



Binge Drinking

Lorena Siqueira, MD, MSPH, FAAP, Vincent C. Smith, MD, MPH, FAAP, COMMITTEE ON SUBSTANCE ABUSE

abstract

Alcohol is the substance most frequently abused by children and adolescents in the United States, and its use is associated with the leading causes of death and serious injury at this age (ie, motor vehicle accidents, homicides, and suicides). Among youth who drink, the proportion who drink heavily is higher than among adult drinkers, increasing from approximately 50% in those 12 to 14 years of age to 72% among those 18 to 20 years of age. In this clinical report, the definition, epidemiology, and risk factors for binge drinking; the neurobiology of intoxication, blackouts, and hangovers; genetic considerations; and adverse outcomes are discussed. The report offers guidance for the pediatrician. As with any high-risk behavior, prevention plays a more important role than later intervention and has been shown to be more effective. In the pediatric office setting, it is important to ask every adolescent about alcohol use.

Adolescence is a time of exploration and limit testing; therefore, it is no coincidence that this is the chief period for initiating substance use. Alcohol is the substance most frequently used by children and adolescents in the United States, and its use in youth is associated with the leading causes of death and serious injury at this age (ie, motor vehicle accidents, homicides, and suicides).¹ Drinking levels that may cause little or no problem for adults may be dangerous for adolescents.² Recent studies indicate that alcohol use during this period of growth may interrupt key processes of brain development, possibly leading to cognitive impairment and an elevated risk of developing a chronic alcohol use disorder.³

Twenty-one percent of youth acknowledge having had more than a sip of alcohol before 13 years of age, and most (79%) have done so by 12th grade.⁴ Among youth who drink, the proportion who drink heavily is higher than among adult drinkers, rising from approximately 50% in those 12 to 14 years of age to 72% among those 18 to 20 years of age.⁵ The original definition for binge drinking (5 drinks in 2 hours) was based on size and body composition for men. A standard drink has been defined by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) as one that contains 14 g of pure alcohol (about 0.6 fluid oz or 1.2 tablespoons), as is found in one 12-oz beer, one 5-oz glass of wine, or one 1.5-oz shot of distilled spirits.⁶ The NIAAA has defined binge drinking as the pattern of drinking that brings a person's blood alcohol concentration (BAC) to 0.08% or greater, which derives from concentrations for drunk driving for

FREE

This document is copyrighted and is property of the American Academy of Pediatrics and its Board of Directors. All authors have filed conflict of interest statements with the American Academy of Pediatrics. Any conflicts have been resolved through a process approved by the Board of Directors. The American Academy of Pediatrics has neither solicited nor accepted any commercial involvement in the development of the content of this publication.

Clinical reports from the American Academy of Pediatrics benefit from expertise and resources of liaisons and internal (AAP) and external reviewers. However, clinical reports from the American Academy of Pediatrics may not reflect the views of the liaisons or the organizations or government agencies that they represent.

The guidance in this report does not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

All clinical reports from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

www.pediatrics.org/cgi/doi/10.1542/peds.2015-2337

DOI: 10.1542/peds.2015-2337

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

Copyright © 2015 by the American Academy of Pediatrics

those 21 years or older (the legal intoxication level for adults in all 50 states in the United States). A quantitative definition of drinking makes results comparable across studies and is also important from a clinical point of view, because binge drinkers are more likely to experience alcohol-related problems than drinkers who do not binge. In adults, binge drinking refers to the consumption of 5 or more alcoholic drinks in a row by men and 4 or more drinks in a row by women, over a 2-hour period.⁷ On average, women weigh less than men and have lower body water percentages; therefore, they reach higher BACs than men with similar quantities of alcohol consumed.⁸ The time period used in surveys to record past episodes varies from one or more times in the last 2 weeks to one or more times in the last 30 days.

Binge drinkers have been characterized in studies by the frequency of the binge episodes and by the quantity consumed above the 4- to 5-drink limit to better correlate the drinking pattern with outcomes. Frequent binge drinkers are those who engage in this behavior 3 or more times in the period studied (usually the previous 2 weeks), and occasional binge drinkers are those who drink this way less than 3 times in the previous 2 weeks. Frequent binge drinkers are significantly more likely to endorse reduced health quality of life and mental distress.⁹ They are also more likely to develop alcohol and substance use disorders.¹⁰ Studies have also defined extreme binge drinkers as those who consume dangerously high levels of alcohol on one occasion, with thresholds of ≥ 10 drinks and ≥ 15 drinks.² Studies in youth may underestimate the resulting harm, because the definitions used for binge drinking are not consistent. Because youth typically weigh less than adults, they are likely to reach a BAC much higher than 0.08% with 5 drinks in a 2-hour period.¹¹ Lower cutoff

points to define binge drinking by age and gender in youth have been suggested: for girls 9 to 17 years of age, the cutoff suggested is ≥ 3 drinks. For boys 9 to 13 years of age, the cutoff suggested is ≥ 3 drinks; for boys 14 to 15 years of age, the cutoff suggested is ≥ 4 drinks; and for boys 16 to 17 years of age, the cutoff suggested is ≥ 5 drinks. These cutoffs in boys reflect their greater muscle mass and total body water content. However, fewer drinks than suggested in these cutoffs should not be considered safe. Again, using adult definitions of binge drinking will result in younger children who are at substantial risk of poor outcomes being missed.

EPIDEMIOLOGY OF BINGE DRINKING

Binge drinking is a common problem. In a 2013 report, 22.9% (60.1 million) of Americans 12 years or older surveyed reported binge drinking in the 30 days before the survey.¹² Among those 12 to 20 years of age, 14.2% (5.4 million) reported binge drinking. Additional breakdown by age revealed that 0.8% of 12- to 13-year-olds reported binge drinking and 4.5% of 14- to 15-year-olds reported binge drinking.

