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A Developmental Perspective on Alcohol and Youths 16 to 20 Years of Age

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ABSTRACT

Late adolescence (ie, 16–20 years of age) is a period characterized by escalation of drinking and alcohol use problems for many and by the onset of an alcohol use disorder for some. This heightened period of vulnerability is a joint consequence of the continuity of risk from earlier developmental stages and the unique neurologic, cognitive, and social changes that occur in late adolescence. We review the normative neurologic, cognitive, and social changes that typically occur in late adolescence, and we discuss the evidence for the impact of these transitions on individual drinking trajectories. We also describe evidence linking alcohol abuse in late adolescence with neurologic damage and social impairments, and we discuss whether these are the bases for the association of adolescent drinking with increased risks of mental health, substance abuse, and social problems in adulthood. Finally, we discuss both the challenges and successes in the treatment and prevention of adolescent drinking problems.

IN THE UNITED States, alcohol involvement sometimes starts but more often escalates between 16 and 20 years of age, when youths are also experiencing dramatic physical, emotional, and social changes. Specifically, a variety of forms of hazardous drinking emerge during middle to late adolescence and, for many youths, these problematic patterns of drinking continue to escalate through 18 to 20 years of age, the period of greatest risk for the onset of an alcohol use disorder (AUD).

In seeking to understand youth development and alcohol involvement, it is important to consider all dimensions of functioning, because the interrelated cognitive, biological, social, and affective changes that occur during adolescence not only affect each another but also influence an individual's risk of problem drinking. In particular, the timing, sequence, and synchrony of developmentally specific transitions can affect how well youths master new roles, as well as continuities and discontinuities in their behavior. Therefore, developmental models representing a range of theoretical orientations, including systems theory, behavioral genetics, and developmental psychopathology, hold great promise for advancing our understanding of the processes that underlie adolescent changes, including the emergence of alcohol use and abuse.

The need for developmental approaches is underscored by the existence of a consistent body of research showing that the escalation in drinking and the emergence of AUDs in middle and late adolescence have behavioral, social, and biological roots from earlier developmental stages. Moreover, it is becoming increasingly clear that alcohol involvement in adolescence has both short- and long-term effects on health and well-being at later developmental stages. Importantly, the consequences of adolescent drinking seem to differ from those associated with adult drinking, because there is increasing evidence that adolescents are especially vulnerable to the adverse effects of heavy alcohol use on both biological and social functioning. Problematic drinking has the potential to redirect the normative course of adolescent development in ways that increase risks not only for AUDs but also for a range of mental health and social problems.

From a developmental perspective, changes in contexts, processes, and developmental tasks all play critical roles in the evolution of drinking behavior during the transition from middle adolescence to young adulthood. The complex multilevel changes in youth drinking and associated behaviors both are bounded by and influence the adaptive capacity of the individual and relate directly to the success individuals experience as they transition through critical normative developmental tasks. For youths in this age range, these tasks include (but are not limited to) dating, obtaining a driver's license and driving, graduating from high school, entering/attending college, leaving the rearing home, entering the workforce or military, and marrying and forming a family.

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Key Words

alcohol, late adolescence, development

Abbreviations

AUD—alcohol use disorder

DWI—driving while intoxicated

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In this review, a multisystem multilevel lens is created, through which adolescent alcohol involvement can be viewed in both its normative and maladaptive developmental roles. This lens not only provides a framework for characterizing the features of current prevention and intervention efforts that contribute to their success but also helps to identify new, more-effective approaches.

NORMATIVE PATTERNS OF DEVELOPMENT

Developmental Contexts and Tasks of Late Adolescence

Late adolescence is a remarkably variable period of development. As with earlier stages, changes emerge both within the individual and in the physical, relational, and social contexts within which the individual develops. Pronounced maturational demands emerge as youths transition out of adolescent responsibilities and begin to assume adult roles. Understanding the normal developmental transitions of this period and the processes whereby these changes unfold is critical for understanding the emergence of alcohol use patterns and problems and the interventions that seek their amelioration.

The late adolescent years are characterized by major role transitions in every domain of life.¹ Normative changes center around taking increasing responsibility for one's daily life, behavior, and future; moving toward less-dependent and more-mature relationships with the family of origin; exploring romantic and sexual relationships; and preparing for and initiating adult occupational roles, including pursuing postsecondary education and/or employment. Preparation for adult relationships includes the development of romantic and sexual relationships for most and cohabitation, engagement, marriage, and/or childbearing for some. Preparation for occupational life includes the completion (or leaving) of high school, beginning of formal paid work, and possibly initiation of postsecondary training.

As adolescents move beyond high school, they tend to spend more time in settings away from their homes and neighborhoods. Those who attend college full-time enter a new, age-graded, social environment, adaptation to which provides opportunities for a fresh start, even if it is stressful.² For other youths, this is also the time of life when they leave their rearing homes and establish for the first time their own residences. Regardless of with whom they live, adolescents at this life stage typically assume increasing responsibility for managing their daily lives, including balancing studying and working with their social and leisure pursuits and making decisions regarding practical matters such as when to sleep and what to eat. The major developmental contexts and tasks for this life stage are summarized in Table 1.

Late adolescence is also a time of legal transition, as developing youths begin to assume the rights, privileges, and obligations of adulthood. Adult legal status is generally defined chronologically, and the presumptive "age of majority" in the United States and much of the Western world is now 18 years. At this age, individuals are empowered to vote, to enter binding contracts, and to assume primary responsibility for financial matters.

TABLE 1 Diversity of Developmental Contexts and Tasks During Late Adolescence/Early Adulthood (16–20 Years of Age)

Developmental Contexts or Tasks	Examples
Contexts	
Living arrangements	Alone In dorm With parents With friends With romantic partner
Educational settings	High school College Night school Trade school
Work settings	Part-time vs full-time employment Career initiation Unemployment
Tasks	
Relational	Dating and sexual behavior Marriage Starting a family Socializing with peers
Occupational/educational	Completion of mandatory education Vocational training College/professional education Starting career
Legal	Driver's license Criminal liability Financial responsibility

There are exceptions, however, to the rule that the 18th birthday brings with it all the rights and responsibilities of adulthood. For example, adolescents can be held criminally responsible in adult criminal courts at <18 years of age. Alternatively, a few privileges and responsibilities are delayed until older ages, including the right to purchase alcohol, which is delayed until 21 years of age. These exceptions are very important, because they reflect both strong societal concerns about adolescent alcohol use and the risks and vulnerabilities that continue after the age of majority has been reached.

Aside from the traditional, age-based, categorical classifications, the law reflects a variety of transitional arrangements that attach privileges and responsibilities on a gradual basis. The most important of these are related to the licensing of drivers, which in many states progresses from learner's permits to provisional driver's licenses with restrictions and then to driver's licenses with full rights and responsibilities. Similar graduated transitional rules can be seen in the imposition of curfews, the regulation of youth employment, and mandatory education. Finally, the very purpose of the juvenile court was to serve a transitional role, and it continues to do so within a restricted sphere.

Increasing Importance of Individual Differences

Despite the almost-universal shift toward greater independence during late adolescence, the timing, sequence, and even occurrence of role changes differ greatly among individuals.³ This reflects in part the less age-graded, constricted, and obligatory nature of develop-

mental transitions at this life stage, compared with childhood and early adolescence. As a consequence, the diversity of living contexts is greater in late adolescence than at any earlier developmental stage; characterization of this period in terms of the “typical” adolescent can be misleading. By age 20, for example, ~20% of adolescents are still single and living with their parents while attending college⁴; nearly 25% do not have a drivers’ license. Although 86% of youths complete high school and 62% of those youths enroll in some college the year after they finish high school, only 29% have earned a bachelor degree by age 25. In part because of the diversity of options and available supports, social and cultural influences emerge as important influences at this life stage; academic attainment, the timing of romantic partnerships and childbearing, and the age of leaving the parental home all differ in complex ways according to gender, ethnicity, socioeconomic status, neighborhood, and country of origin.⁵

Historical trends toward achieving traditional markers of adult status (full-time worker, spouse, and parent) at later ages contribute to the broad diversity of developmental contexts seen in late adolescence. The substantial differences seen in the timing, sequence, and even experience of developmental milestones preclude simple, universal, prescriptive descriptions for this age period. These factors also have resulted in this stage evolving to be more a preparation for than the achievement of adult status, leading to this life stage being designated a period of “emerging adulthood.”⁵

Biological Development

Brain Development

Although it was once thought that human brain development was largely complete by the onset of puberty, it is now known that the brain continues to develop throughout adolescence and into young adulthood.⁶ Longitudinal research demonstrates that higher-order association cortices develop later than primary sensorimotor cortices, with the dorsolateral prefrontal cortex developing through the latest stages of adolescence.⁷ These late developments represent complex interactions between synaptic refinement, which results in decreasing cortical gray matter volumes,^{7,8} and myelination,⁹ which is associated with increased cerebral white matter volumes⁸ and increased density, organization, and integrity of white matter pathways.⁹ These white matter changes result in neural signals transmitting more rapidly, permitting greater capacity for more-complex, higher-order reasoning and processing. This emerging view of adolescence as a period of continued neuroplasticity brings with it a recognition that this is also a period of vulnerability to neurotoxic processes, including those attributable to heavy alcohol exposure.

Sleep Changes

In general, teens need ~8.5 to 9.25 hours of sleep per night, and this amount remains stable over adolescence, in contrast to preadolescence, when sleep needs fluctuate.¹⁰ The most significant change in adolescent sleep

patterns is the delay of the intrinsic sleep phase. This means that teens often do not feel physiologically tired enough to fall asleep until late at night, resulting in late bedtimes (often after midnight) and the desire to sleep later in the morning, compared with younger children. This sleep pattern becomes problematic during the school year (when adolescents need to wake up early), resulting in an average weeknight sleep duration of 7 to 7.75 hours per night for high school-aged youths.¹¹ The ensuing sleep debt accumulates over time, despite “catch-up” weekend sleep, resulting in excessive daytime sleepiness in a significant proportion of adolescents. In a 1997 report by the National Institutes of Health, adolescents and young adults were identified as a population at high risk for pathological sleepiness.¹² The consequences of poor sleep and daytime sleepiness are vast and include poor academic performance, accidents while driving drowsy, negative mood, and self-medication with stimulants, alcohol, and other drugs of abuse.^{11,13}

Development of Emotional and Behavioral Self-regulation

Self-regulation refers to the ability to control and to plan one’s behaviors and to resist impulses to engage in behaviors that result in negative consequences. This factor can be measured in many ways (experimentally, self-reports, and parent or teacher reports). Self-regulation, as measured through maternal report for example, increases over childhood and early adolescence but seems relatively stable by late adolescence, with girls reporting higher levels than boys.¹⁴ The inability or unwillingness to inhibit behavioral impulses has long-term prognostic significance. Markers of disinhibited behavior in preschool-aged¹⁵ or elementary school-aged¹⁶ children predict AUD risk in late adolescence and early adulthood, whereas indicators of neurobehavioral disinhibition¹⁷ have been associated with increased risks for the development of adolescent substance use and problems.

The ability to regulate behavioral impulses is critical for successfully dealing with the increased exposure to risk that typically occurs during adolescence. Adolescents, especially those in late adolescence, are more likely than adults to engage in risky behavior, such as unprotected sex, hazardous driving, or heavy drinking.¹⁸ Age differences in risk-taking behavior may not, however, be attributable to adolescent deficits in the cognitive assessment of risk. By 15 years of age, adolescents seem to be as cognitively capable as adults with respect to logical assessment of the likelihood of risk and their vulnerability to risk.¹⁸ Therefore, rather than being attributable to deficiencies in logical analysis, the adolescent propensity for risk-taking may be a consequence of the way in which adolescents process social and emotional cues.¹⁹ For example, differences between adolescents and adults in risk-taking behavior are maximized when individuals are in the presence of peers and minimized when they are alone,²⁰ which suggests that risk-taking may serve a social function in adolescence. Furthermore, the propensity for risk-taking may depend on 2 distinct brain networks, with different maturational trajectories during adolescence,¹⁹ (1) a cognitive control

network, which is involved in planning and self-regulation and is localized primarily to lateral prefrontal and parietal cortical regions, and (2) a socioemotional network, which is involved in the processing of social and emotional stimuli and is localized primarily to the limbic system, including the amygdala, nucleus accumbens, and medial prefrontal cortex.

