Effects of Drink-Stress Sequence and Gender on Alcohol Stress Response Dampening in High and Low Anxiety Sensitive Drinkers

Martin Zack, Constantine X. Poulos, V. Bess Aramakis, Baljit K. Khamba, and Colin M. MacLeod

Background: This study tested the appraisal disruption hypothesis of alcohol stress response dampening (SRD) in male and female high or low anxiety sensitive (AS) undergraduates. The hypothesis predicts that alcohol SRD will be greater when drinking occurs before versus after stress exposure. High AS males' predominant social-evaluative concerns further implied that alcohol SRD to a social stressor (i.e., a speech) would be relatively stronger in high AS males than in high AS females.

Methods: Male and female ($n = 90$/gender) high and low AS participants ($\geq$70th; $\leq$30th percentile on Anxiety Sensitivity Index-Revised) were matched on drinking habits and randomly assigned to 1 of 9 experimental cells. Drink type—alcohol (0.7 g/kg males; 0.63 g/kg females), placebo, soda—was fully crossed with stress condition—drink before stress (DBS), drink after stress (DAS), and no stress control (NSC). Stress was induced by telling participants they would be required to make a self-revealing speech. Stress response dampening was assessed for state anxiety on the Spielberger scale and Stroop interference to threat-related words. Subjective desire for alcohol was also assessed.

Results: Relative to placebo, alcohol (peak blood alcohol concentration, 0.064%) reliably reduced state anxiety in high AS but not in low AS participants. Alcohol decreased STAI scores and Stroop interference to social threat words significantly more in the DBS than the DAS condition in high AS males; high AS females displayed the exact opposite pattern of effects. In contrast to other participants, high AS males also reported relatively strong desire scores under alcohol.

Conclusions: Overall, the results do not support the appraisal disruption hypothesis as a general mechanism of alcohol SRD in undergraduate drinkers. The findings for high AS males do support the hypothesis, while the opposing profile for high AS females implies that the nature of the stressor (i.e., social challenge) may contribute to gender differences in alcohol SRD in high AS individuals.

Key Words: Appraisal Disruption, Stress, Anxiety Sensitivity, Gender.

ALCOHOL IS WIDELY believed to alleviate stress. However, experimental research has yielded inconsistent results, both in the magnitude and direction of alcohol's effects (Pohorecky, 1991). Several trait and state factors have been investigated in an effort to explain this variability, and the findings indicate that both types of factors moderate alcohol's effect on stress, i.e., "stress response dampening" (SRD; e.g., Kushner et al., 1996; Sayette and Wilson, 1991; Sher and Levenson, 1982; Steele and Josephs, 1990).

Alcohol SRD is important, in part, because it may contribute to the development of alcoholism. Accordingly, traits associated with problem drinking are also associated with heightened alcohol SRD. Two such traits are a family history of alcoholism and anxiety sensitivity (AS; Pihl and Peterson, 1995). Studies with children of alcoholic individuals have found that, compared with individuals with no family history of alcoholism, family history-positive individuals experience greater alcohol SRD as measured by cardiovascular and endocrine responses and self-reported anxiety when faced with physical threat (e.g., electric shock) or social threat (e.g., speech; Croissant and Olbrich, 2004; Sinha et al., 1998; Stewart et al., 1992;

From the Clinical Neuroscience Section, Centre for Addiction and Mental Health (CAMH), Toronto, Ontario, Canada (MZ); the Department of Pharmacology, University of Toronto, Toronto, Ontario, Canada (MZ); the Department of Public Health Sciences, University of Toronto, Toronto, Ontario, Canada (MZ); the Department of Psychology, University of Toronto, Toronto, Ontario, Canada (CXP); the AstraZeneca Inc., Mississauga, Ontario, Canada (VBA); the Canadian College of Naturopathic Medicine, Toronto, Ontario, Canada (BKK); the Department of Psychology, University of Waterloo, Waterloo, Ontario, Canada (CMM).

Received for publication August 2, 2006; accepted November 19, 2006.

This research was funded by The Alcoholic Beverage Medical Research Foundation. We wish to thank Professors David Day, William Glassman, Marilyn Haddad, Brian Rabinowicz, and Isaac Smith for their invaluable assistance with recruitment.

Reprint requests: Martin Zack, PhD, CAMH, 33 Russell Street, Toronto, Ontario, Canada M5S 2S1; Fax: 416-595-6618; E-mail: martin_zack@camh.net

DOI: 10.1111/j.1530-0277.2006.00322.x

Zimmermann et al., 2004). No such differences are evident under placebo, indicating that the differential SRD effects were due to pharmacological factors rather than learned expectancies.

Other research has found a similar pattern of increased alcohol SRD in participants with high AS relative to their low AS peers. High AS is characterized by catastrophic responses to subtle or ambiguous symptoms of anxiety (e.g., trembling), and has been aptly described as ''fear of fear'' (Arrindell, 1993). High AS is also strongly linked with the development of disorders that frequently co-occur with alcoholism, like panic disorder and social phobia (Ball et al., 1995; McNally, 2002). The experimental evidence shows that high AS individuals display greater alcohol SRD during anticipation of shock or aversive noise as well as in response to stressors that induce panic states, such as hyperventilation (Conrod et al., 1998; Macdonald et al., 2000; Stewart and Pihl, 1994).

Of the various state moderators of alcohol SRD, 2 are central to theoretical models: distraction and drink-stress sequence. Steele and Josephs' (1990) attention allocation model asserts that alcohol restricts attention to the most salient cues, leading to alcohol SRD when immediate cues are benign but increasing stress responses in the absence of such distraction. A number of studies, using a variety of stressors and methodologies, support this model (e.g., Curtin et al., 1998, 2001; Josephs and Steele, 1990; Steele and Josephs, 1988).

Whereas Steele and Josephs emphasized the importance of distraction, Sayette's appraisal disruption model (Sayette, 1993) focuses on the timing of alcohol consumption with respect to stress. The model asserts that alcohol impairs the ability of a stimulus to activate its associates in memory, thereby reducing its emotional import. When alcohol consumption precedes exposure to a stressor, this will lead to an impoverished mental representation of the stressor and a corresponding decrease in stress responses. However, when drinking follows a stressor, alcohol is predicted to increase stress responses, relative to sober conditions, by impeding access to associates that are incompatible with stress.

An early study on appraisal disruption found that heart rate reactivity was greater when alcohol was consumed before versus after a stressor (advisory of a self-revealing speech) in a heterogeneous sample of social drinkers (Sayette and Wilson, 1991). More recently, Sayette and colleagues (2001) examined appraisal disruption using a modified Stroop task containing words related to the topic of the speech (aspects of physical appearance). These investigators found a complex interaction, in which Stroop interference to Appearance words declined under alcohol when drinking preceded stress, but only in males with a family history of alcoholism. These findings underscore the complex effects of state and trait factors on alcohol SRD.

The appraisal disruption model seems to be particularly relevant to high AS individuals, whose response to stress involves a heightened or distorted appraisal of their own reaction to threat. In addition, high AS individuals have been found to have a well-developed network of threat-relevant associations, as evidenced by Stroop interference to Social Threat words (e.g., ashamed; Stewart et al., 1998). If alcohol disturbs stress-induced activation of this network, intoxicated high AS individuals may resemble sober low AS individuals in interference to social threat words. Based on the literature, high AS participants may also display greater alcohol SRD in self-reported anxiety. If these effects conform to the appraisal disruption model, high AS participants should display greater alcohol SRD (relative to a placebo drink) when drinking comes before rather than after stress. If appraisal disruption contributes to the enhanced alcohol SRD effects of high AS drinkers, the effects of drink-stress sequence should be stronger in high AS than in low AS participants.

Previous research on alcohol SRD in high versus low AS drinkers has used physical stressors (e.g., anticipated shock, aversive noise). In contrast, research on appraisal disruption has used advisory of a self-revealing speech, a stressor that engages more social-evaluative concerns. This is an important distinction because social-evaluative concerns and drinking to manage these concerns have been found to characterize high AS males, whereas physical (i.e., hypochondriacal) concerns have been found to predominate in high AS females (Stewart et al., 1997, 2001). These findings suggest that gender may moderate the effects of the speech stressor in the present study. To the extent that alcohol dampens speech-induced social-evaluative concerns, alcohol SRD may be especially pronounced in high AS males.

