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# A study on the*Hippocampus* of the brain of youth and its relation with alcoholism consumption. High rate of Failures, violence on educator , sexual assault, lack of interest in their studies, only stuck to their phones playing a whole day;Alcoholism, beer, wine , whisky and its negative consequences on Adolescent brain development of the youth of the western region

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***ABSTRACT:*** *The paper examines the negative impact of alcohol on the development of young adults, adolescents the youths. Among the numerous damages alcohol have on their body, the paper focus on the impact of alcohol on their brain development. Substantial evidence suggests that one of the main targets of alcohol toxicity in the brain is the hippocampus .Various studies[1] have compared brainstructure in adolescents who drink heavily with those whodo not (cross-sectional studies). Some of the differencesseen in adolescent heavy drinkers include:*

*• Reductions in the hippocampal volume (the central areaof the brain that controls spatial navigation and shortterm and long-term memory). This suggests that theadolescent hippocampus may be especially vulnerableto alcohol-related damage.• Decreases in prefrontal cortex (that part of the brainresponsible for executive function) of adolescents,although only consistently observed in females.• Reduced integrity of white matter regions throughout thebrain. White matter integrity is important to how the brainlearns and functions.• Reduction in a major white matter tract, the corpuscallosum, in young adult women with alcohol-usedisorders. The corpus callosum has a role in thinkingprocesses, including problem solving.Heavy drinking among youth[23] is associated with accelerated hippocampal/para-hippocampal volume decline. Such risky drinking patterns also seem to be associated with more memory blackouts (episodes of alcohol induced memory loss) and worse memory functioning likely mediated via hippocampal brain volume loss.*

***KEYWORDS: alcohol, youth, brain development, hippocampus, aggressivity***

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## I. INTRODUCTION

An important research done by the SHAAP [1] on alcoholism and development of adolescent brain showed that there is ground to worry for the mental health of the youth who consume alcohol.Adolescence can be defined as: 'the period of physical, psychological and socialtransition between childhood and adulthood. Until recently it was widely believed that the brain wasanatomically mature from childhood. Changes in socialbehaviour during the teens were attributed to hormones,social experience or the changing social environment.However, advances in the field of magnetic resonanceimaging (MRI) have shown that the structure of the brainundergoes continuous and prolonged development untilearly adulthood[2]. Brain development in adolescence is of particularsignificance when considering that the leading causes ofdeaths for 15–24-year-olds in the USA were not linkedto disease, but to behaviour[3]. Similar patterns are alsoevident in the United Kingdom, according to data releasedin 2012.[4]We now know that synaptic density – the number ofconnections (synapses) between neurons – increasesdrastically in the first few months and years of life. After thisthe brain undergoes a period of "pruning"; systematicallydiscarding parts that are inactive and strengtheningconnections that are frequently used. During childhood,this pruning mainly occurs in the areas responsible forsensory (e.g. vision) and motor processes, whereas inadolescence pruning occurs in the prefrontal cortex, thearea responsible for planning, inhibitory control, decisionmaking and social interactions, and in the temporal cortex,which includes regions supporting social cognition.

SHAAP[1] discussed the difference between adolescent and the adult brain. MRI studies have shown that, in addition to thechanges in brain structure observed during adolescencein the temporal and frontal cortex, there are also changesin brain activity and behaviour. Brain activity differencesbetween adolescents and adults have mainly beenreported in two types of situations.First, adolescents show differences in the activation of

"thesocial brain", during the processing of social information,e.g. when adolescents are asked to think about their ownor other people's thoughts, or to look at and interpretemotional expressions on the faces of other people.These changes in social and affective processing – themeans by which we make sense of social and emotionalinformation – may be crucial to understanding adolescentvulnerabilities. For example, adolescents and adults showdifferences in response to social distress; experimentsinvolving ostracism suggest that adolescents' brains

