Introduction to Underage Alcohol Issues

Why is it important to teach students so early—even in middle school—about alcohol prevention?

Underage drinking is a dangerous, persistent, widespread problem for America's youth. About 10.7 million persons aged twelve to twenty (28 percent of this age group) reported drinking alcohol during the past month.[1] Research shows that the risk for alcohol and other drug use skyrockets when children enter the sixth grade, between the ages of twelve and thirteen.[2] By the end of sixth grade, 19 percent of our nation's youth will have used alcohol, and almost 6 percent will have been intoxicated at least once in their lives.[3]

In the 2007 Monitoring the Future (MTF) study conducted by the University of Michigan, the percentage of students reporting ever using alcohol is 39 percent by the end of eighth grade, and it climbs to 72 percent by the end of high school. Approximately 16 percent of eighth graders, 33 percent of tenth graders, and 44 percent of twelfth graders admitted to drinking an alcoholic beverage thirty days prior to being surveyed.[4]

Underage drinkers consume about 10 percent of all alcohol purchased in the United States, and, according to the U.S. Department of Justice, the "vast majority of this alcohol is consumed in a risky fashion."[5] Almost 18 percent of eighth graders and 55 percent of twelfth graders report having been drunk.[6] The more we can do to prevent underage drinking, the better off our children will be in the future.

Why is underage alcohol use sometimes referred to as a "gateway to risk"?

Drinking alcohol during adolescence is dangerous. Car crashes are the leading cause of death for teenagers, and nearly one-quarter of youth in fatal traffic crashes have been drinking.[7] Alcohol-related traffic crashes and risky driving behavior are serious consequences of alcohol use by youth.[8] Nearly one in three high school students nationwide has ridden during the previous month in a car driven by someone who had been drinking, and 10 percent had driven a car after they had been drinking alcohol.[9]

Underage drinking is associated with many other social, emotional, behavioral, and legal problems during adolescence, including alcohol abuse and dependence, violence and injuries, truancy, risky sexual behavior, and other drug use throughout adolescence and into adulthood.[10]

The earlier teens begin to drink alcohol, the more risky it is. Several studies have shown that adolescents who initiate alcohol use early during adolescence are at increased risk for subsequent abuse and dependence into adulthood.[11] The risk associated with alcohol in adulthood is substantial:

- Alcohol is the third leading actual cause of death in the United States.[12]
- Long-term heavy alcohol use increases the risk of
  - several cancers[13]
  - cardiovascular disease[14]
  - stroke[15]

What does research on the adolescent brain contribute to the discussion about underage drinking?

Not all that long ago, scientists believed the human brain was 90 to 95 percent formed by the time a child entered kindergarten, around age six,[16] and by the onset of puberty, brain development was considered largely completed.[17] We now know those ideas to be false. Recent advances using brain imaging techniques called magnetic resonance imaging (MRI), functional MRIs (fMRIs), and studies of event-related potentials (ERPs), combined with animal models and research of teens' performance on neuropsychological measures—standardized tests of thinking and memory skills—are greatly advancing our knowledge about the adolescent brain and the impact of alcohol on its development, including possible long-term negative consequences that may extend into adulthood.[18]

When the thickening of the brain's gray matter is finished at puberty (about eleven years for girls and twelve years for boys), the brain starts a thinning process to eliminate unused neural connections—a process that continues into early adulthood and results in increased speed and better information processing.[19] The "use it or lose it" principle is involved in this synaptic pruning, and its result is believed to be related to a person's ability to sustain high-level cognitive control over behavior.[20] Myelination (the insulation of established neural connections that optimizes transfer of neural information across the central nervous system) is another key feature of adolescent brain maturation,[21] also thought to be related to cognitive control of behavior.[22]

The maturation of the prefrontal cortex—the region of the brain associated with critical thinking and decision making—is not completed until early adulthood.[23] The subcortical gray matter and the limbic system structures (which includes the hippocampus and amygdala) increase in volume during adolescence.[24] Figure 1 shows these and other important areas

