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Alcohol and Fear-Potentiated Startle: The Role of Competing Cognitive Demands in the Stress-Reducing Effects of Intoxication

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Effects of alcohol and cognitive demands on reactions to threat were examined using startle response potentiation to index negative emotion. Men and women received nonalcoholic or alcoholic beverages prior to a series of trial blocks, signaled by light cues indicating that shocks might be delivered ("threat" blocks) or that none would occur ("safe" blocks). Within half of the blocks, participants intermittently viewed pleasant photographic slides. Alcohol attenuated overall startle reactivity, but robust fear potentiation (larger startle magnitudes and shorter latencies during threat versus safe blocks) did not differ by beverage condition. Decomposition of the Beverage × Threat × Slide interaction revealed significant fear potentiation in all conditions, except the one in which alcohol was combined with slides. Thus, dampening of stress response by alcohol may depend on diminished ability to process competing cognitive demands.

Despite its apparent importance to key theories of drinking and alcoholism, the general notion that alcohol reduces tension or dampens response to threatening stimuli has emerged from more than 50 years of intense empirical scrutiny without strong support. Even greater doubt surrounds speculations regarding the processes that might underlie such effects, if and when they occur (Greeley & Oei, in press). Such equivocation and uncertainty are increasingly being interpreted as signs that intoxication does not invariably reduce distress but rather does so only under certain conditions and through mechanisms more complex than direct cause (e.g., Sayette, 1993; Steele & Josephs, 1990). Moreover, indications are that improved understanding of the relevant phenomena will be expedited if alcohol researchers become more willing to embrace contemporary theories and methods of the broader field of emotion (cf. Stritzke, Lang, & Patrick, 1996). We have already made some progress in this arena (e.g., Stritzke, Patrick, & Lang, 1995) and sought to build on it by examining the potential role of competing cognitive demands in alcohol's effect on reactions to threat. This work applied a multidimensional conceptualization of emotion and incorporated state-of-the-art psychophysiological measurements of affect.

During the decade of the 1980s, much of the research on drinking and negative affect revolved around the stress-response dampening (SRD) model (see Sher, 1987, for a review). Proponents of this approach asserted that alcohol intoxication reduces emotional response to aversive stimuli largely by suppressing autonomic reactivity to stress and that this effect could contribute to the reinforcing value of drinking—particularly when it occurs in potentially stressful contexts. Support for the direct attenuation of the stress response in intoxicated participants, however, has been inconsistent. The number of studies in which an SRD effect of alcohol has been found is nearly equaled by the number in which alcohol has shown little or no effect, or even an augmentation of the stress response (Sayette, 1993). Partially in response to the apparent complexity of the relationship between alcohol consumption and stress, theorists have begun to consider possible mediators and moderators of this association, and cognitive mechanisms have figured prominently in these efforts.

Alcohol and Information Processing

It is widely acknowledged that acute alcohol intoxication results in numerous and diverse impairments in cognitive functioning, including deficits in encoding of new material and in retrieval of previously learned information (see Holloway, 1994, for a review). Of particular importance is the consistent and markedly deleterious effect of intoxication on performance of dual tasks involving "divided attention" (i.e., simultaneous attention to competing demands). For example, Moskowitz and Sharma (1974) showed that alcohol-intoxicated people had significantly more difficulty than sober ones in detecting a visual stimulus presented in the periphery when a more central stimulus was presented simultaneously. However, no such differences in performance due to alcohol were observed when the peripheral stimulus was presented alone. Apparently, when intoxication diminishes cognitive capacity to the point where resources are insufficient to respond to competing demands, people devote their limited capacity to central stimuli or tasks, thereby compromising the processing of more subtle or peripheral signals.

Such observations are at the foundation of the attention-allocation (A-A) model (Steele & Josephs, 1990), a relatively re-

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cent and influential formulation developed to explain how alcohol affects emotional and other psychologically important responses and to account for some apparent inconsistencies in research on these phenomena. This approach posits that alcohol intoxication reduces attentional capacity and allocates that which remains on the basis of the "salience" of the stimuli vying for attention. Thus, when alcohol is consumed under stressful conditions but in the presence of "distracting" (and presumably benign) stimuli, some of the drinker's limited attentional capacity should be diverted away from the stressor. The predicted result is a reduction in distress. In contrast, if alcohol is consumed in the absence of a competing stimulus, no stress reduction is predicted because the focus of whatever attention is available should remain on the stressor. Josephs and Steele (1990) have further argued that in this context, alcohol intoxication might even lead to an intensification of the stress response (the "crying-in-one's-beer" effect) by serving not only to focus the inebriate on the stressful cues but also to reduce the likelihood that effective coping responses will be enacted.

Although the concepts of "salience" and "distraction" are potentially troublesome in the context of alcohol and emotion research,[1] and there is not a well-developed empirical or theoretical base for the notion that alcohol intoxication might actually *increase* the distress occasioned by a threat if there is no other stimulus to compete with it,[2] the overall A-A model remains intuitively appealing. To date, however, it has not been subjected to extensive, methodologically rigorous evaluation. All of the pertinent research has relied exclusively on self-report as the means to assess affective state and has not systematically assessed the key parameters of emotional valence and arousal thoroughly. Moreover, experiments conducted thus far have not succeeded in evaluating emotional response at the exact point when both threatening and competing stimuli were present, as the model seems to require. Thus, we sought to advance the status of relevant investigation by applying a more contemporary conceptualization of emotion, together with a sophisticated approach to the measurement of this complex, multidimensional construct. Our design also provided for more precise temporal management of threat as it relates to competing stimuli.

Emotion, Startle, and Intoxication

Contemporary theories of emotion conceive of affective states in terms of "action dispositions" or readiness to engage in adaptive behaviors reflecting the basic motivations to avoid harm and enhance pleasure (e.g., Lang, Bradley, & Cuthbert, 1990). This view maps onto an underlying structure of emotion involving two primary motive systems in the brain: an aversive system governing defensive reactions and an appetitive system governing approach and consummatory behaviors (cf. Gray, 1987). These primitive motivational systems, evident in virtually all mammals, are subcortically based (Lang, 1994) and reflect the valence (pleasantness) dimension of emotion that can be activated at various levels of arousal or intensity (cf. Russell & Mehrabian, 1978). They play a key role in elementary conditioning processes (Konorski, 1967) and broadly organize affective mobilization and expression (Lang, 1995). However, these systems also interact with other regions of the brain—for example, centers governing higher "declarative" memory (LeDoux, 1995)—thereby permitting emotions to be influenced by learning history, by ongoing information processing, and by the unique demands of eliciting circumstances. Consequently, a defensive emotional disposition such as fear can be evoked by a simple sensory cue (e.g., a light signaling shock) or by a complex symbolic stimulus (e.g., a verbal description or image associated with threat), and its overt expression can take widely different forms depending on the context.