In sum, the specific hypotheses were as follows: relative to placebo, (a) alcohol will reduce responses to stress; (b) alcohol SRD will be greater when drinking comes before versus after the stressor; (c) the effects of drink-stress sequence on alcohol SRD will be greater in high AS versus low AS participants; and (d) high AS males should exhibit more robust alcohol SRD than high AS females. The literature indicates that placebo effects do not account for alcohol SRD effects. Therefore, the placebo drink is not expected to lead to SRD, regardless of AS group or drink-stress sequence.

These hypotheses were tested in high AS and low AS male and female university undergraduates who received alcohol, a placebo drink, or soda before or after stress (advisory of a self-revealing speech). A no stress control (NSC) condition assessed reactivity to the experimental setting per se. In line with previous research, alcohol SRD was assessed for self-reported state anxiety and Stroop interference to Appearance and Social Threat words. The incentive-motivational effects of alcohol and drink-stress sequence were explored for self-reported desire for alcohol.

MATERIALS AND METHODS

Study Design

The study used a 2 (gender: male, female) ×2 (AS group: low AS, high AS) ×3 (drink type: alcohol, placebo, soda) ×3 [stress condition: NSC, drink before stress (DBS), drink after stress (DAS)] factorial design. There were 20 participants (5 low AS males; 5 low AS females; 5 high AS males; 5 high AS females) in each of the 9 experimental cells. Drink type was fully crossed with stress condition. Participants were matched on weekly alcohol use and randomly assigned to the 9 cells.

Participants

One hundred and eighty (90/gender) undergraduates were recruited from psychology classes (total $N = 1{,}464$) at Ryerson University in Toronto. The entire cohort completed the Anxiety Sensitivity Index-Revised (ASI; Peterson and Reiss, 1992) at the start of the semester, along with the drinking habits questionnaire (DHQ; Vogel-Sprott, 1992), a measure of typical alcohol use validated for undergraduate students. The DHQ assesses the mean frequency (occasions/week) dose (mL ethanol/kg), duration (hours), and rate (mL/kg/h) of alcohol consumption.

The cut-off for high AS was defined as the 70th percentile for each gender in the full sample; low AS was defined as the 30th percentile. These criteria conform to previous studies with this population (Zack et al., 2003, 2006), and are designed to maximize the group difference while permitting inclusion of enough participants to detect experimental effects reliably. The ASI cut-off values for the high and low AS groups were 27 and 19, respectively, for males, and 31 and 19 for females. Detailed participant characteristics are provided in ''Results.''

To qualify, participants must have had at least 1 alcoholic drink in the week preceding screening, and at least 3 drinks in one sitting in the preceding 90 days. All participants had normal or corrected-to-normal vision and were not color blind. Participants were advised that neither the screening nor test session were related to their academic standing and that they would receive $60 if they were invited to take part in the test session.

Apparatus

Stroop Task. The Stroop task was administered on a PC equipped with MicroExperimental Laboratories (v. 2.01; Psychology Software Tools, Pittsburgh, PA) and run entirely within MS-DOS. Participants faced the screen at a distance of ~60 cm and responded vocally to the stimuli. A microphone attached to the computer by a cable and positioned ~3 cm from the participant's mouth registered vocal response time (RT) to each stimulus with millisecond (ms) accuracy. Participants were instructed not to read the word stimuli but instead to name their color as quickly and accurately as possible. During the task, the experimenter coded response accuracy (correct response; error: incorrect color name or reading the word) after each trial using a button box (Psychology Software Tools, Pittsburgh, PA), also attached to the computer by cable. There were 2 procedurally identical versions of the task: a practice version that used neutral words only and familiarized participants with the task before the experimental manipulations, and a test version containing words from 4 motivationally relevant categories and 1 neutral control category. The categories in the test version were as follows: Physical Threat (e.g., dizzy), Social Threat (e.g., ashamed), Appearance related (e.g., ugly), Alcohol (e.g., vodka), and Neutral (e.g., river). Physical and Social Threat items were taken from Stewart et al. (1998). Appearance-related items were taken from Sayette et al. (2001). Alcohol and Neutral items were taken from a prior Stroop study with social drinkers (Sharma et al., 2001). There were no significant differences across the 5 categories in the mean length or frequency of occurrence in print, ensuring that these nonsemantic features did not account for any differences in RT.

The test version of the task included 20 warm-up trials with Neutral items only. Participants had an opportunity to ask questions before proceeding to the experimental trials. The sequence of events on every trial was the same: the stimulus (1 cm in height) appeared in 1 of 4 colors (red, yellow, blue, or green) in the center of the screen and remained visible until a vocal response was made. Following the response, the screen remained blank for 1,000 ms before another stimulus appeared in the same location. The experimenter coded response accuracy after each vocal response. There were 20 items per category, with items and categories distributed randomly over trials.

A J4-X ALERT (Alcohol Countermeasures Inc., Mississauga, ON, Canada) handheld breathalyzer measured blood alcohol concentration (BAC; %) at the start of the test session for all participants, and at key intervals throughout the session for participants who received alcohol. A mock breathalyzer, designed to closely resemble the ALERT, was used to strengthen the belief that one had consumed alcohol in participants who received placebo. In line with Sayette et al. (2001), the device registered a false BAC of 0.041% and was administered once, 10 minutes after drinking ended. This is the highest credible value for placebo manipulations of this kind (Martin and Sayette, 1993).

Female participants, assigned to receive alcohol or placebo, were required to perform a pregnancy self-test before the start of the test session. They were advised of this requirement during the initial telephone contact before agreeing to participate.

Participants completed state self-report scales at preexperimental baseline and after drink/stress exposure during the test phase (right after the Stroop task; see Table 1). In line with Sayette et al. (2001), the state portion of the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1970) assessed subjective anxiety. A modified visual analog scale (VAS; 0–10 not at all-extremely) measured desire for alcohol in all participants. To assess the perceived intoxicating effects of the alcohol dose and confirm the credibility of the placebo, participants in the alcohol and placebo conditions rated the strength of their drinks in terms of standard (5%) bottles of beer (cf. Vogel-Sprott, 1992) upon completion of all other measures.

Trait scales were administered following the test phase. For participants who received alcohol, this occurred when BAC declined below 0.03%. The scales included the Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993), the trait portion of the STAI (Spielberger et al., 1970), and the Beck Depression Inventory-Short Form (BDI-SF; Beck and Beck, 1972). A family history of alcoholism was assessed with a checklist that participants used to indicate biological relatives who, they believed, had an alcohol problem.

Procedure

The procedure conformed closely with that of Sayette et al. (2001). Eligible participants were contacted by telephone and invited to participate in a study on the behavioral effects of alcohol. In compliance with institutional ethics requirements, they were told that they may receive a low dose of alcohol (less than 2 standard drinks), a moderate dose of alcohol (2–4 standard drinks), or a soft drink, and that their drink condition would be determined randomly. Also, they were advised that some participants may be asked to make a brief speech, and that this would also be determined randomly. This was couched in terms of an assessment of alcohol's effect on verbal behavior. Participants did not know the topic of the speech beforehand. Advisory of the speech topic—''what I like and dislike about my body and physical appearance''—served as the stressor. During the initial telephone contact, participants were instructed to avoid alcohol for 24 hours, caffeine for 4 hours, and food for 3.5 hours before the start of the test session.

