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Intoxication Level and Emotional Response

Keith F. Donohue, John J. Curtin, Christopher J. Patrick, and Alan R. Lang
Florida State University

We used affective modulation of the eye-blink component of the startle reflex to examine effects of three levels of alcohol intoxication and a no-intoxication control on emotional responses to pleasant, neutral, and unpleasant pictures. Non-problematic student drinkers (n = 101; 48 female) were randomly assigned to intoxication groups. Normal inhibition of startle during exposure to pleasant pictures was intact across groups. In contrast, potentiation of startle during viewing of unpleasant pictures was evident in the no- and low-intoxication groups, compared to the intermediate- and high-intoxication groups, in which it was significantly reduced. This pattern suggests that a direct and selective anxiolytic effect of alcohol can occur at higher levels of intoxication without an analogous impact on response to emotionally positive stimuli at similar levels.

Keywords: alcohol, dose response, stress response dampening, emotion, startle response

Although regulation of emotion has been central to influential theories of drinking behavior for decades (Leonard & Blane, 1999), the specific impact of alcohol on response to emotional stimuli—let alone the processes and mechanisms that might underlie it—is still not well understood (see Lang, Patrick, & Stritzke, 1999, for a review). There are certainly indications from analyses of self-report assessments of drinking motives and expectations that alcohol use can be driven by emotion (e.g., Cooper, Frone, Russell, & Mudar, 1995; Kidorf & Lang, 1999), but this work does not touch very directly on how responses to emotional stimuli are actually altered by alcohol intoxication. A concurrent line of research (see Greeley & Oei, 1999, for a review) has provided some insight into the nature of such reactions by manipulating alcohol administration and exposure to stress in individuals who are continuously monitored using indices of autonomic nervous system activity, most notably heart rate (HR), generally regarded to be markers of emotional arousal. Paradigms commonly used in this work rely on a single, extended episode of threat as the primary means of eliciting the emotional response that is evaluated. This approach has contributed to our understanding of connections between alcohol intoxication and response to threat, revealing an effect known as "stress-response dampening," but it has not addressed the possibly unique impact of drinking on more phasic emotional responses, including those associated with positive stimuli. In addition, only the occasional study has considered the possible role of level of intoxication in the alcohol-emotion nexus. The present experiment sought to address these issues and elucidate critical elements of the phenomena of interest through application of a sophisticated model of emotion, controlled administration of an array of both alcohol and affective manipulations, and use of a precise, reliable dependent measure that can tap both negative and positive emotional responses.

The conceptual framework for this work treats emotions as "action dispositions" that can be described in terms of their valence and intensity (Lang, 1995). Valence refers to the directional or qualitative (i.e., pleasant vs. unpleasant) aspect of emotional response thought to reflect the operation of two primary brain motive systems: an aversive system that governs withdrawal behavior and an appetitive system that governs approach behavior. Intensity represents the extent to which either of these systems is activated (also see Cacioppo & Berntson, 1994; Gray, 1987; Watson, Clark, & Tellegen, 1988, for comparable models). Neuroscience research has identified acute effects of alcohol in areas of the brain associated with these systems (Fromme & D'Amico, 1999) and psychosocial investigations have demonstrated that the expected and retrospectively reported effects of drinking primarily involve changes in the quality of emotion (Goldman, Del Boca, & Darkes, 1999). Accordingly, the emphasis here was on the impact that alcohol has on the valence of emotional response, and therefore pleasant as well as unpleasant stimuli were included to evaluate the specificity of effects at various levels of intoxication. This approach represents a departure from that taken in much of the extant literature on how alcohol affects emotion, which, since the early 1980s, has focused on the stress response (Greeley & Oei, 1999), using physiological measures that do not specifically target variations in valence.

Further, few alcohol-emotion studies to date have analyzed dose-response effects. Among the exceptions to this oversight, Sher and Walitzer (1986) examined male participants for the effect of different doses of alcohol on heart rate (HR) reactivity and self-reported anxiety occasioned by exposure to a socially stressful manipulation. They found that at breath alcohol concentrations

Keith F. Donohue, John J. Curtin, Christopher J. Patrick, and Alan R. Lang, Department of Psychology, Florida State University.

John J. Curtin is now at the Department of Psychology, University of Wisconsin-Madison. Christopher J. Patrick is now at the Department of Psychology, University of Minnesota-Twin Cities.

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Correspondence concerning this article should be addressed to John J. Curtin, Department of Psychology, University of Wisconsin, Madison, WI 53706. E-mail: jjcurtin@wisc.edu

(BrACs)[1] of either 0.04 or 0.07, participants showed diminished HR reactivity during a countdown period before the stressful interaction and also during the interaction itself. However, only those in the higher BrAC group reported relatively reduced anxiety during the countdown period—an effect not sustained during the stressor. This suggests that perhaps higher levels of intoxication are required to obtain consistent anxiolytic effects, even when complex appraisal and preparatory behaviors are involved.

Using a simpler and more explicit threat manipulation (electric shock), Stewart, Finn, and Pihl (1992) examined the effect of different levels of intoxication, including some at or above legally defined intoxication, on HR reactivity in male participants who differed in family history of alcoholism. They found that relatively high BrACs (in the range of 0.08 to 0.10) were required just to demonstrate decreased HR during the stressor and then only among participants with a positive, multi-generational family history of alcohol use disorders. The authors did not report whether dampening of HR reactivity to the shock stressor was accompanied by changes in subjective emotional state in this study, but previous research has indicated that BrACs approaching 0.10 are consistently associated with decreased response to a variety of stressors (e.g., Levenson, Sher, Grossman, Newman, & Newlin, 1980; Sher & Levenson, 1982). Taken together, results from these and other stress-response-dampening experiments suggest that reliable alcohol-induced reductions in autonomic reactivity in the presence of distressing stimuli may require relatively high levels of intoxication, a point made by Sher (1987) in his review of the early literature using such paradigms. Moreover, the exact relationship between changes in HR and emotional valence remains somewhat uncertain, partly because even in tightly controlled studies, HR may be subject to influence by processes not directly linked to emotion (cf. Lister, Eckhardt, & Weingartner, 1987).