become more aroused in response to social exclusion thando adult's, and their mood is consequently more affected.[5]Structural changes in white matter during adolescenceare thought to underlie the establishment of longrange connections that support top-down modulationof behaviour.[6]Adolescents show differences inactivation of the frontal cortex in situations that require reasoning, decision-making, or inhibitory control. As aresult, it is suggested that adults are better at mentallycomprehending the longer term impact of behaviour andthat, therefore, appreciation of adverse consequencesplays a greater role in decision-making for adults thanyoung people.A range of mental health conditions commonly emergein adolescence; these include anxiety disorders, bipolardisorder, depression, eating disorders, psychosis andsubstance use experimentations.The link between adolescence and the onset ofpsychopathology may emerge from the fact that "movingparts get broken". Adolescence is characterised bychanges in the neural systems that support reasoning,social interactions, cognitive control of emotions, risk andreward evaluation. When these changes are too extremeor take place at the wrong time, and interact with certainpsychosocial factors (e.g. school or relationships) and/orbiological environmental factors (e.g. pubertal hormones orpsychoactive substances) there is an increase in the riskof cognitive, affective and addictive disorders.[7, 8]

## II. LITERATURE REVIEW

SHAAP[1] stated that Brain development continues through adolescence:synaptic pruning discards synapses that are inactive,heavily used networks are strengthened and myelination,(the 'insulating' of neurones), increases the speed of neuralcommunication. However, different brain regions reachmaturity at different times. In adolescence, though thebrain regions that control desires and motivation are welldeveloped, the frontal regions (responsible for planning,impulse inhibition and abstract thought) are still maturing.As a result, adolescents are less able to resist urges andalso less able to conceptualise the adverse consequencesof behaviour, particularly future problems. Their potentialfor mood volatility, conflict, and risky behaviour is oftenunderstandable. Research demonstrates that adolescent animals are moresusceptible than adults are to the acute effects of alcoholand that the consequences of alcohol exposure persistinto adulthood. Studies of adolescent humans suggeststhat they are also more susceptible than adults to alcoholinduced memory impairment. Animal data suggestshowever that adolescents may be less susceptible tothe motor effects of alcohol.[9]This would mean that thesigns that adults often recognise as indicating intoxication(clumsiness, unsteady walking) would not occur in anadolescent until higher levels of blood alcohol werereached. There is also evidence that adolescent heavydrinking increases the probability of alcohol disordersdeveloping in later life.[10]If adolescent alcohol use can be associated with persistenteffects in adulthood, this suggests that it may be interactingwith adolescent brain development. Various studies have compared brainstructure in adolescents who drink heavily with those whodo not (cross-sectional studies). Some of the differencesseen in adolescent heavy drinkers include:• Reductions in the hippocampal volume (the central areaof the brain that controls spatial navigation and shortterm and long-term memory). This suggests that theadolescent hippocampus may be especially vulnerableto alcohol-related damage.• Decreases in prefrontal cortex (that part of the brainresponsible for executive function) of adolescents,although only consistently observed in females.• Reduced integrity of white matter regions throughout thebrain. White matter integrity is important to how the brainlearns and functions.• Reduction in a major white matter tract, the corpuscallosum, in young adult women with alcohol-usedisorders. The corpus callosum has a role in thinkingprocesses, including problem solving.The most useful studies follow up adolescents over timeto see if the brain develops differently in those who drinkheavily compared to those who do not; these are calledlongitudinal studies. This gets around the problem of notknowing if differences seen when drinkers are comparedto non-drinkers are a cause or consequence of use of thesubstance. Only one study has done this to date, and itreported that alcohol use was associated with reducedintegrity in the superior longitudinal fasciculus, a tractwhich regulates motor behaviour.[11]

**Hippocampus region of the Brain**

Hippocampus is a complex brain structure embedded deep into temporal lobe. It has a major role in learning and memory. It is a plastic and vulnerable structure that gets damaged by a variety of stimuli. Studies have shown that it also gets affected in a variety of neurological and psychiatric disorders. Alcohol affects several brain areas such as the prefrontal cortex, the corpus callosum, the cerebellum, and the hippocampus. Substantial evidence suggests that one of the main targets of alcohol toxicity in the brain is the hippocampus; indeed the alcoholic population shows neuronal loss and a reduction in total hippocampal volume as shown by

