

Brainwave Trust is a not-for-profit organisation that delivers easy to understand presentations to parents, professionals and the wider community using the latest scientific research on brain development.

Alcohol, booze, piss, grog, liquor. Alcohol is a psychoactive drug that easily becomes addictive (NZLC R114, 2010).

any begin to drink it during adolescence and its presence is never far from the media. A Research New Zealand (2008) report prepared for the Alcohol and Liquor Advisory Council (ALAC) observed that relatively few parents identify alcohol-related issues as a concern for parents with teenagers, particularly when compared to issues such as drugs and peer pressure. However, given alcohol use (and abuse) among New Zealand adolescents is far more common than other psychoactive drug use, maybe we don't hear as much as we should. Should we be worried or is this just what teenagers do....?

Adolescence is the period of transition from the dependence of childhood to the independence of adulthood. The adolescent brain is biologically different from an adult brain – not just an adult brain with fewer miles on the clock but a work in progress with important parts seeming to be "closed for construction".

There is a wide and ever-increasing body of literature on alcohol and adolescents. This article provides a brief review of some of the key papers and puts adolescent alcohol use in the New Zealand context.

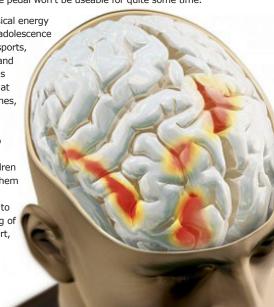
ADOLESCENT BRAIN DEVELOPMENT

Researchers thought that most brain development was finished within the first few years of life. It is only in the last 10 or so years that a second wave of brain changes has become apparent. Exuberant growth of connections, pruning and myelination all happen again during adolescence.

New connections are made in some specific areas of the brain; unused (and thus deemed to be unnecessary) ones are pruned away and the remainder may continue to be myelinated to result in a more efficient adult brain (Spear, 2000).

The limbic areas of the brain which are thought to regulate emotions and are associated with sensation seeking are turned on around puberty, but the parts for exercising judgment, impulse control and self-regulation, for example, are still maturing throughout adolescence, probably into the early 20s for girls and mid-20s for boys - like buying a high-powered car but being told that the brake pedal won't be useable for quite some time.

The emotional and physical energy that is characteristic of adolescence can be channelled into sports, academics, music, art, and various causes as well as in negative directions that produce adverse outcomes, including alcohol use (Dahl and Hariri 2004). Adolescent sensitivity to rewards appears to be different from both children and adults, prompting them to seek higher levels of novelty and stimulation to achieve the same feeling of pleasure (Paulus & Tapert, 2005).





LEGISLATION

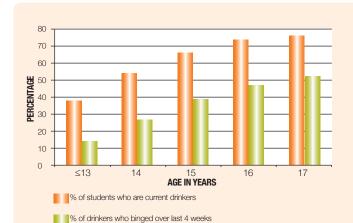
There is no legal restriction on when young people can drink. New Zealand law controls the age of purchase, but not the age of drinking. It has been legal to purchase alcohol at 18 since the limit was reduced in December 1999 – some 10 years after all US states who had previously lowered the drinking age to 18 returned to a legal minimum of 21.

In its submission to the Law Commission on Alcohol Reform (2010), the New Zealand Police described the knock-on effect of lowering the purchase age:

"... whereas before the law change, the effective drinking age was around 17–19 years of age, the de facto drinking age is now around 14–17 or even younger. Some districts are reporting that 11 and 12 year olds are now impacted upon by alcohol. Young females are also seen to be drinking to higher levels of intoxication and are subsequently at increased risk of victimisation."

PREVALENCE

Anyone exposed to adolescents in any capacity will be aware at least anecdotally, that many drink and many drink to excess. But when do they start and how much are they drinking?



Youth'07 is a cross-sectional anonymous se

Youth'07 is a cross-sectional, anonymous, self-report survey of a representative sample (9000+) of New Zealand secondary school students (Ameratunga et al, 2011) primarily aged 13-18, almost all of whom are below the legal purchasing age. Results include:

- 60.6% of students (aged 13-18) are current drinkers.
- · Over 50% of 14 year olds say they currently drink.
- . Beer is most popular choice for 14 and under. RTDs for 15 and over.
- . Binge drinking:
 - both current drinking and binge drinking rates increase with age as can be seen in this graph,
 - 34.3% of all students had engaged in binge drinking in the previous four weeks,
 - among current drinkers, 46.1% reported usually drinking 5+ per session,
 - binge drinking among current drinkers is higher in areas of low deprivation (64.9%) than high (52.0%),
 - proportion of current drinkers reporting binge drinking increased significantly from 48.5% in 2001 to 57.0% in 2007.