Cognitive Development

Unique Cognitive Status of Adolescents

Many individuals working with adolescents perceive them to be “somewhere between childhood and adulthood . . . in terms of cognitive development . . . [such as] the ability to plan and anticipate.”²¹ The cognitive differences between adolescents and adults are more subtle than those between children and adults, however, and primarily involve the domain of executive functioning. For example, inhibitory control develops on a gradient throughout adolescence, although some proportion of adolescents’ improvement may be attributable to simple increases in the speed of processing.²² “Metacognition,” the strategic, supervisory, and self-monitoring aspects of executive functioning,²³ also continues to develop throughout adolescence, particularly in the effective use of available strategies.²⁴ There is also evidence of gender differences in executive function development. Flexible control of verbal processing develops more quickly in girls than in boys, which suggests that boys may not achieve mature verbal control of their behavior until their late teens.²⁵ Girls lag behind boys in the development of certain nonverbal tasks.²⁶

Decision-making

Decision-making refers to the complex process involved in selecting an action among different options with various outcomes. This process involves multiple underlying cognitive abilities, which may develop at different rates. For example, logical reasoning, an obvious component of decision-making, develops throughout adolescence,²⁷ as does one’s management of reasoning biases and one’s judgment of reasoning success.²⁸ Social judgment and social problem-solving also improve through adolescence into early adulthood.²⁹ Some studies have suggested that individuals in middle adolescence have a more-competent decision-making style than do those in early adolescence.³⁰ Decision-making typically involves integrating factual knowledge with internal cognitive capacity, particularly executive functioning. In this regard, it is noteworthy that adolescents have a much larger gap between procedural knowledge (eg, memory skills and goal-oriented planning) and factual knowledge than do adults. This suggests that adolescents may lack strategic options for using extant factual knowledge. Adolescence is likely also a period of practice in applying executive strategies (procedural knowledge) to real-world problems that is crucial for normal development. Obviously, decision-making is related to the likelihood of alcohol abuse, particularly in decisions related to risk-taking and the projected assessment of consequences. In this regard, although most adolescents perform like

adults on a gambling task, adolescents at risk for substance involvement may have decision-making impairments, demonstrating greater risk-taking in decision-making.³¹

Working Memory

Working memory refers to a constellation of interrelated cognitive processes that ultimately result in one’s ability to hold temporarily and to manipulate information on-line.³² This ability is fundamental to intact performance in a variety of other cognitive domains, including language comprehension, abstract reasoning, and learning and memory. Verbal and spatial working memory abilities improve throughout childhood and adolescence,³³ and working memory for spatial stimuli in particular continues to mature during the adolescent years.³³ For example, older adolescents demonstrate better accuracy and faster reaction times than do younger adolescents during spatial working memory tasks.³⁴ Functional MRI research has suggested that adolescents activate similar brain regions as do adults while engaged in working memory tasks³⁵ but they show increased frontal and parietal brain activation, relative to younger adolescents,^{34,36} and greater^{34,36} and more-widespread³⁶ activation, relative to adults.

Poor Executive Functions Predispose Subjects to Alcohol and Other Substance Use Disorders

Because executive functions mediate the complex interplay between thinking, affect, and social judgment, it is not surprising that research has confirmed associations with alcohol and drug abuse. Poorly developed executive functions are prominent in adolescents who are at high risk of developing alcohol/substance abuse problems, including those with conduct disorder,³⁷ those with high Minnesota Multiphasic Personality Inventory MacAndrew Alcoholism Scale scores,³⁸ and children of alcoholics.³⁹ Individuals with attentional disorders (with impaired executive functioning, by definition) have high rates of alcohol and drug abuse and dependence, particularly when conduct disorder is comorbid.⁴⁰ Executive function scores also predict the age at first drink,⁴¹ which is consistent with the finding that students using alcohol before the sixth grade show weaker decision-making skills.⁴² For children of alcoholics, executive function scores predicted the number of drinks per drinking occasion⁴³ and reactive aggression.⁴⁴ Executive functions also combine with coping styles⁴⁵ and temperament⁴⁶ to increase liability for abuse of substances. Poorer executive functioning in social drinkers has been associated with decreased awareness of abuse consequences.⁴⁷ If executive functions do not mature fully until early adulthood, then this lack of maturity is likely to alter the effectiveness of prevention programs.

Adolescent Cognitive Development and Effectiveness of Prevention Programs

Alcohol and other drug abuse prevention efforts are typically aimed at adolescents. Multiple types of programs, from education-based school programs⁴⁸ to risk

TABLE 2 Components of Executive Functions and Sample Behaviors

Component	Behaviors
Goal-directedness	Establishing goal hierarchies, maintaining goals, organizing sequences, evaluating progress, using strategies
Initiation/inhibition	Initiating behavior independently, self-cueing, inhibiting inappropriate behavior, constraining actions with rules
Flexibility/perseveration	Generating novel possibilities, flexibly switching between guidelines, performing contingency-based revisions, strategizing
Abstract reasoning	Using rule-guided thinking, forming concepts, using hierarchical and temporal relationships
Reward appraisal	Evaluating reward likelihood, performing relative valuation, using reward appraisal to guide behavior
Social appraisal	Understanding social norms, apprehending social cues, incorporating social information into decision-making

or skill remediation programs⁴⁹ and even community-wide programs,⁵⁰ have been developed. Despite 3 decades of research, these prevention efforts still show mixed effectiveness.⁵¹ Those that seem most promising operate at multiple levels of intervention.⁵⁰ One way to increase effectiveness might be to tailor these approaches by using information on cognitive changes in late childhood and adolescence.

Because the initiation (or refusal) of use of alcohol and other drugs involves a decision, prevention programs target various components of the decision-making process, such as the quality of information available for consideration,⁴⁸ consideration of consequences,⁵² identification of social pressures for use,⁵³ generation of behavior alternatives,⁵² and problem-solving skills development.⁵⁴ The prevention literature has not benefited from the identification of specific cognitive functions that support high-level processes such as decision-making or the wealth of research that has accumulated from the study of these and other executive functions. Executive functions consist of multiple components that enable consideration of goals, social contexts, and rewards, as well as regulation of response initiation and inhibition. These functions, described in Table 2, are pivotal to the 2 primary cognitive tasks of adolescence, namely, to develop one's capacity to integrate information from diverse sources relevant to a goal and to develop personal rule sets that efficiently guide behavior toward future goals. Strategies that work to facilitate adolescents' self-change behaviors may be particularly promising in this regard.⁵⁵

Social and Emotional Development

Family Relationships

In optimal situations, normative transitions toward independence from the family of origin occur in the context of continued parental support and attachment, which predict getting off to a good start in terms of education, career, and family life.⁵ Parental assistance in

the form of housing and other material support aids late adolescents in pursuing education and launching themselves toward adult life, but availability varies greatly according to parental finances. When adolescents leave the family home, the quantity of interaction with parents often decreases but the quality of relationships typically improves.² Alcohol use tends to increase in parallel with individuation from parents⁵⁶ and decreased parental monitoring.⁵⁷ However, the internalization of healthy parental norms may promote healthy choices in the long term.⁵⁸

Relationships with siblings have received less research attention than those with parents, particularly in later adolescence. However, older siblings' alcohol and drug use predicts early adolescents' subsequent substance use, beyond parental predictors.⁵⁹ Behavioral genetic studies also suggest that youths' perceptions of their older siblings' alcohol use predict younger siblings' alcohol involvement. Furthermore, unlike many other sibling and parental correlations, whose interpretation is confounded by the possibility of passive genotype-environment correlational processes, sibling similarities in alcohol use seem to involve true environmental effects.⁶⁰ Mechanisms through which siblings influence substance use may include modeling, access to alcohol, and direct social influence.

Peer Relationships

Normative age-related changes in the importance of peer relationships and culture heighten exposure to cultural norms and influences and in many cases encourage experimentation with and heavy use of alcohol.⁶¹ Subjectively positive aspects of social life center around drinking contexts, and sociability expressed while drinking can serve as a marker of successful peer relationships and bonding. Media and peer culture heighten exposure to norms, myths, and influences that promote an age-graded period of experimentation. Cultural and peer influences are not, however, monolithic in their power or direction of influence. Individuals tend to seek out and to be selected by peers who have similar goals, values, and behaviors.

Peer influences on risk for alcohol use and abuse can take 3 alternative forms during this age period.⁶¹ First, peer influences may be relatively direct, through modeling processes or the encouragement of alcohol use. Direct peer influences increase in salience during periods of change and adaptation to new environments. Second, peer influences may be self-sustaining, because affiliation with like-minded friends can encourage behavioral continuity and resistance to change processes. Finally, adolescents significantly overestimate the prevalence of their peers' drinking, which indirectly encourages heavy drinking. Peer influences can be amplified by the media and popular culture, which generally depict unhealthy and risky behaviors, such as drinking, physical aggression, interpersonal conflict, and unprotected sex, as both glamorous and risk-free.⁶²

Romantic Relationships and Sexuality

Adolescence brings dramatic changes in sexual feelings, sexual identity, and experimentation with romantic relationships and sexual behaviors. Brooks-Gunn and Paikoff⁶³ identified 4 developmental challenges in the domain of sexuality, namely, becoming comfortable with one's maturing body, accepting feelings of sexual arousal, understanding that sexual behaviors should be mutually voluntary, and practicing safe sex. These challenges are personal and relational, involve complex feelings and shared behaviors, and remain important developmental tasks well beyond adolescence. Romantic relationships are central to late adolescent and young adult development. These new relational expectations and challenges have the potential to influence adolescent development both positively and negatively. Romantic relationships progress from heterosexual group interactions to group dating and ultimately to dyadic romantic relationships in which emotional reliance shifts from friendships (eg, best friends) to romantic partners.⁶⁴ These interactions are strongly tied to friendships and peer relationships. The role of alcohol in the divergent trajectories of relationship involvement during adolescence has not been fully articulated.

At 18 years of age, ~70% of adolescents have engaged in sexual intercourse.⁶⁵ Most, however, have sex infrequently and have done so with only 1 partner. Therefore, many in this age range have relatively little sexual experience, confidence, or skills.⁶³ Coupled with a developmentally normative intense interest in finding a romantic partner, limited sexual experience can lead to unplanned, unreciprocated, or nonconsensual sexual situations. Of note, alcohol involvement is associated with a variety of early sexual activities. Adolescent girls with older boyfriends seem to be a particularly vulnerable population. Girls with boyfriends >2 years older than they are engage in more of all forms of sexual intimacy and are more likely to have sex under the influence of alcohol and to experience sexual coercion.⁶⁶ Alcohol use may be paired with early sexual experiences in several ways. Desire to meet new partners may lead to social contexts where alcohol is served, and positive expectancies about the social and sexual enhancement properties of alcohol may increase motivations to drink. Alcohol consumption in turn can make sexual behaviors in general and unsafe behaviors in particular more likely, by reducing inhibitions, giving courage, and providing an "excuse" for getting wild.

Emotional Changes and Mental Health Problems

Negative affect and emotional lability increase with the onset of and progression through puberty. These changes not only reflect neurohormonal changes and brain maturation patterns but also coincide with increases in environmental stress during this period. Along with school transitions (eg, progression into high school and college), exposure to new social situations, transition to first work environments, elevations in relational aggression, and heightened academic performance expectations contribute to emotional distress experienced during this period. Several types of mental health problems also increase during

this age period, including depression, anxiety, suicidal ideation, and delinquent behaviors.

Disruptive disorders such as conduct disorder and attention-deficit disorder, as well as internalizing disorders of depression and anxiety, increase risk for alcohol involvement and related problems. Conversely, intensive and protracted alcohol use has been shown to provoke deviant behaviors as well as depression and anxiety.^{67,68} In community studies of adolescents, youths with an AUD or substance use disorder have 3 times the rates of cooccurring axis I mental health disorders, compared with youths without alcohol or drug disorders.⁶⁹ Mood disorders and disruptive disorders are more prevalent among youths with alcohol/drug disorders than are anxiety disorders, and rates of cooccurring disorders are substantially higher in clinical populations.⁶⁸ Youths treated for AUDs who also have an axis I mental health disorder have been shown to experience relapse more quickly and after exacerbations in symptoms of the mental health disorder.⁷⁰

NORMATIVE PATTERNS OF ALCOHOL INVOLVEMENT

Epidemiological Perspectives on Typical Drinking Experiences

By any measure, alcohol use and problem drinking are widespread among the nation's high school students. On the basis of the 2005 national survey from the Monitoring the Future study, by the time young people reach 10th grade (modal age: 16 years), almost two thirds have tried alcohol at least once in their lifetime, with more than two fifths reporting being drunk at least once; by 12th grade (modal age: 18 years), more than three fourths have tried alcohol at least once, with nearly three fifths reporting being drunk at least once.⁷¹ In terms of current alcohol use, 33.2% of the nation's 10th-graders and 47.0% of the nation's 12th-graders reported using alcohol at least once in the past 30 days, 17.6% and 30.2%, respectively, reported being drunk in the past 30 days, 21.0% and 28.1%, respectively, reported having ≥ 5 drinks in a row (sometimes called binge drinking) in the past 2 weeks, and 1.3% and 3.1%, respectively, reported daily alcohol use.⁷¹ Historically, the 2005 rates reflect a long-term (but not always progressive) decrease in use over the past few decades. Nonetheless, the 2005 rates are hardly comforting, with >2 of 10 of the nation's 10th-graders and almost 3 of 10 of the nation's 12th-graders reporting binge drinking in the past 2 weeks.

Considerable attention has been given to the increase in alcohol use and problem drinking that occurs in the years immediately after high school, when widespread alcohol use gives way to pervasive use, culminating in the highest rates of use across the entire life span during the early twenties. With regard to alcohol use during the first 2 years after high school (modal ages: 19 and 20 years), lifetime prevalence (based on 2005 follow-up surveys from the Monitoring the Future study) was 81.8%, 30-day use prevalence was 59%, and binge drinking prevalence was 36.3%.⁷² Furthermore, it is notable that, although rates of alcohol use and binge drinking have declined for post-high school youths, the decreases have been relatively modest, compared with

those seen for high school students, as mentioned above.^{61,72} This is especially true for college students, who have higher rates of alcohol use than their noncollege peers, although high school students who are college bound have lower rates of drinking than do high school students who do not proceed to college.^{61,71,72} In 2005, the rate of binge drinking for college students (1–4 years beyond high school) was 40.1%, whereas the rate for their noncollege peers was 35.1%.⁷²

Across the years of high school and the transition to adulthood, alcohol use and problem drinking vary according to various sociodemographic characteristics. The prevalence rates of alcohol use are higher for boys than for girls, higher for white and Hispanic adolescents than for black adolescents, and higher for those living in the North and North Central US regions than for those living in the South and West regions. During early high school, alcohol use is typically negatively related to socioeconomic status (and parent education level); by the end of high school and during the transition to adulthood, this relationship decreases and actually reverses, corresponding to proportionally higher rates of college attendance among youths with higher socioeconomic status. During high school, alcohol use tends to be negatively related to population density (with higher rates in rural areas), but this relationship also reverses during early adulthood.^{71,72}

Patterns and Trajectories of Drinking and Social Subtypes of Youth Drinkers

Overview

The normative nature of the increase in alcohol use between 16 and 20 years of age suggests that it may serve a developmental function, such as those involving autonomy, risk-taking, and social relationships.^{61,73} Indeed, it is unlikely to be a coincidence that alcohol use, problems with alcohol, and other risky behaviors all increase during late adolescence, at the same time that quests for autonomy and identity exploration increase and social relationships intensify. Similarly, the relationship between increased alcohol use/problems and the many transitions that follow high school, particularly those related to college entry, is likely to be attributable at least in part to the changes in social roles and contexts that occur in late adolescence.⁶¹ The notion of alcohol (and other drug) use being developmentally embedded during late adolescence and early adulthood leads to a consideration of both (1) the trajectories of alcohol use and problem drinking over time and (2) the relationship of alcohol use and problems to developmental tasks.