Table 1. Test Session Timeline

Consent		
Baseline measures		
Breathalyzer (BAC-0)		
Pregnancy screen		
Self-report measures (STAI-0, VAS-0)		
Practice STROOP (Neutral words only)		
STRESSOR—(advisory of speech topic)	→	DAS participants
CONTROL—(advisory—NO speech required)	→	NSC participants
Drink administration (3 drinks)		
Breathalyzer (BAC-1)		
STRESSOR—(advisory of speech topic)	→	DBS participants
Breathalyzer (BAC-2)		
Test STROOP (motivationally relevant Neutral words)		**Main Dependent Measures**
Self-report measures (STAI-1, VAS-1)		
Breathalyzer (BAC-3)		
Supplemental consent	→	DAS and DBS participants
Self-report measures (STAI-2, VAS-2, Drink Strength Rating)		
Breathalyzer (BAC-4)		
Videotaped speech	→	DAS and DBS participants
Participants rest		
Breathalyzer (BAC 4)		
Trait Self-Report Scales (AUDIT, BDI-SF, STAI-Trait; BAC $<$ .03%)		
Debriefing		
Payment/dismissal		

Table 1 shows the sequence of events during the test session, and highlights the time when the main dependent measures were taken, which corresponded to peak BAC. The session began at noon. Participants read the consent form, which reiterated the points made on the phone, assured confidentiality, and confirmed that the study had been approved by the Research Ethics Boards of the Centre for Addiction and Mental Health and Ryerson University. After signing the consent form, participants received their baseline breathalyzer assessment. Females in the alcohol or placebo conditions then received their pregnancy test. Baseline ratings of state anxiety and desire for alcohol were also taken. To ensure that the Stroop task was well learned before drink administration, participants performed a set of 80 practice trials with Neutral words only (e.g., names of birds). The Neutral categories and items in the practice task were different from those used in the test version. Participants were advised that they would perform the color-naming task again after their drinks and that the task requirements would remain the same.

Immediately after the baseline measures, participants in the DAS and NSC conditions received their respective experimental challenges. In line with Sayette et al. (2001), DAS participants were told that, in a short time, they would be required to make a 3-minute speech about their physical appearance (as described above). They were advised that the speech would be recorded on videotape and analyzed by graduate students in clinical psychology for openness, defensiveness, and other psychological variables. They were then escorted to a nearby room containing a video-camera mounted on a tripod and attached by cable to a VCR. The experimenter provided them with a piece of paper and pen to write down any points they wished to include in their speech. After a 2-minute interval, participants were escorted back to the waiting room where they received their drinks. At the same time that DAS participants were advised of the speech topic, participants in the NSC condition were advised that they had been assigned to a control condition and would not be required to make a speech. Instead, they were provided with a general interest article from National Geographic (November 2002, pp 124–126), which they were instructed to read in the waiting room adjacent to the laboratory. Participants in the DBS condition rested in the waiting room during this brief interval, and read magazines or watched television at their discretion.

All participants then received their assigned drinks, served in 3 equal-sized portions. In line with Sayette et al. (2001), participants first received a dry bagel plus 8-oz water to help standardize drink absorption. Participants in the alcohol group then received 0.7 g/kg (males) or 0.63 g/kg (females) of vodka, mixed in a ratio of 1:3.5 with noncaffeinated, carbonated soft drink (Mello Yello, The Coca Cola Co., Atlanta, GA). This dose conformed to the original appraisal disruption study (Sayette and Wilson, 1991).[1] Placebo participants received three 8-oz. cups of chilled nonalcoholic beer (Molson Exel, Molson Coors Brewing Co., Toronto, Ontario, Canada). The rim of each cup was misted with 50% alcohol solution to enhance olfactory cues for alcohol. Participants in the soda group received three 8-oz. cups of Mello Yello.

Drinks were served at 10-minute intervals, and participants were instructed to consume them steadily rather than all at once. Blood alcohol concentration readings were taken before and after drinking and at 20-minute intervals thereafter. Ten minutes after completion of the third drink, placebo participants received their false BAC reading of 0.041%. Participants in the DBS condition received the stressor 48 minutes after drinking commenced (28 minutes after receiving the third drink). At this point in the procedure, Sayette et al. (2001) administered the STAI. In the present study, the STAI was administered after participants completed the Stroop task ($\sim$10 minutes later), as some words in the STAI could prime certain categories (e.g., Social Threat) in the task. Thus, the main dependent measures were taken from 50 to 65 minutes after the initiation of drinking, which was designed to coincide with peak BAC.

Following assessment of the main dependent measures, DAS and DBS participants were escorted back to the video-camera room. For ethical reasons (lack of full disclosure about speech topic), participants were provided with a second consent form, which gave them the opportunity to opt out of the speech. Therefore, only measures taken before the speech are relevant.

After the speech (or decision to opt out), participants returned to the waiting room, where they received lunch and watched television

[1]The gender-corrected dose for females was based on the male:female dose ratio used by Sayette et al. (2001).

or read magazines. After lunch, participants completed their trait scales. After the trait scales, they were debriefed, paid, and dismissed. Participants who received alcohol completed their trait scales after their BAC had declined to 0.03%. Before departure, all participants received a pamphlet on safe use of alcohol and the contact number of the principal investigator in case of future questions about the study.

Data Analytic Plan

Chi-square tests of independence were conducted to ensure that a family history of alcoholism was not confounded with any of the experimental factors. A series of 2 (gender: male, female)×2 (AS group: high AS, low AS)×3 (drink type: alcohol, placebo, soda)×3 (stress condition: NSC, DBS, DAS) variance analyses assessed participant background characteristics. The Bonferroni procedure controlled for inflation of family-wise α with 4 variables: age, AUDIT, BDI-SF, and STAI-Trait. A multivariate analysis of variance (MANOVA) assessed the 4 measures of drinking habits on the PDQ: frequency (occasions/week), dose (mL alcohol/kg), duration (hours/occasion), and rate (mL/kg/h). A series of 2×2×3×3 variance analyses assessed the 3 main outcome measures: STAI-State, Stroop interference, and desire for alcohol. In each analysis, experimental effects were assessed using the score taken at peak BAC (50–65 minutes after initiation of drinking; see Table 1). Word type served as a within-subjects variable in the analysis of Stroop scores. Time of test served as a within-subjects variable in the analysis of BAC. For the STAI and desire measures, preexperimental baseline scores were included as covariates in analyses of covariance (ANCOVAs) to control for intrinsic variation and isolate treatment effects (cf. Sayette et al., 2001). Simple effects analyses, using the MS-error term from the corresponding variance analysis (Winer, 1971), decomposed significant interactions. Post hoc analyses, using Dunn's protected *t* test, compared scores as a function of drink type in the NSC condition.

Results

Participants

Background Characteristics. A family history of alcoholism was not significantly associated with any of the experimental factors, p's > 0.058 (Bonferroni p's > 0.46). The total number of participants reporting a family history of alcoholism was 21/180 (12%). Table 2 reports the mean (SD) scores for age and the trait measures in the different groups and conditions. The table shows that participants were uniformly young, with fairly high levels of alcohol misuse as reflected by scores above 8 on the AUDIT; they reported relatively few depressive symptoms (BDI-SF cut-off for depression = 8) apart from high AS females, and moderate levels of trait anxiety (scale range = 20–60).

