American Psychological Association

Psychology of Addictive Behaviors

0893-164X/19/$12.00

2019, Vol. 33, No. 3, 254–265
http://dx.doi.org/10.1037/adb0000454

Real-Time Social Stress Response and Subsequent Alcohol Use Initiation Among Female Adolescents

Renee M. Cloutier and Heidemarie Blumenthal
University of North Texas

Ryan S. Trim
University of California San Diego

Megan E. Douglas
University of North Texas

Kristen G. Anderson
Reed College

Adolescents who are particularly sensitive to social stress may be vulnerable to earlier alcohol consumption and related problems. Although a small literature supports this contention, previous studies mostly relied on retrospective self-report. The current study used discrete-time survival analysis (DTSA) to test whether real-time social stress responding (via laboratory induction) and social anxiety symptoms predicted 12-month alcohol onset in an alcohol-naïve sample of young female adolescents. Anxiety elicited by the task was expected to predict greater and earlier rates of alcohol incidence, particularly among girls with higher levels of self-reported social anxiety symptoms. Participants were 104 community-recruited girls (ages 12–15 years) who completed a modified Trier Social Stress Test and questionnaires; follow-up calls were conducted at 3, 6, 9, and 12 months after the laboratory visit. Self-reported anxiety was assessed in response to the stressor following acclimation (baseline), instruction (anticipation), and speech (posttask). By 12 months, 30.8% of the sample had consumed a full alcoholic beverage. The DTSA revealed that girls with higher levels of social anxiety and greater elevations in anticipatory (but not posttask) anxiety compared to baseline had earlier alcohol initiation. This is the first study to examine the role of both laboratory-induced anxious responding and retrospective reports of social anxiety as prospective predictors of alcohol incidence. These preliminary findings suggest that adolescent girls who are more sensitive to social stress may be at risk for experimenting with alcohol earlier than their peers.

Keywords: adolescent, alcohol incidence, social stress responding, anticipatory anxiety, discrete-time survival analysis (DTSA)

The majority of individuals in the United States will experiment with alcohol use during adolescence (e.g., ~61% by 12th grade; Miech et al., 2017), making early to middle adolescence a key period for prospectively studying the equifinality of initial alcohol use and subsequent risk. Adolescent alcohol use is linked to extensive societal costs and personal consequences (e.g., poor health outcomes, poorer academic achievement; Boden & Fergusson, 2011). Some estimates place the cost of underage drinking in the United States at $61.9 billion annually, including $34.7 billion related to violent crime and $13.7 billion to traffic accidents (Miller, Levy, Spicer, & Taylor, 2006). Although underage alcohol use often reflects experimentation related to normative developmental processes (Jessor, 1991; Johnston, O'Malley, Miech, Bachman, & Schulenberg, 2017), the initiation of alcohol use prior to age 16 years is associated with heightened risk for alcohol-related and other substance use problems (Jenkins et al., 2011; Wittchen et al., 2008). For instance, drawing from data collected in the National Longitudinal Study of Adolescent Health, Moss, Chen, and Yi (2014) found that adolescents who consumed alcohol prior to 16 years of age were significantly more likely to report daily cigarette smoking, recent binge drinking, and prescription and illicit drug misuse (e.g., methamphetamine) in young adulthood

This article was published Online First March 14, 2019.

Renee M. Cloutier and Heidemarie Blumenthal, Teen Stress and Alcohol Research Laboratory, Department of Psychology, University of North Texas; Ryan S. Trim, Department of Psychiatry, University of California San Diego; Megan E. Douglas, Department of Psychology, University of North Texas; Kristen G. Anderson, Department of Psychology, Reed College.

Renee M. Cloutier was partially supported by National Institute on Drug Abuse National Research Service Award 1F31DA041105 during the writing of this article. This project also was partially supported by a National Institute on Alcohol Abuse and Alcoholism grant awarded to Heidemarie Blumenthal (R15 AA026079-01). This article has not been, nor will it be, published by any other journal and is not under consideration for publication elsewhere. Earlier versions of the ideas and data appearing in this article were presented at poster sessions at the Collaborative Perspectives on Addiction Conference (2016) and a Research Society on Alcoholism meeting (2018).

Correspondence concerning this article should be addressed to either Renee M. Cloutier or Heidemarie Blumenthal, Teen Stress and Alcohol Research Laboratory, Department of Psychology, University of North Texas, 1155 Union Circle No. 311280, Denton, TX 76203. E-mail: reneecloutier@my.unt.edu or heidemarie.blumenthal@unt.edu

(ages 24–32) compared to those who had not yet consumed alcohol. Similarly, in a large sample of twin girls, participants who reported initial use prior to age 16 were over 3 times as likely to develop an alcohol use disorder (AUD) by age 30, with 32.9% of early users meeting AUD criteria compared to 12% of those who reported initiation later in life (Jenkins et al., 2011). These findings suggest that initial alcohol use in adolescence (particularly before age 16) may pose a risk for the development of later alcohol-related problems.

Although much of the literature has focused on externalizing pathways to adolescent substance use, a small but growing body of work has begun to highlight the importance of internalizing pathways (Hussong, Jones, Stein, Baucom, & Boeding, 2011) and maladaptive social stress responding in particular (Battista, Stewart, & Ham, 2010). This literature presents mixed findings, supporting positive, negative, and null associations between internalizing symptoms and stress responses with alcohol use indices (e.g., Battista et al., 2010; Colder et al., 2013; Fröjd, Ranta, Kaltiala-Heino, & Marttunen, 2011). One potential explanation for these inconsistent findings is that heterogeneity in internalizing symptoms, when broadly defined to include all depression and anxiety symptoms, may be masking divergent associations with specific symptom clusters (e.g., social anxiety [SA] vs. separation anxiety; Buckner et al., 2008; Marmorstein et al., 2010). This suggests there may be utility in focusing on specific symptom clusters, rather than general internalizing symptoms. SA and related social stress may be particularly important for understanding adolescent alcohol use. Adolescence is a period of increased sensitivity to social rewards and susceptibility to peer influence, and adolescent drinking commonly occurs in the context of peers (e.g., Anderson & Brown, 2010; Steinberg, 2007). Although SA is not consistently associated with use *frequency,* with some notable exceptions (Tomlinson & Brown, 2012), elevated social fears, including social anxiety disorder (SAD), relate to problematic alcohol use among both adolescents and adults (Blumenthal, Leen-Feldner, Badour, & Babson, 2011; Buckner, Heimberg, Ecker, & Vinci, 2013; Buckner & Schmidt, 2009). Although these findings suggest that sensitivity to social stressors are associated with greater alcohol use–related problems, the inconsistent associations with use frequency (e.g., Keough, Badawi, Nitka, O'Connor, & Stewart, 2016) make it difficult to explain how these patterns emerge. Of note, the majority of this literature relies solely on retrospective report of anxiety symptoms, which are subject to several biases (e.g., recall biases, introspective accuracy).

Experimental psychopathology techniques that incorporate real-time assessments can be used to maximize internal validity and minimize common biases of retrospective self-reports (Zvolensky, Lejuez, Stuart, & Curtin, 2001). Indeed, to circumvent potential biases, research conducted with adults has used real-time, laboratory-based tasks and assessments in the examination of acute social stress (e.g., giving an impromptu speech) and alcohol-relevant responses (e.g., sensory bias, reported craving, self-administration; Battista et al., 2010). Several studies have found that anxiety elicited by laboratory-induced social stress is positively correlated with alcohol craving and consumption among heavy-drinking (Field & Powell, 2007; Nesic & Duka, 2006) and socially drinking (de Wit, Söderpalm, Nikolayev, & Young, 2003; Magrys & Olmstead, 2015) adults and that the relation is especially pronounced among adults with relevant internalizing problems (e.g., SAD; Abrams, Kushner, Medina, & Voight, 2002). Further, individuals with a current AUD have been found to respond more strongly to an evaluative speech task than do those presenting with past AUD or no AUD history (Starcke, van Holst, van den Brink, Veltman, & Goudriaan, 2013). Last, in the one laboratory study to date conducted with adolescents (ages 14–17; recent alcohol consumption), symptoms of SA were positively correlated with the acute desire to drink following introduction to the novel, socially relevant laboratory protocol (Blumenthal, Ham, Cloutier, Bacon, & Douglas, 2016). Although these findings suggest *current* drinkers may be prone to consume alcohol in new, potentially stressful social contexts, it is unclear whether sensitivity to social stress might also be related to risk of early initial alcohol use among alcohol-naïve youth.

Finally, based on historical data indicating elevated alcohol use and disorder prevalence among men compared to women, the majority of work has targeted adult men and boys (Nolen-Hoeksema & Hilt, 2006; Schulte, Ramo, & Brown, 2009). However, in the past two decades, the gender gap in alcohol use has begun to close. In fact, recent national data have indicated that prior drinking, recent drinking, and past-year AUD prevalence are now higher among adolescent girls than boys (Forman-Hoffman, Edlund, Glasheen, & Ridenour, 2017; Kann et al., 2016; Miech et al., 2017; Perou et al., 2013). Further, young women evidence greater immediate risk of alcohol-related problems and injury (Stockwell et al., 2002), as well as a faster transition from use to disorder, compared to men (Diehl et al., 2007; Johnson, Richter, Kleber, McLellan, & Carise, 2005). A growing body of work has detailed differential structural and functional neuro-correlates of AUD among adult and adolescent women, suggesting not only distinct but elevated risk for the neurotoxic effects of alcohol among women (Caldwell et al., 2005; Medina et al., 2008). Collectively, sex-specific differences in risk and consequences of use, alongside an underrepresentation of women in this literature (Foster, Hicks, Iacono, & McGue, 2014) and greater rates of SAD diagnoses in girls (Merikangas et al., 2010) emphasize the need for research examining markers, processes, and outcomes among adolescent girls specifically (Brady & Randall, 1999; Medina et al., 2008).

