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The neurocognitive effects of alcohol on adolescents and college students ☆

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Abstract

Background. Adolescents and college students are at high risk for initiating alcohol use and high-risk (or binge) drinking. There is a growing body of literature on neurotoxic and harmful cognitive effects of drinking by young people. On average, youths take their first drink at age 12 years.

Methods. MEDLINE search on neurologic and cognitive effects of underage drinking.

Results. Problematic alcohol consumption is not a benign condition that resolves with age. Individuals who first use alcohol before age 14 years are at increased risk of developing alcohol use disorders. Underage drinkers are susceptible to immediate consequences of alcohol use, including blackouts, hangovers, and alcohol poisoning and are at elevated risk of neurodegeneration (particularly in regions of the brain responsible for learning and memory), impairments in functional brain activity, and the appearance of neurocognitive deficits. Heavy episodic or binge drinking impairs study habits and erodes the development of transitional skills to adulthood.

Conclusions. Underage alcohol use is associated with brain damage and neurocognitive deficits, with implications for learning and intellectual development. Impaired intellectual development may continue to affect individuals into adulthood. It is imperative for policymakers and organized medicine to address the problem of underage drinking.

Keywords: Adolescent; Young adult (qualified by alcohol dependence); Alcohol; Binge drinking; High-risk drinking; Learning; Memory; Brain; Health; Injury

Introduction

Adolescence and college attendance are high-risk periods for initiating alcohol use and engaging in patterns of high-risk drinking. A growing body of research examining the physiologic and medical consequences of drinking by preadult populations is now available. These data indicate that underage and college-age drinkers are at greater risk of neurotoxicity and harmful cognitive effects due to alcohol consumption than those who initiate alcohol use later in life. To consolidate relevant findings on this important issue and inform physicians, public health advocates, educators, parents, students, and state and national policymakers, this report reviews the epidemiology of alcohol use by young people, the physiologic effects (pharmacology) of alcohol, and the evidence linking underage drinking with harmful neurocognitive effects that may have long-term consequences. Strategies to address the problem of underage drinking are presented.

Methods

Literature searches were conducted in the MEDLINE database for English-language articles published from January 1990 through February 2003, using the search terms "adolescent," "youth," and "young adult," qualified by

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"alcohol dependence," "ethanol," and "binge drinking". The results of this search were further qualified using the search terms "cognition," "brain," "health," and "injury," yielding a total of 1,371 articles. Articles were selected based on their provision of information on (1) the epidemiology of alcohol use in adolescents and young adults; (2) the pharmacology of alcohol; and (3) the consequences of alcohol consumption on the health of adolescents and young adults. Additional references were culled from the bibliographies of these pertinent references. Statistics on underage drinking were gathered from the articles described above and from government publications and surveys, including "Monitoring the Future" (National Institute on Drug Abuse, 2002); "Youth Risk and Behavior Surveillance System" and "National Household Survey" (Centers for Disease Control and Prevention, 1998 and 2002); "A Call to Action" (National Institute on Alcohol and Alcoholism); "Traffic Safety Facts 2000" (National Highway Traffic Safety Administration); and "College Alcohol Survey" (Harvard School of Public Health).

Results

Epidemiology of alcohol use in the adolescent population

Underage drinking affects virtually every community in the United States. The age at which young people begin using alcohol has decreased over the last 35 years; on average, youths now take their first drink at the age of 12 years. There are three major national surveys that assess adolescent drug use, including alcohol. "Monitoring the Future" (MTF) is a National Institute on Drug Abuse assessment of adolescents in 8th, 10th, and 12th grades that is administered by the University of Michigan [1]. The MTF surveys approximately 50,000 adolescents annually and also includes past year and past-30-day-use prevalence. The Centers for Disease Control and Prevention conducts the Youth Risk and Behavior Surveillance System (YRBSS) [2]. The YRBSS conducts surveys among students in most states and the District of Columbia, assessing between 10,000 and 18,000 subjects every other year. Both of these national surveys assess alcohol use in a confidential manner. A third major national survey, the National Household Survey on Drug Abuse (NHSDA), sponsored by the U.S. Department of Health and Human Services' Substance Abuse and Mental Health Services Administration, is not strictly confidential, as parents are permitted to attend interviews of those under age 18 years. This may impede assessment since adolescents may be less forthcoming in this setting; this may explain some of the differences in alcohol use prevalence among the NHSDA, MTF, and YRBSS studies.

