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Stress Reactivity, Social Anxiety, and Alcohol Consumption in People With Alcoholism: A Laboratory Study

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Abstract

Objective—Social anxiety may maintain alcohol dependence through increased reactivity to stressful events, a propensity to drink to cope with stressful events, or both. The current study is a secondary analysis of an existing dataset that examined differences between individuals with alcohol dependence and concurrent high and low social anxiety in objective and subjective stress reactivity to a laboratory stressor (Trier Social Stress Test; TSST), as well as consumption of alcohol following the stressor.

Methods—Forty participants with alcohol dependence (20 women) were randomly assigned to the TSST condition as part of the parent study. Post-hoc analysis of social anxiety measures yielded high ($n = 19$) and low ($n = 21$) social anxiety groups. Participants completed the TSST, followed by a small dose of their favorite alcoholic beverage (target blood alcohol concentration 0.03g/dl) to prime subsequent laboratory drinking. Participants received a sham beer taste test of two glasses (710ml total) of beer. Differences between high and low social anxiety groups was assessed via subjective and objective (mean arterial pressure, serum cortisol) reactivity to the TSST and consumption of alcohol during the taste test (total mls consumed, mls/kg of body weight, and likelihood of consuming all the beer available).

Results—No differences emerged in either objective or subjective measures of stress reactivity between high and low socially anxious groups. There were also no differences between social anxiety groups in amount of alcohol consumed during the taste test.

Conclusions—No differences were observed between high and low socially anxious participants with concurrent alcohol dependence on stress reactivity or alcohol consumption following a stressor. Given that all participants in this study had alcohol dependence, negative results may suggest that heightened stress reactivity and drinking to cope are more relevant to the development of alcohol dependence and that other factors may maintain alcohol use once dependence has developed.

Keywords

social anxiety; alcohol dependence; stress reactivity

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DISCLOSURES

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Social anxiety disorder is characterized by intense fear of social evaluation and scrutiny and commonly co-occurs with alcohol dependence (Grant et al., 2005). It is one of the few co-occurring disorders that almost always onsets prior to alcohol dependence (Falk, Yi, & Hilton, 2008). Evidence suggests that drinking to cope with anxiety or stress is one pathway by which individuals with social anxiety disorder may be more susceptible to the development of alcohol use problems than those without social anxiety (Buckner & Heimberg, 2010). Socially anxious individuals drink to cope more often than individuals without social anxiety (Thomas, Randall, & Carrigan, 2003), and may do so because they are more sensitive to the negative effects of stressors, at least regarding subjective interpretation (Levin, Saoud, Strauman, & Gorman, 1993). Whether individuals with and without social anxiety differ on other measures of response to a stressor (e.g., heart rate, cortisol) is unclear (Condren et al., 2002; Furlan et al., 2001; Levin et al., 1993; Roelofs et al., 2009; Shirotaki et al., 2009).

Though drinking to cope with social anxiety has been primarily examined as a mechanism underlying the development of alcohol dependence, it may continue to play a role in disordered drinking once dependence has occurred. Drinking to cope behaviors are frequently endorsed among individuals with comorbid anxiety and substance use diagnoses and are associated with a two-fold increase in likelihood of carrying the substance use disorder two years later, compared to those who did not report using substances to cope (Robinson, Sareen, Cox, & Bolton, 2011). These findings are in line with a mutual maintenance model of anxiety and substance use comorbidity, which suggests that the two disorders engage in a positive feedback loop: anxiety making substance use more likely through the practice of drinking to cope and continued substance use adding to existing stressful situations and anxiety (Stewart & Conrod, 2008). It is unclear whether heightened sensitivity to stress among those with comorbid anxiety and alcohol dependence may play a role, as few studies have examined stress reactivity among people with comorbid anxiety and alcohol dependence (Brady et al., 2006).

Taken together, it is tempting to speculate that social anxiety may not only be a risk factor in the development of alcohol dependence, but may also help to maintain dependence once it has developed - by affecting one's response to a stressor, promoting the practice of drinking in response to stress, or both.