Trajectories of Alcohol Use and Problem Drinking Over Time

Although it is useful to consider the normative increases and decreases in alcohol use at the population level, it is essential from a developmental perspective to investigate how individuals and subgroups differ in their patterns of alcohol use over time by using longitudinal data. Multiwave prospective studies allow for the characterization of both individual and group trajectories of alcohol use in terms of characteristics such as overall level, rate of escalation, age at peak use, and rate of decline.

Such studies generally have taken 1 of 2 forms, focusing on either the normative group trajectory (and considering individual deviations from the normative trajectory in terms of variations around the intercept and slope) or the existence of common trajectory groups (eg, chronically high use, escalating use, or low/no use).⁷⁴ These 2 approaches, which are sometimes referred to as variable-centered and pattern-centered approaches, respectively, are not mutually exclusive, and techniques such as growth mixture modeling combine the 2.⁷⁵ However, the 2 approaches tend to have different assumptions about the extent to which the normative trajectory usefully describes the important features of individual trajectories of alcohol use across time.

Several studies have examined the normative trajectory of alcohol use/problems during adolescence and early adulthood by using growth models representing the level (intercepts) and rate of change over time (slopes). In general, findings indicate that there are normative increases in alcohol use and heavy drinking from early adolescence through the early twenties, with marked individual differences in the rates of change.⁷⁶ The advantages of this approach include the abilities to predict individual differences in intercepts and slopes (with time-invariant or time-varying covariates) and to correlate changes in alcohol use (eg, the slope for an individual trajectory) with changes in other behavioral and social constructs, to understand how alcohol use “travels together” with other variables.

The multiple-trajectory approach essentially groups people according to their similarity in how they change or do not change over time. Rather than assuming that everyone generally follows the same developmental trajectory, this approach aggregates individuals into multiple, relatively homogeneous groups according to their distinctive trajectories. The advantages of this approach include the abilities to draw meaningful distinctions among the groups in terms of antecedents and consequences and to consider the occurrence of similar trajectory groups across different outcomes.⁷⁷ Maggs and Schulenberg⁷⁴ summarized several studies that used this approach to examine alcohol use and problem drinking during late adolescence and early adulthood. Although the studies varied considerably in terms of ages, waves, measures, and analytic strategies, several common trajectory groups were found across the studies (Table 3). The most commonly observed trajectory subgroup included abstainers, light drinkers, or very rarely heavy drinkers across all time periods assessed. Depending on the specific definitions used in different studies for these levels of alcohol consumption, estimates of the proportion of young people in this low-risk group ranged from approximately one fifth⁷⁸ to more than two thirds.³⁹ Members of another common trajectory subgroup, that is, stable moderate drinkers, engage in some steady drinking but limited heavy drinking across adolescence and young adulthood. Across studies, estimates are that approximately one third of adolescents and emerging adults fall into this group.⁷⁹ Together, these 2 broad categories, which include relatively low-risk drinkers, include a large proportion of all young people.

TABLE 3 Alcohol Use Trajectory Groups Identified in Longitudinal Research on Adolescents

Trajectory Group	Alcohol Use Pattern	Approximate Representation, %
Abstainers/light drinkers	Stable low use or nonuse of alcohol	~20–65
Stable moderate drinkers	Stable moderate use, limited heavy use	~30
Fling drinkers	Time-limited periods of heavy use	~10
Decreasers	Early onset but declining course of alcohol use	~10
Chronic heavy drinkers	Early onset and stable course of heavy drinking	<10
Late-onset heavy drinkers	Late onset with rapid escalation to heavy drinking	<10

Many studies also identified groups of chronic heavy drinkers and late-onset heavy drinkers. These 2 groups are distinguished by the age at which the subjects start heavy drinking, but, because this age varies among studies, chronic heavy drinkers and late-onset heavy drinkers are more difficult to compare across studies. Chronic heavy drinkers typically start heavy drinking at a relatively early age and tend not to decrease their drinking in their twenties.^{78,80} Members of the late-onset heavy-drinking subgroup start to drink later, but their use escalates rapidly.^{75,80}

“Fling” drinkers,⁸⁰ who make up 10% to 12% of the adolescent and young adult population, take yet a different trajectory. They experience a period of developmentally limited, heavy drinking that peaks and then declines after late adolescence or the early adult years.⁸⁰ A final subgroup, decreaseers, seems to be more common in older adolescent and young adult samples⁸⁰ than in younger samples. Decreasers begin heavy drinking at an early age and reduce their use significantly during or shortly after high school. Approximately 10% of adolescents and young adults fall into this subgroup. Of note, substantial fluctuations in youth drinking occur within the course of a single year⁸¹ and may be tied to personal change efforts⁸¹ or environmental factors.⁸²

Relationships of Alcohol Use and Problems to Developmental Tasks

From a developmental perspective, it is essential to examine the meaning of substance use from the young person’s perspective, specifically, to consider substance use in relation to the developmental tasks adolescents are expected to accomplish. Escalating alcohol use and patterns of long-term heavy use are likely to be associated with many negative antecedents and consequences, likely interfering with various developmental tasks. For example, alcohol use is often involved in first (and unwanted) sexual encounters and increases as youths initially transition into independent living. In addition to the many destructive aspects of alcohol (and other drug) use, there may be some constructive aspects in terms of developmental tasks, particularly with respect to identity

exploration and bonding with peers.^{73,83} For example, as Maggs⁸⁴ demonstrated, alcohol use during the transition to college may help to achieve valued social goals, such as making new friends, but may threaten safety and short- and long-term health and well-being. During late adolescence and early adulthood, alcohol use may function in part to accomplish particular developmental tasks, reflecting age-normative (although not necessarily optimal) behavior.

Summary of Patterns of Alcohol Involvement

During high school, alcohol use and problem drinking are widespread; after high school, rates of alcohol use and problems increase to lifetime peaks in the early twenties. Post-high school alcohol use varies, to some degree, as a function of social role and context, as illustrated by rapid increases in use and problems among college students. On the basis of multiwave longitudinal data, studies have considered individual- and group-level trajectories of alcohol use and problem drinking, illustrating both the population-level increase that occurs during late adolescence and early adulthood and the distinct trajectory courses that characterize different groups (eg, chronic heavy use, increasing use, and low-level use/abstinence). By examining individual- and group-level trajectories, studies can better address how changes in alcohol use relate to changes in other constructs, providing a stronger foundation for a developmental perspective on alcohol use. Numerous gaps exist in this line of research, indicating the need for more multiwave longitudinal research that includes a wide range of developmental biological and psychosocial phenomena. An important component of a developmental perspective is an emphasis on how alcohol use relates to developmental tasks. In many ways, the field has yet to address adequately the challenge of Baumrind and Moselle⁸⁵ from 2 decades ago regarding the need to document fully the negative effects of alcohol and other drug use on developmental tasks. However, there has been some recognition that, in addition to the destructive aspects, there are potential constructive aspects (from the young person’s perspective) of alcohol use. A more-comprehensive understanding of the destructive and constructive aspects of alcohol use in terms of developmental tasks during late adolescence and early adulthood is needed to advance etiologic research and prevention efforts.

RISK AND PROTECTIVE PROCESSES

There is substantial research literature establishing the major risk and protective factors associated with alcohol use and abuse in late adolescence. For example, we know that adolescent abusers are more likely than non-abusers to have a family history of alcoholism, to have preexisting mental health problems, to have low levels of self-regulation, to come from broken families and/or to have parents who monitor their activities poorly, to be exposed to deviant peer models, to have been victims of sexual or physical abuse, and to hold beliefs that encourage excessive alcohol use. Alternatively, adolescents

who do not abuse alcohol are more likely to be oriented positively toward family and school and to have long-term educational and occupational aspirations. Although we know much about the major dimensions of risk and protection in late adolescence, we know relatively little about the developmental mechanisms that underlie their association with alcohol use outcomes.

The landmark research of Jessor⁷³ showed that adolescent alcohol use and abuse usually does not occur in isolation but typically occurs with other adolescent problem behaviors, including tobacco and illicit drug use, early sexual behavior, antisocial behavior, and poor academic attachment and progress. There are differing perspectives on the mechanisms that underlie these correlations. One perspective, which is reflected in the so-called Gateway Model, posits that adolescent alcohol use increases the likelihood of other adolescent problem behaviors because exposure to alcohol alters the course of adolescent development.⁶⁹ Alternatively, others have argued that the co-occurrence of adolescent problem behaviors arises because adolescent alcohol use is 1 manifestation of a more-generalized disposition to contravene social norms.⁸⁶ In this case, adolescents use alcohol for the same reasons they use or abuse other substances or engage in antisocial behavior (eg, a relatively strong need for stimulation and social reinforcement, coupled with relatively low levels of self-regulation). Although distinct, these 2 perspectives are not necessarily incompatible. Adolescent alcohol use may both reflect an underlying disposition toward undercontrolled behavior and alter the course of adolescent behavior in a way that increases the likelihood of untoward outcomes.⁸⁷ For example, McGue et al⁸⁸ reported that early use of alcohol was associated with a range of preexisting markers of disinhibition, including ratings of hyperactivity/impulsivity and inattention and event-related brain potential differences (reduced P3 amplitude), the latter being a well-known biological marker of alcoholism risk.⁸⁹ Alternatively, Keyes et al⁹⁰ found that early use of alcohol in adolescence was associated with subsequent increases in a range of contextual risks, including increased parent-adolescent conflict, reduced engagement in school, and increased exposure to deviant peer models.

Behavioral genetic research documents the existence of genetic influences on adolescent drinking behavior, several features of which are particularly noteworthy. First, the strength of genetic influences on drinking behavior seems to increase from middle adolescence to late adolescence.⁹¹ Second, in late adolescence and early adulthood, genetic influences on symptoms of problem drinking seem to overlap extensively with genetic influences on other indicators of disinhibited behavior.⁹² Although the latter research suggests that for some youths abusive drinking in late adolescence may be driven substantially by inherited differences in a general disposition toward undercontrolled behavior, it does not rule out the influence of alcohol-specific genetic effects (eg, genetic influences on alcohol sensitivity) or the impact of contextual factors. Indeed, there is mounting evidence that genetic influences on complex behavioral outcomes such as drinking behavior reflect a complex interplay between inherited and environmental factors,⁹³ the im-

plications of which for models of adolescent drinking are only beginning to be explored.⁹¹

The transition to college serves to illustrate the complex nature of contextual influences on drinking behavior in late adolescence. As discussed earlier, research has shown unambiguously that drinking, especially binge drinking, increases markedly during the transition from high school to college. However, differences in drinking behavior between college students and non-college students are not necessarily large,⁹⁴ which suggests that there may be developmentally normative aspects to increases in problem drinking in late adolescence. Moreover, the influence of college on drinking behavior may be transient in most cases.⁹⁵ However, we cannot conclude that the sharp increase in high-density drinking that occurs in late adolescence is without long-term consequences. Students attending college drank less in high school than did students not attending college. Therefore, the college environment is able to elicit high levels of problem drinking in a relatively low-risk population. Which individuals are most susceptible to these contextual influences in late adolescence and the long-term consequences of the sharp increase in binge drinking that occurs in late adolescence remain to be determined.

The timing of other life transitions in late adolescence is also associated with drinking behavior. Although normative at later life stages, the assumption of adult social roles in adolescence, which Bachman and Schulenberg⁹⁶ termed "precocious development," is associated with increased risk of problem drinking and other social problems. For example, adolescents who work >20 hours/week,⁹⁷ marry, or become a parent⁹⁸ are all at increased risk of problem drinking, relative to adolescents whose course of social development is more normative. Although precocious transitions clearly disrupt the normal course of adolescent development, their effects on drinking behavior remain somewhat obscure. The association of early transitions with high levels of drinking may reflect preexisting dimensions of risk or the disruption of normal developmental processes. Research on early puberty may be informative in this regard. Adolescents, particularly girls, who undergo puberty early are at increased risk for early substance use.⁹⁹ They are also at excess risk for the early expression of other antisocial behaviors, although differences in antisocial behaviors between early- and late-maturers decrease with age, as the late-maturers "catch up."¹⁰⁰ It is possible that the principal effect of early puberty on drinking behavior is to advance the age at which drinking is initiated, and early puberty may have limited impact on long-term alcohol outcomes.