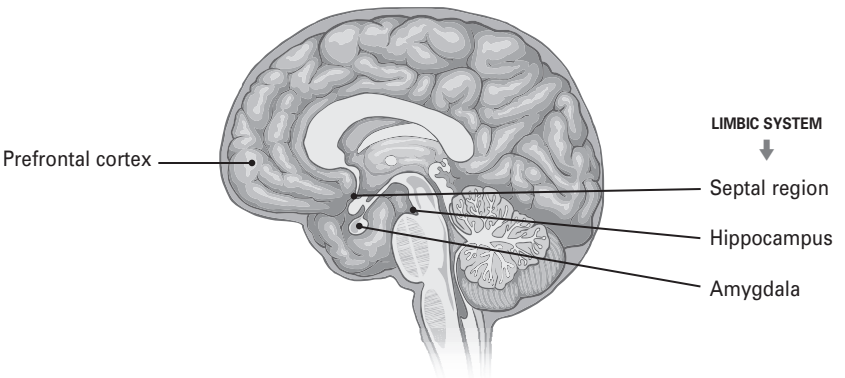
What Is fMRI?

Functional magnetic resonance imaging (fMRI) is a specialized type of neuroimaging scan or MRI. Unlike the MRIs used by doctors as diagnostic tools, the fMRI is primarily used in research studies to learn which regions of the brain are active during a specific cognitive function by measuring the blood flow response to neural activity. Because it is a noninvasive procedure, it can be used in studies of normal children and adolescents.

of the brain. The development of the hippocampus during adolescence may be especially significant given its association with learning and memory.[25] Recent studies using fMRIs suggest that it is both the maturation of these distinct areas of the brain and the brain's increased ability to work in an integrated manner, perhaps due to the processes of synaptic pruning and myelination occurring during adolescence, that results in adult-level cognitive control of behavior.[26]

FIGURE 1

Brain Development



These changes in the brain's underlying structure and physiology are likely related to observed changes in adolescents' emotional and cognitive processes and behavior.[27] During adolescence, changes are observed in several cognitive processes (for example, problem solving, reasoning, working memory, response inhibition, attention allocation), as well as other higher-order functions including mood regulation, decision making, and impulse control.[28] Developmental fMRI studies of response inhibition show that brain function changes with age and improved behavioral performance.[29] Response inhibition, in general terms, is the ability to suppress competing responses or distractions. Response inhibition is present in every voluntary action people perform, since choosing a response requires the rejection of other possible responses. Its development begins in infancy with continued improvement throughout childhood. However, until recently, studies stopped at late childhood, leaving a gap in our knowledge about the development of this crucial ability during adolescence.[30]

Recent fMRI studies of adolescents indicate that while some teens, at times, can demonstrate mature response inhibition, the integration of brain circuitry supporting such cognitive control over behavior does not work at adult levels. Even younger children can perform isolated response inhibition trials accurately, but it is during adolescence that the ability to do so consistently and with greater efficiency appears to develop.[31] This suggests that the underlying brain mechanisms supporting response inhibition, perhaps because they are not yet fully formed in adolescents, may be especially vulnerable under high-demand

situations.[32] So, for example, the belief of some that parents can teach appropriate drinking skills to adolescents by providing alcohol in the "safe" home environment may not translate to other high-demand environments like parties or unsupervised teen gatherings with heightened levels of emotion, motivations to drink, and distracting stimuli and competing tasks because the underlying system or brain circuitry for exerting cognitive control over behavior is not yet sufficiently established.

As research accumulates on the changes occurring in the brain during adolescence, concern is increasing about what the introduction of a potent neurotoxin like alcohol might do to these crucial developmental and maturational processes during the second decade of life.[33] For those surprised to hear alcohol described as a neurotoxin, consider the harm that can be done to a fetus with in utero exposure to alcohol. Such exposure can lead to fetal alcohol syndrome (FAS), a birth defect characterized by central nervous system damage, among other features, resulting from prenatal alcohol exposure.

Adolescents' smaller body mass, their initial lack of tolerance to and experience with alcohol, and a pattern of use favoring heavy and rapid drinking suggest young people may suffer greater negative effects of alcohol use than adults.[34] Adolescent alcohol abuse is associated with damage to the brain and neurocognitive deficits that can have direct implications for learning and other cognitive abilities that could continue into adulthood.[35] However, further research is needed to determine which comes first, the brain and neurocognitive deficits or the adolescent's heavy alcohol use.[36] It is also unclear at present which neurocognitive deficits evident in adolescents with alcohol-use disorders are reversible after long-term abstinence, because longitudinal studies have yet to be conducted.[37] Additional research is urgently needed to determine the nature, extent, and persistence of alcohol-related brain injury in adolescence, given its potential for far-reaching consequences.[38]

