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Adolescent Brain Development and Underage Drinking in the United States: Identifying Risks of Alcohol Use in College Populations

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Abstract

Alcohol use typically is initiated during adolescence, an age period that overlaps with critical structural and functional maturation of the brain. Brain maturation and associated improvements in decision-making continue into the second decade of life, reaching plateaus within the period referred to as “emerging adulthood” (18–24 years). Emerging adulthood is the typical age span of the traditionally aged college student, which includes the age (21 years) when alcohol consumption becomes legal in the United States. This review highlights neurobiological evidence indicating the vulnerabilities of the emerging adult brain to alcohol effects. This review also identifies that reduced sensitivity to alcohol sedation and increased sensitivity to alcohol-related disruptions in memory, positive family history of alcoholism effects on brain structure and function, and emerging co-morbid psychiatric conditions serve as unique vulnerabilities that increase the risks associated with underage alcohol use. These vulnerabilities likely contribute to excessive and unsupervised drinking in college students. Discouraging alcohol consumption until neurobiological adulthood is reached is important for minimizing alcohol-related disruptions in brain development and decision-making capacity, and reducing the negative behavioral consequences associated with underage alcohol use.

Keywords

adolescence; emerging adulthood; brain development; heavy episodic drinking; MLDA

Introduction

The age of onset of alcohol use, the rapid escalation in frequency and quantity of alcohol intake, and the high prevalence of alcohol use disorders all occur during a time that overlaps with the critical period of adolescent brain re-organization.^{1–6} This raises significant public health concerns given that the period of vulnerability of the developing brain extends beyond what is often referred to as adulthood, or reaching age 18, the “age of majority”. Ages 18–24, “emerging adulthood”,⁷ is the typical age span of traditional college students,

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and accordingly has been characterized as having greater functional independence and competence than adolescence, but less so compared to adulthood. This review examines developmental brain research and the neurobiological consequences of alcohol consumption when the emerging adult brain is undergoing the final stages of maturational refinement. The ongoing debate regarding lowering the minimal legal drinking age (MLDA), as a strategy for encouraging more responsible drinking and reducing rates of heavy alcohol consumption, highlights the need for a fact-driven dialogue about the dangers of drinking during “emerging adult”. Integration of these areas is needed to encourage cross talk between the community, scientific researchers and policy makers. This will lead to development of better-informed policies that are based on psychological, social and neurobiological factors associated with heavy alcohol use, which will serve to promote optimal mental health by reducing risk for addictions during the second decade of life.

The Problem in Perspective: Underage Drinking

Excessive consumption of alcohol is the third leading cause of preventable death in the United States (US).⁸ To this end, the extent and consequences of underage heavy alcohol use continues to be a major public health concern. According to the National Survey on Drug Use and Health (NSDUH) in 2009, alcohol use prevalence was shown to increase dramatically during adolescence, from only 9.3% reporting any lifetime use and 2.4% reporting past month use at age 12, up to 89.8% and 71.5% less than ten years later at age 21, respectively.⁹ As this rapid prevalence of use increases with age, drinking patterns and quantity of alcohol consumed likewise increase, reaching heavy episodic levels. Heavy episodic consumption is a pattern of drinking that increases the blood alcohol concentration (BAC) to 0.08 gram % or greater, typically defined as 5+ for adult men and 4+ drinks for adult women over a two hour period,¹⁰ although empirical studies on heavy episodic drinking often do not constrain heavy episodic drinking criterion based on hours, but rather on number of drinks consumed on a drinking occasion. Regardless, past month heavy episodic alcohol consumption prevalence rates in 2009 climbed from 1.0% at age 12 to 30.4% at age 18, and then further increased to 35.7% at age 19, 38.9% at age 20, and 50.5% at age 21, before peaking at 50.5% at age 22.⁹ A study of 10,424 first-semester freshman in the US revealed that 20% of undergraduate males consumed 10.1 drinks and 10% of females consumed 8.1 drinks, twice the heavy episodic threshold, at least once in the previous 2 weeks.¹¹ Frequent heavy episodic drinkers were more likely than infrequent heavy episodic drinkers to consume 2 or 3 times the threshold, indicating subpopulations of extreme drinkers also exist.