Though prospective, longitudinal studies are needed to examine this question conclusively, a more timely and less expensive approach could be to compare individuals with alcohol dependence with and without marked social anxiety using a clinical laboratory setting to examine how they respond to a stressor and the opportunity to drink afterwards. The present study is a secondary analysis of an existing dataset from a laboratory study using a psychosocial stressor, the Trier Social Stress Task, (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), which has been shown to induce a stress response in participants with alcohol dependence (Thomas, Randall, Brady, See, & Drobles, 2011a) and social anxiety (Roelofs et al., 2009).

Those with clinically relevant social anxiety (as determined by a validated self-report instrument) were hypothesized to experience greater subjective and objective (cortisol and mean arterial blood pressure) distress in response to the stressor and greater subsequent alcohol consumption compared to participants with low levels of social anxiety.

METHODS

Participants

A full description of study methods has been previously published (Thomas, Bacon, Randall, Brady, & See, 2011b). Non-treatment-seeking participants with alcohol dependence were recruited via community advertisements and screened via telephone for initial eligibility requirements (quantity/frequency of alcohol use; dependence symptoms). Those meeting pre-eligibility requirements were invited to an in-person assessment visit.

Informed consent and assessment—Upon arrival, participants provided written informed consent. The informed consent document—approved by the university's institutional review board and in accordance with the Declaration of Helsinki—fully explained the procedures of the study. Participants were deceived regarding the purpose of the study (they were told the study was to examine how personality affects one's ability to detect subtle differences in alcoholic drinks) to reduce demand effects regarding the stress induction procedure and alcohol consumption.

Consenting individuals completed a battery of self-report measures, including measures assessing dependence symptoms (Alcohol Dependence Scale, ADS; Skinner & Allen, 1982), motives for drinking (Drinking Motives Questionnaire, DMQ; Cooper, 1994), depressive symptoms (Beck Depression Inventory-II, BDI; Beck et al., 1996), trait anxiety (State/Trait Anxiety Inventory, STAI; Spielberger, 1983), anxiety sensitivity (Anxiety Sensitivity Index, AnxSI; Reiss et al., 1986), and social anxiety symptoms (Social Phobia Inventory, SPIN; Connor et al., 2000). They then completed a structured clinical interview, including the MINI (Sheehan et al., 1998) to assess for Axis I disorders and the Timeline Followback (Sobell & Sobell, 1992) to determine quantity and frequency of alcohol use. Participants were eligible for the challenge visit if they: (1) met diagnostic criteria for alcohol dependence, (2) consumed at least 14 (women) or 20 (men) drinks per week, (3) were not taking any medications known to affect the hypothalamic pituitary axis, (4) had a body mass index <35, and (5) had not received prior medical alcohol detoxification. The requirements of the parent study excluded individuals with other current Axis I disorders, including both current dependence on other drugs (excepting nicotine) and social anxiety disorder. However, none of the individuals excluded on this basis ($n = 7$) did so as a result of meeting full diagnostic criteria for social anxiety disorder.

Eligible participants were then scheduled for the challenge visit within no more than 60 days. A total of 79 (40 women) completed the challenge. The present paper examines the data from the 40 participants (20 women) randomized to the stress condition, as the focus is on comparing those with and without high social anxiety on response to the stressor.

Social anxiety groups—A score of 16 on the SPIN differentiates individuals with social anxiety from individuals without social anxiety who were treated for another psychiatric disorder (Connor et al., 2000). Individuals above and below this cutoff were distinguished as high ($n=19$) and low ($n=21$) social anxiety groups following completion of the study procedures and prior to analyses.

Laboratory Challenge

Upon arrival to the laboratory, participants were fitted with an indwelling catheter in order to obtain blood samples used to measure cortisol.

Stress manipulation—The TSST (Kirschbaum et al., 1993) is an acute psychological stressor considered the gold standard for eliciting an hypothalamic pituitary axis stress

response in a laboratory setting (Dickerson & Kemeny, 2004). Participants were tested singly. They were informed at 5:00 pm that they would soon complete a behavioral personality test in the presence of three individuals who would be assessing body language and behavior. After a five-minute preparation period, participants performed a 5-minute speech, then a 5-minute serial subtraction task before audience members wearing white lab coats.

Taste test—Five minutes following the TSST, participants were provided with a priming dose of their preferred alcoholic beverage with the goal of achieving a target blood alcohol content (BAC) of 0.03g/dl. This BAC level has been demonstrated to induce subsequent drinking in participants with alcohol dependence in clinical laboratory settings, which may not otherwise be conducive to drinking (Anton et al., 2004). Participants were given ten minutes to consume the beverage and 15 minutes for alcohol absorption, before beginning the taste test task.

