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Pathological alcohol involvement: A developmental disorder of young adulthood

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Abstract
In 1987, we began a longitudinal study of the offspring of alcoholic parents and have been following this group of young adults from their freshman year in college throughout their transition into later young adulthood. The goal of this review is to highlight some of the findings we consider most important and relevant to the development of pathological alcohol involvement in young adulthood. Courses of pathological alcohol involvement in young adulthood are outlined. Predictors of both the development and course of pathological alcohol use in young adulthood are also addressed, including family history of alcoholism, personality, alcohol use motivations, and role transitions. While certainly a problem in its own right, pathological alcohol involvement can also affect the attainment of important life tasks and success in various life roles. Consequently, we also examine the effects of pathological alcohol involvement on later role transitions and role attainment. Finally, prevention, policy, and treatment issues surrounding this stage of life are discussed.

Pathological alcohol involvement, including frequent heavy drinking and alcohol use disorders (AUDs [alcohol abuse and dependence]), appears to be primarily a disorder of late adolescence and young adulthood. Based on data from the National Longitudinal Alcohol Epidemiological Survey, the highest prevalence of AUDs (*Diagnostic and Statistical Manual of Mental Disorders*, fourth edition [DSM-IV], American Psychiatric Association [APA], 1994) occur in the 18–29 years age group (Grant, Harford, Dawson, Chou, Dufor, & Pickering, 1994). As illustrated in Figure 1, the rate for AUDs in the youngest group (age 18–29 years) was more than twice that of individuals in the next oldest group (age 30–44 years), which, in turn, was more than twice as high as the next oldest group (age 45–64 years). Also, the overall, strong negative association with age holds true across both abuse and dependence diagnoses and across sex and the racial groupings used in the study (with the possible exception of black females, who show very low rates overall and a maximum prevalence in the 30 to 44-year-old period). Given reports that 21 years is the median age of onset for DSM-III (1980) lifetime AUDs (Christie, Burke, Regier, Rae, Boyd, & Locke, 1988) and frequency of heavy drinking peaks around ages 20–21 years (Johnston, O'Malley, & Bachman, 1996), a more developmentally sensitive reporting of AUD prevalence (e.g., a 20 to 24-year-old cohort) would likely yield even higher rates associated with the late adolescent–early adulthood transition. When these cross-sectional findings are considered with longitudinal findings that most young adults "mature out" of excessive drinking patterns (e.g., Chen & Kandel, 1995; Temple & Fillmore, 1985–1986), it appears that many, if not the majority, of cases of lifetime AUDs are "developmentally limited" (Zucker, 1987).

This is not to say that a developmentally limited form of pathological alcohol involvement in young adulthood is not damaging.

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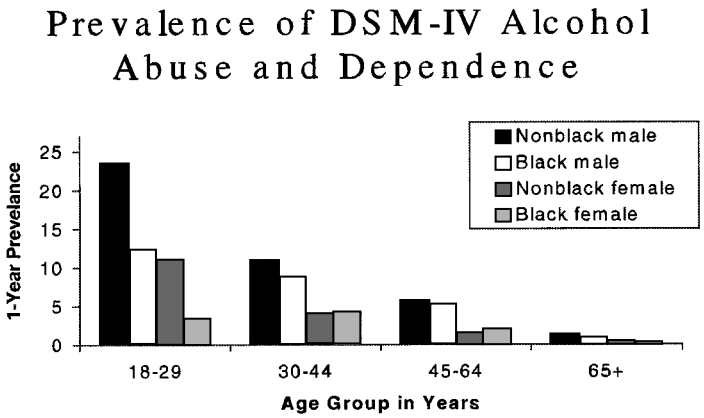


Figure 1. From "Prevalence of DSM-IV Alcohol Abuse and Dependence," by B. F. Grant, T. C. Harford, D. A. Dawson, P. Chou, M. Dufour, and R. Pickering, 1994. *Alcohol, Health, and Research World, 18*, pp. 243–248.

Such problem drinking is associated with increased risk for acute negative consequences such as traumatic injury, rape, and risky sexual behavior resulting in unplanned pregnancy, sexually transmitted diseases, or HIV infection (Commission on Substance Abuse at Colleges and Universities [CASA], 1994; Cooper, 1992; Parker, Harford, & Rosenstock, 1994; Wechsler, Davenport, Dowdall, Moeykens, & Castillo, 1994). Also, there may be long-term consequences of AUDs on educational and occupational attainment (Turnbull, George, Landerman, Swartz, & Blazer, 1990). For some young adults, pathological alcohol involvement may presage chronic drinking problems and serious social and medical problems across the life course. Types of early-onset AUDs are associated with antisociality and aggression (e.g., Buydens–Branchey, Branchey, & Noumair, 1989; Zucker, Fitzgerald, & Moses, 1995) as well as negative affect and an internalizing style (Mezzich, Tarter, Kirisci, Clark, Buckstein, & Martin, 1993). In general, alcoholics with an onset before age 20 years have more antisocial personality traits, drug abuse, bipolar and panic disorders, and suicide attempts (Roy et al., 1991).

Developmental psychopathology (Cicchetti, 1984, 1993; Rutter & Garmezy, 1983; Sroufe & Rutter, 1984) represents an important area of theory and research that can be used to help understand young adult pathological alcohol involvement. This approach examines how developmental tasks associated with the adolescent–early adulthood transition are related to risk factors and how they may modify the expression of a disorder. In addition, developmental psychopathology stresses the importance of studying both stability and instability of processes over time (Lerner et al., 1996), such as trajectories of pathological alcohol involvement in young adulthood. "Age-indexed variations in susceptibility to environmental hazards" (Rutter, 1996, p. 212) appear to be particularly important in understanding the development and persistence of pathological alcohol involvement at this stage of life as some risk factors have more or less impact depending on the age of the individual. For example, parents and peers are more influential on drinking in young adulthood than in later adulthood (e.g., Curran, Stice, & Chassin, 1997; Hawkins, Catalano, & Miller, 1992; Scheier & Botvin, 1997).

Cicchetti's argument that "any psychopathology can be conceived as a distortion or deviation in the normal ontogenetic process" (1993, p. 475) implies that pathological alcohol involvement, including AUDs, can be seen as a disorder of development. Alcohol plays a large role in American society, and even children have elaborate beliefs about the effects of alcohol (Zucker & Fitzgerald, 1991). Most adolescents and young adults develop schemas about the uses and effects of

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alcohol, have early drinking experiences in family situations, and experiment more heavily with alcohol during late high school and the college years. Their alcohol use then typically decreases during the 20s. However, many young adults deviate from this normative process and develop health-damaging, heavy-drinking patterns that may lead to abuse of alcohol or physical dependence. That this process occurs during a time of major life transition may make young adults more susceptible to developing pathological drinking habits. As Caspi and Moffitt (1993) suggested, predispositions (in this case, risk factors related to alcoholism) are accentuated by individuals in an attempt to make sense of the "novel, ambiguous, and uncertain circumstances" (p. 247) during life-transition periods.