Treating emotional reactivity as a reflection of the intricate interplay of primitive action-mobilization centers with other brain centers, including those involved in higher informational processing, enables one to accommodate a wide variety of specific emotions and emotional blends. Moreover, it points to ways in which alcohol might influence affective reactions in a "top-down" manner rather than through direct alteration of emotional processing at the fundamental level of appetitive or defensive motive systems (Stritzke et al., 1996).

Given this multidimensional, multilevel conceptualization, it is evident that meaningful analysis of emotional response must consider more than just peripheral activation as typically indexed by changes in autonomic arousal (e.g., heart rate or skin conductance response). Such measures of reactive intensity are important but do not adequately capture the valence dimension of affect. It is within this context that the startle reflex emerges as a powerful index of affective response. Extensive research has shown that reflexive startle response to a noxious stimulus probe (e.g., a sudden, loud noise burst) can provide valuable information about an organism's underlying emotional set (Lang et

[1] We believe continued use of the terms *salience* and *distraction* or their variants is troublesome for research on cognitive factors in alcohol and emotion because they have not been clearly defined in the relevant literature and seem to unnecessarily cloud understanding of the phenomena and processes under consideration. The answer to the question of what makes a stimulus *salient* seems to be "that one attends to it," which also seems to be what defines a stimulus as *distracting*. In our view, this does not advance understanding. It is the fact that two or more stimuli or tasks simultaneously demand attention and other cognitive processing resources that makes them important to the alcohol–emotion relationship. Thus, we have chosen to frame our exposition in terms of *competing* stimuli, demands, or tasks.

[2] Four A-A experiments, reported in two articles (Josephs & Steele, 1990; Steele & Josephs, 1988), were characterized by Steele and Josephs (1990) as "reliably" showing an anxiogenic effect of alcohol intoxication when a "strong, upcoming, and salient" stressor was presented in the absence of "distraction" (p. 931). However, a careful reading of these articles suggests that such an effect is, at best, a relatively weak one. In three of the four experiments, the alcohol/no-distraction condition did *not* produce significantly greater changes (increases) in anxiety than those observed in at least one of the critical comparison conditions. Even accepting that a combination of data from these experiments yielded a reliable pattern, the mechanism underlying such an effect is elusive and seems distinct from that understood to be responsible for the anxiolytic effect of alcohol. The abundant evidence that alcohol diminishes overall attention and compromises performance on divided-attention tasks is relatively easy to apply to the reduced distress observed in individuals whose exposure to threat coincides with exposure to competing stimuli. It is far less clear why alcohol alone would increase one's response to threat. Thus, our focus here is on the role of alcohol, together with competing cognitive demands, in attenuating negative emotional response to aversive stimuli.

al., 1990). Specifically, it appears that this defensive reflex is enhanced when it matches the ongoing action disposition (i.e., the disposition is also defensive) and that the reflex is inhibited when elicited in the context of an opposing, appetitive action set. In this way, differences in reflexive reactions to startle probes provide a sensitive and reliable index of the valence of the underlying affective state.

The augmentation of startle in animals during exposure to threat or fear cues, an effect referred to as *fear-potentiated startle,* has been observed for a variety of conditioned fear stimuli (both auditory and visual) and across different probe modalities (Davis, 1986). Recently, this research has been extended to humans through a series of laboratory studies demonstrating that participants reliably exhibit exaggerated startle responses when exposed to probes in the presence of a light cue signaling the potential administration of shock (Grillon, Ameli, Woods, Merikangas, & Davis, 1991; Hamm, Stark, & Vaitl, 1990). These results fortified earlier work (Vrana, Spence, & Lang, 1988) showing that the magnitude and latency of human eyeblink response to startle probes were modulated by simultaneous exposure to photographic slides depicting pleasant, neutral, or aversive emotional content.

Of particular relevance to the current study is the examination of drug effects on fear-potentiated startle. Anxiolytic drugs (e.g., diazepam and flurazepam) diminish the usual potentiation of startle in animals exposed to conditioned fear stimuli, without affecting baseline startle reactivity (Davis, 1979). Conversely, anxiogenic drugs (e.g., piperoxan and yohimbine) facilitate fear-potentiated startle while leaving baseline startle intact (e.g., Davis, Redmond, & Baraban, 1979). Finally, a selective anxiolytic effect of diazepam on fear-potentiated startle—again, without concomitant attenuation of overall startle magnitude—has also been demonstrated in humans (Patrick, Berthot, & Moore, 1996).

Although animal researchers have not evaluated the effects of alcohol on fear-potentiated startle, they have examined the effects of alcohol on overall startle reactivity. A significant suppression of the startle reflex has been reliably observed in rats exposed to an ethanol challenge (Brick, Pohorecky, Faulkner, & Adams, 1984; Deturk & Pohorecky, 1987; Pohorecky, Brick, & Carpenter, 1986). Moreover, this decline in reactivity is dose dependent, with greater attenuation occurring at higher doses.

In the first investigation of alcohol and startle in humans, Stritzke et al. (1995) studied the effect of alcohol on both overall startle reactivity and on affective modulation of the startle reflex in a sample of social drinkers. After consuming a moderate dose of ethanol or an equivalent volume of a nonalcoholic beverage, participants were exposed to a series of pleasant, neutral, or unpleasant photographic slides. Consistent with findings for animals, overall startle reactivity was diminished by alcohol. Despite this reduction in baseline reactivity, however, there was no selective effect of alcohol on responding as a function of slide content. In particular, alcohol and control groups both showed robust and comparable levels of the potentiated startle effect in connection with probes introduced during viewing of the aversive slides. Such results cast doubt on the notion that alcohol acts selectively to dampen reactivity to unpleasant stimuli.

Although these findings are troublesome for proponents of a direct SRD model of alcohol effects on emotion, the absence of a selective effect of intoxication on the startle index of affective disposition can be accommodated by more cognitively oriented theories. Those subscribing to such a perspective would argue that cognitive complexity and related features of the stimulus situation play a key role in determining whether alcohol dampens responses to stress. With this thesis in mind, we designed the present experiment to replicate and extend the findings of Stritzke et al. (1995), while offering a more conceptually and methodologically sophisticated test of the A-A model than has been conducted to date.

The Present Study

Stritzke et al. (1995) investigated the effects of alcohol on overall startle reactivity and on affectively modulated startle in humans, but because this is the only such study in the literature, it warranted the conceptual replication reported here. Moreover, it remained to be determined whether the absence of a selective attenuating effect of alcohol on startle in the presence of unpleasant photographic slides is indicative of a more general inability of alcohol to reduce fear. Thus, here we chose threat of electric shock to manipulate affective state because of its greater potency and its more direct connection to fear.