Although assessments of autonomic reactivity, tracked and averaged over relatively long time periods, can be useful in characterizing ongoing or tonic mood states and manipulated alterations of them, they may not provide the optimal level of specificity and precision desirable for evaluations across broad dimensions of emotional response (Dawson, Schell, & Bohmelt, 1999; Dawson, Schell, & Filion, 2000). Consequently, alternative measures that are more sensitive to phasic changes in affective valence should be considered. The startle reflex in general and particularly its eye-blink component in humans provides one such index that is easy to elicit and measure and has proved to be quite sensitive to the valence of emotional state (Lang, Bradley, & Cuthbert, 1990).

The magnitude of startle response to a sudden, intense stimulus probe (e.g., a burst of white noise) varies with the valence of emotional state, such that it is augmented if the probe occurs in the context of an unpleasant stimulus and attenuated if the probe coincides with a pleasant stimulus (Lang et al., 1990). This affective modulation is thought to reflect the synergistic match or antagonistic mismatch between the action disposition elicited by the stimulus (defensive or appetitive) and reflexive reaction to the probe (which is defensive). Brain lesion studies in non-human animals have demonstrated that the neural pathway for the potentiation of startle during unpleasant stimuli involves projections from the central nucleus of the amygdala (CeA) to the nucleus reticularis pontis caudalis (nRPC) (for a reviews, see Davis, 1986, 1989; Davis, Walker, Lee, 1999), and brain-imaging studies of humans reveal activation of the amygdala during viewing of threatening stimuli (e.g., Hariri, Mttay, Tessitore, Fera, & Weinberg, 2003). Thus, augmentation of startle in organisms primed by aversive stimulation is thought to directly reflect activation of the defensive motivational system. The neural substrates of attenuation of startle during pleasant stimulation are less well established, but recent work by Steidl, Li, and Yeomans (2001) implicates the nucleus accumbens and its links to both the appetitive (reward) system and the primary startle circuit.

Stritzke, Patrick, and Lang (1995) took advantage of startle methodology to examine emotional response to pleasant, neutral, and unpleasant pictures from the International Affective Picture System (IAPS; Center for the Study of Emotion and Attention, 1997) in participants who consumed alcohol to a moderate level of intoxication (BrAC = 0.07) or consumed only non-alcoholic beverages. Together with a corrugator ("frown") electromyogram (EMG) measure, startle reflex was used to index the valence of emotional response to the affect-laden pictures, relative to neutral pictures. Results indicated that alcohol did not alter the highly replicable pattern of affective modulation of startle; that is, startle was still potentiated during viewing of unpleasant picture and inhibited during viewing of pleasant ones, both relative to neutral. EMG results corroborated this pattern, suggesting that there was no significant effect of alcohol on emotional response at a moderate level of intoxication when a simple, passive picture-viewing paradigm was used to manipulate affect.

This outcome, however, runs contrary to findings from two subsequent alcohol-challenge experiments in which participants at a similar level of intoxication showed diminished response to startle probes presented during exposure to threat of electric shock (Curtin, Lang, Patrick, & Stritzke, 1998; Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001). Critically, however, in both of these studies the observed anxiolytic effect of alcohol, as indexed by reduced startle potentiation, occurred only under conditions where the threat was paired with a task that competed for participants' attention. When the shock threat was presented alone, the potentiation of startle to probes presented concurrently was comparable across moderately intoxicated and sober participants. The investigators concluded that a moderate level of intoxication can reduce emotional response to aversive stimuli, but that the effect is probably moderated by the complexity and cognitive demands of the context in which exposure to emotional stimuli occurs.

This does not, however, rule out the possibility that at higher levels of intoxication a different process might operate—one in which the impact of alcohol on response to emotional stimuli is direct and independent of competing attentional demands. We sought to test this

[1] Blood alcohol concentration (BAC) is commonly expressed in g/100ml of blood or gms%, often denoted without units, as is the case here. However, as with most alcohol challenge studies involving human participants, the present experiment used estimates of BAC derived from breath alcohol concentration (BrAC), which is analyzed as g/210 l of breath. Numerous studies indicate that these indices are highly correlated (r = .95–0.98, see Jones & Andersson, 2003), and breath testers, such as the ones used in the present study, provide reliable estimates of BAC to two decimal places (Intoximeters, 2006). The effects of different BrACs are, of course, variable dependent upon individuals' tolerance, but .04 is generally regarded as producing mild stimulation/euphoria with relatively minor cognitive impairment, .07 reliably compromises complex cognitive processing and may impair psychomotor performance (note: .08 legally defines intoxication for driving purposes in most states), .10 significantly diminishes cognitive and behavioral control in most drinkers.

hypothesis in the present experiment by using the same simple picture viewing paradigm as Stritzke et al. (1995), but including low-, intermediate-, and high-intoxication conditions as well as a no-intoxication control. This afforded an opportunity to evaluate the effects of levels of alcohol intoxication on response to both positive and negative emotional stimuli in the absence of complex cognitive demands. Based on prior stress-response-dampening research, we predicted that affective modulation of the startle reflex would be altered at higher levels of intoxication such that potentiation of startle during exposure to aversive stimuli would be diminished among these participants, whereas it would remain intact among those with no- or low-intoxication levels. The expected effect of alcohol on inhibition of startle to probes occurring during presentation of positive pictures was more speculative, although there is some reason to believe that this effect might be greatest at low levels of intoxication, which have been associated with mild euphoria (e.g., Little, 1999).

Method

Participants

We recruited undergraduates (n = 101; 48 female), aged 21 or older, from introductory psychology classes for a study of "alcohol and emotion." All prospective participants had to report recent and non-problematic experiences with doses of alcohol comparable to the highest administered in this study. Screening assessments conducted over the telephone included three components. First, self-reports of drinking behavior (average frequency of drinking occasions and average quantity of consumption per occasion over the past year) were collected to evaluate the recent experience criteria. Men said they drank a mean of 1.3 occasions per week (SD = 1.0) and 5.4 "drinks" (12-oz beers, 5-oz glasses of wine, or 1.5 oz distilled spirits "shots" straight or with mixers) per occasion (SD = 2.4) for an average of about seven drinks/week. Women said they drank a mean of 1.2 times per week (SD = 1.0) and 3.5 drinks per occasion (SD = 1.5) for an average of 4.2 drinks/week. High levels of consumption ($>$ 35 drinks/week for men; $>$28 for women) automatically disqualified participants. Second, the Short Michigan Alcoholism Screening Test (SMAST; Selzer, Vinokur, & Rooijan, 1975) was used to evaluate alcohol problems, and participants were excluded if their scores exceeded 2 (the probable alcoholism standard). Third, a detailed medical history questionnaire was administered and persons with histories of any medical condition contraindicating alcohol use were excluded.