Other researchers have provided similar conceptualizations of pathological alcohol involvement (e.g., Devor, 1994). Tarter and Vanyukov (1994) described alcoholism as a developmental disorder, postulating an epigenetic process model of alcoholism development. Genetic predispositions, as manifested in temperament, can initiate a chain of liability, interacting with behaviors and environmental contexts to increase or decrease the liability up to the threshold level for the diagnosis of alcoholism. Zucker and colleagues (e.g., Zucker, 1987, 1994; Zucker et al., 1995) also take a biopsychosocial epigenetic perspective and have constructed several pathways of risk that lead to different subtypes of young adult alcoholism, including antisocial, developmentally limited, and negative affect types (as discussed later). The initiation of substance use and early involvement with psychoactive substances (including alcohol) have been conceptualized in many ways. Petraitis, Flay, and Miller (1995) reviewed broad models of experimental substance use, most of which are also applicable to more pathological forms of alcohol involvement. Such models implicate cognitive-affective, social learning, conventional commitment and social attachment, and intrapersonal factors. Others have taken a more integrative approach involving sources of influence at biological, psychological, and social levels of explanation. In our own work, which has focused primarily on explicating the role of family history of alcoholism, we have outlined three prototypical "metamodels" or mechanisms of the risk process (Sher, 1991; Sher & Trull, 1994). A pharmacological vulnerability metamodel posits that individual differences in sensitivity to the reinforcing and punishing effects of alcohol represent one risk mechanism leading to the development or persistence of high alcohol consumption levels. Existing research suggests that individual differences in alcohol effects are associated with temperamental traits related to behavioral undercontrol (Pihl, Peterson, & Finn, 1990) and prospectively predict the development of AUDs (Schuckit & Smith, 1996). Another metamodel of risk mechanisms, affect regulation, proposes that drinking behavior is motivated by a desire to alter or control affective states. In negative affect regulation, individuals who experience high levels of distress drink to self-medicate (Kushner, Sher, & Beitman, 1990). In positive affect regulation, individuals drink to enhance positive emotional states. Both positive and negative affect regulation appear related to basic personality dimensions and outcome expectancies concerning the reinforcing properties of alcohol (Cooper, Frone, Russell, & Mudar, 1995). A third prototypical metamodel, deviance proneness, theorizes that early "difficult" temperament in transaction with deficient socialization leads to impulsivity and disinhibition, association with deviant peers, and initiation into substance use including alcohol involvement (see Sher & Trull, 1994; Zucker, 1994; Zucker et al., 1995).

In conceptualizing these metamodels, it is important to bear in mind that they represent classes of models and are not specific models themselves. For example, although the pharmacologic vulnerability model assumes that individual differences in ethanol sensitivity contribute to risk for AUDs, specific submodels differ substantially in their focus and their predictions (e.g., some models predict that greater sensitivity is associated with alcoholism risk, some models predict less sensitivity is associated with risk). With respect to the affect regulation model, some models fo-

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cus on regulating negative affect (e.g., self-medication), and others focus on regulating positive affect (e.g., direct reinforcement; Cooper et al., 1995). Likewise, one submodel of deviance proneness sees socialization failure as arising primarily from genetically based temperamental characteristics while another views these outcomes as stemming directly from defective parenting or the influence of pathogenic social environments. Although the three metamodels of alcoholism vulnerability are conceptually distinct, they are not viewed as statistically independent. Certain etiologically relevant variables (e.g., alcohol expectancies, behavioral undercontrol) relate to multiple etiologic mechanisms. Thus, in conceptualizing the relation between etiological factors and alcohol-related outcomes, key constructs can be viewed as playing multiple roles in causal chains linking biological risk to AUDs. Although these metamodels are not exhaustive of the kinds of constructs used to explain the development of substance use involvement (see Petraitis et al., 1995), they are heuristic in that they highlight how familial risk can be manifested in psychosocial processes over the course of development.

In 1987, we began a longitudinal study of the offspring of alcoholic parents and have been following this group of young adults from their freshman year in college throughout their transition into later young adulthood. The goal of this review is to highlight some of the findings that we consider most important and most relevant to the problem of pathological alcohol involvement in young adulthood. Rutter (1996) defined two research strategies for developmental psychopathology, "the study of continuities and discontinuities over the course of development and the study of continuities and discontinuities over the span of behavioral variation" (p. 211). In discussing our research, we adopt both strategies first by reviewing the course of pathological alcohol involvement throughout young adulthood and then by noting several key factors that affect its development and persistence or remission. In addition, ways in which young adult alcoholism affects other developmental processes (e.g., cognitive functioning, young adult role transitions) will be considered.

Overview of Our Longitudinal Study on Offspring of Alcoholic Parents

In the fall of 1987, 3,156 (80% of total class of 3,944) first-time freshmen at a large, Midwestern university were screened for the presence of family history of alcoholism, drug abuse, and antisocial personality disorder. Of those screened, 489 participants were chosen for the study. Participants were classified as family-history positive for alcoholism (FH+; $n = 252$) if they reported biological paternal alcoholism on both a version of the Short Michigan Alcoholism Screening test (SMAST; Selzer, Vinokur, & van Rooijen, 1975) adapted to measure paternal drinking problems (F-SMAST; Crews & Sher, 1992) and a section of the Family History-Research Diagnostic Criteria interview (FH-RDC; Endicott, Andreasen, & Spitzer, 1978) that assessed paternal drinking habits. Participants were classified as family-history negative (FH–; $n = 237$) if they reported an absence of substance use disorders in all biological first- and second-degree relatives and an absence of antisocial personality disorder in all biological first-degree relatives. (For a more complete description, see Sher, Walitzer, Wood, & Brent, 1991.) The majority of the sample is White (94%), and at Year 1 the average age was 18.6 years.

Participants from the baseline sample were contacted early in their 1st year of undergraduate work and asked to participate in a research project assessing health behaviors during the college years. Participants were assessed at baseline when they were freshmen (Year 1), at three subsequent yearly intervals (Years 2, 3, and 4, corresponding to the sophomore, junior, and senior years of college for the majority of subjects), and again 3 years later at Year 7. Attempts were made to retain and track all participants, regardless of whether they remained at the university. By Year 7, 93.7% ($n = 458$) were still participating, and 451 had participated at all five waves (although the sample size in particular studies

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may vary according to differing patterns of missing data and exclusion criteria).

At each wave of assessment, participants completed a structured diagnostic interview to assess the presence or absence of Axis I disorders using criteria from the DSM-III (1980). The Diagnostic Interview Schedule (DIS) Version III-A (Robins, Helzer, Croughan, Williams, & Spitzer, 1985) was administered at Years 1 and 2, and the DIS III-R (Robins, Helzer, Cottler, & Goldring, 1989) was administered at Years 3, 4, and 7. In addition, at each wave of assessment, participants completed a self-report questionnaire that measured a range of health behaviors including tobacco, alcohol, and drug use, motivational factors for and consequences of alcohol and drug use, and several personality scales. Also, at baseline participants completed a neurocognitive assessment, and 1 year after the Year 7 assessment a targeted follow-up was made wherein a subgroup of study participants was given neurocognitive and formal reasoning tasks. Although at Years 2–7 attempts were made to have all participants complete the assessment materials in the laboratory, some participants completed phone interviews and questionnaires that had been mailed to them (e.g., 27% at Year 7).

Course of Alcoholism in Young Adulthood

Perhaps the defining attribute of developmental psychopathology theory is its insistence on examining trajectories of continuity and discontinuity in behaviors (Cicchetti & Rogosch, 1996). Not only should psychopathology be studied across time, rather than in cross-sectional pieces, but it also should be studied at the level of the individual in order to resolve time-dependent patterns of symptomatology. Most studies use variable-centered approaches that "provide information about change over time in variables aggregated across individuals, whether the focus is on predictors of deviations from average change over time (e.g., longitudinal structural equation models) or on group-level differences in average change over time (e.g., repeated measures analysis of variance)" (Schulenberg, Wadsworth, O'Malley, Bachman, & Johnston, 1996, p. 661). From a developmental psychopathology perspective these approaches do not provide a complete picture, because they do not describe individual patterns of stability and change over time and across developmental periods (Achenbach, 1990). For example, although most college students mature out of heavy alcohol use patterns and graduate from college, there are subgroups of students who continue a pattern of frequent binge drinking during young adulthood and are less likely to graduate (Schulenberg, O'Malley, Bachman, Wadsworth, & Johnston, 1996).

Several research groups have studied the course of alcohol use during young adulthood to help explicate pathways of risk for development or maintenance of pathological alcohol involvement. Schulenberg, Wadsworth, et al. (1996) used a pattern-centered approach that outlined trajectories of frequent binge drinking based on conceptually meaningful categories (e.g., chronic, decreased, increased, fling, rare, never). Cluster analyses were then conducted to provide support for the categories (see also Tubman, Vicary, von Eye, & Lerner, 1990). Alternatively, a process-oriented approach, growth curve analysis (Chassin, Curran, Hussong, & Colder, 1996; White, Bates, & Labouvie, in press), models mean levels as well as linear and nonlinear trends in alcohol involvement, allowing for representation of quantitative changes or individual trajectories (in and out of alcohol use).