A series of 2×2×3×3 ANOVAs yielded the following results. There were no significant effects for age, Bonferroni p's > 0.056. On the AUDIT, a main effect emerged for gender, $F(1, 145) = 12.34$, Bonferroni's $p = 0.004$,

Table 2. Mean (*SD*) Characteristics of Low and High Anxiety-Sensitive (AS) Participants ($N = 180$)

	Stress Condition											
	DAS				DBS				NSC			
	Low AS		High AS		Low AS		High AS		Low AS		High AS	
	M	F	M	F	M	F	M	F	M	F	M	F
Soft drink												
Age	21.4	20.5	20.8	22.7	21.3	22.0	23.0	21.4	20.4	22.8	23.0	24.1
	(2.3)	(1.7)	(2.7)	(3.9)	(2.4)	(4.8)	(1.9)	(2.6)	(1.7)	(2.9)	(3.8)	(3.3)
AUDIT	11.0	6.7	8.6	7.2	5.8	5.0	12.8	5.8	9.4	5.0	11.8	4.4
	(3.5)	(3.6)	(3.7)	(2.6)	(1.9)	(2.9)	(6.7)	(4.3)	(4.6)	(2.8)	(6.4)	(1.5)
BDI-SF	2.4	0.8	2.2	6.3	2.2	1.0	7.0	5.0	1.2	1.0	7.8	3.4
	(2.6)	(1.0)	(2.0)	(5.4)	(1.3)	(1.4)	(4.6)	(1.9)	(0.8)	(1.7)	(4.3)	(5.6)
STAI-T	39.0	38.0	43.8	48.0	41.0	34.5	52.2	46.6	35.6	35.8	48.2	42.6
	(10.3)	(4.2)	(7.2)	(10.9)	(4.6)	(7.8)	(3.5)	(6.7)	(5.8)	(4.9)	(7.6)	(3.9)
Placebo												
Age	22.0	20.6	22.3	20.4	22.4	20.6	24.0	21.6	23.2	20.5	23.8	21.0
	(2.2)	(1.5)	(2.9)	(1.9)	(1.5)	(1.8)	(4.0)	(1.8)	(2.6)	(1.9)	(2.2)	(2.3)
AUDIT	10.3	10.4	10.7	8.8	8.5	7.4	6.3	8.2	8.0	5.5	8.6	7.5
	(2.8)	(7.0)	(5.8)	(2.9)	(6.6)	(3.4)	(3.0)	(7.2)	(4.4)	(3.1)	(4.5)	(2.5)
BDI-SF	1.5	3.6	5.3	6.2	0.6	4.6	5.0	5.2	3.6	2.3	1.6	3.3
	(2.4)	(4.4)	(4.4)	(7.5)	(1.3)	(2.9)	(3.6)	(5.8)	(3.9)	(3.2)	(1.5)	(3.4)
STAI-T	31.0	41.8	41.3	52.0	36.0	40.8	45.5	45.0	38.6	42.3	36.4	43.5
	(4.5)	(8.0)	(6.2)	(9.4)	(5.9)	(9.6)	(9.9)	(3.6)	(3.6)	(10.7)	(1.5)	(7.3)
Alcohol												
Age	21.4	22.0	21.0	22.0	22.2	20.2	22.2	21.0	23.2	21.0	23.4	19.8
	(1.8)	(1.9)	(2.0)	(2.5)	(2.8)	(1.8)	(2.2)	(1.9)	(4.6)	(2.6)	(2.1)	(1.1)
AUDIT	12.4	4.6	11.0	9.4	7.4	5.4	11.0	10.2	11.0	7.2	11.3	13.6
	(7.2)	(1.9)	(2.6)	(4.6)	(4.7)	(2.9)	(3.7)	(6.1)	(4.3)	(3.1)	(4.3)	(6.1)
BDI-SF	1.6	1.0	4.0	6.6	5.4	4.2	7.1	8.0	2.6	2.4	3.0	4.8
	(2.3)	(1.4)	(5.2)	(7.4)	(3.4)	(2.8)	(5.5)	(10.8)	(2.5)	(1.5)	(4.7)	(3.8)
STAI-T	34.3	36.2	46.3	49.2	44.0	35.6	51.4	45.8	32.0	31.2	40.8	47.6
	(3.3)	(6.8)	(11.9)	(6.2)	(4.3)	(7.5)	(9.3)	(12.9)	(5.8)	(5.6)	(8.7)	(7.8)

DAS, drink-after-stress; DBS, drink-before-stress; NSC, no-stress control; AUDIT, Alcohol Use Disorders Identification Test; BDI-SF, Beck Depression Inventory-Short Form; STAI-T, State-Trait Anxiety Inventory–Trait subscale.

reflecting higher problem drinking scores for males. On the BDI-SF, a main effect of AS group, $F(1, 145) = 19.05$, $p < 0.001$, reflected higher overall depressive symptoms in high AS participants. On the STAI-Trait, a main effect of AS group, $F(1, 145) = 61.39$, $p < 0.0001$, reflected higher trait anxiety scores in high AS participants. The STAI-Trait analysis also yielded a significant gender×drink type interaction, $F(2, 145) = 5.24$, Bonferroni's $p = 0.024$, reflecting higher scores for females versus males in the placebo condition, and a significant gender×stress condition interaction, $F(2, 145) = 4.97$, Bonferroni's $p = 0.032$, reflecting higher scores for females versus males in the DBS condition. Follow-up analyses were conducted to rule out the possible influence of these background differences on experimental variables (see end of "Results").

Drinking Habits. A 2×2×3×3 MANOVA assessing the mean drinking frequency, dose, duration, and rate of consumption scores on the PDQ yielded no significant multivariate effects, p's > 0.06. The results conform very closely to norms for undergraduate drinkers on the PDQ (Vogel-Sprott, 1992): 4.1 standard drinks per occasion, consumed over 3.75 hours, at a rate of 1.1 drinks per hour, 1.5 times per week.

Experimental Effects

Drink Strength Ratings. A 2×2×3×2 (drink type) ANOVA assessed drink strength ratings at the end of the test phase in participants who received alcohol or placebo. The analysis yielded a main effect of drink type, $F(1, 65) = 31.07$, $p < 0.0001$, and no other significant effects, p's > 0.30. The mean (SD) drink strength ratings in terms of bottles of 5% beer were 3.7 (1.4) for alcohol participants and 2.1 (1.3) for placebo participants. The placebo rating significantly ($p < 0.001$) exceeded 0, supporting the credibility of the manipulation.

Blood Alcohol Concentration. A 2×2×3×7 (time of test) ANOVA of BACs in participants who received alcohol yielded a main effect of time of test, $F(6, 114) = 203.91$, $p < 0.0001$, and no other significant effects, p's > 0.07. The dose resulted in a mean (SD) peak BAC of 0.064 (0.009)% at 70 minutes after drinking commenced, which coincided with the assessment of the main dependent measures (BAC-3, Table 1). The mean (SD) BAC immediately before the Stroop task, when DBS participants received the stressor, was 0.063 (0.001)%.

State Anxiety (STAI). A preliminary 2×2×3×3 ANOVA of baseline STAI-State anxiety scores yielded a main effect of AS group, $F(1, 142) = 61.63$, $p < 0.0001$, and no other significant effects, p's > 0.09. Preexperimental state anxiety was greater in high AS ($M = 40.3$) than low AS ($M = 30.2$) participants. The range in possible scores on the STAI is 20 to 60. These baseline results confirm that floor or ceiling effects did not restrict response to the treatments.

Figure 1A–1D presents the state anxiety scores for all experimental cells. The bars depict means adjusted for pretest baseline; the lines show unadjusted means. The 4 panels show that the pattern of baseline-adjusted and unadjusted means was very similar across groups and conditions, indicating that extraexperimental variance did not account for treatment effects. Figures 1A and 1B reveal a generally similar pattern of scores for low AS males and females. Figures 1C and 1D indicate that, in contrast to low AS participants, alcohol consistently decreased anxiety relative to placebo in high AS participants, and that the effect of drink-stress sequence on alcohol anxiolysis appeared to differ for males versus females.