Manipulations, Apparatus, Materials, and Measures

Level of intoxication (beverage). Participants were randomly assigned, within gender, to one of four intoxication conditions. A high-intoxication level with a target BrAC of 0.10 was chosen to evaluate alcohol effects on emotional response at a level beyond the legally defined DUI intoxication (0.08 in the U.S.) that is clearly associated with impairment of cognitive and psychomotor performance in most people, which seems to induce stress-response dampening fairly reliably and which is to some extent representative of levels typically attained by drinkers whose patterns of alcohol use might be described as pathological. An intermediate-intoxication condition with a target BrAC of 0.07 was chosen to replicate the level used in many alcohol-emotion studies whose results for emotional responding have proved to be equivocal or dependent on factors other than intoxication alone. A low-intoxication condition with a target BrAC of 0.04 was chosen to capture the possible stimulant or euphoric effects sometimes associated with minimal intoxication (e.g., Little, 1999). Finally, a no-intoxication (zero BrAC) condition served as the comparison group.

The no-intoxication control was used in lieu of a placebo condition for several reasons. First, as noted by Greeley and Oei (1999) in their review of an extensive research literature on alcohol and tension reduction, placebo effects are rarely observed in this arena. Further, they concluded that when alcohol stress-response dampening occurs, it appears to have a purely pharmacological basis, apparently even where self-report measures are concerned (cf. Sher & Walitzer, 1986). Moreover, the dose-response focus of the present study introduced additional practical obstacles to the already difficult task of developing adequate placebos even for a single intoxication level. Hence, the simple no-alcohol control seemed most appropriate to our research goals.

All participants, except those assigned to the no-intoxication group, were told that they would be consuming alcoholic beverages, but were not apprised of the actual dose or its equivalence in standard drinks. They were given beverages consisting of seven parts mixer (orange/cranberry juice combination) to one part 190-proof (95%) ethyl alcohol. Total beverage volume and alcohol dose for each participant were calculated using a computer program (Curtin, 2000) based on total body water volume (estimated using each participant's age, gender, height, and weight) and duration of the drinking period (Watson, 1989).[2] Participants assigned to the no-intoxication group were told that they would not be receiving any alcohol and were given mixer-only beverages in a volume comparable to drinks administered to participants assigned to the intermediate level of intoxication.[3]

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[2] The procedure used to determine alcohol dosage in the present study was developed using formulae available from Watson (1989). It is predicated on the assumption that to reach a target blood alcohol concentration (BAC), the alcohol dose administered should be a function of each participant's total body water (TBW), duration of the drinking period (DDP), time to peak BAC (TPB), and alcohol metabolism rate (MR). Specifically, Alcohol dose (g) = $(10 \times \text{BAC} \times \text{TBW})/0.8 + 10 \times \text{MR} \times (\text{DDP} + \text{TPB}) \times (\text{TBW}/0.8)$. By convention, 0.015 g/100 ml/hr is used as the average metabolism rate for all participants. In addition, it was assumed, based on fasting, beverage concentrations, and drinking rates in this study, that participants reached their peak BAC approximately 0.5 hr after cessation of drinking. TBW was determined from gender-specific regression equations provided by Watson: Men's TBW = 2.447 – 0.09516 × age (years) + 0.1074 × height (cm) + 0.3362 × weight (kg). Women's TBW = – 2.097 + 0.1069 × height (cm) + 0.2466 × weight (kg). Finally, alcohol dose was converted from grams to milliliters by dividing by the density of alcohol at 24 °C, 0.7861 g/ml.

[3] We are aware that that this procedure resulted in consumption of total beverage volumes that, on average, differed systematically across targeted intoxication conditions, with those in the high-intoxication condition consuming relatively larger volumes than those in the low-intoxication condition, with the intermediate- and no-intoxication groups in between. This could produce mild bloating in the high group and perhaps other experiential differences across the groups. However, with a 20-min absorption period following drinking and a restroom break, such effects were judged to be minor and no participant spontaneously reported any discomfort. Also, any alternative procedure (e.g., equating volumes across groups) would involve a trade-off in terms of other variables (e.g., beverage taste). Inasmuch as none of these effects seemed critical to affective modulation of startle in response to the valent images, the simplest approach was applied.

Alcohol use questionnaires. Participants completed an expanded version of the self-report measure of drinking behavior used in the screening. This instrument included a 10-option item for the frequency of alcohol consumption (ranging from *once a month or less* to *21 times or more a week*) and a 10-option item for the number of drinks typically consumed during each drinking occasion (ranging from *less than one drink* to *more than 12 drinks*). In addition, it included a 10-option item for frequency of getting "at least somewhat high or intoxicated" from drinking (ranging from *once a month or less* to *seven times or more a week*) and a 10-option item for the number of drinks required to achieve this state (ranging from *less than one drink* to *more than 12 drinks*). Finally, the questionnaire included a 10-option item for frequency of getting "drunk" (ranging from *once a month or less* to *seven times or more a week)* and a 10-option item for the number of drinks required to feel "drunk" (ranging from *less than one drink* to *more than 12 drinks*). For all items, one "drink" was again defined as one 12-oz beer, one 5-oz glass of wine, or one 1.5-oz shot of liquor (straight or in a mixer). Participants also completed the SMAST again.