High school is a time when drinking rates increase drastically. Between 36% and 50% of high school students currently drink alcohol, and 28% to 60% of them report binge drinking.^{13,14} Nearly two-thirds of these students reported binge drinking on more than one occasion in the past 30 days. One in 10 high school seniors report drinking 10 or more drinks in a row, and 5.6% of high school seniors report consuming 15 or more drinks in a row.²

The rates of alcohol consumption and binge drinking in girls and young women have been increasing but are still lower than among boys and young men. In 2013, male and female survey respondents 12 to 20 years of age had similar rates of drinking in the past month (23% and 22.5%,

respectively), but male respondents were more likely to report binge drinking (15.8% and 12.4%, respectively).¹²

Among high school students, boys (24.7%) were more likely than girls (15%) to participate in all levels of binge drinking.²

The trends in binge drinking among youth also vary by race or ethnicity and geography. Binge drinking was far more common among white high school students (23.8%) than among black high school students (7.6%).² Hispanic (22.4%) and non-Hispanic white (21.7%) high school girls had a higher prevalence of binge drinking than did non-Hispanic black girls (10.3%).¹⁵ Young men, students from more rural areas, and youth of white race or ethnicity had particularly high rates of extreme binge drinking. Rates of binge drinking vary within and between states in the United States.¹⁶ The regions with the highest rates of underage binge drinking were located in Massachusetts, New Hampshire, Rhode Island, Vermont, District of Columbia, Florida, West Virginia, Indiana, North Dakota, Ohio, and Wyoming.¹⁶ Teens from the Midwest were more likely than those in other geographic regions to report extreme binge drinking.¹⁷ Socioeconomic status was less consistently predictive of binge drinking behavior. Youth of higher socioeconomic status are at greater risk of binge drinking but lower risk of extreme binge drinking; however, youth of lower socioeconomic status and those from rural areas may be at higher risk of very extreme binge drinking (ie, ≥ 15 drinks on one occasion).²

Lesbian, gay, bisexual, and transgender (LGBT) youth are at elevated risk of alcohol misuse, with drinking patterns that significantly increase linearly over time, more rapidly among male compared with female LGBT youth.¹⁸ However, the overall pattern of results and correlates of alcohol use are strikingly similar to those of non-LGBT youth.¹⁸

Rates of drinking were significantly lower in African American LGBT youth than in those of all other racial groups. Furthermore, alcohol use in these youth has been associated with their experience of victimization, threats, or violence¹⁹ and by the chronic stress associated with societal stigmatization.²⁰

Underage drinkers typically obtain alcohol from adults of legal drinking age, including at home from parents, siblings, or other relatives, and they drink most frequently in their own or others' homes.²¹ A recent study showed that 13- to 20-year-olds drank hard liquor, especially vodka, in almost 44% of their recent binge-drinking sessions; beer was involved in less than a third of all the reported binge-drinking episodes.²²

Binge drinking is a costly problem. Excessive alcohol consumption in 2006 cost states a median of \$2.9 billion (\$703 per person or approximately \$1.91 per drink), with more than 70% of the cost attributable to binge drinking.²³ Although the majority of these costs were related to adult drinking, underage drinking alone was responsible for a median of \$361.4 million in economic costs.²³ These costs include health care expenditures, loss of worker productivity, criminal justice system expenses, property damage from motor vehicle crashes and fires, and special education needed for those with fetal alcohol spectrum disorders.²³ Because the costs described relate only to the directly attributable costs, they are likely to underestimate the actual economic impact of binge drinking.

RISK FACTORS FOR BINGE DRINKING IN THE CONTEXT OF ADOLESCENT DEVELOPMENT

The onset and progression in alcohol use are influenced by the developmental changes in children as they enter adolescence.²⁴ Epidemiologic studies clearly indicate

that adolescence is a critical risk period for initiation of alcohol use, and the earlier onset is associated with greater risk of developing alcohol abuse and dependence.^{25,26} A number of individual risk factors for alcohol misuse have been identified, and some may be relevant to our understanding of the onset and course of binge drinking, in particular. According to Bandura's Social Learning Theory, 2 constructs are central to the initiation and maintenance of human behavior (ie, outcome expectancies and self-efficacy).²⁷ The anticipated effects of alcohol ingestion, referred to as "alcohol expectancies," play a crucial role in decision-making for children and adolescents. These expectancies may be both positive and negative. Positive expectancies are often shaped by alcohol advertising, which is ubiquitous and portrays its use as being a normal part of adolescent and adult social life. As a result, the negative expectancies usually present in children are slowly replaced by positive expectancies as they enter adolescence.²⁸

Greater positive expectancies have been associated with binge drinking and have been noted more frequently in boys and men.²⁹ Among girls and women, those with a history of childhood maltreatment endorse the reduction of ongoing tension as a positive expectancy, and this is associated with later heavy drinking. Bandura's second construct would refer to drinking refusal and self-efficacy; that is, youth with low resistance skills would be more susceptible to peer pressure to binge drink.^{27,30}

Similar to outcome expectancies, another risk factor described is one's "drinking motives." Drinking to enhance positive emotional states (enhancement motives) has been related to heavy drinking and in turn is linked to certain personality characteristics, such as sensation seeking,³¹ low inhibitory control,³²

and impulsivity.³³ Sensation seeking is a personality characteristic that indicates the need for novel experiences and the willingness to take physical and psychological risks for these experiences.³³ The connection of sensation seeking to earlier onset of substance use among teenagers and young adults and to higher levels of substance use has been established.³⁴