The taste test procedure is a covert way of assessing alcohol consumption without demand characteristics (Caudill & Marlatt, 1975; Higgins & Marlatt, 1973). At 5:45 pm, participants were provided two 355ml glasses of beer and were instructed that they had 15 minutes to taste from each glass in order to determine if the glasses contained identical or different kinds of beer. Participants were informed that they could consume whatever amount necessary to make an accurate decision. After explaining the procedure, the research assistant left the room for the duration of the task and returned to collect the glasses and rating sheet.

The two glasses contained the same beverage: a mixture of equal parts Budweiser and O'Douls non-alcoholic beer. Non-alcoholic beer was provided to minimize intoxication in those consuming all the beer available (710ml or 24oz).

At the end of the challenge (7:15 pm), participants were debriefed regarding the rationale for the study and study procedures and compensated \$200. Once their BAC levels dropped below 0.03 g/dl, participants were permitted to leave by taxi.

Laboratory Assessment

Stress reactivity—Stress reactivity was determined by subjective rating of distress, serum cortisol (ug/dl), and mean arterial pressure (mm/Hg). Subjective distress was measured on a 10-point scale, from 1 (not at all distressed) to 10 (severely distressed). Blood samples for cortisol were collected in iced EDTA tubes and assayed using the ADVIA Centaur XP immunoassay system (Siemens Healthcare Diagnostics, Flanders, NJ).

All stress reactivity measures were collected nine times, twice pre-stressor (20 minutes and 5 minutes prior) and seven times post-stressor (at 0, 15, 45, 60, 75, 90, and 105 minutes). For the purposes of the current analyses, the two baseline measures (BL1 and BL2) and the first four post-stressor indices are reported (see Figure 1).

Alcohol consumption—Alcohol consumption during the taste test was measured with three indices: amount of beer consumed (in total mls and mls/kg body weight) and probability of drinking all the beer provided.

Data Analytic Plan

Demographics, pre-challenge alcohol use, and psychological assessment descriptive measures were analyzed with one-way (social anxiety group: high, low) analysis of variance or chi-square tests for dichotomous variables.

Stress reactivity measures (cortisol, mean arterial pressure, and subjective distress) were analyzed in a series of repeated-measures analysis of variance, with time (pre-, post-TSST) as the within-subjects variable and social anxiety group (high, low) as the between-subjects variable. The time by social anxiety group interaction was used to assess for differences in stress reactivity to the TSST between the high and low social anxiety groups. For cortisol, the reading 15 minutes after the completion of the stress task was used to index peak cortisol response (Kirschbaum et al., 1993). Mean arterial pressure and subjective response measures taken immediately after the TSST were used as dependent variables in those analyses.

For alcohol consumption outcomes, amount of beer consumed in mls and mls/kg were analyzed using analyses of variance, with social anxiety group as the between-subjects variable. Probability of drinking the total amount of beer was analyzed with a logistic regression. The relation between stress reactivity measures and alcohol consumption was assessed via bivariate correlations. Unadjusted means are reported unless otherwise noted.

RESULTS

Participants

Demographics, alcohol use, and psychological descriptive measures are detailed in Table 1. One female participant in the high social anxiety group reported alcohol consumption in the month prior to participation that was greater than 1.5 times the interquartile range. Her drinking data were removed from analyses. As expected, mean scores on the Social Phobia Inventory differed significantly between high ($M=26.3$, $SD=11.1$) and low ($M=8.24$, $SD=4.3$) social anxiety groups, $F(1, 38) = 47.52$, $p < .001$. The high social anxiety group had higher scores on measures of general anxiety, including trait anxiety, $F(1, 38) = 13.64$, $p = .001$, and anxiety sensitivity, $F(1, 38) = 25.05$, $p < .001$. As compared to the low social anxiety group, the high social anxiety group also had greater severity of depressive symptoms, $F(1, 38) = 8.60$, $p = .006$, drinking to cope with negative affect, $F(1, 38) = 8.51$, $p = .006$, and severity of alcohol dependence, $F(1, 38) = 9.73$, $p = .003$. These measures were examined as potential covariates in analyses of stress reactivity, but dropped if their inclusion did not alter results.