In addition to replicating and extending key features of Stritzke et al. (1995), we sought to test predictions arising from the A-A model of alcohol and emotional reactivity through an analysis of the effects of concurrent exposure to competing stimuli. To do so, the presence or absence of pleasant slide stimuli was systematically manipulated concurrently with threat, across beverage conditions. Prior studies of the A-A model have relied primarily on self-reports of distress collected *after* withdrawal of a competing task that occurred during exposure to a protracted, ongoing threat. This paradigm precludes examination of the *simultaneous* effects of threat and competing stimuli thought to be crucial to alcohol's action in reducing distress. By using psychophysiological measures of affect, we were able to assess the influence of competing stimuli "on line" (i.e., simultaneous with threat). In addition, whereas most prior studies of the alcohol–stress link have relied primarily on heart rate change and self-report, we used multiple measures of autonomic arousal and included the startle probe reflex as a specific index of emotional valence (fear). Finally, our paradigm allowed for repeated administrations of the stressor, an approach designed to increase measurement reliability.

The primary goals and hypotheses of the current study were as follows:

1. *To replicate and extend initial findings concerning the effects of alcohol on affect-modulated startle.* On the basis of Stritzke et al.'s study (1995) and related animal studies, it was predicted that (a) alcohol would reduce overall startle reactivity. In addition, given the lack of consistent evidence of a selective effect of alcohol on negative affect in humans, it was hypothesized that—contrary to a simple, direct model of SRD—(b) *both* alcohol and no-alcohol control groups would exhibit significant fear-potentiated startle.

2. *To evaluate predictions, derived from the A-A model, regarding fear-potentiated startle.* Our primary hypothesis was that (a) fear-potentiated startle would be reduced by alcohol only when intoxication was combined with a competing stimulus (viz., a pleasant slide) capable of capturing attention. Support for this hypothesis would take the form of a significant Beverage × Threat × Slide interaction, with simple effects analyses re-

vealing significant fear-potentiated startle in all conditions, except the one combining alcohol with competing stimuli (i.e., slides). We were also able to evaluate the less compelling hypothesis that (b) alcohol intoxication in the absence of competing stimuli might increase fear potentiation. Support for this notion would derive from a significant Beverage × Threat interaction, with simple effects tests showing greater potentiation under alcohol/no-slides than no-alcohol/no-slides conditions.

Method

Participants

Forty-eight students (24 men) seeking to complete a research participation requirement were recruited from introductory psychology classes at Florida State University. Inclusion criteria specified that participants should be at least 21 years old, have recent experience with the doses of alcohol comparable with those to be administered in our study, have no history of alcohol-related problems or any medical condition that might contraindicate alcohol consumption, and have a commitment to arrange safe transportation home from the experimental site at the conclusion of participation. Appropriate volunteers were scheduled and instructed to abstain from all drugs for at least 24 hr and from all food and beverages for at least 4 hr prior to arrival for appointments.

Procedure

Consent, screening, and baseline. On arrival for the experiment, each participant was seated in a comfortable room where later drinking would occur. All signed consent forms which included an agreement to remain at the research site until their blood-alcohol levels (BALs) were sufficiently low to permit safe release. They also completed a drinking and medical history questionnaire, providing more detailed coverage of the queries already made during initial telephone contact. All women were subjected to a urine sample pregnancy test (ICON II HGC by Hybritech of San Diego, CA) during this initial period, with a negative result required for further participation.

Following consent and screening procedures, eligible participants were escorted to the psychophysiology lab for baseline measures. A breath sample was collected to verify an initial BAL of 0.00 (BAC Verifier by Verax Corporation of Fairport, NY). Electrodes were attached so baseline data could be collected on all physiological measures used in the experimental session. The format of the baseline session was a modified and shortened version of the experimental session. Participants were presented with four 40-s trial blocks. Two of these were designated "slide" blocks and contained two 6-s slide presentations of a simple character display (viz., a "+" sign). In the interval between slide presentations, a uniform gray background was projected onto the screen. The remaining two blocks were designated "no slide" blocks. During these blocks, only the gray background was projected for the entire block. The start of each block was accompanied by illumination of either a red or green light (balanced across slide and no-slide blocks) located beneath the slide screen. Although these lights were used as discriminative stimuli for the shock threat manipulation in the main session, no information about the lights was given to participants during the baseline session. After this preliminary assessment, electrodes were removed and participants returned to the drinking room.

Beverage manipulation. Half of the male and half of the female participants were randomly assigned to the alcohol condition. This group received a beverage containing orange juice mixed with pure ethyl alcohol in a 6:1 ratio. They were accurately informed of their beverage condition, were instructed that the dose they received was roughly equivalent to 3 or 4 standard drinks in 1 hr for a 150-lb (approx. 68-kg) person, and were permitted to observe the digital readout during later breath test analyses. The ethanol dose needed to produce the target peak BAL of 0.075 g/100 ml at the start of the main experimental session was computed for each individual participant, using a computer program (Curtin, 1995a) developed for this purpose.[3] Participants assigned to the no-alcohol group received only orange juice in a volume equivalent to the total amount that would have been administered had they been in the alcohol condition. They were told simply that they were in the no-alcohol comparison group.[4] All beverages were evenly divided into two drinks, each to be consumed in 20 min, for a total drinking period of 40 min. A 20-min absorption period followed the drinking. After the drinking phase, participants were returned to the psychophysiology lab room where recording electrodes were reattached.

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[3] 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 BAL, 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 BAL (TPB), and alcohol metabolism rate (MR). Specifically,

$$\text{Alcohol dose (g)} = (10 + \text{BAL} + \text{TBW})/0.8 + 10 \cdot \text{MR} \cdot (\text{DDP} + \text{TPB}) \cdot (\text{TBW}/0.8).$$

We used 0.015 g/100 ml/hr as the average metabolism rate for all participants. In addition, we assumed that participants reached their peak BAL at 0.5 hr after cessation of drinking. TBW was determined from gender-specific regression equations provided by Watson:

$$\text{Men's TBW} = 2.447 - 0.09516 \cdot \text{age} + 0.1074 \cdot \text{height (cm)} + 0.3362 \cdot \text{weight (kg)}.$$

$$\text{Women's TBW} = -2.097 + 0.1069 \cdot \text{height (cm)} + 0.2466 \cdot \text{weight (kg)}.$$

Finally, alcohol dose was converted from grams to milliliters by dividing by the density of alcohol at 24°C, 0.7861 g/ml. Application of this procedure in several recent studies in our lab has resulted in both exceptional accuracy and minimal variability in observed peak BALs—especially relative to what is typically reported in alcohol challenge studies. A simple Windows-based program that performs these calculations is available from Alan R. Lang upon request.