According to NHSDA, between 1995 and 2000, the number of young people aged 12–17 years who first used alcohol increased from 2.2 to 3.1 million. The prevalence of alcohol use increases with age, from 2.6% at 12 years of age to 67% of persons aged 21 years. More than 10 million American youth aged 12 –20 years report past-30-day use of alcohol. This represents more than 28% of this age group [3–5]. The pattern of alcohol use is significant as well. Although it is illegal for high school students and most undergraduate college students to purchase alcoholic beverages, alcohol use is widespread, as are occasions of heavy drinking, particularly among college students. Of the 10 million American youth who reported drinking alcohol in the past 30 days, 6.8 million or 19% were binge drinkers (defined by NHSDA as consuming five or more alcoholic beverages on the same occasion on at least 1 day in the past 30 days) and 2.1 million or 6% were heavy drinkers (defined by NHSDA as consuming five or more drinks on the same occasion on at least five different days in the past 30 days) [3–6]. Binge drinking is also called "heavy episodic," "high risk" or "dangerous" drinking. It must be noted that there is a lack of common definitions to measure heavy drinking; however, the prevalence estimates obtained by NHSDA are much lower than those obtained by other surveys and probably underestimate the extent of the problem. According to MTF, 49% of 12th graders report past-30-day use and more than 60% of these drinkers had been drunk during that period. These figures are in agreement with those obtained by the YRBSS, which found that in 2001, 47% of high school student were current drinkers and 30% of these students had engaged in heavy episodic (or binge) drinking in the past 30 days. In other words, close to half of all high school students are current drinkers and approximately 60% of current drinkers binge.

According to NHSDA, among full-time college students aged 18–22 years, 42% have reported binge drinking and 18% have reported heavy drinking in the past month. The percentage of underage persons who binge drink increases with age, ranging from approximately 1% in 12-year-olds to 48% in 21-year-olds [3–5]. The 2002 National Institute on Alcohol Abuse and Alcoholism report on college drinking, *A Call to Action*, indicated that 31% of college students met the diagnostic criteria for alcohol abuse and 6% met criteria for alcohol dependence in the past year based on self-reported drinking behavior [7].

The acute health and safety consequences of alcohol use in the younger population are well established and familiar to the public health community. Alcohol use in adolescents is associated with alcohol poisoning [8], motor vehicle crashes [9], risky sexual behaviors [10,11], suicide attempts [12–14], drowning [15], and other drug use [16]. Alcohol use is a significant contributor to injury in adolescence and may play a role in more than 50% of traumatic brain injuries in adolescents [17,18]. In 1998, more than 1400 college students aged 18–24 years died as a result of alcohol-related unintentional injuries, including motor vehicle crashes. Additionally, more than 500,000 college students were unintentionally injured while under the influence of alcohol and more than 600,000 were assaulted by another student who had been drinking [19].

While chronic diseases are relatively uncommon in adolescents, those who misuse alcohol report significantly more symptoms or medical conditions—including appetite changes, weight loss, eczema, headaches, and sleep disturbance [20]. Serum enzymatic markers of liver damage are elevated in alcohol-abusing adolescents.

Although some consider alcohol use to be a "rite of passage" for many adolescents and young adults, problematic alcohol consumption is not a benign condition that resolves with age. Individuals who first use alcohol in the age range of 11–14 years are at much greater risk of subsequently developing alcohol abuse or alcohol dependence; an estimated 40% of children who start drinking before the age of 15 years will develop such alcohol use disorders [21]. Early drinkers (past-30-day use) and even those students who experiment with alcohol (three or more drinks annually but no past month use) are more likely than nondrinkers to report academic problems, substance use, and delinquent behavior in both middle and high school. By young adulthood, early alcohol use is associated with employment problems, other substance use, and other behavioral problems [22]. Additionally, drinking onset at ages younger than 21 years is associated with experiencing alcohol-related injuries later in life [23]. The question of whether early alcohol use per se is the cause of alcohol use disorders in later life, or whether both alcohol use disorders and early drinking reflect more of a underlying vulnerability to a variety of problems, is not settled [24]. Nevertheless, if drinking onset is delayed by 5 years, a child's risk of serious alcohol problems later in life is reduced by 50% [25]. Prolonged alcohol abuse has harmful effects not only on the liver, but also on the lungs, pancreas [26], kidneys [27], endocrine system [28], immune system [29,30], cardiovascular system [31], and brain [32].

