

Does alcohol adversely affect an adolescent's brain?

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The latest version of the National Health and Medical Research Council's Australian guidelines to reduce health risks from drinking alcohol was published in 2009. It superseded the 2001 version entitled Australian alcohol guidelines: health benefits and health risks. One of the guidelines that completely changed related to recommendations for children and young people up to 18 years of age. Guideline 3 now recommends that for children and young people up to 18 years of age, not drinking alcohol is the safest option. Prior to this the recommendation was that if young people chose to drink then they should keep any drinking to a minimum, not drink beyond the maximum daily levels of two standard drinks per day for women and four for men, and most importantly, should not drink to intoxication. A gradual supervised introduction to alcohol was also recommended compared to the now recommended delayed initiation of drinking for as long as possible until late adolescence or early adulthood.

The rationale for a gradual supervised introduction to alcohol was that drinking by young people in Australia is common. For example, the 2001 National Drug Strategy Household Survey showed that Australian children as young as 14 years of age consumed alcohol on a daily and weekly basis (Australian Institute of Health and Welfare 2002). In 2004, 25% of Australian 14- to 19-year-olds were reported as drinking alcohol on either a daily or weekly basis during the past 12 months, compared with 50% of the general population 14 years and over (Australian Bureau of Statistics 2006). Numerous cultural groups, however, choose to introduce children to dilute alcohol at a young age in order to help them learn about alcohol and its effects in a safe and supportive environment (Engels and Knibbe 2000). This is aimed at helping them manage their alcohol drinking to minimize the risk to themselves and others, both when they are adolescents and then as adults.

Surely this rationale is relevant for 2010?

No, or at least, not necessarily. This is because it was initially thought that brain development was essentially completed by adolescence, but it is now accepted that the brain continues to develop during childhood and adolescence into adulthood (Paus et al. 2001; Paus 2005). From new human

neuropsychological and neuroimaging studies, the prefrontal cortex, frontal and temporal lobes including limbic brain regions such as the amygdala, hippocampus, white matter myelin, and reward circuits, all undergo active development during adolescence (Spear 2000; Chambers et al. 2003; Gogtay et al. 2004). Consequently, these structures and their functions of cognitive, behavioral, and emotional regulation may be particularly vulnerable to the adverse effects of alcohol.

Alternatively, it has been argued that deficits or developmental delays in these structures and their functions may predispose adolescents to accelerated alcohol consumption patterns and, potentially, alcohol dependence (Clark et al. 2008). For example, the brains of adolescents who habitually consume large amounts of alcohol have smaller prefrontal white matter volumes compared to those of control adolescents (De Bellis et al. 2005; Medina et al. 2008), which correlates with impaired cognitive function; the hippocampus volume, which influences both inhibitory and excitatory mechanisms, is similarly smaller in the former group (De Bellis et al. 2000).

For example, in the developing foetus the basic brain structure of cells is mapped out. In childhood, connections or wiring between brain cells occurs accompanied by the development of simple motor, sensory and language functions, and behavioural and emotional control occurs and continues until adolescence or puberty (Gogtay et al. 2004; Paus et al. 2008). This is seen in the brain as an increase in the grey matter of the brain. Indeed, the brain weight reaches adult values (approximately 1.45 kg) between 10 and 12 years of age. The fastest growth occurs during the first three years of life so that by the age of 5 years the child's brain weighs about 90% of the adult value (Dekaban 1978), such that changes to brain morphology in later childhood and adolescence are more subtle than those in the first four or five years of life.

During adolescence after the onset of puberty, the brain changes certain previously formed connections between brain cells. This is to make the connections more efficient, enabling the more complex behavioural and emotional control expected of an adult. This is observed visually as a reduction in the

grey matter of the brain (Gogtay et al. 2004), which contains the nerve cell bodies. Changes also occur to the white matter, which contains the myelin-insulated nerve fibres that carry information between the nerve cells and the spinal cord. The white matter increases in volume and becomes more myelinated during adolescence, improving the communication or processing of information between brain regions. In addition, the frontal lobes and temporal lobes containing the amygdala and hippocampus develop and change (Figure 1). The amygdala is involved in emotions such as anxiety and fear, and well in as learning and memory, while the hippocampus is involved in memory. The frontal lobe, which develops last in late adolescence/early adulthood, is involved in reasoning, problem solving, judgment and impulse control, emotions such as empathy and altruism, and also in motor control and memory. It has been suggested that development of the frontal lobe in adulthood is critical to the inhibition of childish immature, instinctive and impulsive behaviours and emotions to more considered and controlled actions (Brown et al. 2008; Fryer et al. 2008).

So, what does alcohol do to the developing brain?

Although recent research suggests that heavy alcohol consumption during adolescence and early adulthood is associated with poor cognitive function,

including impaired learning and memory formation and weakened attention and visuo-spatial skills, the results research about the effects of any alcohol on the developing adolescent brain are not definitive (Tapert et al. 2002; Clark et al. 2008).