It is therefore not surprising that significant consequences of alcohol use are observed in college populations, typically 18–24 years old, which include 1,825 deaths from alcohol-related unintentional injuries, 599,000 alcohol-related injuries, 646,000 assaults, and 97,000 sexual assault or date rape cases per year.¹² Heavy alcohol consumption is not only associated with these negative health and life consequences, but also is directly related to increased rates of alcohol abuse and dependence diagnoses.¹³ For instance, of 14,000 students surveyed from 119 4-year colleges, 31% met the criteria for alcohol abuse and 6% met the criteria for alcohol dependence within the past year. Students identified as frequent heavy episodic drinkers had thirteen times greater odds for alcohol abuse and nineteen time

greater odds for alcohol dependence.¹⁴ Rates of alcohol abuse and dependence are also strongly influenced by age of onset of alcohol use,^{15, 16} as evidenced by epidemiological data demonstrating that a drinking onset before age 15 is associated with a 38% prevalence rate of alcohol dependence.¹⁶ If alcohol use onset is delayed until age 17, the associated prevalence rate of developing alcohol dependence later in life is 28%.¹⁶ Within the emerging adulthood period, associated prevalence rates of dependence are 15%, 17%, and 11% for an age of onset of alcohol use at 18, 19, or 20, respectively.¹⁶ While the prevalence rate is lower if individuals wait until the legal age to drink, 21 years of age in the US, the associated rate of prevalence is still significant at 10%.

To the extent that traditionally-aged college students are predominantly within the age span of emerging adulthood, it is noteworthy that exposure to a college environment has been reported to moderate alcohol consumption since drinking behaviors are more likely to be promoted among peers.¹⁷ While emerging adults that do not attend college tend to drink more as adolescents than those who attend college, college students surpass non-college peers in overall alcohol use and demonstrate a greater genetic influence on the quantity of drinks consumed per episode in emerging adulthood.¹⁷ Thus, those in the college population may have an increased vulnerability to the behavioral, biological and neurobiological consequences of alcohol use given the more episodic nature and increased amount of drinks consumed per occasion.

Reaching Neurobiological Adulthood: Period of Opportunity and Vulnerability

Consistent with physiological and social maturation from adolescence through emerging adulthood, magnetic resonance imaging (MRI) has revealed adolescence as being a critical period for brain development, including changes in cortical gray and white matter tissue from approximately 4 to 20 years of age,^{18–20} and occurring in as little as a 7 month span during adolescence.²¹ Significant evidence of brain changes from childhood through adolescence have been reported, measured using neuroimaging techniques. These studies have established that structural brain changes continue into emerging adulthood (up to age 22).^{22, 23} For instance, college students (mean age 18.6 years) scanned at the beginning of their freshman year and again 6 months later demonstrate significant structural brain changes in multiple brain regions, including the frontal lobe.²³

Cognitive processing and intellectual functioning also improve significantly during this age period, with the most dramatic improvements observed for executive functioning, which emerge towards the end of adolescence and into emerging adulthood.^{24–27} Age-related improvements in executive functions are thought to be related to the reorganization and refinement of the frontal lobe, including improved functional white matter connectivity within and between brain cortical and subcortical brain regions.^{20, 28–31} There is also an increased propensity to seek out novel stimulation and engage in risk-taking behaviors during this time,^{32, 33} perhaps due in part to immature cognitive and behavioral response inhibition abilities. Accordingly, functional MR studies document increased frontal lobe activity during executive function task performance, suggesting the importance of this region in contributing to age-related improvements in self-regulatory control.^{28, 34–36} Thus,

evidence of age-related structural and functional changes that continue into emerging adulthood suggests that the period of vulnerability of the brain extends beyond the conventional age of 18 (age of majority for some legal rights, but not for consuming alcohol in the US). Indeed, initiation of alcohol use and escalation of use to heavy episodic drinking prior to when neurobiological adulthood is reached (up to age 22) could have detrimental implications for the final stages of brain maturation.