Gender differences in drinking behavior, which are minimal at earlier life stages, begin to emerge and to become consequential during late adolescence.¹⁰¹ Although adolescent girls are as likely to have tried alcohol as adolescent boys, they are less likely to be diagnosed as having an AUD and to engage in problematic or binge drinking. Gender differences in drinking behavior are associated with different profiles of risk. Adolescent girls, for example, are more likely than adolescent boys to be

victims of abuse¹⁰² and to suffer from depression and anxiety,¹⁰³ both of which are associated with elevated rates of drinking. Adolescent boys are more likely to suffer from externalizing disorders (eg, conduct disorder) and to score high on measures of impulsivity,¹⁵ which also are markers of risk. Although we know much about the existence and correlates of gender differences in adolescent drinking behavior, we know relatively little about the mechanisms that underlie those differences and consequently the reasons for the apparent convergence in male and female drinking behaviors over time.¹⁰⁴

Resilience refers to positive developmental outcomes for individuals who face risk or adversity. Resilience among children has been studied extensively over the past 30 years. Patterns of resilient behavior provide important information for models of normal development and models of the development of problem behaviors and may also yield insights into effective preventive strategies. Given the pronounced maturational development across all domains of youth behavior that occurs in late adolescence, along with the substantial increases in alcohol-related risks, resilience is a useful perspective from which to evaluate the pathways of success for youths regarding alcohol problems (eg, abstinence or nonproblematic drinking or rapid resolution of hazardous drinking or abuse). Resilience has particular applicability to youths with genetic or environmental risks for alcohol dependence, many of whom are able to overcome their developmental liabilities.¹⁰⁵ Biological, cognitive, social, and emotional characteristics of youths can contribute to their resiliency. In particular, certain integrative skills (such as self-organization and emotional regulation) that emerge during this time period promote resilience and may protect against trajectories of protracted heavy alcohol involvement.¹⁰⁶

CONSEQUENCES OF ADOLESCENT ALCOHOL USE AND ABUSE

Developmental Perspective on the Consequences of Adolescent Alcohol Use

To the extent that alcohol consumption during adolescence has effects on later development, the costs to the individual and to society increase greatly beyond the obvious short-term consequences. Addressing how and to what extent adolescent alcohol use affects subsequent development, however, involves distinguishing the effects of alcohol use from influences that precede or occur with adolescent alcohol involvement. For example, early externalizing disorders (eg, conduct disorder) are associated with risks for excessive alcohol and drug involvement and with risks for a range of other enduring, adverse, behavioral outcomes. In addition, some developmental consequences may result from brief exposures to alcohol during a window of developmental vulnerability, whereas other effects may emerge only as a result of protracted alcohol use or drinking in conjunction with other substance involvement. Consequently, demonstration of an adverse developmental consequence that can be attributed to alcohol involvement requires longi-

tudinal research that demonstrates an effect of alcohol exposure over and above these frequent comorbidities.

As noted above, a number of developmental models have been proposed to explain the role of alcohol involvement and problems in youth development. For example, the theoretical analysis of the effects of alcohol and drug abuse by Baumrind and Moselle⁸⁵ suggests that adolescent substance use could result in negative effects on the attainment of formal operational capacities, transitions in attitudes toward social convention, the normative transition from conventional to principled morality, normative increases in self-centeredness, enhancement of abilities to understand the perspective of others, increases in peer group significance and changes in social contexts, development of ego identity, and development of self-esteem. Also, Baumrind and Moselle⁸⁵ proposed specific mechanisms regarding how alcohol and other substance involvement could affect development, by obscuring the context of work and the context of play, promoting a false consciousness of reality, reinforcing a sense of being special, enabling the adolescent to avoid realistic confrontations with the environment, consolidating the cultural relativism and negative identity characteristics in (early) adolescence, and masquerading as an emancipatory effort. In addition, affective and behavioral systems may be affected by an amotivational syndrome. These proposed mechanisms exemplify ways in which alcohol and other substance involvement may interfere with the development of basic competencies in intellectual functioning, moral development, personality development, schooling and vocational readiness, and development of interpersonal skills (both generally and in forming intimate relationships). Alcohol and other substance use may preempt normal development by providing shortcuts to achieving various roles without the underlying competencies or eliminating the need to develop competencies by maintaining a social context that is tolerant of functioning typical of earlier ages.

Effects of Alcohol Use on Adolescent Development

Typically, alcohol-related problems and consequences refer to a variety of negative life experiences that arise from drinking, such as social problems (eg, physical or verbal aggression or relationship difficulties), legal problems (eg, arrests for driving while intoxicated [DWI] or public inebriation), educational/vocational problems (eg, academic difficulties, termination from employment, or failure to achieve career goals), and medical problems (eg, unintentional injury, liver disease, or central nervous system disease). Although alcohol problems may be direct consequences of consumption, other factors associated with alcohol consumption may act independent of or interact with actual consumption to predict problems.¹⁰⁷ Although some types of outcomes (eg, blackouts, alcohol-related arrests, or having people complain about one's drinking) are clearly alcohol-related, others are more ambiguous (eg, getting into fights, skipping classes because of one's drinking, or depression) and still others are related to associated behaviors (eg, concomitant drug use resulting in arrest for drug procurement). Youths may not attribute problems to alcohol even when they are a consequence of alcohol

TABLE 4 Possible Mechanisms Producing Associations Between Alcohol Use and Outcomes¹⁰⁸

Temporal Features	Alcohol Use Produces Outcome	Putative Outcome Produces Alcohol Use	Other Factor Produces Alcohol Use and Outcome
Acute/situational	Acute intoxication leads to DWI	Failure on a test leads to a drinking binge	Being at a party at a friend's known for fighting and drinking means one is more likely to drink and to be assaulted while drinking
Chronic/dispositional	Long-term pattern of consumption leads to depression resulting from neuroadaptational changes	Social marginalization leads to reduced social controls and increased drinking	Conduct disorder leads to both repeated binge drinking and high-risk sexual encounters

involvement (eg, conflicts with parents); therefore, assessment is a challenge. Youth drinking and adverse consequences may have 3 types of functional relationships, that is, (1) alcohol use produces the outcome, (2) the putative outcome leads to alcohol use, and (3) another behavior or factor leads to both alcohol use and the outcome.¹⁰⁸ Furthermore, the effects may occur temporarily or be more protracted (ie, acute versus chronic), as illustrated in Table 4.

Table 4 illustrates direct, indirect, and cooccurring noncausal relationships between drinking and youth outcomes. Both person-based analyses and event-based analyses showing an association between alcohol and a presumed outcome can potentially lead to incorrect inferences unless care is taken to rule out these alternative explanations. Clearly, prospective studies and event-based data on alcohol use and associated personal and environmental characteristics are needed to determine which of these groups of mechanisms are at work.

A variety of studies have shown associations between adolescent alcohol involvement and a range of adverse consequences, including academic problems, social problems, hangovers, unplanned and risky sex, aggression and victimization, unintentional injuries (especially motor vehicle crashes), various physical and emotional problems, and suicidality.⁷⁰ Strong evidence exists for adolescent alcohol involvement directly causing physical outcomes such as blackouts, unplanned sexual behavior, and motor vehicle crashes. Less clear is the casual contribution of alcohol involvement to the multiple behavioral, emotional, social, and academic problems that are correlated with teen drinking.

Alcohol and the Developing Adolescent Brain

A particularly important consequence of adolescent alcohol exposure concerns the neurologic effects of alcohol on the developing adolescent brain. As noted earlier, adolescence is now recognized as a time of rapid neuro-maturation in many key brain regions involved in self-regulation, emotional tone and reactivity, and a broad range of higher-level cognitive functions. Moreover, we know from studies comparing adult alcoholic and non-alcoholic subjects¹⁰⁹ and individuals exposed in utero to high doses of alcohol¹¹⁰ that alcohol is a potent neurotoxin. Adolescents may be especially vulnerable to perturbations in the course of neurodevelopment attributable to alcohol. Moreover, because those with a range of developmental psychopathological conditions (both in-

ternalizing and externalizing) are at highest risk for excessive alcohol involvement and the same individuals are most likely to have compromised developmental competencies and brain function before alcohol and other substance involvement, there may exist subpopulations of adolescents who not only are likely to become problematically involved in alcohol use but also are likely to experience more-severe long-term consequences because of earlier developmental compromise.

Reviews of research on neurocognitive effects of alcohol on adolescents and college students, as well as animal models of adolescence,^{6,70} concluded that adolescent alcohol use is associated with damage to the brain and neurocognitive deficits, with implications for learning and other cognitive abilities that may continue to affect the individual into adulthood. For example, adolescents with an AUD have deficits in memory retrieval and in visuospatial functioning.¹¹¹ Research relating alcohol use to brain structure and functioning supports the conclusion that heavy alcohol use in adolescence can result in selective long-term cognitive impairments. Imaging studies of brain structure¹¹² and longitudinal neurocognitive evaluations of clinical samples of adolescent drinkers¹¹¹ suggested differences between heavily alcohol-involved adolescents and control subjects, with a variety of cognitive abilities deteriorating for late adolescents and young adults who persist in heavy drinking. How extensive adolescent drinking must be before brain damage is significant and protracted, the extent of variation in vulnerability to alcohol-related brain damage, the rate and pattern of neurocognitive recovery, and the extent to which structural and functional changes are attributable solely to alcohol are not yet clear. It is clear that determining the nature, extent, and persistence of alcohol-related brain injury in adolescence is a high priority for research, because of its potential far-reaching consequences across multiple domains of human functioning.

Because of the rigorous experimental control that is possible, animal research has the power to demonstrate and to characterize the neurotoxic properties of alcohol in the developing adolescent brain.¹¹³ Much of the existing relevant research, primarily with rodents, has focused on the structure and function of the hippocampus, showing it to be especially sensitive to both acute and chronic ethanol exposure during adolescence. Acute alcohol exposure at both high and low doses seems to inhibit memory formation, and adolescent rats seem to

be unusually sensitive to these cognitive ability-impairing effects.¹¹⁴ Chronic ethanol exposure has also been shown consistently to result in long-term cognitive impairments; again, adolescent rats seem to be especially sensitive to these effects.¹¹⁵ Studies with rats also suggested that repeated periods of heavy drinking followed by withdrawal may be particularly neurotoxic, especially in adolescence.¹¹⁶ For example, chronic intermittent ethanol exposure is the rat analog of human binge drinking. Chronic intermittent ethanol exposure was associated with reduced neurogenesis in the rat hippocampus¹¹⁷ and long-term alteration of serotonergic function,¹¹⁸ likely resulting in permanently altered forebrain functioning. Therefore, adolescent vulnerability to the neurotoxic effects of alcohol may be exacerbated by the typical pattern of adolescent drinking, which often involves intermittent bouts of heavy drinking (ie, periodic binge drinking).^{116,119} Specifically, chronic intermittent ethanol exposure was associated with damage to the frontal association cortex and other frontal regions in adolescent but not adult rats.¹¹⁶

In contrast to their heightened sensitivity to the cognitive ability-impairing effects of alcohol, adolescents seem to show reduced sensitivity to other effects of alcohol. Adolescent rats are less sensitive to the motor function-impairing effects of ethanol¹²⁰; they also are less sensitive to the sedating effects of alcohol.¹²¹ This reduced sensitivity may be further decreased by typical patterns of adolescent drinking; chronic intermittent ethanol exposure has been associated with both increased tolerance to ethanol's impairing effects in a spatial learning task¹²² and altered sensitivity to ethanol's aversive effects.¹²³ Chronic intermittent ethanol exposure in adolescence also has been linked to reduced sensitivity to the motor function-impairing effects of alcohol in adulthood.¹²⁴ Moreover, in humans, reduced sensitivity to the motor function-impairing and aversive effects of alcohol has been shown to be a potent risk factor for the development of alcoholism.¹²⁵ Consequently, adolescent drinking may be especially risky because (1) some brain regions, such as the hippocampus and the frontal cortical regions, may be especially vulnerable during adolescence to the neurotoxic effects of alcohol, (2) adolescent exposure may lead to a long-term reduction in sensitivity to the cues that typically moderate alcohol consumption (ie, sedation and locomotor impairment), and (3) both of these effects are amplified by the pattern of drinking that typifies youth.

Adolescent Alcohol Involvement and Later Alcohol and Other Substance Involvement

An increasing number of studies have tracked the longer-term outcomes of adolescents who drink during later adolescence (ie, 16–20 years of age) to later time points in their third decade of life. Across a diverse range of samples and measures of alcohol involvement, there is clear evidence that drinking patterns during late adolescence are associated with alcohol-related outcomes during later life stages. Of particular interest are studies that examined developmental patterns of drinking and later diagnoses of AUDs.^{79,126,127} Not surprisingly, alcohol use during late ad-

olescence is associated with AUDs during the third decade of life, with higher probabilities of being diagnosed being associated with higher intensity of use. For example, Wells et al¹²⁷ categorized drinking patterns at age 16 and found that the maximal number of drinks consumed on a single occasion in the recent past was a key indicator of more-severe drinking patterns, both cross-sectionally and longitudinally. Across a number of studies, the relationship between alcohol involvement in later adolescence and alcohol involvement in early adulthood seems to be robust even when a host of competing factors are considered.

A number of studies have attempted to relate various aspects of middle or late adolescent drinking behavior to later outcomes in other developmentally important domains. A study by Wells et al¹²⁷ of a birth cohort in New Zealand is particularly instructive. In their analyses, those authors examined the relationship between the extent of alcohol involvement at 16 years of age and a broad range of outcomes at 21 and 25 years of age, including other (nonalcohol) substance dependence, major depression, anxiety disorders, suicidality, educational and vocational outcomes, sexual relationships, and criminal offending. Although there were many strong associations between drinking patterns at 16 years of age and these outcomes, very few could be attributed exclusively to early alcohol use. When demographic and background variables were controlled, alcohol involvement at 16 years of age predicted independently noncannabis drug dependence at 25 years of age, number of sexual partners, and property and violent offenses. Alcohol might have been an important, non-unique, contributing factor to other developmentally important outcomes. Clearly, adolescent alcohol involvement is embedded in layers of associated risk factors at the individual, intrafamilial, and extrafamilial environmental levels. Research is needed to examine the unique and combined effects of alcohol and other factors on important developmental outcomes, which represents a major challenge.