For multiple reasons, scientists have turned to animal research to address some of the questions about the neurotoxic effects of alcohol on the developing adolescent brain. Laboratory animals allow for better experimental control of the amount and type of neurotoxins to introduce to the brain than do observational studies of teens already identified as having alcohol or other drug abuse problems. Researchers today are reluctant to use adolescents in studies about the neurotoxic effects of alcohol on the developing brain because of ethical and legal concerns about providing alcohol to underage research participants given the known risks.[39]

Animal models of underage drinking suggest that adolescents may be more sensitive to some effects of alcohol and less sensitive to others. Adolescent rats show more sensitivity than adult rodents to alcohol's social facilitation effects, which could encourage increased intake of alcohol.[40] Adolescent rats also show more sensitivity than do adult rats to the effects of alcohol on memory and learning that are believed to result from alcohol's action

on the hippocampus.[41] Chronic alcohol exposure consistently results in long-term cognitive impairments, with adolescent rats especially sensitive to these consequences.[42] The rat equivalent to adolescent binge drinking—chronic intermittent alcohol exposure—was associated with damage to the frontal association cortex and other frontal regions in adolescent, but not adult, rats.[43] Binge drinking, a pattern of typical adolescent use, increases future susceptibility to the memory-impairing effects of alcohol in animal models.[44]

This parallels what we know from humans about the important changes in the hippocampus and prefrontal cortex during adolescence, described above. In addition, a preliminary study of humans that compared twelve adolescents with diagnosed alcohol dependence or abuse with twenty-four comparison adolescents found a reduction in the size of the hippocampus among those with alcohol disorders.[45] Further research is needed to clarify if these changes persist into adulthood and how they relate to brain functioning in humans.

There are effects of alcohol that adolescent rats appear to have less sensitivity to than do adult rats, including alcohol-related motor impairment and alcohol-induced sedation.[46] Unfortunately, these negative effects of alcohol intake can serve to moderate use because difficulty walking and/or becoming sleepy can serve as cues to limit intake; furthermore, these adolescent-related insensitivities to alcohol may be most pronounced during early adolescence.[47] A rare study in human children and adolescents ages eight to fifteen years demonstrated similar insensitivity.[48] Subjects in this study, eleven boys with a family history of alcoholism and eleven age-matched controls, were given a mixture of 0.5 ml/kg of pure ethanol with 12 ounces of Diet 7UP to drink within three minutes, resulting in blood alcohol levels within the intoxicating range for adults.[49] However, "little gross behavioral change occurred in the children," and none behaved grossly "intoxicated" compared to what the researchers observed with their "adult practice subjects."[50]

This brief overview of the exciting new research on the adolescent brain development and maturation can be used to inform decisions today about prevention strategies for adolescent alcohol use.[51] More answers are likely forthcoming as research techniques improve, more data are collected, and theories are developed about the brain/behavior connections and age-related changes. Current adolescent brain research supports prevention programs that have clear no-use messages, given what is now known about the significant maturation and development occurring in the brain during adolescence. Alcohol is a neurotoxin that may interfere with these important processes.

THERE IS NO SAFE LEVEL OF ALCOHOL USE FOR TEENS. ALL UNDERAGE DRINKING SHOULD BE DISCOURAGED BY RESPONSIBLE ADULTS.

No information is available to recommend a "safe" level of alcohol intake for adolescents. Furthermore, adolescents are cognitively immature in neurological processes related to decision making.[52] Public policies to discourage underage drinking, like the maintenance and enforcement of the minimum drinking age, are essential given the neurocognitive effects of alcohol on adolescents and college students.[53] Given that most people are currently unaware of the association of underage alcohol use with brain damage and neurocognitive deficits, efforts are needed to better publicize this information so that policy makers, physicians, educators, parents, and adolescents better appreciate the risk of underage drinking.[54] Comprehensive programs like Project Northland, which include interventions for the individual to learn and rehearse behavioral skills, as well as programs for the family, school, and community to provide environments conducive to no-use norms, best match the immature neurological development of adolescence.[55]

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Notes

1. Substance Abuse and Mental Health Services Administration, Office of Applied Studies, Results from the 2007 *National Survey on Drug Use and Health: National Findings,* NSDUH Series H-34, DHHS Publication No. SMA 08-4343 (Rockville, MD: 2008).