Course of AUDs in the study of offspring of alcoholic parents

Although the previously described ways of modeling the course of pathological alcohol involvement have greatly advanced our knowledge, the clinical relevance of these findings is somewhat unclear because consumption levels alone do not necessarily reflect "problems" with or dependence on alcohol. Consequently, we sought to characterize the course of AUDs over 7 years, beginning with freshman year in college. Considering the presence or absence of an AUD diagnosis (past-year DSM-III [1980] alcohol abuse or dependence) at five points in time (Years 1,

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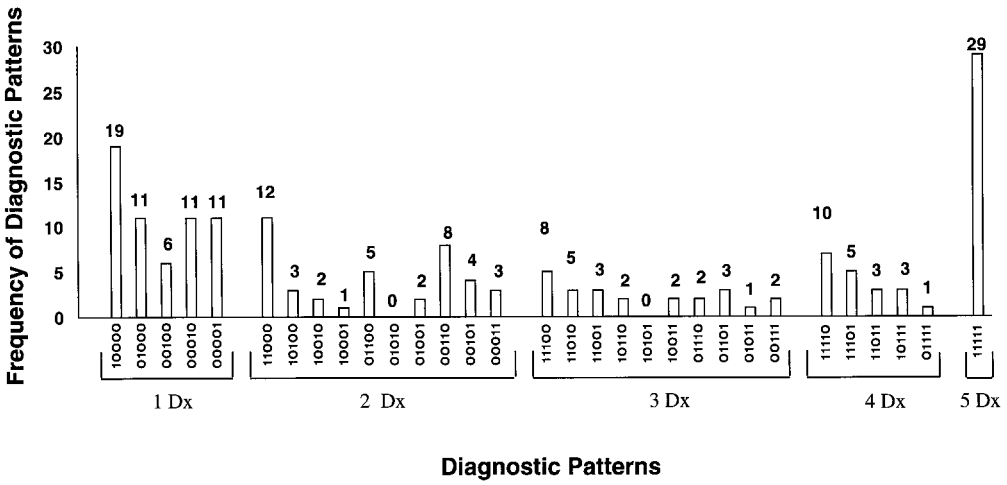


Figure 2. Patterns of diagnosing with a past-year DSM-III AUD diagnosis, Years 1–7 (n = 177).

2, 3, 4, and 7 of the study) results in 32 (2^5) possible temporal patterns of diagnosing. Of the 32 possible patterns, 30 patterns were identified in the 451 participants who completed all five assessments. For present purposes, different patterns are represented using a string of "0"s (not diagnosing) and "1"s (diagnosing), with the serial position of the 0s and 1s corresponding to successive years of data collection. Thus, the 274 individuals who did not diagnose with an AUD at any of Years 1–7 are represented by the pattern "00000." Figure 2 depicts how many of our participants who diagnosed at least once during the course of the study (n = 177) fall into each of the other patterns. Patterns are grouped according to total number of diagnoses throughout the study.

In considering Figure 2, note the different trajectories or courses of AUDs. For example, several patterns appear to represent a developmentally limited trajectory, including those who diagnosed only at Year 1, Year 2, or both but then did not diagnose later (e.g., 10000, 11000, 01000). Some individuals appear to have diagnosed several times but by later years appear to have recovered from their AUDs (e.g., 11100, 11110). A late-onset trajectory is exemplified by individuals with a pattern such as 00011 or 00001. Finally, a disturbing number of individuals appear to have chronic patterns of AUD diagnoses (e.g., 11111, 01111, 10111).

Similar to Schulenberg and colleagues (Schulenberg, O'Malley, et al., 1996; Schulenberg, Wadsworth, et al., 1996), in one study we rationally distinguished courses of diagnoses using conceptually meaningful categories (Sher, Gotham, Watson, & Meier, 1999). "Chronics" (n = 29) were classified if they met diagnostic criteria for an AUD at all five waves. "Recoverers" (n = 33) met diagnostic criteria for an AUD at baseline and at least one other time during Years 2, 3, or 4, and did not diagnose or have any past-year symptoms of an AUD at Year 7. "Nondiagnosers" (n = 274) did not meet criteria for a past-year AUD at any of the five waves. These three groups were then examined to determine whether they were differentially predicted by other study variables (as will be discussed later).

Another method of distinguishing among courses is configural frequency analysis (CFA; von Eye, 1990). CFA is a method that searches for "types," meaning configurations of scores on categorical variables, that occur more frequently than expected by chance. Expected cell frequencies are estimated for each configuration, and then a nonparametric binomial test is used to determine whether the expected and observed cell frequencies are sta-

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tistically different from one another. A CFA conducted on Years 1–4 yielded four types: chronic diagnosers (1111), developmentally limited diagnosers (1000 and 1100), and nondiagnosers (0000; P. K. Wood, Sher, & von Eye, 1994). However, a recent reanalysis with Years 1–7 yielded three types of chronic diagnosers (11111), developmentally limited diagnosers (11110), and nondiagnosers (00000; P. K. Wood, 1998). These findings, although consistent with patterns examined by other researchers, illustrate two limits of CFA: (a) findings can be quite variable with the addition of new waves of observations, and (b) as many participants are left unclassified, it is unclear how their patterns of AUD diagnoses should be typified.

One method that does lead to the classification of all participants is latent class analysis (LCA; see examples in Bucholz et al., 1996; Heath et al., 1994). LCA assumes that a number of groups (classes) corresponding to specific response profiles underlie a set of nominally scored variables. Each variable has an estimated item endorsement probability (also called a conditional probability, similar to a factor loading in factor analysis). In addition, the latent class probability refers to the probability of a particular individual falling into a particular class. Thus, based on each person's highest latent class probability, each can be assigned to his or her "most likely" class (Bucholz et al., 1996). An LCA was conducted on all 5 years of AUD diagnoses for those participants with complete data who were between 18 and 20 years of age at Year 1 ($n = 431$; Gotham, Sher, & Wood, 1999). A four-class model best fit the data. Based on a plot of the probability of diagnosing at each year (Figure 3), the classes were labeled as chronic, late onset, developmentally limited, and nondiagnosing.

Finally, several variants of path analysis have been used to examine the course of pathological alcohol involvement over time. Although path analysis represents a variable-centered rather than a pattern-centered approach, longitudinal autoregressive path analysis takes into account the influence of previous measurement occasions on future ones. In addition to autoregressive models of AUDs

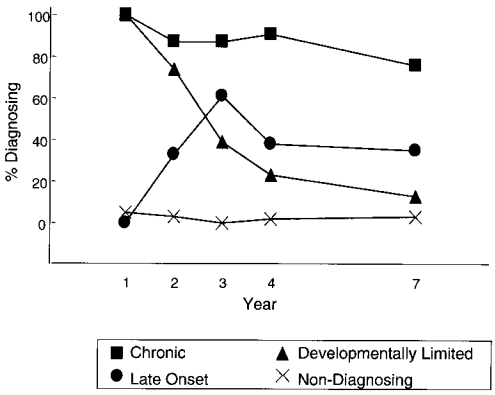


Figure 3. Four latent classes of past-year DSM-III AUD diagnoses, Years 1–7 ($n = 431$).

(e.g., Kushner, Sher, & Erickson, 1999; Sher, Gotham, Erickson, & Wood, 1996), we also use state–trait path analysis, in which AUDs are considered to be comprised of both state- and traitlike qualities (Erickson, Sher, & Wood, 1997; Sher & Wood, 1997). Whereas the traditional autoregressive model only accounts for the effect of the preceding occasion on diagnosing at the next occasion (e.g., diagnosing at Year 1 affects the likelihood of diagnosing at Year 2), a state–trait model also accounts for a traitlike factor that spans measurement occasions (Sher & Wood, 1997; Steyer, Ferring, & Schmitt, 1992). Thus, diagnosing at any occasion is attributable to the trait factor, the autoregressive carryover from the previous occasion, and unique sources of variation such as time-specific covariates and measurement error that are specific to that occasion (Sher & Wood, 1997).

