



Underage Drinking
Research Initiative
NATIONAL INSTITUTE ON ALCOHOL
ABUSE AND ALCOHOLISM

ALCOHOL AND THE DEVELOPING ADOLESCENT BRAIN

It is evident that as humans move from childhood, through adolescence, and into adulthood, dramatic changes take place in their bodies and behavior. Many of the changes that occur during adolescence, such as an increased emphasis on social interactions with peers and a greater tendency to take risks and seek novel experiences, can be good things—helping teens gradually separate from their parents and eventually function as independent adults. But these same changes can also increase the risk for harmful behaviors, including alcohol use. Until recently, what has been less evident is the extent of change in many parts of the brain that occurs during this period and how the process of brain maturation influences overall adolescent development.

NEW UNDERSTANDING OF ADOLESCENT BRAIN DEVELOPMENT

Those who interact with children and adolescents are often struck by the ease with which they are able to learn a wide range of skills from speaking a foreign language to playing a musical instrument to mastering a sport to programming a computer. This facility for learning is due in large part to the tremendous adaptability (plasticity) of the developing brain. Imaging studies of normal brain development show an inverted U-shaped trajectory of change in gray matter volume; for girls, volume peaks at around age 8 1/2, while for boys it peaks at around age 10 1/2 (Lenroot et al., 2007). It has been postulated that the initial increase reflects an overproduction of synapses and that the subsequent thinning of cortical gray matter during adolescence may be due to a “use it or lose it” phenomenon, i.e., synapses that are not used are lost, whereas those that are used are reinforced. This may help explain the increase in processing efficiency as the brain matures, although more research will be needed to confirm this hypothesis.

Research has also shown that the brain is not fully physiologically mature until a person’s mid-twenties, and that maturational processes in the brain do not occur uniformly throughout it (Gogtay et al., 2004). These differences in maturational timing can have important

implications for behavior. Perhaps most important for understanding adolescent behavior is the maturational gap between the limbic system and the prefrontal cortex. Early in adolescence, developmental changes in the limbic system result in alterations in the control of emotions and motivation. This occurs well before the cognitive systems involving the prefrontal cortex that are responsible for self-regulation, planning, and reasoning become sufficiently mature to exert control over the impulsive and emotional reactions generated in the limbic system. The emotional intensity characteristic of adolescence may in part be explained by the uneven timing in development across these regions of the brain.

POTENTIAL VULNERABILITIES ARISING FROM ALCOHOL EXPOSURE TO THE DEVELOPING ADOLESCENT BRAIN

The remarkable plasticity of the brain during adolescence, which confers significant advantages in terms of learning, may also make the teen brain particularly vulnerable to the effects of alcohol and other drugs (Spear 2000; Teicher et al. 1995). Recent research indicates that adolescent alcohol consumption may affect cognitive functioning and/or change the developing brain in ways that increase the risk for future dependence.



COGNITIVE FUNCTIONING

Studies using animal models (rodents) indicate that alcohol has a greater adverse impact on learning and memory-related brain functions in adolescents compared to adults (e.g., see White and Swartzwelder 2005). A study in humans also showed that a single, moderate dose of alcohol can disrupt learning more powerfully in people in their early twenties, compared with those in their late twenties (Acheson et al. 1998). The effects of repeated alcohol consumption during adolescence may also be long-lasting. Studies in humans have detected cognitive impairments in adolescent alcohol abusers weeks after they stopped drinking (Brown et al. 2000), and a different pattern of brain response to tests of memory than among non-abusers (Tapert et al. 2004). Research using imaging techniques to study brain structure in humans has found adolescent-onset alcohol abuse to be associated with a reduction in the size of the hippocampus (DeBellis et al. 2000; Nagel et al. 2005), a part of the brain involved in memory and spatial navigation.

While some of these differences in brain structure and functioning appear to predate drinking, others may be a result of the adolescent alcohol exposure per se. Indeed, the magnitude of deficits in certain cognitive abilities is correlated with the number of episodes of heavy drinking and withdrawal during late adolescence (Tapert and Brown 1999). And finally, in animal studies, exposure to high levels of alcohol during adolescence has been found to produce more extensive brain damage in certain brain regions in adolescents than in adults (Crews et al. 2000).

FUTURE DEPENDENCE

Early alcohol use in humans is correlated with future alcohol dependence. Forty percent of people who report drinking before the

age of 15 also describe their drinking behavior at some point in their lives in a manner consistent with a diagnosis of alcohol dependence. In addition, in rats bred to voluntarily drink high levels of alcohol, repeated intake of alcohol during adolescence increases alcohol intake in adulthood, results in craving-like behavior, and increases potential for alcohol relapse (McBride et al. 2005).

DIFFERENTIAL SENSITIVITY OF THE ADOLESCENT BRAIN TO ALCOHOL MAY CONTRIBUTE TO RISK

Research in animals suggests that adolescents may be more sensitive to the social stimulating effects of alcohol and less sensitive to its intoxicating and sedating effects than adults (see Spear and Varlinskaya 2005). For example, adolescent rats, on their first exposure to alcohol, are less susceptible than adult rats to alcohol's sedative effects, as well as its effects on balance and motor coordination (Silveri and Spear 1998; White et al. 2002). Although it is not known whether these differences occur in humans, these findings suggest that adolescents might be able to stay awake (and perhaps continue drinking) at higher blood alcohol levels than adults, perhaps contributing to the greater incidence of binge drinking seen among adolescents than adults.

WHAT TO DO NOW?

The accumulating evidence showing that alcohol can adversely affect the developing brain is compelling. Given how pervasive drinking and binge drinking are among adolescents, the prevention and reduction of underage drinking must be a priority for everyone. Continuing research will determine the magnitude and nature of the adverse effects of alcohol on the developing brain and the extent to which they resolve over time.

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