Pictures. Stimulus control software (DMDX; Forster & Forster, 2003) was used to present pictures on a 54-cm high-resolution video monitor placed approximately 90 cm in front of participants. Prior to beverage consumption, participants completed a baseline recording session in which they viewed a series of seven solid black geometric shapes against solid white backgrounds. After beverage consumption, participants completed the main testing session in which they viewed a series of 36 pictures from the IAPS (Center for the Study of Emotion and Attention, 1997) used in earlier similar work by Stritzke et al. (1995). The series included twelve pleasant (e.g., erotic or other appetitive items or activities), 12 neutral (e.g., household objects or faces with neutral expressions), and 12 unpleasant pictures (e.g., depictions of assaults or other threats).[4] Pleasant and unpleasant pictures were matched for complementary valence and equivalent intensity using IAPS norms for both genders.

Pictures were ordered randomly within three blocks, each of which contained four pleasant, four unpleasant, and four neutral pictures. Picture order was counterbalanced so that each picture occurred equally often in each block, within each intoxication group, and across the intoxication groups. Each picture was presented for 6 s, and the intervals between pictures ranged randomly from 11 to 17 s.

Startle probes. Acoustic startle probes (50-ms, 100 db white noise, instantaneous rise time) were presented binaurally through ear canal speakers (ER4-S microPro, Etymotic Research, Elk Grove Village, Il) during 4 of the 7 pictures in the baseline recording session and during 24 of the 36 pictures in the main recording session at 3, 4, or 5 s after the onset of the picture. Probes were balanced across picture valence categories.

Startle response. Startle responses were recorded with Ag/AgCl electrodes filled with electrode gel and positioned beneath the left eye in accordance with published standards (Fridlund & Cacioppo, 1986). The locations for the electrodes were cleaned until the measured impedance of the skin was below 5 *K*ohms.

NeuroScan Scan 4.1 software sampled data from SynAmps amplifiers (Neuroscan Labs, Sterling, VA) at 2000 samples/second, with 500Hz low pass, 28Hz high pass, and 60Hz notch filters. Data were reduced off-line using NeuroScan software and scored for base-to-peak amplitude. Individual responses less than within-baseline deflections were scored as no-responses and trials with baseline deflections exceeding 20 μvolts were excluded from analysis due to unstable baseline.

Procedure

Experimental sessions were scheduled to begin in the late afternoon. Participants were instructed to abstain from any alcohol for 24 hours and from any drugs for 72 hours prior to sessions. In addition, they were instructed to fast for four hours prior to their appointments. Participants presented proof of age, signed consent forms, and were administered BrAC tests (Alco-Sensor IV, Intoximeters, St Louis, Missouri) to confirm that all had initial BrACs of 0.00. Urine-sample pregnancy tests (One-Step Dipstick Pregnancy Test, LW Scientific, Tucker, GA) were administered to all female participants, with no non-negative results.

After participants were seated in the testing room, the experimenter applied electrodes and ear canal speakers and ran the baseline recording session. Participants were then notified of their beverage group assignments and consumed half of their total beverage volumes in each of two consecutive 20-min periods (total drinking time = 40 min). After a 20-min absorption period, the experimenter administered a second breath test and conducted the main recording session, and then administered a third breath test. After 15 min, a fourth breath test was administered.

Upon completion of the study, participants were debriefed and detained at the experimental site until their BrACs were at or below 0.02 and were then released to escorts who drove or walked them home. Participants were compensated with research credits for class or cash ($5/hour), and agreed not to drive or consume alcohol or drugs for at least four (4) hours after the experiment. Because participants' targeted levels of intoxication were related to the amount of time that they were required to stay in the laboratory after the experiment (while waiting for their BrAC to fall to 0.02), cash compensation tended to differ across intoxication conditions, with participants in the high-intoxication condition earning relatively more cash money than those in the intermediate group, who in turn earned more than the low-intoxication group, whereas the no-intoxication group earned the least. However, inasmuch as there was no task performance aspect to this study and compensation differences occurred merely as a function of time spent after experimental testing had concluded, there was little reason to think compensation might confound the effects of intoxication level on affective modulation of startle.

Main Data Analytic Strategy

Intoxication Group (no-, low-, intermediate-, and high-) was analyzed as a between-subjects factor and Picture Valence (unpleasant, neutral, pleasant) was analyzed as a within-subjects factor. Gender was also included as a between-subjects factor in all initial analyses, but when it yielded no main or interactive effects pertinent to the main hypotheses, data were collapsed across genders to simplify presentation in the analyses reported here.

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[4] IAPS slide numbers. Pleasant: 4607, 4609, 4641, 4650, 4680, 4690, 5621, 8030, 8034, 8180, 8190, and 8420. Neutral: 2440, 2480, 2570, 2870, 5510, 7010, 7060, 7100, 7130, 7175, 7491, and 9360. Unpleasant: 3060, 3071, 3110, 3530, 3500, 6244, 6260, 6270, 6370, 6510, 6560, and 6570.

Significant effects involving Intoxication Group were decomposed into planned orthogonal contrasts (POCs): POC1 (no- vs. low-intoxication), POC2 (no- and low-intoxication vs. intermediate- and high-intoxication), and POC3 (intermediate-intoxication vs. high-intoxication). These contrasts addressed both ordinal Intoxication Group effects (supported if all POCs proved significant) and potentially contrasting intoxication effects between different levels of intoxication (e.g., a low-intoxication contrast effect, of particular interest for exploring emotional responses to positive stimuli, would be supported if only POC1 were significant). Significant effects involving Picture Valence were decomposed into Negative Potentiation (unpleasant vs. neutral) and Positive Inhibition (pleasant vs. neutral) comparisons to address the effect of Intoxication Group on distinct types of emotional response modulation.

Results

Intoxication Level Groups, Pharmacokinetics, and Possible Covariates

As noted above, alcohol dose was calibrated to achieve targeted levels of intoxication. BrACs measured immediately before and after the main recording session (i.e., at the second and third BrAC assessment) were used to verify attained level of intoxication. Based on the maximum value observed across these measurements, each participant was assigned to one of four attained Intoxication Groups (see Table 1): no-intoxication ($n = 25$); low-intoxication ($n = 26$; peak BrAC range: 0.02–0.05); intermediate-intoxication ($n = 27$; peak BrAC range: 0.06–0.08); and high-intoxication ($n = 23$; peak BrAC range: 0.09–0.10).