Lastly, peer use is another important risk factor. The social experience of drinking in a group has been correlated with youth binge drinking.³⁵ As opposed to drinking alone, group involvement leads to greater perceived euphoria and also greater consumption. As a cautionary note, adolescents who drink alone are in a very high-risk group and need referral for treatment. Studies indicate that students often seek out others to drink with, and when they enter college this tendency may influence their choice of living arrangements. Living in dormitories correlates with higher drinking levels than living off campus.³⁶

The biological basis for binge drinking is also being researched. Developmental stages during adolescence influence the effects of alcohol and the risk of heavy drinking. The pharmacokinetics of alcohol (the way alcohol is absorbed, distributed, and eliminated) vary by gender and by age, with higher BACs in younger subjects as compared with adults with similar levels of consumption, as noted previously. Hormonal changes during puberty may also affect sensitivity to alcohol, making adolescents less sensitive to the effects of intoxication (ie, sedation and loss of coordination). Lack of sensitivity to alcohol, in turn, may be related to the developmental immaturity of the neurotransmitter receptor system in youth.

The rising limb theory postulates that in the early phase of drinking, binge drinkers are more sensitive to the subjective positive euphoric effects of

alcohol than light drinkers but are less sensitive to the sedative effects in both the early rise and the fall in alcohol concentrations. Drinkers who do not binge drink, on the other hand, are more likely to have greater sedation in the early phase of drinking.³⁷ Thus, young people, who are more sensitive to the subjective euphoric effects in the early phases of drinking, are more likely to become binge drinkers.

The question now is whether there is a common factor that influences both this biological response to alcohol and the associated personality and neurocognitive variables. Research on brain development in adolescence has determined that areas of the brain that control cognition, impulsivity, and sensitivity to rewards mature at different rates in different adolescents, and a delay in this development may contribute to externalizing behaviors, such as the initiation of substance use.

NEUROBIOLOGY OF INTOXICATION, BLACKOUT, AND HANGOVER

Intoxication is the feeling of drunkenness or inebriation associated with alcohol consumption. As alcohol enters the bloodstream, the liver must metabolize it for excretion, which occurs at a fixed rate. Consequently, intoxication, which usually occurs at a BAC of 50 to 150 mg/dL, is the consequence of alcohol entering the bloodstream faster than it can be metabolized by the liver. Because alcohol readily crosses the blood–brain barrier, the concentration of alcohol in the brain parallels the concentration in the blood.³⁸ Symptoms of intoxication include initial euphoria followed by incoordination, imbalance, ataxia, sleepiness, loss of social inhibitions, loquacity, depression, and hostility.³⁸ When the BAC exceeds 150 mg/dL, more depressant symptoms, including lethargy, bradycardia, hypotension, and respiratory depression, may occur.³⁸ When

a person is in an alcohol-induced torpor, vomiting and pulmonary aspiration may occur.³⁸ Alcohol poisoning may occur when the BAC increases beyond the depressant level, usually around a median of 450 mg/dL.³⁸ Manifestations include stupor, coma, and death by respiratory depression with respiratory acidosis and hypotension.³⁸

Binge drinking may result in a “blackout,” or losing memory of events that occurred during a drinking episode.³⁸ The loss of memory is usually temporary but may persist hours to days after the drinking episode and is probably the result of acute dysfunction of the hippocampus.³⁸ Alcohol specifically interferes with the brain’s ability to make long-term memories from short-term memories and experiences.³⁹ Blackouts are based on the amount of alcohol consumed and are more common in adolescents than adults.³⁸ On average, among adolescents and college students males consume 9 drinks and females consume 5 drinks before a blackout.³⁸ During a blackout, drinkers are disinhibited and may engage in high-risk behaviors or suffer an adverse outcome, including having unprotected sexual intercourse, which may lead to unplanned pregnancy.⁴⁰

A “hangover” is a constellation of symptoms that usually occur within 6 to 24 hours after a heavy or prolonged drinking episode.^{38,41} The symptoms include headache, drowsiness, dizziness, inability to concentrate, dry mouth, gastrointestinal complaints including nausea and vomiting, sweating, hyperexcitability, muscle weakness or pain, tremors, tachycardia, hyperventilation, depression, irritability, and anxiety.^{38,41} Thought processes and learning are also impaired.³⁸ Adolescence is a period of ongoing brain maturation that is characterized by specific and

ordered changes in brain structure that hone the functioning of neural circuits in the service of achieving optimal cognitive, emotional, and higher-order executive functioning.⁴² Thus, the potential for alcohol to disrupt critical processes underlying adolescent brain maturation is concerning, given accumulating supporting scientific evidence to that effect. Studies indicate that cumulative alcohol use together with the severity of hangover and withdrawal symptoms predicts cognitive functioning.⁴³

BINGE DRINKING AND ALCOHOLISM

Current research estimates that approximately half the risk for alcoholism comes from genetic factors, and the other half comes from environmental risk factors. Environmental influences probably have more of a role in initiation of alcohol use, whereas genetics have more of an influence on development of an addiction. An ongoing study to determine candidate genes, called the Collaborative Study on the Genetics of Alcoholism and supported by the NIAAA, recognizes that no single gene explains all facets of this condition.⁴⁴ Rather, variations in many different genes interacting with the environment place some people at significantly higher risk for the disease. DNA regions with susceptibility genes have been found on chromosomes 1 and 7, and DNA regions with protective genes have been found on chromosome 4. Of great interest, the region on chromosome 4 is in the general vicinity of the alcohol dehydrogenase (ADH) genes. The primary enzymes involved in the metabolism of alcohol in the body are ADH and aldehyde dehydrogenase (ALDH). Both of these enzymes occur in several forms encoded by different genes and influence both the level of consumption and the risk of alcoholism.⁴⁵ Most of the numerous

variants of these genes involve single nucleotide polymorphisms. ADH molecules do their work primarily in the stomach and the liver and convert alcohol to acetaldehyde.