In our data, state–trait conceptualizations of AUD diagnoses produce excellent fitting models with most of the variability in diagnosis on a single occasion due to the trait factor and with relatively small effects due to influences of the previous diagnostic occasion. In Figure 4 (from Erickson et al., 1997) we model the effects of family history of alcoholism and sex on AUDs over a 7-year period. In this model, we posit that the effects of both family history and sex on the diagnosis of an AUD at a particular point in time are due to their effects on the AUD trait. (In order to control for effects associated with the method bias of assessing Year 1 AUDs, sex, and fam-

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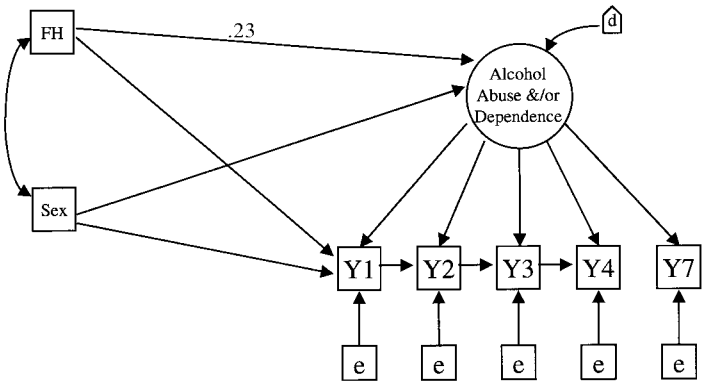


Figure 4. State-trait model of past-year DSM-III AUD diagnoses, Years 1–7.

ily history at the same point in time, we also posit direct effects from these exogenous variables to Year 1 AUD.) In addition to fitting the data well, state–trait models have several advantages for studying AUDs and psychopathology more generally. Perhaps most important, they quantitatively resolve persistence as the key construct (i.e., the trait of tending to diagnose over time) and can better resolve the effects that temporally specific variables (e.g., changes in role transitions) have on diagnosing at specific occasions (Sher & Trull, 1996).

Summary

From this delineation of various approaches to describing the course of AUDs in our sample of young adults, we find evidence for several meaningful trajectories that correspond to other researchers' conceptualizations. For example, similar to work on heavy-drinking occasions across young adulthood from the Monitoring the Future project (e.g., Schulenberg, O'Malley, et al., 1996; Schulenberg, Wadsworth, et al., 1996), we found evidence for chronic, rare or never (our nondiagnosing), decreased (our developmentally limited), and increased (our late-onset) trajectories. Our data also correspond to Zucker et al.'s (1995) suggestion that there are several forms of young adult alcoholism, including a developmentally limited form and what appears to be an emerging chronic form. However, we also believe that state–trait models represent another viable, albeit less developmental, approach to modeling the course of AUDs over time. From a state–trait perspective, individuals have a "tendency" to diagnose (i.e., the trait) and specific diagnostic occasions can be viewed as caused by the trait, short-term carryover or momentum from preceding occasions, and temporally specific third variables.

Factors Involved in the Development and Course of Pathological Alcohol Involvement in Young Adulthood

Given our evidence for different courses of pathological alcohol involvement during young adulthood, examining predictors of these different courses was a logical next step. In the following section, we highlight a few key findings with respect to predictors of the development and course of pathological alcohol involvement in young adulthood. These results (both replications of others and new findings) cover a range of topics: (a) the influence of family history of alcoholism on young adult pathological alcohol involvement, (b) the relation between personality factors (e.g., behavioral undercontrol, negative affectivity) and drinking motivations on AUDs and the course of pathological alcohol involvement, (c) the effect of role transitions on patterns of alcohol use during young adulthood, (d) the interaction of person–variables and role transitions in predicting alcohol consumption, and (e) the possibility that some risk factors are developmentally specific.

Influence of family history of alcoholism

Perhaps the most often noted risk factor for the development of pathological alcohol in-

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volvement in young adulthood is family history of alcoholism. It has long been observed that alcoholism runs in families (Cotton, 1979), and behavior–genetic studies over the past 2 decades have established an important role of heredity (Heath, Slutske, & Madden, 1997). A biological family history of alcoholism is associated with increased alcohol involvement and risk of alcoholism in adolescents and young adults (see Hawkins et al., 1992; Sher, 1991). In our own study (Sher, Gotham, et al., 1996), AUDs and frequency of heavy drinking were associated with a positive family history of alcoholism throughout the study.

However, as it is not inevitable that familial alcoholism leads one to pathological alcohol involvement, family history (as a distal variable) must be transduced by more proximal variables. In several studies from our project, the effect of family history of alcoholism on pathological alcohol involvement appears to be mediated by several other variables. For example, the influence of family history on alcohol involvement at Year 1 was mediated by behavioral undercontrol, a construct denoting impulsivity, sensation seeking, and antisociality (Sher et al., 1991).

This also appears to hold up prospectively for relations between family history and AUDs. To develop a phenotypic measure of risk that could be used in mediational analyses, we empirically constructed a "vulnerability index" using logistic regression. More specifically, this vulnerability index was constructed from a weighted composite of constitutional variables (e.g., basic dimensions of personality, measures of cognitive ability) assessed at Year 1 that best discriminated individuals with and without a family history of alcoholism. The index loaded heavily on childhood conduct problems and personality traits related to impulsivity (Erickson et al., 1997) and can be viewed as a measure of behavioral undercontrol (see below). Of greatest interest, the vulnerability index was a strong mediator of the relation between family history of alcoholism and a state–trait model of AUDs from Years 1 to 7. In Figure 5, we simultaneously modeled three purported mediators of family history risk: alcohol expectancies, childhood stressors (see Sher, Gershuny, Peterson, & Raskin, 1996), and the vulnerability index. Comparison of Figures 4 and 5 indicates that most, but not all, of the family history effect on AUDs is accounted for by these mediators (the vulnerability index is responsible for the vast majority of this mediation, with a lesser contribution from alcohol expectancies). (Childhood stressors are very strong correlates of family history but do not mediate the family history effect on AUDs, highlighting that correlates of a risk factor are not necessarily etiologically relevant; McNeil & Kaij, 1979.)

Although most of our analyses to date have focused on variables that appear to mediate risk, other researchers have found several variables that act as moderators to the family history effect and appear to serve as protective or exacerbating factors. For example, Baer, Kivlahan, and Marlatt (1995) reported that family history did not predict changes in drinking overall for young adults during the critical transition from high school to college, but it did interact significantly with sex and living environment to predict college drinking for some groups. That is, the liability to develop drinking problems appeared to be potentiated or inhibited by certain environmental contexts. In earlier research, we found that personality traits related to self-consciousness moderated the family history effect (Rogosch, Chassin, & Sher, 1990). Thus, exacerbating or protective factors can presumably take many forms and bring home the point that risk is not the same as destiny.

Influence of behavioral undercontrol

In our study, measures related to behavioral undercontrol appear to mediate most of the family history effect on offspring AUDs in young adulthood. As we use the term, behavioral undercontrol refers to a number of personality traits and outward behaviors encompassing sensation seeking, aggressiveness, antisociality, hyperactivity, and impulsivity (Sher et al., 1991) that have been associated with pathological alcohol involvement in adolescents and young adults (see reviews by Chassin & DeLucia, 1996; Sher & Trull, 1994; Sher, Trull, Bartholow, & Vieth, 1999; Zucker et al., 1995). A recent twin study

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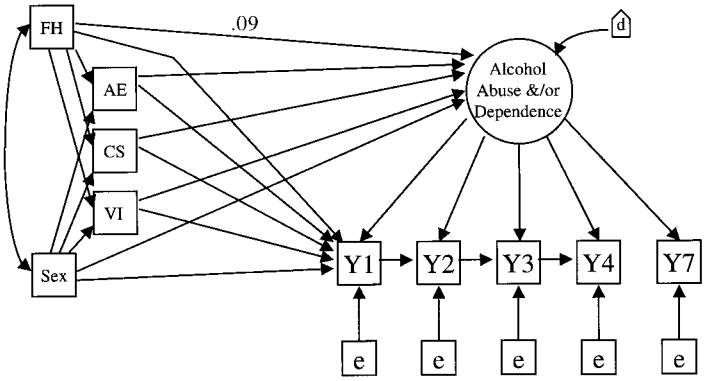


Figure 5. Mediational state-trait models of past-year DSM-III AUD diagnoses, Years 1–7.