BINGE DRINKING

The Way We Drink, a 2004 study of the current attitudes and behaviours of New Zealanders suggests that most think binge drinking means having more than 14 standard drinks in a session. Is that consistent with the literature? Although there is some variability internationally among the definitions of binge drinking, the most common for those aged 18 to 25 is 5 **standard** drinks in a 2-4 hour period (often 4 drinks for girls) and 7 for adults.

And a **standard** drink? This also is variable, but most commonly accepted as 10 grams of pure alcohol e.g. 100 mls of table wine, or a 330ml can of 4% beer. Ready to Drinks (RTDs) vary widely in both bottle/can size and alcohol content (typically 4-8%) but usually range between 1.1 and 2.1 standard drinks. A bottle of wine is approximately 8.

Binge drinking among adolescents is not peculiar to New Zealand – it is a global problem with New Zealand's rate of around 42% of 15-16 year olds having drunk 5 or more drinks in the last 30 days being in the mid range for European countries, similar to France at 43% and lower than the UK (54%) (ESPAD, 2007). Comparability of data may be an issue, but the general point is made.

ALCOHOL AND THE ADOLESCENT BRAIN

There are many obvious and well researched risks of heavy drinking, including car accidents, injury or death, crime, violence, injuries, sexual risk taking including unprotected and unwanted sex leading to sexually transmitted infections (STI), pregnancy and abortion for example, as well as mental health disorders like depression and suicidal behaviour (Ferguson & Boden, 2011); nicotine dependence, illicit substance use, antisocial personality, conduct disorder and academic underachievement (McGue, et al 2001); alcohol poisoning ... the list goes on.

Harm from alcohol can be both immediate and long term. It is only more recently that the less easily observable potential harm to brain development has become more apparent. It is the very plasticity of the adolescent brain which makes the brain vulnerable to toxic insults such as alcohol and other drugs.

Alcohol affects a developing adolescent brain differently from an adult brain. The mechanisms which explain why and how are often not yet well understood. Alcohol exposure during adolescence not only has an immediate impact on brain function; it also may lead to consequences for various brain functions that last even into adulthood (Hiller-Sturmhofel & Swartzwelder, 2004/5).

THE RESEARCH

Numerous research studies, particularly since the beginning of this century, have begun to investigate the effect of alcohol on adolescents and their brains. Many of the early studies were in rats and non-human primates. Spear (2000) points out that increased peer-directed social interactions and elevations in exploratory and risk seeking behaviours is a characteristic of adolescence that occurs in many species, including rats and humans. Ethical issues prohibit giving alcohol to young people for research purposes so it is difficult to design studies to test the biological sensitivity of human adolescents to alcohol. Much of this evidence is now being corroborated by longitudinal and cross-sectional human studies. Findings include:

1. DEPENDENCE

The earlier alcohol use starts (more than small sips), the dramatically higher likelihood of both alcohol dependence and abuse: dependence was evident in more than 40% of those who started drinking before 15 and fell to roughly 10% among those who started drinking at 20 or older;

 rates of lifetime abuse declined from just over 11% among those who started at 16 or younger to approximately 4% among those who started at 20+ (Grant & Dawson, 1997).

2. SENSITIVITY TO ALCOHOL

Conventional wisdom would say that adolescent drinkers, typically being less experienced than adult drinkers, would be more sensitive to the effects of alcohol. However in fact the reverse appears to be true. Adolescents are relatively insensitive to the negative effects but show greater sensitivity to the pleasure effects, which may explain the high levels of binge drinking observed in adolescents (Windle et al, 2008):

- Adolescent rats are substantially less sensitive than adults to aspects
 of intoxication like sleepiness and impairment to motor activity the
 ability to walk in a straight line, for example. So, adolescents can drink
 significantly more than an equivalent adult before sleepiness, slurring of
 words etc start to kick in usual indicators of excess which can serve as
 protective factors for older drinkers;
- In a time before ethical conditions became paramount, Behar et al (1983) found no behavioural signs of intoxication in 8 to 15 year old boys who were given a dose of pure ethanol that induced peak blood alcohol levels to within the intoxicating range for adults;

- Doremus et al (2003) found that adolescents are also less sensitive to the "hangover" effect;
- These insensitivities to alcohol may be particularly pronounced during the early stages of adolescence (Varlinskaya and Spear 2004);
- Adolescent rats are more sensitive than adults to some of alcohol's
 pleasurable effects such as less shyness in social settings. Adolescents
 may enjoy the positive sensation more than adults.