2. J. E. Donovan, S. L. Leech, R. A. Zucker, C. J. Loveland-Cherry, J. M. Jester, H. E. Fitzgerald, L. I. Puttler, M. M. Wong, and W. S. Looman, "Really Underage Drinkers: Alcohol Use among Elementary Students," *Alcoholism: Clinical and Experimental Research* 28, no. 2 (2004): 341–49.

3. Donovan et al., "Really Underage Drinkers."

4. L. D. Johnston, P. M. O'Malley, J. G. Bachman, and J. E. Schulenberg, "Overall, Illicit Drug Use by American Teens Continues Gradual Decline in 2007" (Ann Arbor, MI: University of Michigan News Service, 2007). Online at http://www.monitoringthefuture.org.

5. D. K. Eaton, L. Kann, S. Kinchen, S. Shanklin, J. Ross, J. Hawkins, W. A. Harris, R. Lowry, T. McManus, D. Chyen, C. Lim, N. D. Brener, and H. Wechsler, "Youth Risk Behavior Surveillance United States, 2007," *Morbidity and Mortality Weekly Report* 57, no. 4 (2008): 1–131.

6. Johnston et al., "Overall, Illicit Drug Use."

7. National Highway Traffic Safety Administration, "Fatality Analysis Reporting System Data Files 1976–2002" (Washington, DC: National Center for Statistics and Analysis, 2006).

8. National Highway Traffic Safety Administration, "Fatality Analysis"; J. S. Zakrajsek and J. T. Shope, "Longitudinal Examination of Underage Drinking and Subsequent Drinking and Risky Driving," *Journal of Safety Research* 37, no. 5 (2006): 443–51.

9. National Center for Chronic Disease Prevention and Health Promotion, "YRBSS Youth Online: Comprehensive Results," http://www.cdc.gov/HealthyYouth/yrbs (retrieved September 12, 2008).

10. B. F. Grant and D. A. Dawson, "Age of Onset of Drug Use and Its Association with DSM-IV Drug Abuse and Dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey," *Journal of Substance Abuse* 10, no. 2 (1998): 163–73; B. F. Grant, F. S. Stinson, and T. Harford, "Age at Onset of Alcohol Use and DSM-IV Alcohol Abuse and Dependence: A 12-year Follow-up," *Journal of Substance Abuse* 13 (2001): 493–504; E. Gruber, R. J. DiClemente, M. Anderson, and M. Lodico, "Early Drinking Onset and Its Association with Alcohol Use and Problem Behavior in Late Adolescence," *Preventive Medicine* 25, no. 3

(1996): 293–300; R. Hingson, T. Heeren, S. Levenson, A. Jamanka, and R. Voas, "Age of Drinking Onset, Driving After Drinking, and Involvement in Alcohol Related Motor-Vehicle Crashes," *Accident Analysis and Prevention* 34, no. 1 (2002): 85–92; R. Hingson, T. Heeren, R. Zakocs, M. Winter, and H. Wechsler, "Age of First Intoxication, Heavy Drinking, Driving After Drinking and Risk of Unintentional Injury among U.S. College Students," *Journal of Studies on Alcohol* 64, no. 1 (2003): 23–31; R. Hingson, T. Heeren, and R. Zakocs, "Age of Drinking Onset and Involvement in Physical Fights After Drinking," *Pediatrics* 108, no. 4 (2001): 872–77; R. Hingson, T. Heeren, M. R. Winter, and H. Wechsler, "Early Age of First Drunkenness as a Factor in College Students' Unplanned and Unprotected Sex Attributable to Drinking," *Pediatrics* 111, no. 1 (2003): 34–41; R. W. Hingson, T. Heeren, and M. R. Winter, "Age at Drinking Onset and Alcohol Dependence—Age at Onset, Duration, and Severity," *Archives of Pediatrics & Adolescent Medicine* 160, no. 7 (2006): 739–46.