There are significant aspects of the current cultural context that should be also considered when identifying unique risks contributing to heavy alcohol consumption in college-aged populations. There has been a boom in the alcoholic beverage industry that has increased alcohol-related drinking options beyond beer, wine, and spirits. Since the marketing of the first wine cooler in 1981, several similar beverages have been marketed as "malternative beverages", and introduced to the beverage industry market as "alcopops". "Alcopop", is a colloquial term more recently used to describe flavored alcoholic beverages, including malt beverages, beverages containing wine, e.g., wine coolers, and distilled alcohol. More recently, alcohol products that combine alcohol and caffeine are being promoted based on the ability of caffeine to mask alcohol effects^{37, 38} and to intensify intoxication, e.g., Red Bull mixed with vodka, or premixed beverages such as Four Loko. Recently, Four Loko was required by the Food and Drug Administration to remove caffeine and other stimulants from the drink based on incidents in which individuals were hospitalized or died following consumption.³⁹ Today's alcohol consumers have an entirely new class of alcoholic beverages available that 1) have a high alcohol content that is often masked by sugar or caffeine, 2) are marketed in brightly colored packages, similar to sports and energy drinks, which is highly appealing to youth, 3) are regularly and abundantly available in liquor stores, grocery stores and minimarts, and 4) are sold at low cost. Marketing geared towards youth and greater availability of alcoholic beverage choices increase levels of drinking and have additional neurobiological consequences, as well as increase dangers associated with drinking, such as drinking and driving incidents and fatalities, in the already neurobiologically vulnerable adolescent and college student populations.

Do They Know How Much They are Drinking?

It is important to examine whether college aged alcohol consumers in particular provide realistic estimates of their drinking quantities. The potential neurobiological consequences (discussed in detail in subsequent sections) of heavy and rapid alcohol intoxication are often based on the self-report of consumption of alcohol measured in units of standard drinks. Indeed, if college aged consumers do not realistically estimate drinking frequencies and quantities, then the associated magnitude of neurobiological and behavioral consequences should be considered in this context.

In a recent study, undergraduate students shown to overestimated the amount of alcohol in a standard drink and therefore underestimated their alcohol quantities consumed, and when provided with feedback regarding their definition of a standard drink, readjusted their self-reported levels to be higher than originally reported.⁴⁰ This finding highlights the need to provide education on the definition of a standard drink, in order to improve the validity of college students' responses on alcohol surveys. Some inaccuracies in self-report have also

been observed at the highest levels of alcohol consumption.⁴¹ For instance, the correlation between BAC, measured from an in vivo breath test, and next day retrospective estimates of BAC (eBAC) for the same drinking event was statistically significant in a sample of undergraduate students. However, the association between BAC and eBAC was weaker at higher intoxication levels, and was no longer significant when the BAC was over 0.08%. The authors suggest that alcohol-related impairments in memory likely disrupt accurate recall of more frequent consumption.⁴¹ Furthermore, while a large-scale clinical trial of treatment matching for alcoholism has provided evidence for reliable self-report of alcohol use in adult alcoholics, collateral informant reports in that trial did not contribute to self-report accuracy.⁴² Reasonable accuracy of self-report has been reported in college aged populations, and although correlations were only in the moderate range, collateral informants did corroborate college student self-report of alcohol consumption.⁴³ However, there is some evidence of underreporting by collaterals that was deemed to be either intentional or protective.⁴⁴

At present, these findings suggest that college student self report of alcohol consumption may have reasonable accuracy, although the data are mixed with regard to how well college students quantify a standard drink. Feedback regarding the quantities of alcohol consumed by college student drinkers may help put perspective on dangerous levels of drinking that have negative consequences, both behavioral and neurobiological, in this at-risk population. This is particularly important for those that have yet to reach neurobiological adulthood, i.e., younger than 21, given the increased vulnerability of the maturing brain to alcohol effects. Such feedback may also help college students contemplate their drinking habits (frequency and quantity), and when necessary, promote change in order to reduce dangerous behaviors associated with alcohol consumption.