3. CHANGES TO BRAIN STRUCTURE AND FUNCTION

There are numerous studies which have investigated in either, or both, rats and humans the effects of alcohol on the structure and function of the adolescent brain. The effects, while not always dramatic, can be significant. Structural changes include:

- a decrease in the volume of the hippocampus, a key region for learning new information, in heavy-drinking adolescents (Medina et al, 2007);
- reduction in the prefrontal cortex in heavy drinkers (de Bellis et al, 2005);
- reductions in the integrity of the corpus callosum (which connects the brain's two hemispheres) among heavy drinkers (Tapert et al, 2003);
- preliminary results showing widespread reductions across 18 regions of
 the brain in white matter (myelinated axons) integrity in binge drinkers
 who did not have a history of alcohol use disorder. Teens reporting
 multiple hangover symptoms showed more compromised white matter
 in several brain areas, but there was no relationship with drinking
 frequency or duration(McQueeny et al, 2009);
- it is the drinking to the point of being drunk which appears to be causing damage to cognitive functioning such as spatial working memory and pattern recognition, rather than how often and how much (Weissenborn & Duka, 2003).

4. GENDER DIFFERENCES

There are a number of subtle differences in the changes to brain structure and function between girls and boys. Alcohol may affect frontal lobe functioning more readily in females than in males, for example (Tapert et al, 2004/5).

5. BRAINS HAVE TO WORK HARDER

The brain of a chronic drinker has to "work harder" to keep things in mind, like remembering a phone number. Although there are similar changes in adolescents with only 1-2 years of heavy drinking, the young brain can compensate for any subtle alcohol-related disturbances by working other brain regions a little harder. If heavy drinking continues, by young adulthood the brain may not be able to compensate as effectively, and performance may begin to decline (Tapert al, 2001).

6. RISK OF FOETAL ALCOHOL SPECTRUM DISORDER (FASD)

According to Statistics NZ, over the last 10 years to 31 March 2012 an average of 4345 babies have been born every year to young women under 20, including an average of 34 per year to girls under 15. In addition to the risks alcohol use poses to adolescents themselves, there are additional, significant, and long-term risks for children of adolescents consuming alcohol during pregnancy.

FAS (Foetal Alcohol Syndrome) is associated with high alcohol consumption throughout pregnancy, including binge drinking (Valenzuela et al, 2012). Although heavy drinking poses the highest risk to the foetus, there is no known safe level below which no damage will occur (Ministry of Health, 2010). Alcohol use in pregnancy results in "a spectrum of defects" known as FASD (Niccols, 2007). FAS is at the most severe end and includes "growth deficiency, characteristic facial anomalies, and central nervous system dysfunction" (Fast & Conry, 2009). A child with FAS is identified by the distinctive facial anomalies but there are many other children exposed to alcohol in utero, whose effects fall short of FAS but demonstrate foetal alcohol effects, which include being hyper-responsive to stress (Fast & Conry, 2009; Niccols, 2007), and deficits in working memory and processing speed, attention difficulties, and impaired social behaviour (Boris, 2009). These foetal alcohol effects may affect a child for life but are not easy to diagnose. FAS is the most common known cause of intellectual disability (Valenzuela et al, 2012).



MARKETING TO ADOLESCENTS

This section reviews some aspects of marketing and their affect on adolescents.

1. IT HAPPENS

Research shows that alcohol is heavily marketed to younger drinkers. Longitudinal studies (Anderson et al, 2009) suggest exposure to media and commercial communications on alcohol is associated with the likelihood that adolescents will start to drink alcohol, and with increased drinking if they are already using.

2. RTDS AND PRICING

Ready to Drink (RTDs) are premixed spirits mixed with soft drinks. When four x 1.25 litre bottles of vodka (5%) mixed with your choice of raspberry, orange or cola are available for \$10, a mere \$2.50 for 5 standard drinks, pricing does not provide a barrier to access for most.

3. STANDARD DRINKS

The number of standard drinks is on all alcohol bottles/cans and is either prominently displayed or in tiny print, depending on the target market. Teenagers seem well-versed in the number of standard drinks in a bottle, even if it is so they can maximise their intake - 25% of young people who currently drink admit they do so "to get drunk" (The Way We Drink, 2004).

SHAKE COCKTAILS

DO PARENTS HAVE A ROLE?