Despite this caution, longitudinal studies have clarified the long-term correlates of late adolescent drinking and demonstrated significant effects of adolescent alcohol involvement on adult outcomes. Several studies have shown that alcohol involvement is associated with adult drug dependence,^{126–128} antisocial behavior,^{127,128} and depression^{126,127}; however, the latter association has been inconsistent.¹²⁸ Adult antisocial behavior is associated with being drunk by 18 years of age, even in the absence of childhood conduct problems.

Unfortunately, there has been considerably less study of the association between adolescent alcohol involvement and other domains of adult functioning. Clearly, adolescent alcohol involvement is associated with educational problems in both secondary and higher education,^{127,128} although it is not clear whether this is uniquely attributable to alcohol. The data regarding adult outcomes associated with productive involvement in society and parental role functioning are limited but suggest possible areas of developmental impairment.¹²⁸

IMPLICATIONS FOR DEVELOPMENT OF MORE-EFFECTIVE INTERVENTIONS AND PREVENTION

Challenges in Treatment of Adolescent AUDs

The developmental framework proposed in this report provides direction to efforts to enhance interventions for youths as they transition from middle adolescence to late adolescence and into young adulthood. The salient shifts in physical, relational, social, and cultural contexts create both opportunities and challenges for efforts designed to reduce adverse alcohol-related consequences at large-scale (eg, costs to society) and small-scale (eg, individual outcomes) levels. Of note, despite the high rates of problematic alcohol involvement by teens, remarkably small proportions of teens currently seek and receive treatment specifically for alcohol use problems. In fact, traditional approaches to intervention do not seem to be optimal for adolescents and fail to reach teens before concomitant drug use problems ensue.¹²⁹ Changes in the health care system, a high threshold for care, and delays in treatment entry result in failure to capitalize on transient high motivational states of youths who want to make changes in their drinking, resulting in poor engagement of youths in the treatment process. The vast majority of treated adolescents do not seek treatment in part because of negative social stereotypes associated with traditional approaches to treatment.^{81,130}

Few forms of alcohol treatment for adolescents have clearly demonstrated efficacy. Even with treatment, the high relapse rates common among alcohol-dependent adults are paralleled among adolescents.¹³¹ School-based programs often lack ongoing supports needed for youths to sustain abstinence.¹³² Given the remarkable variety of deviant externalizing and internalizing behaviors adolescents display when entering treatment for alcohol and/or drug problems, it is not surprising that the tertiary interventions considered most promising for alcohol-abusing adolescents require substantial resources in multiple contexts to provoke and to sustain necessary lifestyle changes.¹³³ Clearly, interventions targeted at youths in earlier stages of the development of alcohol problems hold much promise for reaching a larger population, providing cost savings, and avoiding considerable personal and family hardship.

Developmentally Related Resolution of Drinking Problems

As with adults, many youths who develop and resolve alcohol problems never receive formal treatment.⁸¹ Studies examining the contexts and mechanisms of change or recovery from alcohol problems among youths are needed. Studies indicate that ~15% to ~20% of drinkers in high school make purposeful attempts to decrease or to stop their drinking and brief, developmentally tailored interventions are successful in facilitating quitting efforts.⁸¹

Even youths who receive specific treatment for alcohol and drug abuse seem to experience improvement or to maintain behavioral changes through means other than those recommended by treatment programs, reflecting developmental context and process shifts. For example, after treatment only one half of adolescent alcohol and drug abusers comply with the primary be-

havioral prescription of treatment programs, namely, attending aftercare or 12-step program meetings.¹²⁹ Approximately one third of nonattendees used other methods to abstain consistently or to have minimal alcohol involvement during the first year after treatment.¹²⁹

Identification of strategies that adolescents use in their natural environments to resolve their own alcohol problems is needed. Such information could facilitate our understanding of the process of youth change at this stage of development, potentially enhance the effectiveness of all intervention efforts, and allow treatment to reach adolescents who do not currently seek treatment.¹³⁴

Adult studies examining the self-change process for alcohol problems outside treatment have focused primarily on intentional change, such as those who want to change on their own¹³⁵ or triggers for change.¹³⁰ These studies help clarify the degree of motivation, the process of purposeful progression through change, precipitants to self-change efforts, and types of activities in which individuals engage. The issue of motivation may be particularly important for adolescents, as indicated by difficulties in enrolling adolescents into substance use cessation studies.¹³⁶ Alcohol and smoking cessation interventions among adolescents that are based on motivational interviewing principles¹³⁷ have demonstrated efficacy, and motivational enhancement techniques (eg, cognitive-behavioral skills training) have been integrated into other forms of treatment for alcohol-abusing youths. The only voluntary alcohol intervention in high schools that has been shown to increase personal quit attempts is based on these motivational enhancement principles, combined with youth preferences.⁵⁵

Few studies have examined what might be considered contextually related alcohol change, that is, change related to transitions that occur with adolescent development, such as role shifts and the socialization process.¹³⁸ Yamaguchi and Kandel¹³⁹ theorized that developmental changes in life stage-specific roles (social, academic, work, and parenting roles) influence drinking decisions as part of the process of self-selection, preparation for new roles, and anticipatory socialization. Clear examples of alcohol and drug use behavior changes with role and context changes are abundant in the adult literature (eg, heroin cessation with transition out of the Vietnam War and alcohol and drug cessation during pregnancy) and also have been demonstrated for adolescents.⁸¹ Escalation of both alcohol and drug involvement has been demonstrated among youths as they initially move to independent living¹⁰⁸ and into college environments of lower constraint. Deescalation from problem drinking levels has been reported to follow changes in peer group affiliation among 10th- to 12th-grade high school students¹⁴⁰ and to occur during periods of high academic demands among college students.⁸²

Learning theory has been applied to models of both development and treatment of alcohol problems. Learning-based models may be equally applicable to deescalation of alcohol involvement among youths in middle to late adolescence. Within this conceptual framework, alcohol use decisions result from cognitive appraisal and evaluation processes,^{135,141} which are dependent on intentionality

(motivation) and personal risk-resource balances. In particular, the developmental, social information-processing model used in the study of deviant behaviors among youths⁸¹ postulates that proximal cognitive and emotional states lead to engagement in specific behaviors (drinking) within a social context (eg, peer drinking). Grounded in cognitive science research on the manner in which individuals store and retrieve information, distribute cognitive processing resources, and ultimately solve problems, this model links distal youth risk factors (eg, biological risks, cultural experiences, and family risk and resilience factors) with proximal situations (eg, peer use, alcohol availability, reinforcement expectancies, and emotional and motivational states). Longitudinal research with adolescents suggests that these factors play a role in both the deliberate (intentional) and automatic (incidental) decisions of youths regarding alcohol use. External contingencies (eg, sports drug testing, parental threats, or changes in peer group) and/or personal experiences (eg, alcohol problems such as blackouts, nausea, or high-risk sexual behavior) may create the perception of a need for change for the adolescent. If personal resources are sufficient to cope, then more-automatic processing ensues (to use or not to use, depending on context, motivation, and personal learning history). However, when personal resources are limited (eg, undeveloped executive functioning skills, poor coping skills, or low self-efficacy) or personal or contextual risks are high (eg, abusing peers or family members, limited financial resources for alternative activities, or depression), then more-deliberative processing results. Furthermore, depending on the stage of neurocognitive development (eg, myelination of frontal and prefrontal lobes, which occurs throughout puberty), immediate and/or long-term consequences (eg, social criticism and role expectations) are incorporated into the social cognition. Such a decision-making process may result in potentially different strategies and self-regulated change efforts, as well as outcomes, during early and middle adolescence, compared with later adolescence and young adulthood.¹⁴²

The developmental social cognition model suggests that developmental factors related to initial actions in support of reduction/cessation of alcohol/drug use (eg, motivation, decisional balance, immediate contingencies, and coping skills) are different from those required to sustain behavior changes (eg, self-monitoring skills and available alternative reinforcers). Motivated youths may make efforts to reduce or to stop drinking but, without appropriate environmental "scaffolding," there may be limited likelihood of permanent change.⁸¹ This 2-phase process (ie, initial change efforts and maintenance efforts) has support in the literature with adult spontaneous remitters¹⁴¹ and treated samples¹⁴³ and with adolescent alcohol and nicotine abusers.⁵⁵

Developmental Context and Traditional Treatment Efforts

Development is a critical consideration in the design and implementation of effective interventions for adolescent alcohol problems. The rapid biological, social, behavioral, and neurocognitive changes of adolescence and the acceleration in stressful life experiences demand substantial social adjustments and skill development. A number of psy-

chosocial tasks of adolescent development are associated with alcohol use and reduction in alcohol involvement. For example, development of new social roles with same-gender and opposite-gender peers and with family members (identity), development of social skills related to new social roles, separation and individuation within the family (autonomy), and coping with developmentally specific stressors, which vary with age, gender, culture, socioeconomic status, and environment, may act to provoke or to facilitate changes (eg, successful coping, which refines skills and enhances self-esteem) or in other cases to interfere with successful task accomplishment (eg, identity as an alcohol or drug user).⁸¹

Given the diversity and intensity of developmental demands and the variations in vulnerability and resilience, it is unlikely that a singular intervention is appropriate or optimal for youths 16 to 20 years of age. As articulated in the research on college drinking, involvement of multiple systems will be necessary to produce substantial changes in high-risk drinking. At the individual level, optimal interventions to reduce or to stop alcohol involvement and to maintain change for adolescents must (1) provide personal choice to address the diversity of problems, (2) fit developmental, stage-specific motivation, (3) be consonant with youth needs to optimize engagement (eg, contextually convenient), (4) be perceived as helpful, and (5) be sufficiently diverse to reflect the heterogeneity of adolescents who experience alcohol problems (eg, social leaders, behaviorally undercontrolled youths, and youths with a history of abuse and family problems) and sensitive to alcohol-related and concomitant problems (eg, depression, anxiety, or conduct disorder).¹⁰⁵

Current, empirically validated treatments, although holding promise for youths, are limited in their context of application and generalizability to the heterogeneity of youths in need of treatment. Although pharmacotherapy is a common adjunct to psychosocial interventions for adolescents with AUDs, limited empirical evidence is available, and developmentally tailored studies are needed.¹⁴⁴ For example, despite the high rates of psychiatric comorbidity among youths with alcohol and other drug problems, few behavioral or pharmacologic studies have focused on these populations.¹⁴⁵ Treatment outcome studies clearly indicate the disadvantage to abusing/dependent youths of concomitant axis I mental health disorders and distinctive process and context differences challenging such youths.¹⁴⁶ In addition, traditional models of treatment outcomes are lacking when applied to youths, because of salient developmental differences in contextual risks, neurocognitive development, motivational distinctions, and developmentally related resources for success.

Research on youth treatment needs to incorporate developmental considerations into both design and methods. Treatment assessment procedures should be tailored to developmental stage (eg, age- and gender-typical and diagnostically subthreshold problems), using procedures and formats (eg, self-reports and computer-based biological samples) to enhance the accuracy of youths' self-reports. Studies of treatment processes (eg,

social skills competence changes in group, family, and individual interventions) and outcome measurements are needed. The timing and context of changes in relapse risk are poorly understood for youths in middle to late adolescence. Conceptual frameworks and strategies to match temporal patterns of change are needed to elucidate the diversified trajectories of use outcomes for alcohol and other drugs and are critical to improvements in treatment designs for youths. For example, multiple research groups have identified common patterns of success, fluctuations, and failure after treatment for alcohol problems; however, little is known about the impact of these outcomes on functioning as youths transition through late adolescence and into young adulthood.⁷⁰

Public Policy and Prevention of Adolescent Drinking Problems

Rationale

The most-effective interventions would be those that prevent rather than treat adolescents' problem drinking. A range of public policy initiatives have been implemented in an attempt to regulate and to eliminate teen drinking and its problems. Although these programs vary widely in their effectiveness, research suggests that some have been effective in either delaying the onset of drinking or reducing heavy problematic drinking, DWI, and alcohol-related crash involvement among people <21 years of age. A description of the most-effective policy initiatives follows.

Adolescent Drinking and Driving

Despite the legal scaffolding to protect adolescent drivers from the dangers of driving and drinking, 16- to 20-year-old youths still accounted for 12% of the 80 million trips driven in 1999 by drivers with blood alcohol concentrations of $\geq 0.10\%$.¹⁴⁷ An analysis found that there was an 11-fold increased risk of single-vehicle fatal crash involvement in all age and gender groups at a blood alcohol concentration of 0.08%, compared with 0%. However, for 16- to 20-year-old male drivers with blood alcohol concentrations of 0.08%, there was a 52-fold increase in single-vehicle fatal crash risk.¹⁴⁸ Unfortunately, progress in reducing alcohol-related fatal crashes among adolescents has stalled. The proportions of fatally injured, 16- to 20-year-old drivers with blood alcohol concentrations of $\geq 0.08\%$ were 29% in both 1995 and 2004.

