Annu. Rev. Clin. Psychol. 2005. 1:493–523
doi: 10.1146/annurev.clinpsy.1.102803.144107

First published online as a Review in Advance on October 29, 2004

THE DEVELOPMENT OF ALCOHOL USE DISORDERS

Kenneth J. Sher, Emily R. Grekin, and Natalie A. Williams
Department of Psychological Sciences, University of Missouri-Columbia, Columbia, Missouri 65211-0001; email: Sherk@missouri.edu; grekine@missouri.edu; nawzpf@mizzou.edu

Key words alcohol dependence, alcohol abuse, etiology, personality, genetics

■ **Abstract** Pathological alcohol use is a complex and costly problem. This chapter focuses on recent developments in the etiology of alcohol use disorders. Literature is reviewed from the fields of epidemiology, genetics, personality, neuropsychology, parenting, and social influences. In addition, theoretical models that describe pathways to the development of alcohol use disorders are presented. Particular emphasis is given to ways in which genetic, environmental, psychopharmacological, and personological literatures can inform one another.

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INTRODUCTION

The etiology of harmful drinking is a vibrant research area informed by studies of epidemiology, genetics, socialization, social learning, personality, behavioral pharmacology, neuropsychology, and psychopathology. This review provides a broad overview of factors that lead to the development of pathological alcohol use. Although a discussion of treatment issues is beyond the scope of this paper, several recent reviews are available to interested readers who seek more information (Anton & Swift 2003, Berglund et al. 2003, Edwards et al. 2003).

DEFINING PATHOLOGICAL ALCOHOL USE

Alcohol Dependence

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association 1994) defines substance dependence as "a maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following symptoms occurring during the same 12-month period: (1) tolerance, (2) withdrawal, (3) the substance is often taken in larger amounts or over a longer period than intended, (4) a persistent desire or unsuccessful efforts to cut down or control substance use, (5) a great deal of time is spent in activities necessary to obtain the substance, use the substance or recover from its effects, (6) important social, occupational, or recreational activities are given up or reduced because of substance use, and (7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance." In contrast to DSM-III (American Psychological Association 1980) and earlier diagnostic criteria (e.g., National Council on Alcoholism 1972) that restricted the diagnosis of alcohol dependence to cases where there was clear evidence of physiological dependence (i.e., tolerance and/or withdrawal), the phenotype of alcohol dependence in DSM-IV is broader and physiological criteria are neither necessary nor sufficient for diagnosis.

Alcohol Abuse

According to DSM-IV, "the essential feature of Substance Abuse is a maladaptive pattern of substance use manifested by recurrent and significant adverse

consequences related to the repeated use of substances. There may be repeated failure to fulfill major role obligations, repeated use in situations in which it is physically hazardous, multiple legal problems, or recurrent social and interpersonal problems" (American Psychiatric Association 1994, p. 182). Within the DSM-IV, alcohol abuse is a residual category that is superseded by a current or past diagnosis of alcohol dependence.

The Abuse Versus Dependence Distinction

Although the abuse/dependence distinction is codified in the DSM and reified in clinical thinking, empirically, the distinction is somewhat suspect. Various psychometric approaches (e.g., factor analysis, item response theory analysis) fail to provide evidence for distinct abuse and dependence syndromes (e.g., Harford & Muthen 2001, Langenbucher et al. 2004, Martin et al. 1996). What these analyses do show is that some symptoms of alcohol use disorder appear to be more severe than others, but "severity" does not map cleanly onto the abuse/dependence distinction. Further evidence is needed to address the validity of the distinction as currently defined.

Binge Drinking

In recent years, a large number of studies have focused on heavy episodic or binge drinking. The term typically refers to consuming five or more drinks "in a sitting" or "in a row" (although some studies have used a cut-off of four or more drinks for women). Several researchers have argued that the term "binge" is misleading in that it is easily confused with the clinical concept of a binge or a bender (i.e., a period of two or more days of sustained heavy drinking) and does not take into account moderating factors such as body mass, duration of consumption, and tolerance. In 2004, the Advisory Council of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) defined the term "binge" as "a pattern of drinking alcohol that brings blood alcohol concentration (BAC) to 0.08 gram percent or above. For the typical adult, this pattern corresponds to consuming 5 or more drinks (male), or 4 or more drinks (female), in about 2 hours" (NIAAA, unpublished data). Thus, the NIAAA Council recommends that measures of excessive consumption be based upon blood alcohol levels but, at the same time, it provides rough guidelines regarding what types of drinking behavior might produce these elevated BACs.

EPIDEMIOLOGY

Over the past 25 years, four large-scale, population-based epidemiological surveys using structured diagnostic interviews have provided estimates of alcohol use disorders in the United States. These include the Epidemiologic Catchment Area Survey (Helzer et al. 1991, Robins & Price 1991); the National Comorbidity Survey (Kessler et al. 1994, 1997); the National Longitudinal Alcohol Epidemiologic Survey (Grant 1997, Grant & Pickering 1996, Grant et al. 1994b), and, most

recently, the National Epidemiologic Survey on Alcohol and Related Conditions (Grant et al. 2004b). Each of these major studies indicates very high past-year and lifetime prevalences of alcohol use disorders (AUDs) in the U.S. population (13.8% lifetime and 6.8% past year DSM-III in the Epidemiologic Catchment Area Survey; 23.5% lifetime and 7.7% past year DSM-III-R in the National Comorbidity Survey; 18.2% lifetime and 7.41% past year DSM-IV in the National Longitudinal Alcohol Epidemiologic Survey; and 8.46% past year in DSM-IV in the National Epidemiologic Survey on Alcohol and Related Conditions).