Peak BrAC was analyzed using a one-way ANOVA with Intoxication Group as the independent variable. The main effect for Intoxication Group was, of course, highly significant, $F(3, 97) = 812.63$, $p < .001$, $\eta_p^2 = 0.96$,[5] and linear, with p-values $< .001$ for all three POCs (see Table 1). This indicates that the Intoxication groups all differed significantly from each other in terms of peak BrACs. To explore possible differences in pharmacokinetics as a function of intoxication condition, an additional analysis was conducted using only those participants who actually received alcohol. BrAC data from the tests that occurred just before and just after the main recording session were included as a within-subjects repeated variable (Time: Pre/Post) in a mixed-model ANOVA with Intoxication Group (low, intermediate, high) as a between-subjects factor. Again, the main effect for Intoxication Group was highly significant, $F(2, 73) = 236.60$, $p < .001$, $\eta_p^2 = 0.87$. The main effect for Time was also significant, $F(1, 73) = 10.71$, $p < .01$, $\eta_p^2 = 0.03$, although the effect size was small and an examination of the means (see Table 1) revealed a near plateau of BrAC during the approximately 20-min main data recording period. Perhaps more important, the Time × Intoxication Group interaction was not significant, $F(2, 73) = 1.00$, $p = .371$, $\eta_p^2 = 0.03$ indicating no difference in time to peak BrAC or pharmacokinetics across the three intoxication groups in which alcohol was consumed.

Drinking behavior and drinking problems (assessed by the SMAST) were also compared across Intoxication Group and Gender. Due to previously undetected technical problems with self-reports entered by participants via computer, data from four individuals were missing. Analyses of the data from the remaining participants indicated little systematic difference across intoxication groups, although some predictable gender differences were evident (see Table 2). There were no significant main effects for Intoxication Group on any aspect of drinking behavior or on SMAST scores. There were predictable significant main effects for Gender on quantity of drinks per drinking occasion, $F(1, 89) = 21.35$, $p < .001$, $\eta_p^2 = 0.19$; number of drinks required to feel intoxicated, $F(1, 89) = 19.99$, $p < .001$, $\eta_p^2 = 0.18$; and number of drinks to feel drunk, $F(1, 89) = 31.35$, $p < .001$, $\eta_p^2 = 0.26$. Not surprisingly, men said they consumed more drinks per occasion than women did and also required more drinks to feel intoxicated or drunk than women did. However, Gender did not interact with Intoxication Group.

None of the self-reported frequencies and quantities of drinking behavior was significantly correlated with baseline startle reactivity (all rs < 0.10, $p > .10$), so these variables were not included as covariates in any analysis.

Startle Response

Startle response data from the main testing session were analyzed using a 2-way mixed model ANOVA with Intoxication Group and Picture Valence[6] as independent variables.[7] The main effect of Picture Valence was significant, $F(2, 194) = 22.56$, $p < .001$, $\eta_p^2 = 0.19$. Significant effects for the Negative Potentiation comparison (neutral pictures vs. unpleasant pictures), $t(100) = 4.59$, $p < .001$, and the Positive Inhibition comparison (neutral pictures vs. pleasant pictures), $t(100) = 2.56$, $p < .01$, indicated that that startle magnitude was significantly larger during unpleasant pictures and significantly smaller during pleasant pictures, both relative to magnitude during neutral pictures.

The main effect of Intoxication Group was also significant, $F(3, 97) = 12.03$, $p < .001$, $\eta_p^2 = 0.27$. POC1 (no- vs. low-intoxication) was significant, $F(1, 97) = 5.28$, $p = .024$, $\eta_p^2 = 0.05$, indicating that overall startle magnitude was smaller in the low- than in the no-intoxication group. POC2 (no/low- intoxication vs. intermediate/high-intoxication) was also significant, $F(1, 97) = 31.15$, $p < .001$, $\eta_p^2 = 0.24$, indicating that startle was smaller within the intermediate- and high-intoxication groups than within the no- and low-intoxication groups. However, POC3 (intermediate- vs. high-intoxication) was not significant, indicating that startle did not differ between these intoxication groups, $F(1, 97) = 0.17$, $p = .679$, $\eta_p^2 = 0.00$. The observed trend toward reduced vigor of reflexive responding as a function of increasing

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[5] Partial eta-squared (η_p^2) effect size estimates are reported to document the magnitude of either theoretically or methodologically critical effects. $\eta_p^2 = SS_{effect} / (SS_{effect} + SS_{error})$ and is analogous to a squared partial correlation from multiple regression models.

[6] Huynh-Feldt corrected p values are reported for all effects involving the three-level picture valence factor to correct for possible violations of sphericity.

[7] Picture order was included as a between-subjects variable in initial analyses, but no significant main effects or interactions involving this variable were observed. The reported analyses were therefore collapsed across picture orders to simplify presentation. In addition, participants' startle during the main testing session was residualized on their baseline startle to control for individual differences in overall startle magnitude. As expected, baseline startle was positively related to startle during the main session, $F(1, 99) = 184.52$, $p < .001$, $r = .81$.

Table 1
Means for Breath Alcohol Concentrations (BrACs) by Intoxication Group and Gender

	Intoxication group							
Gender	No		Low		Intermediate		High	
Female	(n = 12)		(n = 13)		(n = 14)		(n = 9)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
BrAC	0.00	0.00	0.04	0.04	0.07	0.06	0.09	0.09
Peak BrAC	0.00		0.04		0.07		0.09	
Male	(n = 13)		(n = 13)		(n = 13)		(n = 14)	
BrAC	0.00	0.00	0.04	0.04	0.07	0.07	0.09	0.09
Peak BrAC	0.00		0.04		0.07		0.09	

Note. BrAC unit is g alcohol/210 l breath estimated by breath samples. Pre = the breath test taken immediately before main data recording; Post = the breath test taken immediately after main data recording; Peak BrAC = the mean of the higher of these two measurements for each subject. (Follow-up breath tests administered 15 minutes after the main recording session indicated that both male and female participants in the high-intoxication group had BrAC = 0.08, so their peak was during main data recording). Standard deviations for all BrACs in the alcohol groups were 0.01 (g/210 l).

intoxication is a highly replicable one in studies of both human and non-human subjects, but it is incidental to the effect of intoxication on affective modulation of the emotional valence construct under investigation here.