The Current Study

Early alcohol use initiation (before age 16) poses significant risk for experiencing problems in adulthood, particularly among female adolescents; therefore, it is critical to obtain a better understanding of the processes that may underlie early alcohol use. When paired with longitudinal designs, laboratory analogues can be used to establish the temporal ordering of co-occurring phenomenon (e.g., SA and AUD) and provide enhanced sensitivity to prodromal symptoms (e.g., preclinical SA; Zvolensky et al., 2001). To date, several laboratory studies with adults and at least one study with adolescents have suggested that elevated social stress responses may lead to increased alcohol use among current drinkers. The present study seeks to build upon this work by examining whether alterations in stress responding might *precede* initial alcohol use among adolescents.

Specifically, the current study tests whether real-time social stress responding (elicited in the laboratory) would predict 12-month alcohol incidence in an alcohol-naïve sample of young

female adolescents, using discrete-time survival analysis (DTSA). It was expected that anxiety elicited by the task would positively relate to initiation of alcohol use over the following 12-month period. Individuals with higher levels of SA symptoms more generally are known to be particularly sensitive to social stressors and may therefore be more likely to initiate alcohol use as a means of coping with their acute anxiety. Accordingly, we also tested the potential interactions of trait-level SA symptoms with anxious reactivity to the task as predictors of alcohol incidence. Given their noted history with alcohol incidence, age, ethnicity, and race were considered as potential covariates (Forman-Hoffman et al., 2017). Though alcohol-naivety was considered "never consuming a *full* alcoholic beverage," there is recent evidence to suggest that youth often try small amounts of alcohol months or years before consuming a full alcoholic beverage (Colder, Shyhalla, & Frndak, 2018). Therefore, in the current study, baseline alcohol sipping history was included as a statistical control variable.

Method

Participants

Participants were drawn from a larger laboratory-based investigation on pubertal development and emotional vulnerability among community-recruited girls between the ages of 12 and 15 (N = 138). Recruitment for this project ran concurrently with a second project screening for adolescent boys and girls (ages 14–17) who consumed a full alcoholic beverage within the past year (Blumenthal, Cloutier, Douglas, Kearns, & Carey, 2019). Because participants could not participate in both studies, the current sample comprises alcohol-naïve girls or girls who have never consumed a full standard alcoholic beverage at screening (n = 119). Following the initial laboratory component, participants completed telephone follow-up interviews for 1 year at 3-month intervals; retention rates (from baseline) were 82.6% (3 months), 71.0% (6 months), 62.3% (9 months), and 53.6% (12 months).

The current analyses include only girls who reported no lifetime alcohol use or less than a full standard alcoholic beverage at baseline and had complete data on the primary baseline variables; that is, scores on all three Subjective Units of Distress Scale anxiety assessments (SUDS; Gotlib, Traill, Montoya, Joormann, & Chang, 2005; Wolpe, 1958) and the Revised Child Anxiety and Depression Scale—Social Phobia subscale (RCADS–SA; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000) as well as on at least one follow-up call. Thus, the final sample includes 104 early adolescent girls with a mean age of 13.9 at baseline. The racial composition of the final sample was comparable to that of the local community (Denton County, TX; U.S. Census Bureau, 2016): 65.7% Caucasian, 11.4% African American, 3.8% Asian, 2.9% Native American, 15.2% multiracial, and 1.0% "other." However, the proportion of Hispanics–Latinos was slightly lower (14.0%) than that of the local community (19.2%; Denton County, TX; U.S. Census Bureau, 2016). A series of independent-samples t tests and chi-square tests with baseline data indicated that there were no statistically significant differences in attrition or selection rates by age, SA symptoms, stress responsivity, ethnicity, race, or sipping status.

Measures and Task

Alcohol use. Participants were asked about their alcohol use at initial screening; during the laboratory visit; and through telephone interviews 3, 6, 9, and 12 months following the laboratory appointment.

Baseline alcohol use—Sipping at baseline. At initial screening, participants were asked "Have you ever consumed an alcoholic beverage, even just one or two sips?" If any alcohol use was endorsed, participants were asked to indicate whether they had consumed a full alcoholic beverage. Only participants who reported never consuming more than a few sips at the initial telephone screening were included in the current study. To control for sipping history, all screening responses were treated as a dichotomous variable such that 0 reflects "no alcohol use" and 1 reflects "endorsed sipping."

For 27 of 109 cases, telephone screening data were not available (i.e., ages 12–13; drinking status not assessed at screening for those under age 14) but otherwise had complete baseline and follow-up data. To retain these cases and maintain statistical power, we used data from the Adolescent Alcohol and Drug Involvement Scale (AADIS; Moberg, 2000) completed in the laboratory to infer use history. Specifically, the alcohol item asks how often participants have used alcohol but does not confirm whether positive endorsements refer to full alcoholic beverages or just a few sips. Most participant responses from the laboratory assessment reflected no lifetime alcohol use (n = 22), which was coded as 0 for "no alcohol use" on the sipping variable. However, the five cases that endorsed trying alcohol "once or twice" were excluded, resulting in a final sample of 104.

12-month alcohol use. During the follow-up calls participants reported on their alcohol use frequency via the Adolescent Alcohol and Drug Involvement Scale (AADIS; Moberg, 2000; Moberg & Hahn, 1991). The AADIS was completed in an interview format with a trained research assistant who would read the instructions and list the substance (i.e., alcohol) and each of the response items. Participants are asked to select a single response that best reflected how often they used alcohol on a scale of 0 (*never used*) to 7 (*several times a day*). Each response point is accompanied by a written descriptor, and higher numbers reflect increased frequency of use (e.g., 2 = *several times a year;* 3 = *several times a month*). If participants endorsed drinking, research assistants confirmed that they had consumed a full alcoholic beverage (i.e., not just sips). For the current analyses, responses were dichotomized such that reporting *never used* or *sips only* by 12 months was coded as 0, and any other positive endorsement at any follow-up call was coded as 1 so long as the endorsement included full alcoholic beverages. The AADIS is a face-valid scale that has been shown to strongly correspond with clinical assessments of use patterns (i.e., nonuse, abuse, dependence; Moberg & Hahn, 1991) and has been successfully employed in prior work examining substance use in similar community-based samples of adolescents (e.g., Blumenthal, Leen-Feldner, Frala, Badour, & Ham, 2010).

Social anxiety. The Revised Child Anxiety and Depression Scale—Social Phobia subscale (RCADS–SA; Chorpita et al., 2000) was used as a continuous measure of typical, or traitlike, SA symptoms. This nine-item self-report subscale includes items such as "I am afraid of looking foolish in front of other people" and "I worry about what others think of me," which participants rate on

a 4-point Likert-type scale (0 = *never,* 1 = *sometimes,* 2 = *often,* and 3 = *always*) as to how often each statement reflects how they typically feel. Responses are summed to create a total SA score. The RCADS evidences sound psychometric properties, demonstrating convergent validity with existing measures of childhood anxiety and anxiety disorders and test–retest reliability (SA test–retest coefficient = .80), as well as internal consistency (e.g., current sample Cronbach's α = .86; Chorpita et al., 2000). The RCADS also has been used in other studies of adolescent social anxiety and substance use (e.g., Cloutier, Blumenthal, & Mischel, 2016).

Social stress induction. Social stress was elicited through the Modified Trier Social Stress Test (TSST-M; Yim, Quas, Cahill, & Hayakawa, 2010). Participants were informed that they would complete a 5-min speech task for which they should imagine that they were introducing themselves to a new classroom and convincing the class that they would be a good student. Participants also had to include at least one good thing and one bad thing about themselves. They were given 3 min to prepare with paper and pencil but were informed that they would not be allowed to use any notes during the actual speech. Participants also were told that researchers would be viewing and evaluating their speech from the adjoining room via the camera located above them. Each participant prepared for her speech in private; a research assistant then reentered the room and instructed her to stand up, look directly into a camera, and begin the speech as soon as the researcher left the room and said "Go." Participants then spoke freely; however, if they stopped speaking for more than 10 s, a researcher in the adjoining room would remind them to continue speaking. Because the rooms were separated by a doorway, the researcher could provide prompts to participants without their seeing the researcher. If, after several prompts, they continued to stop speaking, a researcher would ask open-ended questions relevant to their speech. The TSST has a long history of safely and successfully eliciting socially relevant psychobiological stress among adults, adolescents, and children (Kudielka, Hellhammer, & Kirschbaum, 2007; Yim et al., 2010).