As shown in Figure 1, AUDs are more than twice as prevalent in men as in women, with larger sex differences in older cohorts. Moreover, across virtually all demographic strata (sex and race-ethnicity), and for both alcohol abuse and dependence, there is a monotonic decrease in the past 12-month prevalence of AUDs associated with age. This suggests either a marked developmentally limited condition that tends to remit in the third decade of life, or secular changes occurring in the prevalence of AUDs resulting in more-recently born cohorts having higher prevalences. A comparison of estimates from the National Longitudinal Alcohol Epidemiologic Survey (conducted in 1991–1992) and the National Epidemiologic Survey on Alcohol and Related Conditions (2001–2002) reveals an overall increase in the prevalence of alcohol abuse (from 3.03% to 4.65%) and a slight decrease in the prevalence of alcohol dependence (from 4.38% to 3.81%). Of particular interest, the strong age-gradient remained and was especially prominent for alcohol dependence. Prospective studies of heavy, episodic alcohol use in young adulthood

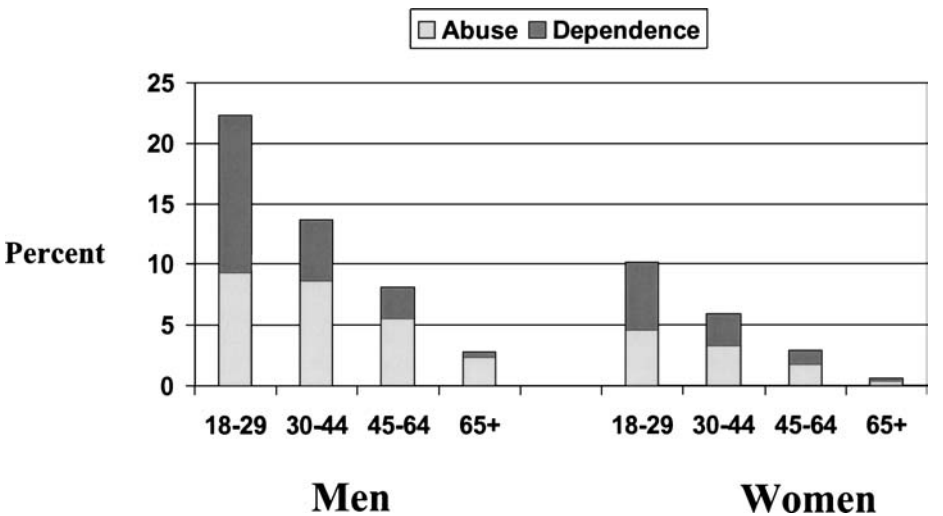

Figure 1 Past 12-month prevalence of alcohol abuse and dependence for men and women (as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, American Psychiatric Association, 1994). Data taken from the National Epidemiologic Survey on Alcohol and Related Conditions (Grant et al. 2004a).

(e.g., Chen & Kandel 1995, Schulenberg et al. 1996a) show similar patterns and suggest that the age-related decline in prevalence is primarily a developmental phenomenon and is not attributable to secular trends in consumption patterns (Grant 1997, Sher & Gotham 1999).

Trajectories of Alcohol Involvement

Cross-sectional and prospective studies of alcohol use and AUDs reveal a mean trend for alcohol involvement to increase during adolescence, peak during late adolescence and early adulthood, and then gradually decrease as adulthood progresses. Against these normative trends, there is increasing recognition of individual variability in course. Several recent empirical studies (e.g., Bennett et al. 1999; Chassin et al. 2002; Colder et al. 2002; Schulenberg et al. 1996a,b; Tucker et al. 2003) reveal the existence of distinct prototypical courses of alcohol involvement (e.g., a nonuser/stable low-user course, a chronic or high-user course, a "developmentally limited" course, and a later-onset course). Although developmentalists have embraced typological, trajectory-based approaches, alternative models for studying alcohol involvement across the life span have been proposed. For example, the "state-trait" model proposes that the tendency to develop an AUD is attributable to a stable trait that is indicated by the presence or absence of symptoms at multiple assessment occasions. The occurrence of an AUD at a particular point in time is a joint function of trait AUD and situational and developmental variables that tend to inhibit or promote the expression of the trait symptomatically at specific points in time. Both trajectory and state-trait perspectives suggest that current diagnoses and lifetime diagnoses (based on meeting of diagnostic criteria at some point in the individual's life) fail to capture developmental aspects of AUDs and the related dimension of chronicity, both of which may be important clinically and etiologically.

ETIOLOGIC MODELS OF ALCOHOL USE DISORDERS

A number of theories of alcoholism etiology have received empirical support. These models are not mutually exclusive and may represent multiple pathways into pathological alcohol involvement both between and within individuals. Four etiological models described by Sher & Slutske (2003) are highlighted here: (*a*) positive affect regulation, (*b*) negative affect regulation, (*c*) pharmacological vulnerability, and (*d*) deviance proneness.

Positive Affect Regulation

Most drinkers expect alcohol consumption to be a positive experience that directly produces pleasurable experiences (Goldman et al. 1999a). Drinking for positive reinforcement or "enhancement" (e.g., drinking "to get high" and "because it makes you feel good") (Cooper et al. 1992) is strongly associated with positive expectancies for enhancement as well as personality traits related to reward seeking (i.e.,

sensation seeking; Cooper et al. 1995) and appears to mediate these expectancy and personality effects on drinking outcome. Presumably, these motivations for positive reinforcement from alcohol are based on alcohol's neuropharmacological effects on the brain centers involved in basic reward mechanisms. For example, alcohol, like other drugs of abuse, has been shown to stimulate mesolimbic dopamine activity that is believed to be involved in basic reward mechanisms (Koob 2000). In addition, alcohol has been shown to increase activity in brain opioid systems (Gianoulakis 1996).

Negative Affect Regulation

One of the most enduring etiological perspectives on alcoholism is that AUDs develop because alcohol relieves negative affect. There is considerable evidence in support of this model, which has sometimes been referred to as "self-medication" or the tension-reduction hypothesis (Cappell & Herman 1972, Greeley & Oei 1999, Sayette 1999, Sher 1987). Many individuals hold strong expectations that alcohol is anxiety or stress reducing (Fromme et al. 1993). In addition, many people report that they drink to cope with negative affect (e.g., " to forget your worries") (Cooper et al. 1992). These coping motivations are strongly related to both alcohol consumption and problems (see Sher 1987) and appear to mediate the effects of negative affect and tension-reduction expectancies on drinking outcomes (Cooper et al. 1995).