The critical Intoxication Group × Picture Valence interaction was significant, $F(6, 194) = 2.59$, $p = .021$, $\eta_p^2 = 0.07$ (see Figure 1), and simple effects tests for Picture Valence within each Intoxication Group revealed significant effects of Valence for the no-, $F(2, 48) = 9.37$, $p = .001$, $\eta_p^2 = 0.28$, and low-intoxication groups, $F(2, 50) = 10.77$, $p = .001$, $\eta_p^2 = 0.30$, but not for the intermediate, $F(2, 52) = 2.29$, $p = .134$, $\eta_p^2 = 0.08$, or high-intoxication groups, $F(2, 44) = 2.74$, $p = .097$, $\eta_p^2 = 0.11$. Consistent with these simple effects, the interaction between POC2 and Picture Valence was significant, $F(2, 194) = 6.28$, $p = .002$, $\eta_p^2 = 0.06$, meaning that the Picture Valence effect was larger among participants in the no- and low-intoxication groups ($\eta_p^2 = 0.28$) than among participants in the two higher intoxication groups ($\eta_p^2 = 0.08$). In contrast, neither POC1 nor POC3 significantly interacted with Picture Valence (ps = .373 and .606, respectively), indicating that significant differences in the magnitude of the Picture Valence effect were not evident across these other Intoxication Group contrasts.

Analyses of the Negative Potentiation and Positive Inhibition comparisons were conducted to further examine the observed

Table 2
Means (and Standard Deviations) for Individual Differences in Self-Reported Drinking Variables by Intoxication Group and Gender

	Intoxication group							
	No		Low		Intermediate		High	
Drinking behavior/problem	Female (n = 12)	Male (n = 11)	Female (n = 13)	Male (n = 12)	Female (n = 14)	Male (n = 12)	Female (n = 9)	Male (n = 14)
Frequency drinking	1.8 (1.16)	2.6 (1.48)	2.2 (1.82)	2.3 (1.25)	2.4 (0.84)	2.00 (1.11)	2.3 (1.58)	2.1 (1.45)
Quantity drinking	3.1 (1.08)	5.0 (2.19)	3.8 (1.92)	6.7 (3.22)	3.5 (1.02)	5.7 (3.62)	3.8 (1.48)	5.2 (2.19)
Frequency intoxicated	0.8 (1.15)	1.0 (0.86)	0.9 (0.77)	1.6 (1.27)	1.3 (1.23)	1.1 (0.81)	2.0 (1.80)	1.5 (1.22)
Quantity intoxicated	3.7 (1.61)	5.8 (1.89)	3.8 (1.91)	5.8 (2.62)	3.1 (1.35)	5.3 (2.77)	3.6 (0.73)	4.9 (2.51)
Frequency drunk	0.7 (0.67)	0.6 (0.59)	0.9 (0.85)	1.2 (1.25)	1.0 (1.18)	1.1 (1.08)	1.6 (1.21)	1.2 (0.95)
Quantity drunk	4.5 (1.68)	8.7 (2.37)	4.1 (2.85)	7.8 (2.83)	4.5 (1.94)	7.0 (2.86)	4.2 (1.09)	6.2 (3.04)
SMAST score	0.6 (1.21)	1.0 (0.95)	0.6 (0.79)	0.6 (1.19)	0.6 (0.78)	1.1 (1.18)	0.8 (1.30)	1.1 (1.21)

Note. Frequency variables refer to the number of occasions per week. Quantity variables refer to the number of drinks consumed per occasion; one "drink" = 1 beer, 1 glass of wine, or 1 single shot–straight or mixed. Drinking variables refer to occasions of consuming any alcohol. Intoxicated variables refer to occasions of consuming enough alcohol to feel "somewhat high or intoxicated." Drunk variables refer to occasions of consuming enough alcohol to feel "drunk." SMAST score refers to the total score for the Short Michigan Alcoholism Screening Test.

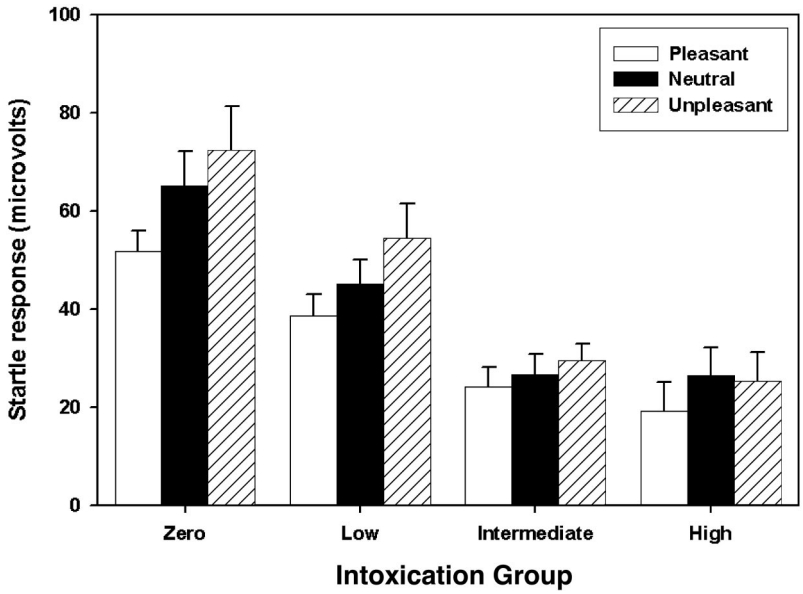

Figure 1. Interaction of intoxication group and picture valence (pleasant, neutral, and unpleasant) to determine magnitude of startle response.

alteration in emotional response within the two higher Intoxication groups. The interaction between POC2 and Negative Potentiation was significant, $F(1, 97) = 4.27$, $p = .041$, $\eta_p^2 = 0.04$. Simple effects tests within each level of this contrast revealed that the Negative Potentiation was significant among participants in the no- and low-intoxication groups, $t(50) = 2.97$, $p = .005$, but not among those in the intermediate and high intoxication groups, $t(49) = 0.43$, $p = .671$. POC1 and POC3 did not significantly interact with the Negative Potentiation in any comparison (ps = .653 and .454, respectively), indicating that negative emotional reactivity to unpleasant pictures did not differ between the no- and low-intoxication groups (POC1), or between the intermediate-intoxication and high-intoxication groups (POC3). Thus, it appears that, although low BrACs were not sufficient to alter emotional response to negative pictures, there was a BrAC above which such responses.

