more complete understanding of the factors that contribute to problem drinking and the risk of developing AUDs.

References


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