(Slutske et al., 1998) reported a strong genetic association between childhood conduct disorder and alcohol dependence, suggesting that the two disorders have common genetic risk factors. This finding is consistent with our findings regarding the mediation of family history.

We have consistently found evidence for a relationship between personality measures of behavioral undercontrol (novelty seeking, psychoticism, agreeableness, and conscientiousness) and various measures of pathological alcohol involvement (e.g., AUDs, frequent heavy drinking; Martin & Sher, 1994; Sher, Bartholow, & Wood, 1999; Sher, Gotham, et al., 1999; Trull & Sher, 1994). Moreover, the development of AUDs is related to significantly more DSM-III (1980) Conduct Disorder and Antisocial Personality Disorder (ASP) symptoms at Year 1, and with continuing to have at least one new past-year adult ASP symptom over time (Sher, Gotham, et al., 1999). In addition, DSM-III (1980) Conduct Disorder and ASP symptoms at Year 1 presaged a chronic course of AUDs. Alcohol-specific factors related to behavioral disinhibition also influence the course of pathological alcohol use, as young adults who reported more sensation seeking reasons for drinking at Year 1 were more likely to develop an AUD and continue on a chronic course.

To put our findings in a larger context, the relations we have noted between behavioral undercontrol and pathological alcohol involvement in young adults are consistent with other work on an antisocial subtype of early-onset alcoholism (e.g., Zucker et al., 1995). In addition to research on subtypes specifically, Bates and Labouvie (1995) suggested that groups of personality and environment variables "personality–environment constellations" (p. 23) related to impulsivity, disinhibition, and deviant peers are associated with trajectories of increased alcohol use and alcohol-related problems during adolescence. Notions of such personality–environment constellations are similar to the deviance proneness (and perhaps the pharmacological vulnerability) metamodels of risk mechanisms outlined earlier.

Influence of negative affectivity

Measures of negative affectivity, including neuroticism, depression, and anxiety, are related to young adult pathological alcohol involvement (e.g., Hawkins et al., 1992; Kilbey, Downey, & Breslau, 1998), and Zucker et al. (1995) hypothesized a negative affect subtype in young adulthood. Young adults (ages 18–30 years) with a prior anxiety or depression disorder are at doubled risk for developing substance abuse disorders including AUDs (Christie et al., 1988).

Several reports from our project support that negative affectivity is related to pathological alcohol use in young adulthood. Martin and Sher (1994), Sher, Bartholow, et al. (1999), and Sher, Gotham, et al. (1999) found that several measures of neuroticism predicted AUDs during the course of the study. Negative affect, in the form of DSM-III (1980)

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anxiety disorders, DSM-III (1980) major depression, and a measure of psychological distress, also predicted diagnosing with an AUD (Sher, Gotham, et al., 1999; see also Kushner & Sher, 1993). In addition, among reasons for drinking at Year 1, mood modification motives most clearly differentiated Chronic and Recovering courses of AUDs.

Changes in negative affectivity over time are linked to trajectories of AUDs. For example, although overall levels of distress decreased over time, those who experienced AUDs (both Chronics and Recoverers) were more distressed throughout the study and showed an increase in distress from Year 4 to Year 7 of the study. Chronics and Recoverers reported similar mood modification drinking motives early in the study. Over time, however, the Recoverers' motives decreased to those of Nondiagnosers while Chronics' motives remained stable. Another set of analyses (Kushner et al., 1999) noted reciprocal relations over time between DSM-III (1980) anxiety disorders and alcohol dependence over time at Years 1, 4, and 7. For example, the odds of developing a new anxiety disorder at Year 7 were 3–5 times higher if one had alcohol dependence at Year 1 or 4, and the odds of diagnosing with alcohol dependence at Year 7 were comparably increased for those with an anxiety disorder at Year 1 or 4.

It is important to highlight that high levels of negative affectivity may not be sufficient to motivate drinking by itself. Both in our study (Kushner, Sher, Wood, & Wood, 1994) and in others' (Cooper, Russell, Skinner, Frone, & Mudar, 1992), it appears that negative affectivity or stress is only associated with alcohol consumption among subgroups of individuals holding beliefs that alcohol is effective for relieving stress or tension. Additionally, laboratory studies indicate that stress-induced drinking is only likely to occur when more effective coping alternatives are not available (Sher, 1987).

Influence of role transitions

Young adults are confronted with many role transitions that may exacerbate or inhibit the expression of psychological disorders. Examination of the effect of these transitions represents a key aspect of a developmental approach. Role transitions associated with the adolescent to early adulthood transition include completing one's education, entering the workforce, developing intimate relationships and perhaps getting married, and becoming a parent (Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 1997). One mechanism by which role transitions affect pathological alcohol involvement, role socialization, was proposed by Kandel and Yamaguchi (Kandel, 1980; Yamaguchi, 1990) and suggests that a person changes "one's drug involvement so as to make the behavior compatible with the expectations from one's social roles" (Yamaguchi, 1990, p. 132).

Research into the effects of educational and employment status on alcohol involvement provides conflicting support for a role socialization model, however. Bachman, O'Malley, and Johnston (1984) described that changes in alcohol use were related to entering college, but after controlling for high school alcohol use, employment status was not a significant predictor of level of young adult alcohol use. Similarly, Schulenberg, O'Malley, et al. (1996) reported that role transitions such as changes in marriage and parenthood predicted binge drinking trajectories while employment status did not. The relation between alcohol involvement and earnings appears curvilinear with respect to age. No discrepancies have been found in incomes between young male alcoholics and nonalcoholics. During middle age, however, there is a large discrepancy. In later years, the discrepancy decreases (Mullahy & Sindelar, 1992). Perhaps young-adult drinkers who do not attend college or leave school have initially higher wages, but over time those who attend school or do not drink heavily earn more. This curvilinear relationship may also depend on amount of alcohol consumed.

Strong socialization effects have been noted in longitudinal studies examining the development of intimate relationships and the marital role transition (becoming married, separated, divorced, or widowed). In early adulthood, getting married leads to a reduction in alcohol involvement, whereas remain-

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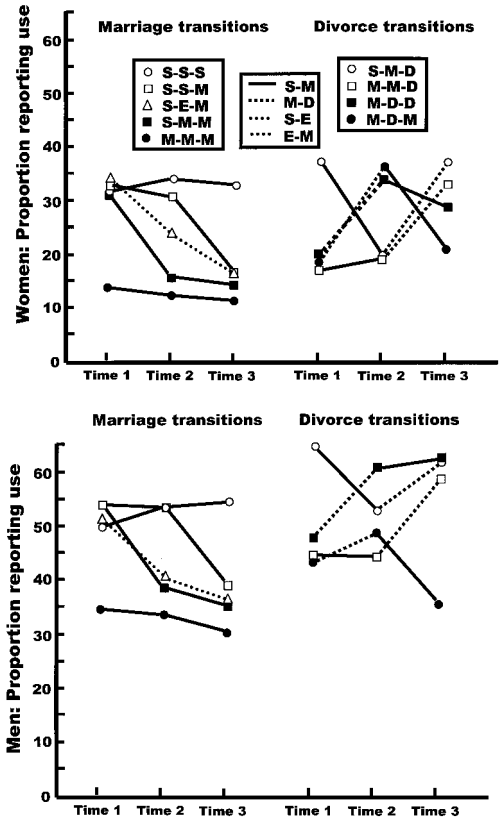


Figure 6. From "Heavy alcohol use and transitions in marital status," by J. G. Bachman, K. N. Wadsworth, P. M. O'Malley, J. Schulenberg, and L. D. Johnston, in *Parenthood During the Transition to Young Adulthood: Impacts on Drug Use and Abuse* (p. 261), 1997, New York: Cambridge University Press. Copyright 1997 by Cambridge University Press. Reprinted with permission.

ing single (and perhaps becoming divorced or separated) usually leads to an increase in drinking (Labouvie, 1996; Schulenberg, O'Malley, et al., 1996). However, becoming involved in a committed relationship or becoming engaged begins this trend toward decreased drinking (Miller–Tutzauer, Leonard, & Windle, 1991; Sadava & Pak, 1994). Bachman, Wadsworth, O'Malley, Schulenberg, and Johnston (1997) reported that young adults (ages 18–28 years) who moved (across 2-year intervals) from single to engaged to married showed a decrease in drinking in the transition to engagement (Figure 6). Individuals who were single at Times 1 and 2 and married at Time 3 did not show the decline in drinking until the assessment at which they were married. Furthermore, becoming divorced resulted in increases in drinking, suggesting that the effects of marital dissolution during young adulthood are important.