Real-time social stress response. Participants self-reported their acute emotional state immediately before task instructions (i.e., baseline), postinstructions–prespeech (i.e., in anticipation of task), and postspeech via the well-established Subjective Units of Distress Scale (SUDS; Gotlib et al., 2005; Wolpe, 1958). Specifically, a single-item measurement of how anxious the participant felt was rated on a scale of 0 (*Not at all*) to 10 (*A lot*). To create change scores reflecting increases in anxiety in anticipation of and immediately following the task, we subtracted baseline anxiety values from the postinstructions–prespeech anxiety values and postspeech anxiety values, respectively. This resulted in a possible range of −10 (high baseline anxiety; low anticipatory or posttask anxiety) to 10 (low baseline anxiety; high anticipatory or posttask anxiety).

Procedure

All study procedures were approved by the university Institutional Review Board. Participants were passively recruited from the local community with flyers and information booths at local events. Interested guardians and youth who contacted the laboratory were informed about study procedures. Eligibility (i.e., ages 12–15, female sex) was assessed via a brief telephone screener, at which point the initial laboratory visit was scheduled. Upon arrival, written guardian consent (for child) and assent was obtained for the lab visit as well as the follow-up calls. Thereafter, youth completed 45 min of questionnaires, followed by the baseline SUDS assessment. Participants then completed the social stress task described earlier as well as a series of interviews and questionnaires unrelated to the current study. At the end of the 2.5-hr protocol, participants were fully debriefed and compensated $30 for their time. Participants then scheduled their first, 3-month follow-up call.

Follow-up calls were always scheduled with respect to the period since the laboratory date (e.g., 90 days from lab appointment). When participants did not complete their scheduled call, research assistants were instructed to attempt contact–call completion up to 30 days after their original call date, at which point their call was skipped until the next scheduled call (e.g., 180 days from lab appointment) or the participant asked to withdraw from the study. At the completion of each call, participants scheduled their next follow-up call and were mailed $5 for their time.

Data Structure

The raw data were prepared based on recommendations by Muthén and Masyn (2005) so that the DTSA could be modeled in a latent variable framework. A total of four binary time-specific event indicators were constructed to reflect the 3-month intervals between the earliest postlab visit initiation onset (i.e., within 3 months) and the latest postlab visit initiation onset (i.e., between 9 and 12 months) that was assessed. For ease, the binary time-specific indicators is referred to by the month of assessment (i.e., 3-, 6-, 9-, and 12-month interview). The participant was considered to have experienced the event of interest in the interval in which they reported drinking at least one full standard alcoholic beverage for the first time. First-time endorsements of alcohol use were coded as 1 on the binary time-specific event indicators; prior event indicators were coded as 0, meaning the event had not occurred; and subsequent event indicators were coded as missing because they already experienced the event.

Analytic Approach

Repeated-measures analysis of variance (ANOVA) was conducted as a manipulation check to confirm that anxiety increased in anticipation of the speech and following the speech compared to baseline. Descriptive statistics were computed for the entire sample, and a series of independent-samples *t* tests (age) and chi-square tests (ethnicity, race, sipping history) were conducted to identify potential baseline differences among girls who initiated alcohol use and those who did not by the 12-month follow-up.

For the primary analyses, the relations between the selected baseline covariates and the alcohol onset outcomes were examined using a series of DTSA with a latent hazard function representing the event time distribution. The discrete-time hazard is the conditional probability that an adolescent will consume her first alcoholic beverage in a time period, given that she did not report alcohol use in previous time periods (Singer & Willett, 2003). The DTSA model provides information on whether the participant consumes an alcoholic beverage (i.e., event occurrence) as well as

when it occurs within the assessment period. The survival function is the sample's cumulative probability of not reporting any alcohol use across the 1-year assessment period; in other words, it is the probability of "surviving" through the 12-month follow-up as a nonuser (Muthén & Masyn, 2005). This survival function approximates the Cox regression model used in traditional continuous time survival models and is preferred when the data are categorical with fewer than 20 categories (Asparouhov, Masyn, & Muthén, 2006).

Based on the procedures outlined by Muthén and Masyn (2005), the DTSA was modeled as a special case within the latent variable framework in which a single-class latent class analysis corresponded to binary time-specific indicators. First, the constancy of the hazard rate assumption was tested to determine whether changes in the survival rate were constant across time. An unconditional survival model was fit that allowed the hazard rate to vary across the four, binary time-specific event indicators for alcohol incidence. This model was then compared to one that constrained the hazard rate to equality across intervals using a likelihood ratio test from the model deviance statistics. The hazard assumption is met if the constrained model fits as well as, or better than, the unconstrained model.

Second, the proportionality assumption for each of the predictors and covariates was tested to evaluate whether the effect of each covariate on the latent hazard function was equivalent across time. For each predictor–covariate, a model constraining their effect to equality was compared to a model allowing the variable to vary across time. The proportionality assumption for each variable is met if the constrained models fit as well as, or better than, the unconstrained models.

Third, a multivariate model with all the predictors was estimated such that the latent hazard function (with its four binary time-specific event indicators) was regressed on the set of predictors. This model provided estimates for the individual effects of each variable on alcohol incidence as well as the interactions of theoretical interest after controlling for all the other variables in the model. Two separate models that include the main and interaction effects of anticipatory anxiety change scores with RCADS SA symptoms (Model 1) and posttask anxiety change scores with RCADS SA symptoms (Model 2) are reported. To increase confidence in the final maximum likelihood values, we used automatically generated starting values with random perturbations (100 random sets of starting values with 30 full optimizations) for all models.

Results

Manipulation Check of the Stressor Task

First, the repeated-measures ANOVA indicated statistically significant within-subject differences in anxiety, $F(2, 206) = 12.946$, $p < .001$, $\eta_p^2 = .112$, across time. Specifically, pairwise comparisons confirmed statistically significant increases in anxiety both in anticipation of the speech ($M = 4.16$, $SD = 3.13$, $p < .001$) and following the speech ($M = 4.20$, $SD = 3.11$, $p < .001$) compared to baseline levels ($M = 3.15$, $SD = 2.74$).

Demographics and Covariate Selection

Table 1 presents baseline demographic information of the total sample and by overall initiation status.[1] By 12 months, 30.8% of the sample had consumed a full alcoholic beverage. Specifically, 13.5% of the sample newly initiated at 3 months, 8.7% newly initiated at 6 months, 3.9% newly initiated at 9 months, and 4.8% newly initiated at 12 months. Adolescents who initiated alcohol use were slightly older, $t(101) = -2.69$, $p = .008$, and were more likely to have sipped alcohol, $\chi^2(1, N = 104) = 19.13$, $p < .001$, at baseline compared to noninitiators. There were no differences in terms of SA symptoms, $t(101) = -1.32$, $p = .190$; ethnicity, $\chi^2(1, N = 103) = .41$, $p = .524$; race, $\chi^2(4, N = 99) = 1.874$, $p = .759$; or grade in school, $\chi^2(5, N = 103) = 6.907$, $p = .228$, across initiation groups. Additional t tests exploring whether individuals who reported a sipping history (compared to those without a sipping history) differed on any of the acute measures of anxiety during the TSST-M (i.e., baseline, anticipatory, posttask, anticipatory–baseline change scores, posttask–baseline change scores) or RCADS–SA were not statistically significant (ps $> .05$; data not shown). Based on these initial analyses, the only covariates included in the final model were age and sipping history.

Discrete Time Survival Analyses

Unconditional survival model. The unconditional hazard function in Figure 1 demonstrates a gradual decrease in rates of "surviving" alcohol onset across the 1-year follow-up period. Approximately 69.2% of girls did not report any alcohol use by their final interview. The likelihood ratio test comparing the unconditional hazard model with time-varying hazard rates to one that constrained the hazard rates to equality was not statistically significant, meeting the constancy of the baseline hazard rate assumption. Therefore, the hazard rate function was constrained to equality across all models.

Univariate covariate effects. None of the likelihood ratio tests comparing the models where the effects of each of the predictor variables on alcohol onset were allowed to vary, against models where the effects were constrained across time, were significant (analyses not shown). Because all the predictor variables met the proportionality assumption, the effect of each predictor was estimated to be proportional across time.

The parameter estimates (i.e., log hazard odds) for the univariate effects of each covariate on the hazard probabilities are listed in Table 2, under the Univariate Effects heading. Three baseline predictors were statistically significant in the expected direction: older age, sipping history, and higher change scores on posttask anxiety from baseline were all associated with increased alcohol onset. Univariate change scores in anticipatory anxiety from baseline were also in the expected direction ($p = .06$).

Multivariate survival model. In Table 2, the log hazard odds for each variable are presented under the Full Model heading. After adjusting for the effects of all other variables, sipping history and the interaction term for change scores in anticipatory anxiety and SA symptoms were statistically significant (ps $< .05$). The interaction term between posttask anxiety change scores and SA did not reach the threshold for statistical significance ($p = .06$).

[1] A small number of cases (n = 1–5) were missing data on some or all of the demographic characteristics tested. The preliminary analyses were run on the data that was available, resulting in slightly variable N's and df reported, though do not ultimately change the primary analyses or final interpretation of findings.