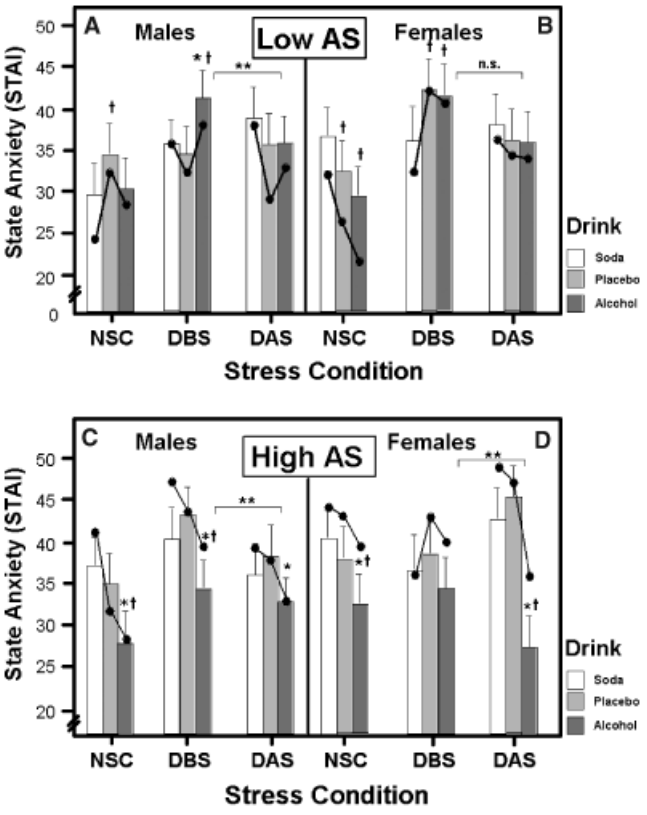

Fig. 1. Mean (SEM) state anxiety on the State-Trait Anxiety Inventory (Spielberger et al., 1970) in low and high anxiety sensitive (AS) undergraduate drinkers administered soda, a placebo drink, or alcohol in no stress control (NSC), drink before stress (DBS), and drink after stress (DAS) conditions. (**A**) Low AS males; (**B**) low AS females; (**C**) high AS males; (**D**) high AS females ($n = 45$ for each level of AS group×gender). *Significant ($p < 0.05$) difference from placebo; †significant difference from soda; and **significant difference in alcohol stress response dampening (SRD; alcohol minus placebo) between DBS and DAS conditions.

A 2×2×3×3 ANCOVA of STAI scores, with baseline scores as the covariate, yielded significant main effects of drink type, $F(2, 134) = 5.13$, $p = 0.007$, and stress condition, $F(2, 134) = 6.20$, $p = 0.003$, an AS group×drink type interaction, $F(2, 134) = 5.49$, $p = 0.005$, and no other significant effects, p's > 0.17.

The AS group×drink type interaction in the ANCOVA confirmed the consistent dampening effect on STAI scores for alcohol ($M = 31.5$) versus placebo ($M = 39.7$) in high AS participants ($p < 0.001$), with no consistent difference for

alcohol ($M = 37.2$) versus placebo ($M = 37.0$) in low AS participants, $p > 0.50$. Relative to placebo, scores for the soda condition did not differ significantly in high AS participants ($M = 38.8$) or low AS participants ($M = 37.1$), p's > 0.40. Thus, in high AS participants, alcohol reliably dampened state anxiety relative to a placebo beverage, whereas placebo had no reliable effects relative to a soft drink.

The main effect of stress condition reflected significantly greater anxiety in both the DBS ($M = 38.9$) and DAS ($M = 37.5$) conditions relative to the NSC ($M = 34.2$) condition (p's < 0.001), and no overall difference in anxiety between the DBS versus DAS conditions, $p > 0.20$.

Simple Effects[2]

In high AS males, the difference in STAI for alcohol versus placebo in the DBS condition ($M = 9.42$) was significantly greater than in the DAS condition ($M = 4.61$), $t(134) = 3.67$, $p < 0.001$ (Fig. 1C). By contrast, in high AS females, the difference in STAI for alcohol versus placebo in the DBS condition ($M = 3.55$) was significantly smaller than in the DAS condition ($M = 18.23$), $t(134) = -11.28$, $p < 0.0001$ (Fig. 1D). Thus, consistent with the appraisal disruption hypothesis, alcohol SRD as measured by state anxiety was greater when drinking came before rather than after stress in high AS males. In high AS females, the results were inconsistent with those predicted based on appraisal disruption.

Figures 1A and 1B shows that, for both male and female low AS participants, STAI values for placebo and alcohol were virtually identical in 3 of the 4 active stress conditions, t's < 1, p's > 0.50. The sole exception was the DBS condition in low AS males, where STAI was significantly greater under alcohol, $t(134) = -5.45$, $p < 0.001$, than placebo or soda, which did not differ from one another. Thus, in direct contrast to high AS participants, alcohol had no overall anxiolytic effects when stress was present in low AS participants, and led to increased anxiety in low AS males who drank before stress.

Stroop Interference. A preliminary $2\times2\times3\times3$ ANOVA of RT to neutral words yielded no significant effects, p's > 0.14. Therefore, any differences in interference (RT difference from Neutral for motivationally relevant words) are not due to differences in overall mental fluency to motivationally neutral stimuli. A preliminary $2\times2\times3\times3\times5$ (Word Type) ANOVA of errors (reading the word, misnaming the color) yielded a marginal AS group×drink type interaction ($p = 0.052$), and no other significant or marginal effects, p's > 0.11. The marginal result reflected a slightly higher mean error rate under alcohol (0.48/20) versus placebo (0.20/20) or soda (0.23/20) in low AS participants as against a slightly lower error rate under alcohol (0.24/20) versus placebo (0.26/20) or soda (0.36/20) in high AS participants. The low absolute error rate (< 2.5% of trials) ensures that the mean RT scores for the Stroop task were reliable.

A $2\times2\times3\times3\times5$ ANOVA of RT scores to Physical Threat, Social Threat, Appearance, Alcohol, and Neutral words yielded a 5-way interaction, $F(16, 564) = 1.70$, $p = 0.044$, and no significant between-subjects effects (i.e., no overall effects collapsed across word type), p's > 0.13. Within-subjects contrasts, comparing the 4 motivationally relevant word types with Neutral words, found that the interaction was due solely to differences in relative RT to Social Threat versus Neutral words across the experimental cells. A follow-up $2\times2\times3\times3$ ANOVA of RT difference scores to Social Threat (minus Neutral) words alone yielded a 4-way interaction, $F(4, 145) = 2.88$, $p = 0.025$, and no other significant effects involving drink type, p's > 0.09.

The RT difference needed for significant within-cell interference (Social Threat slower than Neutral) or facilitation (Social Threat faster than Neutral) was ± 9 ms, where 0 = Neutral RT. The difference needed for a significant between-cell effect (e.g., alcohol vs placebo) in the size of the RT difference score was ± 21 ms.

Figures 2A–2D shows the Stroop RT difference scores (Social Threat minus Neutral words). Dashed lines mark the boundaries for significant within-cell effects, relative to Neutral. Positive scores denote interference and negative scores denote facilitation to Social Threat.

Figure 2A shows that, in low AS males, significant interference to Social Threat words emerged only when drinking came after stress and only when the drink was soda or placebo. Alcohol negated interference to Social Threat relative to both of these drink conditions and induced a level of facilitation similar to that seen in the other stress conditions.

Figure 2B shows that, in low AS females, significant interference occurred in the absence of stress under soda and placebo and alcohol negated these effects. The most prominent effect in these participants was the marked interference in those who drank placebo after stress. Relative to placebo, alcohol negated this interference but also negated the facilitation observed in the soda condition.

Figures 2C and 2D show that high AS participants displayed interference in 5 of 6 cells in the NSC condition, consistent with their expected response to Social Threat words in the absence of stress. Figure 2C shows that in high AS males, placebo reversed the interference seen under soda in the NSC condition. Alcohol in turn reversed the facilitation seen under placebo, leading to significant but more modest interference than soda. In the 2 active stress conditions, the effects of drink type were essentially opposite depending on the drink-stress sequence. In high AS males who drank before stress, facilitation emerged under soda whereas interference emerged under placebo. Facilitation also emerged under

[2]When an a priori hypothesis exists, it is permissible to test simple effects in the absence of a significant F statistic in the ANCOVA (Howell, 1992).