From even before the birth of their children, parents are subjected to many and often contradictory messages about how they should parent. With regard to alcohol, these two studies might help to inform the debate.

1. HARM MINIMISATION OR ZERO TOLERANCE?

Harm-minimization accepts that alcohol is a part of normal adolescent development and that parents should supervise their children's use to encourage responsible drinking.

Zero-tolerance, on the other hand, suggests that all underage alcohol use should be discouraged.

McMorris et al (2011) studied whether adult-supervised alcohol use is a risk factor, as predicted by zero-tolerance policy, or a protective factor for harmful alcohol use, as predicted by harm-minimization policy. Adult-supervised settings for alcohol use resulted in higher levels of harmful alcohol consequences. These findings challenge the harm-minimization position that supervised alcohol use or early-age alcohol use will reduce the development of adolescent alcohol problems.

2. RISKS AND PROTECTIVE FACTORS

There are a range of risk and protective factors when considering the relationship between early alcohol use and later problems.

- Risk factors can include aspects of temperament, personality and childhood behaviour problems, family factors such as marital conflict, stressful events and violence, for example. Alcohol-specific risk factors include a family history of alcoholism, the influence of older siblings and peer factors for example;
- Protective factors include temperament, religiosity and parenting factors such as warm and supportive parenting, parental monitoring etc.
 Alcohol-specific protective factors also include the drinking behaviour of older siblings or non-drinking peers (Windle et al, 2008).

CONCLUSION

The evidence is rapidly accumulating that alcohol affects the developing brains of adolescents and affects them differently from adults. Many adolescents are drinking regularly and drinking heavily. What can be done? There are, regrettably, no silver bullets but there are a number of evidence based policy interventions (NZLR 114, 2011) which could be effective in reducing alcohol-related harm to adolescents.

Of course, alcohol hasn't just started affecting the brains of adolescents – it has done so through history – but now we better understand the potential harm. Isaac Asimov, the biochemist and science fiction writer, drew our attention to this quandary when he said "the saddest aspect of life right now is that science gathers knowledge faster than society gathers wisdom."

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REFERENCES

Ameratunga, S, Waayer, D, Robinson, E, Clark, T, Crengle, S, Denny, S, Sheridan, J & Teevale, T (2011)
Youth 07: The Health and Wellbeing of Secondary School Students in New Zealand. Young People and Alcohol.
Auckland, New Zealand: The University of Auckland, Adolescent Health Research Group.

Anderson, P, de Bruijn A, Angus K, Gordon, R, Hastings, G. (2009)
Impact of Alcohol Advertising and Media Exposure on Adolescent Alcohol Use: A Systematic Review of Longitudinal Studies. Alcohol and Alcoholism. 44(3):229-243. Oxford University Press.

Behar D, Berg C, Rapoport J et al (1983)
Behavioral and physiological effects of ethanol in high-risk and control children: A pilot study. Alcoholism: Clinical and Experimental Research 7(4):404–410.

Boris NW (2009)

substance abuse. In C. H. Zeanah (Ed.), Handbook of Infant Mental Health (3rd ed., pp. 171-179). New York, NY: The Guilford Press

BRC Marketing and Social Research (2004)
The Way We Drink: A Profile of Drinking Culture in New Zealand. Wellington: Alcohol Advisory Council of New Zealand.

Dahl, R and Hariri, A (2004)

Dani, A and Parin, A (2004)
Frontiers of Research on Adolescent Decision Making—Contributions from the Biological, Behavioral, and Social Sciences. Background paper prepared for the Planning Meeting on Adolescent Decision Making and Positive Youth Development: Applying Research to Youth Programs and Prevention Strategies. National Research Council/Institute of Medicine Board on Children, Youth, and Families, Committee on Adolescent Health and Development.

De Bellis, M., Clark, D., Beers, S et al (2000) Hippocampal volume in adolescent-onset alcohol use disorders. American Journal of Psychiatry 157:737–744.

Doremus T, Brunell S, Varlinskaya E, Spear L (2003)
Anxiogenic effects during withdrawal from acute ethanol in adolescent and adult rats. Pharmacology, Biochemistry, and Behavior 75(2):411–418

The European School Survey Project on Alcohol and Drugs (ESPAD) Report (2003)
Alcohol and Other Drug Use Among Students in 35 European Countries available online at http://www.monitoringthefuture.org/pubs/espadusa2003.pdf

Fast, DK. & Conrv. J. (2009)

Fetal alcohol spectrum disorders and the criminal justice system. Developmental Disabilities Research Reviews, 15(3), 250-257. doi: 10.1002/ddrr.66

Fergusson, D & Boden, J (2011)
Chapter 19: Alcohol use in adolescence, Improving the Transitions: Reducing Social and Psychological Morbidity During Adolescence. Auckland: Office of the Prime Minister's Science Advisory Committee.