Table 1
Baseline Demographics for the Total Sample and by Initiation Status

Variable	Total (N = 104)		Never initiated (n = 72)		Initiated (n = 32)	
	M (*SD*)	%	*M* (*SD*)	%	*M* (*SD*)	%
Age (years)	13.95 (.97)		**13.78 (.99)**		**14.31 (.82)**	
Social anxiety symptoms	12.70 (6.73)		12.20 (6.55)		14.08 (6.97)	
Anticipatory anxiety change score	1.01 (2.27)		.78 (2.42)		1.47 (1.90)	
Posttask anxiety change score	1.05 (2.21)		.79 (2.32)		1.50 (1.81)	
Ethnicity (Hispanic/Latino)		12.9		14.3		9.7
Race						
Caucasian/White		68.7		68.7		68.8
African American/Black		10.1		9.0		12.5
Asian American		4.0		4.5		3.1
Native American		3.0		4.5		.0
Other/Multiracial		14.1		13.4		15.6
Year in school						
Sixth grade		5.8		5.6		6.3
Seventh grade		10.7		12.7		6.3
Eighth grade		16.5		21.1		6.3
Ninth grade		42.7		40.8		46.9
10th grade		23.3		18.3		34.4
Other (homeschooled)		1.0		1.4		.0
Sipping		25.2		**12.7**		**53.1**

Note. Bolded values were statistically different at $p < .05$ for initiators and noninitiators.

To provide a visual representation of the interaction terms, we created a series of tables (data not shown) ordering the new alcohol initiations at 3, 6, 9, and 12 months by dichotomized subgroups of the main effect variables. Median splits were used to create binary variables for RCADS–SA (<11 vs. ≥11), anticipatory anxiety change scores from baseline (<1 vs. ≥1), and posttask anxiety change scores from baseline (<1 vs. ≥1). For both sets of interactions, four subgroups were created, with the survival function calculated for each subgroup individually graphed across time. Figure 2 shows the rate of alcohol onset (i.e., decreased survival–abstinence rates) across the four subgroups formed by crossing high–low categories of SA and anticipatory anxiety change scores from baseline. The only group to not show an immediate decrease in survival at 3 months was the high SA–low anticipatory change group (100% survival rate), though they did show a gradual decline at 6, 9, and 12 months. Both of the low SA groups and the high SA - high anticipatory change group showed immediate sharp declines in survival at 3 months (78%–82%); however, whereas

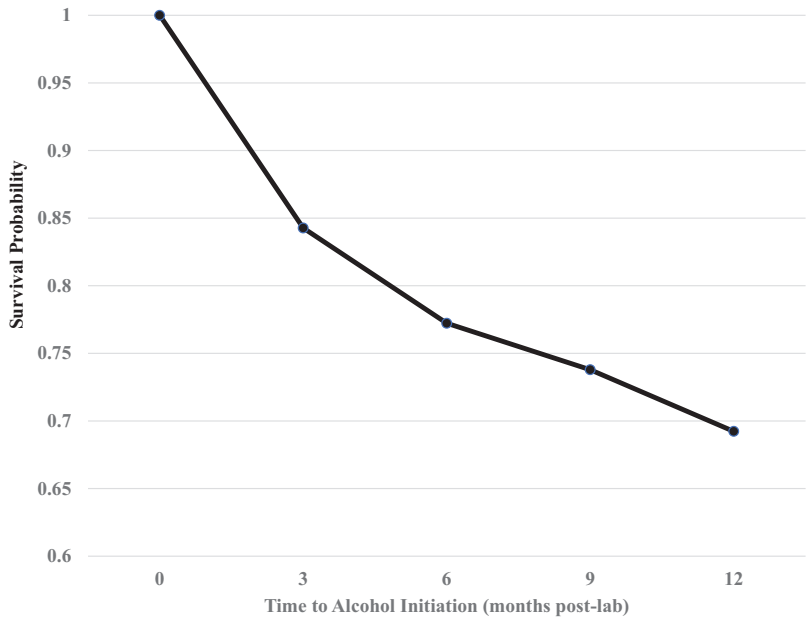

Figure 1. Fitted survival probabilities for onset of alcohol use following lab assessment. See the online article for the color version of this figure.

Table 2
Hazard Estimates of Alcohol Onset From Baseline SA and Social Stress Response (n = 104)

Model and variable	Univariate effects			Full model		
	b (*SE*)	*t*	*OR* [95% CI]	*b* (*SE*)	*t*	*OR* [95% CI]
Model 1: Anticipatory anxiety						
Age	**.60 (.24)**	**2.47**	**1.83 [1.13, 2.95]**	.24 (.27)	.88	1.26 [.75, 2.14]
Any sipping	**1.87 (.44)**	**4.29**	**6.50 [2.77, 15.28]**	**1.89 (.49)**	**3.85**	**6.61 [2.52, 17.31]**
Social anxiety (SA)	.03 (.03)	.89	1.03 [.97, 1.08]	.01 (.03)	.21	1.01 [.94, 1.08]
Antic. anxiety Δ	.14 (.07)	1.88	1.14 [.99, 1.31]	.17 (.09)	1.92	1.18 [.99, 1.40]
SA × Antic. Anxiety Δ				**.03 (.01)**	**2.17**	**1.03 [1.00, 1.05]**
Model 2: Posttask anxiety						
Age	**.60 (.24)**	**2.47**	**1.83 [1.13, 2.95]**	.38 (.24)	1.62	1.47 [.92, 2.33]
Any sipping	**1.87 (.44)**	**4.29**	**6.50 [2.77, 15.28]**	**1.77 (.45)**	**3.96**	**5.89 [2.45, 14.18]**
SA	.03 (.03)	.89	1.03 [.97, 1.08]	.02 (.03)	.74	1.02 [.96, 1.09]
Posttask anxiety Δ	**.15 (.07)**	**2.07**	**1.16 [1.01, 1.34]**	.15 (.09)	1.72	1.17 [.98, 1.39]
SA × Posttask Anxiety Δ				.02 (.01)	1.83	1.02 [.99, 1.05]

Note. Model 1 tests the interaction of SA and anticipatory anxiety as the primary predictor while controlling for main effects and baseline variables; Model 2 tests the interaction of SA and posttask anxiety as the primary predictor while controlling for main effects and baseline variables. Bold indicates effects that are statistically significant ($p < .05$). *OR* = odds ratio; CI = confidence interval; Antic. Anxiety Δ = anticipatory anxiety change score (calculated by subtracting the baseline report of Subjective Units of Distress Scale [SUDS] anxiety from pretask SUDS anxiety); posttask anxiety Δ = posttask anxiety change score (calculated by subtracting the baseline report of SUDS anxiety from posttask SUDS anxiety).

the low SA–groups appeared to stabilize through the 12-month assessment (74%–85%), the high SA–high anticipatory change group continued to decline steadily to ~48% survival rate.

Figure 3 shows the rate of alcohol onset (i.e., decreased survival–abstinence rates) across the four subgroups formed by crossing high–low categories of SA and posttask anxiety change scores from baseline. Overall, the findings were similar to the interaction findings between SA and anticipatory anxiety change scores. The only group to not show an immediate decrease in survival at 3 months was the high SA–low posttask anxiety change group (100% survival rate), though they did show a gradual decline at 6, 9, and 12 months. The low SA–low posttask anxiety change group had a decline in survival that was slightly more gradual (e.g., 90% at 3 months) than did the corresponding anticipatory anxiety change interaction noted earlier (e.g., 82% at 3 months), though it did stabilize again at 82% between 6 and 12 months. The low SA–high posttask anxiety change group had a steeper immediate decline in survival (80% at 3 months) than did the corresponding anticipatory anxiety interaction noted earlier (90%), but then it stabilized through 12 months. Finally, the high SA–high posttask anxiety change group evidenced the steepest decline in survival rates, starting at 3 months (80%) and continuing through 12 months (51%).

Discussion

Early alcohol use initiation (before age 16) is often associated with alcohol-related problems in adulthood. To date, most studies examining internalizing pathways have been limited to retrospec-

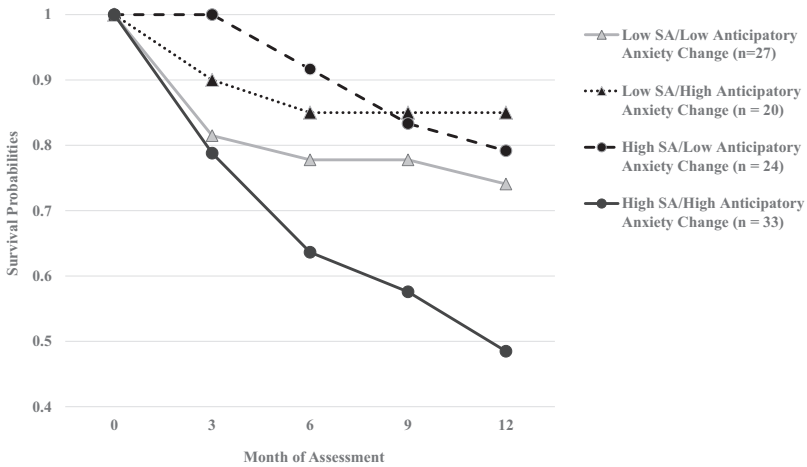

Figure 2. Survival probabilities of alcohol onset by high/low social anxiety–anticipatory anxiety change score subgroups. SA = social anxiety.