The high and low social anxiety groups did not differ on demographic variables except for greater representation by Caucasian participants in the low social anxiety group ($\chi^2 = 4.27$, $p = .04$). On average, participants consumed 11 standardized drinks per drinking day ($SD = 5$) and 56 standardized drinks per week ($SD = 35$).

Stress Reactivity to the TSST

The TSST proved to be an effective method of inducing stress. Analyses from the parent study indicated significant differences in subjective distress, cortisol, and mean arterial pressure between participants who completed the TSST compared to those in a control condition (Thomas et al., 2011b).

High and low social anxiety groups showed similar baseline levels for both cortisol, $F(1, 38) < 1$, $p = .97$, and mean arterial pressure, $F(1, 38) < 1$, $p = .93$. Those in the high social anxiety group reported greater overall baseline subjective distress ($M = 3.4$, $SD = 2.5$) than those in the low social anxiety group ($M = 1.3$, $SD = 1.4$), $F(1, 38) = 10.77$, $p = .002$.

Objective stress reactivity—Inclusion of covariates in the model did not affect the result that there was no significant time by social anxiety group interaction on cortisol response to the TSST, $F(1, 37) < 1$, $p = .50$, $\eta_p^2 = .01$ (Figure 1a). The time by social anxiety group interaction for mean arterial pressure was also not significant, $F(1, 77) < 1$, p

$= .40$, $\eta_p^2 = .01$ (Figure 1b). For mean arterial pressure there were significant main effects for the covariates of gender, $F(1, 36) = 11.78$, $p = .002$, $\eta_p^2 = .25$, and trait anxiety, $F(1, 36) = 4.52$, $p = .04$, $\eta_p^2 = .11$, though inclusion of these variables in the model did not alter results.

Subjective stress reactivity—On subjective stress reactivity, there was a significant main effect for social anxiety group such that the high social anxiety group ($M = 4.71$, $SE = 0.45$) reported higher subjective distress overall compared to the low anxiety group ($M = 2.98$, $SE = 0.43$), $F(1, 38) = 7.92$, $p = .01$, $\eta_p^2 = .17$. There was also a significant main effect for the covariate of severity of alcohol dependence ($F(1, 32) = 4.15$, $p = .05$, $\eta_p^2 = .12$), but its inclusion did not alter the result that there was no significant time by social anxiety group interaction for subjective distress, $F(1, 38) < 1$, $p = .40$, $\eta_p^2 = .02$ (Figure 1c).

Alcohol Consumption

Priming drink—The priming dose effectively achieved the target BAC of 0.03 g/dL, with no significant difference between the high ($M = .030$, $SD = .009$) and low ($M = .025$, $SD = .010$) social anxiety groups, $F(1, 38) = 2.50$, $p = .12$, $\eta_p^2 = .06$.

Correlates with voluntary alcohol consumption—For the consumption of alcohol in the taste task, there was a significant correlation with gender and amount of beer consumed (Spearman's $\rho = .42$, $p = .007$), and probability of consuming all the beer available (Spearman's $\rho = .44$, $p = .005$). There was a trend for a positive correlation between drinks per week and total amount of beer consumed ($r = .31$, $p = .05$). Gender and drinks per week were included in analyses as covariates in the alcohol consumption analyses, and were removed if their inclusion did not account for significant variance in the dependent variable.

Amount of beer consumed (mls and mls/kg)—After controlling for the significant effect of gender ($F(1,37) = 8.63$, $p = .006$, $\eta_p^2 = .19$) there was not a significant difference in mls of alcohol consumed between high ($M_{adjusted} = 517\text{ml}$, $SE = 44$) and low ($M_{adjusted} = 466\text{ml}$, $SE = 42$) social anxiety groups, $F(1, 37) < 1$, $p = .42$, $\eta_p^2 = .02$.

For amount of beer consumed as a function of body weight, covariates did not account for a significant portion of the variance in the dependent variable. There was no significant difference in mls of beer consumed as a function of body weight between high ($M = 5.92$, $SE = 0.56$) and low ($M = 6.10$, $SE = 0.54$) social anxiety groups, $F(1, 38) < 1$, $p = .82$, $\eta_p^2 = .001$.

