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ADOLESCENTS AND ALCOHOL

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Abstract

The high levels of alcohol consumption characteristic of adolescence may be in part biologically based, given that elevated consumption levels are also evident during this developmental transition in other mammalian species as well. Studies conducted using a simple animal model of adolescence in the rat has shown adolescents to be more sensitive than adults to social facilitatory and rewarding effects of alcohol, but less sensitive to numerous alcohol effects that may serve as cues to limit intake. These age-specific alcohol sensitivities appear related to differential rates of development of neural systems underlying different alcohol effects as well as to an ontogenetic decline in rapid brain compensations to alcohol, termed “acute tolerance”. In contrast, these adolescent-typical sensitivities to alcohol do not appear to be notably influenced by pubertally-related increases in gonadal hormones. Although data are sparse, there are hints that similar alcohol sensitivities may also be seen in human adolescents, with this developmentally decreased sensitivity to alcohol’s intoxicating effects possibly exacerbated by genetic vulnerabilities also characterized by an insensitivity to alcohol intoxication, thereby perhaps permitting especially high levels of alcohol consumption among vulnerable youth.

Keywords

Adolescent; alcohol; puberty; intoxication; rodent model

Alcohol use becomes normative during adolescence and reaches high levels in some youth. Among 12th graders, over 1 in 4 report binge use of alcohol (defined as ≥ 5 drinks consumed in a drinking episode) within the past 2 weeks (Johnston, O’Malley, Bachman & Schulenberg, 2007). In a recent field study conducted in a college bar area, alcohol consumption levels of young underage and legal drinkers averaged 8 standard drinks, overall mean breathalyzer levels were above the cut-off for drunk driving (>80 mg%), and 75% of the individuals reported scored above the standard cut-off for risky alcohol consumption on the Alcohol Use Disorders Identification Test (AUDIT) (Celio, Vetter-O’Hagen, Lisman, Johansen & Spear, 2011). Indeed, on average adolescents drink more than twice as much per drinking occasion than adults (SAMHSA, 2006). Consequences of adolescent alcohol use can be devastating, not only from immediate effects of intoxication such as alcohol-related automobile accidents and other injuries (e.g., see Alcohol Research and Health, 2005), but also in terms of potential longer-lasting effects. Early use of alcohol is associated with an

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increased probability of later alcohol/drug dependence (Grant & Dawson, 1998), and amount of use has been correlated with certain neural and neuropsychological measures, although causality cannot be conclusively determined from such correlational analyses (see Clark, Thatcher & Tapert, 2008, for discussion).

Despite the importance of determining contributing factors to and consequences of alcohol use among adolescents, ethical concerns against administering alcohol to underage individuals have constrained empirical studies investigating these issues in youth and have led some researchers to study these issues using simple animal models of adolescence. When defined as the transition from a state of immaturity and parental dependence to that of relative independency and sexual maturity, adolescence is a developmental transition widely identifiable across mammalian species. During this transition, similar biological changes are seen across mammalian species, including rises in sex-related hormones and other physiological transformations of puberty, alterations in release of other hormones, a growth spurt, and notable transformations in the brain. Maturation extends through adolescence in frontal regions (such as the prefrontal cortex) that are important for cognitive control (e.g., Casey & Jones, 2010), with these developmental immaturities sometimes proving seemingly insufficient to countermand activity in subcortical emtion- and reward-relevant regions (such as the amygdala and nucleus accumbens) that may be especially reactive to socially arousing and emotional circumstances during adolescence (see Spear, 2011a).

Along with these neural transformations are a variety of behavioral proclivities that are seen across a variety of mammalian species, including an increasing focus on and time spent interacting socially with peers, and increases in novelty-seeking, risk-taking, sensation-seeking and related behaviors (see Spear, 2010). Likewise, the propensity for enhanced alcohol consumption during adolescence is seen not only in humans, but also in other mammalian species as well (e.g., Doremus, Brunell, Pottayil & Spear, 2005), thereby suggesting that this propensity may in part have evolutionarily conserved, neurobiological roots and supporting the use of simple mammalian (e.g., rodent) models to study alcohol and other drugs in adolescence. Of course, the use of alcohol and other drugs among adolescents is determined by multiple factors only some of which are amenable to study in laboratory animals. Hence, the appropriateness, validity and applicability of the animal model needs to be carefully considered, a point to which we return after reviewing alcohol studies using a simple animal model of adolescence in the rat.