Why Is Alcohol Intake So High in Emerging Adulthood and What are the Neurobiological Consequences?

Due to ethical considerations against administering alcohol to human youth, animal studies have proven useful in identifying age-related differences in the sensitivity to alcohol effects, as well as the development of alcohol tolerance and withdrawal, and the consequences of alcohol on the brain and behavior in adolescent versus adult animals, under controlled laboratory conditions. There have been numerous reports of ontogenetic differences in alcohol sensitivity, with substantial evidence for age-related increases in sensitivity to many alcohol effects, including alcohol-induced sedation^{45–48}, motor-impairment^{45, 49, 50} (but also see⁴⁸), and hypothermia.^{51–53} In general, animal data support that the outward signs of intoxication, such as sedation and motor-impairment, which serve as important self-limiting factors for alcohol consumption, are reduced in adolescents. This is important given that reduced sedation and motor impairment permit greater levels of intake. Greater alcohol intake (quantity and frequency), in turn, leads to the development of alcohol tolerance, which means that increasing amounts of alcohol are necessary to achieve the same desired effect, again permitting even greater levels of alcohol intake. Accordingly, reports of age-differences in the development of tolerance^{47, 50, 53–60} and expression of alcohol withdrawal^{61–64} (but also see⁶⁵) also exist, providing additional neurobiological

mechanisms that permit excessive alcohol intake during adolescence and into emerging adulthood.

While this body of evidence has suggested a relative resistance to alcohol-induced sedation and motor impairment during adolescence and early adulthood, other work using animal models has shown that young animals are more sensitive to alcohol-induced alterations in hippocampal long-term potentiation,^{66, 67} spatial memory deficits^{68, 69} and impairments in trace conditioning.⁷⁰ Notably, some alcohol-related memory impairments persist beyond the period of intoxication⁷⁰, and for extended durations in adolescents relative to adults.⁷¹ Human studies have likewise demonstrated consequences of alcohol use on learning and memory during adolescence, illustrating alcohol-related memory impairments on spatial working memory and visuospatial memory, as well as verbal and nonverbal recall domains.^{72–74} In a study of emerging adults (21–24 years) and young adults (25–29 years), the former group demonstrated greater impairment on acquisition of semantic verbal memory (word list learning) and figural memory following an acute alcohol challenge compared to the older group.⁷²

Animal models have also suggested that heavy episodic alcohol exposure during adolescence is associated with significant deficits in memory retention,^{58, 75} producing greater cumulative damage than chronic exposure,^{76, 77} and leading to long-term changes in cognition.⁷⁸ Heavy episodic patterns of alcohol consumption may therefore make the brain more susceptible to damage,^{79, 80} particularly at a time when heavy episodic drinking levels peak and the brain is undergoing the final stages of maturational refinement in emerging adulthood. Thus, there is converging evidence from both animal and human studies that the acquisition of learning and memory are vulnerable to alcohol use during adolescence and into emerging adulthood. Indeed, structural and functional brain changes likely serve as neurobiological substrates contributing to performance decrements associated with alcohol use.⁸¹

Long-term neurobiological consequences of heavy alcohol consumption have been well characterized in adult cohorts, demonstrating deficits across several domains of human cognition,^{82–85} with executive functioning and memory domains being the most vulnerable to disruptions by alcohol.^{86, 87} This has been demonstrated under conditions of acute alcohol challenges in both adult non-drinkers and drinkers, and in populations of heavy episodic drinkers, chronic heavy drinkers, alcohol dependent and recently abstinent alcohol dependent individuals.^{88–94} MRI and fMRI, employed to characterize neurobiological factors underlying alcohol-related cognitive deficits in adult populations have revealed alterations in brain structure^{95–100} and brain activation during performance of cognitive tasks^{101–105} (for a comprehensive review see¹⁰⁶). Magnetic resonance spectroscopy (MRS) studies have also identified alcohol-related abnormalities in neurochemistry, or brain metabolites, across a variety of alcohol drinking cohorts, although these findings have been limited to adults (for a comprehensive review see¹⁰⁷). These alcohol-related alterations largely include reductions in neuronal integrity (N-acetyl-aspartate or NAA) and cell membrane metabolism (choline), mostly in frontal lobe regions of interest, in active heavy drinkers and alcohol dependent individuals, relative to light drinking or non-drinking adult comparison subjects.