Probability of consuming all the beer provided—The covariates of gender ($p = .01$) and drinks per week ($p = .02$) each independently predicted (and together predicted) whether participants would drink all the beer provided; $\chi^2(2) = 13.22$, $p = .001$. Half of the men (50%) vs. 10% of the women drank all the beer available. Those who drank all the beer had a greater number of drinks per week ($M = 76$, $SD = 51$) than those who did not drink all the beer available ($M = 47$, $SD = 20$). The addition of the social anxiety group variable failed to predict who would drink all the beer available; Wald $\chi^2(1) = 0.07$, $p = .80$. In the low social anxiety group 33% of participants consumed all the beer available vs. 26% in the high social anxiety group.

Association Between Stress Reactivity and Alcohol Consumption

To examine whether stress reactivity to the TSST was associated with voluntary alcohol consumption, alcohol consumption measures (mls, mls/kg, and whether the participant consumed all the beer provided) were examined for correlations with stress reactivity measures (percentage change from baseline for mean arterial pressure, cortisol and

subjective distress), using a partial correlation to control for the effects of gender. No corrections were used to adjust for inflated type I error rate for this exploration. No significant relationships emerged either for the overall sample or in the high social anxiety group. In the low social anxiety group, there were significant positive correlations between percentage change in mean arterial pressure and all alcohol consumption measures (r 's .49 to .56, p 's < .05), reflecting that individuals with low social anxiety and with greater blood pressure changes to the stressor consumed higher amounts of alcohol.

DISCUSSION

Using a clinical laboratory paradigm, the present study examined whether individuals with high vs. low levels of social anxiety and co-occurring alcohol dependence differed in their reaction to a psychosocial stressor and/or alcohol consumption following stress induction. The extant literature suggests that social anxiety may enhance problematic alcohol consumption in individuals with alcohol dependence, either by increasing post-stress alcohol consumption (Buckner & Heimberg, 2010; Robinson et al., 2011; Thomas et al., 2003), by making stressors more aversive (Condren et al., 2002; Levin et al., 1993; Roelofs et al., 2009), or both. Our results did not show that individuals with both alcohol dependence and high social anxiety had different stress reactivity or alcohol consumption following a stressor than those with alcohol dependence and low social anxiety. It is important to clarify that these negative results do not necessarily indicate that differences in stress reactivity or propensity to drink following stress induction are irrelevant risk factors. They may, in fact, markedly contribute to the *development* of alcohol use problems among socially anxious individuals, but not in *maintaining* problematic alcohol use once dependence develops.

The present study used a validated stressor that induces a stress response (cortisol, mean arterial pressure and subjective distress) in individuals with alcohol dependence (Thomas et al., 2011b), so the negative results are not due to lack of internal validity of the manipulation. Additionally, though the present study was not designed and powered to specifically examine differences between high and low social anxiety groups, the means and variability for the high and low social anxiety groups are virtually identical across all stress reactivity measures (as seen in Figure 1a–c). This similarity suggests that the negative effects were not due to inadequate power to detect group differences. A more likely explanation is that individuals with alcohol dependence have a dampened stress response (Errico et al., 1993; Panknin et al., 2002), which may have negated the heightened stress response found in those with high social anxiety (Condren et al., 2002; Levin et al., 1993; Roelofs et al., 2009). While this study was the first to examine stress response differences between those with comorbid social anxiety and alcohol dependence compared to alcohol dependence only, other researchers have also found no differences in stress response between comorbid posttraumatic stress disorder and alcohol dependence compared to either disorder independently (Brady et al., 2006). More investigation of stress response among individuals with both social anxiety and alcohol dependence is warranted.

The present study also found no significant differences between individuals with alcohol dependence and high versus low social anxiety in amount of alcohol consumed following the stressor. Though contrary to hypotheses, these results do align with results from treatment studies which find that reducing social anxiety in those with alcohol dependence via medication (Thomas, Randall, Book, & Randall, 2008) or psychotherapy (Randall, Thomas, & Thevos, 2001) did not decrease drinking. The present study found that individuals with alcohol dependence and high social anxiety did not show greater post-stressor drinking than their low socially anxious counterparts, and stress reactivity was not associated with higher quantity of post-stress drinking in socially anxious participants. In total, studies seem to consistently show that once dependence develops, social anxiety does

not appear to be the primary driver of drinking in those with concurrent alcohol dependence and high social anxiety.