11. J. D. Grant, J. F. Scherrer, M. T. Lynskey, M. J. Lyons, S. A. Eisen, M. T. Tsuang, W. R. True, and K. K. Bucholz, "Adolescent Alcohol Use Is a Risk Factor for Adult Alcohol and Drug Dependence: Evidence from a Twin Design," *Psychological Medicine* 36, no. 1 (2006): 109–18; J. Guo, L. M. Collins, K. G. Hill, and J. D. Hawkins, "Developmental Pathways to Alcohol Abuse and Dependence in Young Adulthood," *Journal of Studies on Alcohol* 61, no. 6 (2000): 799–808; J. D. Hawkins, R. F. Catalano, and J. Y. Miller, "Risk and Protective Factors for Alcohol and Other Drug Problems in Adolescence and Early Adulthood—Implications for Substance-Abuse Prevention," *Psychological Bulletin* 112, no. 1 (1992): 64–105; Hingson et al., "Age at Drinking Onset."

12. A. H. Mokdad, J. S. Marks, D. F. Stroup, and J. L. Gerberding, "Actual Causes of Death in the United States, 2000," *Journal of the American Medical Association* 291, no. 10 (2004): 1238–45.

13. V. Bagnardi, M. Biangiardo, C. La Vecchia, and G. Corrao, "Alcohol Consumption and the Risk of Cancer: A Meta-analysis," *Alcohol Research and Health* 25, no. 4 (1997): 263–70.

14. G. Corrao, V. Bagnardi, A. Zambon, and C. LaVecchia, "A Meta-analysis of Alcohol Consumption and the Risk of 15 Diseases," *Preventive Medicine* 38, no. 5 (2004): 613–19; G. Corrao, L. Rubbiati, V. Bagnardi, A. Zambon, and K. Poikolainen, "Alcohol and Coronary Heart Disease: A Meta-analysis," *Addiction* 95, no. 10 (2000): 1505–23; J. Rehm, G. Gmel, C. T. Sempos, and M. Trevisan, "Alcohol-related Morbidity and Mortality," *Alcohol Research & Health* 27, no. 1 (2003): 39–51.

15. Corrao et al., "A Meta-analysis of Alcohol Consumption"; K. Reynolds, L. B. Lewis, J. D. L. Nolen, G. L. Kinney, B. Sathya, and J. He, "Alcohol Consumption and the Risk of Stroke: A Meta-analysis," *Journal of the American Medical Association* 289, no. 5 (2003): 579–88.

16. B. Lopez, S. J. Schwartz, G. Prado, A. E. Campo, and H. Pantin, "Adolescent Neurological Development and Its Implications for Adolescent Substance Abuse Prevention," *Journal of Primary Prevention* 29 (2008): 5–35.

17. S. A. Brown, M. McGue, J. Maggs, J. Schulenberg, R. Hingson, S. Swartzwelder, C. Martin, T. Chung, S. F. Tapert, K. Sher, K. C. Winters, C. Lowman, and S. Murphy, "A Developmental Perspective on Alcohol and Youths 16 to 20 Years of Age," *Pediatrics* 121, no. S4 (2008): 290–310.

18. S. E. Tapert, L. Caldwell, and C. Burke, "Alcohol and the Adolescent Brain: Human Studies," *Alcohol Research & Health* 28, no. 4 (2004/2005): 205–12.

19. Lopez et al., "Adolescent Neurological Development"; B. Luna and J. A. Sweeney, "The Emergence of Collaborative Brain Function: fMRI Studies of the Development of Response Inhibition," *Annals of the New York Academy of Sciences* 1021 (June 2004): 296–309.

20. Ibid.

21. Brown et al., "Developmental Perspective on Alcohol and Youths"; Lopez et al., "Adolescent Neurological Development"; D. W. Zeigler, C. C. Wang, R. A. Yoast, B. D. Dickinson, M. A. McCaffree, C. B. Robinowitz, and M. L. Sterling, "The Neurocognitive Effects of Alcohol on Adolescents and College Students," *Preventive Medicine* 40 (2005): 23–32.

22. Luna and Sweeney, "Emergence of Collaborative Brain Function."

23. S. Hiller-Sturmhöfel and H. S. Swartzwelder, "Alcohol's Effects on the Adolescent Brain: What Can Be Learned from Animal Models," *Alcohol Research & Health* 28, no. 4 (2004/2005): 213–21; Lopez et al.,

"Adolescent Neurological Development"; Luna and Sweeney, "Emergence of Collaborative Brain Function"; E. F. Wagner, "Developmentally Informed Research on the Effectiveness of Clinical Trials: A Primer for Assessing How Developmental Issues May Influence Treatment Responses among Adolescents with Alcohol Use Problems," *Pediatrics* 121, no. S4 (2008): 337–47.