There is a growing body of literature that demonstrates alterations in cognitive performance,^{73, 108} and in brain structure and function, particularly in the frontal networks and hippocampus, in adolescent and young adults with alcohol use disorders (AUDs)^{101, 109, 110} and with heavy episodic alcohol consumption histories.^{110–113} For instance, adolescents with AUDs examined following three weeks of abstinence demonstrate poorer verbal learning and visual reproduction than non-AUD comparison adolescents.⁷³ These findings are consistent with MRI data, demonstrating reduced prefrontal cortex and hippocampal volumes in adolescents with AUDs.^{109, 114–116} Furthermore, adolescents with AUDs, aged 16.8 ± 0.7 years and reporting consumption of 41.5 ± 31.3 alcohol beverages/month, show greater fMRI blood oxygen level dependent (BOLD) responses in bilateral parietal regions and lower response in cerebellar areas during spatial working memory performance compared to adolescents without an AUD, aged 16.5 ± 0.8 years and reporting 2.0 ± 4.5 alcohol beverages/month.¹¹⁰ Lifetime number of drinks consumed predicted less spatial working memory response in frontal lobe regions, as well as reduced performance on a verbal learning and memory task, providing evidence for altered brain activation and function in youth who have a relatively brief problematic drinking history. To date, MRS studies have generally examined older recently detoxified alcohol dependent men (>35 years old), however, no MRS data are available for adolescent or emerging adult cohorts or from samples that stratify by consumption pattern (heavy episodic versus non-heavy, except¹¹⁷).

While sensitivity to alcohol-related sedation and motor-impairment reduces the outward signs of the level of intoxication in adolescents and emerging adults, early damage to the neurobiological systems contributing to learning and memory likely go undetected during the period of intoxication. Indeed, heightened levels of alcohol consumption, particularly at high doses, are associated with impaired decision-making, leading to even greater levels of alcohol intake, and potentially having serious implications for the transition from adolescence to adulthood. Therefore, in this regard, the age that one drinks is in part responsible for excessive drinking; the younger the age, the lesser the alcohol response, the greater the capacity for consumption. There are several excellent reviews available on the neurobiology of addiction, as well as on the neuropsychological sequelae and behavioral dysregulation associated with addiction (for example, see^{118–121}). In general, addiction may begin to emerge as alcohol intake escalates and tolerance develops, with the periods between episodes of alcohol use now including craving, or increased desire, for alcohol.^{118, 119} As craving for alcohol increases, symptoms can lead to alcohol addiction, characterized by compulsive drinking, loss of control over drinking, and impairments in social, behavioral and cognitive functioning.^{118–121} While this is an overly simplified, theoretical description of the cycle of addiction, and that the emergence of tolerance is neither a simple process nor an immediate pre-cursor to physiological dependence on alcohol, given the significant age differences in the sensitivity to alcohol effects and the development of tolerance, diagnosis of alcohol addiction should be considered within a developmental context (see^{122,123,124}). Data from animal models that provide empirical neurobiological support for the multiple components of responsiveness to alcohol that can lead to addiction have been successfully translated from animal studies to the human condition.^{125, 126} Regardless, it should be emphasized that the cycle of addiction begins with the initiation of alcohol use, which occurs and escalates during adolescence, peaking in emerging adulthood.