Individuals who increase their binge drinking from age 18 to 24 years and those who consistently binge drink at least once a week during this period may have problems attaining the goals typical of the transition from adolescence to young adulthood (i.e., educational attainment, employment, and financial independence) [33]. Thus, the younger one starts drinking and the greater the consumption, the greater the risk of problems now and in the future.

Pharmacology of alcohol

Pharmacokinetics

Alcohol (ethyl alcohol; ethanol) is a nonionized, lipid-soluble compound that is completely miscible with water, is readily absorbed from the stomach, small intestine, and colon, and distributes throughout total body water. Because of its high lipid solubility, alcohol readily penetrates the central nervous system (CNS). Under normal circumstances, approximately 98% of the ingested dose is oxidized, mostly by alcohol dehydrogenase (present in the stomach and liver) to acetaldehyde, with a small amount eliminated unchanged via the kidneys. A small portion of the ingested amount is also oxidized in the liver by a specific cytochrome p450 isoform (CYP2E1). This enzyme is inducible; with chronic exposure to alcohol, a larger proportion of the ingested dose may be oxidized by this enzyme. The average rate of metabolism is approximately 120 mg kg^{-1} h^{-1}, or about 30 ml (1 oz) in 3 h. A constant amount of alcohol is eliminated per hour (zero order or saturation kinetics). This amount is not affected by the blood alcohol concentration (BAC). Therefore, if alcohol is consumed at a rate greater than the amount being eliminated, BAC will continue to rise until drinking is discontinued [34].

Pharmacodynamics

Alcohol has been shown to affect brain function through many different mechanisms including effects on ion channels, neurotransmitter receptors, and cell signaling molecules. Alcohol also affects behavior and feelings, which are influenced by personal expectations and the environment in which drinking occurs.

Alcohol acts largely as a CNS depressant, interacting with a number of neurotransmitter systems. It potentiates the action of gamma-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the human brain, and inhibits the action of glutamate, the major excitatory neurotransmitter in the human brain. This contributes to the depressive effects on cognition and motor skills caused by alcohol consumption. However, alcohol consumption also is associated with increased CNS activity in certain brain regions. Alcohol stimulates the release of endorphins, which induce a brief state of euphoria that can reinforce the desire to consume alcohol. Alcohol also can stimulate dopamine release, which activates the dopaminergic reward system. This system is an important mediator to substance abuse behavior and is highly responsive to acute and repeated alcohol administration, as well. Not only does short-term alcohol exposure enhance dopamine release and "reward" activity, "conditioning" associated with repeated administration of alcohol can further promote drug expectancy effects [35,36].

Alcohol can impair learning and memory. Under normal conditions, glutamate interacts with a specific glutamate receptor, the *N*-methyl-D-aspartate (NMDA) receptor, which plays a role in mediating long-term potentiation (LTP). LTP is a persistent increase in the efficiency of a neuron's response to its neurochemical signal and is believed to underscore learning and memory processes. Alcohol dampens the activity of NMDA receptors while it is present, thereby impairing learning and memory.

Excessive alcohol use can result in brain damage and cognitive deficits. It has been proposed that alcohol-induced neurotoxicity results from increased NMDA receptor activity during withdrawal from long-term, excessive alcohol intake. Inhibition of NMDA receptors caused by excessive alcohol exposure leads to the up-regulation or sensitization of NMDA receptors (particularly in the hippocampus). In addition, calcium channels may be up-regulated after chron-

ic ethanol exposure. Upon alcohol withdrawal, the excessive activity resulting from the increased number of NMDA receptors (and perhaps calcium channels) leads to enhanced NMDA-mediated calcium influx into neurons, causing increased calcium-induced excitotoxicity that contributes to neurodegeneration and cell death [37]. Excessive glucocorticoid release induced by the stress of a withdrawal response may exacerbate the responses of already overactive NMDA receptors [37]. Thus, periods of binge drinking followed by abstinence may trigger a cycle of responses that leads to increased neurotoxicity and cognitive deficits. Similarly, chronic alcohol exposure may cause down-regulation of GABA receptors. Upon withdrawal of alcohol, diminished GABA-mediated inhibition may further contribute to increased CNS excitation that could account for many of the symptoms that characterize the withdrawal syndrome in subjects who are physically dependent on alcohol [35].