Grant B, Dawson D (1997) Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. Journal of Substance Use 9:103–110.

Hiller-Sturmhofel S & Swartzwelder H (2004)

hol's effects on the adolescent brain: what can be learned from animal models. Alcohol Res Health 28(4):213–221.

Medina, K, Schweinsburg, A, Cohen-Zion, M, Nagel, B, & Tapert, S (2007)
Effects of alcohol and combined marijuana and alcohol use during adolescence on hippocampal volume and asymmetry. Neurotoxicology & Teratology, 29, 141-152.

New Zealand Law Commission Report 114 (NZLC R114) (2010)
Alcohol in our lives: Curbing the Harm. A Report on the Review of the Regulatory Framework for the Sale and Supply of Liquor. New Zealand Law Commission.

McGue, M, Iacono W, Legrand N & Elkins, I (2001)
Origins and consequences of age at first drink: familial risk and heretability. Alcohol Clin Exp Res. 25:1166-1173

McMorris B, Catalona, K, Kim M, Toumbourou J, Hemphill S (2011)

Influence of family factors and supervised alcohol use on adolescent alcohol use and harms: similarities between youth in different alcohol policy contexts. J Stud Alcohol Drugs. 72(3):418-28.

McQueeny, T, Schweinsburg, B, Schweinsburg, A, Jacobus, J, Bava, S, Frank, L & Tapert, S (2009) Altered White Matter Integrity in Adolescent Binge Drinkers Alcoholism: Clinical and Experimental Volume 33, Issue 7, pages 1278–1285.

Ministry of Health. (2010)
Alcohol and Pregnancy: A practical guide for health professionals. Wellington, NZ: Ministry of Health, Retrieved from http://www.moh.govt.nz

Monti P, Miranda R, Nixon K, Sher K, Swartzwelder H, Tapert S, White A, Crews F (2005)
Adolescence: Booze, brains, and behavior. Alcoholism: Clinical and Experimental Research 29:207–220

Niccols, A (2007)
Fetal alcohol syndrome and the developing socio-emotional brain. Brain and Cognition, 65(1), 135 – 142.

Paulus, M and Tapert, S (2005)
State-of-Science Review: SR-E8. Neurocognition and Social Cognition in Adolescent Drug Users: Vulnerability and Consequences. A review commissioned as part of the UK Government's Foresight Project, Mental Capital and Wellbeing.

Spear L (2000) The adolescer

scent brain and age-related behavioral manifestations. Neurosci Biobehav Rev. 24(4):417–463.

Tapert, S, Caldwell, L & Burke, C (2004-2005) Alcohol and the adolescent brain: Human studies. Alcohol Research & Health, Vol 28(4), 205-212

Tapert, S, Cheung, E, Brown, G, Frank, L, Paulus, M, Schweinsburg, A, Meloy, M, & Brown, S (2003) Neural response to alcohol stimuli in adolescents with alcohol use disorder. Arch Gen Psychiatry, 60, 727-735.

Tapert, S, Theilmann, R, Schweinsburg, A et al (2003)Reduced fractional anisotropy in the splenium of adolescents with alcohol use disorder. Proceedings of the International Society of Magnetic Resonance Medicine 11:8217.

Tapert S, Brown G, Kindermann S, Cheung E, Frank L, & Brown S (2001)
MRI measurement of brain dysfunction in alcohol dependent young women. Alcoholism: Clinical and Experimental Research, 25, 236-245.

Valenzuela, CF, Morton, RA, Diaz, MR, & Topper, L. (2012)
Does moderate drinking harm the fetal brain? Insights from animal models. Trends in Neurosciences, 35(5), 284-292

Varlinskaya El, Spear LP (2004)
Changes in sensitivity to ethanol-induced social facilitation and social inhibition from early to late adolescence.
Annals of the New York Academy of Sciences. 1021:459–461.

Windle, M, Spear, L, Fuligni, AJ et al (2008)
Transitions into underage and problem drinking: Developmental processes and mechanisms between 10 and 15 years of age. Pediatrics 121 (Suppl. 4) S273–S289.

Weissenborn, R & Duka, T (2003)
Acute alcohol effects on cognitive function in social drinkers: Their relationship to drinking habits.
Psychopharmacology, 165, 306-312.