At the same time, however, negative affective states, by themselves, are not strongly related to alcohol consumption or problems, and laboratory-based investigations of the effect of alcohol on negative affect have yielded confusing and contradictory evidence (Greeley & Oei 1999, Sayette 1999, Sher 1987, Stritzke et al. 1996). Reviewers of the literature on alcohol and stress have tended to conclude that negative affect regulation from drinking is highly conditional upon both intraindividual and situational factors (expectancies, genetics, stress-inducing environments, etc.) (Greeley & Oei 1999).

Pharmacological Vulnerability

The pharmacological vulnerability model (Sher 1991) proposes that individuals differ in their responses to the acute and/or chronic effects of alcohol and that these individual differences are etiologically relevant. The model itself incorporates several submodels that would appear to offer opposing predictions. For example, it can be hypothesized that some individuals are at risk for alcohol-related difficulties because they are especially sensitive to reinforcement (either positive or negative) and are therefore more likely to use alcohol because they get comparatively greater effect from it. Alternatively, it can be hypothesized that some individuals are relatively insensitive to reinforcement and thus must consume relatively high amounts of alcohol to achieve a desired effect, thereby exposing themselves to high blood alcohol levels and putting themselves at higher risk for physiological dependence.

Deviance Proneness

A final model to consider concerns what has been termed deviance proneness (Sher 1991). The key notion here is that excessive alcohol involvement comes about not because of attempts to regulate affective states or because of any particular vulnerability to alcohol as a drug, but because alcohol use is part of a more general, deviant pattern that has its roots in childhood and is attributable to deficient socialization. In a probing review of the early development of alcohol problems, Zucker et al. (1995) note consistency across extant longitudinal studies of alcoholism that begin in childhood. These studies highlight a number of common AUD correlates including a history of childhood antisocial behavior problems, childhood achievement problems, poorer childhood interpersonal relations, heightened activity in childhood, less parent-child contact, and inadequate parenting. Several explanatory models have been put forth to explain the relation between these correlates, early alcohol use, and other problem behavior. Perhaps the best known of these is problem behavior theory (e.g., Jessor & Jessor 1977), which posits that a range of personality, family, peer, and other environmental variables causally relate to involvement in a range of deviant behaviors including early alcohol use, illicit drug use, precocious sexual activity, and school failure. From this perspective, alcohol involvement is just one indicator of a broader factor of general deviance (Windle & Davies 1999). Although this model emphasizes deficient socialization as evidenced by decreased attachments to family, school, religious institutions, and involvement with deviant peers, personality and temperamental variables are often viewed as distal influences on these social, developmental processes (Petraitis et al. 1995). Consequently, genetic influences on personality are probably very relevant to these ostensibly social processes. Moreover, the same personality traits that put in place these problematic behavior trajectories (e.g., impulsivity) can also have proximal effects on alcohol use in the form of risky decisions about alcohol use (Sher et al. 1999).

GENETICS

The fact that AUDs are strongly familial has been long established (e.g., Cotton 1979); a number of twin and adoption studies published in the past 25 years have demonstrated that most of this familiality is attributable to genetic factors (see Heath 1995a and McGue 1999 for reviews). At present there appears to be an emerging consensus that genetic factors are important in both men and women, that multiple genes are responsible for the genetic effect, and that the nature of the genetic vulnerability remains to be discovered (McGue 1999, NIAAA 2000).

The search for specific genes that contribute to alcoholism risk is still at an early stage. However, recent advances in molecular genetics now allow us to scan many genetic loci for possible associations with alcoholism. Several chromosomal regions that appear to contain genes associated with alcoholism have been tentatively identified (Bierut et al. 2002, Reich et al. 1998), and it seems likely

that in the next several years, specific genes associated with alcoholism risk will be definitively identified and their mode of influence characterized.

One source of genetic influence on alcoholism risk appears to be mediated by individual differences in ethanol metabolism. First, variation in two of the genes (ADH2 and ADH3) responsible for the enzymes that break down alcohol into its metabolite, acetaldehyde, appear to be related to alcoholism risk in Asian populations (Reich et al. 1998). One recent review concludes, "it can now be regarded as firmly established that . . . [genetic variants] encoding faster metabolizing forms of ADH2 and ADH3 reduce the risk that carriers of these [genetic variants] will develop alcoholism" (NIAAA 2000, p. 176). Additionally, variation in one of the genes for aldehyde dehydrogenase (the enzyme that breaks down acetaldehyde, the toxic metabolite of alcohol, into acetic acid) is associated with alcoholism risk in Asians (e.g., Peng et al. 1999). Those with a specific variant of the gene are at very low risk for alcoholism (i.e., they are protected). The relevance of this effect to those of European ancestry, however, is unclear because the prevalence of this genetic variation is very rare in Caucasians. Still, this finding indicates one pathway through which specific genes can have an effect on alcoholism risk.

Much of the current research on specific alcoholism genes has focused on genes related to central nervous system functioning, where a number of candidate genes have been proposed (Diamond & Gordon 1995). These include genes associated with the gamma-aminobutyric acid/benzodiazepine receptor complex (important in anxiolysis), the *N*-methyl-D-asparate receptor (an excitatory glutamate receptor known to be extremely sensitive to alcohol in physiological doses), calcium channels, cyclic adenosine monophosphate, and G proteins. Additionally, there has been recent interest in genes regulating dopamine and serotonin transport (Dick et al. 2004, Lichtermann et al. 2000, Repo et al. 1999) and genes regulating enzymes important in the metabolism of dopamine and serotonin (e.g., catechol-*O*-methyltransferase and monoamine oxydase; Henderson et al. 2000, Snell et al. 2002). However, to date, no genes related to brain function have been firmly linked to alcoholism risk. Previous reports that one genetic variant of the dopamine D2 receptor is associated with alcoholism have proven controversial and are not widely accepted (McGue 1999, NIAAA 2000). However, there is currently much interest in the possible association between the D4 receptor gene and a range of impulse control disorders including alcohol misuse (Zuckerman & Kuhlman 2000).