Although the mechanism(s) by which alcohol affects brain development are yet to be fully explained, it is anticipated that alcohol adversely affects the developing white matter tracts and grey matter, especially in the frontal lobe. These adverse effects may partly explain the impulsive drinking and risk-taking behaviour in young adults that abuse alcohol (Crews and Boettiger 2009).

For example, fractional anisotropy is a measure of structure and orientation (directional coherence) of white matter fiber tracts and is used in neuroimaging. Neuroimaging of binge or heavy episodic drinking adolescents aged 16-19 years has recently demonstrated widespread reductions of fractional anisotropy in major white matter pathways (McQueeney et al. 2009). Changes and reductions in the structure and orientation of white matter fiber tracts would translate into less efficient communication of information between grey matter regions. This was specifically seen in the binge drinking adolescents' frontal and temporal lobes (including the neural bridge corpus callosum that connects the right and

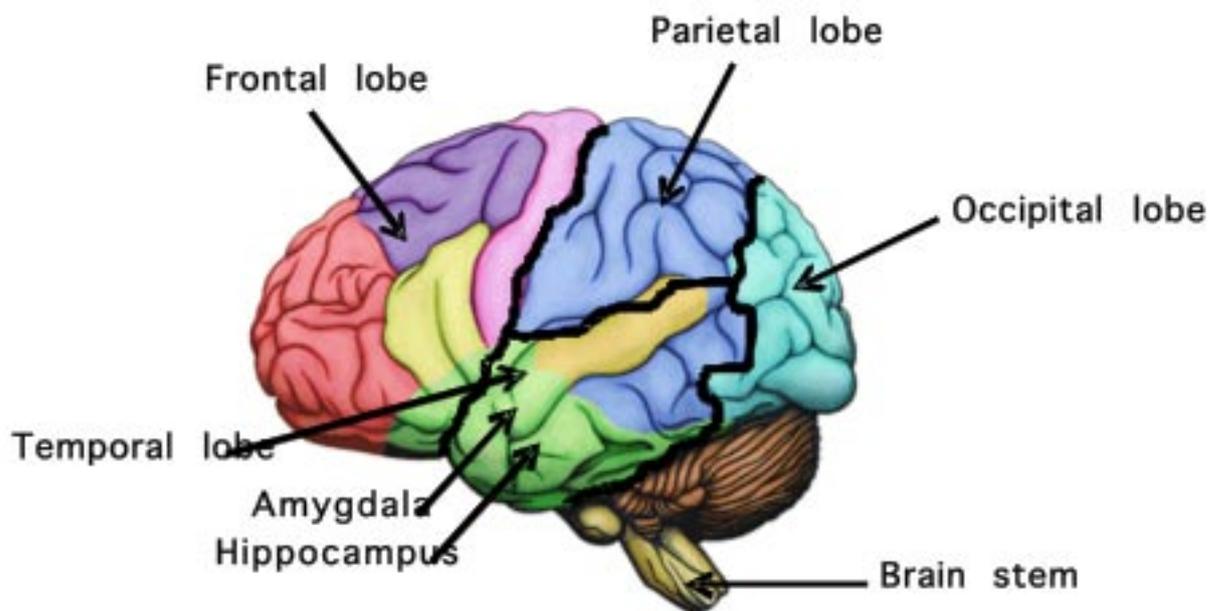


Figure 1. Schematic diagram of regions in the human brain

left brain hemispheres) as well as cerebellar, and parietal regions, which was also seen in alcohol-dependent or alcohol abusive adults (Pfefferbaum and Sullivan 2005; Schulte et al. 2005; Pfefferbaum et al. 2006a; Pfefferbaum et al. 2006b; Harris et al. 2008). In addition, the affect of alcohol on the structure and orientation of white matter fibres was dose-dependent (McQueeney et al. 2009).

The hippocampus, which is involved in information classifying, processing and learning, and the formation of long-term memories, also appears to be adversely effected by alcohol, especially during adolescence (Nagel et al. 2005; Medina et al. 2007; Squeglia et al. 2009). This was demonstrated in neuroimaging as changes and reductions in the structure and orientation in the fiber tracts extending to the hippocampus.

Interestingly, there appear to be gender differences in the effects of alcohol on the developing brain (Medina et al. 2008), which may reflect gender differences in the rate and timing of brain development in adolescence. The brains of females may be more sensitive to the adverse, or neurotoxic, affects of alcohol (Hommer et al. 2001; Schweinsburg et al. 2003).

Conclusion

Research into alcohol and the developing brain is relatively new. What we know is that alcohol has adverse effects on the developing brain, which appear to be dose-related. The effects also appear to be related to the duration and frequency of the alcohol consumption, such that regular binge drinking may result in impairments to brain structure and function, possibly predisposing to the continuation of alcohol abusive behaviours and development of alcohol dependence. We don't necessarily know, however, all the mechanisms of the adverse effects on the brain's structure and on its subsequent function, and whether they are reversible. Animal models are starting to suggest that abstinence may initiate new nerve cell growth in grey matter (Crews and Nixon 2009).

Therefore, it is recommended that the initiation of drinking be delayed for as long as possible until late adolescence or early adulthood.

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