Physiology & Behavior 148 (2015) 122–130

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

Review

Adolescent alcohol exposure: Are there separable vulnerable periods within adolescence?

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HIGHLIGHTS

- Consequences of early vs. late adolescent alcohol exposure may differ.
- Timing-specific effects of adolescent insults may extend to other drugs/stressors.
- Brain ontogeny imparts changing landscape of possible timing-related vulnerabilities.

ARTICLE INFO

Article history:
Received 3 September 2014
Received in revised form 20 January 2015
Accepted 22 January 2015
Available online 23 January 2015

Keywords:
Adolescence
Ethanol
Nicotine
Lasting effects
Stressors
Neurobehavioral consequences
Puberty
Timing-specific effects

ABSTRACT

There are two key alcohol use patterns among human adolescents that confer increased vulnerability for later alcohol abuse/dependence, along with neurocognitive alterations: (a) early initiation of use during adolescence, and (b) high rates of binge drinking that are particularly prevalent late in adolescence. The central thesis of this review is that lasting neurobehavioral outcomes of these two adolescent exposure patterns may differ. Although it is difficult to disentangle consequences of early use from later binge drinking in human studies given the substantial overlap between groups, these two types of problematic adolescent use are differentially heritable and hence separable to some extent. Although few studies using animal models have manipulated alcohol exposure age, those studies that have have typically observed timing-specific exposure effects, with more marked (or at least different patterns of) lasting consequences evident after exposures during early-mid adolescence than late-adolescence/emerging adulthood, and effects often restricted to male rats in those few instances where sex differences have been explored. As one example, adult male rats exposed to ethanol during early-mid adolescence (postnatal days [P] 25–45) were found to be socially anxious and to retain adolescent-typical ethanol-induced social facilitation into adulthood, effects that were not evident after exposure during late-adolescence/emerging adulthood (P45–65); exposure at the later interval, however, induced lasting tolerance to ethanol's social inhibitory effects that was not evident after exposure early in adolescence. Females, in contrast, were little influenced by ethanol exposure at either interval. Exposure timing effects have likewise been reported following social isolation as well as after repeated exposure to other drugs such as nicotine (and cannabinoids), with effects often, although not always, more pronounced in males where studied. Consistent with these timing-specific exposure effects, notable maturational changes in brain have been observed from early to late adolescence that could provide differential neural substrates for exposure timing-related consequences, with for instance exposure during early adolescence perhaps more likely to impact later self-administration and social/affective behaviors, whereas exposures later in adolescence may be more likely to influence cognitive tasks whose neural substrates (such as the prefrontal cortex [PFC]) are still undergoing maturation at that time. More work is needed, however to characterize timing-specific effects of adolescent ethanol exposures and their sex dependency, determine their neural substrates, and assess their comparability to and interactions with adolescent exposure to other drugs and stressors. Such information could prove critical for informing intervention/prevention strategies regarding the potential efficacy of efforts directed toward delaying onset of alcohol use versus toward reducing high levels of use and risks associated with that use later in adolescence.

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http://dx.doi.org/10.1016/j.physbeh.2015.01.027
0031-9384/

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1. Introduction

Research assessing potential long-term consequences of adolescent alcohol exposure has begun relatively recently in both humans and laboratory animals, driven in part by the increasing recognition of developmental transformations in the brain during adolescence that could provide a period of vulnerability to lasting effects of alcohol exposure on later neuropsychological function and abuse propensity. Two key alcohol use patterns among human adolescents that are thought to confer vulnerability for later alcohol abuse/dependence are: (a) early initiation of use, and (b) high rates of binge drinking during late adolescence (e.g., high school and college age individuals) (e.g., see [89], for review). The central thesis of this mini-review is that neurobehavioral consequences of these two adolescent exposure patterns may differ, based in part on evidence of notable differences in the developmental alterations occurring in the brain across the broad span of adolescence, as well as emerging data in laboratory animals showing notable dissimilarities in the consequences of alcohol/drug exposures early versus later in adolescence. Data to be reviewed include studies examining adolescent-timing relevant consequences following exposure to alcohol as well as nicotine, cannabinoids and stressors, followed by a brief discussion of timing-related brain changes within adolescence.

2. Human studies

Alcohol use typically is initiated during adolescence, with alcohol use becoming normative in the United States by about 15 years of age (e.g., [38]). Monitoring the Future data have shown that >25% of 8th grades (~13 years.), 50% of 10th graders, and ~2/3rds of 12th graders used alcohol in the past year. Some of this use is extensive, particularly among older individuals within this group, with 25% of 12th graders reporting having been drunk within the past month, and 22% reporting that they had engaged in binge drinking (consumption of 5 + drinks on a given occasion) within the past two weeks, with a majority of the latter group reporting that they had done so on multiple occasions [47]. Prevalence rates for alcohol dependence are highest among 18–20 year old individuals, followed by 21–24 year olds, with rates declining into adulthood ([79]; cited by [25]).

2.1. Early exposure effect

In 1997, Grant and Dawson [23] reported that rates of alcohol dependence among those individuals that began drinking prior to the age of 14 were over 4 fold greater than those who did not initiate alcohol use until after 20 years of age. Hazards associated with age of first alcohol use (defined as the age when an individual begins to drink, excluding small tastes or sips of alcohol) are non-linear, with markedly elevated risks for the development of alcohol-related problems between 11 and 14 years that peak at 11–12 years. For instance, DeWit and colleagues [16] report eventual diagnoses of dependence in 15.9% of those first using alcohol at 11–12 years, dropping to 9% of those beginning use at 13–14 years, and declining rapidly there thereafter to reach an incidence rate of 1% among individuals first using alcohol when they were 19 or older [16].

Although age of first alcohol is a reliable and robust risk factor for later alcohol abuse and dependence (e.g., [56,92]), factors contributing to this effect are less clear. One possibility is that this use increases the probability of interacting with environmental factors (e.g., older alcohol-using peers) that favor escalation of use, although it is not obvious why this effect would be restricted to pre-/early-adolescence. It is also possible that early use merely represents a marker for some other problem (e.g., externalizing disorders) or neural vulnerability that elevates the probability of later abuse/dependence, although if this were the case, it would be expected that risk might be elevated at even younger ages (<11 years) rather than peaking at 11–12 years. It is also possible that there is a direct causal relationship, with early use altering normal brain development that is occurring at that time to increase the probability of later abuse/dependence (e.g., see [29]). Genetic contributors may vary with age to enhance vulnerabilities for alcohol use disorders, with perhaps pre-/early-adolescence representing a window of vulnerability to consequences of alcohol consumption on neurophysiological function (e.g., [12]).

2.2. Adolescent binge drinking characteristic of later adolescent period

Adolescents, particularly older adolescents, typically consume more alcohol per occasion than adults [38]. These elevated consumption levels develop over the course of adolescence, as does the emergence