In contrast, the interaction between POC2 and Positive Inhibition was not significant, and simple effects tests within each level of this contrast confirmed that the consistency of Positive Inhibition comparisons across intoxication groups. They were significant for participants within both the no- and low-intoxication groups, $t(50) = 4.43$, $p < .001$, and also within the intermediate- and high-intoxication groups, $t(49) = 2.09$, $p < .05$. Likewise, neither POC1 nor POC3 interacted significantly with Positive Inhibition, indicating that positive emotional reactivity to pleasant pictures was constant across the no- and low-intoxication groups (POC1) and the intermediate- and high-intoxication groups (POC3). Thus, emotional response to pleasant stimuli appeared to be unaffected by Intoxication at any level tested in this experiment.

Discussion

Recent reviews of the relevant literature (e.g., Greeley & Oei, 1999; Lang et al., 1999) cast doubt on the notion that alcohol intoxication directly and selectively attenuates emotional response to aversive stimuli. Indeed, some investigations have provided evidence that the impact of alcohol intoxication on emotional response may be conditional, dependent upon a variety of factors including the context in which affective responses are elicited and/or evaluated (e.g., Curtin et al., 2001), as well as perhaps certain individual differences (e.g., Stewart et al., 1992). In particular, there are indications that, to the extent that defensive responding to unpleasant stimuli is dampened by intoxication, the effect may be mediated by the deleterious impact of alcohol on complex cognitive functions required to process threats when other situational demands compete for attention. Unfortunately, most studies pertinent to these conditional effects have used single doses of alcohol yielding BrACs that did not exceed .07.

Considering results from the limited number of dose-response studies (Sher & Walitzer, 1986; Stewart et al; 1992) and also from some single-dose studies that evaluated effects of doses resulting in BrACs as high as .10 (cf. Sher, 1987), there are indications that stress-response-dampening effects of alcohol might be reliable and relatively independent of context only when levels of intoxication are high. Accordingly, the present study sought to test this premise using a range of intoxication levels and repeated assessments of emotion. To examine the specificity of alcohol effects, we also included evaluation of how level of intoxication influences responses to pleasant as well as unpleasant stimuli using a simple, passive picture-viewing paradigm that involved minimal cognitive demand.

Our results for response to negative emotional stimuli revealed a direct and specific effect of alcohol in dampening defensive responding—even in the absence of competing contextual demands—but only when levels of intoxication were relatively high. In contrast, there was no evidence of a significant impact of alcohol on responses to positive emotional stimuli at any level of

intoxication. The latter null result is of interest in light of so-called "biphasic" effects of alcohol, consisting of the mild stimulation and euphoria that accompany low levels of intoxication, especially on the ascending limb of the blood-alcohol curve, before giving way to sedation and possible tension reduction at higher levels of intoxication and on the descending limb (cf. Martin, Earlywine, Musty, Perrine, & Swift, 1993). However, it is the former, concurrent and contrasting finding of selectively reduced response to aversive stimuli at elevated BrACs that is most intriguing. It suggests that relatively low levels of intoxication are not, in and of themselves, sufficient to diminish defensive responding to unpleasant, threatening, or otherwise negative stimuli. Instead, any anxiolytic effects of drinking at these levels may depend upon the presence of distracting stimuli or task demands that compete with unpleasant stimuli for attention that has been compromised by alcohol (cf. Steele & Josephs, 1990). However, as intoxication level increases, its independent capacity to attenuate responses to such stimuli may increase.

This key effect was evident in the present study, as indicated by significant fear-potentiated startle (i.e., the Negative Potentiation comparison, reflecting the difference in startle responses to probes presented during unpleasant vs. during neutral pictures) among participants in the no-/low-intoxication group, but not among those in the intermediate-/high-intoxication group. Evidently, as the level of intoxication increases, its impact in dampening response to aversive stimuli becomes less dependent on cognitive mediation. This, of course, does not necessarily mean that at higher BrACs alcohol's impairment of cognitive processing becomes irrelevant to its effects on negative emotion—compromised cognition could still exert an additive or even multiplicative effect—but simply that it is no longer required for attenuation of response to stress.

In this context, some comparisons should be drawn between results reported here and those obtained in a prior experiment (Stritzke et al., 1995), using a similar paradigm without the dose-response analysis. The earlier study did not find a significant attenuating effect of moderate alcohol intoxication on startle potentiation during viewing of unpleasant pictures, whereas the present study did, at least when intermediate and high BrAC groups were aggregated in the critical contrast. Importantly, however, there was no significant interaction between Negative Potentiation and POC3 (comparison of intermediate vs. high BrAC), indicating that it was only the combination of Intermediate and High BrAC groups that differed from the No-/Low-BrAC groups (POC2). In addition, although the mean peak BrAC in Stritzke et al. (1995) was comparable to that obtained in the intermediate BrAC condition of the current study, the range of BrACs was wider in that study because it was based on a single dose, whereas the BrAC groups in the present study were developed post hoc, based on BrACs that were actually obtained. Thus, more participants in the earlier study may have fallen below the BrAC needed for direct reduction of emotional response to aversive stimuli. Furthermore, participants per beverage condition in that experiment were smaller than in the current study (18 vs. about 24), thereby reducing the power available in Stritzke et al. to detect an alcohol effect. It is also noteworthy that in two of our group's other studies of the effects of comparable BrACs on responses to cues signaling the delivery of electric shock, rather than unpleasant pictures as the aversive stimuli (Curtin et al., 1998, 2001), we found alcohol had no impact on fear-potentiated startle when the cue was presented alone, i.e., in the absence of competing stimuli or tasks. This suggests that the effects of alcohol on emotional responses to negative stimuli may vary as a function of the potency of the affective cue (i.e., the directness of its association with punishment) as well as BrAC. Higher doses of ethanol may be required to block fear associated with potent aversive cues such as shock.