magnetic resonance imaging [24, 25.] The hippocampus is a structure located under the cerebral cortex in the limbic system. It has a unique horseshoe-like shape and contains two regions, the cornuammonis (CA)and the dentate gyrus (DG). The CA is further divided into four zones, namely, CA1, CA2, CA3, and CA4, all of them principally containing pyramidal cells. The connectivity of these zones is especially depicted in a trilaminar loop, wherein afferences via the axons of the entorhinal cortexproject into the DG. The granule cells in the DG project mossy fibers onto the dendrites of the CA3 pyramidal neurons, and the axons from the CA3 connect to the CA1 neurons in a so-called Schaffer collateral pathway. From there, signals leave the hippocampus to return to the respectivesensory cortices. The hippocampus is one of the most-studied brain structures and is involved in complex processes such as learning and memory, including recognition memory and spatialprocessing/navigation [26 , 27 ·] Evidence shows that the dorsal (posterior in human) hippocampus develops this function, and damaging this portion strongly impairs the acquisition of learning and memory tasks [28, 29.]

## III. DISCUSSION

**Toxic effects of alcohol on the brain[12]**

National Institute on Alcohol Abuse and Alcoholism argued that alcohol interferes with the brain's communication pathways and can affect the way the brain looks and works. Alcohol makes it harder for the brain areas controlling balance, memory, speech, and judgment to do their jobs, resulting in a higher likelihood of injuries and other negative outcomes. Long-term heavy drinking causes alterations in the neurons, such as reductions in their size. Below are a few key topics related to alcohol and the brain.Adolescent brains are more vulnerable to the negative effects of alcohol than adult brains. Misuse of alcohol during adolescence can alter brain development, potentially resulting in long-lasting changes in brain structure and function.Alcohol misuse can cause alcohol-induced blackouts. Blackouts are gaps in a person's memory of events that occurred while they were intoxicated. These gaps happen when a person drinks enough alcohol that it temporarily blocks the transfer of memories from short-term to long-term storage—known as memory consolidation—in a brain area called the hippocampus.Continuing to drink despite clear signs of significant impairments can result in an alcohol overdose. An alcohol overdose occurs when there is so much alcohol in the bloodstream that areas of the brain controlling basic life-support functions—such as breathing, heart rate, and temperature control—begin to shut down. Symptoms of alcohol overdose include mental confusion, difficulty remaining conscious, vomiting, seizure, trouble breathing, slow heart rate, clammy skin, dulled responses (such as no gag reflex, which prevents choking), and extremely low body temperature. Alcohol overdose can lead to permanent brain damage or death.[12] As individuals continue to drink alcohol over time, progressive changes can occur in the structure and function of their brains. These changes can compromise brain function and drive the transition from controlled, occasional use to chronic misuse, which can be difficult to control and lead to alcohol use disorder (AUD). Individuals with moderate to severe AUD may enter a cycle of alcohol addiction. The extent of the brain's ability to return to normal following long-term sobriety is not fully understood, but a growing number of studies indicate that at least some AUD-induced brain changes—and the changes in thinking, feeling, and behaving that accompany them—can improve and possibly reverse with months of abstinence from drinking. (More detail about the neuroscience of AUD is provided in the Neuroscience: The Brain in Addiction and Recovery section of The Healthcare Professional's Core Resource on Alcohol.)Prenatal alcohol exposure can cause brain damage, leading to a range of developmental, cognitive, and behavioral problems, which can appear at any time during childhood. Alcohol can disrupt fetal development at any stage during a pregnancy—including at the earliest stages and before a woman knows she is pregnant.[12]

## IV. FINDINGS

Harmful and underage college drinking are significant public health problems, and they exact an enormous toll on the lives of students on campuses across the United States.[12] Drinking at college has become a ritual that students often see as an integral part of their higher education experience. Some students come to college with established drinking habits, and the college environment can lead to a problem. According to the 2021 National Survey on Drug Use and Health (NSDUH), 49.3% of full-time college students ages 18 to 22 drank alcohol in the past month. Of those, about 27.4% engaged in binge drinking during that same time frame. For the purposes of this survey, binge drinking was defined as consuming 5 drinks or more on one occasion for males and 4 drinks or more for females. However, some college students drink at least twice that amount, a behavior that is often called high-intensity drinking.[13]

**Consequences of Harmful and Underage College Drinking[12]**

Drinking affects college students, their families, and college communities.