One of the most successful interventions has been the adoption of age 21 as the legal drinking age. One national study of laws raising the drinking age to 21 indicated that persons who grew up in states with a drinking age of 21, relative to those who grew up in states with lower legal drinking ages, drank less not only when they were <21 years of age but also when they were 21 to 25 years of age.¹⁴⁹ A review of 49 studies of changes in the legal drinking age revealed that, in the 1970s and 1980s, when many states lowered the drinking age, alcohol-related traffic crashes increased 10%. In contrast, when states increased the legal drinking age to 21, alcohol-related crashes among people <21 years of age decreased an average of 16%.¹⁵⁰ The National Highway Traffic Administration estimates that a legal drinking age

of 21 has prevented >21 000 traffic deaths since 1976. Furthermore, zero-tolerance laws, which make it illegal for drivers <21 years of age to drive after any drinking, have been associated with ~20% decreases in alcohol-related fatal crashes and DWIs.¹⁵¹

A number of school-based institutional programs have sought to reduce driving after drinking or riding with a drinking driver by targeting life skills and refusal skills. A review of 5 studies involving 6 programs¹⁵² indicated reductions in riding with drinking drivers and reductions in driving after drinking in conjunction with several of those programs. Furthermore, experimental studies in trauma center and emergency departments with youths who experienced alcohol-related injuries indicated that screening and brief intervention counseling for alcohol problems in this population were associated with reductions in drinking and driving offenses and alcohol-related injuries.

Even with laws such as zero-tolerance laws in place, many youths continue to drink and to drive after drinking. Education about the laws and how they are enforced and actual enforcement by police and courts are critical to the optimal success of these laws in reducing rates of alcohol-impaired driving and related crash involvement. Enforcement of these laws generally has not been very vigorous.¹⁵³ Moreover, studies have found that young drivers are substantially underrepresented in the DWI arrest population, relative to their contributions to the alcohol crash problem.¹⁵⁴ One reason for this may be that younger drivers are more likely to drink at locations where DWI enforcement resources are less likely to be deployed. Furthermore, given the lower sedative impact of alcohol on adolescents, young drivers with high blood alcohol concentrations are more likely to be missed by police at sobriety checks.¹⁵⁵ Enforcement of zero-tolerance laws is hindered in some states because of the way the laws are written. For example, in some states the implied-consent laws require either an arrest for DWI or probable cause for a DWI arrest before the evidentiary test can be performed to prove a zero-tolerance violation. Therefore, in practice, zero-tolerance laws often are not enforced independently of DWI. In states such as New Mexico, where this situation exists, the majority of teenagers are not aware that there is a zero-tolerance law.¹⁵⁶ Of note, a combined media and enforcement campaign in 2 Maryland counties after enactment of a zero-tolerance law in that state yielded greater awareness of the law and a one third greater decrease in alcohol-related crashes among people <21 years of age in those counties, compared with the rest of the state.¹⁵⁷

Other programs may also be effective in reducing the prevalence of teen drinking and driving. Systematic reviews of the relevant research literature found that sobriety checkpoints and mass media campaigns¹⁵⁸ can reduce rates of alcohol-related traffic crashes. However, the specific effects on drivers <21 years of age have not been analyzed. Targeting servers and sellers with the enforcement of laws governing the purchase of alcohol can also be an effective strategy for reducing problem teen drinking.¹⁵⁹ Research on the effects of price on

alcohol consumption indicates that, as price increases, consumption decreases.¹⁶⁰ Furthermore, increasing the price of alcohol has, with rare exceptions, been associated with reductions in rates of motor vehicle deaths.¹⁶¹ Studies of the effects of keg registration are limited but suggest that registration of kegs is negatively correlated with traffic fatality rates.¹⁶²

Comprehensive Community Interventions

Several carefully conducted, community-based interventions have had particular success in reducing alcohol-related traffic crashes and deaths. These programs typically coordinate efforts of city governments, schools, health agencies, police, private citizens and their organizations, students, parents, and merchants who sell alcohol. Six comprehensive community programs have shown significant reductions in alcohol problems, including driving after drinking, among adolescents and adults, that is, the Communities Mobilizing for Change Program,^{159,163} the Community Trials Program,¹⁶⁴ the Saving Lives Program,¹⁶⁵ the Matter of Degree Program,¹⁶⁶ a college community intervention,¹⁶⁷ and the Fighting Back Program.¹⁶⁸ Two programs^{159,163,164} concentrated efforts on underage purchase attempt surveys, with feedback to alcohol sellers and the community about the proportion of attempts that resulted in sales and the penalties for continued violation. Three programs^{163,164,166} used environmental interventions to reduce alcohol availability to underage drinkers. Three programs^{164,165,167} focused on publicized police enforcement of drunk driving laws, and 1 program targeted risky motorist behaviors engaged in disproportionately by drinking drivers, such as speeding, running red lights, and failing to wear safety belts and yield to pedestrians in crosswalks.¹⁶⁵ Most recently, another group of communities combined efforts to increase participation in individually oriented substance abuse treatment with environmental interventions to reduce alcohol availability.¹⁶⁸

Thinking About Legal Transitions and Access to Alcohol

At the present time, society's aim of curtailing youthful drinking is implemented by legal prohibition, using an approach that is both binary (legal or not) and categorical (based on a simple age classification). As discussed above, this relatively simple approach to regulating adolescent access to alcohol has been shown to be effective in reducing some forms of problem drinking. However, some have advocated a more-complex framework in which a transitional phase of conditional access (a "learning period") is inserted between the periods of prohibited access and unrestricted access to alcohol. The concept of a learning period or supervised drinking has often been mentioned, not as an interim legal classification but as an argument for adopting a drinking age of 16 or 18 (instead of 21). The argument for doing this is that the proper social response to inevitable youthful drinking is to teach youths to drink responsibly, rather than trying to repress use altogether. However, it is possible to envision the learning period as a period of legal transition, rather than as an argument for embracing a lower

legal drinking age. We already have a model available for such a transition phase in the licensing of drivers, in which young drivers (typically at age 16) are licensed for driving during the daytime and without backseat passengers.

Of interest, many countries throughout the world allow transitional access to alcohol at much younger ages than in the United States (typically age 18, but in some cases age 16 or even younger).¹⁶⁹ In most of those countries, however, youth access to driving is either prohibited or severely limited, by law, custom, or economic necessity. Consequently, hazardous drinking while driving is much less a problem in those countries than it is in the United States. In any case, in the absence of research, we can only speculate regarding whether a transitional period of access to alcohol during adolescence would be beneficial or harmful. Indeed, casting the issue in this dichotomous manner may itself be overly simplistic. Although provisional access might be beneficial for a large number of adolescents, it might be especially harmful for the minority of adolescents with high levels of preexisting risk (eg, externalizing psychopathological conditions or family dysfunction).

CONCLUSIONS

The period from 16 to 20 years of age, late adolescence, is a period of extensive and rapid transition in virtually every domain of life functioning. It is also a period of continued neurologic, cognitive, and social maturation. These developmental changes and contexts set the stage for late adolescents' increasing involvement with alcohol. Increased autonomy, reduced parental monitoring, and greater involvement with peers all create the opportunity for psychological growth; they also create a context for the emergence of problem drinking. An alarming number of late adolescents, especially those who are out of high school, engage regularly in hazardous drinking. Although these problematic drinking practices are widespread among late adolescents, they are not universal. Adolescents with a history of behavioral problems and a family history of alcoholism seem especially prone to engaging in problem drinking.

Although problem drinking may represent a transient phase in the lives of many adolescents, for others it can have profound and life-altering effects. Adolescence is now recognized as a period of continued neurologic development, and the adolescent brain may be especially vulnerable to the neurotoxic effects of alcohol, especially given the typical ways in which youths drink. Problem drinking in late adolescence is directly linked to increased risk of an AUD in early adulthood. Less clear is how problem drinking influences other life outcomes, including risk for mental health problems and social achievement. Although there have been some notable successes, most efforts to treat and/or to prevent adolescent drinking have had limited success. This may be because many of the interventions have been modeled on those used with adults, rather than being tailored to the unique cognitive and social context of late adolescence.

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REFERENCES

- Schulenberg J, Maggs JL, Hurrelmann K. *Health Risks and Developmental Transitions During Adolescence*. New York, NY: Cambridge University Press; 1997
- Aseltine RH, Gore S. Mental health and social adaptation following the transition from high school. *J Res Adolesc*. 1993; 3(3):247–270
- Hogan D, Astone N. The transition to adulthood. *Annu Rev Sociol*. 1986;12:109–130
- Fussell E, Furstenberg FFJ. The transition to adulthood during the twentieth century: race, nativity, and gender. In: Settersten RA, Furstenberg FFJ, Rumbaut RG, eds. *On the Frontier of Adulthood: Theory, Research, and Public Policy*. Chicago, IL: University of Chicago Press; 2005:29–75
- Settersten RA, Furstenberg FFJ, Rumbaut RG, eds. *On the Frontier of Adulthood: Theory, Research, and Public Policy*. Chicago, IL: University of Chicago Press; 2005
- Spear LP. The adolescent brain and age-related behavioral manifestations. *Neurosci Biobehav Rev*. 2000;24(4):417–463
- Gogtay N, Giedd JN, Lusk L, et al. Dynamic mapping of human cortical development during childhood through early adulthood. *Proc Natl Acad Sci USA*. 2004;101(21):8174–8179
- Giedd JN, Blumenthal J, Jeffries NO, et al. Brain development during childhood and adolescence: a longitudinal MRI study. *Nature Neurosci*. 1999;2(10):861–863
- Paus T, Zijdenbos A, Worsley K, et al. Structural maturation of neural pathways in children and adolescents: in vivo study. *Science*. 1999;283(5409):1908–1911
- Carskadon MA. Patterns of sleep and sleepiness in adolescents. *Pediatrician*. 1990;17(1):5–12
- Wolfson AR, Carskadon MA. Sleep schedules and daytime functioning in adolescents. *Child Dev*. 1998;69(4):875–887
- National Institutes of Health. *Problem Sleepiness in Your Patient*. Bethesda, MD: National Institutes of Health; 1997
- Roehrs T, Beare D, Zorick F, Roth T. Sleepiness and ethanol effects on simulated driving. *Alcohol Clin Exp Res*. 1994;18(1): 154–158
- Raffaelli M, Crockett LJ, Shen YL. Developmental stability and change in self-regulation from childhood to adolescence. *J Genet Psychol*. 2005;166(1):54–75
- Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 predict adult psychiatric disorders: longitudinal evidence from a birth cohort. *Arch Gen Psychiatry*. 1996; 53(11):1033–1039
- Elkins IJ, McGue M, Iacono WG. Prospective effects of attention-deficit/hyperactivity disorder, conduct disorder, and sex on adolescent substance use and abuse. *Arch Gen Psychiatry*. 2007;64(10):1145–1152
- Kirisci L, Tarter RE, Vanyukov M, Reynolds M, Habeych M. Relation between cognitive distortions and neurobehavior disinhibition on the development of substance use during adolescence and substance use disorder by young adulthood: a prospective study. *Drug Alcohol Depend*. 2004;76(2):125–133
- Reyna VF, Farley F. Risk and rationality in adolescent decision making: implications for theory, practice, and public policy. *Psychol Sci Public Interest*. 2006;7(1):1–44
- Steinberg L. Risk taking in adolescence: what changes, and why? *Ann NY Acad Sci*. 2004;1021:51–58
- Gardner M, Steinberg L. Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: an experimental study. *Dev Psychol*. 2005;41(4): 625–635
- Nowinski J. *Substance Abuse in Adolescents and Young Adults*. New York, NY: Norton; 1990
- Christ SE, White DA, Mandernach T, Keys BA. Inhibitory control across the life span. *Dev Neuropsychol*. 2001;20(3): 653–669
- Jarman RF, Vavrik J, Walton PD. Metacognitive and frontal lobe processes: at the interface of cognitive psychology and neuropsychology. *Genet Soc Gen Psychol Monogr*. 1995;121(2): 153–210
- Suzuki-Slakter N. Elaboration and metamemory during adolescence. *Contemp Educ Psychol*. 1988;13(3):206–220
- Levin HS, Culhane KA, Hartmann J, et al. Developmental changes in performance on tests of purported frontal lobe functioning. *Dev Neuropsychol*. 1991;7(3):377–395
- Davies PL, Rose JD. Assessment of cognitive development in adolescents by means of neuropsychological tasks. *Dev Neuropsychol*. 1999;15(2):227–248
- Mueller U, Overton WF, Reene K. Development of conditional reasoning: a longitudinal study. *J Cogn Dev*. 2001;2(1): 27–49
- Klaczynski PA. Analytic and heuristic processing influences on adolescent reasoning and decision-making. *Child Dev*. 2001;72(3):844–861
- Cauffman E, Steinberg L. (Im)maturity of judgment in adolescence: why adolescents may be less culpable than adults. *Behav Sci Law*. 2000;18(6):741–760
- Ormond C, Luszcz MA, Mann L, Beswick G. A metacognitive analysis of decision making in adolescence. *J Adolesc*. 1991; 14(3):275–291
- Ernst M, Grant SJ, London ED, Contoreggi CS, Kimes AS, Spurgeon L. Decision making in adolescents with behavior disorders and adults with substance abuse. *Am J Psychiatry*. 2003;160(1):33–40
- Baddeley A. The episodic buffer: a new component of working memory? *Trends Cogn Sci*. 2000;4(11):417–423
- Luna B, Garver KE, Urban TA, Lazar NA, Sweeney JA. Maturation of cognitive processes from late childhood to adulthood. *Child Dev*. 2004;75(5):1357–1372
- Kwon H, Reiss AL, Menon V. Neural basis of protracted developmental changes in visuo-spatial working memory. *Proc Natl Acad Sci USA*. 2002;99(20):13336–13341
- Thomas KM, King SW, Franzen PL, et al. A developmental functional MRI study of spatial working memory. *Neuroimage*. 1999;10(3):327–338
- Klingberg T, Forssberg H, Westerberg H. Increased brain activity in frontal and parietal cortex underlies the development of visuospatial working memory capacity during childhood. *J Cogn Neurosci*. 2002;14(1):1–10
- Kim MS, Kim JJ, Kwon JS. Frontal P300 decrement and executive dysfunction in adolescents with conduct problems. *Child Psychiatry Hum Dev*. 2001;32(2):93–106
- Deckel AW. Tests of executive functioning predict scores on the MacAndrew Alcoholism Scale. *Prog Neuropsychopharmacol Biol Psychiatry*. 1999;23(2):209–223
- Hill SY, Steinhauer S, Park J, Zubin J. Event-related potential characteristics in children of alcoholics from high density families. *Alcohol Clin Exp Res*. 1990;14(1):6–16
- Milberger S, Biederman J, Faraone SV, Wilens T, Chu MP. Associations between ADHD and psychoactive substance use disorders: findings from a longitudinal study of high-risk siblings of ADHD children. *Am J Addict*. 1997;6(4):318–329