Immediate neurological consequences of alcohol use in adolescents and young adults

Acute alcohol intoxication

Because alcohol readily crosses the blood-brain barrier, the concentration of alcohol in the brain parallels the concentration established in the blood. For sporadic drinkers, obvious intoxication occurs at BACs of 50–150 mg/dL. Symptoms vary directly with the rate of drinking and may include euphoria, incoordination, ataxia, drowsiness, loss of inhibitions, garrulousness, gloominess, and belligerence. With increasing BACs, the direct depressant effects of alcohol predominate and subjects may experience lethargy, bradycardia, hypotension, and respiratory depression, sometimes complicated by vomiting and pulmonary aspiration. As BACs increase further, alcohol poisoning manifests with the development of stupor, coma, and death, usually secondary to respiratory depression with respiratory acidosis and hypotension. The median lethal BAC is approximately 450 mg/dL [34].

Adolescents typically have smaller bodies (less body mass) than adults and initially have not developed a physiological or behavioral tolerance to alcohol and its effects. Thus, they often do not need to drink very much to become intoxicated. They are also more prone to drink heavily and rapidly until intoxicated because their social, emotional control, thinking, and decision-making skills are less developed. Moreover, they are more likely than adults to lose control and are more prone to risk taking [38].

Blackouts

An episode of heavy drinking may cause a "blackout" or loss of memory for events that occurred during the drinking episode. Blackouts appear to be caused by acute dysfunction of the hippocampus [39]. Because the occurrence of blackouts is based on the amount of alcohol consumed at one drinking episode, they are common in binge drinkers, including college students [40]. Typically, an individual wakes up the morning after an episode of heavy drinking and cannot remember what occurred the previous evening, only to later learn that he or she had engaged in risky behavior [41]. One in four college students who drank reported forgetting where they were or what they did while drinking during the school year. The incidence doubled (54%) among frequent binge drinkers [40]. Females blacked out with an average of only five drinks. Males averaged nine drinks per occurrence [37]. Adolescents also seem to have more blackouts and forgetting events than adult drinkers [20]. The memory loss is generally temporary but may persist for some time after the drinking episode that caused it [39].

Hangover

Generally, within 6–24 h following heavy or prolonged drinking, drinkers experience short-term subacute symptoms or "hangover," which is a manifestation of withdrawal symptoms. These typically include headache, dizziness, nausea and vomiting, muscle weakness or pain, tremors, tachycardia, hyperventilation, sweating, depression, and irritability. Thought processes and learning also are impaired [40,42,43].

Development of the brain in adolescence

Adolescence is a period of significant neuromaturation, during which the brain's efficiency is enhanced through increased myelination and selective removal of synapses (synaptic pruning) [44]. In particular, the hippocampus and prefrontal cortex develop more actively in adolescence than in adulthood, and subcortical gray matter and limbic system structures (septal area, hippocampus and amygdala) increase in volume. At the same time, cortical structures such as the prefrontal cortex decrease in volume, perhaps as a result of synaptic pruning [45]. Through this process, the prefrontal area becomes more efficient as it matures into adulthood and enhances the ability of the adult brain (relative to the adolescent brain) to execute such tasks as planning, integrating information, abstract thinking, problem solving, judgment, and reasoning.

The brain maintains a high rate of energy expenditure to sustain this neurodevelopment during adolescence, not tapering off toward adult levels until about age 20 years. Because the adolescent brain undergoes dynamic changes, it may be more susceptible to damage from alcohol than the relatively stable adult brain. This hypothesis is supported by findings of differential changes in neurochemistry, gross morphology, and neurocognitive function between adolescents and adults in both animal and human studies.

Disruption of learning and memory by alcohol: effects on the hippocampus

Because it is unethical and illegal to provide alcohol to minors for the purpose of research, the majority of

controlled studies have been performed on laboratory animals, primarily rodents. Findings from several of these studies suggest that the adolescent brain is uniquely sensitive to alcohol. For example, intermittent exposure to ethanol during adolescence causes greater sensitization of GABA receptors compared to adults [46]. Table 1 presents some of the effects observed in studies of rodents exposed to alcohol.

