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Developmental differences in the effects of alcohol and stress on heart rate variability

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HIGHLIGHTS

- Effects of stress and ethanol on heart rate variability during ontogeny were assessed.
- A possible ethanol-induced sympathetic deactivation in adolescents was noted.
- Ethanol, but not stressor, effects on sympathovagal balance were seen in adolescents.
- Ethanol-induced autonomic effects were more pronounced in adults following stress.
- Effects of prior stress on hormone levels were less in adolescents than adults.

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ABSTRACT

Adolescent rats differ in their responses to stress and ethanol from their adult counterparts, although not much is known about the contribution of the autonomic nervous system (ANS) to these differences. This study assessed the impact of stress, ethanol, and their combination on parameters of heart rate variability (HRV) in adolescent and adult male Sprague–Dawley rats. Animals were habituated to the testing box and neck sensors (MouseOX, STARR Life Sciences Corp.) used for recording heart rate (HR). After 8–10 min of baseline recording, animals were restrained for 90 min or returned home, followed by intraperitoneal injection of 0, 0.5, 1.0, or 1.5 g/kg ethanol. The 8–10 min test recording occurred 30 min post-injection. Ethanol-related decreases in LF (an index of sympathetic activity) were evident under non-stressed conditions in adolescents but only after stress in adults, perhaps in part due to apparent ethanol-induced sympathetic deactivation in adolescents. Parasympathetic tone, indexed by HF, was unaffected by both ethanol and stress in adolescents, while again both the 1.0 and 1.5 g/kg ethanol doses decreased HF in adults following stress. Ethanol also decreased low frequency/high frequency tone (LF/HF), an index of sympathovagal balance, only in adolescents, with no decrease evident in adults. Further, stressed adults, and not adolescents, had significantly lower CORT and PROG values than their non-stressed counterparts. Taken together, these results demonstrate notable age differences in the ANS response to ethanol under stressful vs. non-stressful circumstances, reflected by ethanol-mediated autonomic effects that were more pronounced following stressor exposure in adults but under non-stressed conditions in adolescents.

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1. Introduction

Adolescents often differ from adults in their behavior, including enhanced novelty seeking and impulsivity [5], as well as greater per occasion alcohol consumption (see [61,62] for a review). However, little is yet known regarding the mechanisms underlying these unique age-dependent responses. Studies using rat models have shown adolescents to be relatively insensitive to many ethanol effects when compared to adults, especially those consequences of ethanol that presumably serve as cues to limit intake (for review, see [12,28]). For instance, adolescent rats are less sensitive than adults to ethanol-induced dysphoria (indexed by conditioned taste aversions; [2,55]), sedation [58], and motor impairment [85], as well as the social impairment that emerges at moderate or higher doses of ethanol [75]. Adolescents may also exhibit an attenuated sensitivity to certain post-intoxification effects, with this age group failing to demonstrate the anxiogenic effect of acute withdrawal from a high dose of ethanol that is seen in adults [15]. Adolescents are not less sensitive than adults, however, to all ethanol effects. Relative to adults, adolescents exhibit greater sensitivity to the social facilitating effects induced by low doses of ethanol [76] as well as to alcohol-induced impairments in memory and brain plasticity [68,69].

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Responsiveness to stressors also differs developmentally, and may contribute to the propensity of adolescents to exhibit certain age-typical behaviors. For instance, human developmental studies have shown that the transition into adolescence is marked by alterations in the HPA axis, with greater basal and stress-induced activity of the HPA axis (indexed via the glucocorticoid cortisol) seen after stressors than in adults [29,84]. The autonomic nervous system (ANS) is another intriguing possible contributor to age-related differences in responsivity to stressors, given that activation patterns within the somatic system have been shown to be used as feedback cues for self-attribution of affect (see [17] for a review). Generally speaking, relative levels of functioning of both the sympathetic and parasympathetic branches of the ANS are thought to reflect a stable individual characteristic, although recent findings have shown some malleability and ontogenetic changes during childhood and adolescence [1,31,54,80]. Indeed, ANS reactivity may not be stable during the transition from adolescence to adulthood, given evidence for a developmental switch from parasympathetic to sympathetic dominance that occurs during adolescence [33,49]. Utilizing a rodent model to examine autonomic responding to an aversive auditory stimulus during ontogeny, Kurtz and Campbell [39] found evidence that the ANS response to this stimulus was characterized by parasympathetic withdrawal and sympathetic activation in adolescents versus adults, respectively. Similar results have been seen in developing youth, suggesting that ANS reactivity may be more plastic to environmental manipulations throughout ontogeny, whereas tonic ANS functioning remains a relatively stable characteristic [13,16,19]. Yet despite increases in ANS emotional reactivity during adolescence, there is some evidence that the ability to link these physiological reactions to perceived emotions may not be as developed in adolescence as adulthood (see [29,65]). The ANS is of particular interest developmentally in that its activation patterns are influenced by multiple brain systems, many of which are undergoing developmental change during adolescence ([8]; also see [11] for a review). Collectively, these findings lead to the question of whether developmental differences in activation of the ANS could contribute to age-related differences between adolescents and adults in sensitivity to stressors and alcohol.