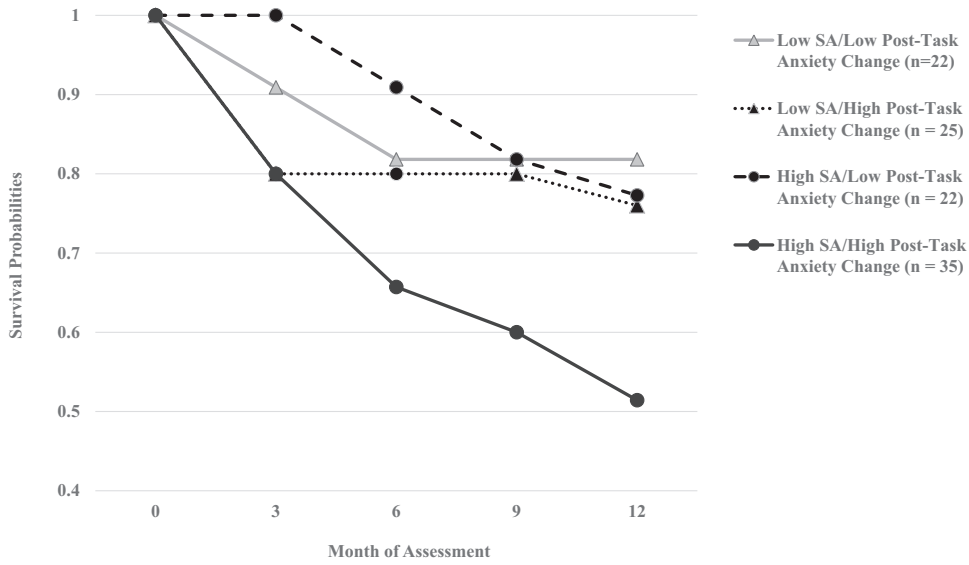

Figure 3. Survival probabilities of alcohol onset by high/low social anxiety–posttask anxiety change score subgroups. SA = social anxiety.

tive self-report, often of global symptoms. Although there has been some evidence to suggest sensitivity to social stress is related to acute alcohol desires among current drinkers, whether exaggerated stress responses might *precede* initial alcohol use and related problems among adolescents has not been well understood. The present study sought to build upon the existing literature by testing whether social stress reactivity elicited in the laboratory was related to 12-month incidence of drinking among young female adolescents who never consumed a full alcoholic beverage. Consistent with hypotheses, increased anxiety elicited by the task was associated with greater and earlier rates of alcohol initiation over the following 12-month period. However, the moderating effects of SA was only statistically significant with anticipatory anxiety and not posttask anxiety. Girls with elevated anticipatory anxiety and greater levels of retrospectively reported SA symptoms appeared to have the greatest and earliest rates of onset. Findings were robust to the inclusion of age and alcohol sipping history at baseline. Building off a tension-reduction framework, it is possible that the exaggerated responses to a social stressor may be a marker of inadequate coping mechanisms and therefore risk for alcohol initiation. Indeed, social relationships with peers are a central concern to adolescents (e.g., Steinberg, 2007)—and alcohol may be viewed as a means of coping with social stress, which in turn, prompts the initiation of early alcohol use (Kuntsche, Knibbe, Engels, & Gmel, 2007).

Prior research has found that exaggerated stress–anxiety responding is associated with alcohol craving among *current* drinkers and those with an AUD (e.g., Blumenthal et al., 2016; Starcke et al., 2013). We extend this work by examining the prospective association between laboratory-induced social stress–anxiety and alcohol initiation. Theoretical accounts of why internalizing symptoms, such as social stress and anxiety, are associated with alcohol use have been based in large part on self-medication and tension reduction models, proposing that people use alcohol as a means of coping with acute and/or anticipated emotional distress (Khantzian, 1985). Self-medication models have been expanded to include a variety of relevant factors (e.g., genetics, social factors; Buckner et al., 2008; Hussong et al., 2011), and an implicit assumption in this work is that people need to experience the anxiolytic effects of alcohol in order to self-medicate. One interpretation of these models suggests that social stress and anxiety may not be germane to understanding initiation and early experimentation of alcohol use (cf. heavy use or AUD). In contrast, the present findings suggest that exaggerated anxiety responses to anticipated social stressors may temporally precede alcohol incidence and is involved in the early stages of adolescent drinking.

That this effect was statistically significant in terms of only anticipatory anxiety (cf. task anxiety) is consistent with several findings in which anticipation of stressors often elicits greater anxiety–stress responses than does the experience of the stressor itself (e.g., Evans, Greaves-Lord, Euser, Franken, & Huizink, 2012; van den Bos, de Rooij, Miers, Bokhorst, & Westenberg, 2014). It may also be important to view anticipatory anxiety through a developmental lens when considering initiation of alcohol use. During adolescence, there is an increase in sensitivity to potentially negative social evaluations (Ollendick & Hirshfeld-Becker, 2002; Westenberg, Drewes, Goedhart, Siebelink, & Treffers, 2004). In a 2-year prospective study where children and adolescents (ages 8–17 at baseline) completed two speech tasks, children evidenced the greatest stress responding during the speech, whereas adolescents evidenced the greatest response in anticipation of the speech (van den Bos et al., 2014). With emerging and shifting expectancies about alcohol in early adolescence (Dunn & Goldman, 2000; Smit et al., 2018), adolescents may initiate alcohol use as a coping strategy (Johnston et al., 2017; Steinberg, 2007) or in an attempt to "fit in" or avoid negative evaluations (Blumenthal et al., 2011; Buckner & Schmidt, 2009).

Of note, the findings somewhat diverge from a significant body of work that argues *blunted* stress–anxiety reactivity is a precursor to alcohol initiation and problems (e.g., Evans et al., 2012). The

underlying argument is that certain individuals are less reactive to their environment, including stressors, and therefore begin using alcohol as a means of enhancing their internal state (e.g., sensation seeking; Goeders, 2003). Although we found that the low SA–low anticipatory anxiety change group evidenced drops in survival similar to those in the high SA–high anticipatory change group at 3 months, their rates of survival plateaued through the 12-month assessment. It is possible that the cross-sectional examinations of blunted stress–anxiety with clinical samples are capturing associations later in the etiological process (i.e., blunted responding develops after alcohol onset). It is also possible that with a longer evaluation period, the low SA–low anticipatory change group would have caught up with or even surpassed the high SA–high anticipatory change group, supporting the equifinality of alcohol-related risk as well as highlighting two distinct high-risk groups for alcohol initiation–later alcohol problems. Nonetheless, as the first study to report these findings, replication is key. In particular, future studies should incorporate specific physiological and subjective measures of stress responding as well as repeated stress task administrations beginning before alcohol initiation (e.g., ages 12–15) with sufficient follow-ups to assess changes in both stress responses and various alcohol patterns (e.g., incidence to problematic drinking). Although this would require substantially larger samples and more complex analyses to account for normative changes in incidence and progression, such findings would provide clear and comprehensive insight into the underlying physiological and cognitive processes that drive changes in alcohol use.

Although measuring internalizing symptomology as anxious reactivity to a social stressor is a novel, methodological strength, we did not include real-time assessments of other internalizing symptoms (e.g., feeling sad). Future researchers should consider the extent to which the findings are specific to anxiety (cf. internalizing symptoms–negative affect more broadly). The current study also did not include potential covariates from the externalizing domain. Externalizing symptoms and related variables are robust correlates of initiation of adolescent alcohol use, and recent work has highlighted the importance of examining internalizing and externalizing symptoms concurrently (e.g., Colder et al., 2013; Colder et al., 2017; Hussong, Ennett, Cox, & Haroon, 2017). It is possible that the association between anticipatory anxiety and alcohol incidence would be better accounted for by co-occurring externalizing symptoms such as impulsivity or behavioral disruptiveness (Gibbons et al., 2016; Kessler et al., 2006). For example, externalizing behaviors may further moderate the association between acute anxiety symptoms and alcohol incidence (Colder et al., 2017; Keough et al., 2016). Further, recent studies have shown that impulsivity moderates the association between SA symptoms, coping-motivated drinking, and other drinking outcomes that could help explain the lack of main effects for SA in the present study (e.g., Keough et al., 2016). Laboratory tasks that tap into certain aspects of externalizing such as impulsivity (e.g., BART; Lejuez, Aklin, Zvolensky, & Pedulla, 2003; Lejuez et al., 2002) or inattentiveness (e.g., Conners' Adult ADHD Rating Scales; Conners, Erhardt, & Sparrow, 1999) may aid in parsing apart these relations. Future researchers should consider additional ways in which externalizing behaviors (e.g., behavioral disruption) might be modeled statistically as well as in laboratory settings. Finally, most of the literature has examined externalizing- and internalizing-type problems as (at least statistically) mutually exclusive; research seeking to understand the additive, interactive, and reinforcing links among behaviors traditionally conceptualized as internalizing or externalizing is needed (e.g., Colder et al., 2013).

In the current study, sipping history was included as a potential covariate based on recent literature indicating that youth begin trying alcohol months, or even years, before consuming their first full alcoholic beverage (Colder et al., 2018). As expected, we found that girls who reported sipping alcohol at baseline were more likely to initiate alcohol use within the following year and at earlier rates than were those who did not report any sipping at baseline. Although this finding is consistent with the limited work in this area, it is worth noting that the 95% confidence intervals for that variable were fairly large, preventing us from drawing any conclusions regarding precisely how strong the effect of sipping behaviors has on alcohol initiation. Notably, wide confidence intervals generally occur with low base rate behaviors and small sample sizes. Although we are fairly confident that sipping history would lead to earlier rates of alcohol onset, larger epidemiological studies on this new topic area are certainly warranted.

