Psychoneuroendocrinology 100 (2019) 137–144

Contents lists available at ScienceDirect

Psychoneuroendocrinology

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

Binge drinking in adolescence predicts an atypical cortisol stress response in young adulthood

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ARTICLE INFO

Keywords:
Cortisol
Adolescence
Young adulthood
Alcohol
Binge drinking

ABSTRACT

Adolescence is a sensitive developmental period in which substance use can exert long-term effects on important biological systems. Emerging cross-sectional research indicates that problematic alcohol consumption may be associated with dysregulated neuroendocrine system functioning. The current study evaluated the prospective effects of binge drinking in adolescence on cortisol stress reactivity in young adulthood among individuals who had experienced parental divorce in childhood (N = 160; Mean age = 25.55, SD = 1.22; 46.9% Female; 88.8% White Non-Hispanic). Youth completed validated measures of problematic drinking during adolescence (aged 15–19) and participated in a standardized social stress task nine years later in young adulthood. Latent growth modeling was conducted within a structural equation modeling framework. Greater binge drinking during adolescence was associated with a significantly lower cortisol stress response in young adulthood, controlling for young adult drinking, sex, childhood externalizing problems, and socioeconomic status. Findings suggest problematic alcohol consumption during mid-to-late adolescence may have important effects on the neuroendocrine stress response system at subsequent developmental stages.

1. Introduction

Alcohol is the most commonly abused substance among youth (Johnston et al., 2018). The rate of binge drinking (defined as the consumption of at least 5 drinks for males and 4 drinks for females in a 2-hour period) is alarmingly high in adolescence, with as many as 18% of youth reporting at least one binge drinking episode in a 30-day period (Kann et al., 2015). For adolescents who experience family disruption, such as parental divorce, the rate of problematic alcohol consumption is even higher (Barrett and Turner, 2006; Pilowsky et al., 2009). For example, parental divorce has been associated with an earlier age of onset of adolescent drinking (Jackson et al., 2016) and an increased risk for later alcohol abuse (Arkes, 2013). Adolescence is a developmental period of intense biological change. Problematic alcohol consumption during this sensitive period has been associated with abnormal brain development and emotion regulation deficits (Jones et al., 2016; Trantham-Davidson et al., 2017). The hypothalamic-pituitary-adrenal (HPA) axis is a prime mediator of the effects of alcohol on the body in the short-term and a potential pathway by which alcohol might exert long-term effects on biological systems; however, the lasting impact of problematic alcohol use on neuroendocrine functioning among youth has not been examined.

The HPA axis is the primary arm of the neuroendocrine stress system and is activated by both ascending (from the brainstem) and descending (from limbic structures) inputs (Herman et al., 2005). Superimposed on a diurnal rhythm, the stress-related activation of the HPA axis initiates a hormonal cascade that results in accelerated synthesis and secretion of cortisol. During stress, cortisol facilitates an increase in cardiovascular activity, alterations in cognitive and sensory thresholds, an increase in alertness, promotion of stress-induced analgesia, suppression of nonessential functions (e.g., growth, digestion, and reproduction), and the processing and consolidation of emotionally-laden memory (Ulrich-Lai and Herman, 2009). High levels of cortisol then trigger a negative feedback cycle in which the subsequent

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https://doi.org/10.1016/j.psyneuen.2018.10.002
Received 23 August 2018; Received in revised form 2 October 2018; Accepted 4 October 2018

release of hormones is inhibited, ultimately leading to a decrease in cortisol to basal levels and a return to a pre-stress state (Tsigos and Chrousos, 2002). Thus, a typical cortisol response to stress involves a period of reactivity (a rise in cortisol levels that are sustained for an appropriate amount of time to meet the demands of the situation) and a period of recovery (a decline in cortisol levels back to baseline). Dysregulation of this typical response is observed when cortisol reactivity continues when no longer needed or, conversely, is not of sufficient magnitude to meet the demands of the situation (McEwen, 2007).

Associations between alcohol consumption and cortisol activity are complex. In a non-stress context, consumption of alcohol has a stimulating effect on the HPA axis, resulting in an initial increase in cortisol output (Magrys et al., 2013). However, many experimental studies have shown that when alcohol is consumed immediately prior to or following a discrete psychosocial stressor it can have an attenuating effect among some individuals such that cortisol reactivity is much lower than expected or does not appear at all (Balodis et al., 2011; Dai et al., 2002; Schrieks et al., 2016).

Emerging cross-sectional literature documents associations between alcohol use outside of the experimental context and alterations to the expected cortisol stress response profile (e.g., Orio et al., 2017). Only one study to our knowledge has prospectively examined alcohol use and biological stress system functioning. In an examination of a community sample of youth, it was found that a flattened diurnal cortisol rhythm at age 11 predicted greater alcohol use between ages 15–18, and heavier alcohol use predicted further flattening of the diurnal rhythm six months later (Ruttle et al., 2015). No study to our knowledge has evaluated the association between excessive alcohol consumption at one developmental stage and cortisol reactivity to social stress at a subsequent developmental stage. This is a critical oversight given that problematic substance use earlier in life is likely to have pervasive and enduring consequences for central nervous system functioning. The long-term detrimental effects of alcohol consumption may be especially likely to occur when consumption takes place during adolescence – a period when the neurobiological stress system undergoes critical developmental alterations (Casey and Jones, 2010). Consistent with this idea, animal models show that alcohol exposure during this developmental period alters the neural circuitry underlying the activation of the stress response (specifically the functioning of the paraventricular nucleus) resulting in a blunted stress response later in life (Allen et al., 2011).