One study on humans has also demonstrated age-dependent effects on memory, albeit in a slightly older population. The acute effects of alcohol (0.6 g/kg) on the acquisition of both semantic and figural memory were tested in 12 healthy adults (aged 21–29 years) using a repeated measures, placebo-controlled experimental design. The acute dose of alcohol, which produced peak BACs in the 70 mg/dL range (less than the legal driving limit), significantly impaired memory acquisition in both domains. Subjects in a younger subgroup (aged 21–24 years) were significantly more impaired in memory measures than subjects aged 25–29 years. Individuals in the age range of many college students may be at significant risk for alcohol-induced memory dysfunction [51]. These findings are consistent with data from animal research indicating that acute alcohol exposure is a more potent antagonist of memory and memory-related hippocampal activity in adolescents compared with adults.

Because adolescence is marked by progressive hippocampal development, this region of the brain may be particularly susceptible to the effects of alcohol. Indeed, some of the most severe alcohol-related neurological damage in adolescents may be in the areas of the brain responsible for learning and memory. In one study on humans, magnetic resonance imaging was used to measure the hippocampal volume in 12 adolescents and young adults (aged 13–21 years; mean age, 17.2 years) with adolescent onset alcohol use disorder [determined by Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV criteria] and 24 control subjects matched for age, sex, and handedness. Both left and right hippocampal volumes were significantly smaller (approximately 10%) in subjects with alcohol use disorders than in nonusers. Total hippocampal volume correlated positively with the age of onset and negatively with the duration of the alcohol use disorder [45].

Table 1
Effects of alcohol exposure on rodents

- Acute exposure to a relatively small amount of alcohol (equivalent to two beers) disrupts NMDA receptor-mediated long-term potentiation in the adolescent hippocampus (an area involved in learning and memory) at doses that have little or no effect on the hippocampus in adults [47].
- Alcohol significantly impairs spatial memory acquisition in adolescents [48,49].
- Shorter periods of alcohol exposure during adolescence cause long-term electrophysiological effects as measured by the electroencephalogram and event-related potentials in the hippocampus compared to adults [50].

Heavy underage drinking and functional neurological deficits

A number of neuropsychological deficits have been documented in adults with alcohol use disorders, including deficits in verbal and nonverbal performance; memory and learning; problem-solving; abstract reasoning; visuospatial function; and perceptual motor skills [52–57]. Human studies involving adolescents have typically recruited known alcohol users. Findings in these subjects indicate that adolescent alcohol users are susceptible to neurodegeneration, impairments in functional brain activity, and the appearance of neurocognitive deficits.

Functional magnetic resonance imaging (fMRI) is a noninvasive method used to measure the neural substrates of both normal and disordered brain function. It can be used to visualize brain function, by reflecting changes in the chemical composition or blood flow in brain areas (as a proxy for neural activity) during the performance of specific tasks. This technique was used to measure the functional brain activity associated with completion of a spatial working memory task in 10 young women (aged 18–25 years) with histories of alcohol dependence (for which they had received treatment) compared with 10 controls. Alcohol-dependent subjects demonstrated significantly less functional activity in the frontal and parietal regions of the brain, particularly in the right hemisphere, during tasks of spatial working memory. This type of neural activity is operational; for example, during an activity such as remembering the location of one's car in a parking lot, copying a complex picture, or solving a puzzle [58].

Functional deficits also can be measured through neurocognitive testing. Several studies have examined the neurocognitive function of adolescent and young adult alcohol users. In one study, 489 first-year college undergraduates underwent a battery of neuropsychological tests. Participants with alcohol use disorders showed deficits in visuospatial ability, while those with alcohol dependence showed deficits in both visuospatial ability and motor speed relative to participants who abused alcohol [59]. Visuospatial tasks involve visually encoding items, maintaining those images, and manipulating them. Such "inner sketchpads" are useful in everything from rearranging furniture to fitting luggage into the trunk of the car.