Adolescent Alcohol Sensitivity

Like their human counterparts, adolescent rats typically voluntarily consume 2–3 times more alcohol than do adults (e.g., Doremus et al., 2005). Adolescent rats not only consume more alcohol, but differ in their sensitivity to various acute effects of alcohol when compared with adults. As summarized in Table 1, the nature of this developmental difference varies with the alcohol effect examined. For a few restricted effects of alcohol, adolescent rats are more sensitive than adults. These findings include data from the Swartzwelder group that adolescent are more vulnerable than adult rats to alcohol-induced disruptions in memory and in brain plasticity (indexed via long-term potentiation – i.e., an increase in transmission of signals between neurons after prior stimulation of those neurons)(see White &

Swartzwelder, 2005, for review). There is also emerging evidence that adolescent rats may be more sensitive than adults to the rewarding effects of alcohol (Pautassi, Myers, Spear, Molina & Spear, 2008; Ristuccia & Spear, 2008). In basic animal studies, adolescents have also been shown to be uniquely sensitive to alcohol-induced social facilitation. Under familiar, low stress circumstances, social behavior is stimulated by alcohol in adolescent but not adult rats (Varlinskaya & Spear, 2002). This alcohol-induced social facilitation in adolescent rats is specific to social behavior and not evident in terms of overall activity, and can be seen both with adolescent-typical (play fighting) and adult-typical (social investigation – i.e., body sniffing) social behaviors.

In marked contrast to these few enhanced alcohol sensitivities, adolescents have been shown in animal studies to be notably less sensitive than adults to a variety of other alcohol effects – many of which seemingly serve as cues to help regulate intake. Effects of alcohol for which adolescent rats are relatively resistant include the suppression of social behavior that begins to emerge at moderate doses of alcohol (Varlinskaya & Spear, 2002), as well as alcohol-induced motor impairment (Silveri & Spear, 2001), disruption of locomotion (Little, Adams & Cicero, 1996) and sedation (Silveri & Spear, 1998). Using conditioned taste aversions, adolescents were likewise found to be more resistant to the aversive effects of alcohol, with adolescent rats requiring more alcohol than adults to develop an aversion to a novel taste paired with alcohol (Vetter-O'Hagen, Varlinskaya & Spear, 2009). Animal studies have also shown adolescent rats to be less likely to display certain "hangover" symptoms during the post-intoxication recovery period than adults, including hangover-related increases in anxiety (Varlinskaya & Spear, 2004).

At least some of these adolescent-typical sensitivities may extend beyond alcohol. For instance, relative to adults, adolescents are not only more sensitive to the rewarding and less sensitive to the aversive properties of alcohol, but also to other drugs including nicotine (Wilmouth & Spear, 2004; Shram, Funk, Li & Le, 2006), cocaine and amphetamine (Infurna & Spear, 1979; Brenthouse & Andersen, 2008), as well as to even appetitive and aversive taste stimuli (Wilmouth & Spear, 2009). Such adolescent-typical reward/aversion biases may reflect very basic reward and motivational systems undergoing transformation during adolescence (e.g., see Spear, 2011a, for review).

Contributors to Adolescent-typical Alcohol Sensitivities

One potential explanation for age-related alterations in alcohol sensitivity could be developmental changes in how rapidly alcohol gets into and out of the system, although this possibility seems unlikely to be able to explain both the attenuated and accentuated sensitivities that adolescents show to different effects of alcohol. Adolescents do tend to have higher metabolic rates than adults, and age differences in alcohol levels are occasionally observed, although such differences are generally insufficient to account for the altered alcohol sensitivity of adolescents. For instance, adolescent rats given a sedative dose of alcohol not only recover in about half the time as do adults given the same dose, but they recover at higher brain levels of alcohol, suggesting that their brains are more resistant to the sedative properties of alcohol (Silveri & Spear, 1998). Findings such as these support the

conclusion that adolescent-typical alcohol sensitivities are not simply a function of pharmacokinetic differences, but rather reflect age differences in brain sensitivity to alcohol.