[4] The decision to use a no-alcohol, as opposed to a placebo, comparison group was a reasoned one. First, as noted by Greeley and Oei (in press) in their review of the last decade of alcohol and tension reduction research, placebo effects are rarely observed in this area. They further concluded that the majority of SRD effects, when present, appear to have a clearly pharmacological basis. This, of course, does not mean that a placebo condition might not help make a more convincing case, even if only by producing null results. However, because this was the first study to investigate the A-A predictions by using more sophisticated measures of affect obtained in the context of manipulations of competing stimulus presentations, we felt use of an extreme-groups design was justified. Basically, we first wanted to demonstrate that there was an effect unique to the combination of alcohol and competing stimuli before turning to a more fine-grained analysis of its underlying causal mechanisms (i.e., expectancy vs. pharmacology). We also worried about the effects of a possible artifact in the form of suspicion that invariably accompanies attempted placebo deceptions. Specifically, we were concerned that participants in a placebo condition would devote some of their cognitive resources to trying to figure whether they had actually received alcohol or trying to determine how much they had received. If so, this might constitute an unintended, competing cognitive task and thereby cloud interpretation of results.

Stimulus materials and counterbalancing. Each participant viewed 24 positively valenced (pleasant) color slides depicting content from the following domains: appetizing foods, erotica, stimulating action scenes, and cute children and animals. These competing stimulus slides were selected from the International Affective Picture System (Lang, Ohman, & Vaitl, 1988), a large and varied pool of slides for which normative rating data on valence, arousal, and dominance are available. All slides were chosen for their high positive valence and high arousal.

The slides were arranged into four blocks of six slides each, such that the content across blocks and the order of content within blocks was balanced. Each of the four content categories occupied the first slide position in one of the four blocks. In addition, assignments were made to ensure that slides in each block were approximately equivalent on standardized ratings of valence and arousal.

The acoustic startle stimulus consisted of a 50-ms presentation of a 100 dB burst of white noise with instantaneous rise time. It was produced using a Coulbourn white noise generator (Coulbourn Instruments, Allentown, PA), gated through an audio mixer amplifier. Each block contained six startle probes: three during slide presentation and three during the intervals between slides (i.e., during presentation of gray foreground). Probes occurred randomly during the 6-s slide presentation (either 3, 4, or 5 s after slide onset). A similar format was followed for startle probes presented between slides, with probes occurring at 7, 8, or 9 s into the presentation of the gray foreground. Two counterbalancing conditions were formed such that, across participants, each slide had an equal probability of being probed. In addition, timing of the startle stimulus during slide presentation was balanced such that each slide category received an equal number of startle probes at each of the three startle times.

During the experimental session, each participant sat in a comfortable chair positioned approximately 2 m from the slide screen in a dimly lit, sound-attenuated room. Two Kodak slide projectors (Eastman Kodak, Rochester, NY), one for the color slides and one for the neutral gray background presented in the absence of the color slides, were located in an adjoining room. A small rectangular metal box containing a red and green light (positioned vertically with red light on top) was located beneath the screen, approximately 1 m above the floor. A device, conspicuously labeled *shock generator,* was positioned right next to each participant's chair so leads from it could be easily attached to their right forearms. Also, to the participants' right was a wall-mounted Panasonic video camera (Panasonic Corp., Denver, CO) used for monitoring throughout the session.

Physiological measures. The presentation and timing of stimuli, and the collection of physiological, self-report, and viewing-time data, were controlled by an IBM-compatible PC equipped with Virtual Psychophysiology Monitor (VPM) stimulus control and data-collection software (Cook, Atkinson, & Lang, 1987).

The eyeblink component of startle responses was measured by recording activity from the orbicularis oculi muscle beneath the left eye. A Coulbourn HiGain Bioamplifier (Coulbourn Instruments, Allentown, PA) was used to amplify the raw electromyogram (EMG) signal, and frequencies below 90 Hz and above 250 Hz were filtered out. The integrated signal (time constant = 80 ms) was sampled at 1000 Hz for 50 ms preceding, and 250 ms following, each startle stimulus onset. Eyeblink data were reduced off-line using a program (Winstar; Curtin, 1995b) that scores startle-elicited blinks for amplitude in arbitrary analog to digital (A–D) units and for onset latency in ms.

To explore additional indices of positive and negative emotional reactivity, facial EMG activity indicative of the valence of expression (corrugator for frown, zygomatic for smile) were recorded unilaterally (left) with Ag-AgCl miniature electrodes positioned in accordance with published guidelines (Fridlund & Cacioppo, 1986). This EMG activity was sampled at 20 Hz throughout each trial block and for the 5 s preceding each block.

To provide opportunities for comparison with typical alcohol and affect research, heart rate (HR) was recorded, using standard Ag-AgCl electrodes placed on the inside of participants' left and right forearms. The signal was filtered using a Coulbourn Bioamplifier and a Schmitt Trigger (Coulbourn Instruments) that interrupted the computer each time it detected a cardiac R-wave. Interbeat intervals were recorded to the nearest millisecond for the entirety of each trial block, as well as for the 5 s preceding each block.

Skin conductance (SC), included as an index of general physiologic arousal commonly used in emotion research, was recorded from standard Ag-AgCl electrodes placed on the hypothenar eminence of the left palmar surface. The electrodes were filled with Unibase paste thinned slightly with physiological saline, as recommended by Lykken and Venables (1971). The signal was acquired by a Coulbourn S71-23 skin conductance coupler (Coulbourn Instruments), which produces a constant 0.5 V across the electrodes. Tonic SC was recorded continuously, and phasic skin conductance response was obtained for both the cue onset (i.e., the red and green lights) and the slide stimulus onset.

Experimental task instructions. After beverage absorption, participants were escorted to the session room and seated in a padded recliner. Electrodes for the physiological measures were attached, and an overview of the session and specific instructions were given. Just prior to the start of the session, a second breath sample was collected for BAL testing.

Participants were told that they would be presented with a series of trial blocks and that the onset of a colored light would mark the start of each block and define its nature. If the light was red, the block was a "shock" block, and participants were warned that an electric shock might be administered at any time during such a block. They were advised that if the light was green, the block was "safe" and no shock would be administered. The colored light remained on during the entire block, and the offset of the light signaled the termination of that block.

Participants were informed that they could receive up to three electric shocks over the course of the entire experiment, but that they might not receive any. They were also instructed that shock intensity would increase with time (i.e., any shock received later in the session would be more intense than those administered early). Although no shocks were actually administered to any participant in this procedure, similar threat manipulations have proved successful in maintaining elevated arousal and distress across extended periods (e.g., Grillon et al., 1991).