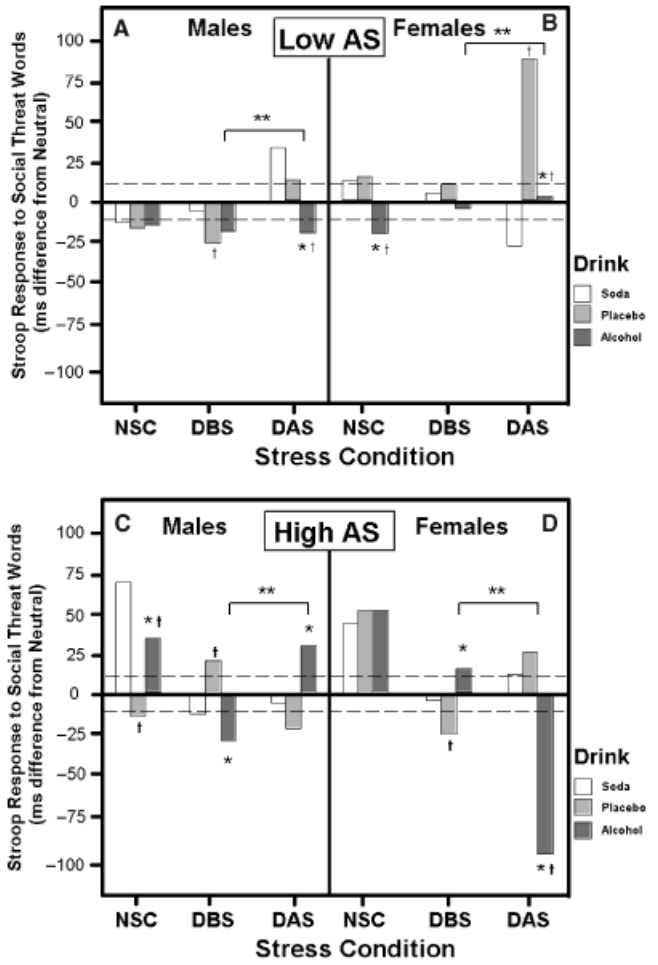

Fig. 2. Mean difference in color-naming response time (ms; Social Threat minus Neutral) on a modified Stroop task in low and high anxiety sensitive (AS) undergraduate drinkers administered soda, a placebo drink, or alcohol in no stress control (NSC), drink before stress (DBS), and drink after stress (DAS) conditions. (**A**) Low AS males; (**B**) low AS females; (**C**) high AS males; (**D**) high AS females ($n = 45$ for each level of AS group×gender). Dashed lines indicate a significant ($p < 0.05$) within-cell difference from neutral. Positive scores reflect interference; negative scores reflect facilitation to Social Threat versus Neutral words. *Significant ($p < 0.05$) between-cell difference from placebo; †significant between-cell difference from soda; and **significant difference in alcohol stress response dampening (SRD; alcohol minus placebo) between DBS and DAS conditions.

alcohol, and the magnitude of this effect was greater than under soda but only marginally so, $p < 0.10$. In high AS males who drank after stress, neither interference nor facilitation emerged under soda. Significant facilitation (relative to neutral baseline) emerged under placebo but the size of this effect did not differ from soda, $p > 0.10$. Under alcohol, participants displayed interference, which differed significantly from placebo but only marginally from soda, $p < 0.10$.

Figure 2D shows the scores for high AS females, and reveals a pattern of effects very different from that of high AS males. In the absence of stress, high AS females displayed significant and comparable interference in all 3 drink conditions. In the presence of stress, the effects of drink-stress sequence were marked, but opposite to the pattern seen in high AS males. Specifically, in high AS females who drank before stress, placebo led to facilitation, which differed significantly from soda. Alcohol in turn, led to interference that differed significantly from the placebo but not the soda condition. In high AS females who drank after stress, significant interference emerged under soda and under placebo, but there was no difference between these conditions. Alcohol induced a dramatic facilitation effect, which differed significantly from the other 2 drink conditions.

In sum, Stroop interference to Social Threat words was less common in low AS than high AS participants. Interference did emerge in low AS males who drank soda or placebo after stress and in low AS females who drank placebo after stress. In high AS participants, gender significantly moderated the effects of drink-stress sequence and drink type, with high AS males displaying facilitation (i.e., SRD) when they drank alcohol before stress and high AS females displaying marked facilitation when they drank alcohol after stress. The reversal in the pattern of effects of alcohol as a function of drink-stress sequence in high AS males versus females on the Stroop task parallels the gender-related reversal in the pattern of alcohol's effects on state anxiety.

Desire for Alcohol. Figures 3A–3D show the desire scores for the different groups and conditions. As with the other indices, baseline-adjusted and unadjusted scores were similar so that extraexperimental variation did not appear to account for treatment effects.

A 2×2×3×3 ANCOVA of postdrink/stress desire scores, controlling for baseline, yielded several significant effects. The highest order effect was an AS group× gender×drink type interaction, $F(2, 145) = 4.66$, $p = 0.011$. Simple effects analyses found that, in low AS males, overall desire scores did not differ in the soda versus placebo conditions, $p > 0.10$, each of which was significantly greater than the alcohol condition, t's $(145) > 2.08$, p's < 0.05 (Fig. 3A). In low AS females, desire scores were higher in the alcohol condition than in the soda or placebo conditions, $t(145) = 2.50$, $p < 0.02$, which did not differ from one another, $p > 0.90$ (Fig. 3B). In high AS males, overall desire scores were higher in the soda than in the placebo condition, $t(145) = 2.92$, $p < 0.01$, and higher in the alcohol than in the soda condition, $t(145) = 2.62$, $p < 0.01$ (Fig. 3C). In high AS females, there were no significant pair-wise differences in desire among the soda, placebo, or alcohol conditions, p's > 0.40 (Fig. 3D).

In sum, alcohol primed desire more than the other 2 types of drink in high AS males, whereas drink type had no appreciable effect on desire in high AS females. Low AS participants showed the opposite pattern across gender, with stronger alcohol priming effects observed in females. Compared with soda, placebo selectively reduced (i.e.,

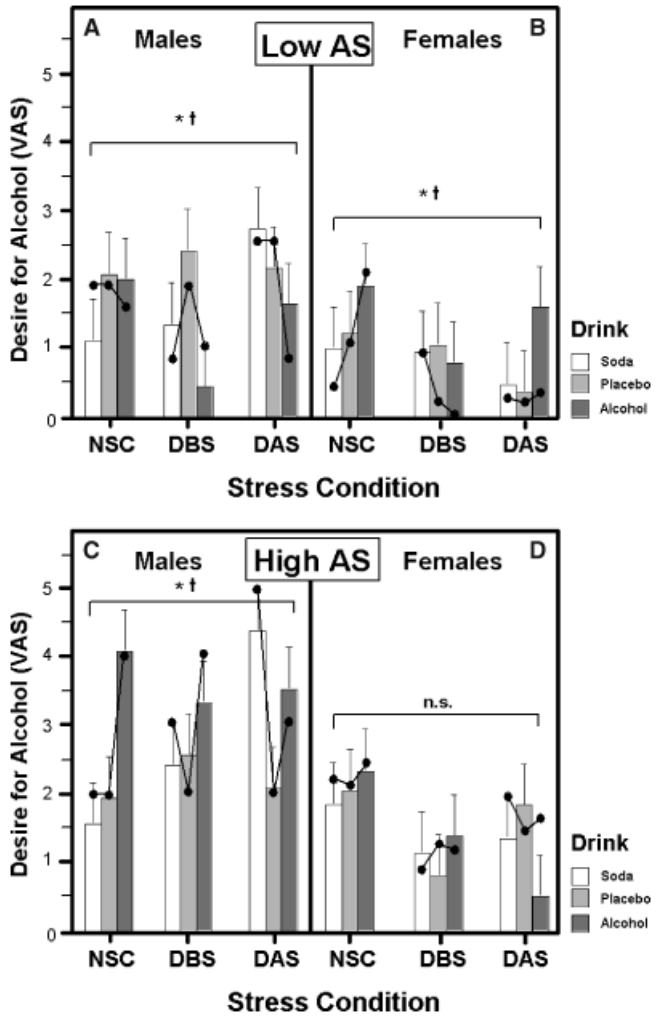

Fig. 3. Mean (SEM) ratings of desire for alcohol on a modified Visual Analog Scale (VAS; 0–10) in low and high anxiety-sensitive (AS) undergraduate drinkers administered soda, a placebo drink, or alcohol in no stress control (NSC), drink before stress (DBS), and drink after stress (DAS) conditions. (**A**) Low AS males; (**B**) low AS females; (**C**) high AS males; (**D**) high AS females ($n = 45$ for each level of AS group×gender). *Significant overall difference from placebo, aggregated across drink-stress sequence and †denote a significant overall difference from soda, aggregated across drink-stress sequence.

inhibited) desire for alcohol in high AS males alone, although Fig. 3C suggests that this was due primarily to the marked decrease in desire under placebo in the DAS condition. Overall, stress reduced the priming effect of alcohol.[3]

[3]The analyses of participant background characteristics revealed that, despite matching and random assignment, some experimental cells differed significantly in mean weekly alcohol dose (based on follow-up univariate ANOVA), BDI-SF, and trait anxiety. Supplemental ANCOVAs were conducted for the 3 experimental measures including each of these 3 background measures as covariates. The pattern of significant effects was identical to that of the original analyses. These results confirm that the effects reported in the original analyses were due to the experimental factors and were not mediated (accounted for) by differences in background characteristics.