Studies have begun to explore neural contributors to age differences in brain sensitivity to alcohol. Unlike most other drugs, alcohol acts widely throughout the brain and interacts with multiple neurotransmitter systems, including the major inhibitory and excitatory neurotransmitters and their receptor systems: gamma aminobutyric acid [GABA] (and $GABA_A$ and $GABA_B$ receptors) and glutamate (and n-methyl-d-aspartate NMDA] receptors), respectively. Adolescent-typical alcohol sensitivities appear to be related in part to ontogenetic changes that occur through adolescence in these and other alcohol-sensitive neurotransmitter systems, such as the opiate and dopaminergic systems. For example, facilitatory effects of alcohol during adolescence appear related in part to mu opiate receptors, given that mu-receptor-specific opiate antagonists block this adolescent-specific effect (Varlinskaya & Spear, 2009). Unusually potent inhibition by alcohol of developmentally over-expressed NMDA receptors has been suggested to contribute to the greater sensitivity to adolescents to alcohol-induced disruptions in long-term-potentiation (see White and Swartzwelder, 2005, for review), whereas attenuated sedative effects of alcohol appear related in part to delayed development of the GABA system (Silveri & Spear, 2002).

Attenuated alcohol sensitivities of adolescents also appear related in part to the very rapid onset of compensatory processes in their brains that serve to partially counter alcohol's effects – a phenomenon termed "acute tolerance" (see Silveri & Spear, 2001, for further discussion and references). This rapid decline in alcohol sensitivity occurs within a single alcohol exposure and can be indexed various ways, all generally based on the expression of less impairment at later points than earlier in the intoxication period relative to the amount of alcohol that is present at those times. When tested using measures such as alcohol-induced sedation (Silveri & Spear, 1998, 2002) and social inhibition (Varlinskaya & Spear, 2006), adolescent rats often exhibit notable acute tolerance under test conditions that do not support expression of acute tolerance in adults. This rapid adaptation, however, does not appear to be solely responsive for the attenuated alcohol sensitivities of adolescents, given that disruption expression of acute tolerance in adolescents by blocking NMDA receptors does not eliminate age differences in alcohol sensitivity (Silveri & Spear, 2002; Silveri & Spear, 2004).

Potentially important contributors to adolescent-typical alcohol sensitivities are changes related to puberty. Some neural transformations of adolescence may be pubertally-dependent and driven by rising gonadal hormone levels, whereas other changes, particularly in hypothalamic regions and associated forebrain regulatory areas, may predate puberty and help precipitate pubertal processes. Still other alterations may be associated with maturational changes unrelated to puberty. For instance, consider developmental changes during adolescence in dopamine (DA), a neurotransmitter system through to be critical for processing of rewarding stimuli. There is evidence for both gonadal hormone-dependent and -independent development within the DA system, with pre-pubertal gonadectomy, for instance, blocking post-adolescent declines in DA release from DA cell bodies in the

substantial nigra (Knoll et al, 2000), while not affecting developmental declines in density of D1 and D2 subtypes of the DA receptor in striatum (Andersen et al, 2002).

One challenge when attempting to parse pubertally-dependent versus independent processes in studies with humans is that, in contrast to the many biomarkers of pubertal status, there is no good proxy of non-pubertal-related maturational state. Age, though typically used, at best likely is only marginally effective, given well-known differences in developmental rate that likely impact the relative maturational rate of both pubertal and non-pubertal-related processes. As research identifies aspects of cognition and behavior unrelated to pubertal status, it is possible that maturational changes in these measures (or neural alterations correlated with these functional changes) could perhaps be used to index general, non-pubertally-related maturational rate.