Some have argued that clinical phenotypes such as the DSM AUD syndromes are too complex to use in the search for individual genes and that narrower behavioral phenotypes, or "endophenotypes," thought to be foundational to the clinical syndrome might be more useful for studying the effects of individual genes. Given the putative association of dopamine with drug or drug-cue-induced incentive value, arousal, and euphoria (Weiss & Koob 1991), genes related to dopamine receptor function are logical candidate genes to examine in studies of endophenotypes. This approach is well demonstrated by Hutchison and colleagues (Hutchison et al. 2002), who examined the potential moderating role of the D_4 dopamine receptor gene in relations between craving and responses to alcohol and

tobacco (or related cues) and found large individual differences in alcohol-related craving associated with different variants of the gene for both substances (but not in the reinforcing or stimulatory effects of either drug). These findings provide intriguing preliminary data on the relevance of specific genes for influencing alcohol (and other drug) seeking.

ENVIRONMENTAL INFLUENCES

Researchers have examined a variety of environmental factors that affect AUDs. These environmental influences include both distal (e.g., prenatal exposure to alcohol) and proximal (e.g., the effects of drinking contexts) factors that differ widely in their presumed mechanism of action.

Intrafamilial Influences

PRENATAL SUBSTANCE EXPOSURE Prenatal alcohol exposure has been implicated as a specific risk factor for the development of serious psychiatric disorders that are often comorbid with substance use disorders (Famy et al. 1998, O'Conner et al. 2002) in addition to fetal alcohol syndrome, a clinical syndrome characterized by growth retardation, facial dysmorphology, and central nervous system deficits (Larkby & Day 1997). Studies examining school-aged outcomes for children affected by fetal alcohol syndrome reveal a range of cognitive and behavioral problems that often persist and worsen as children mature, leading to high rates of antisocial behavior and the development of AUDs (Baer et al. 2003, Steinhausen et al. 1993, Streissguth et al. 1994). These types of deficits and related behavior problems are not restricted to children with fetal alcohol syndrome, but are also common in children prenatally exposed to alcohol without syndromal fetal alcohol syndrome (Streissguth et al. 1990, Testa et al. 2003, Willford et al. 2004).

The research on fetal drug effects suggests that prenatal alcohol exposure may operate to increase risk for substance use in two ways: (*a*) through a pathway related to general deviance proneness as indicated by findings suggesting a spectrum of antisocial outcomes related to fetal alcohol syndrome and (*b*) through a pathway related to pharmacological vulnerability. Specifically, research in animal models suggests that fetal exposure to alcohol may lead to the development of specific drug sensitivities and preferences (Abel et al. 1981, Dominguez et al. 1998, Osborn et al. 1998). These findings have particular relevance for our understanding of the etiology of alcohol problems in humans, given data implicating the role of drug sensitivity in the development of AUDs (Griesler & Kandel 1998, Newlin & Thomson 1990).

PARENTAL SUBSTANCE USE Although it is firmly established that children of alcoholic parents are at high risk for the development of alcohol use disorders in adolescence and adulthood, the extent to which there is an environmental component

to this familial risk is controversial. Genetic epidemiological studies indicate that about half of the liability to alcohol dependence is environmental (Kaprio et al. 2002, Rose et al. 2001, Viken et al. 1999). Interestingly, however, biometric models on twin data indicate that the majority of these environmental influences are unique (i.e., unshared). Thus, the role of shared familial environments on offspring drinking outcomes remains unclear. Notably, recent evidence suggests that shared environmental influences may be important in the context of gene/environment interactions. For example, Jacob et al. (2003) found that the environmental experience of growing up in a home with an alcoholic parent can have an etiological effect in the presence of a genetic diathesis for alcoholism.

Several environmental factors have been proposed to explain the intergenerational transmission of alcohol misuse. Children of alcoholic parents are often exposed to problematic parental drinking and may emulate this behavior as they get older (Ellis et al. 1997). However, adoption studies tend to show little evidence of modeling, at least as a main effect, since rates of alcoholism in the adoptive children of alcoholics do not appear to be elevated (Hopfer et al. 2003). Moreover, a study finding that risk for the development of alcohol use disorders in the children of alcoholic parents was unchanged over a five-year period regardless of whether parents were actively drinking suggests that the link between parental alcohol use and increased risk of alcohol use disorders in offspring is not accounted for by modeling alone (Chassin et al. 1999).

PARENTING PRACTICES Parents who abuse substances often demonstrate poor family management practices, such as lax and inconsistent discipline and inadequate supervision and monitoring, which are strongly linked with higher rates of internalizing and externalizing problems in children. In addition, poor parental monitoring puts children at risk for association with substance-using peers, which has been identified as a critical risk factor for early alcohol initiation and the development of alcohol problems (Hawkins et al. 1992). At the family level, parental substance abuse is associated with greater family conflict, hostility, marital problems, and disruption of family rituals (Jacob & Johnson 1997).

Moreover, considerable evidence suggests a link between parenting and offspring antisocial behavior, including the early initiation of alcohol use and the development of alcohol use disorders in adolescence and early adulthood. Investigations in this area have focused on both general parenting style and specific parenting practices in relation to the development of conduct problems and antisocial behavior. In general, a parenting style characterized by high warmth and behavioral control (e.g., authoritative parenting) appears to protect adolescents from negative outcomes such as antisocial behavior and the development of alcohol use problems (Adalbjarnardottir & Hafsteinsson 2001, Patock-Peckham et al. 2001).

Finally, research suggests that parental communications regarding alcohol consumption norms that deter alcohol use are more likely to be internalized by

children and adolescents within the context of a warm and supportive parent-child relationship (Brody et al. 2000, Fletcher & Jefferies 1999, Johnson & Pandina 1991, Kosterman et al. 2000). Despite the growing influence of peers and declining influence of parents on children's behavior throughout adolescence, some research suggests that the parent-child relationship and certain parenting variables, such as warmth, may moderate the extent to which adolescents are influenced by association with substance-using peers (Marshal & Chassin 2000).

In contrast, negative parenting is associated with adjustment problems in children and adolescents. Harsh and inconsistent discipline in response to oppositional behavior in early childhood promotes further undesirable child behavior, and establishes a pattern of interactions marked by increasingly hostile and coercive behaviors (Ary et al. 1999). This parenting style is associated with earlier initiation of alcohol use, as well as with other predictors of alcohol use disorders (e.g., conduct problems, poor self-regulatory skills, and antisociality) (Beyers et al. 2003, Kumpfer & Bluth 2004, Repetti et al. 2002).