24. Zeigler et al., "Neurocognitive Effects of Alcohol."

25. Tapert et al., "Alcohol and the Adolescent Brain."

26. Luna and Sweeney, "Emergence of Collaborative Brain Function."

27. Brown et al., "Developmental Perspective on Alcohol and Youths"; Lopez et al., "Adolescent Neurological Development"; Luna and Sweeney, "Emergence of Collaborative Brain Function."

28. Lopez et al., "Adolescent Neurological Development."

29. Luna and Sweeney, "Emergence of Collaborative Brain Function."

30. Ibid.

31. Ibid.

32. Ibid.

33. Brown et al., "Developmental Perspective on Alcohol and Youths"; Lopez et al., "Adolescent Neurological Development"; C. O'Brien, "Brain Development as a Vulnerability Factor in the Etiology of Substance Abuse and Addiction," *Adolescent Psychopathology and the Developing Brain: Integrating Brain and Prevention Science,* ed. D. Romer and E. F. Walker (New York: Oxford University Press, 2007), 388–98; Tapert et al., "Alcohol and the Adolescent Brain"; Zeigler et al., "Neurocognitive Effects of Alcohol."

34. Zeigler et al., "Neurocognitive Effects of Alcohol."

35. Brown et al., "Developmental Perspective on Alcohol and Youths"; Lopez et al., "Adolescent Neurological Development"; Hiller-Sturmhöfel and Swartzwelder, "Alcohol's Effects on the Adolescent Brain"; Wagner, "Developmentally Informed Research"; Zeigler et al., "Neurocognitive Effects of Alcohol."

36. Wagner, "Developmentally Informed Research."

37. O'Brien, "Brain Development as a Vulnerability Factor."

38. Brown et al., "Developmental Perspective on Alcohol and Youths."

39. Hiller-Sturmhöfel and Swartzwelder, "Alcohol's Effects on the Adolescent Brain"; M. D. Wood and K. J. Sher, "Risks of Alcohol Consumption in Laboratory Studies Involving Human Research Participants," *Psychology of Addictive Behaviors* 14, no. 4 (2000): 328–34.

40. O'Brien, "Brain Development as a Vulnerability Factor"; M. Windle, L. P. Spear, A. J. Fuligni, A. Angold, J. D. Brown, D. Pine, G. T. Smith, J. Giedd, and R. E. Dahl, "Transitions into Underage and Problem Drinking: Developmental Processes and Mechanisms between 10 and 15 Years of Age," *Pediatrics* 121, no. S (2008): 273–89.

41. Brown et al., "Developmental Perspective on Alcohol and Youths"; Hiller-Sturmhöfel and Swartzwelder, "Alcohol's Effects on the Adolescent Brain"; O'Brien, "Brain Development as a Vulnerability Factor."

42. Brown et al., "Developmental Perspective on Alcohol and Youths."

43. Ibid.

44. Zeigler et al., "Neurocognitive Effects of Alcohol."

45. M. D. DeBellis D. B. Clark, S. R. Beers, P. H. Soloff, A. M. Boring, J. Hall, A. Kersh, and M. S. Keshavan, "Hippocampal Volume in Adolescent-Onset Alcohol Use Disorders," *American Journal of Psychiatry* 157, no. 5 (2000): 737–44.

46. Brown et al., "Developmental Perspective on Alcohol and Youths"; Hiller-Sturmhöfel and Swartzwelder, "Alcohol's Effects on the Adolescent Brain"; O'Brien, "Brain Development as a Vulnerability Factor."

47. Windle et al., "Transitions into Underage and Problem Drinking."

48. D. Behar, C. J. Berg, J. L. Rapoport, and W. Nelson, "Behavioral and Physiological Effects of Ethanol in High-Risk and Control Children: A Pilot Study," *Alcoholism: Clinical and Experimental Research* 7, no. 4 (1983): 404–10.

49. Ibid.

50. Ibid.

51. Lopez et al., "Adolescent Neurological Development"; Zeigler et al., "Neurocognitive Effects of Alcohol."

52. Lopez et al., "Adolescent Neurological Development."

53. Zeigler et al., "Neurocognitive Effects of Alcohol."

54. Ibid.

55. Lopez et al., "Adolescent Neurological Development"; Zeigler et al., "Neurocognitive Effects of Alcohol."

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