DISCUSSION

This study tested the appraisal disruption hypothesis of alcohol SRD in male and female undergraduates with high or low levels of AS. The hypothesis predicted that alcohol SRD would be greater when drinking came before rather than after exposure to a stressor. The possible disruption of appraisal by alcohol seemed especially relevant for high AS participants, who tend to overinterpret their involuntary responses to threat and use alcohol to cope under these circumstances. Gender differences in the importance of social-evaluative concerns in high AS participants further implied that alcohol SRD to the social stressor of a self-revealing speech would be greater in high AS males than in high AS females. Scores on the STAI were significantly increased by the stressor relative to NSC. The alcohol dose yielded a peak BAC of 0.064% at the time alcohol SRD was assessed, comparable to previous research with the same dose (Sayette and Wilson, 1991). The drink strength ratings for alcohol participants further indicated that they accurately registered the size of the dose. Also, the drink-strength ratings for placebo participants significantly exceeded 0, indicating that the placebo was credible. These results confirm the effectiveness of the stress, alcohol, and placebo manipulations and thus indirectly support the validity of the other findings.

As in previous studies, alcohol significantly reduced state anxiety in high AS participants. In low AS participants, alcohol had no significant overall effect on state anxiety. In low AS males who drank before stress, alcohol resulted in increased anxiety relative to placebo or soda. This effect was absent in low AS males who drank alcohol after stress, a pattern that does not conform to the appraisal disruption hypothesis. Interestingly, the findings for low AS males in the DBS condition do conform to the attention allocation hypothesis (Steele and Josephs, 1990), which predicts increased anxiety under alcohol when drinking precedes stress and there is no distraction. Of the various subgroups in the present study, only high AS males displayed a pattern of state anxiety scores consistent with the appraisal disruption hypothesis. In these participants, alcohol led to a significantly greater decline in anxiety relative to placebo in the DBS versus DAS condition.

The results from the modified Stroop task indicated that, of the 4 motivationally relevant types of words (Physical Threat, Social Threat, Appearance related, Alcohol), only Social Threat words engendered significant interference effects that differed as a function of the experimental factors. Consistent with previous research with no experimental stressor, significant interference to Social Threat words emerged in the NSC condition in high AS males and high AS females (Stewart et al., 1998). Also in line with the literature, male and female low AS participants displayed either very modest interference or facilitation of RT to

Social Threat versus Neutral words in the NSC, a pattern that typifies nonanxious individuals (e.g., Li et al., 2005). In the active stress conditions, alcohol significantly reduced interference to Social Threat words relative to placebo in the DAS condition in low AS males, low AS females, and high AS females. No such alcohol SRD effects were evident in the DBS condition in these 3 subgroups. These findings are inconsistent with the appraisal disruption hypothesis, and instead indicate that, in a majority of young drinkers, alcohol reliably reduces attention to threat cues (i.e., increases SRD) when drinking comes after a social stressor.

In contrast to the prevailing pattern for the sample on the Stroop task, high AS males again displayed distinct effects that were consistent with the appraisal disruption hypothesis: a greater dampening effect of alcohol, relative to placebo, in the DBS versus DAS condition. In the latter condition, alcohol actually increased interference relative to placebo in high AS males. Simple effects analyses confirmed that the effects of drink-stress sequence were statistically significant. In sum, although alcohol effects on Stroop interference to Social Threat words generally did not support the appraisal disruption hypothesis, the Stroop findings for high AS males, as in the case of state anxiety, did support the hypothesis. The lack of anxiolysis on the STAI or facilitation to threat words on the Stroop under placebo, coupled with the presence of these effects under alcohol, indicates that when drinking precedes a stressor, the pharmacological effect of alcohol rather than expectancy effects account for alcohol SRD in high AS males.

Desire for alcohol on the modified VAS reflected the priming or incentive-motivational effects of the dose. Relative to placebo, the priming effects of alcohol varied markedly across the experimental cells. Modest priming effects emerged in low AS but not high AS females. In low AS males, alcohol was associated with decreased desire scores relative to placebo or soda. In contrast to the other 3 subgroups, high AS males displayed robust and consistent priming effects of alcohol, although these were somewhat attenuated by stress. The consistency and magnitude of alcohol priming relative to placebo in high AS males, together with the reliable anxiolytic effect of alcohol versus placebo in these participants, imply that high AS males may derive a different pharmacological effect from alcohol than either high AS females or low AS individuals of either gender.

Overall, the results of this study do not lend support to the appraisal disruption hypothesis as a general mechanism accounting for alcohol SRD in undergraduate drinkers. However, the findings for high AS males do support the hypothesis, both in subjective stress dampening and decreased activation of the social threat memory network. High AS males also differed from the other 3 subgroups in the priming effects of alcohol, suggesting that high AS males may experience relatively greater positive reinforcement (i.e., incentive motivation) as well as greater negative reinforcement (i.e., SRD) from alcohol compared with other young drinkers.

The findings for high AS females were directly opposite to those for high AS males. High AS females displayed greater alcohol SRD in the DAS than the DBS condition on the state anxiety scale and on the Stroop task. Also in contrast to high AS males, high AS females exhibited no significant alcohol priming effects. These gender differences on the 3 experimental indices demonstrate that gender reliably moderates alcohol SRD to a social stressor and imply that alcohol may recruit different processes in high AS males versus females. Based on the pattern of effects, one possibility is that alcohol encourages approach motivation and concomitant willingness to engage a social challenge to a relatively greater degree in high AS males than in high AS females.

As noted in the introduction, high AS females report predominantly physical or hypochondriacal concerns and drink alcohol to manage these (Stewart et al., 1997, 2001). This suggests a possible explanation for the present results: in high AS females, the proximal cause of distress may have been the physiological arousal evoked by advisory of the speech. The interoceptive effects of alcohol in high AS females could have obscured this arousal, providing a sharp contrast to their predrink state in the DAS condition, but not in the DBS condition, where they had not yet encountered the stressor. Thus, differential arousal immediately before drink administration may have translated into greater perceived alcohol SRD (i.e., greater decline in arousal relative to predrink state) in the DAS than the DBS condition in high AS females.

In contrast to their female counterparts, in high AS males, the primary cause of anxiety may have been the perceived need to perform well in the speech. Alcohol may have reduced these concerns about good performance. Specifically, alcohol has been found to reduce emotional concern for the adverse consequences of one's actions (Zeichner et al., 1982). High AS males in the DBS condition would have received the full benefit of this effect. In contrast, high AS males in the DAS condition would have initially considered the potential consequences of their actions (e.g., embarrassment) while sober, permitting full activation of their social threat network. In this condition, alcohol's dampening effect on concern for consequences may have been less effective because the social threat network was already activated. This explanation is consistent with the appraisal disruption hypothesis.