Peer Influences

The adolescent substance abuse literature has consistently shown that adolescents and young adults resemble their peers with respect to substance use. Researchers have explained this similarity through two processes: socialization or causation and selection (Andrews et al. 2002). Socialization occurs when an individual's alcohol use is shaped by influence from the peer group. In this case, affiliations with substance-using peers may encourage greater involvement with alcohol through various mechanisms, including social learning, peer group influence, modeling, and social facilitation (Deater-Deckard 2001, Fergusson et al. 2002). Conversely, the process of selection occurs when adolescents seek affiliation with peers who display similar patterns of substance use or deviant behavior. Research suggests that adolescents from disadvantaged, dysfunctional, or disturbed environments, or those with a predisposition toward antisocial behavior, are most likely to become involved with deviant peer groups through the selection process (Fergusson et al. 1999). In either case, the proportion of peer associates who use alcohol and engage in deviant behavior is a powerful predictor of the development of alcohol abuse and dependence in adolescence (Fergusson et al. 2002, Windle 2000).

ALCOHOL AND PERSONALITY

The relationship between personality, alcohol use, and AUDs is complex. Research over the past 50 years has consistently failed to find a particular constellation of personality traits that uniquely predicts alcoholism. However, although studies do not support the existence of a specific "alcoholic personality," a variety of personality traits have been reliably associated with both the development and the manifestation of alcohol use disorders. We focus here on three broad personality

dimensions that are frequently discussed in the alcoholism literature—neuroticism/negative emotionality, impulsivity/disinhibition, and extraversion/sociability—and that, broadly considered, correspond to "Big Three" models such as those proposed by Eysenck (1994) and Tellegen (1994).

It is important to note that traits that characterize clinical alcoholics (individuals meeting diagnostic criteria for alcoholism) often differ from those that characterize prealcoholics (nonalcoholic individuals who later become alcoholic) and children of alcoholics (individuals at genetic risk for alcoholism). Some of this discrepancy results from the fact that alcoholism tends to cause personality change (e.g., increases in anxiety and depression; Sher et al. 1999).

Neuroticism/Negative Emotionality

A growing body of evidence suggests a relationship between neuroticism/negative emotionality and clinical alcoholism. For example, several researchers have found high rates of anxiety and depression among alcoholic samples (Gratzer et al. 2004, Hasin & Nunes 1997, Weitzman 2004). In addition, individuals with AUD diagnoses tend to score higher on self-report measures of neuroticism (Jackson & Sher 2003, McCormick et al. 1998, Prescott et al. 1997) and negative emotionality (Martin et al. 2000, McGue et al. 1999, Swendsen et al. 2002) than do nonalcoholic controls.

The relationship between neuroticism/negative emotionality and future alcoholism, however, is unclear. Zimmerman et al.(2003) found that baseline anxiety disorders predicted the subsequent onset and course of alcohol use disorders in a prospective community sample. In addition, Elkins et al. (2004) found high rates of negative emotionality among nonalcoholic adolescents with a parental history of alcoholism. Similarly, Merikangas et al. (1998) found high rates of alcoholism in relatives of panic disorder patients, suggesting a common, underlying etiology between the two disorders. Other studies, however, indicate that neuroticism/negative emotionality is a consequence, rather than a cause, of alcoholism. For example, Sutherland (1997) found a decrease in neuroticism over time among recovering alcoholics, suggesting that neuroticism is the result of long-term, problematic alcoholic use. In addition, Slutske et al. (2002) found that negative emotionality accounted for an extremely small proportion of the genetic variation in risk for alcoholism (Sher et al. 2000) Additional prospective, high-risk studies are needed to clarify the relationship between negative emotionality and the development of alcoholism.

Impulsivity/Disinhibition

Impulsivity and disinhibition have consistently been associated with clinical alcoholism. Individuals who meet AUD criteria score high on both self-report (Baker & Yardley 2002, Bennett et al. 1999, McGue et al. 1997, Trull et al. 2004) and laboratory (Kollins 2003; Petry 2001, 2002) measures of these traits. In addition,

alcoholics tend to exhibit high rates of Cluster B (dramatic/impulsive) personality disorders, such as antisocial personality disorder (Bucholz et al. 2000, Kanzler & Rosenthal 2003) and borderline personality disorder (Rohde et al. 2001; Trull et al. 2000, 2004). Additionally, recent evidence suggests that the genetic variance in behavioral undercontrol accounts for a significant proportion of the genetic variance in alcohol dependence (Slutske et al. 2002). It should be noted, however, that the strength of the alcoholism/impulsivity relationship might depend on the definition of impulsivity being employed (Whiteside & Lynam 2003). Moreover, some recent studies suggest that alcoholism is no longer related to disinhibition after controlling for drug use (McGue et al. 1999) and psychopathy (Whiteside & Lynam 2003). Future studies with large sample sizes and adequate control measures are needed to test these hypotheses.

Disinhibition and impulsivity have also been related to future alcoholism. For example, offspring of alcoholics have been found to exhibit high levels of externalizing and disinhibited behaviors (Loukas et al. 2001, 2003; Puttler et al. 1998). This relationship is particularly strong for offspring with multiple alcohol-abusing relatives, suggesting that the alcoholism/disinhibition association may be, in part, heritable. Perhaps more directly relevant are prospective studies that indicate that individuals with disinhibited traits are at heightened risk for the development of alcoholism. For example, Sher and colleagues (Sher et al. 2000) found that disinhibited traits prospectively predicted alcohol use disorders in a high-risk sample. Other recent studies have yielded similar findings (Caspi et al. 1998, Schuckit 1998).