Mitochondrial ALDH oxidizes acetaldehyde to acetate. High-activity ADH variants increase the rate of acetaldehyde generation, and a low-activity ALDH2 variant is associated with an inability to metabolize this compound. The resultant pathophysiologic effect of this altered alcohol metabolism may be mediated by the accumulation of acetaldehyde. Many East Asians and American Indians experience a “flush syndrome” after alcohol consumption, with facial flushing, tachycardia, headaches, nausea, and vomiting that may deter additional use of alcohol. This is similar to the effect of disulfiram (a medication used to treat alcoholism). Similar variants have also been found in the Caucasian population and may influence the maximum number of drinks a person can handle.⁴⁶ Low tolerance may be protective, whereas high tolerance may make someone more vulnerable to alcoholism.

A search is under way to determine whether there are genes that specifically influence binge drinking. Investigators have evaluated the role of γ -aminobutyric acid receptors in the brain in rat models and found a role for them in excessive drinking. Another candidate gene is the serotonin transporter gene (5-HTT). Studies indicate that students who are homozygous for its short version are at risk for anxiety and depression and that they also report higher levels of alcohol intake, suggesting that they may use alcohol for tension reduction.⁴⁷ Those who are heterozygous consume a smaller number of drinks at each episode. Other authors have noted that students with this gene drink expressly for the purpose of becoming drunk.⁴⁸ These initial studies hold hope that a common factor that underlies the various risk

factors that influence binge drinking and the subsequent development of alcohol dependence will be found.

ADVERSE OUTCOMES ASSOCIATED WITH DRINKING

Drinking alcohol is associated with numerous adverse outcomes in underage drinkers, and binge drinking increases these risks. Adolescents who binge drink are more likely to exhibit poor judgment and engage in drunk driving, riding in the car with an impaired driver, riding a bicycle without a helmet, and or other risky behavior.⁴⁰ Alcohol use is involved in each of the major causes of mortality in adolescents (ie, accidents, suicides, and homicides). Drinking alcohol impairs driving ability,² and among 15- to 20-year-olds, nearly a third of all fatal automobile crashes involve alcohol.⁴⁰ Binge drinking increases the risk of suicide and attempted suicide.^{40,49} Drinking alcohol is also associated with an elevated risk for nonautomobile accidents with other potentially fatal outcomes, including severe injuries and drowning.² In the United States, 50% of all head injuries in adolescents are associated with alcohol consumption.⁴⁰ Underage drinkers are susceptible to immediate consequences of alcohol use, including blackouts, hangovers, and acute alcohol poisoning.³⁸ Binge drinking can also lead to fatal alcohol poisoning.² Many of these alcohol-associated adverse outcomes could lead to hospitalization or, in severe cases, death. Adolescent binge drinking may also lead to liver disease, hypertension, heart disease, stroke, and breast and other cancers in the future. Adolescents who start drinking before 15 years of age are at 4 times the risk of developing alcohol dependence as those who start drinking after 20 years of age.^{38,40} The developing adolescent brain is more vulnerable to alcohol-induced brain damage and cognitive impairment than the adult brain.⁵⁰

The brain changes make the adolescent more susceptible to both addiction and increased substance use severity. Binge drinkers are also more likely to use other substances at a younger age.⁵¹ Binge drinking may affect school performance by impairing study habits and may erode the development of transitional skills to adulthood.³⁸ In fact, adolescents who reported binge drinking tend to have lower grade point averages.⁵¹

Binge drinking has been associated with earlier sexual activity and more frequent changing of sexual partners.⁴⁰ It has also been associated with higher rates of unwanted and teenage pregnancy, sexually transmitted infections, and infertility.⁴⁰ Adolescents are at higher risk of becoming the victims of unwanted sexual activity, actual or attempted forced sexual activity, and sexual victimization when binge drinking.^{40,52}

Pregnant adolescents who binge drink put their growing fetuses at risk, because prenatal alcohol exposure causes a broad range of adverse developmental effects collectively referred to as fetal alcohol spectrum disorder.⁵³ Binge drinking exposes infants to high BACs over a short period of time, and in animal studies it was more harmful to the fetus than more continuous drinking patterns, even if the overall amount of alcohol consumed was less.⁵⁴ Longitudinal human studies have shown severe cognitive and behavioral deficits in children of mothers who were binge drinkers during pregnancy.⁵⁴

There is evidence of brain volume reduction in adolescents who are heavy drinkers. A longitudinal study demonstrated that 3 areas in the brain's frontal region and cerebellar white matter had preexisting volume differences in heavy drinkers and nondrinkers, and over the next 3 years, those who drank showed greater volume reductions in subcortical and temporal regions than

did the nondrinkers undergoing normal adolescent developmentally related neural pruning.⁵⁵ The frontal regions found to be affected in this study are regions involved in executive control, including inhibitory functioning, attention, impulsivity, and self-regulation.^{56,57} Poorer inhibitory functioning in those who have not used substances has been found to predict future substance use and could therefore explain the transition to heavy drinking in the study subjects.⁵⁵ Changes noted after drinking were in brain regions involved in language and visuospatial abilities and may account for these deficits noted in adolescent drinkers.

