

Early exposure to alcohol leads to the development of neurological mechanisms that are primed for the future development of addictive behaviour.

EARLY-ONSET ADOLESCENT ALCOHOL CONSUMPTION AND FUTURE SUBSTANCE USE

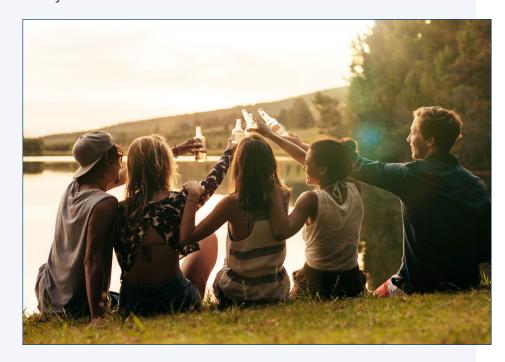
Adolescent substance use is a significant health problem worldwide^{1,2}. In the US, nearly one third of 8th grade students report ever using alcohol and 15% have used marijuana³. Given the impact of alcohol and other drug use on later life,⁴ it is important to examine the factors that influence the development of future substance use. The following piece will provide a brief overview of the neurological evidence.

While media coverage has been limited, one recent article in the Washington post has shed light on the issue⁵. In an article examining the pitfalls of the current strategy, Elizabeth Heubeck argues that the recent trends in high adolescent alcohol consumption are hampered by parental harm reduction strategies. Citing a recent study from the Medical University

of South Carolina⁶, Heubeck contends that informing adolescents of the neurological effects of alcohol misuse is a more useful strategy. This longitudinal study of 500 adolescents aged 12-19 demonstrated that parental warmth and accountability was predictive of reduced proneness to heavy drinking.6 In addition, neurological features that led to increased substance use during adolescence included poorer neuropsychological functioning on test of inhibition, working memory, reduced psychomotor speed, and lower overall IQ6.

While preliminary, these results suggest that early-onset alcohol use has and can potentially lead to broader detrimental effects on the developing brain.

Accordingly, a neurological study led by Subhash Pandey from the University of Chicago found similar results7. Analysing the brains of 11 individuals who began drinking seriously before and after the age of 21, the researchers focused on the area of the brain responsible for emotion regulation; the amygdala7. Moreover, the amygdalae of those who drank before 21 had increased amount of a molecule known as BDNF-AS, a non-coding RNA. Put simply, this molecule regulates the production of a protein called BDNF, which is vital for the normal function and maintenance of synapses throughout the brain. This means that when in BDNF-AS increases, BDNF decreases. Consequently, early-onset drinkers had 30-40 per cent lower BDNF when compared to lateonset drinkers. According to Pandey, this then



leads to emotional dis-regulation and increased anxiety⁷. It therefore seems clear that the neurological effects are not limited to cognition, but emotion regulation as well.

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There is also preliminary evidence that Alcohol can be a gateway drug to other substances. For instance, a secondary analysis of the 12th grade cohort of the 2008 Monitoring the future data⁸. Using the Guttman scale to analyse initiation into alcohol. A coefficient of reliability and scalability was calculated to evaluate fit. Using cross-tabulations and chi square, the Guttman scale demonstrated alcohol represented a gateway drug leading to tobacco, marijuana and other illicit substance use. In other words, alcohol use was associated with a future likelihood of both licit and illicit drugs.

Moreover, a conditioned place preference study with water pre-exposed mice⁹, demonstrated that mice that were exposed to ethanol early, had enhanced perception of cocaine. This experiment provides evidence that early exposure to alcohol leads to the development of neurological mechanisms that are primed for the future development of addictive behaviour.

In conclusion, it is clear from the emerging body of evidence (some contained herein), that current 'harm reduction' strategies of 'controlled drinking' or 'parent supervised early on set' drinking to combat harms from early-onset adolescent alcohol use are insufficient, and counterproductive. Myth perpetuating cultural adages like, 'we did it when we were young, didn't seem to do much harm' are now known to cause, not prevent harm.

It has been shown that early imitation has detrimental effects with respect to:

- future consumption behaviour
- cognitive function; and
- emotion regulation.

In summary, early-onset adolescent alcohol use has been shown to be detrimental over time.

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REFERENCES

- 1. Young SE, Corley RP, Stallings MC, Rhee SH, Crowley TJ, Hewitt JK. Substance use, abuse and dependence in adolescence: prevalence, symptom profiles and correlates. Drug and alcohol dependence. 2002 Dec 1;68(3):309-22.
- 2. Catalano RF, Fagan AA, Gavin LE, Greenberg MT, Irwin Jr CE, Ross DA, Shek DT. Worldwide application of prevention science in adolescent health. The Lancet. 2012 Apr 28;379(9826):1653-64.
- 3. Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE, Miech RA. Monitoring the Future national survey results on drug use, 1975-2013: Volume I, Secondary school students.
- 4. Toumbourou JW, Stockwell T, Neighbors C, Marlatt GA, Sturge J, Rehm J. Interventions to reduce harm associated with adolescent substance use. The Lancet. 2007 Apr 21;369(9570):1391-401.
- 5. http://www.essentialkids.com.au/health/health-wellbeing/safe-teen-drinking-heres-why-parents-shouldnt-facilitate-it-20190402-htd2licited1/6/20at13:10pm
- 6. Squeglia LM, Gray KM. Alcohol and drug use and the developing brain. Current psychiatry reports. 2016 May 1;18(5):46.
- 7. https://www.sciencedaily.com/releases/2019/02/190206144521.htm cited 1/6/20 at 13:10pm
- 8. Kirby T, Barry AE. Alcohol as a gateway drug: a study of US 12th graders. Journal of school health. 2012 Aug;82(8):371-9.
- 9. Molet J, Hervé D, Thiébot MH, Hamon M, Lanfumey L. Juvenile ethanol exposure increases rewarding properties of cocaine and morphine in adult DBA/2J mice. European Neuropsychopharmacology. 2013 Dec 1;23(12):1816-25.

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