Extraversion/Sociability

Although most studies have found that clinical alcoholics and controls exhibit similar levels of extraversion/sociability (Barnes 1983, Cox 1987, Sher et al. 1999), a relationship has been found between extraversion/sociability and drinking onset (Hill et al. 2000, Hill & Yuan 1999) as well as between extraversion and alcohol consumption among nonalcoholics (Cook et al. 1998, Flory et al. 2002, Grau & Ortet 1999, Martsh & Miller 1997). In addition, two recent studies have found that extraversion prospectively predicts the development of alcohol problems among community samples (Kilbey et al. 1998, Wennberg 2002). It is important to note, however, that many studies of extraversion and alcohol problems/consumption have yielded negative findings (e.g., LoCastro et al. 2000, Stacy & Newcomb 1998). In addition, other studies suggest that the extraversion/alcohol problem relationship may be stronger for women than for men (Prescott et al. 1997). There are several potential reasons for these mixed findings. First, it is possible that extraverted individuals are prone to heavy drinking but as alcohol dependence develops, levels of sociability decrease. Alternatively, it is possible that characteristics of extreme extraversion are more reflective of disinhibition than of sociability. More research is needed to verify these hypotheses.

EXECUTIVE DYSFUNCTION AS A RISK FACTOR FOR ALCOHOL USE DISORDERS

For the past 20 years, there has been intense interest in the idea that some cognitive functions related to executive control are important risk factors in the development of AUDs. Individuals with a family history of alcoholism have been shown to perform more poorly than their peers on tests of planning, abstract conceptualization, conceptual shifting, and psychomotor functioning (Corral et al. 2003, Najam et al. 1997, Nigg et al. 2004, Nixon & Tivis 1997, Poon et al. 2000), although null findings have also been reported (Bates & Pandina 1992, Leonard & Eiden 2002). Moreover, recent laboratory studies have found reduced P300 amplitudes among children of alcoholic parents compared with those of offspring of nonalcoholics (Carlson et al. 2002, Hill et al. 1999). The causes of the executive functioning deficits in children of alcoholic parents are unknown, although it seems likely that genetic mechanisms are important (Carlson et al. 2002, Corral et al. 1999, Porjesz et al. 2002).

Some studies have found that executive functioning deficits are more common among individuals with a family history of both alcoholism and antisocial personality disorder (Poon et al. 2000), which suggests these deficits may be associated with a specific subtype of alcohol dependence. Given that executive deficits are implicated in a range of disorders of childhood and adulthood, especially those that are risk factors for AUDs, such as attention deficit hyperactivity disorder and conduct disorder (see section below on Child Psychopathology and Developmental Precursors), the specificity of executive deficits, if any, to AUD beyond comorbid conditions needs to be determined. Although status as a child of alcoholic parents has been associated with impaired cognitive performance, no studies to date have examined whether executive functioning mediates the relationship between a family history of alcoholism and the development of an AUD.

INDIVIDUAL DIFFERENCES IN THE EFFECTS OF ALCOHOL

Central to the pharmacological vulnerability model is the idea that there are important individual differences in alcohol effects and that these individual differences tend to promote or inhibit risk for AUDs. Twenty-five years ago, Marc Schuckit (1980) found that men with a positive family history of alcoholism showed a less-intense subjective response to a moderate dose of alcohol in comparison to men without a positive family history. This finding was later replicated in Shuckit's lab (Schuckit 1984) and in other labs (Heath & Martin 1992, McCaul et al. 1991, Morzorati et al. 2002, Moss et al. 1989, Neale & Martin 1989, Pollock et al. 1986, Pollock 1992). Importantly, Schuckit & Smith (1996) demonstrated the potential etiological significance of these findings by showing that over a 10-year follow-up period, low subjective response to alcohol predicted the onset of alcohol

dependence in a young adult sample (Schuckit 1995; see also Volavka et al. 1996). It should be noted, however, that a number of contradictory findings (Conrod et al. 1997b, de Wit & McCracken 1990, Kaplan et al. 1988, McCaul et al. 1990, Morzorati et al. 2002, Nagoshi & Wilson 1987) and null findings (e.g., Bauer & Hesselbrock 1993, Heath et al. 1999, Vogel-Sprott & Chipperfield 1987) have been reported in this area.

Other individual-difference studies examine the stress-response-dampening effects of alcohol (i.e., the effect of alcohol on response to a discrete stressor) (Sher 1987). In general, these studies find a pattern of *increased* sensitivity to alcohol in individuals with a family history of alcoholism (Carlson et al. 2002, Finn & Pihl 1987, Finn et al. 1990, Levenson et al. 1987). This response has been associated with other correlates of alcohol misuse (e.g., sensation seeking) and seems to be particularly strong among individuals with a multigenerational family history of alcoholism (Conrod et al. 1997a, Finn et al. 1992).

Another body of literature has examined individual differences in subjective intoxication. For example, using an innovative intravenous approach to administering alcohol, Morzorati et al. (2002) found that individuals with a positive family history for alcoholism report feeling more intoxicated than do controls during the period between baseline and the beginning of the clamping interval (when ethanol infusion was controlled to maintain a constant blood alcohol concentration). However, during the clamping interval (that is, while breath alcohol concentration is held constant for an extended period), family history positive subjects' reports of intoxication were not significantly different from those of controls. Taken together, these data on familial risk for alcoholism and ethanol response suggest that those with a family history of alcoholism may be more likely to experience enhanced reinforcement from alcohol early in the course of intoxication. Later on in the drinking episode (when blood alcohol levels are either stable or decreasing and the profile of reinforcement and punishment shifts), those at high risk seem either to become less sensitive to alcohol than do those at lower risk or at least to not differ from them in alcohol response.

ANTECEDENT AND COMORBID PSYCHOPATHOLOGY

Child Psychopathology and Developmental Precursors

Contemporary theorists (Clark & Winters 2002, Vanyukov & Tarter 2000) propose that childhood emotional and behavioral regulation difficulties and adolescent alcohol problems share common causes such as behavioral dysregulation or inhibition deficits, genetic factors, or environmental influences (Clark & Winters 2002, DB Clark et al. 2002). Accumulating evidence that suggests childhood internalizing and externalizing problems mediate the relation between certain variables (e.g., parental alcohol use) and the development of alcohol use and abuse in adolescence and adulthood is consistent with this conceptual framework (Colder & Chassin 1997, Loeber et al. 1995, Reinherz et al. 2000).