The present study includes several additional limitations that need to be addressed. First, the present study recruited only young adolescent girls as a means of reducing gender biased effects. Although these findings contribute to a literature that has historically focused on male-only samples, and there are recent data suggesting minimal gender differences in terms of substance use rates and stress responding (e.g., Miech et al., 2017; van den Bos et al., 2014), the lack of male representation in the current study precludes the possibility of directly testing potential gender differences. Male and female individuals may differ in the way they react to stressors, particularly those that are more socially oriented. Second, a modified version of the Trier Social Stress Test (Yim et al., 2010) was selected as the social stressor, backed by decades of empirical data showing that it effectively elicits both subjective and psychobiological stress responses (Kudielka et al., 2007). However, performance-oriented stressors such as the speech task may not be ideally suited to model real-life stressors that relate to alcohol use, particularly among adolescents. Rejection and peer-evaluation tasks (e.g., Cyberball, Yale Interpersonal Stressor; Stroud, Tanofsky-Kraff, Wilfley, & Salovey, 2000; Williams & Jarvis, 2006) may elicit responses that are more consistent with real-life stressors related to alcohol use. Third, although we statistically controlled for age and early sipping history, there were several additional covariates not assessed that should be considered in future work (e.g., parent–friend substance use; Ennett et al., 2016). Last, although the aim of the current project was focused exclusively on predicting alcohol incidence, there is a wealth of data highlighting adolescence as a period of trying a variety of substances beyond alcohol (e.g., cigarettes, cannabis; Johnston et al., 2017) as well as the role of anxious reactivity in the development of problematic use associated with those substances (Blumenthal et al., 2011). Future work should consider the role of real-time anxious reactivity and SA symptoms as predictors of initiating multiple substances across a longer time frame. Further research is also needed to examine the extent to which real-time anxious reactivity and SA symptoms might predict varying *levels* of use or use patterns by including continuous measures of alcohol use frequency and additional measures of alcohol-related behaviors–consequences (e.g., Savage et al., 2016).

Together, the current study found that girls who were more sensitive to acute social stress also were more likely to initiate alcohol use in the following year. This is the first study to recruit largely alcohol-naïve youth from the community, collect real-time assessments of reactions to a stressor task, and conduct prospective follow-ups. The current findings support the potential utility of targeting social stress responding and management of such distress in the development of selective intervention efforts. Indeed, despite decades of research, early intervention efforts aimed at reducing adolescent alcohol use incidence and problems evidence mixed findings (e.g., Cuijpers, 2002; Sandler et al., 2014). Some of the most consistent, promising data emerge from selective intervention efforts built upon preclinical work identifying risk markers and factors (e.g., high anxiety sensitivity) and related mechanisms (e.g., coping with acute distress). For example, the PreVenture program tailors intervention modules to match one of four identified risk factors (i.e., anxiety sensitivity, hopelessness, impulsivity, sensation seeking) to reduce rates of alcohol initiation and risky-drinking behavior among adolescents (e.g., binge drinking; Conrod et al., 2013; Conrod, Stewart, Comeau, & Maclean, 2006). Consistent with the development and testing of this program, continued efforts aimed at clarifying the nature and boundaries of the SA–alcohol use risk link will aid in further refining such targeted intervention. It is important to note that no other studies to date have examined the role of acute social stress responses and subsequent alcohol initiation among adolescents; therefore, replication of these initial effects as well as work targeting moderators (e.g., use expectancies, coping styles, peer affiliation) and mediators (e.g., use willingness) of this association is needed (Anderson et al., 2014; Fromme & D'Amico, 2000). Additional research pairing a range of real-time assessments (e.g., laboratory stressors, ecological momentary assessment) with prospective follow-ups is needed, particularly with adolescent samples.

References

Abrams, K., Kushner, M. G., Medina, K. L., & Voight, A. (2002). Self-administration of alcohol before and after a public speaking challenge by individuals with social phobia. *Psychology of Addictive Behaviors, 16,* 121–128. http://dx.doi.org/10.1037/0893-164X.16.2.121

Anderson, K. G., Brackenbury, L., Quackenbush, M., Buras, M., Brown, S. A., & Price, J. (2014). A-SIDE: Video simulation of teen alcohol and marijuana use contexts. *Journal of Studies on Alcohol and Drugs, 75,* 953–957. http://dx.doi.org/10.15288/jsad.2014.75.953

Anderson, K. G., & Brown, S. A. (2010). Middle school drinking: Who, where and when. *Journal of Child & Adolescent Substance Abuse, 20,* 48–62. http://dx.doi.org/10.1080/1067828X.2011.534362

Asparouhov, T., Masyn, K., & Muthén, B. (2006). Continuous time survival in latent variable models. *Proceedings of the Joint Statistical Meeting in Seattle* (pp. 180–187). Chesterfield, MO: Mira Digital.

Battista, S. R., Stewart, S. H., & Ham, L. S. (2010). A critical review of laboratory-based studies examining the relationships of social anxiety and alcohol intake. *Current Drug Abuse Reviews, 3,* 3–22. http://dx.doi.org/10.2174/1874473711003010003

Blumenthal, H., Cloutier, R. M., Douglas, M. E., Kearns, N. T., & Carey, C. (2019). *The desire to drink as a function of laboratory-induced social stress among adolescents*. Manuscript submitted for publication.

Blumenthal, H., Ham, L. S., Cloutier, R. M., Bacon, A. K., & Douglas, M. E. (2016). Social anxiety, disengagement coping, and alcohol-use behaviors among adolescents. *Anxiety, Stress, & Coping, 29,* 432–446. http://dx.doi.org/10.1080/10615806.2015.1058366

Blumenthal, H., Leen-Feldner, E. W., Badour, C. L., & Babson, K. A. (2011). Anxiety psychopathology and alcohol use among adolescents: A critical review of the empirical literature and recommendations for future research. *Journal of Experimental Psychopathology, 2,* 318–353. http://dx.doi.org/10.5127/jep.012810

Blumenthal, H., Leen-Feldner, E. W., Frala, J. L., Badour, C. L., & Ham, L. S. (2010). Social anxiety and motives for alcohol use among adolescents. *Psychology of Addictive Behaviors, 24,* 529–534. http://dx.doi.org/10.1037/a0019794

Boden, J. M., & Fergusson, D. M. (2011). The short and long term consequences of adolescent alcohol use. In J. B. Saunders and J. M. Rey (Eds.), *Young people & alcohol: Impact, policy, prevention treatment* (pp. 32–46). Chichester, United Kingdom: Wiley-Blackwell.

Brady, K. T., & Randall, C. L. (1999). Gender differences in substance use disorders. *Psychiatric Clinics of North America, 22,* 241–252. http://dx.doi.org/10.1016/S0193-953X(05)70074-5

Buckner, J. D., Heimberg, R. G., Ecker, A. H., & Vinci, C. (2013). A biopsychosocial model of social anxiety and substance use. *Depression and Anxiety, 30,* 276–284. http://dx.doi.org/10.1002/da.22032

Buckner, J. D., & Schmidt, N. B. (2009). Understanding social anxiety as a risk for alcohol use disorders: Fear of scrutiny, not social interaction fears, prospectively predicts alcohol use disorders. *Journal of Psychiatric Research, 43,* 477–483. http://dx.doi.org/10.1016/j.jpsychires.2008.04.012

Buckner, J. D., Schmidt, N. B., Lang, A. R., Small, J. W., Schlauch, R. C., & Lewinsohn, P. M. (2008). Specificity of social anxiety disorder as a risk factor for alcohol and cannabis dependence. *Journal of Psychiatric Research, 42,* 230–239. http://dx.doi.org/10.1016/j.jpsychires.2007.01.002

Caldwell, L. C., Schweinsburg, A. D., Nagel, B. J., Barlett, V. C., Brown, S. A., & Tapert, S. F. (2005). Gender and adolescent alcohol use disorders on BOLD (blood oxygen level dependent) response to spatial working memory. *Alcohol and Alcoholism, 40,* 194–200. http://dx.doi.org/10.1093/alcalc/agh134

Chorpita, B. F., Yim, L., Moffitt, C., Umemoto, L. A., & Francis, S. E. (2000). Assessment of symptoms of *DSM–IV* anxiety and depression in children: A revised Child Anxiety and Depression Scale. *Behaviour Research and Therapy, 38,* 835–855. http://dx.doi.org/10.1016/S0005-7967(99)00130-8

Cloutier, R. M., Blumenthal, H., & Mischel, E. R. (2016). An examination of social anxiety in marijuana and cigarette use motives among adolescents. *Substance Use & Misuse, 51,* 408–418. http://dx.doi.org/10.3109/10826084.2015.1110174

Colder, C. R., Scalco, M., Trucco, E. M., Read, J. P., Lengua, L. J., Wieczorek, W. F., & Hawk, L. W., Jr. (2013). Prospective associations of internalizing and externalizing problems and their co-occurrence with early adolescent substance use. *Journal of Abnormal Child Psychology, 41,* 667–677. http://dx.doi.org/10.1007/s10802-012-9701-0

Colder, C. R., Shyhalla, K., & Frndak, S. E. (2018). Early alcohol use with parental permission: Psychosocial characteristics and drinking in late adolescence. *Addictive Behaviors, 76,* 82–87. http://dx.doi.org/10.1016/j.addbeh.2017.07.030

Colder, C. R., Shyhalla, K., Frndak, S., Read, J. P., Lengua, L. J., Hawk, L. W., Jr., & Wieczorek, W. F. (2017). The prospective association between internalizing symptoms and adolescent alcohol involvement and the moderating role of age and externalizing symptoms. *Alcoholism: Clinical and Experimental Research, 41,* 2185–2196. http://dx.doi.org/10.1111/acer.13512

Conners, C. K., Erhardt, D., & Sparrow, E. P. (1999). *Conners' Adult ADHD Rating Scales*. North Tonawanda, NY: Multi-Health Systems.