Similarly, transitions to parenthood generally are associated with decreases in alcohol use (e.g., Labouvie, 1996; Power & Estaugh, 1990; Waterson, Evans, & Murray–Lyon, 1990). However, this relationship may be confounded with marital status and the process of developing intimate relationships. Bachman, Wadsworth, O'Malley, Schulenberg, et al. (1997) reported that consistently single women and men who became parents showed a slight decrease in heavy alcohol use, whereas single nonparent women did not change and single nonparent men increased. Both men and women who became married greatly decreased their drinking, whether or not they became parents. Finally, men and women who were married at both time points decreased their drinking slightly, whether or not they became parents.

To evaluate the relation between role transitions associated with graduation from college and AUDs prior to and following the transition, we have employed both pattern- and variable-centered approaches. Gotham et al. (1999) used two structural equation modeling approaches: (a) an autoregressive approach, in which AUDs at Years 1, 2, 3, 4, and 7 were modeled as an autoregressive process, and (b) a typological approach, in which results of an LCA from Year 1 to 4 were dummy coded to represent three groups of participants—never diagnosing, chronically diagnosing, and late diagnosing trajectories. Preadulthood variables such as sex, family history of alcoholism, high school class rank, and religious involvement at Year 1 were included, as well as achievement of various role transitions by Year 7 (completion of a baccalaureate degree, currently working full time, and currently married). (Because only a small percentage of participants had completed the role transitions by Year 4, they are assumed to have occurred between Year 4 and Year 7.) The preadulthood variables were allowed to covary, the developmental tasks were allowed to covary, and all lagged paths between the

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AUD diagnoses were modeled. (Thus, all models were just-identified.) Most important for this discussion, in both models we found protective effects of baccalaureate degree completion and marital status on AUD diagnosis at Year 7. Individuals who got married between Years 4 and 7 or completed their BA or BS degree during that time were less likely to diagnose with an AUD at Year 7. It is important to point out that both of these analyses controlled for prior AUDs. Thus, the alternative hypothesis that the ostensibly beneficial effect of marriage and obtaining a college degree is an artifact of individuals who are not problem drinkers getting married and completing college more frequently (what is called a "role selection" effect) is a less plausible explanation than the socialization effect. We believe that role socialization is a major contributor to the maturing-out process observed during early adulthood.

Influence of person-related variables during role transitions

In addition to examining the effect of role transitions themselves on pathological alcohol involvement in young adulthood, it is also important to consider individual difference or person-related variables that may, as noted by Caspi and Moffitt (1993), have a particular impact at these critical junctures in development. For example, men are more likely than women to continue problem drinking or increase binge drinking from high school or college to later young adulthood (Schulenberg, Wadsworth, et al., 1996).

We conducted a more limited examination of the influence of individual difference variables on changes in frequency of intoxication over the transition from undergraduate college to other roles (Gotham, Sher, & Wood, 1997). Such variables included sex, family history, and personality variables from Costa and McCrae's (1992) "Big Five" factor model of personality (e.g., neuroticism, extraversion, openness to experience, agreeableness, conscientiousness). In a subsample of our participants ($n = 288$) who were full-time students at Year 4 (average age: 21.2 years) and had earned a bachelor degree by Year 7, being male, being relatively more open to experience, and entering the workforce full time were associated with decreases in frequency of intoxication from Year 4 to Year 7. Interestingly, individuals in this subsample who were relatively more extraverted had an increased likelihood of continuing a pattern of frequent intoxication from Year 4 to Year 7. This study was somewhat limited in that it used a variable-centered approach that examined a modal pattern. It did, however, provide an interesting look at this subgroup making a major role transition and lead to a further project examining the effects of preadulthood variables and the course of AUDs on later role transitions (Gotham et al., 1999).

Developmental specificity of risk factors

An issue not discussed earlier is that role transitions such as marriage and parenthood have different impacts on pathological alcohol use depending on the age of the young adult. These may be examples of what Rutter (1996) described as "age-indexed variations in susceptibility to environmental hazards" (p. 212). Despite the general negative relation between alcohol use and parenthood, it appears that there is a trajectory of early heavy alcohol involvement that leads to increased premarital pregnancy (Mensch & Kandel, 1992). Therefore, early heavy alcohol use, especially in women, might discriminate between precocious pregnancy in adolescence and normative childbearing in later young adulthood (Newcomb, 1996). Several other issues related to age-indexed variations are relevant. Some variables, such as alcohol outcome expectancies, are correlated to alcohol involvement during both adolescence and young adulthood, suggesting a continuing pattern of expectancies. However, in adolescence, expectancies build and increase over time (Miller, Smith, & Goldman, 1990; Smith, Goldman, Greenbaum, & Christiansen, 1995), whereas in early adulthood alcohol expectancies decrease (Sher, Wood, Wood, & Raskin, 1996). Thus, normative changes in alcohol expectancies roughly track changes in alcohol involvement.

We are particularly interested in variables

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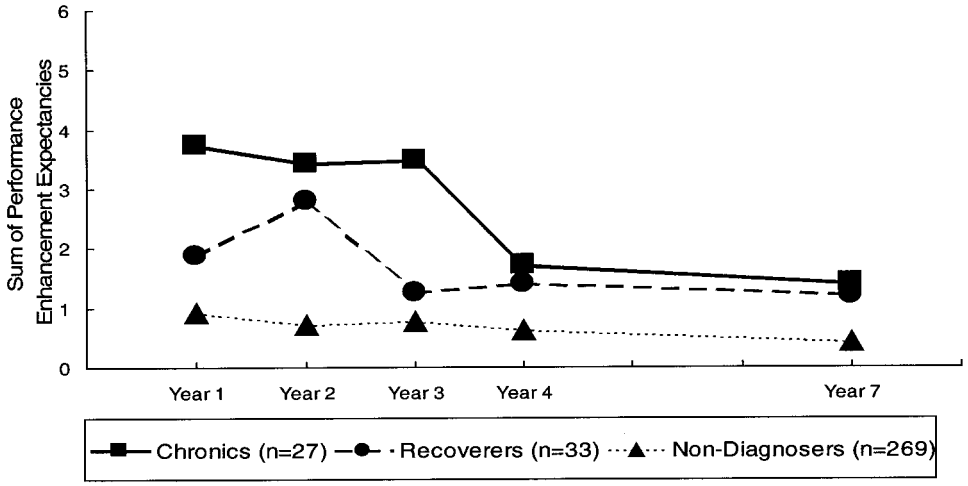


Figure 7. Level of Performance Enhancement alcohol expectancies over time by course of AUDs.

that hold different prognostic significance at different stages of development (i.e., variables that are good prospective predictors in late adolescence but lose prognostic significance several years later). As seen in Figure 7, Chronic diagnosers reported much stronger performance enhancement alcohol expectancies (e.g., "drinking helps me perform certain tasks better") than Recoverers at Year 1, but by Year 4 the two groups were similar (Sher, Gotham, et al., 1999). These differences between the Chronic and Recoverer groups observed at earlier time points—differences that disappear over time—suggest that some risk markers are developmentally limited.

Implications of Young Adult Pathological Alcohol Involvement on Later Development

Thus far we have considered role transitions as predictors of either the development or course of young adult pathological alcohol involvement. In addition, part of what makes pathological alcohol involvement in young adults a disorder of development is its effects on the normative outcomes of young adulthood—that is, the cognitive and intellectual attainment and role attainment and transitions that are hallmarks of the transition from adolescence to young adulthood.