Adverse outcomes associated with underage drinking include legal considerations for youth and their parents. Because the legal drinking age in every state is 21 years, minors who are caught consuming or under the influence of alcohol could suffer fines, forced community service, suspension of driving privileges, mandatory alcohol awareness classes, or jail time, depending on the circumstances.⁵⁸ Social hosting liability laws and dram shop laws vary widely by state, but parents of minors who supply alcohol or allow underage drinking in their home are subject to civil and criminal penalties.²¹ Such liability may include criminal or civil charges, depending on the state, and may include costs of police, fire, or other emergency services.

GUIDANCE FOR PEDIATRICIANS

Universal Preventive Interventions

As with any high-risk behavior, prevention plays a more important role than later intervention. Preventive measures are important, because underage heavy drinking is difficult to control and cannot be successfully addressed by intervening with youth alone. In a society in which alcohol use is normative, a collective strategy is believed to be

more important.⁵⁹ A multipronged approach has been suggested that includes a national adult-oriented media campaign, targeting alcohol advertising and the entertainment media, increasing community interventions, limiting access to alcohol, and increasing alcohol excise taxes. School-based health education programs should teach life skills in addition to knowledge of the dangers of drug use and should include training that promotes healthy alternatives to risky behavior through activities designed to teach students the necessary skills to resist social (peer) pressure to smoke, drink, and use drugs. These life skills programs also help students develop greater self-esteem and self-confidence.⁶⁰ Pediatricians should encourage their local schools to consider adopting this approach.

Anticipatory Guidance

In the office setting, there are programs designed to help pediatricians deliver messages to parents. Surveys indicate that children start to think positively about alcohol between ages 9 and 13 years. The more young people are exposed to alcohol advertising and marketing, the more likely they are to drink, and if they are already drinking, this exposure leads them to drink more. Therefore, it is very important to start talking to children about the dangers of drinking as early as 9 years of age. One such program developed by the Substance Abuse and Mental Health Services Administration is *Talk. They Hear You* (www.underagedrinking.org). Resources available through the Web site include background information, talking points, and other material to distribute in an office setting. All of this material can be customized to include the pediatrician's office address and contact information. Web-based role playing simulations that can be played on a TV monitor in waiting areas are also included. Physicians can also inform parents

that 80% of teenagers say their parents are the biggest influence on their decision whether to drink.⁶¹ In a 2013 study, parental communication on alcohol use before college entry was more likely to prevent nondrinking students from transitioning to heavy drinking status and was also 20 times more likely to reduce heavy drinking patterns among those who had started this drinking pattern before college entry than among students who did not receive parental advice.⁶²

Screening and Brief Intervention

During office visits, because alcohol use is so common, it is important for pediatricians to screen every adolescent for alcohol use. Just using one's clinical impression can underestimate substance use, and therefore structured screening instruments are recommended.⁶³ When time does not permit, alcohol-only screening tools may be a reasonable approach.²⁴ A quick screening tool developed by the NIAAA in collaboration with the American Academy of Pediatrics can quickly identify youth at risk for alcohol-related problems.⁶⁴ This screening tool consists of 2 questions that vary slightly for elementary, middle, and high school patients. For those in elementary school (9–11 years of age), ask the “friends (any drinking)” question first: “Do you have any friends who drank beer, wine, or any drink containing alcohol in the **past year**?” Ask the “patient (any drinking)” question next: “How about you—have you **ever** had more than a few sips of beer, wine, or any drink containing alcohol?” For those in middle school (11–14 years of age), after the same “friends (any drinking)” question, the patient question is, “How about you—in the **past year, on how many days** have you had more than a few sips of beer, wine, or any drink containing alcohol?” For those in high school (14–18 years of age), ask the “patient (any drinking)” question first, followed by

the “friends (how much?)” question: “If your friends drink, **how many drinks** do they usually drink on an occasion?” This very brief screen can detect risk early, is empirically based,^{65,66} and is a good predictor of current and future negative consequences of alcohol use. The screen result categorizes adolescents as low, moderate, or high risk on the basis of their responses to the questions, and the guide outlines different levels of intervention depending on the level of risk. Lower-risk subjects are given brief advice. Moderate-risk subjects are given brief advice and motivational interviewing, and the highest-risk category requires both motivational interviewing and possible referral. Motivational interviewing is a brief patient-centered communication style designed to help the subject consider the benefits of change. The practitioner helps highlight the discrepancy between the subject’s current status and where he or she wants to be and thus motivates him or her to change.⁶⁷ Motivational interviewing delivered over ≥ 1 sessions and based in health care or educational settings is effective at reducing levels of consumption and alcohol-related harm.⁶⁸ More research is needed on the benefit of these brief behavioral counseling methods for long-term morbidity and mortality.⁶⁹

The approach to substance use screening, brief intervention, and referral to treatment for pediatricians has also been outlined in detail in a policy statement from the American Academy of Pediatrics.⁷⁰ The issue of college binge drinking is beyond the scope of this document.

Selected Resources for Pediatricians

Bright Futures. Guidelines for Health Supervision of Infants, Children and Adolescents. brightfutures.aap.org

National Institute on Alcohol Abuse and Alcoholism. *Alcohol Screening and Brief Intervention for Youth*. A

Practitioner’s Guide. www.niaaa.nih.gov/YouthGuide

Substance Abuse and Mental Health Service Administration. *Talk. They Hear You*. www.samhsa.gov/underage-drinking

The Guide to Community Preventive Services. *Preventing Excessive Alcohol Consumption*. www.thecommunityguide.org/alcohol

US Preventive Services Task Force. *Alcohol Misuse: Screening and Behavioral Counseling Interventions in Primary Care*. www.uspreventiveservicestaskforce.org/uspstf/uspdsdrin.htm

National Institute on Alcohol Abuse and Alcoholism. *Make a Difference: Talk to Your Child About Alcohol*. <http://pubs.niaaa.nih.gov/publications/children.pdf>