41. Deckel AW, Bauer L, Hesselbrock V. Anterior brain dysfunctioning as a risk factor in alcoholic behaviors. *Addiction*. 1995; 90(10):1323–1334
42. Sobock J, Abbey A, Agius E, Clinton M, Harrison K. Predicting early adolescent substance use: do risk factors differ depending on age of onset? *J Subst Abuse*. 2000;11(1):89–102
43. Deckel AW, Hesselbrock V. Behavioral and cognitive measurements predict scores on the MAST: a 3-year prospective study. *Alcohol Clin Exp Res*. 1996;20(7):1173–1178
44. Giancola PR, Moss HB, Martin CS, Kirisci L, Tarter RE. Executive cognitive functioning predicts reactive aggression in boys at high risk for substance abuse: a prospective study. *Alcohol Clin Exp Res*. 1996;20(4):740–744
45. Giancola PR, Shoal GD, Mezzich AC. Constructive thinking, executive functioning, antisocial behavior, and drug use involvement in adolescent females with a substance use disorder. *Exp Clin Psychopharmacol*. 2001;9(2):215–227
46. Giancola PR, Parker AM. A six-year prospective study of pathways toward drug use in adolescent boys with and without a family history of a substance use disorder. *J Stud Alcohol*. 2001;62(2):166–178
47. Blume AW, Marlatt GA, Schmalting KB. Executive cognitive function and heavy drinking behavior among college students. *Psychol Addict Behav*. 2000;14(3):299–302
48. Moskowitz JM. Preventing adolescent substance abuse through drug education. *NIDA Res Monogr*. 1983;47:233–249
49. Botvin GJ. Preventing drug abuse in schools: social and competence enhancement approaches targeting individual-level etiologic factors. *Addict Behav*. 2000;25(6):887–897
50. Perry CL, Williams CL, VeblenMortenson S, et al. Project Northland: outcomes of a communitywide alcohol use prevention program during early adolescence. *Am J Public Health*. 1996;86(7):956–965
51. McCoy CB, Metsch LR, Inciardi JA. *Intervening With Drug-Involved Youth*. Thousand Oaks, CA: Sage Publications; 1996
52. Snow DL, Tebes JK, Arthur MW, Tapasak RC. 2-Year follow-up of a social-cognitive intervention to prevent substance use. *J Drug Educ*. 1992;22(2):101–114
53. Ellickson PL, Bell RM, McGuigan K. Preventing adolescent drug use: long-term results of a junior high program. *Am J Public Health*. 1993;83(6):856–861
54. Perry CL, Williams CL, Forster JL, et al. Background, conceptualization and design of a community-wide research program on adolescent alcohol use: Project Northland. *Health Educ Res*. 1993;8(1):125–136
55. Brown SA, Anderson KG, Schulte MT, Sintov ND, Frissell KC. Facilitating youth self-change through school-based intervention. *Addict Behav*. 2005;30(9):1797–1810
56. Baer PE, Bray JH. Adolescent individuation and alcohol use. *J Stud Alcohol Suppl*. 1999;13:52–62
57. Barnes GM, Reifman AS, Farrell MP, Dintcheff BA. The effects of parenting on the development of adolescent alcohol misuse: a six-wave latent growth model. *J Marriage Fam*. 2000;62(1):175–186
58. Brody GH, Ge X, Katz J, Arias IA. A longitudinal analysis of internalization of parental alcohol-use norms and adolescent alcohol use. *Appl Dev Sci*. 2000;4(2):71–79
59. Windle M. Parental, sibling, and peer influences on adolescent substance use and alcohol problems. *Appl Dev Sci*. 2000; 4(2):98–110
60. McGue M, Sharma A, Benson P. Parent and sibling influences on adolescent alcohol use and misuse: evidence from a US adoption cohort. *J Stud Alcohol*. 1996;57(1):8–18
61. Schulenberg JE, Maggs JL. A developmental perspective on alcohol use and heavy drinking during adolescence and the transition to young adulthood. *J Stud Alcohol Suppl*. 2002;(14): 54–70
62. Brown JD, Witherspoon EM. The mass media and American adolescents' health. *J Adolesc Health*. 2002;31(6 suppl): 153–170
63. Brooks-Gunn J, Paikoff R. "Sex is a gamble, kissing is a game": adolescent sexuality and health promotion. In: Millstein SG, Petersen AC, Nightingale EO, eds. *Promoting the Health of Adolescents: New Directions for the Twenty-First Century*. New York: 180–208
64. Kuttler AF, La Greca AM. Linkages among adolescent girls' romantic relationships, best friendships, and peer networks. *J Adolesc*. 2004;27(4):395–414
65. Alan Guttmacher Institute. *Sex and America's Teenagers*. New York, NY: Alan Guttmacher Institute; 1994
66. Gowen LK, Feldman SS, Diaz R, Yisrael DS. A comparison of the sexual behaviors and attitudes of adolescent girls with older vs similar-aged boyfriends. *J Youth Adolesc*. 2004;33(2): 167–175
67. Brown SA, Abrantes AM. Substance use disorders. In: Wolfe DA, Mash EJ, eds. *Behavioral and Emotional Disorders in Adolescents*. New York, NY: Guilford Press; 2005:226–256
68. Abrantes AM, Brown SA, Tomlinson K. Psychiatric comorbidity among inpatient substance abusing adolescents. *J Child Adolesc Subst Abuse*. 2003;13(2):83–101
69. Kandel DB, Johnson JG, Bird HR, et al. Psychiatric comorbidity among adolescents with substance use disorders: findings from the MECA Study. *J Am Acad Child Adolesc Psychiatry*. 1999;38(6):693–699
70. Tomlinson KL, Brown SA, Abrantes A. Psychiatric comorbidity and substance use treatment outcomes of adolescents. *Psychol Addict Behav*. 2004;18(2):160–169
71. Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE. *Monitoring the Future: National Survey Results on Drug Use, 1975–2005, Vol 1: Secondary School Students*. Bethesda, MD: National Institute on Drug Abuse; 2006. NIH publication 06–5883
72. Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE. *Monitoring the Future: National Survey Results on Drug Use, 1975–2005, Vol 2: College Students and Adults Ages 19–45*. Bethesda, MD: National Institute on Drug Abuse; 2006. NIH publication 06–5884
73. Jessor R. Risk behavior in adolescence: a psychosocial framework for understanding and action. *J Adolesc Health*. 1991; 12(8):597–605
74. Maggs JL, Schulenberg JE. Initiation and course of alcohol consumption among adolescents and young adults. *Recent Dev Alcohol*. 2005;17:29–47
75. Muthén BO, Muthén LK. Integrating person-centered and variable-centered analyses: growth mixture modeling with latent trajectory classes. *Alcohol Clin Exp Res*. 2000;24(6): 882–891
76. Schulenberg J, Maggs JL. Moving targets: modeling developmental trajectories of adolescent alcohol misuse, individual and peer risk factors, and intervention effects. *Appl Dev Sci*. 2001;5(4):237–253
77. Jackson KM, Sher KJ, Schulenberg JE. Conjoint developmental trajectories of young adult alcohol and tobacco use. *J Abnorm Psychol*. 2005;114(4):612–626
78. White HR, Johnson V, Buysse S. Parental modeling and parenting behavior effects on offspring alcohol and cigarette use: a growth curve analysis. *J Subst Abuse*. 2000;12(3):287–310
79. Chassin L, Pitts SC, Prost J. Binge drinking trajectories from adolescence to emerging adulthood in a high-risk sample: predictors and substance abuse outcomes. *J Consult Clin Psychol*. 2002;70(1):67–78
80. Schulenberg J, O'Malley PM, Bachman JG, Wadsworth KN, Johnston LD. Getting drunk and growing up: trajectories of

- frequent binge drinking during the transition to young adulthood. *J Stud Alcohol*. 1996;57(3):289–304
81. D'Amico EJ, Metrik J, McCarthy DM, Appelbaum M, Frissell KC, Brown SA. Progression into and out of binge drinking among high school students. *Psychol Addict Behav*. 2001;15(4):341–349
 82. Greenbaum PE, Del Boca FK, Darkes J, Wang CP, Goldman MS. Variation in the drinking trajectories of freshmen college students. *J Consult Clin Psychol*. 2005;73(2):229–238
 83. Maggs JL, Almeida DM, Galambos NL. Risky business: the paradoxical meaning of problem behavior for young adolescents. *J Early Adolesc*. 1995;15(3):344–362
 84. Maggs JL. Alcohol use and binge drinking as goal-directed action during the transition to post-secondary education. In: Schulenberg J, Maggs JL, Hurrelmann K, eds. *Health Risks and Developmental Transitions During Adolescence*. New York, NY: Cambridge University Press; 1997:345–371
 85. Baumrind D, Moselle KA. A development perspective on adolescent drug abuse. *Adv Alcohol Subst Abuse*. 1985;4(3–4):41–67
 86. Vanyukov MM, Tarter RE, Kirisci L, Kirillova GP, Maher BS, Clark DB. Liability to substance use disorders, part I: common mechanisms and manifestations. *Neurosci Biobehav Rev*. 2003;27(6):507–515
 87. Hussong AM, Curran PJ, Moffitt TE, Caspi A, Carrig MM. Substance abuse hinders desistance in young adults' antisocial behavior. *Dev Psychopathol*. 2004;16(4):1029–1046
 88. McGue M, Iacono WG, Legrand LN, Malone S, Elkins I. Origins and consequences of age at first drink, part I: associations with substance-use disorders, disinhibitory behavior and psychopathology, and P3 amplitude. *Alcohol Clin Exp Res*. 2001;25(8):1156–1165
 89. Begleiter H, Porjesz B, Bihari B, Kissin B. Event-related brain potentials in boys at risk for alcoholism. *Science*. 1984;225(4669):1493–1496
 90. Keyes MA, Iacono WG, McGue M. Early onset problem behavior, young adult psychopathology, and contextual risk. *Twin Res Hum Genet*. 2007;10(1):45–53
 91. Rose RJ, Dick DM, Viken RJ, Kaprio J. Gene-environment interaction in patterns of adolescent drinking: regional residency moderates longitudinal influences on alcohol use. *Alcohol Clin Exp Res*. 2001;25(5):637–643
 92. Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. *J Abnorm Psychol*. 2002;111(3):411–424
 93. Rutter M, Silberg J. Gene-environment interplay in relation to emotional and behavioral disturbance. *Annu Rev Psychol*. 2002;53:463–490
 94. Slutske WS, Hunt-Carter EE, Nabors-Oberg RE, et al. Do college students drink more than their non-college-attending peers? Evidence from a population-based longitudinal female twin study. *J Abnorm Psychol*. 2004;113(4):530–540
 95. Bartholow BD, Sher KJ, Krull JL. Changes in heavy drinking over the third decade of life as a function of collegiate fraternity and sorority involvement: a prospective, multilevel analysis. *Health Psychol*. 2003;22(6):616–626
 96. Bachman JG, Schulenberg JE. How part-time work intensity relates to drug use, problem behavior, time use, and satisfaction among high school seniors: are these consequences or merely correlates? *Dev Psychol*. 1993;29(2):220–235
 97. Mortimer JT, Staff J. Early work as a source of developmental discontinuity during the transition to adulthood. *Dev Psychopathol*. 2004;16(4):1047–1070
 98. Martino SC, Collins RL, Ellickson PL. Substance use and early marriage. *J Marriage Fam*. 2004;66(1):244–257
 99. Lanza ST, Collins LM. Pubertal timing and the onset of substance use in females during early adolescence. *Prev Sci*. 2002;3(1):69–82
 100. Moffitt TE, Caspi A, Rutter M, Silva PA. *Sex Differences in Antisocial Behaviour: Conduct Disorder, Delinquency and Violence in the Dunedin Longitudinal Study*. New York, NY: Cambridge University Press; 2001
 101. Wallace JM Jr, Bachman JG, O'Malley PM, Schulenberg JE, Cooper SM, Johnston LD. Gender and ethnic differences in smoking, drinking and illicit drug use among American 8th, 10th and 12th grade students, 1976–2000. *Addiction*. 2003;98(2):225–234
 102. Champion HL, Foley KL, DuRant RH, Hensberry R, Altman D, Wolfson M. Adolescent sexual victimization, use of alcohol and other substances, and other health risk behaviors. *J Adolesc Health*. 2004;35(4):321–328
 103. Poulin C, Hand D, Boudreau B, Santor D. Gender differences in the association between substance use and elevated depressive symptoms in a general adolescent population. *Addiction*. 2005;100(4):525–535
 104. Johnston LD, O'Malley PM, Bachman JG. *Monitoring the Future: National Survey Results on Drug Use, 1975–2000, Vol 1: Secondary School Students*. Rockville, MD: National Institute on Drug Abuse; 2001. NIH publication No. 01-4924
 105. Zucker RA, Wong MA, Puttler LI, Fitzgerald HE. Resilience and vulnerability among sons of alcoholics: relationships to developmental outcomes between early childhood and adolescence. In: Luthar SS, ed. *Resilience and Vulnerability: Adaptation in the Context of Childhood Adversities*. New York, NY: Cambridge University Press; 2003:76–103
 106. Cicchetti D, Rogosch FA. The role of self-organization in the promotion of resilience in maltreated children. *Dev Psychopathol*. 1997;9(4):797–815
 107. Stice E, Barrera M, Chassin L. Prospective differential prediction of adolescent alcohol use and problem use: examining the mechanisms of effect. *J Abnorm Psychol*. 1998;107(4):616–628
 108. Stice E, Myers MG, Brown SA. Relations of delinquency to adolescent substance use and problem use: a prospective study. *Psychol Addict Behav*. 1998;12(2):136–146
 109. Oscar-Berman M, Marinkovic K. Alcoholism and the brain: an overview. *Alcohol Res Health*. 2003;27(2):125–133
 110. Testa M, Quigley BM, Eiden RD. The effects of prenatal alcohol exposure on infant mental development: a meta-analytical review. *Alcohol Alcohol*. 2003;38(4):295–304
 111. Brown SA, Tapert SF, Granholm E, Delis DC. Neurocognitive functioning of adolescents: effects of protracted alcohol use. *Alcohol Clin Exp Res*. 2000;24(2):164–171
 112. De Bellis MD, Clark DB, Beers SR, et al. Hippocampal volume in adolescent-onset alcohol use disorders. *Am J Psychiatry*. 2000;157(5):737–744
 113. Hiller-Sturmhofel S, Swartzwelder HS. Alcohol's effects on the adolescent brain: what can be learned from animal models. *Alcohol Res Health*. 2004;28(4):213–221
 114. Blitzer RD, Gil O, Landau EM. Long-term potentiation in rat hippocampus is inhibited by low concentrations of ethanol. *Brain Res*. 1990;537(1–2):203–208
 115. Sircar R, Sircar D. Adolescent rats exposed to repeated ethanol treatment show lingering behavioral impairments. *Alcohol Clin Exp Res*. 2005;29(8):1402–1410
 116. Crews FT, Braun CJ, Hoplight B, Switzer RC III, Knapp DJ. Binge ethanol consumption causes differential brain damage in young adolescent rats compared with adult rats. *Alcohol Clin Exp Res*. 2000;24(11):1712–1723
 117. Nixon K, Crews FT. Binge ethanol exposure decreases neurogenesis in adult rat hippocampus. *J Neurochem*. 2002;83(5):1087–1093
 118. Obernier JA, White AM, Swartzwelder HS, Crews FT. Cogni-