- **Death**

The most recent statistics from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) estimate that about 1,519 college students ages 18 to 24 die from alcohol-related unintentional injuries, including motor vehicle crashes.[14]

- **Assault**

The most recent NIAAA statistics estimate that about 696,000 students ages 18 to 24 are assaulted by another student who has been drinking.[15]

- **Sexual Assault**

Although estimating the number of alcohol-related sexual assaults is exceptionally challenging—since sexual assault is typically underreported—researchers have confirmed a long-standing finding that 1 in 5 college women experience sexual assault during their time in college.16 A majority of sexual assaults in college involve alcohol or other substances.17, 18 Research continues in order to better understand the relationships between alcohol and sexual assault among college students. Additional national survey data are needed to better estimate the number of alcohol-related assaults.

**Academic Problems**

About 1 in 4 college students report experiencing academic difficulties from drinking, such as missing class or getting behind in schoolwork.[19]In a national survey, college students who binge drank alcohol at least three times per week were roughly six times more likely to perform poorly on a test or project as a result of drinking (40% vs. 7%) than students who drank but never binged. The students who binge drank were also five times more likely to have missed a class (64% vs. 12%).[20]

**Alcohol Use Disorder**

Around 13% of full-time college students ages 18 to 22 meet the criteria for past-year alcohol use disorder (AUD), according to the 2021 NSDUH. [21]

**Other Consequences**

Other consequences include suicide attempts, health problems, injuries, unsafe sexual behavior, and driving under the influence of alcohol, as well as vandalism, damage, and involvement with the police.

**Factors Affecting Student Drinking**

Although some students come to college already having some experience with alcohol, certain aspects of college life—such as unstructured time, widespread availability of alcohol, inconsistent enforcement of underage drinking laws, and limited interactions with parents and other adults—can lead to a problem. In fact, college students have higher binge-drinking rates and a higher incidence of driving under the influence of alcohol than their noncollege peers.The first 6 weeks of freshman year are a vulnerable time for heavy drinking and alcohol-related consequences because of student expectations and social pressures at the start of the academic year.Factors related to specific college environments also are significant. Students attending schools with strong Greek systems or prominent athletic programs tend to drink more than students at other types of schools. In terms of living arrangements, alcohol consumption is highest among students living in fraternities and sororities and lowest among commuting students who live with their families.An often-overlooked preventive factor involves the continuing influence of parents. Research shows that students who choose not to drink often do so because their parents discussed alcohol use and its adverse consequences with them.

**Addressing College Drinking**

Ongoing research continues to improve our understanding of how to address the persistent and costly problem of harmful and underage student drinking. Successful efforts typically involve a mix of strategies that target individual students, the student body as a whole, and the broader college community.

**Strategies Targeting Individual Students**

Individual-level interventions target students, including those in higher risk groups such as first-year students, student athletes, members of Greek organizations, and mandated students. The interventions are designed to change student knowledge, attitudes, and behaviors related to alcohol so they drink less, take fewer risks, and experience fewer harmful consequences.

**Categories of individual-level interventions include the following[12.]**

- Education and awareness programs ,Cognitive-behavioral skills-based approaches, Motivation and feedback-related approaches, Behavioral interventions by health professionals, Strategies Targeting the Campus and Surrounding Community, Environmental-level strategies target the campus community and student body as a whole. They are designed to change the campus and community environments where student drinking occurs. Often, a major goal is to reduce the availability of alcohol because research shows that reducing alcohol availability cuts consumption and harmful consequences on campuses as well as in the general population.

**Alcohol Overdose and College Students**

Thousands of college students are transported to the emergency room each year for alcohol overdose, which occurs when there is so much alcohol in the bloodstream that areas of the brain controlling basic life-support functions—such as breathing, heart rate, and temperature control—begin to shut down.

**Signs of this dangerous condition can include the following:**

- Mental confusion, stupor, Difficulty remaining conscious or inability to wake up
- Vomiting, Seizures,Slow breathing (fewer than eight breaths per minute)

- Irregular breathing (10 seconds or more between breaths), Slow heart rate
- Clammy skin, Dulled responses, such as no gag reflex (which prevents choking)
- Extremely low body temperature, bluish skin color, or paleness
- Alcohol overdose can lead to permanent brain damage or death, so a person showing any of these signs requires immediate medical attention. Do not wait for the person to have all the symptoms, and be aware that a person who has passed out can die. Call Police.