Participants were instructed to attend to the screen at all times and that, during some blocks, slides would be presented intermittently. They were further informed that, in the absence of slides, only a gray background would illuminate the screen. They were also told that from time to time a brief noise resembling loud static (the startle probe) would be heard through the headphones they wore, but that they should disregard it. No additional information was offered. Eight trial blocks, each 2.5 min in duration, were presented. The interval between blocks varied randomly from 15 s to 25 s. Blocks alternated between threat and no-threat conditions (cf. Grillon et al., 1991). Four consecutive blocks contained slides, and the other four contained no slides. Controlling for beverage group and gender group, half of the participants were randomly assigned to each order (slide blocks first or no-slide blocks first). During each slide block, participants viewed six slides, each for a 6-s duration. In the interval between each slide, and throughout all no-slide blocks, a gray background was projected onto the screen. The time between slides ranged from 13 s to 19 s (M = 16 s). Slide order (i.e., slide blocks first or no-slide blocks first) and threat order (i.e., first block threat or no threat) were balanced across conditions. After this main phase of the experimental session, a third BAL test was conducted.

Postsession slide rating. Following completion of the above procedures, participants were presented with the same 24 slides that they had viewed in the experimental session. They were instructed to view each slide for as long as they chose (maximum = 30 s) and then to press a

button to terminate the slide presentation. Elective viewing time (in ms) was recorded for each slide as an unobtrusive index of interest. After the termination of each slide, participants rated their affective experience of the slide on dimensions of valence and arousal, using a computerized version of the Self-Assessment Mannequin (SAM; Lang, 1980). Participants also rated their interest in the slide using a Likert scale with *not at all interesting,* and *extremely interesting* as its anchors. All three of these ratings used 21-point scales (0 to 20), with higher numbers reflecting greater pleasantness, arousal, and interest, respectively.

Debriefing and dismissal. All participants were then debriefed and those in the no-alcohol condition were dismissed, whereas those who had received alcohol got BAL tests until the last two results were below 0.04% and declining before they were driven or escorted home.

Results

Individual Differences, Baselines, and Manipulation Checks

Individual differences and predrinking baseline physiological response. To assess the possibility of predrinking differences in the physiological responding of the two groups later assigned to alcohol and no-alcohol beverage treatments, we conducted one-way analyses of variance (ANOVAs), with beverage group as the independent variable and startle magnitude, startle latency, HR, SC, corrugator EMG, and zygomatic EMG as dependent variables. No significant predrinking differences in responding were found.

Beverage manipulation. All participants had a predrinking baseline BAL of zero. Participants in the no-alcohol beverage condition, of course, maintained a zero BAL through the two subsequent assessment points (postdrinking/pre-main session and after completion of main session). Those in the alcohol condition had a mean BAL of .075 g/100 ml (SD = .016) at the postdrinking assessment just prior to the primary data-collection period, and .069 g/100 ml (SD = .013) at the completion of the main session. As expected, a Beverage × Gender × Time repeated measures multivariate analysis of variance (MANOVA) revealed that participants in the alcohol condition achieved significantly higher BALs than those in the no-alcohol condition, $F(1, 44) = 665.82$, $p < .001$, but there was no main effect of gender of participant and no interaction between beverage condition and gender. In addition, the BALs at the two postdrinking assessments did not differ significantly from one another.

Slide ratings of valence, arousal, and interest. Separate Beverage × Gender ANOVAs for each of the three subjective ratings (i.e., valence, arousal, and interest) of the slides revealed no main effects and no interactions for either between-groups variable on any of these dependent measures. Overall, the slides, as rated on 21-point scales, were viewed as moderately pleasant (M = 14.80, SD = 1.76), arousing (M = 9.93, SD = 3.32), and interesting (M = 13.35, SD = 2.33).

Assessment of Primary Research Questions Regarding Startle

A doubly multivariate analysis of variance (i.e., a repeated measures MANOVA with, in this case, two dependent variables) was conducted with beverage (alcohol vs. no alcohol) as the between-groups variable, threat (shock threat block vs. safe block) and slide (present vs. absent) as within-groups variables, and startle magnitude and latency as variates. Significant multivariate results were followed up with univariate, repeated measures ANOVAs. Table 1 displays cell means and standard deviations for the startle measures in all condition combinations.

The effect of beverage condition on overall startle reactivity. Although the designated beverage groups did not differ in startle response magnitude or latency at predrinking baseline, the predicted significant multivariate main effect for beverage condition was found after drinking, $F(2, 44) = 4.14$, $p < .05$. Intoxicated participants evidenced generally smaller startle magnitudes, univariate $F(1, 46) = 8.51$, $p < .01$, and a trend toward longer startle latencies overall, univariate $F(1, 45) = 3.68$, $p = .06$.

The effect of threat versus safe condition on startle. A highly significant multivariate main effect for threat (i.e., the expected fear-potentiated startle effect) was observed, $F(2, 44) = 11.50$, $p < .001$, with a greater startle response magnitude, univariate $F(1, 46) = 12.78$, $p < .001$, and a faster response latency, univariate $F(1, 45) = 19.20$, $p < .001$, in the shock threat as compared with the safe condition. Also as predicted, fear-potentiated startle was present and robust in both intoxicated and sober participants, a result that is at variance with the hypothesis, derived from an SRD model, of a selective attenuating effect of alcohol on fear potentiation. There was neither a multivariate nor any univariate interaction between beverage and threat.[5]

Separate and joint effects of beverage, threat, and slides on startle. As predicted in our principal hypothesis and consistent with the A-A model, a significant multivariate Beverage × Threat × Slide interaction was obtained, $F(2, 44) = 4.33$, $p < .05$. Follow-up univariate analyses (ANOVAs) indicated that this three-way interaction was present for both startle magnitude, $F(1, 46) = 3.94$, $p < .05$, and latency, $F(1, 45) = 7.13$, $p < .01$ (see Table 1).

Simple effects analyses were used to explore and further clarify this three-way interaction. Separate analyses of the impact of the competing stimuli (i.e., the slide manipulation) on the multivariate threat effect were conducted within the alcohol and no-alcohol groups, using startle magnitude and latency as the variates. Fear-potentiated startle, manifested as heightened and speeded startle reactivity during threat versus safe periods, was

[5] A question could be raised about possible erosion of the effectiveness of the threat manipulation across trials if participants realized they might not ever receive a shock. Potentially more troublesome would be the risk that a confound could arise if there was differential evidence or rates of erosion across beverage conditions. To rule this out, we also conducted our doubly multivariate analysis of startle potentiation (magnitude and latency) with block included as a within-subjects variable. This MANOVA indicated that there was a significant overall (habituation-like) reduction in startle potentiation across blocks, $F(6, 40) = 2.64$, $p < .05$, although follow-up ANOVAs showed this was significant for magnitude only, $F(3, 44) = 5.05$, $p < .01$. In addition, the multivariate, simple effect of threat on startle was still significant in the final trial block, $F(2, 45) = 5.42$, $p < .01$, and univariate follow-up indicated significance for both magnitude, $F(1, 47) = 6.20$, $p < .05$, and latency, $F(1, 46) = 8.35$, $p < .01$. More important, however, was the absence of any Beverage × Block interaction. This result effectively ruled out confounding of the key startle potentiation variable by differential effectiveness of the threat manipulation across time.