Conrod, P. J., O'Leary-Barrett, M., Newton, N., Topper, L., Castellanos-Ryan, N., Mackie, C., & Girard, A. (2013). Effectiveness of a selective, personality-targeted prevention program for adolescent alcohol use and misuse: A cluster randomized controlled trial. *Journal of the American Medical Association Psychiatry, 70,* 334–342. http://dx.doi.org/10.1001/jamapsychiatry.2013.651

Conrod, P. J., Stewart, S. H., Comeau, N., & Maclean, A. M. (2006). Efficacy of cognitive-behavioral interventions targeting personality risk

factors for youth alcohol misuse. *Journal of Clinical Child and Adolescent Psychology, 35,* 550–563. http://dx.doi.org/10.1207/s15374424jccp3504_6

Cuijpers, P. (2002). Effective ingredients of school-based drug prevention programs: A systematic review. *Addictive Behaviors, 27,* 1009–1023. http://dx.doi.org/10.1016/S0306-4603(02)00295-2

de Wit, H., Söderpalm, A. H. V., Nikolayev, L., & Young, E. (2003). Effects of acute social stress on alcohol consumption in healthy subjects. *Alcoholism: Clinical and Experimental Research, 27,* 1270–1277. http://dx.doi.org/10.1097/01.ALC.0000081617.37539.D6

Diehl, A., Croissant, B., Batra, A., Mundle, G., Nakovics, H., & Mann, K. (2007). Alcoholism in women: Is it different in onset and outcome compared to men? *European Archives of Psychiatry and Clinical Neuroscience, 257,* 344–351. http://dx.doi.org/10.1007/s00406-007-0737-z

Dunn, M. E., & Goldman, M. S. (2000). Validation of multidimensional scaling-based modeling of alcohol expectancies in memory: Age and drinking-related differences in expectancies of children assessed as first associates. *Alcoholism: Clinical and Experimental Research, 24,* 1639–1646. http://dx.doi.org/10.1111/j.1530-0277.2000.tb01965.x

Ennett, S. T., Jackson, C., Cole, V. T., Haws, S., Foshee, V. A., Reyes, H. L. M., . . . Cai, L. (2016). A multidimensional model of mothers' perceptions of parent alcohol socialization and adolescent alcohol misuse. *Psychology of Addictive Behaviors, 30,* 18–28. http://dx.doi.org/10.1037/adb0000119

Evans, B. E., Greaves-Lord, K., Euser, A. S., Franken, I. H., & Huizink, A. C. (2012). The relation between hypothalamic-pituitary-adrenal (HPA) axis activity and age of onset of alcohol use. *Addiction, 107,* 312–322. http://dx.doi.org/10.1111/j.1360-0443.2011.03568.x

Field, M., & Powell, H. (2007). Stress increases attentional bias for alcohol cues in social drinkers who drink to cope. *Alcohol and Alcoholism, 42,* 560–566. http://dx.doi.org/10.1093/alcalc/agm064

Forman-Hoffman, V. L., Edlund, M., Glasheen, C., & Ridenour, T. (2017). Alcohol initiation and progression to use, heavy episodic use, and alcohol use disorder among young adolescents ages 12–14 living in U.S. households. *Journal of Studies on Alcohol and Drugs, 78,* 853–860. http://dx.doi.org/10.15288/jsad.2017.78.853

Foster, K. T., Hicks, B. M., Iacono, W. G., & McGue, M. (2014). Alcohol use disorder in women: Risks and consequences of an adolescent onset and persistent course. *Psychology of Addictive Behaviors, 28,* 322–335. http://dx.doi.org/10.1037/a0035488

Fröjd, S., Ranta, K., Kaltiala-Heino, R., & Marttunen, M. (2011). Associations of social phobia and general anxiety with alcohol and drug use in a community sample of adolescents. *Alcohol and Alcoholism, 46,* 192–199. http://dx.doi.org/10.1093/alcalc/agq096

Fromme, K., & D'Amico, E. J. (2000). Measuring adolescent alcohol outcome expectancies. *Psychology of Addictive Behaviors, 14,* 206–212. http://dx.doi.org/10.1037/0893-164X.14.2.206

Gibbons, F. X., Kingsbury, J. H., Wills, T. A., Finneran, S. D., Dal Cin, S., & Gerrard, M. (2016). Impulsivity moderates the effects of movie alcohol portrayals on adolescents' willingness to drink. *Psychology of Addictive Behaviors, 30,* 325–334. http://dx.doi.org/10.1037/adb0000178

Goeders, N. E. (2003). The impact of stress on addiction. *European Neuropsychopharmacology, 13,* 435–441. http://dx.doi.org/10.1016/j.euroneuro.2003.08.004

Gotlib, I. H., Traill, S. K., Montoya, R. L., Joormann, J., & Chang, K. (2005). Attention and memory biases in the offspring of parents with bipolar disorder: Indications from a pilot study. *Journal of Child Psychology and Psychiatry, 46,* 84–93. http://dx.doi.org/10.1111/j.1469-7610.2004.00333.x

Hussong, A. M., Ennett, S. T., Cox, M. J., & Haroon, M. (2017). A systematic review of the unique prospective association of negative affect symptoms and adolescent substance use controlling for externalizing symptoms. *Psychology of Addictive Behaviors, 31,* 137–147. http://dx.doi.org/10.1037/adb0000247

Hussong, A. M., Jones, D. J., Stein, G. L., Baucom, D. H., & Boeding, S. (2011). An internalizing pathway to alcohol use and disorder. *Psychology of Addictive Behaviors, 25,* 390–404. http://dx.doi.org/10.1037/a0024519

Jenkins, M. B., Agrawal, A., Lynskey, M. T., Nelson, E. C., Madden, P. A., Bucholz, K. K., & Heath, A. C. (2011). Correlates of alcohol abuse/dependence in early-onset alcohol-using women. *American Journal on Addictions, 20,* 429–434. http://dx.doi.org/10.1111/j.1521-0391.2011.00151.x

Jessor, R. (1991). Risk behavior in adolescence: A psychosocial framework for understanding and action. *Journal of Adolescent Health, 12,* 597–605. http://dx.doi.org/10.1016/1054-139X(91)90007-K

Johnson, P. B., Richter, L., Kleber, H. D., McLellan, A. T., & Carise, D. (2005). Telescoping of drinking-related behaviors: Gender, racial/ethnic, and age comparisons. *Substance Use & Misuse, 40,* 1139–1151. http://dx.doi.org/10.1081/JA-200042281

Johnston, L. D., O'Malley, P. M., Miech, R. A., Bachman, J. G., & Schulenberg, J. E. (2017). *Monitoring the Future national survey results on drug use, 1975–2016: Overview, key findings on adolescent drug use*. Ann Arbor: Institute for Social Research, The University of Michigan.

Kann, L., McManus, T., Harris, W. A., Shanklin, S. L., Flint, K. H., Hawkins, J., . . . Zaza, S. (2016). Youth Risk Behavior Surveillance—United States, 2015. *MMWR Surveillance Summaries, 65,* 1–174. http://dx.doi.org/10.15585/mmwr.ss6506a1

Keough, M. T., Badawi, G., Nitka, D., O'Connor, R. M., & Stewart, S. H. (2016). Impulsivity increases risk for coping-motivated drinking in undergraduates with elevated social anxiety. *Personality and Individual Differences, 88,* 45–50. http://dx.doi.org/10.1016/j.paid.2015.08.036

Kessler, R. C., Adler, L., Barkley, R., Biederman, J., Conners, C. K., Demler, O., . . . Zaslavsky, A. M. (2006). The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. *American Journal of Psychiatry, 163,* 716–723. http://dx.doi.org/10.1176/ajp.2006.163.4.716

Khantzian, E. J. (1985). The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. *American Journal of Psychiatry, 142,* 1259–1264. http://dx.doi.org/10.1176/ajp.142.11.1259

Kudielka, B. M., Hellhammer, D. H., & Kirschbaum, C. (2007). Ten years of research with the Trier Social Stress Test—Revisited. In E. Harmon-Jones & P. Winkielman (Eds.), *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 56–83). New York, NY: Guilford Press.