## V. CONCLUSION

Psychologist Dr. John F. Kelly [22], of the Harvard Medical School professor of psychiatry and director of the Massachusetts General Hospital Recovery Research Institute stated recently in the wake of the death of famous actor Matthew Perry that "Nobody ever plans on becoming addicted," he said. "Yes, people make a decision to use alcohol and use other drugs. But no one ever says, 'I want to be addicted to a drug.'"More than 140,000 people die from excessive alcohol use in the United States each year, and another more than 106,000 die from drug overdoses, according to the US Centers for Disease Control and Prevention.To understand why the problem is so widespread, people must recognize the complexities around not just getting sober — but staying sober[22.]

## REFERENCES


[1]. SHAAP Scottish health action on alcohol problems,Alcohol and the Developing Adolescent Brain: Evidence Review 2014.
[2]. Giedd JN and Rapoport JL (2010). Structural MRI of pediatric brain development: what have we learned and where are we going? Neuron, 67:728–734.
[3]. Borse NN, Gilchrist J, Dellinger AM, Rudd RA, Ballesteros MF, Sleet DA (2008).
[4]. CDC Childhood Injury Report: Patterns of unintentional injuries among 0-19 year olds in the United States, 2000–2006. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
[5]. Office of National Statistics Office (2013). 'Leading cause of death in England and Wales' and General Register Office for Scotland (2011). Births, deaths and other vital events: Causes of death.
[6]. Sebastian CL, Tan GCY, Roiser JP, Viding E, Dumontheil I, Blakemore, S-J (2011). Developmental influences on the neural bases of responses to social rejection: implications of social neuroscience for education. NeuroImage, 57:686–694.
[7]. Luna B, Padmanabhan A, O'Hearn K (2010). What has fMRI told us about the development of cognitive control through adolescence? Brain and Cognition, 72:101–113.
[8]. Goddings A-L, Burnett Heyes S, Bird G, Viner RM, Blakemore S-J (2012). The relationship between puberty and social emotion processing. Developmental Science, 15:801–811.
[9]. White A M; Bae J G; Truesdale M C; et al (2002). Chronic-intermittent ethanol exposure during adolescence prevents normal developmental changes in sensitivity to ethanol-induced motor impairments. Alcoholism: Clinical and Experimental Research 26:960–968.
[10]. Welch KA, Carson A, Lawrie SM (2013) Brain structure in adolescents and young adults with alcohol problems: Systematic Review of Imaging Studies Alcohol and Alcoholism. 48:433–444.
[11]. Bava S, Jacobus J, Thayer RE et al. (2012) Longitudinal changes in white matter integrity among adolescent substance users. Alcoholism: Clinical and Experimental Research. Jan 2013; 37(Suppl 1): E181–E189.
[12]. National Institute on Alcohol Abuse and Alcoholism NIAAA.2022. Alcohol's Effects on Health
[13]. Available at https://www.niaaa.nih.gov/publications/alcohol-and-brain-overview
[14]. Hingson RW, Zha W, White AM. Drinking beyond the binge threshold: predictors, consequences, and changes in the U.S. Am J Prev Med, 2017;52(6):717–27. PubMed PMID: 28526355.
[15]. Methodology for arriving at estimates described in Hingson R, Zha W, and Smyth D. Magnitude and trends in heavy episodic drinking, alcohol-impaired driving, and alcohol-related mortality and overdose hospitalizations among emerging adults of college ages 18–24 in the United States, 1998–2014. J Stud Alcohol Drugs. 2017;78(4):540–48. PubMed PMID: 28728636 .
[16]. Methodology for arriving at estimates described in Hingson R, Heeren T, Winter M, Wechsler H. Magnitude of alcohol-related mortality and morbidity among U.S. college students ages 18–24: changes from 1998 to 2001. Annu Rev Public Health. 2005;26:259–79. PubMed PMID: 15760289.
[17]. Muehlenhard C, Peterson Z, Humphreys T, Jozkowski K. Evaluating the one-in-five statistic: women's risk of sexual assault while in college. J Sex Res. 2017;54(4-5):549–76. PubMed PMID: 28375675.
[18]. Carey KB, Durney SE, Shepardson RL, Carey MP. Incapacitated and forcible rape of college women: prevalence across the first year. J Adolesc Health. 2015;56(6):678–80. PubMed PMID: 26003585.
[19]. Lawyer S, Resnick H, Bakanic V, Burkett T, Kilpatrick D. Forcible, drug-facilitated, and incapacitated rape and sexual assault among undergraduate women. J Am Coll Health. 2010;58(5):453–60. PubMed PMID: 20304757.
[20]. Wechsler H, Lee JE, Kuo M, Seibring M, Nelson TF, Lee H. Trends in college binge drinking during a period of increased prevention efforts. Findings from 4 Harvard School of Public Health College Alcohol Study Surveys: 1993-2001. J Am Coll Health. 2002;50(5):203–17. PubMed PMID: 11990979.
[21]. Presley CA, Pimentel ER. The introduction of the heavy and frequent drinker: a proposed classification to increase accuracy of alcohol assessments in postsecondary educational settings. J Stud Alcohol. 2006;67(2):324–31. PubMed PMID:16562416.
[22]. SAMHSA, Center for Behavioral Statistics and Quality. 2021 National Survey on Drug Use and Health. Table 8.31B—Alcohol use disorder in past year: among people aged 18 to 22, by college enrollment status and demographic characteristics: percentages, 2021
[23]. CNN Health, 2023. Matthew Perry went to rehab 15 times before getting sober. Here's why it's so hard. Madeline Holcombe.
[24]. Available at https://edition.cnn.com/2023/11/02/health/staying-sober-matthew-perry-wellness/index.html
[25]. Meda SA, Hawkins KA, Dager AD, Tennen H, Khadka S, Austad CS, Wood RM, Raskin S, Fallahi CR, Pearlson GD. Longitudinal Effects of Alcohol Consumption on the Hippocampus and Parahippocampus in College Students. Biol Psychiatry CognNeurosci Neuroimaging. 2018 Jul;3(7):610-617. doi: 10.1016/j.bpsc.2018.02.006. Epub 2018 Mar 14. PMID: 29680476; PMCID: PMC6062479.