HealthyChildren.org, Official Consumer Web Site of the AAP. www.healthychildren.org/English/ages-stages/teen/substance-abuse

Prevention Research Center, The Pennsylvania State University. *A Parent Handbook for Talking With Adolescents About Alcohol*. www.bucknell.edu/Documents/Communication/WHPS/BU_ParentAlcoholHandbook2014.pdf

Mothers Against Drunk Driving. *Underage Drinking*. www.madd.org/underage-drinking

Substance Abuse and Mental Health Service Administration. *National Registry of Evidence-Based Programs and Practices*. www.nrepp.samhsa.gov/AdvancedSearch.aspx

LEAD AUTHORS

Lorena M. Siqueira, MD, MSPH, FAAP
Vincent C. Smith, MD, MPH, FAAP

COMMITTEE ON SUBSTANCE ABUSE, 2014–2015

Sharon Levy, MD, MPH, FAAP, Chairperson
Seth D. Ammerman, MD, FAAP
Pamela K. Gonzalez, MD, FAAP
Sheryl A. Ryan, MD, FAAP
Lorena M. Siqueira, MD, MSPH, FAAP
Vincent C. Smith, MD, MPH, FAAP

LIAISONS

Vivian B. Faden, PhD – *National Institute of Alcohol Abuse and Alcoholism*

Gregory Tau, MD, PhD – *American Academy of Child and Adolescent Psychiatry*

STAFF

Renee Jarrett, MPH

REFERENCES

1. Johnston LD, O’Malley PM, Bachman JG, Schulenberg JE. *Monitoring the Future: National Survey Results on Drug Use, 1975–2012*, Vol. I: *Secondary School Students*. Ann Arbor, MI: Institute for Social Research, University of Michigan; 2012
2. Patrick ME, Schulenberg JE, Martz ME, Maggs JL, O’Malley PM, Johnston LD. Extreme binge drinking among 12th-grade students in the United States: prevalence and predictors. *JAMA Pediatr*. 2013;167(11):1019–1025
3. Guerri C, Pascual M. Mechanisms involved in the neurotoxic, cognitive, and neurobehavioral effects of alcohol consumption during adolescence. *Alcohol*. 2010;44(1):15–26
4. Centers for Disease Control and Prevention (CDC). Vital signs: binge drinking prevalence, frequency, and intensity among adults—United States, 2010. *MMWR Morb Mortal Wkly Rep*. 2012;61(1):14–19
5. Office of Juvenile Justice and Delinquency Prevention. *Drinking in America: Myths, Realities, and Prevention Policy*. Washington, DC: US Department of Justice, Office of Justice Programs, Office of Juvenile Justice and Delinquency Prevention; 2005
6. National Institute on Alcohol Abuse and Alcoholism. What is a standard drink? Available at: www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/what-standard-drink. Accessed June 25, 2015
7. Wechsler H, Davenport A, Dowdall G, Moeykens B, Castillo S. Health and behavioral consequences of binge drinking in college. A national survey of students at 140 campuses. *JAMA*. 1994; 272(21):1672–1677
8. Mello NK. Drug use patterns and premenstrual dysphoria. In: Ray BA, Braude MC, eds. *Women and Drugs: A New Era for Research*. NIDA Research