Kuntsche, E., Knibbe, R., Engels, R., & Gmel, G. (2007). Drinking motives as mediators of the link between alcohol expectancies and alcohol use among adolescents. *Journal of Studies on Alcohol and Drugs, 68,* 76–85. http://dx.doi.org/10.15288/jsad.2007.68.76

Lejuez, C. W., Aklin, W. M., Zvolensky, M. J., & Pedulla, C. M. (2003). Evaluation of the balloon analogue risk task (BART) as a predictor of adolescent real-world risk-taking behaviours. *Journal of Adolescence, 26,* 475–479. http://dx.doi.org/10.1016/S0140-1971(03)00036-8

Lejuez, C. W., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., . . . Brown, R. A. (2002). Evaluation of a behavioral measure of risk taking: The balloon analogue risk task (BART). *Journal of Experimental Psychology: Applied, 8,* 75–84. http://dx.doi.org/10.1037/1076-898X.8.2.75

Magrys, S. A., & Olmstead, M. C. (2015). Acute stress increases voluntary consumption of alcohol in undergraduates. *Alcohol and Alcoholism, 50,* 213–218. http://dx.doi.org/10.1093/alcalc/agu101

Marmorstein, N. R., White, H., Chung, T., Hipwell, A., Stouthamer-Loeber, M., & Loeber, R. (2010). Associations between first use of substances and change in internalizing symptoms among girls: Differences by symptom trajectory and substance use type. *Journal of Clinical Child and Adolescent Psychology, 39,* 545–558. http://dx.doi.org/10.1080/15374416.2010.486325

Medina, K. L., McQueeny, T., Nagel, B. J., Hanson, K. L., Schweinsburg, A. D., & Tapert, S. F. (2008). Prefrontal cortex volumes in adolescents with alcohol use disorders: Unique gender effects. *Alcoholism: Clinical and Experimental Research, 32,* 386–394. http://dx.doi.org/10.1111/j.1530-0277.2007.00602.x

Merikangas, K. R., He, J. P., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., . . . Swendsen, J. (2010). Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Survey Replication—Adolescent Supplement (NCS-A). *Journal of the American Academy of Child & Adolescent Psychiatry, 49,* 980–989. http://dx.doi.org/10.1016/j.jaac.2010.05.017

Miech, R. A., Johnston, L. D., O'Malley, P. M., Bachman, J. G., Schulenberg, J. E., & Patrick, M. E. (2017). *Monitoring the Future national survey results on drug use, 1975–2016: Vol. I. Secondary school students*. Ann Arbor: Institute for Social Research, The University of Michigan.

Miller, T. R., Levy, D. T., Spicer, R. S., & Taylor, D. M. (2006). Societal costs of underage drinking. *Journal of Studies on Alcohol, 67,* 519–528. http://dx.doi.org/10.15288/jsa.2006.67.519

Moberg, D. P. (2000). *The Adolescent Alcohol and Drug Involvement Scale*. Retrieved from https://psycentre.apps01.yorku.ca/wp/adolescent-alcohol-and-drug-involvement-scale-aadis-and-adolescent-drug-involvement-scale-adis/

Moberg, D. P., & Hahn, L. (1991). The Adolescent Drug Involvement Scale. *Journal of Adolescent Chemical Dependency, 2,* 75–88.

Moss, H. B., Chen, C. M., & Yi, H.-y. (2014). Early adolescent patterns of alcohol, cigarettes, and marijuana polysubstance use and young adult substance use outcomes in a nationally representative sample. *Drug and Alcohol Dependence, 136,* 51–62. http://dx.doi.org/10.1016/j.drugalcdep.2013.12.011

Muthén, B., & Masyn, K. (2005). Discrete-time survival mixture analysis. *Journal of Educational and Behavioral Statistics, 30,* 27–58. http://dx.doi.org/10.3102/10769986030001027

Nesic, J., & Duka, T. (2006). Gender specific effects of a mild stressor on alcohol cue reactivity in heavy social drinkers. *Pharmacology, Biochemistry and Behavior, 83,* 239–248. http://dx.doi.org/10.1016/j.pbb.2006.02.006

Nolen-Hoeksema, S., & Hilt, L. (2006). Possible contributors to the gender differences in alcohol use and problems. *Journal of General Psychology, 133,* 357–374. http://dx.doi.org/10.3200/GENP.133.4.357-374

Ollendick, T. H., & Hirshfeld-Becker, D. R. (2002). The developmental psychopathology of social anxiety disorder. *Biological Psychiatry, 51,* 44–58. http://dx.doi.org/10.1016/S0006-3223(01)01305-1

Perou, R., Bitsko, R. H., Blumberg, S. J., Pastor, P., Ghandour, R. M., Gfroerer, J. C., . . . Huang, L. N. (2013). Mental health surveillance among children—United States, 2005–2011. *MMWR Surveillance Summaries, 62*(Suppl 2), 1–35.

Sandler, I., Wolchik, S. A., Cruden, G., Mahrer, N. E., Ahn, S., Brincks, A., & Brown, C. H. (2014). Overview of meta-analyses of the prevention of mental health, substance use, and conduct problems. *Annual Review of Clinical Psychology, 10,* 243–273. http://dx.doi.org/10.1146/annurev-clinpsy-050212-185524

Savage, J. E., Kaprio, J., Korhonen, T., Pulkkinen, L., Rose, R. J., Verhulst, B., & Dick, D. M. (2016). The effects of social anxiety on alcohol and cigarette use across adolescence: Results from a longitudinal twin study in Finland. *Psychology of Addictive Behaviors, 30,* 462–474. http://dx.doi.org/10.1037/adb0000183

Schulte, M. T., Ramo, D., & Brown, S. A. (2009). Gender differences in factors influencing alcohol use and drinking progression among adolescents. *Clinical Psychology Review, 29,* 535–547. http://dx.doi.org/10.1016/j.cpr.2009.06.003

Singer, J. D., & Willett, J. B. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. http://dx.doi.org/10.1093/acprof:oso/9780195152968.001.0001

Smit, K., Voogt, C., Hiemstra, M., Kleinjan, M., Otten, R., & Kuntsche, E. (2018). Development of alcohol expectancies and early alcohol use in children and adolescents: A systematic review. *Clinical Psychology Review, 60,* 136–146. http://dx.doi.org/10.1016/j.cpr.2018.02.002

Starcke, K., van Holst, R. J., van den Brink, W., Veltman, D. J., & Goudriaan, A. E. (2013). Physiological and endocrine reactions to psychosocial stress in alcohol use disorders: Duration of abstinence matters. *Alcoholism: Clinical and Experimental Research, 37,* 1343–1350. http://dx.doi.org/10.1111/acer.12103

Steinberg, L. (2007). Risk taking in adolescence: New perspectives from brain and behavioral science. *Current Directions in Psychological Science, 16,* 55–59. http://dx.doi.org/10.1111/j.1467-8721.2007.00475.x

Stockwell, T., McLeod, R., Stevens, M., Phillips, M., Webb, M., & Jelinek, G. (2002). Alcohol consumption, setting, gender and activity as predictors of injury: A population-based case-control study. *Journal of Studies on Alcohol, 63,* 372–379. http://dx.doi.org/10.15288/jsa.2002.63.372

Stroud, L. R., Tanofsky-Kraff, M., Wilfley, D. E., & Salovey, P. (2000). The Yale Interpersonal Stressor (YIPS): Affective, physiological, and behavioral responses to a novel interpersonal rejection paradigm. *Annals of Behavioral Medicine, 22,* 204–213. http://dx.doi.org/10.1007/BF02895115

Tomlinson, K. L., & Brown, S. A. (2012). Self-medication or social learning? A comparison of models to predict early adolescent drinking. *Addictive Behaviors, 37,* 179–186. http://dx.doi.org/10.1016/j.addbeh.2011.09.016

U.S. Census Bureau. (2016). *QuickFacts: Denton County, Texas: All facts*. Retrieved from https://www.census.gov/quickfacts/fact/table/dentoncountytexas/PST045216

van den Bos, E., de Rooij, M., Miers, A. C., Bokhorst, C. L., & Westenberg, P. M. (2014). Adolescents' increasing stress response to social evaluation: Pubertal effects on cortisol and alpha-amylase during public speaking. *Child Development, 85,* 220–236. http://dx.doi.org/10.1111/cdev.12118

Westenberg, P. M., Drewes, M. J., Goedhart, A. W., Siebelink, B. M., & Treffers, P. D. (2004). A developmental analysis of self-reported fears in late childhood through mid-adolescence: Social-evaluative fears on the rise? *Journal of Child Psychology and Psychiatry, 45,* 481–495. http://dx.doi.org/10.1111/j.1469-7610.2004.00239.x

Williams, K. D., & Jarvis, B. (2006). Cyberball: A program for use in research on interpersonal ostracism and acceptance. *Behavior Research Methods, 38,* 174–180. http://dx.doi.org/10.3758/BF03192765

Wittchen, H. U., Behrendt, S., Höfler, M., Perkonigg, A., Lieb, R., Bühringer, G., & Beesdo, K. (2008). What are the high risk periods for incident substance use and transitions to abuse and dependence? Implications for early intervention and prevention. *International Journal of Methods in Psychiatric Research, 17*(Suppl 1), S16–S29. http://dx.doi.org/10.1002/mpr.254

Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition*. Stanford, CA: Stanford University Press.

Yim, I. S., Quas, J. A., Cahill, L., & Hayakawa, C. M. (2010). Children's and adults' salivary cortisol responses to an identical psychosocial laboratory stressor. *Psychoneuroendocrinology, 35,* 241–248. http://dx.doi.org/10.1016/j.psyneuen.2009.06.014

Zvolensky, M. J., Lejuez, C. W., Stuart, G. L., & Curtin, J. J. (2001). Experimental psychopathology in psychological science. *Review of General Psychology, 5,* 371–381. http://dx.doi.org/10.1037/1089-2680.5.4.371

Received March 28, 2018
Revision received January 23, 2019
Accepted January 24, 2019 ■