In another study, 38 adolescent (mean age 15.5 years) alcohol abusers (as determined by DSM-III-R criteria) and 69 comparison subjects underwent a battery of neuropsychological tests. Verbal and full-scale IQ scores were significantly lower for the adolescent alcohol abusers, who also demonstrated inferior performance in measures of reading recognition, total reading, and spelling achievement compared with controls [60].

Eckhardt et al. [61] subjected 101 alcohol-dependent individuals (as determined by DSM-III or DSM-III-R criteria) aged 18–35 years who had consumed excessive amounts of alcohol (average of 114 g four to five times

per week for 6 years) to an extensive battery of neuropsychological tests. Greater lifetime consumption was predictive of lower test scores, while abstinence predicted higher scores. In particular, greater lifetime consumption was associated with worse performance in measures of memory, abstract thought, and language.

In a more recent study, 33 alcohol-dependent (as determined by DSM-III-R criteria) adolescents aged 15–16 years with more than 100 lifetime alcohol consumption episodes and without dependence on other substances underwent an extensive neuropsychological test battery. Compared to 24 control subjects, alcohol-dependent adolescents demonstrated significant differences in test performance. Protracted alcohol use was associated with poorer performance on verbal and nonverbal retention in the context of intact learning and recognition discriminability. Recent alcohol withdrawal was associated with poor visuospatial functioning, whereas the number of lifetime alcohol withdrawal episodes was associated with poorer retrieval of verbal and nonverbal information. The alcohol abusers scored worse on verbal skills, vocabulary, general information, altered perception of spatial relationships, memory, recalling previously learned information, transferring information between different forms, and identifying groups of information that are related to each other in performance-meaningful ways. Alcohol use had the greatest impact on information recall, both verbal and nonverbal. The mental performance of alcohol-using adolescents was 10% less than their nondrinking peers. Therefore, heavy drinking in early and middle adolescence leads to measurable deficits in the retrieval of verbal and nonverbal information and in visuospatial functioning [62].

Long-term consequences of alcohol use

Alcohol is neurotoxic. Studies on human subjects have repeatedly documented neurodegeneration in alcoholics [63–65]. Neurodegeneration and structural brain lesions occur in adults with alcohol use disorders, most notably ventricular enlargement; cerebellar degeneration; alterations in the morphology of the diencephalon, medial temporal lobe structures, basal forebrain, and frontal cortex; and the specific macroscopic lesions characteristic of Wernicke's encephalopathy, central pontine myelinolysis, and Marchiafava–Bignami syndrome. There is recent evidence that alcohol causes differential brain damage in adolescents. In a binge-drinking rodent model, neurodegeneration was more severe in adolescent brains, including regions that correspond to the prefrontal cortex and parts of the limbic system in humans [66]. In humans, these regions play important roles in personality, planning and goal-directed behaviors, stress responses, and impulse control.

As previously discussed, earlier onset underage drinking is associated with a higher risk of developing alcohol use disorders in adulthood, as well as a higher risk of suffering injury. Based on animal models, binge pattern or repeated exposure to alcohol in adolescence increases susceptibility to the memory-impairing effects of alcohol during adulthood [67].

Other ancillary effects of alcohol use

Underage drinking, the occurrence of drinking episodes, and a pattern of binge drinking directly impair study habits and erode the development of transitional skills needed for progression to adulthood. In addition to the alcohol-induced toxicities and functional impairments discussed above, other neurologic effects of alcohol use may contribute to impaired learning and intellectual development in adolescents. In particular, alcohol disrupts the sleep–wake cycle, altering total sleep time, sleep latency (the time required to fall asleep), and the sequence and duration of sleep states, exacerbating daytime sleepiness and impairing school performance [68,69]. Teens with alcohol use disorders (aged 16 years) have high rates of sleep disturbance [20].

Depression is commonly comorbid with alcohol dependence and abuse. Two hypotheses have been proposed to explain this relationship: (1) people drink because they are depressed (the self-medication hypothesis); and (2) depression occurs mainly as a toxic effect of alcohol dependence. For individuals, depression comorbid with alcohol use disorder may be attributed to one of these hypotheses or a combination of the two [70]. When alcohol use is the primary disorder, altered neurochemical function has been suggested as a mechanism for depression. Research demonstrates a significant reduction in the locus coeruleus neurons of alcoholics, suggesting a change in noradrenergic neurotransmission. This may in turn be associated with symptoms of depression and memory loss [71].