Chicken or the Egg: Family History of Alcoholism, Brain Structure and Function

Previous studies have shown that there are multiple consequences of alcohol use and abuse during early adolescence and adulthood, on brain structure and cognitive function. However, it is unclear whether structural and functional abnormalities are a direct result of the toxic effects of alcohol on the brain (consequence of use), or whether pre-existing brain conditions (antecedent to the initiation of use) are present that differentiate those who begin using at a young age compared to those who do not. One research approach for examining this “chicken or the egg” question is to compare adolescents who have no or minimal alcohol exposure, but who have a positive family history (FH+) for alcoholism, compared with age-matched adolescents who have a negative family history (FH-) for alcoholism. The use FH+ group is an ideal genetic risk model, as a positive family history of alcoholism is associated with an earlier onset on and higher magnitude of use,^{127–132} as well as a higher prevalence of alcohol use disorders in adolescents and young adults.^{133–135}

To date, there is growing evidence that youth at risk for alcohol abuse, prior to their initiation of alcohol consumption, demonstrate deficits in abstract reasoning and planning, have lower IQ scores, exhibit poorer academic performance, and have slower trajectories in cognitive improvement at one-year follow-up when compared with children of non-alcoholics.^{136–139} It is important to note that intellectual functioning, albeit lower in youth at risk for alcoholism, still falls within the average range,^{140–142} however some studies failed to document statistically significant evidence of a deficit.^{143–145} From a neurobiological perspective, FH+ youth exhibit smaller overall total brain volumes than FH- counterparts, although whole brain gray and white matter tissue volumes do not differ between FH groups.¹⁴³ FH+ females also demonstrate altered relationships between white matter volume and cognitive processing speed¹⁴³ and FH+ males exhibit larger hippocampi, predictive of poorer delayed visual memory.¹⁴⁶ Importantly, baseline hippocampal volumes did not predict substance use 4.6 years later, suggesting that hippocampal alterations may manifest as the result of alcohol exposure rather than being a pre-existing structural difference prior to the onset of use.¹⁴⁶ FH+ adolescents also have been reported to demonstrate reduced amygdalar volumes,¹⁴⁷ larger cerebellar volumes,¹⁴⁸ and abnormal laterality,¹⁴⁹ relative to FH- youth. In addition, FH+ children and adolescents demonstrate greater fMRI BOLD activation in superior frontal lobe regions during rest and less BOLD activation during a simple vigilance condition, suggestive of reduced inhibition of task-irrelevant stimuli, cognitive efficiency and goal-directed behavior.¹⁵⁰ Functional activation on the Go No-Go and Stroop tasks also demonstrate FH-related differences in frontal lobe activation during these response inhibition tasks.^{151, 152} While genetic influences on alcohol dependence symptoms have been found to be negligible,¹⁵³ studies conducted in FH+ youth suggest supporting evidence for cognitive and neurobiological vulnerabilities, e.g., reduced neuronal efficiency and/or recruitment of needed neuronal resources, that are antecedent to initiation of alcohol consumption, and which may confer a greater risk for developing an alcohol abuse problem later in life. Thus, whereas a genetic predisposition for alcoholism might influence alcohol effects on the brain and be associated with cognitive dysfunctions,¹⁵³ there is also mounting longitudinal evidence that alcohol initiation or escalation of consumption,

suggesting alcohol-related neurotoxicity, also leads to a decline in functioning (e.g.,^{73, 74, 108, 146}). Either way, alcohol use in adolescents and emerging adults can be particularly detrimental, while leading to even greater rates of future consumption.