With one exception (e.g., Jones et al., 2013), cross-sectional studies with humans have shown an attenuation of the neuroendocrine response to stress (i.e., a decrease in levels across an acute stressor) among adults who report binge drinking or other forms of heavy alcohol use (Lovallo et al., 2000; Orio et al., 2017). Atypical patterns of cortisol reactivity, such as a blunted response, have been associated with a wide range of physical and mental health problems (for a review, see Phillips et al., 2013). For example, attenuated cortisol reactivity may have implications for the development of and recovery from substance use disorders (Back et al., 2010; Blaine and Sinha, 2017) and different forms of psychopathology (Petrowski et al., 2013; Scott et al., 2013). As such, the potential prospective effects of adolescent binge drinking on later neuroendocrine system functioning may have a number of consequences. Yet, little is known about the relation between problematic drinking in adolescence and stress reactivity in young adulthood, especially with regard to individuals who experienced parental divorce in childhood. To address this critical gap in the literature, the current study tested the hypothesis that greater binge drinking during adolescence (ages 15–19) would predict an attenuated cortisol response to social stress (i.e., lower cortisol reactivity) in young adulthood among individuals who experienced parental divorce in childhood, even after statistically adjusting for a range of covariates known to be associated with cortisol reactivity, including participant sex, smoking, family socioeconomic status, childhood externalizing problems, and binge drinking in young adulthood.

2. Materials and methods

2.1. Participants

Participants were a subsample of families who were part of a longitudinal study of divorced families that participated in a randomized trial of a prevention intervention. Participant recruitment and eligibility are described in detail by Wolchik and colleagues (Wolchik et al., 2000) and only briefly reviewed here. Potential participants were identified by reviewing randomly selected divorce decrees (divorced within 2 years prior to baseline assessment) of families with children between ages 9 and 12. Families were recruited through letters and telephone calls; 20% of the sample was recruited through supplemental methods (e.g., media, referrals). The primary eligibility criteria were: primary residential parent was female, neither child nor mother was in treatment for mental health problems, mother had not remarried, and custody arrangements were anticipated to be stable. Families were excluded and referred for treatment if the child scored above 17 on the Children's Depression Inventory or 97th percentile on the Externalizing subscale of the Child Behavior Checklist or endorsed suicidal ideation.

Although not the subject of the current study, the larger project included a randomized controlled trial of a preventive intervention, the New Beginnings Program, which was designed to reduce children's post-divorce mental health problems. The original trial included 240 families, a sample size selected so that small to medium effects of the program could be detected with power equal to or greater than 0.80. Of the original 240 offspring enrolled in the trial, 194 participated in the 15-year follow-up. The current study is based on participants in the 15-year follow-up who supplied salvia samples, across intervention group assignment. Of the 194 individuals in the 15-year follow-up, 12 did not participate in the stressor task or provide saliva samples, and two had a cortisol concentration that was outside normal physiological parameters (> 50 nmol/L; Nicolson et al., 2008), indicating assay interference. Of the remaining 180 participants, 20 were excluded a priori due to pregnancy or breast-feeding (n = 9), use of steroidal medications or chronic health conditions (n = 9), violation of protocol by smoking within 30 min of the first saliva sample (n = 1), or only one viable saliva sample (n = 1). Thus, the final sample included 160 offspring (53.1% male; 88.8% White Non-Hispanic) between ages 24 and 28 ($M = 25.55$, $SD = 1.22$). By the 15-year follow-up, 40.2% of young adults had completed at least some college education. Young adults' median pre-tax annual household income was $59,500.

2.2. Procedures

The current study comprises families who were randomized to participate in a literature control or an intervention (mother-only program and mother-plus child program version of a preventive intervention for divorced families) (Wolchik et al., 2013, 2000). Because neither intervention condition was shown to have direct effects on cortisol, intervention and control groups were combined and intervention condition was included as a covariate in all analyses. Because previous analyses reported an age x intervention effect on cortisol reactivity in this sample (Luecken et al., 2015), analyses were repeated with this interaction term. However, the interaction was not significant in relation to cortisol and model fit deteriorated, thus the more parsimonious model is presented here.

All procedures and measures were approved by the Arizona State University Institutional Review Board. Six waves of assessment were conducted: baseline, post-test, 3-months later, 6-months later, 6 years later and 15 years later. In the current study, only data from the baseline, 6-year and 15-year follow-up assessments were used. All assessments were conducted by trained staff in participants' homes. At each assessment, confidentiality was explained, and mothers signed consent forms; offspring younger than 18 signed assent forms and offspring 18 or older signed consent forms. Families received $45