Table 1
Means and Standard Deviations for Startle Response Magnitude and Latency by Beverage, Threat, and Slide Conditions

	Startle magnitude (μV)					Startle latency (ms)				
	Safe		Threat			Safe		Threat		
Condition	*M*	*SD*	*M*	*SD*	*F*(1, 23)	*M*	*SD*	*M*	*SD*	*F*(1, 23)
No alcohol										
No slide	19.9	17.8	26.8	26.2	5.63*	37.7	10.6	33.5	9.6	4.59*
Slide	17.7	16.9	26.7	26.0	7.51**	36.1	10.6	33.6	10.4	10.01**
Alcohol										
No slide	7.1	7.5	12.6	12.2	10.56**	43.2	13.8	37.9	12.4	20.58***
Slide	7.7	8.2	10.6	10.9	2.27	39.1	13.2	38.4	11.6	1.02

Note. *F*s are for the simple effect of threat versus safe for each combination of beverage and slide conditions.
* $p < .05$. ** $p < .01$. *** $p < .001$.

evident and significant in all no-alcohol conditions—regardless of the presence or absence of slides, $F(2, 22) = 3.91$, $p < .05$, and, $F(2, 22) = 6.32$, $p < .01$, respectively. In contrast, participants in the alcohol condition evidenced significant fear-potentiated startle only in the absence of slides, $F(2, 21) = 11.76$, $p < .001$. Significant fear-potentiated startle was not observed in intoxicated participants assessed during slide exposure, $F(2, 21) = 1.09$, $p = .35$.

Separate univariate analyses of startle magnitude and latency were also conducted, and results for fear potentiation, as indexed by both measures, were consistent with the multivariate pattern described above (see Table 1). For startle magnitude, significant potentiation was observed in the no-alcohol group both with slides, $F(1, 23) = 7.51$, $p < .01$, and without slides, $F(1, 23) = 5.63$, $p < .05$, as well as in the alcohol group when not engaged in slide viewing, $F(1, 23) = 10.56$, $p < .001$. There was, however, not a significant effect of threat on startle magnitude for intoxicated participants assessed during concurrent slide viewing, $F(1, 23) = 2.27$, $p = .15$.

For startle latency, significant potentiation (more rapid eyeblink response in threat vs. safe condition) was observed in the no-alcohol group, regardless of whether they were simultaneously viewing slides, $F(1, 23) = 10.01$, $p < .01$, or not, $F(1, 23) = 4.59$, $p < .05$. The alcohol/no-slide group showed similar startle potentiation, $F(1, 22) = 20.58$, $p < .001$, but as was the case for startle magnitude, there was no significant effect of threat on startle latency for intoxicated participants during slide viewing, $F(1, 22) = 1.02$, $p = .32$.[6]

The notion that response to threat can be increased by alcohol intoxication alone, if there is no concurrent stimulus or task to compete for cognitive resources, was not borne out by the data. A doubly multivariate, repeated measures MANOVA, with beverage and threat as the independent variables, was conducted on the magnitude and latency of startle responses elicited in intervals between slide presentations. The critical Beverage × Threat interaction was not significant.

Autonomic and Facial EMG Measures

Tonic levels during no-slide blocks. Repeated-measures ANOVAs were conducted to investigate how beverage and threat conditions affected tonic levels of each of the remaining dependent variables, including skin conductance, heart rate, and facial (corrugator and zygomatic) EMG. These measures provide indices of ongoing psychophysiological activity associated with two key manipulated variables (alcohol and threat). Tonic levels for each block were calculated by subtracting the average activity for the 5 s prior to the start of the block from the average level of activity during that block. Additionally, the first 5 s of each block were omitted from the computation of the average tonic level to exclude changes in activity associated with phasic reactions to the light cue. Analyses for tonic levels of each variable were based on no-slide blocks only to avoid contamination of the measures by phasic reactions to the slides (see below).

For tonic skin conductance level (SCL), a main effect of threat was observed, $F(1, 44) = 7.89$, $p < .01$, with higher tonic levels during threat blocks ($M = 0.11$ μS, $SD = 0.36$) than during safe blocks ($M = -0.09$, $SD = 0.43$). No main effect of beverage condition on SCL was observed, and there was no interaction between beverage and threat. Analysis of tonic levels of heart rate during the no-slide blocks yielded no main effects or interactions for beverage or threat. For tonic corrugator level, a main effect of threat was found, $F(1, 44) = 7.08$, $p < .05$, with higher tonic levels associated with shock-threat blocks ($M = 0.31$ μV, $SD = 1.11$) as compared with safe blocks ($M = -0.11$, $SD = 1.32$). No main effect for beverage condition on tonic corrugator level was observed, and there was no Beverage × Threat interaction. Similar analyses of zygomatic EMG did not yield any significant results.

[6] To examine the possibility that participant gender might exert a main or interactive effect on key startle potentiation measures, we included it as a variable in supplementary analyses. In the resulting doubly multivariate repeated measures MANOVA, we thus used gender and beverage as between-subjects variables and threat and slide as within-subjects variables. This analysis revealed an overall multivariate, main effect for gender, $F(2, 42) = 3.81$, $p < .05$, but this variable did not interact significantly with any other independent variable. Univariate follow-up analyses showed the main effect was significant for magnitude only, $F(1, 44) = 7.93$, $p < .01$. Women exhibited higher startle magnitude ($M = 919.5$, $SD = 1081.4$) than men ($M = 458.3$; $SD = 575.7$), but the two groups did not differ on startle latency.

Phasic reactions to the light cues. To examine the impact of light cues signaling threat versus safe periods and the possible effects of alcohol on them, we analyzed phasic reactions. Phasic skin conductance response (SCR) to the light cue was calculated by scoring the largest reaction occurring in the window between 0.9 and 4 s after light onset. Participants exhibited significantly larger SCRs to the light cues, $F(1, 46) = 19.56$, $p < .001$, signaling the onset of threat blocks ($M = 61.74$, $SD = 103.02$) than to those signaling the onset of safe blocks ($M = 17.33$, $SD = 47.96$), but there was no effect of beverage condition and no Beverage × Threat interaction for this dependent measure.

