SHORT COMMUNICATION

Longitudinal associations between diurnal cortisol slope and alcohol use across adolescence: A seven-year prospective study

Paula L. Ruttle [a,*], Julie Maslowsky [b,1], Jeffrey M. Armstrong [a], Linnea R. Burk [c], Marilyn J. Essex [a]

[a] Department of Psychiatry, School of Medicine and Public Health, University of Wisconsin—Madison, Madison, WI 53719-1176, USA
[b] Department of Population Health Sciences, School of Medicine and Public Health, University of Wisconsin, Madison, WI 53719-1176, USA
[c] Department of Psychology, University of Wisconsin, Madison, WI 53719-1176, USA

Summary   A large body of research has linked hypothalamic—pituitary—adrenal (HPA) axis function and alcohol consumption, including work suggesting that flatter diurnal cortisol slopes are associated with greater alcohol use. A lack of longitudinal studies and a focus on adult and alcoholic populations leaves unclear whether such associations are also present in younger, non-clinical populations and whether flatter diurnal slopes are a consequence of or preexisting risk factor for alcohol use; however, theory suggests such associations may be mutually reinforcing. In a longitudinal, community sample of 200 (55% female) adolescents, the current study demonstrates that flatter diurnal cortisol slope at age 11 predicts higher levels of alcohol use from ages 15—18, and that heavier alcohol use in turn predicts further flattening of diurnal cortisol rhythm at age 18.5. This is the first study to demonstrate a longitudinal chain of associations between diurnal cortisol slope and alcohol use. Findings support contemporary theoretical models of the neurobiological processes underlying alcohol use and can inform future research on risk factors for and consequences of underage drinking.
© 2015 Elsevier Ltd. All rights reserved.

* Corresponding author. Tel.: +1 608 263 7887.
  E-mail addresses: ruttle@wisc.edu, plruttle@gmail.com (P.L. Ruttle).
[1] Present address: Department of Kinesiology and Health Education, University of Texas, Austin, TX, USA.

1. Introduction

Although underage alcohol use is fairly common, it can have serious short- and long-term consequences. These include increased risk of accidental injury, risky sexual behavior, and lower educational attainment (U.S. Department of Health and Human Services, 2007; Masten et al., 2008), all of which increase the risk of morbidity and mortality. As such, it is essential to understand underlying neurobiological processes associated with underage drinking. Theory (Schepis et al., 2011) suggests that alcohol use and hypothalamic—pituitary—adrenal (HPA) axis activity are mutually reinforcing over time, but this has yet to be tested empirically, highlighting the need for longitudinal studies examining whether a longitudinal chain of associations links preadolescent cortisol functioning, alcohol use across adolescence, and later cortisol functioning.

As outlined by Schepis et al. (2011), different forms of HPA-axis dysfunction may act as risk factors for later alcohol use and abuse, and chronic alcohol consumption in turn may contribute to further disturbances in HPA-axis functioning (e.g., flattened diurnal cortisol slopes due to hypocortisolism). This theoretical work rests on existing preclinical (e.g., Allen et al., 2011) and human (e.g., Gianoulakis et al., 2003; Boschloo et al., 2011) studies. However, human research in this area has largely focused on cross-sectional associations in adults, often alcoholics, with cortisol response to acute stressors or to experimental administration of alcohol predominating (e.g., Adinoff et al., 2005). Nevertheless, basal cortisol levels and diurnal patterns have also been examined (e.g., Gianoulakis et al., 2003; Boschloo et al., 2011). Such work is integral to understanding health and behavior because deviations from the expected diurnal rhythm can reflect impaired integrity of day-to-day HPA-axis functioning, which is often indicative of more persistent health problems. Flatter diurnal cortisol slopes have been associated with greater alcohol consumption (Badrick et al., 2008), echoing links between flattened cortisol slopes and greater mental health symptoms in adolescence (Shirtcliff and Essex, 2008; Ruttle et al., 2011), but findings are inconsistent. For example, one cross-sectional study of adolescent alcohol-use onset found no association with diurnal cortisol (Evans et al., 2012), and one prospective study yielded null associations between morning, afternoon, and evening cortisol levels at ages 10—12 and alcohol use concurrently and at ages 13—14 (Huizink et al., 2009). However, both studies measured alcohol use very early in adolescence. Given that a variety of mental and physical health conditions (e.g., externalizing and internalizing symptoms, burnout, chronic fatigue) have been associated with hypocortisolism (Fries et al., 2005; Ruttle et al., 2011), it may be that more persistent drinking patterns, rather than acute drinking or early experimentation reflecting behavior at a single time point, are associated with flattened diurnal—cortisol patterns (Gianoulakis et al., 2003).

The present study takes an important first step to addressing the gaps in the literature by testing for a longitudinal chain of effects of HPA-axis function and alcohol consumption in a community sample of adolescents. We hypothesized that flatter diurnal cortisol slope at age 11 would relate to higher levels of alcohol use across ages 15—18, which would in turn relate to further flattening of diurnal cortisol slope at age 18.5. We also examined whether this latter association is present net of age 11 cortisol slope to ensure that the observed finding does not simply reflect developmental changes in HPA-axis function, such as increased basal cortisol levels across adolescence (Gunnar et al., 2009), which may result in the flattening of diurnal cortisol slope. Given prior work (Evans et al., 2012), we also considered whether other key concurrent factors linked to cortisol activity and/or adolescent drinking behavior (i.e., pubertal development, externalizing symptoms, and internalizing symptoms) might explain these associations.

2. Methods

2.1. Participants

Participants are drawn from a longitudinal study of 560 families recruited during mother's pregnancy and followed through the target child's adolescence (Hyde et al., 1995). The sample is primarily Caucasian (93%); initial annual family income ranged from $7500 to >$200,000 (median = $47,000). The current study focused on the sub-sample ($n = 200$) of participants who provided saliva samples at ages 11 and 18.5. Participants did not differ from non-participants on demographics, mental health symptoms, or alcohol use. All procedures were approved by University of Wisconsin Institutional Review Boards.

3. Measures

3.1. Alcohol use

At modal ages 15, 16, 17, and 18 — the spring of grades 9—12 — adolescents reported whether they had ever had an alcoholic drink and, if so, the typical number of drinks consumed per occasion in the past 30 days as in major studies (e.g., Johnston et al., 2012). Responses were scored as 0 (I don't drink alcohol), 0.5 (Less than a drink), 1 (1 drink), 2 (2 drinks), 3 (3 drinks) and 5 (5 drinks). At each grade, number of drinks consumed per occasion ranged from 0 to 5: grade 9 $M = 0.84$, $SD = 1.34$; grade 10 $M = 1.29$, $SD = 1.68$; grade 11 $M = 1.62$, $SD = 1.74$; grade 12 $M = 1.97$, $SD = 1.86$. Alcohol use was averaged over time to create a summary measure of drinking habits across high school.

3.2. Salivary cortisol

Diurnal cortisol was assessed in the summers following grade 7 (modal age 11) and grade 12 (modal age 18.5, approximately 6 months after the last alcohol use measure). Saliva was collected three times per day for three days at set target times: shortly after waking, between 3:00 and 7:00 PM, and before bed. Adolescents were instructed to record collection time, collect samples before eating, and freeze samples upon collection. The vast majority of participants were able to provide samples across all three days of sampling at both assessments (n missing one day: age 11 = 6, age 18.5 = 1; n missing two days: age 11 = 0, age 18.5 = 1). Samples were transported to the laboratory by researchers and