The presence of mood disorders is a risk factor for suicide. Suicide is the third leading cause of death among Americans aged 15–24 years. Many suicides among adolescents are related to the use of alcohol. According to the Core Survey, 6.1% of college students who drink (5.1% of college students respondents) confided that they had suicidal thoughts and 1.6% (1.9% of drinkers) revealed they had actually tried to commit suicide within the last year due to drinking or other drug use [72]. Other researchers report that between 1.2% and 1.5% of student drinkers indicated that they tried to commit suicide within the past year due to drinking or drug use [5]. Youths aged 12–17 years who report past-year alcohol or illicit drug use are more likely than youths who did not use these substances to be at risk for suicide [73].

The social environment of underage drinking

It is important to keep in mind that the surrounding environment influences underage drinking. Historically, management and prevention of alcohol-related problems focused on changing individual behaviors, knowledge, and attitudes in isolation from the factors that gave rise to them.

Problems associated with alcohol consumption were viewed as arising out of human weakness and individual error. However, public health researchers and practitioners have learned that the environment in which people live, work, play, and consume heavily affects their attitudes, decisions, and behaviors regarding alcohol consumption. Some of the major environmental influences are the availability and cost of alcohol (number and placement of sales outlets, price); alcohol serving practices; national and local advertising, marketing, and promotion; public policies regarding these factors, as well as governing how, when, and where alcohol is consumed; social norms and policies regarding acceptable and unacceptable behaviors related to drinking; and enforcement of those norms and policies [74,75]. While these influences will not affect the effects of alcohol on the brain and body, they will influence the age at which drinking is initiated, the frequency and magnitude of consumption, and several other variables including expectations about behavior when intoxicated, access to alcohol and its affordability, peer pressure, and whether alcohol is perceived as either a "drug" or as a socially accepted consumable with little or no impact other than the pleasure it may provide.

Discussion

Alcohol use is common among adolescents and young adults. Contrary to the belief by some that underage drinkers readily recover from the toxic effects of alcohol, these individuals often suffer greater negative effects than adults due to smaller body mass, patterns of alcohol use (i.e., binge drinking), and at least initially lack of tolerance to and experience with alcohol. Alcohol use is a significant contributor to injury and death in adolescents and it is associated with increased physical symptoms and health complaints. Underage alcohol use also increases the risk of progression to alcohol use disorders and concomitant diseases.

Underage alcohol use is associated with brain damage and neurocognitive deficits. Animal studies demonstrate that adolescent brains are more susceptible than adult brains to neurochemical changes, neurodegeneration, long-lasting changes in functional activity, and impairments in spatial memory acquisition. They also demonstrate that binge-pattern exposure to alcohol during adolescence increases future susceptibility to the memory-impairing effects of alcohol. Research on humans demonstrates that adolescent alcohol use is associated with neurodegeneration; changes in functional brain activity; and neurocognitive impairments, including deficits in visuospatial ability and retrieval of verbal and nonverbal information. Memory also appears to be more strongly affected by acute alcohol use in younger individuals than in their older counterparts.

All of these effects have implications for the learning abilities and intellectual development of underage drinkers. Traumatic brain injury and neurotoxic insults may directly inhibit cognitive abilities, but other effects, including sleep disturbance, depression, and symptoms of alcohol intoxication and withdrawal, also may hinder academic performance. In a nationwide survey of more than 14,000 students at 4-year colleges, 31% and 62% of occasional and frequent binge drinkers, respectively, reported missing a class (compared to 9% of nonbinge drinkers); while 26% and 46%, respectively, reported falling behind in schoolwork (compared to 10% of nonbinge drinkers) [40]. Impaired intellectual development may continue to affect individuals even after they have entered adulthood. This supports the view that strong action is justified to protect adolescents from promotions, marketing, and cultural beliefs that normalize alcohol use and high-risk consumption.

Strategies to counteract youth drinking

Effective public policy is the best tool for changing the environmental factors that encourage underage drinking. A wide range of effective strategies are available, especially the establishment and maintenance of a minimum legal drinking age [76]. Raising the costs of purchasing alcohol (e.g., increasing prices, license fees, and excise taxes) reduces consumption by young people and can also influence their drinking behaviors by discouraging consumption of large quantities in a short period of time [77–79]. Increasing alcohol excise taxes not only represents one potentially effective means to create healthier environments and save young lives, but can also generate revenues to support programs that address alcohol problems through prevention, treatment, law enforcement, and research.