Cognitive performance and neurocognitive impairment

Baumrind and Moselle (1985) argued that alcohol and other substance use during late adolescence and early young adulthood may disrupt intellectual development, and bring changes in self-esteem and identity, social and moral reasoning, and the ability to assume adult roles. Pathological alcohol involvement during this critical phase can interfere, directly and indirectly, with ambient socialization experiences and formal education that constitute the critical environments for developing higher intellectual functions. Using data collected during the targeted follow-up after Year 7, P. K. Wood, Sher, and Bartholow (1999) found almost no evidence for deficits in neurocognitive performance or cognitive abilities due to AUDs during the college years unless a heavy-drinking pattern was maintained after college. These results suggest that, at least for nonclinical samples, cognitive deficits attributed to alcohol involvement during the college years may reflect prior cognitive impairment or deficits associated with

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recent alcohol involvement (i.e., short-term effects).

Educational/academic attainment

Although in cross-sectional studies alcohol use is related to negative educational outcomes such as poor grades and dropping out (CASA, 1994; Presley, Meilman, & Lyerla, 1993), researchers have offered that other third variables better account for these relations (Cook & Moore, 1993; Jessor, Donovan, & Costa, 1991). Also, our work suggests that results vary depending on the measure of academic attainment used. P. K. Wood, Sher, Erickson, and DeBord (1997) found that the bivariate relation between alcohol involvement at Year 1 (freshman year) and later academic problems was accounted for by variables occurring before the freshman year, including high school class rank, academic aptitude, and parent's education. Likewise, obtaining a baccalaureate degree by Year 7 was significantly predicted by preadulthood variables (e.g., sex, family history of alcoholism, high school class rank, and religious involvement) but not by AUDs at Years 1–7 or dummy variables for AUD course (Gotham et al., 1999). However, using level of degree attained (including different kinds of postbaccalaureate work), collegiate AUDs (Years 1–4; and in separate analyses, collegiate heavy drinking) predicted lower overall level of educational attainment at Year 7, even after controlling for preadulthood variables (M. D. Wood, Sher, & McGowan, in press). High school class rank moderated the relation between collegiate AUDs and later educational attainment. Individuals with higher high school class ranks showed a strong negative relation whereas those with lower high school class ranks showed a negligible relation between AUDs and educational attainment.

Although we did not find associations between trajectories of AUDs and educational attainment, Schulenberg, O'Malley, et al. (1996) reported that individuals who completed more years of education were more likely to have slightly increased and then decreased binge drinking across young adulthood as compared to other binge drinking trajectories (e.g., increased, decreased, rare). These results were observed after controlling for a number of preadulthood variables, current marital status, and employment. This "fling" trajectory appears similar to the normative maturing-out effect of increased alcohol involvement during the college years and then decreased use after approximately age 21 years (Grant, Harford, & Grigson, 1988; Johnston et al., 1996; Temple & Fillmore, 1985–1986).

Occupational attainment

Similar to educational attainment, research on occupational attainment and pathological alcohol involvement yields different results depending on the age of the participants, how occupational attainment is operationalized, and whether occupational attainment is adjusted for the effects of prior alcohol use and educational attainment. Mullahy and Sindelar (1989) reported that onset of AUD symptoms before age 22 years was related to lower job status (after controlling for demographic factors but not current symptoms of alcoholism). However, we found (M. D. Wood et al., in press) no evidence for a relation between collegiate AUDs or heavy drinking and occupational status 7 years postmatriculation. Using full-time work status instead of occupational attainment (Gotham et al., 1999), a significant relation was documented between Year 4 AUD (senior year in college for most participants) and full-time work status at Year 7. That only the most proximal AUD diagnosis had a harmful effect on later work status may reflect a flexibility of work roles at this age, especially given that the majority of our participants were enrolled in school during Years 1–3.

When examined from a developmental perspective that takes age into account, some resolution of disparate findings is possible. Alcohol use during high school signals early involvement in the workforce, which leads to initially higher wages (precocious development; Newcomb, 1996; Newcomb & Bentler,

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1988). Over time, however, continued heavy alcohol use may lead to lower work statuses and more job changes (Kandel, Davies, Karus, & Yamaguchi, 1986; Kandel & Yamaguchi, 1987). Also, while young adults who go to college may have lower alcohol involvement in high school, they appear to "catch up" upon entering college (Bachman et al., 1984). It is likely that individuals who experiment with heavy alcohol use in college, but mature out near the end of college, are those who have the highest occupational attainment in later young adulthood (Gotham et al., 1997; Mullahy & Sindelar, 1992).

Social transitions of marriage and parenthood

Generally, pathological alcohol involvement during young adulthood is associated with lower rates of marriage and parenthood (e.g., Mullahy & Sindelar, 1989; Power & Estaugh, 1990; Schulenberg, O'Malley, et al., 1996). We have shown that individuals who get married in young adulthood are less likely to drink in adolescence or young adulthood (Gotham et al., 1997, 1999). Similarly, most young adults who become parents are initially less involved with alcohol and then decrease their consumption compared to those who do not become parents (Power & Estaugh, 1990; Schulenberg, O'Malley, et al., 1996).

However, as noted earlier, a very early onset of alcohol use or abuse may lead to precocious development, which results in increased risk for early marriage or parenthood (e.g., Newcomb & Bentler, 1985, 1987). Power and Estaugh (1990) reported that women who were heavy drinkers at age 16 years were more likely than other groups to have had a first partnership (either marriage or cohabitation for at least 6 months) during their teens. There also appears to be a trajectory of early heavy alcohol involvement that leads to increased risky sexual behavior, pregnancy, and parenthood (Parker et al., 1994). Mensch and Kandel (1992) documented that teens (up to age 19 years) who had an earlier initiation into alcohol use were more likely to become pregnant, although multivariate analyses revealed that illicit drug use was a more robust predictor of premarital pregnancy. Taken together, the research suggests that the meaning of "early marriage or cohabitation" differs greatly depending upon the age group studied. In adolescence, early marriage is a sign of unconventionality and is associated with higher levels of substance use, while in early adulthood it is a sign of conventionality and is associated with less substance use. More generally, the timing of various role transitions needs to be viewed in a broad developmental context.

Prevention, Policy, and Treatment Issues

If research leads us to conceive of problematic alcohol involvement as a disorder of development, it follows that our prevention and treatment strategies need to incorporate a developmental perspective. Toward that end, we will discuss some implications of our research for interventions designed to prevent and ameliorate pathological alcohol involvement in young adulthood. Because prevention can occur at multiple levels (governmental, institutional, family or individual), we consider policy and prevention together. We also use Institute of Medicine prevention classifications: universal (general public or specific groups), selective (individuals with above average risk), and indicated (individuals with noted risk factors; Mrazek & Haggerty, 1994). Note that prevention, policy, and treatment programs can be directed towards children and adolescents before they begin drinking (or quite early in their drinking careers), as well as young adults who are in the midst of alcohol use and pathological alcohol involvement.

Prevention and policy

Prevention programs and policies aimed at factors that increase risk for pathological alcohol involvement (rather than decreasing actual alcohol use), such as behavioral undercontrol and alcohol-specific factors (e.g., alcohol expectancies) can be implemented before young adulthood. For instance, early treatment of conduct disorder may be preventative of later

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problematic alcohol use. Also, children with a family history of alcoholism or who exhibit personality traits related to behavioral undercontrol could be targeted for special programming to foster self-regulation (e.g., Maguin, Zucker, & Fitzgerald, 1994). Regarding alcohol-specific factors, by age 6 years many children can differentiate the smell of alcohol from other substances (see review by Zucker & Fitzgerald, 1991). Just a few years later children have developed expectancies about the effects of alcohol (Miller et al., 1990). School-based universal prevention efforts that focus on the development of alcohol use norms, expectancies, and mores (Adolescent Alcohol Prevention Trial; Hansen, Graham, Wolkenstein, & Rohrbach, 1991) have been implemented. Other programs intervene at the level of peer-related variables such as peer pressure and resistance training (Shope, Dielman, Butchart, Campanelli, & Kloska, 1992). Children and adolescents also develop alcohol-related schemas and beliefs through advertising in the mass media (Grube & Wallack, 1994). Thus, efforts at minimizing the effects of mass media (Collins, 1995; Grube, 1995) are necessary. Such strategies include restricting advertising, consulting with producers of alcohol advertising, counter- and educational advertising, and providing educational and skills training programs within schools that help adolescents and young adults reduce the impact of alcohol-related advertising (Collins, 1995; Grube, 1995). Each of these areas of prevention and policy are thought to affect the behavioral precursors or environmental context of alcohol involvement.