CONDUCT DISORDER Extant literature has established that antisocial behaviors in childhood, including oppositional defiant disorder and conduct disorder (CD), as well as subsyndromal antisociality, are key components in the developmental pathway to later substance use disorders (DB Clark et al. 2002). Elevated rates of CD and oppositional defiant disorder are found in clinical and community samples of adolescents with substance use disorders (Clark et al. 1997). In addition, prospective longitudinal studies have demonstrated that childhood antisocial behaviors precede and predict adolescent involvement with alcohol and are strongly associated with the development of alcohol problems (Clark et al. 1998), and behavior genetic investigations show a strong genetic correlation between the two disorders (Slutske et al. 1998). Some research suggests that the association between antisocial behaviors and problematic alcohol involvement is particularly salient in male children with a family history of paternal substance use disorders (Clark et al. 1999). Difficulty with executive functioning has been hypothesized to be a critical developmental precursor to disruptive behavior problems and subsequent alcohol use problems (Barkley 1997, Giancola & Tarter 1999, Nigg 2001). Studies examining this relationship have provided mixed empirical support for this hypothesis (C Clark et al. 2000, 2002; Riggs et al. 2003), with recent data suggesting that executive function deficits (e.g., poor self-regulation, difficulty resisting short-term gratification to obtain long-term goals) affect high- and low-risk children in different ways (Nigg et al. 2004).

ATTENTION DEFICIT/HYPERACTIVITY DISORDER Studies of adolescents receiving treatment for substance use disorder have found attention deficit/hyperactivity disorder (ADHD) rates to be as high as 30% (Molina et al. 2002). Although significant associations have been reported between ADHD and substance use in both cross-sectional and prospective studies, data from several studies indicate that the significant relation of ADHD to substance use problems is greatly attenuated, if not eliminated, after controlling for co-occurring conduct problems (Beiderman et al. 1998, Disney et al. 1999). Additionally, the relationship between ADHD and alcohol use and dependence is mediated by level of cognitive functioning, such that individuals with lower cognitive ability are more likely to use and abuse alcohol as a coping strategy to deal with the hyperactivity characteristic of ADHD (Dawes et al. 2000, Span & Earleywine 1999).

Findings that ADHD and CD occur together in 30% to 50% of cases have led researchers to examine other interpretations for the relationship between ADHD and substance use problems. These explorations have resulted in some evidence for the interactive effects of these two disorders on substance use and abuse; however, results indicate that this effect may be limited to tobacco and illicit drug use as opposed to alcohol use problems (Flory et al. 2003, Molina et al. 2002). Thus, although individuals with comorbid ADHD and CD had the highest rates of some types of substance use in young adulthood, more research is needed to determine if comorbidity between ADHD and CD is predictive of higher rates of alcohol use and dependence independent of substance use disorders more generally.

MOOD AND ANXIETY DISORDERS Although somewhat mixed findings have been reported on the temporal association between depression and alcohol problems (Deas & Thomas 2002), depressive symptomatology appears to be one aspect of a larger profile of risk for the development of alcohol use disorders in adolescents. In a recent review of community studies examining psychiatric comorbidity with substance use, Armstrong & Costello (2002) reported high rates of comorbidity between substance use disorders and depressive symptomatology (median = 18.8%) and anxiety disorders (median = 16.2%). Prospective evidence indicates that anxiety symptoms in childhood or early adolescence predict later substance use and dependence (Rohde et al. 1996), and clinical studies indicate that anxiety symptoms often precede the onset of substance use disorders (Deas-Nesmith et al. 1998).

Adult Psychopathology and Comorbidity

In the National Comorbidity Survey (Kessler et al. 1997), lifetime alcohol dependence was robustly associated with higher rates of lifetime diagnoses in both men and women of all anxiety, affective, drug, and antisocial behavior disorders surveyed. Consistent with data from the Epidemiologic Catchment Area Survey (Helzer & Pryzbeck 1988, Helzer et al. 1991), the disorders most strongly associated with alcohol dependence were mania, drug use disorder, and antisocial personality disorder. However, comorbidity with alcohol abuse was less consistent, and those relations that were significant were less generalizable across gender.

Most National Comorbidity Survey participants with an AUD had at least one of the disorders surveyed. Establishing whether the comorbidity with AUD was potentially causal, consequential, or attributable to some common etiological process is a major area of current interest. In order to characterize the nature of comorbidity better, many investigators have attempted to classify AUDs as either primary or secondary (Schuckit 1985) on the basis of the sequencing of onset of AUDs and of co-occurring conditions. That is, when an AUD occurs prior to a comorbid condition it is considered primary; when it occurs subsequent to a comorbid condition it is considered secondary. In the National Comorbidity Survey, alcohol dependence was typically found to be secondary to other comorbid disorders (Kessler et al. 1997). This is not surprising because, by definition, the onset of some disorders (e.g., conduct disorder) occurs before mid-adolescence. Moreover, prior lifetime disorders tended to positively predict the onset of lifetime alcohol dependence across all disorders assessed. However, prior lifetime disorders did not consistently predict the onset of alcohol abuse, and when they did predict the onset, the patterns were difficult to interpret (Kessler et al. 1997). It seems likely that these seemingly anomalous results represent a statistical artifact of segregating out a mild form of AUD (abuse) from more severe forms (dependence). Thus, it might be useful to simply consider broadband diagnosis (abuse and/or dependence combined) and narrow-band dependence alone.

Unfortunately, there are few prospective studies of AUD comorbidity that would help to unravel direction of causality. Those studies that do exist cover either early

periods of development (e.g., Costello et al. 1999), where participants have yet to pass through much of their period of risk for disorders, or begin later in development (e.g., Kushner et al. 1999), when extensive symptomatology is already in place.