The Frontal Lobe: Developmental Capacity for Responsibility and Link to Psychiatric Comorbidity

There is a solid neurobiological framework supporting the notion that executive functions, which include decision-making, likely continues to improve with continued brain development from 18 to 21 year of age and beyond. This decision-making capacity includes being able to make safe choices about frequency and quantity of alcohol use, regardless of drinking experience. Alcohol has been shown to have a greater impact on learning and memory prior to age 25, so it should not be assumed that learning to drink responsibly is a viable approach to reducing heavy alcohol consumption, particularly in light of the scientific evidence that alcohol itself impairs judgment and decision-making.⁸⁹ In a sample of college students followed longitudinally, disadvantageous decision-making was found to be a predictor of heavy drinking in males, but not females.¹⁵⁴ Similar evidence has been reported in adolescent cohorts, demonstrating that diminished performance on neurocognitive inhibition tasks and tasks evaluating rewards and losses over time successfully predict the later development of substance abuse or addictive behaviors like problem gambling.^{155, 156} Consistent with human data, preclinical rodent data demonstrate that adolescents that consumed high levels of alcohol also showed a higher risk preference that lasted up to 3 months after alcohol use was discontinued, relative to control animals.¹⁵⁷ To the extent that decision-making capacity reflects frontal lobe development and can effectively predict disadvantageous alcohol use in nonclinical populations of adolescents and emerging adult college students, neurobiological and cognitive evidence together provide a strong argument as to why the legal drinking age should remain at age 21, an age when neurobiological adulthood confers more optimized decision-making compared to younger ages.

While brain maturation is important for the refinement of decision-making capacity, alterations in the structure and function of the prefrontal cortex and the hippocampus, regions that are likewise susceptible to alcohol effects, have also been implicated in psychiatric conditions. Interestingly, mental health problems often manifest during adolescence and emerging adulthood, with significant prevalence rates of depression and anxiety being evident in college students.¹⁵⁸ Prevalence rates of depression, panic or generalized anxiety, and suicidal thoughts (within the month prior to survey completion) of 13.8%, 4.2%, and 2.5%, respectively, have been reported from a sample of 1,181 undergraduate students in 2005.¹⁵⁹ College students that have higher levels of depression also report greater alcohol-related problems.¹⁶⁰ In a sample of 143 emerging adult college students, negative urgency ("a tendency to behave rashly and without concern for consequences in response to negative affect"¹⁶⁰) and drinking to cope were significant mediators of the relationship between depression and alcohol problems. Thus, alcohol use is particularly problematic for students with elevated depression, as negative affect is detrimental to short term impulse control and decision-making.¹⁶⁰ Similar reports have also been published on the relationship between anxiety and alcohol-related problems in

individuals in their early to mid-twenties.^{161–163} Individuals with an anxiety disorder are 2–3 times more likely to develop an AUD than those without an anxiety disorder, and those with an AUD are 10 times more likely to develop an anxiety disorder than those without an AUD.¹⁶⁴ Positive associations between anxiety sensitivity and problem drinking also have been reported,¹⁶⁵ suggesting a dose-response relationship between these variables. Although it may be difficult to discern which condition is primary, college students should be educated about the dangers of self-medicating psychiatric illnesses with alcohol at time when rates of alcohol consumption and psychiatric illnesses are both elevated, further increasing the vulnerability of the young brain in its final developmental stages.

An Integrated New Paradigm: Future Directions

The integration of neuroscience findings with epidemiological alcohol use data provides an opportunity to develop an alternative perspective for understanding the consequences of alcohol use in vulnerable adolescent and college-aged populations. Information about the extended time course of brain development, from early adolescence into adulthood, should be incorporated with the appreciation that the young brain is relatively insensitive to some, but not all alcohol effects. This combined information provides a novel direction for a new paradigm that permits a focus on the developing brain and the capacity to make optimal decisions, an ability that increases with age and which minimizes risk and negative consequences. Together, these important data highlight the need for improved screening tools for early and problematic alcohol use, for the development of educational interventions aimed at prevention of early onset alcohol use and problematic college drinking behaviors, as well as the identification of novel treatment strategies for alcohol use disorders. All of these measures could be translated into practical institutional and societal policies, and incorporated directly into campus-based interventions aimed at reducing heavy alcohol consumption in college student populations. Thus, overall integration of these areas will encourage important cross talk and new conversations between the community, scientific researchers and policy makers, potentially leading to a reduction of maladaptive alcohol use, not just in underage populations, but at all ages.

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