- Monograph 65. Rockville, MD: National Institute on Drug Abuse; 1986
9. Okoro CA, Brewer RD, Naimi TS, Moriarty DG, Giles WH, Mokdad AH. Binge drinking and health-related quality of life: do popular perceptions match reality? *Am J Prev Med.* 2004;26(3):230–233
 10. Chassin L, Pitts SC, Probst 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
 11. Donovan JE. Estimated blood alcohol concentrations for child and adolescent drinking and their implications for screening instruments. *Pediatrics.* 2009; 123(6). Available at: www.pediatrics.org/cgi/content/full/123/6/e975
 12. Substance Abuse and Mental Health Services Administration. *Results From the 2013 National Survey on Drug Use and Health: Summary of National Findings.* NSDUH Series H-48, HHS Publication SMA 14-4863. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2014
 13. 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
 14. Hingson RW, Zha W, Iannotti RJ, Simons-Morton B. Physician advice to adolescents about drinking and other health behaviors. *Pediatrics.* 2013;131(2): 249–257
 15. Centers for Disease Control and Prevention (CDC). Vital signs: binge drinking among women and high school girls—United States, 2011. *MMWR Morb Mortal Wkly Rep.* 2013;62(1):9–13
 16. Substance Abuse and Mental Health Services Administration, Center for Behavioral Health Statistics and Quality. *The NSDUH Report: Underage Binge Alcohol Use Varies Within and Across States.* Rockville, MD: Substance Abuse and Mental Health Services Administration; August 7, 2014
 17. Johnston LD, O'Malley PM, Miech RA, Bachman JG, Schulenberg JE. *Monitoring the Future National Survey Results on Drug Use, 1975–2013. 2013 Overview: Key Findings on Adolescent Drug Use.* Ann Arbor, MI: Institute for Social Research, University of Michigan; 2014
 18. Newcomb ME, Heinz AJ, Mustanski B. Examining risk and protective factors for alcohol use in lesbian, gay, bisexual, and transgender youth: a longitudinal multilevel analysis. *J Stud Alcohol Drugs.* 2012;73(5):783–793
 19. Goldbach JT, Tanner-Smith EE, Bagwell M, Dunlap S. Minority stress and substance use in sexual minority adolescents: a meta-analysis. *Prev Sci.* 2014;15(3): 350–363
 20. Schragger SM, Goldbach JT, Holloway IW. Platform session: male health & substance use. Presentation on the application of minority stress theory to binge drinking among lesbian and gay adolescents (E-PAS2014:1675.5). Pediatric Academic Societies' Meeting; May 3, 2014; Vancouver, BC
 21. Dills AK. Social host liability for minors and underage drunk-driving accidents. *J Health Econ.* 2010;29(2):241–249
 22. Naimi TS, Siegel M, DeJong W, O'Doherty C, Jernigan D. Beverage- and brand-specific binge alcohol consumption among underage youth in the US [published online ahead of print May 30, 2014]. *J Substance Use.* doi:10.3109/14659891.2014.920054
 23. Sacks JJ, Roeber J, Bouchery EE, Gonzales K, Chaloupka FJ, Brewer RD. State costs of excessive alcohol consumption, 2006. *Am J Prev Med.* 2013; 45(4):474–485
 24. Masten AS, Faden VB, Zucker RA, Spear LP. Underage drinking: a developmental framework. *Pediatrics.* 2008;121(suppl 4):S235–S251
 25. Kroutil L, Colliver J, Gfroerer J. *OAS Data Review: Age and Cohort Patterns of Substance Use Among Adolescents.* Rockville, MD: Substance Abuse and Mental Health Services Administration, Office of Applied Studies; 2010:1–9
 26. Dawson DA, Goldstein RB, Chou SP, Ruan WJ, Grant BF. Age at first drink and the first incidence of adult-onset DSM-IV alcohol use disorders. *Alcohol Clin Exp Res.* 2008;32(12):2149–2160
 27. Bandura A. Self-efficacy: toward a unifying theory of behavioral change. *Psychol Rev.* 1977;84(2):191–215
 28. Zucker RA, Donovan JE, Masten AS, Mattson ME, Moss HB. Early developmental processes and the continuity of risk for underage drinking and problem drinking. *Pediatrics.* 2008; 121(suppl 4):S252–S272
 29. Blume AW, Schmalting KB, Marlatt AG. Predictors of change in binge drinking over a 3-month period. *Addict Behav.* 2003;28(5):1007–1012
 30. Connor JP, George SM, Gullo MJ, Kelly AB, Young RM. A prospective study of alcohol expectancies and self-efficacy as predictors of young adolescent alcohol misuse. *Alcohol.* 2011;46(2):161–169
 31. Comeau N, Stewart SH, Loba P. The relations of trait anxiety, anxiety sensitivity, and sensation seeking to adolescents' motivations for alcohol, cigarette, and marijuana use. *Addict Behav.* 2001;26(6):803–825
 32. Leeman RF, Patock-Peckham JA, Potenza MN. Impaired control over alcohol use: An under-addressed risk factor for problem drinking in young adults? *Exp Clin Psychopharmacol.* 2012;20(2): 92–106
 33. Shin SH, Hong HG, Jeon SM. Personality and alcohol use: the role of impulsivity. *Addict Behav.* 2012;37(1):102–107
 34. Zuckerman M. Sensation seeking and the endogenous deficit theory of drug abuse. *NIDA Res Monogr.* 1986;74:59–70
 35. Clapp JD, Shillington AM. Environmental predictors of heavy episodic drinking. *Am J Drug Alcohol Abuse.* 2001;27(2): 301–313
 36. Wechsler H, Nelson TF. What we have learned from the Harvard School of Public Health College Alcohol Study: focusing attention on college student alcohol consumption and the environmental conditions that promote it. *J Stud Alcohol Drugs.* 2008;69(4): 481–490
 37. Holdstock L, King AC, de Wit H. Subjective and objective responses to ethanol in moderate/heavy and light social drinkers. *Alcohol Clin Exp Res.* 2000; 24(6):789–794
 38. Zeigler DW, Wang CC, Yoast RA, et al; Council on Scientific Affairs, American Medical Association. The neurocognitive effects of alcohol on adolescents and college students. *Prev Med.* 2005;40(1): 23–32
 39. Acheson SK, Stein RM, Swartzwelder HS. Impairment of semantic and figural memory by acute ethanol: age-