Regarding Positive Inhibition, neither Stritzke et al. (1995) nor the present experiment yielded any evidence that alcohol facilitated the typical attenuation of startle during viewing of pleasant pictures relative to neutral pictures. Instead, such attenuation was observed more or less uniformly across all levels of intoxication in this study, without any amplification in the low-intoxication condition as might have been expected based on biphasic effects of alcohol. However, it is not altogether clear that a general increase in tonic positive affect or in a specific feeling of mild euphoria or stimulation due to low levels of intoxication represents a robust phenomenon or one that should necessarily be manifested in a synergistic interaction with the viewing of pleasant pictures to further reduce startle. Nonetheless, it is noteworthy that attenuation of startle response in the presence of pleasant stimuli remained intact across all doses of alcohol because it argues against the possibility that the contrasting reduction in augmentation of startle response in the presence of unpleasant stimuli observed at higher levels of intoxication was attributable to an overall dampening in the startle response or related insensitivity of the affective modulation paradigm. Moreover, the proposed interpretation of selective effects on the defensive motivation system resonates with the disinhibitory effects of alcohol observed in classic reward-punishment ("conflict") paradigms in animals (cf. Gray, 1987), and with the well-known facilitating effects of alcohol on stimulation seeking and risk taking in humans (e.g. Cohen & Fromme, 2002; Dunn, Bartee, & Perko, 2003).

In this research, we share the conceptualization of emotional response as a hierarchical process in which subcortical motivational systems directly activate positive-appetitive and negative-defensive action tendencies that are modified by higher brain systems (Lang, 1994; LeDoux, 1995). As a function of this interplay, impairments in cognitive functions such as divided attention, memory, and context processing can lead to alterations in affective processing. Substantial data exist to indicate that higher, controlled processes of this kind begin to show impairment at relatively modest levels of intoxication in humans (see Holloway, 1994, for a review). At these levels, affective processing that relies on such higher cognitive functions is likely to be compromised. Support for this hypothesis comes from studies indicating that alcohol impairs contextual fear learning in non-human animals (e.g., Melia, Corodimas, Ryabinin, Wilson, & LeDoux, 1994) and threat-cue processing under conditions of distraction in humans (Curtin et al., 1998, 2001), but further research will be needed to clarify the role of cognition in the effects of alcohol on emotional response to pleasant stimuli.

Among the additional points deserving mention here is the obvious observation of a general linear effect of intoxication on the absolute magnitude of startle, such that there was an overall decrease in startle as the level of intoxication increased. This robust effect was one that has been reported consistently across a range of species and paradigms. Startle, however, is a reflexive

response and care must be taken not to confuse this main effect with the dampening of traditional sympathetic nervous system indices (e.g., skin conductance response [SCR]) typically linked to the arousal component of emotional state. Of course, it is possible that the general decrease in startle response associated with rising levels of intoxication could reflect a reduction in activity at the level of the nRPC, which, in turn, could reflect an overall reduction in arousal state. In other words, nRPC activity might covary with general brainstem reticular activation. However, this is by no means a given and other measures (e.g., SCR, EEG) would be needed to substantiate this interpretation. Even then, its implications for our use of startle to index emotional valence are unclear as we were able to take advantage of the way in which it reliably covaries with the pleasantness or unpleasantness of emotional state to track changes in affective responding as a function of increasing alcohol intoxication.

Several other possible uncertainties and related directions for future research emerge from reflection on this study. First is the advantage that might accrue from the use of noise probes of varying intensity to rule out possible scaling problems due to the main effect of alcohol in reducing absolute startle magnitude. Specifically, it would be interesting to include higher and lower intensity probes as a means of equating baseline startle levels across dose groups to facilitate comparisons of modulatory effects.

Some consideration might also be given to moving beyond exclusive use of static pictorial stimuli (IAPS images) to elicit emotional responses. Evaluation of alcohol effects on responses to more varied, dynamic, and contextually relevant stimuli would be attractive. Although our paradigm for studying affective modulation of startle has well-established reliability and internal validity in the assessment of changes in overall aspects of emotional responding, its external validity appears to be somewhat limited. Certainly, the effects of alcohol on emotional responding could vary as a function of the nature and potency of affective cues and the context in which they occur, as well as a consequence of intoxication level.

In connection with alcohol administration, possible biphasic and limb effects on the blood-alcohol curve and their potential impact on emotional response were not adequately captured by our study's design. Calculations of total alcohol doses and the timing of the absorption period were made to ensure that the main recording session coincided with peak levels of intoxication, and analyses of BrAC measurements before and after this period indicated that comparable pharmacokinetic profiles were achieved across the intoxication groups. Thus, although the study was able to examine differences in emotional responding associated with differences in peak levels of intoxication, it could not evaluate pharmacokinetic variations in alcohol effects on emotional responding, so a fuller understanding of them will have to await future research.

One final consideration for future research would be assessment for the effects of individual difference variables, such as family history of alcohol-related problems and personality factors associated with risk for alcohol use and abuse. Previous studies have highlighted the relationship between certain of these variables and differences in stress-response dampening effects of alcohol (e.g. Sher & Levenson, 1982; Stewart et al., 1992), so their exploration as both moderators and mediators of affective responding under alcohol could prove interesting.

In summary, the present study contributes to the literature on alcohol and emotional response by providing new evidence of an interaction between level of intoxication and valence of the stimuli used to elicit affective reactions. The findings suggested a selective reduction of responses to negative stimuli, but only at higher levels of intoxication. Specifically, alcohol blocked fear-potentiated startle at relatively high levels of intoxication, even in the absence of stimuli or tasks that competed for attention to aversive cues. This suggests that at elevated levels of intoxication, alcohol appeared to directly and selectively compromise emotional responding associated with the subcortical defensive system, impairing affective reactions to explicit threat cues. However, when alcohol affects emotional distress or defensive responding at lower levels of intoxication, it appears to depend at least partly on the compromising of higher cognitive processing in contexts that involve competition for attention to threats. Our data also provided evidence of asymmetric effects of intoxication on appetitive versus defensive systems, with inhibition of startle in the presence of pleasant or appetitive stimuli remaining relatively impervious to any of the levels of intoxication tested. This result could help to account for the well-known disinhibitory effects of alcohol on goal-directed behavior under conditions of conflict.

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