- tive deficits and CNS damage after a 4-day binge ethanol exposure in rats. *Pharmacol Biochem Behav.* 2002;72(3):521–532
119. Miller JW, Naimi TS, Brewer RD, Jones SE. Binge drinking and associated health risk behaviors among high school students. *Pediatrics.* 2007;119(1):76–85
 120. White AM, Ghia AJ, Levin ED, Swartzwelder HS. Binge pattern ethanol exposure in adolescent and adult rats: differential impact on subsequent responsiveness to ethanol. *Alcohol Clin Exp Res.* 2000;24(8):1251–1256
 121. Little PJ, Kuhn CM, Wilson WA, Swartzwelder HS. Differential effects of ethanol in adolescent and adult rats. *Alcohol Clin Exp Res.* 1996;20(8):1346–1351
 122. Silvers JM, Tokunaga S, Mittleman G, O'Buckley T, Morrow AL, Matthews DB. Chronic intermittent ethanol exposure during adolescence reduces the effect of ethanol challenge on hippocampal allopregnanolone levels and Morris water maze task performance. *Alcohol.* 2006;39(3):151–158
 123. Graham DL, Diaz-Granados JL. Periadolescent exposure to ethanol and diazepam alters the aversive properties of ethanol in adult mice. *Pharmacol Biochem Behav.* 2006;84(3):406–414
 124. White AM, Bae JG, Truesdale MC, Ahmad S, Wilson WA, Swartzwelder HS. Chronic-intermittent ethanol exposure during adolescence prevents normal developmental changes in sensitivity to ethanol-induced motor impairments. *Alcohol Clin Exp Res.* 2002;26(7):960–968
 125. Schuckit MA, Smith TL. An 8-year follow-up of 450 sons of alcoholic and control subjects. *Arch Gen Psychiatry.* 1996;53(3):202–210
 126. Brook DW, Brook JS, Zhang CS, Cohen P, Whiteman M. Drug use and the risk of major depressive disorder, alcohol dependence, and substance use disorders. *Arch Gen Psychiatry.* 2002;59(11):1039–1044
 127. Wells JE, Horwood LJ, Fergusson DM. Drinking patterns in mid-adolescence and psychosocial outcomes in late adolescence and early adulthood. *Addiction.* 2004;99(12):1529–1541
 128. Hill KG, White HR, Chung IJ, Hawkins JD, Catalano RF. Early adult outcomes of adolescent binge drinking: person- and variable-centered analyses of binge drinking trajectories. *Alcohol Clin Exp Res.* 2000;24(6):892–901
 129. Brown SA. Recovery patterns in adolescent substance abuse. In: Baer JR, Marlatt GA, McMahan RJ, eds. *Addictive Behaviors Across the Lifespan: Prevention, Treatment, and Policy Issues.* Beverly Hills, CA: Sage Publications; 1993
 130. Tucker JA. Predictors of help-seeking and the temporal relationship of help to recovery among treated and untreated recovered problem drinkers. *Addiction.* 1995;90(6):805–809
 131. Brown SA, Myers MG, Mott MA, Vik PW. Correlates of success following treatment for adolescent substance abuse. *Appl Prev Psychol.* 1994;3(2):61–73
 132. Zunz SJ, Ferguson NL, Senter M. Post-identification support for substance dependent students in school-based programs: the weakest link. *J Child Adolesc Subst Abuse.* 2005;14(4):77–92
 133. Waldron HB, Flicker SM, Hersen M. Alcohol and drug abuse. In: Hersen M, ed. *Clinical Behavior Therapy: Adults and Children.* Hoboken, NJ: Wiley; 2002:474–490
 134. Sobell LC, Cunningham JA, Sobell MB. Recovery from alcohol problems with and without treatment: prevalence in two population surveys. *Am J Public Health.* 1996;86(7):966–972
 135. Sobell LC, Sobell MB, Toneatto T, Leo GI. What triggers the resolution of alcohol problems without treatment. *Alcohol Clin Exp Res.* 1993;17(2):217–224
 136. Peltier B, Telch MJ, Coates TJ. Smoking cessation with adolescents: a comparison of recruitment strategies. *Addict Behav.* 1982;7(1):71–73
 137. Colby SM, Monti PM, Barnett NP, et al. Brief motivational interviewing in a hospital setting for adolescent smoking: a preliminary study. *J Consult Clin Psychol.* 1998;66(3):574–578
 138. Watson AL, Sher KJ. Resolution of alcohol problems without treatment: methodological issues and future directions of natural recovery research. *Clin Psychol Sci Pract.* 1998;5(1):1–18
 139. Yamaguchi K, Kandel DB. On the resolution of role incompatibility: a life event history analysis of family roles and marijuana use. *Am J Sociol.* 1985;90(6):1284–1325
 140. Stice E, Myers MG, Brown SA. A longitudinal grouping analysis of adolescent substance use escalation in de-escalation. *Psychol Addict Behav.* 1998;12(1):14–27
 141. Klingemann HK. The motivation for change from problem alcohol and heroin use. *Br J Addict.* 1991;86(6):727–744
 142. Tapert SF, Brown SA, Myers MG, Granholm E. The role of neurocognitive abilities in coping with adolescent relapse to alcohol and drug use. *J Stud Alcohol.* 1999;60(4):500–508
 143. Cronkite RC, Moos RH. Determinants of the post-treatment functioning of alcoholic patients: a conceptual-framework. *J Consult Clin Psychol.* 1980;48(3):305–316
 144. Dawes MA, Johnson BA. Pharmacotherapeutic trials in adolescent alcohol use disorders: opportunities and challenges. *Alcohol Alcohol.* 2004;39(3):166–177
 145. Cornelius JR, Clark DB, Bukstein OG, Kelly TM, Salloum IM, Wood DS. Fluoxetine in adolescents with comorbid major depression and an alcohol use disorder: a 3-year follow-up study. *Addict Behav.* 2005;30(4):807–814
 146. McCarthy DM, Tomlinson KL, Anderson KG, Marlatt GA, Brown SA. Relapse in alcohol- and drug-disordered adolescents with comorbid psychopathology: changes in psychiatric symptoms. *Psychol Addict Behav.* 2005;19(1):28–34
 147. National Highway Traffic Safety Administration. *Traffic Safety Facts 2000: Alcohol.* Washington, DC: National Highway Traffic Safety Administration; 2000. Report DOT HS-809–323
 148. Zador PL, Krawchuk SA, Voas RB. Alcohol-related relative risk of driver fatalities and driver involvement in fatal crashes in relation to driver age and gender: an update using 1996 data. *J Stud Alcohol.* 2000;61(3):387–395
 149. O'Malley PM, Wagenaar AC. Effects of minimum drinking age laws on alcohol use, related behaviors and traffic crash involvement among American youth: 1976–1987. *J Stud Alcohol.* 1991;52(5):478–491
 150. Shults RA, Elder RW, Sleet DA, et al. Reviews of evidence regarding interventions to reduce alcohol-impaired driving. *Am J Prev Med.* 2001;21(4 suppl):66–88
 151. Voas RB, Tippetts AS, Fell JC. Assessing the effectiveness of minimum legal drinking age and zero tolerance laws in the United States. *Accid Anal Prev.* 2003;35(4):579–587
 152. Elder RW, Nichols JL, Shults RA, Sleet DA, Barrios LC, Compton R. Effectiveness of school-based programs for reducing drinking and driving and riding with drinking drivers: a systematic review. *Am J Prev Med.* 2005;28(5 suppl):288–304
 153. Jones RK, Lacey JH. *Alcohol and Highway Safety 2001: A Review of the State of Knowledge.* Washington, DC: National Highway Traffic Safety Administration; 2001. Report DOT HS-809–383
 154. Preusser DF, Ulmer RB, Preusser CW. *Obstacles to Enforcement of Youthful (Under 21) Impaired Driving.* Washington, DC: National Highway Traffic Safety Administration; 1992. Report DOT HS-807-878
 155. Wells JK, Greene MA, Foss RD, Ferguson SA, Williams AF. Drinking drivers missed at sobriety checkpoints. *J Stud Alcohol.* 1997;58(5):513–517
 156. Ferguson SA, Williams AF. Awareness of zero tolerance laws in three states. *J Safety Res.* 2002;33(3):293–299
 157. Blomberg RD. *Lower Legal BAC Limits for Youth: Evaluation of the Maryland 0.02 Law.* Washington, DC: US Department of Transportation; 1992. Report DOT HS-806-807

158. Elder RW, Shults RA, Sleet DA, Nichols JL, Thompson RS, Rajab W. Effectiveness of mass media campaigns for reducing drinking and driving and alcohol-involved crashes: a systematic review. *Am J Prev Med.* 2004;27(1):57–65
159. Wagenaar AC, Murray DM, Gehan JP, et al. Communities mobilizing for change on alcohol: outcomes from a randomized community trial. *J Stud Alcohol.* 2000;61(1):85–94
160. Wagenaar AC, Toomey TL. Effects of minimum drinking age laws: review and analyses of the literature from 1960 to 2000. *J Stud Alcohol Suppl.* 2002;(14):206–225
161. Kenkel DS. Drinking, driving, and deterrence: the effectiveness and social costs of alternative policies. *J Law Econ.* 1993;36(2):877–913
162. Howat P, Sleet D, Elder R, Maycock B. Preventing alcohol-related traffic injury: a health promotion approach. *Traffic Inj Prev.* 2004;5(3):208–219
163. Wagenaar AC, Murray DM, Toomey TL. Communities Mobilizing for Change on Alcohol (CMCA): effects of a randomized trial on arrests and traffic crashes. *Addiction.* 2000;95(2):209–217
164. Holder HD, Gruenewald PJ, Ponicki WR, et al. Effect of community-based interventions on high-risk drinking and alcohol-related injuries. *JAMA.* 2000;284(18):2341–2347
165. Hingson R, McGovern T, Howland J, Heeren T, Winter M, Zakocs R. Reducing alcohol-impaired driving in Massachusetts: the Saving Lives Program. *Am J Public Health.* 1996;86(6):791–797
166. Weitzman ER, Nelson TF, Lee H, Wechsler H. Reducing drinking and related harms in college: evaluation of the “A Matter of Degree” program. *Am J Prev Med.* 2004;27(3):187–196
167. Clapp JD, Johnson M, Voas RB, Lange JE, Shillington A, Russell C. Reducing DUI among US college students: results of an environmental prevention trial. *Addiction.* 2005;100(3):327–334
168. Hingson R, Heeren T, Winter M, Wechsler H. Magnitude of alcohol-related mortality and morbidity among US college students ages 18–24: changes from 1998 to 2001. *Annu Rev Public Health.* 2005;26:259–279
169. Klingberg T, Fernell E, Olesen PJ, et al. Computerized training of working memory in children with ADHD: a randomized, controlled trial. *J Am Acad Child Adolesc Psychiatry.* 2005;44(2):177–186

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