To date, population-based epidemiology of comorbidity between AUDs and psychopathology has focused on the DSM Axis I disorders, with the important exception of antisocial personality disorder (and its childhood precursors). Although the database for examining AUD/Axis II comorbidity is not well developed, existing studies (Grant et al. 2004a, Sher et al. 1999, Trull 2004) suggest a strong relation between AUDs and both antisocial and borderline personality disorder, two disorders characterized by disinhibition. Other personality disorders associated with AUDs in multiple studies include borderline, histrionic, narcissistic, and avoidant. It is possible that much of the comorbidity among AUDs and Axis 1 disorders is mediated via personality disorder or closely related traits. Moreover, common third variables might influence both alcohol involvement and comorbid conditions. For example, behavior-genetic investigations suggest that common genetic vulnerabilities are partially responsible for comorbidity between alcohol dependence and nicotine dependence (True et al. 1999), conduct disorder (Jang et al. 2000, Slutske et al. 1998), and to a lesser degree, anxiety disorders (Kendler et al. 1995, Merikangas et al. 1998) and depression (Prescott et al. 2000).

ALCOHOL OUTCOME EXPECTANCIES

Alcohol outcome expectancies can be defined as beliefs that people have about the affective, cognitive, and behavioral effects of drinking alcohol (Goldman et al. 1987). Varying psychometric methods (e.g., exploratory and confirmatory factor analysis, multidimensional scaling) have been employed in the development of self-report expectancy measures designed to assess particular types of beliefs about drinking and to examine their relations with alcohol use and problems (e.g., Fromme et al. 1993, Goldman et al. 1991). Although the specific content of empirically derived factors varies across methods and measures, factors related to tension reduction, social and/or sexual facilitation, and enhanced cognitive or motor performance have been replicated across studies. Goldman et al. (1999) suggest that outcome expectancies can be categorized along three basic dimensions: (*a*) positive versus negative expected outcomes (e.g., increased sociability versus increased aggressiveness); (*b*) positive versus negative reinforcement (e.g., social facilitation versus tension reduction); and (*c*) arousal versus sedation (e.g., stimulant versus depressant effects). A growing body of research utilizing implicit assessment of outcome expectancies has also demonstrated associations with alcohol use (Palfai & Wood 2001, Stacy 1997, Wiers et al. 2002).

The bulk of outcome expectancy research is cross-sectional, with consistent demonstration of robust associations between outcome expectancies and measures of alcohol use and problems across drinking patterns ranging from abstention to alcohol dependence and among diverse subject populations including adolescents, college students, and adults (e.g., Goldman et al. 1999b). Prospective studies have

demonstrated that outcome expectancies predict alcohol use onset, subsequent use, and problematic use (Christiansen et al. 1989, Newcomb et al. 1988, Sher et al. 1996, Smith et al. 1995, Stacy et al. 1991). Contemporary research is increasingly focusing on the relation of expectancies to more distal risk factors such as genetics (McCarthy et al. 2000) and personality (Henderson et al. 1994, Sher et al. 1991), based on the hypothesis that expectancies represent a common final pathway of diverse biopsychosocial influences on alcohol use and misuse (Goldman et al. 1999a, Sher 1991).

PERSISTENCE AND DESISTENCE

The epidemiological data reviewed above indicate strong normative trends toward decreasing levels of alcohol involvement during the third decade of life, and the data on individual variation in life course trajectories indicate that although many cases of early onset AUDs "mature out," others persist (and some smaller percentage have later onsets). In order to understand the nature of AUDs over the life course, it is important to understand not only what factors lead to the development and maintenance of alcohol problems but also what factors lead to their reduction. The developmental gradient in the prevalence of AUDs provides a strong clue, but what is it about early adulthood that leads to normative reductions in alcohol-related problems?

The reduction in AUDs and other alcohol-related behaviors that tends to occur in the midtwenties and continue into later adulthood is thought to reflect, in large part, a maturational process in which individuals move into adult roles that are incompatible with drinking (Miller-Tutzauer et al. 1991, Newcomb & Bentler 1988). Such role incompatibilities are occasioned by developmental transitions such as finding a mate, beginning a career, and becoming a parent (Bachman et al. 1984, 2002; Havighurst 1972). Although most of these reductions in alcohol involvement appear to be a function of situational variables that directly inhibit a lifestyle characterized by heavy alcohol consumption in combination with exits from environments that promote heavy drinking (e.g., college, the military), it is clear that this "maturing out" effect is moderated by individual difference variables such as personality as well as drinking experiences that promote problem recognition (e.g., Sobell et al. 2000). Given the very high rates of AUDs that remit during the third decade of life, understanding chronic forms of AUDs requires a more complete understanding of normative mechanisms that tend to inhibit the continuation of problematic drinking patterns.

CONCLUSIONS

AUDs are prevalent disorders in our society and show a strong age gradient with typical onset during late adolescence. Although many individuals who experience AUDs appear to "mature out" of them, a significant number show more life-course

persistent forms. A number of etiological factors have been associated with the development of AUDs. Genetic factors appear to be both specific to alcohol as a drug (e.g., ethanol metabolism) and nonspecific (e.g., relating to the incentive value of various experiences, self-regulation, and negative affectivity). Although it has been hard to identify specific socializing experiences that relate to risk for alcoholism, there is evidence that some types of family experiences (e.g., drinking in the home and certain types of parenting) promote the development of AUDs, at least in vulnerable individuals.

It is becoming increasing clear that alcohol misuse and AUDs have their roots in childhood and are closely associated with a range of both internalizing and externalizing childhood disorders. These disorders probably promote the development of AUDs via multiple mechanisms, including negative affect regulation and deviance proneness. The strong association between AUDs and other psychological disorders continue into adulthood, and understanding comorbid processes may be particularly important for understanding more chronic versus benign courses.

The study of AUD etiology is illustrative of how genetic, environmental, psychopharmacological, and personological approaches can inform each other. We are approaching the point where we can see the potential influence of individual genes on specific risk mechanisms and how these mechanisms relate to each other and are moderated by early experience and situational constraints on alcohol-involved lifestyles. A fuller understanding of these mechanisms will facilitate the development of a broad range of intervention strategies in the areas of prevention and treatment.

ACKNOWLEDGMENTS

This study was supported by National Institute on Alcohol Abuse and Alcoholism grants R37AA7231, R01AA13987, and T32AA13526 to Kenneth J. Sher, and P50 AA11990 to Andrew C. Heath.

The *Annual Review of Clinical Psychology* is online at http://clinpsy.annualreviews.org

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