The study is strengthened by the convergence of findings across objective and subjective measures of stress reactivity in a well-validated stress induction paradigm, and controlled assessment of alcohol consumption in the laboratory. There are, however, notable limitations. The present study is an analysis of an existing study designed to examine whether stress induces drinking in individuals with alcohol dependence (Thomas et al., 2011b) and was not specifically conducted to examine the role of social anxiety. Thus, high and low social anxiety groups were determined retrospectively using responses to a self-report questionnaire and a validated threshold for identifying the presence of clinically relevant social anxiety (Connor et al., 2000). Though responses on related measures of anxiety supported that the differences between the social anxiety groups represented valid differences in social anxiety, future studies should include participants with a diagnosis of social anxiety disorder determined through structured interview.

In summary, results of the present study indicate that the presence of social anxiety in non-treatment seeking individuals with alcohol dependence is not associated with altered objective or subjective stress reactivity to a psychosocial stressor or greater stress-induced alcohol consumption. Though counter to hypotheses, the results do integrate with the extant literature - that is, while social anxiety and drinking to cope with anxiety may be significant risk factors for the development of alcohol dependence (Buckner & Heimberg, 2010; Buckner et al., 2008; Thomas et al., 2003), social anxiety may play a lesser role in the maintenance of continued alcohol use in those who have become dependent.

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Figure 1. Stress reactivity responses to the TSST by social anxiety group across time. Note. TSST = Trier Social Stress Test; SA = Social Anxiety; BL1, BL2 = baseline 1 and 2 prior to stressor; PT1 – PT4 = post-stressor measurements at 15 minute intervals. Bars indicate standard error.

Table 1

Demographics, alcohol use, and psychological assessment variables by social anxiety group ($N=40$)

	Low Social Anxiety ($n = 21$)	High Social Anxiety ($n = 19$)	$\chi^2 / F, p\text{-value}$
Demographics			
Caucasian, n (%)	19 (91%)	12 (63%)	$\chi^2 = 4.27, p = .04$; Low > High
Age, M (SD)	34.90 (12.97)	34.42 (10.50)	$F(1, 38) = 0.02, p = .90$
Employed full-time, n (%)	11 (52%)	5 (26%)	$\chi^2 = 2.82, p = .09$
Married, n (%)	5 (24%)	1 (5%)	$\chi^2 = 2.69, p = .10$
Smoker, n (%)	8 (38%)	11 (58%)	$\chi^2 = 1.57, p = .21$
College graduate, n (%)	9 (43%)	4 (21%)	$\chi^2 = 2.16, p = .14$
Alcohol Use, M (SD)			
Drinks per week *	51.8 (23.9)	61.4 (44.4)	$F(1, 37) = 0.73, p = .40$
Drinks per drinking day *	9.9 (3.5)	11.3 (6.1)	$F(1, 37) = 0.85, p = .36$
Dependence severity, ADS [0–47]	12.0 (4.8)	18.1 (7.5)	$F(1, 38) = 9.73, p = .003$; High > Low
Age onset alcohol dependence **	25.5 (8.7)	23.0 (7.2)	$F(1, 36) = 0.91, p = .35$
Psychological assessment, M (SD)			
Trait anxiety, STAI [20–80]	37.4 (10.0)	50.1 (11.6)	$F(1, 38) = 13.64, p = .001$; High > Low
Anxiety sensitivity, AnxSI [0–64]	15.7 (7.7)	29.3 (9.5)	$F(1, 38) = 25.05, p < .001$; High > Low
Depressive symptoms, BDI [0–63]	9.1 (7.6)	17.3 (10.1)	$F(1, 38) = 8.60, p = .006$; High > Low
Drinks to cope, DMQ [0–4]	2.2 (0.7)	2.8 (0.6)	$F(1, 38) = 8.51, p = .006$; High > Low

Note. ADS = Alcohol Dependence Scale; SPIN = Social Phobia Inventory; STAI = State/Trait Anxiety Inventory; AnxSI = Anxiety Sensitivity Index; BDI = Beck Depression Inventory; DMQ = Drinking Motives Questionnaire; ns = not significant.

* One participant in the high social anxiety group reported baseline drinking greater than 1.5 times the interquartile range; her data was removed from drinking analyses

** Data missing for one male in the low social anxiety group and one female in the high social anxiety group.