The dilemma of prevention in young adulthood is that this stage of life represents the ages of peak alcohol involvement, and individuals are already well into their period of risk for AUDs. Approximately 40% of those who will develop an AUD have their first symptom between ages 15 and 19 years (Helzer, Burnam, & McEvoy, 1991). In many ways, "the horse is already out of the barn." Moreover, it does not appear that high levels of consumption typically result from individual drinkers "losing control" of intake once they start drinking. For example, it appears that level of alcohol intoxication achieved is closely related to preepisode intent (Glindemann, Geller, & Ludwig, 1996). For these reasons, indicated approaches may be most relevant to this stage of life. Such approaches can focus either on attempts to have the drinker moderate his or her consumption or on creating environments that foster moderation.

Because most young adults with AUDs will never come in contact with alcohol treatment specialists, alternative intervention strategies are needed. In recent years, there has been increasing recognition of the role primary care physicians can play in screening for alcohol problems and effecting positive outcomes through brief interventions (National Institute on Alcohol Abuse and Alcoholism, 1997). However, despite the demonstrated efficacy of brief physician interventions, implementation of screening and intervention procedures remains rare. Practice guidelines and favorable managed-care reimbursement procedures could facilitate broader adoption of screening and brief intervention protocols in primary-care settings in the community, student health services, and in the military.

A more specialized strategy of indicated prevention, Marlatt's High Risk Drinkers Project (Baer, 1993; Kivlahan, Marlatt, Fromme, Coppel, & Williams, 1990), is targeted at young adults who are already heavily alcohol involved. This approach includes motivational interviewing (e.g., having individuals evaluate the role of alcohol in their lives and consider altering their consumption patterns; Miller & Rollnick, 1991), information about alcohol involvement (e.g., models of addiction, overview of the immediate and delayed effects of alcohol, the role of expectancies), and alcohol-related behavioral skills (e.g., assertiveness, drink refusal skills, setting limits). In intervening with young adults, Marlatt's group recognizes that abstinence is not a viable goal for many and encourages moderation as a way of both avoiding problems and maximizing the benefits of drinking. It is also possible to intervene by focusing on drinking contexts. Excessive alcohol consumption in young adults most typically occurs in social settings such as parties and bars. Engineering these settings to encourage moderation by requiring greater effort to obtain

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beverages (e.g., use of bartenders vs. self-service at fraternity parties; Geller & Kalsher, 1990) or promoting the use of low alcohol beverages (Geller, Kalsher, & Clarke, 1991) can reduce levels of intoxication.

Treatment

Perhaps the greatest treatment-related paradox is that although early young adulthood is the time of highest pathological alcohol involvement, treatment for AUDs appears to peak in later adulthood. Moreover, of all psychiatric disorders except for antisociality and cognitive impairment, individuals with diagnosable alcohol abuse are least likely to tell a doctor about their symptoms (Robins, Locke, & Regier, 1991). Clearly, the group with the largest potential need is not seeking or receiving treatment. Perhaps this is because young adults do not view their alcohol use patterns and negative consequences as problematic. Young adults have been shown to overestimate their peers' alcohol use (Baer, Stacy, & Larimer, 1991) relative to their own. Given the normative heavy drinking that accompanies young adulthood, particularly in college settings, it is likely that young adults fail to see that they are engaging in potentially problematic behaviors associated with risk for long-term health consequences. Indeed, in our own study less than 35% of chronic diagnosers reported having concerns about their drinking. These reported concerns decreased over time despite the fact that these individuals were experiencing persistent problems.

Because young adults often do not view their pathological alcohol involvement as problematic, treatment approaches that serve to increase problem recognition would appear to be well suited to this target population. Motivational interviewing (Miller & Rollnick, 1991) was in large part developed to assist individuals who are, in Prochaska and DiClemente's (1984) terms, precontemplative and have not yet considered altering their drinking behavior. For these individuals, a primary therapeutic goal is to "raise doubts—increase the client's perception of risks and problems with current behavior" (Miller & Rollnick, 1991, p. 18).

The role socialization influences discussed earlier suggest that interventions serving to increase success in school, the workplace, and developing intimate relations would have salutary effects on drinking behavior. That is, one approach to treatment could be to facilitate those normative transitions that appear to assist in socializing individuals out of excessive drinking patterns. Such socialization influences due to transitions into educational or work settings can provide a powerful opportunity to change drinking trajectories as has been seen in such adult treatment programs as the Community Reinforcement Approach that combines alcohol treatment with job training and recreational counseling (Smith & Meyers, 1995).

Regarding individual variables related to increased drinking over transitions, we have shown that comorbid disorders related to behavioral undercontrol (e.g., conduct disorder, ASP) and negative affectivity (e.g., anxiety disorders, major depression) are not only associated with the development of pathological alcohol involvement but are also associated with a chronic course of AUDs during young adulthood. Thus, treatment of such comorbid disorders can have beneficial effects on pathological alcohol involvement in young adulthood (Kaminer, 1994; Miller, 1995).

As noted by Bukstein (1994), until recently most adolescents and young adults who were treated for AUDs were treated in the same facilities and with the same treatment programs as older adults. Although this review did not specifically discuss differences in pathological alcohol involvement between younger and older adults, there are several characteristics of young adult AUDs and problematic drinking relevant to treatment. Issues such as young adults' developing emotional and intellectual maturity, the strong influence of parents and peers, and the role transitions discussed in this review need to be addressed or at least considered when planning treatment strategies. Also, as noted in the previous section, assertiveness, drink-refusal skills training (e.g., social skills training; Monti, Abrams, Kadden, & Cooney, 1989), and challenges to schemas regarding alcohol (e.g., alcohol expectancies) may be most relevant to young adults who are developing new

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peer networks and have not necessarily had long drinking careers.

Assessment of severity is an important consideration in treatment planning with young problem drinkers. Babor, DelBoca, McLaney, Jacobi, Higgins–Biddle, and Hass (1991) described an identification, referral, and treatment plan called Youth Evaluation Services (YES) that places adolescents in school-based programs, after-school programs, intensive outpatient programs, or therapeutic community or inpatient programs according to the level of severity of the problem. Similar "stepped care" approaches can be implemented with young adults. That is, the level of treatment intensity can be titrated to the persistence of the AUD. Subsequently, the level of treatment intensity is systematically adjusted, as needed, based on treatment responsiveness (Institute of Medicine, 1990). Given the vulnerability of young adults to develop long-standing problems with alcohol, treatment at an earlier stage of problem development, in addition to the proactive prevention strategies mentioned earlier, may help some young adults from following a chronic course of AUDs throughout later adulthood.

Summary

Early adulthood represents the stage of life most associated with alcohol use disorders. Many of those who experience AUDs will quickly mature out of these problems, others may suffer severe consequences with long-term implications (e.g., traumatic injuries, unwanted pregnancy, HIV infection), and still others might progress into a life-course persistent form of AUDs. Understanding not only the determinants of AUD onset but also the course of AUDs is critical and requires a broad psychological perspective that incorporates an appreciation of the effects of earlier development, stable individual differences, and the unique developmental challenges facing the young adult. Just as development affects the onset and persistence of AUDs, they can have important influences on the course of later development. An important life-task of late adolescence and young adulthood is the building of human capital (in the form of education and early vocational training) that holds dramatic implications for quality of life and economic well-being over the course of adulthood. To the extent that AUDs undermine the accrual of this capital, the long-term consequences can be devastating. However, we are still in the early stages of charting the long-term consequences of AUDs in young adulthood. Part of the difficulty facing researchers is that AUDs occur in the context of other psychological characteristics (e.g., behavioral undercontrol, negative affectivity) that can exert important effects of their own. The development of more effective prevention and treatment strategies requires that we adopt a broad perspective that simultaneously examines the reciprocal associations between presumed causes and effects of AUDs over the life span.

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