[26]. Jernigan, T. L., Butters, N., Ditraglia, G., Schafer, K., Smith, T., Irwin, M., et al (1991). Reduced cerebral grey matter observed in alcoholics using magnetic resonance imaging. Alcohol. Clin. Exp. Res. 15, 418–427. doi: 10.1111/j.1530-0277.1991.tb00540.x
[27]. Harper, C. (1998). The neuropathology of alcohol-specific brain damage,or does alcohol damage the brain? Brain Behav. Immun. 57, 101–110.doi: 10.1097/00005072-199802000-00001
[28]. Bird, C. M., and Burgess, N. (2008). The hippocampus and memory: insights from spatial processing. Nat. Rev. Neurosci. 9, 182–194. doi: 10.1038nrm2335
[29]. Stella, F., Cerasti, E., Si, B., Jezek, K., and Treves, A. (2012). Self-organization of multiple spatial and context memories in the hippocampus. Neurosci. Biobehav. Rev. 36, 1609–1625. doi: 10.1016/j.neubiorev.2011.12.002
[30]. Moser, M. B., Moser, E. I., Forrest, E., Andersen, P., and Morris, R. G. (1995). Spatial learning with a minislab in the dorsal hippocampus. Proc. Natl. Acad.Sci. U S A 92, 9697–9701. doi: 10.1073/pnas.92.21.9697
[31]. Pothuizen, H. H., Zhang, W. N., Jongen-Rêlo, A. L., Feldon, J., and Yee, B. K.(2004). Dissociation of function between the dorsal and the ventral hippocampus in spatial learning abilities of the rat: a within-subject, within-task comparison of reference and working spatial memory. Eur. J. Neurosci. 19,705–712. doi: 10.1111/j.0953-816x.2004.03170.x