The role of pubertal hormones in adolescent-typical neurobehavioral function can be systematically examined in laboratory animals by examining consequences of pre-pubertal removal of the gonads. Work using this approach has found that animals that do not experience normal developmental increases in pubertal hormones exhibit deficits in later expression of a variety of reproductive behaviors and other adult-typical, sexually dimorphic behaviors (see Sisk & Zehr, 2005, for review). In contrast, we have found pubertal hormones to exert surprisingly modest influences on alcohol intake and sensitivity (Vetter-O'Hagen & Spear, 2011, 2012). For instance, gonadectomy in males (but not females) either prepubertally or in adulthood increased alcohol intake in adulthood, an increase largely reversed by testosterone replacement (Vetter-O'Hagen & Spear, 2011; Vetter-O'Hagen, Sanders & Spear, 2012); this pattern of findings is consistent with an activational role of testosterone in adulthood rather than pubertally-related processes. Moreover, neither pre-pubertal or adult gonadectomy altered sensitivity to alcohol's social inhibitory effects, although the microstructure of social behavior was affected in both males and females (Vetter-O'Hagen & Spear, 2012). Studies such as these support the suggestion that adolescent-typical alcohol sensitivities are not notably dependent on pubertally-related processes.

Relevance to Human Adolescent Alcohol Use and Emergence of Alcohol Use Disorders?

Although available data are limited, studies conducted in human adolescents hint of comparable age-related differences to those seen in laboratory animals. For instance, reminiscent of the alcohol-induced social facilitation seen in adolescent rats, social interactions are also of particular importance for facilitating drinking among youth (e.g., Kelly, Chan, Toumbourou, O'Flaherty, Homel, Patton & Williams, 2012). Likewise, late adolescent individuals (in their early twenties) were found to be more vulnerable to alcohol-induced disruptions in memory than those in their late twenties (Acheson, Stein & Swartzwelder, 1998). Conversely, in terms of intoxicating effects of alcohol that may serve as cues to moderate intake, insensitivities similar to those seen in adolescent rats were reported in an older study of 8–15 year old boys given a battery of laboratory tests of intoxication, with the researchers noting that they "were impressed by how little gross behavioral change occurred in the children…after a dose of alcohol which had been

intoxicating in an adult population" (Behar, Berg, Rapoport, Nelson, Linnoila, Cohen, Bozevich & Marshall, 1983, p.407).

Any adolescent insensitivity to intoxicating effects that normally help curb intake could contribute to the greater per episode drinking seen among adolescents. Indeed, decreased sensitivity to alcohol's intoxicating, aversive and sedative effects has been shown in genetic studies in humans (e.g., Schuckit, 1994) and mice (Green & Grahame, 2008) to be a major risk factor for problematic alcohol use and enhanced alcohol intake. Thus, normal insensitivities to alcohol intoxication during adolescence may be exacerbated among genetically vulnerable individuals to encourage especially high levels of alcohol consumption, thereby elevating exposure of the still-developing brain of the adolescent to alcohol, and potentially precipitating a variety of long-term consequences (see Spear, 2011b, for review).

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TABLE 1

Adolescent-typical alcohol sensitivities seen in laboratory animals.

Adolescent rats are MORE sensitive than adults to alcohol-induced:

- Impairment in brain plasticity & spatial memory (White & Swarzwelder, 2005)*
- Social Facilitation (Varlinskaya & Spear, 2002)
- Rewarding effects (Pautassi et al, 2008; Ristuccia & Spear, 2008)

Adolescent rats are LESS sensitive than adults to alcohol-induced:

- Aversive Effects (Vetter-O'Hagen et al, 2009)
- Social Inhibition (Varlinskaya & Spear, 2002)
- Sedation (Silveri & Spear, 1998)
- Motor impairment (Silveri & Spear, 2001)
- Disruption in locomotion (Little et al, 1996)
- Hangover effects (Varlinskaya & Spear, 2004)

*Sample references provided parenthetically – for additional references see the text, recommended readings, and the references cited in the articles listed in this Table.