Differential responses to the speech stressor may also have been influenced by physiological factors. The effects of speech stress, unlike those of physical stressors, are strongly influenced by serotonin—and particularly the 5-HT-2 receptors (Graeff et al., 2003). Notably, agents that reduce serotonin transmission at 2A/C receptors (e.g., nefazodone) also decrease the dopaminergic effects of alcohol and alcohol self-administration in animal models

(Olausson et al., 1998). Taken together, this evidence suggests that decreased activation of 5-HT2A/C receptors (particularly 5-HT2A; Pehek et al., 2006), with concomitant effects on dopamine, may have contributed to the effects of the speech stressor in the present study and that alcohol SRD involved modulation of this process. Serotonin and dopamine have both been shown to influence stress-induced and alcohol-induced critically priming in animals (Le and Shaham, 2002). Thus, the preservation of alcohol-priming effects in the stress conditions in high AS males raises the possibility that these individuals differ in 5-HT2A/C receptor sensitivity or, more directly, in their dopaminergic response to alcohol. Given the consistent link between alcohol SRD and problem drinking (e.g., Croissant and Olbrich, 2004; Pihl and Peterson, 1995; Sinha et al., 1998; Zimmermann et al., 2004), direct assessment of the neurochemical basis of alcohol SRD in high AS males and females would appear to be an important avenue for future investigation.

REFERENCES

Arrindell WA (1993) The fear of fear concept: stability, retest artifact and predictive power. Behav Res Ther 131:139–148.

Ball SG, Otto MW, Pollack M, Uccello R, Rosenbaum JF (1995) Differentiating social phobia and panic disorder: a test of core beliefs. Cogn Ther Res 19:473–481.

Beck AT, Beck RW (1972) Screening for depressed patients in family practice: a rapid technique. Postgrad Med 52:81–85.

Conrod PJ, Pihl RO, Vassileva J (1998) Differential sensitivity to alcohol reinforcement in groups of men at risk for distinct alcoholism subtypes. Alcohol Clin Exp Res 22:585–597.

Croissant B, Olbrich R (2004) Stress response dampening indexed by cortisol in subjects at risk for alcoholism. J Stud Alcohol 65: 701–707.

Curtin JJ, Lang AR, Patrick CJ, Stritzke WG (1998) Alcohol and fear-potentiated startle: the role of competing cognitive demands in the stress-reducing effects of intoxication. J Abnorm Psychol 107: 547–557.

Curtin JJ, Patrick CJ, Lang AR, Cacioppo JT, Birbaume N (2001) Alcohol affects emotion through cognition. Psychol Sci 12:527–531.

Graeff FG, Parente A, Del-Ben CM, Guimaraes FS (2003) Pharmacology of human experimental anxiety. Braz J Med Biol Res 36:421–432.

Howell DC (1992) *Statistical Methods for Psychology*, 3rd ed., Duxbury, Boston, MA.

Josephs RA, Steele CM (1990) The two faces of alcohol myopia: attentional mediation of psychological stress. J Abnorm Psychol 99: 115–126.

Kushner MG, Mackenzie TB, Fiszdon J, Valentiner DP, Foa E, Anderson N, Wangensteen D (1996) The effects of alcohol consumption on laboratory-induced panic and state anxiety. Arch Gen Psychiatry 53:264–270.

Le A, Shaham Y (2002) Neurobiology of relapse to alcohol in rats. Pharmacol Ther 94:137–156.

Li X, Li X, Luo YJ (2005) Anxiety and attentional bias for threat: an event-related potential study. Neuroreport 16:1501–1505.

MacDonald AB, Baker JM, Stewart SH, Skinner M (2000) Effects of alcohol on the response to hyperventilation of participants high and low in anxiety sensitivity. Alcohol Clin Exp Res 24:1656–1665.

Martin CS, Sayette MA (1993) Experimental design in alcohol administration research: limitations and alternatives in the manipulation of dosage-set. J Stud Alcohol 54:750–761.

McNally RJ (2002) Anxiety sensitivity and panic disorder. Biol Psychiatry 52:938–946.

Olausson P, Ericson M, Petersson A, Kosowski A, Soderpalm B, Engel JA (1998) Nefazodone attenuates the behavioral and neurochemical effects of ethanol. Alcohol 15:77–86.

Pehek EA, Nocjar C, Roth BL, Byrd TA, Mabrouk OS (2006) Evidence for the preferential involvement of 5-HT2A serotonin receptors in stress- and drug-induced dopamine release in the rat medial prefrontal cortex. Neuropsychopharmacology 31:265–277.

Peterson RA, Reiss S (1992) *Anxiety Sensitivity Index Manual*, 2nd ed., International Diagnostic Systems, Worthington, OH.

Pihl RO, Peterson JB (1995) Alcoholism: the role of different motivational systems. J Psychiatry Neurosci 20:372–396.

Pohorecky LA (1991) Stress and alcohol interaction: an update of human research. Alcohol Clin Exp Res 15:438–459.

Saunders JB, Aasland OG, Babor TF, de la Fuente JR, Grant M (1993) Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption—II. Addiction 88: 791–804.

Sayette MA (1993) An appraisal–disruption model of alcohol's effects on stress responses in social drinkers. Psychol Bull 114:459–476.

Sayette MA, Martin CS, Perrott MA, Wertz JM, Hufford MR (2001) A test of the appraisal–disruption model of alcohol and stress. J Stud Alcohol 62:247–256.

Sayette MA, Wilson GT (1991) Intoxication and exposure to stress: effects of temporal patterning. J Abnorm Psychol 100:56–62.

Sharma D, Albery IP, Cook C (2001) Selective attentional bias to alcohol related stimuli in problem drinkers and non-problem drinkers. Addiction 96:285–295.

Sher KJ, Levenson RW (1982) Risk for alcoholism and individual differences in the stress-response-dampening effect of alcohol. J Abnorm Psychol 91:350–367.

Sinha R, Robinson J, O'Malley S (1998) Stress response dampening: effects of gender and family history of alcoholism and anxiety disorders. Psychopharmacology 137:311–320.

Spielberger CD, Gorsuch RL, Lushene RE (1970) *Manual for the State Trait Anxiety Inventory*. Consulting Psychologists Press, Palo Alto, CA.

Steele CM, Josephs RA (1988) Drinking your troubles away. II: an attention–allocation model of alcohol's effect on psychological stress. J Abnorm Psychol 97:196–205.

Steele CM, Josephs RA (1990) Alcohol myopia. Its prized and dangerous effects. Am Psychol 45:921–933.

Stewart SH, Conrod PJ, Gignac ML, Pihl RO (1998) Selective processing biases in anxiety-sensitive men and women. Cognit Emotion 12: 105–134.

Stewart SH, Finn PR, Pihl RO (1992) The effects of alcohol on the cardiovascular stress response in men at high risk for alcoholism: a dose response study. J Stud Alcohol 53:499–506.

Stewart SH, Pihl RO (1994) Effects of alcohol administration on psychophysiological and subjective-emotional responses to aversive stimulation in anxiety-sensitive women. Psychol Addict Behav 8: 29–42.

Stewart SH, Taylor S, Baker JM (1997) Gender differences in dimensions of anxiety sensitivity. J Anxiety Disord 11:179–200.

Stewart SH, Zvolensky MJ, Eifert GH (2001) Negative-reinforcement drinking motives mediate the relation between anxiety sensitivity and increased drinking behavior. Person Individ Diff 31:157–171.

Vogel-Sprott M (1992) *Alcohol Tolerance and Social Drinking: Learning the Consequences*. Guilford Press, New York.

Winer BJ (1971) *Statistical Principles in Experimental Design*, 2nd ed., McGraw-Hill, New York.

Zack M, Poulos CX, Fragopoulos F, MacLeod CM (2003) Effects of negative and positive mood phrases on priming of alcohol words in young drinkers with high and low anxiety sensitivity. Exper Clin Psychopharmacol 11:176–185.

Zack M, Poulos CX, Fragopoulos F, Woodford TM, MacLeod CM (2006) Negative affect words prime beer consumption in young drinkers. Addict Behav 31:169–173.

Zeichner A, Pihl RO, Niaura R, Zacchia C (1982) Attentional processes in alcohol-mediated aggression. J Stud Alcohol 43:714–724.

Zimmermann U, Spring K, Kunz-Ebrecht SR, Uhr M, Wittchen HU, Holsboer F (2004) Effect of ethanol on hypothalamic–pituitary–adrenal system response to psychosocial stress in sons of alcohol-dependent fathers. Neuropsychopharmacology 29: 1156–1165.