The current study compared the ANS response patterns of adolescents and adults in response to alcohol and stress, as indexed via heart rate variability (HRV), a measure often used to index relative ANS sympathetic and parasympathetic responding. HRV is used to describe the cyclical variations in both instantaneous heart rate and beat-to-beat (RR) intervals, information that is reflected in the standard deviation of the RR (beat-to-beat) intervals (SDNN) that represents that total variability in the HR. HRV is also influenced by relative activity of the sympathetic and parasympathetic (vagal) components of the ANS on the sinus node of the heart, components that can be partially separated through spectral density analysis (see [42] for a review). The high frequency (HF) component of the spectrum arises primarily from contributions from the parasympathetic component of the ANS, whereas attribution of the low frequency (LF) component is more controversial. Some research supports the suggestion that the LF is a general marker of sympathetic excitation [43], whereas other work has concluded that the LF component reflects activity in both the sympathetic and the parasympathetic/vagal systems [34]. The ratio of LF to HF components, often referred to as sympathovagal balance, in turn, examines proportional distribution of power between the LF and HF components. This study assessed the effects of an acute stressor and ethanol alone or in combination on measures of HRV in adolescent and adult rats.

2. Methods

2.1. Subjects

A total of 195 Sprague–Dawley male rats bred and reared in our colony at Binghamton University were used in this study. The day after birth, all litters were culled to 8–10 pups and housed with their mother until weaning on postnatal day (P) 21, at which time animals were pair-housed with same-sex littermates. Animals were maintained in a temperature controlled (20–22 °C) vivarium on a 12-/12-h light/dark cycle (lights on at 0700) with ad libitum access to food (Purina Rat Chow, Lowell, MA) and tap water. All procedures were conducted in accord with guidelines established by the National Institutes of Health using protocols approved by the Binghamton University Institutional Animal Care and use Committee.

2.2. Experimental design and animal assignment

The design of this study was a 2 age (adolescent, adult) × 4 dose (saline, 0.5 g/kg, 1.0 g/kg, and 1.5 g/kg ethanol) × 2 stress condition (stress, nonstress) factorial. An additional non-manipulated control group (NM) was included at each age that received the same habituation and test sessions, but was not stressed nor injected prior to test. Adolescents were tested at P32–33 whereas adults were tested at P72–74. Ethanol was administered interperitoneally (i.p.) as an 18.9% (v/v) solution in physiological saline, while control subjects were injected with 0.9% saline isovolumetric to the highest dose of ethanol administered. Animals in the stress condition received 90 min of acute restraint stress on test day, whereas the non-stress group did not. Animals were assigned to groups randomly, with the constraint that no more than one animal from any given litter was placed in a particular test group to avoid confounding litter with treatment effects [88]. Both littermates within each pair were either stressed or non-stressed, but were assigned to different dose/manipulation conditions.

2.3. Apparatuses

Testing was conducted in a Plexiglas® chamber (30.48 cm × 20.32 cm × 16.51 cm) with an adjustable plastic divider used to adjust the chamber size for adults (19.05 cm × 20.32 cm × 16.51 cm) and adolescents (11.43 cm × 20.32 cm × 16.51 cm). For restraint stress, different sized Tailveiner® restrainers (Braintree Scientific) were used at each age (RTV-190 for adults and RTV-180 for adolescents). Apparatuses were cleaned with 6% hydrogen peroxide and dried between animals. Data were collected using two MouseOx® recording collars, one for each age (size large for adolescents; extra-large for adults), and MouseOx® software (STARR Life Sciences, Alleghany, PA). The MouseOx® collected and recorded heart rate data (pulse strength, duration and frequency). Spectral analyses of these data were performed using Lab Chart, 7.3 (AD Instruments, Colorado Springs, CO), which converted the signal into a series of interbeat (RR) intervals and computed the frequency spectrum of this tachogram using Fast Fourier Transforms. The power contained within specific frequency bands (i.e. HF or LF) was computed by Lab Chart by integrating the periodogram over the desired bands.

2.4. Procedure

All testing was conducted between 0900 and 1600. Beginning on either P30–31 for the adolescent age group or P70–72 for the adults, each animal was separately habituated for 30 min to the testing container. On the second day, each animal was again placed in the testing container for 30 min, this time while also wearing the testing collar. During this second habituation period, the experimenter remained in the room with the animal to ensure the safety of the animal, collar integrity and placement, as well as to habituate the animal to the presence of the experimenter in the room. On day 3, testing occurred, with the experimenter again remaining in the room throughout the test session. On test day, a recording collar was again placed on the test animals and it was placed in the apparatus for ~15 min for collection of baseline HR data. The collar was then removed and the animal was placed in a restraint tube in a nearby room for 90 min or returned to its home cage and littermate. Immediately after this 90-minute period, all but NM

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