- dependent effects. *Alcohol Clin Exp Res*. 1998;22(7):1437–1442
40. Stolle M, Sack PM, Thomasius R. Binge drinking in childhood and adolescence: epidemiology, consequences, and interventions. *Dtsch Arztebl Int*. 2009; 106(19):323–328
 41. Stephens R, Ling J, Heffernan TM, Heather N, Jones K. A review of the literature on the cognitive effects of alcohol hangover. *Alcohol*. 2008;43(2):163–170
 42. Tau GZ, Peterson BS. Normal development of brain circuits. *Neuropsychopharmacology*. 2010;35(1):147–168
 43. Jacobus J, Tapert SF. Neurotoxic effects of alcohol in adolescence. *Annu Rev Clin Psychol*. 2013;9:703–721
 44. Edenberg HJ. The Collaborative Study on the Genetics of Alcoholism: an update. Available at: <http://pubs.niaaa.nih.gov/publications/arh26-3/214-218.htm>. Accessed June 25, 2015
 45. Crabb DW, Matsumoto M, Chang D, You M. Overview of the role of alcohol dehydrogenase and aldehyde dehydrogenase and their variants in the genesis of alcohol-related pathology. *Proc Nutr Soc*. 2004;63(1):49–63
 46. Saccone NL, Kwon JM, Corbett J, et al. A genome screen of maximum number of drinks as an alcoholism phenotype. *Am J Med Genet*. 2000;96(5):632–637
 47. Tartter MA, Ray LA. The serotonin transporter polymorphism (5-HTTLPR) and alcohol problems in heavy drinkers: moderation by depressive symptoms. *Front Psychiatry*. 2011;2:49
 48. Herman AI, Philbeck JW, Vasilopoulos NL, Depetrillo PB. Serotonin transporter promoter polymorphism and differences in alcohol consumption behaviour in a college student population. *Alcohol*. 2003;38(5):446–449
 49. Windle M. Suicidal behaviors and alcohol use among adolescents: a developmental psychopathology perspective. *Alcohol Clin Exp Res*. 2004;28(5 suppl):29S–37S
 50. Spanagel R. Alcoholism: a systems approach from molecular physiology to addictive behavior. *Physiol Rev*. 2009; 89(2):649–705
 51. 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
 52. 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
 53. American Academy of Pediatrics, Committee on Substance Abuse. Fetal alcohol spectrum disorder. *Pediatrics*. 2015; (in press)
 54. Maier SE, West JR. Drinking patterns and alcohol-related birth defects. *Alcohol Res Health*. 2001;25(3):168–174
 55. Squeglia LM, Rinker DA, Bartsch H, et al. Brain volume reductions in adolescent heavy drinkers. *Dev Cogn Neurosci*. 2014; 9:117–125
 56. Fjell AM, Walhovd KB, Brown TT, et al; Pediatric Imaging, Neurocognition, and Genetics Study. Multimodal imaging of the self-regulating developing brain. *Proc Natl Acad Sci USA*. 2012;109(48): 19620–19625
 57. Goldberg E. *The Executive Brain: Frontal Lobes and the Civilized Mind*. New York, NY: Oxford University Press; 2001
 58. Fell JC, Fisher DA, Voas RB, Blackman K, Tippetts AS. The impact of underage drinking laws on alcohol-related fatal crashes of young drivers. *Alcohol Clin Exp Res*. 2009;33(7):1208–1219
 59. Bonnie R, O'Connell ME, eds. Institute of Medicine, Committee on Developing a Strategy to Reduce Underage Drinking. *Reducing Underage Drinking: A Collective Responsibility*. Washington, DC: The National Academies Press; 2004
 60. Botvin GJ, Griffin KW. Life skills training as a primary prevention approach for adolescent drug abuse and other problem behaviors. *Int J Emerg Ment Health*. 2002;4(1):41–47
 61. Jackson C. Perceived legitimacy of parental authority and tobacco and alcohol use during early adolescence. *J Adolesc Health*. 2002;31(5):425–432
 62. Turrisi R, Mallett KA, Cleveland MJ, et al. Evaluation of timing and dosage of a parent-based intervention to minimize college students' alcohol consumption. *J Stud Alcohol Drugs*. 2013;74(1):30–40
 63. Wilson CR, Sherritt L, Gates E, Knight JR. Are clinical impressions of adolescent substance use accurate? *Pediatrics*. 2004;114(5). Available at: www.pediatrics.org/cgi/content/full/114/5/e536
 64. National Institute on Alcohol Abuse and Alcoholism. Alcohol Screening and Brief Intervention for Youth: A Practitioner's Guide. NIH Publication no. 11-7805. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism; 2011. Available at: www.niaaa.nih.gov/YouthGuide. Accessed June 25, 2015
 65. Chung T, Smith GT, Donovan JE, et al. Drinking frequency as a brief screen for adolescent alcohol problems. *Pediatrics*. 2012;129(2):205–212
 66. Brown SA, Donovan JE, McGue MK, et al. Youth Alcohol Screening Workgroup II: determining optimal secondary screening questions. *Alcoholism Clin Exp Res*. 2010;34(suppl S2):267A
 67. Miller WR, Rollnick S. *Motivational Interviewing: Preparing People for Change*. New York, NY: The Guilford Press; 2002
 68. Patton R, Deluca P, Kaner E, Newbury-Birch D, Phillips T, Drummond C. Alcohol screening and brief intervention for adolescents: the how, what and where of reducing alcohol consumption and related harm among young people. *Alcohol*. 2014;49(2):207–212
 69. US Preventive Services Task Force. Screening and behavioral counseling interventions in primary care to reduce alcohol misuse: recommendation statement. *Ann Intern Med*. 2004;140(7): 554–556
 70. Levy SJ, Kokotailo PK; Committee on Substance Abuse. Substance use screening, brief intervention, and referral to treatment for pediatricians. *Pediatrics*. 2011;128(5). Available at: www.pediatrics.org/cgi/content/full/128/5/e1330

Binge Drinking

Lorena Siqueira, Vincent C. Smith and COMMITTEE ON SUBSTANCE ABUSE

Pediatrics 2015;136:e718

DOI: 10.1542/peds.2015-2337 originally published online August 31, 2015;

Updated Information & Services	including high resolution figures, can be found at: http://pediatrics.aappublications.org/content/136/3/e718
References	This article cites 54 articles, 7 of which you can access for free at: http://pediatrics.aappublications.org/content/136/3/e718#BIBL
Subspecialty Collections	This article, along with others on similar topics, appears in the following collection(s): Current Policy http://www.aappublications.org/cgi/collection/current_policy Committee on Substance Use and Prevention http://www.aappublications.org/cgi/collection/committee_on_substance_abuse Adolescent Health/Medicine http://www.aappublications.org/cgi/collection/adolescent_health_medicine_sub
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://www.aappublications.org/site/misc/Permissions.xhtml
Reprints	Information about ordering reprints can be found online: http://www.aappublications.org/site/misc/reprints.xhtml

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™



PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Binge Drinking

Lorena Siqueira, Vincent C. Smith and COMMITTEE ON SUBSTANCE ABUSE

Pediatrics 2015;136:e718

DOI: 10.1542/peds.2015-2337 originally published online August 31, 2015;

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://pediatrics.aappublications.org/content/136/3/e718>

Pediatrics is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. Pediatrics is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2015 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 1073-0397.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