For HR and facial EMG, phasic responses to the light cue were calculated by subtracting the 1-s prestimulus baseline from the largest response (largest deceleration for HR and largest increase for corrugator or zygomatic) in a 6-s window after light onset. There was a main effect of threat on phasic HR change, $F(1, 46) = 7.78$, $p < .01$, with greater HR deceleration to the light cue signaling the onset of a threat block ($M = -5.48$ beats per min, $SD = 7.02$) as compared with the cue signaling the onset of a safe block ($M = -3.65$, $SD = 6.07$). No significant phasic effects of threat, alcohol, or their interaction were observed for either facial EMG measure.

Phasic reactions to the slides. Phasic responses to slide onset were also analyzed, using calculation procedures analogous to those described above for each measure. There was no significant effect of beverage, threat, or their interaction on SCRs to slide onset. A main effect of beverage, $F(1, 46) = 8.39$, $p < .01$, was the only significant result for HR changes in response to slide onset. HR deceleration to the slide stimulus was more pronounced among participants in the no-alcohol group ($M = -6.17$, $SD = 6.48$) than among those in the alcohol group ($M = -4.62$, $SD = 5.63$). Phasic facial EMG responses to slide onset were not significantly affected by threat, beverage, or their interaction.

Discussion

A major aim of the current study was to evaluate two prominent models of alcohol intoxication and negative affect. The use of startle methodology to assess emotional reactions offered unique advantages over previous research that has relied almost exclusively on subjective, self-report measures or on psychophysiologic indices (e.g., heart rate and skin conductance), which are better suited to assessment of general physiological arousal than affective valence specifically. By focusing on fear-potentiated startle, we were also able to capitalize on an extensive literature (e.g., Davis, 1986; Lang et al., 1990) explicitly linking this phenomenon to a defensive disposition indicative of aversive emotional state.

The main results of this experiment included replication of Stritzke et al. (1995), who found that alcohol produced overall attenuation of startle reflex, without influencing affective modulation of the response. Our data provide compelling evidence that fear-potentiated startle is robust in both sober and intoxicated individuals, a finding that is at odds with the general thesis of SRD that alcohol should consistently and selectively reduce distress responses to threatening stimuli. More important, as predicted by the A-A model, we found that only the *combination* of alcohol intoxication with benign stimuli capable of competing for cognitive resources attenuated distress as indexed by fear potentiation. There was no evidence that alcohol alone increased distress reactions to threat; that is, the so-called crying-in-one's-beer hypothesis was not supported.

Alcohol Effects on General Startle Reflex Reactivity and Possible Underlying Mechanisms

As we noted above, alcohol had the predicted significant main effect of reducing overall startle reactivity. Intoxicated participants exhibited significantly lower startle magnitudes than did nonintoxicated participants. There was also a trend toward longer startle latencies among participants who received alcohol compared with those who did not. These results replicated prior findings of Stritzke et al. (1995), who observed a similar attenuation of overall startle reactivity in intoxicated humans, without any concomitant reduction in affective modulation of the startle response. An established neuroanatomical model exists to interpret these effects.

Specifically, animal studies have provided evidence that the two components of the startle reflex (general reactivity and affective modulation) are mediated by different neural pathways. Using a fear conditioning procedure and brain-lesioning techniques with animals, Davis (1986) identified two independent neural pathways that interact to produce the fear-potentiated startle effect. The primary reflex pathway is the brainstem circuit along which impulses from the sensory receptors are transmitted to the peripheral effectors, by way of the pontine reticular formation (i.e., nucleus reticularis pontis caudalis). Lesions anywhere along this pathway completely eliminate the startle reflex. Potentiation of the reflex by fear conditioning, however, involves a secondary pathway that modulates this primary circuit. Monosynaptic projections from the amygdala (a limbic structure involved in aversive stimulus processing and defensive behavior) to the primary circuit have been shown to play a key role in the potentiation of the reflex by fear.

On the basis of these findings, it appears that alcohol's nonselective attenuation of startle in intoxicated human participants may be due entirely to the drug's inhibitory action on the primary reflex pathway, leaving intact the modulatory process by which the reflex is potentiated under aversive cuing conditions. In contrast, other drugs that are known for their specific anxiolytic effects (e.g., diazepam; see Patrick et al., 1996) seem capable of disrupting affective modulation of startle without dampening general startle reactivity. It remains for further research to determine where precisely along the primary acoustic startle pathway alcohol exerts its effects. Because studies of brainstem auditory-evoked potentials have (e.g., McRandle & Goldstein, 1973) indicated that moderate doses of alcohol do not directly inhibit acoustic sensory-processing systems, it seems probable that the effect of alcohol on general startle reactivity is attributable to a dampening of peripheral motoric responsivity or pontine reticular activation, or some combination of these processes.

General Effect of Alcohol on Negative Affect

Another key prediction tested here was one derived from the general SRD model. It holds that there should be a *selective*

effect of alcohol intoxication on emotion, such that it reduces response to affectively negative stimuli. This hypothesis was evaluated with particular reference to startle magnitude and latency. If SRD assumptions are broadly applicable, one would expect to see diminished fear-potentiated startle in intoxicated participants. However, strong and roughly equivalent fear-potentiated startle was observed in both alcohol and no-alcohol groups. There was no evidence of the predicted Beverage × Threat interaction for either the combined or separate magnitude and latency measures of fear-potentiated startle, suggesting that the impact of the shock threat was not generally dampened by alcohol intoxication. A similar pattern of results was observed in analyses of alcohol's impact on reactivity to the shock-threat cue using corrugator EMG activity, which has been demonstrated to be a reliable index of negative affect. These findings for specific indices of aversive emotional state raise questions about the broad applicability of the SRD model.

In addition to its influence on relatively direct measures of negative affect, the threat manipulation increased participants' overall physiological arousal, as indexed by SCL. Entertaining a liberal interpretation of the SRD model, it could be argued that although alcohol does not appear to attenuate aversive reactivity directly, it might serve to diminish a person's general emotional arousal in response to fearful stimuli. In other words, perhaps intoxicated participants should be expected to exhibit a smaller increase in arousal to shock threat than would sober participants. However, as with the specific indices of negative emotional state, no such arousal effect was observed for SCL.

It should be noted that the observed lack of support for predictions derived from the SRD model is not easily attributable to mismatched methodological features of the present experiment. Indeed, it appears to have fulfilled many key requirements essential to a strong test of the model. First, the manipulated stressor produced a reliable and robust stress response. Physiological measures indicated that the shock threat was physiologically arousing (tonic skin conductance effect) and affectively negative (startle magnitude, startle latency, and corrugator EMG effects) for participants. In addition, participants evidenced relatively greater orienting response (HR deceleration and phasic skin response) to cues for threat versus safe blocks, indicating that they perceived the threat block cue as a potent stimulus because of its association with the possibility of receiving an electric shock. Furthermore, the physiologic measures used here permitted assessment of how alcohol affected participants' emotional reactions to stressors in terms of both specific negative affect and general arousal. Finally, because it has previously been demonstrated that the array of measures used here is sensitive to the effects of the threat of electric shock, it can be argued that if the alcohol dose used here does indeed have the general effect of reducing stress reactions, these measures should have detected it. They did not.