There are numerous other strategies that are effective in controlling how alcohol is sold, served, and promoted. For example, limiting the number and type of alcohol outlets in a neighborhood and regulating how those outlets serve alcohol may reduce availability and sales to minors, prevent overservice to intoxicated individuals, and increase the costs of alcohol by reducing competitive pressures to lower prices [79–82]. Measures to reduce the promotion of alcohol and proconsumption social messages influencing young people show potential for positive impacts. Among these are social norms campaigns to correct misperceptions about levels of consumption and activities to better understand the impact of advertising viewed by and promotions targeting young consumers. In fact, emerging data on the susceptibility of the adolescent brain to the harmful effects of alcohol create an imperative to critically examine alcohol advertising and marketing practices at the federal level. Eliminating alcohol service and promotion in public places frequented by young people, such as parks and recreation areas, can change the social norms perceived by them. In addition, active, adequately financed, and publicly supported enforcement of existing laws designed to prevent underage drinking and alcohol-related problems, such as drinking and driving, may discourage alcohol use and overuse. While no one measure

will suffice, comprehensive community-wide efforts can make change possible.

Physician involvement in community efforts to implement environmental changes can be helpful. On the individual patient level, physicians can be encouraged to ascertain the alcohol use behaviors of their adolescent patients—and to learn how to use effective new techniques for screening and brief office interventions [83] with adolescents [84]. While research on effective screening and intervention techniques for adolescents has been limited compared with adults, some of the same principles and strategies appear to be useful when appropriately adapted for adolescents [85–87]. When problem use is identified, knowledge of and referral to available intervention and treatment resources is vital. Physicians can encourage parents and other adult child guardians to learn more about alcohol's effects on adolescents and to monitor their own child's behaviors regarding alcohol. Discussion of parental modeling regarding alcohol consumption and intervention with parents who themselves may have alcohol-related problems are important ways to help prevent or diminish existing adolescent consumption. Physician expression of concerns to adolescent patients may also increase patient awareness of the seriousness of this health issue and its potential for creating problems while giving credibility to prevention messages they hear elsewhere.

Educating youths and their parents on the harmful effects of alcohol is another essential element. The development and availability of comprehensive school health programs with a curriculum that addresses preventable unhealthy behaviors, including underage drinking, and that fosters good decision-making skills are an important part of a community-wide approach to reducing adolescent alcohol use. Other strategies to reduce early onset and high-risk alcohol use include mandatory education in school, prenatal education and programs, and public education about alcohol that targets children and parents.

Policymakers and organized medicine need to address the problem of underage drinking through primary prevention, targeting those who may be considering drinking; secondary intervention to encourage those who already are drinking to discontinue this practice; and tertiary treatment for those individuals with alcohol use disorders. Additional research can lend weight to their efforts. For example, more research on the harmful effects of alcohol on the development of the adolescent brain; on the long-term effects and permanence of the neurologic damage; and to define the relationship between the neurocognitive effects of heavy alcohol use and comorbidities such as depression, suicide, and aggression can stimulate the development of new policies and stronger application of existing ones. Investigation into and quantification of factors that influence alcohol use by youths (e.g., how much parental service of alcohol to minors and parental modeling encourage the use of alcohol by youths) is helpful for determining education and policy needs. Other questions must be answered to aid in the development of effective interventions: What is the best way to train physicians and provide incentives for them to conduct screening and brief interventions for young people in primary care and emergency care settings? What is the best way to educate parents and their children on the consequences of alcohol use? What is the most effective school-based curriculum design for promoting healthy behaviors and discouraging unhealthy ones (including underage drinking)? What particular policies or actions in the home and community are most effective at discouraging underage drinking in each youth age group?

As additional research yields evidence that underage alcohol use is associated with brain damage and neurocognitive deficits, efforts are needed to bring this to the attention of policymakers, physicians, educators, parents, and their children. The overwhelming majority of people are currently unaware of this research and its implications for youth learning and intellectual development, public health, and policy. A young person's brain is too precious to waste.

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