Interactive Effects of Alcohol and Competing Stimuli on Negative Affect in the Face of Threat

Unlike the SRD model, the A-A model posits that alcohol intoxication alone does not necessarily reduce a person's stress response to negative stimuli. Instead, this theory proposes that alcohol intoxication produces two important cognitive effects that may indirectly influence response to stress. Specifically, alcohol intoxication is thought to reduce attentional capacity and to increase focus on the most "salient" stimuli in the environment. Given these putative effects, it can be hypothesized that alcohol intoxication will succeed in reducing reactivity to stress only when combined with competing attentional demands that overload cognitive capacity already limited by intoxication and thereby compromise processing of the threat. A corollary prediction is that in the absence of competing stimuli, the threat-induced distress reactions of intoxicated individuals might even be intensified, although the mechanisms underlying such an effect have not been clearly articulated.

In any event, startle probe methodology is especially well suited to testing predictions derived from the A-A model because this approach can be used to assess the valence of participants' affective response to an ongoing stressor during concurrent exposure to competing stimuli (see Stritzke et al., 1996, for a detailed discussion of this issue). Multivariate analysis of the magnitude and latency of startle response revealed a significant three-way interaction between the beverage, threat, and slide conditions of the present experiment. Univariate analyses indicated that this interaction was consistent for both startle magnitude and latency, and simple-effects analyses helped to elucidate its exact form.

Significant fear potentiation was exhibited by sober participants, both when viewing slides and in the absence of any competing stimuli. A parallel effect was also evident in intoxicated participants during no-slide periods. However, significant fear potentiation was not observed (for either startle magnitude or latency) in intoxicated participants when they were simultaneously engaged in slide viewing. This suggests a unique effect of the combination of alcohol intoxication and competing stimuli on negative emotional reactivity to threat. Benign, cognitively demanding slide stimuli appeared to reduce intoxicated participants' negative reaction to the stressful shock cue, whereas such competing stimuli did not reduce the stress response of sober participants. This result is consistent with the main prediction derived from the A-A model. In contrast, there was no indication that alcohol intoxication alone either decreased or increased stress responses manifested as fear-potentiated startle. Thus, the modification of affective response by alcohol appeared to occur only when participants were *both* intoxicated *and* engaged in slide viewing that evidently diverted cognitive resources away from the processing of threat, thereby producing a significant reduction in distress.

Models and Mechanisms to Explain Alcohol–Stress Relationships: Future Directions

There is considerable intuitive appeal to Steele and Josephs' (1990) theory that the effects of alcohol on stress reactivity are mediated by its impact on higher level cognitive processing. In addition, there is plausibility to the specific proposition that alcohol intoxication reduces overall attentional capacity and might also facilitate a focus on the most demanding of the stimuli vying for attention. Under stressful conditions involving the simultaneous presence of competing stimuli, some of an inebriated person's compromised attentional capacity could be diverted to or at least confused by the competing stimuli, re-

sulting in diminished processing of and responsivity to the stressful cues. However, one potential difficulty with this formulation lies in the concept of "salience," which appears somewhat circular as an explanatory construct—the most salient stimulus is defined as the one commanding the most attention, but this begs the question of why. There is also uncertainty about whether and, if so, why alcohol intoxication should lead to an increased focus on any particular stimulus rather than simply diminished processing of any and all stimuli.

Sayette's (1993) appraisal–disruption model addresses these issues to some extent by proposing that alcohol interferes with the processing of aversive cues via a general disruption of higher associative functioning. Specifically, Sayette theorized that "alcohol acts pharmacologically to disrupt appraisal of stressful information by constraining the spread of activation of associated information previously established in long term memory (LTM)" (p. 463). In other words, when initially appraising a stressor, alcohol interferes with the ability to activate the associated representations in LTM that supply the emotional content to the stimuli (cf. Bower, 1981), resulting in a diminished stress response.

From this perspective, the attention-allocation and appraisal–disruption models may be viewed as complementary rather than as competing perspectives, a point made by Sayette (1993). If Steele and Josephs' term *salience* is conceptualized as the degree to which a stimulus primes associated memory representations, then both models appear to posit that alcohol's impact on the stress response is mediated by activation of competing associations. However, a key difference in the theories is in the importance attributed to the time course of events. Sayette's model proposes that a reduction in the stress response is likely to occur only if alcohol is consumed prior to initial appraisal of the stressor. Once the stressor is appraised and associated memory structures are activated, alcohol should have minimal anxiolytic effects. In contrast, the A-A model postulates that the interposition of a competing stimulus can attenuate stress even after an aversive stimulus has been recognized and responded to, provided that the competing stimulus is sufficiently demanding.

The latter perspective appears more consonant with the findings of the present study. Our participants were apprised of the stressor (electric shock) in connection with informed consent procedures occurring prior to alcohol administration, and the specific shock-threat cue (red light) was introduced before the first slide presentation. Despite this advance warning of impending threat, intoxicated participants showed a reduction in startle reflex potentiation when engaging slides were introduced. Nonetheless, a synthesis of the two models might provide a more complete and conceptually satisfying picture of the interactions of alcohol, cognitive processing, and stress. We propose that any conditions or cues potent enough to disrupt processing of threat stimuli could result in a reduction of the stress response. These might take the form of initial impairment of threat encoding due to intoxication or activation of competing memory representations by other demanding stimuli (cf. Stritzke et al., 1996).

Even adoption of this expanded perspective, however, leaves a number of key questions unanswered. Certainly, the matter of dose-response effects on alcohol–emotion relationships needs attention, as does the possibility of divergent results on the ascending versus descending limbs of the blood-alcohol curve. There is also considerable uncertainty about what characteristics of a stimulus event or array determine its predominance or ability to diminish the impact of other stimuli or associations in individuals under the influence of alcohol. The present research demonstrated that the presence of a highly pleasant and arousing stimulus can, under conditions of moderate intoxication, attenuate the stress response occasioned by an ongoing cue that signals the possibility of a noxious event. Yet, one wonders exactly what the key features and mechanisms operating in this situation might be. For example, must the valence of the stimulus competing with the threat be incompatible with it, or could the presence of other negative stimuli reduce distress simply by compromising the processing of it? The internal versus external nature of competing stimuli might also influence the anxiolytic effect of drinking. Through future parametric research, it should be possible to address issues such as these and elucidate the dimensions of intoxication and properties of the competing stimuli that underlie the modification of emotional response by alcohol. This, in turn, should advance understanding of the mechanisms that mediate such effects. Because there can be little doubt that affect and alcohol use, abuse, and dependence are intimately linked, we look forward to further developments in these areas.

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