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Research Report

ALCOHOL AFFECTS EMOTION THROUGH COGNITION

John J. Curtin,[1] Christopher J. Patrick,[2] Alan R. Lang,[3] John T. Cacioppo,[4] and Niels Birbaumer[5,6]

[1]Department of Psychology, University of Wisconsin; [2]Department of Psychology, University of Minnesota; [3]Department of Psychology, Florida State University; [4]Department of Psychology, University of Chicago; [5]Institute of Medical Psychology and Behavioral Neurobiology, University of Tübingen, Tübingen, Germany; and [6]Institute of General Psychology, University of Padua, Padua, Italy

Abstract—*Determining how cognition and emotion interact is pivotal to an understanding of human behavior and its disorders. Available data suggest that changes in emotional reactivity and behavior associated with drinking are intertwined with alcohol's effects on cognitive processing. In the study reported here, we demonstrated that alcohol dampens anticipatory fear and response inhibition in human participants not by directly suppressing subcortical emotion centers, as posited by traditional tension-reduction theories, but instead by impairing cognitive-processing capacity. During intoxication, reductions in fear response (assessed via startle potentiation) occurred only under dual-stimulus conditions, and coincided with reduced attentional processing of threat cues as evidenced by brain response (assessed via P3 event-related potentials). The results are consistent with higher cortical mediation of alcohol's effects on fear, and illustrate more broadly how disruption of a cognitive process can lead to alterations in emotional reactivity and adaptive behavior.*

Emotion is central to an understanding of human behavior because it is the primal force that drives action and its inhibition (Gray, 1987; Izard, 1993; P.J. Lang, 1995). Excesses and deficits in basic emotional reactivity have been posited as explanations for many forms of behavior disorder. However, the subcortical systems that directly activate positive (appetitive) and negative (defensive) responses do not operate in isolation. Rather, they interact extensively with higher brain regions (P.J. Lang, 1995; LeDoux, 1995), raising the possibility that disturbances in cognitive functions such as attention, memory, and appraisal may contribute to impairments in affective processing (A.R. Lang, Patrick, & Stritzke, 1999). The disruption of emotional behavior that occurs during acute alcohol intoxication is, we argue, an illustrative case in point.

Long regarded to be an anxiolytic drug, alcohol can induce behavioral changes—including disinhibition of punished responses, risk taking, and aggressiveness (Leonard & Blane, 1999)—that have been ascribed to its direct impact on the fear response system. However, theorists have begun to challenge this notion (A.R. Lang et al., 1999; Sayette, 1993; Steele & Josephs, 1990), and preliminary data (Curtin, Lang, Patrick, & Stritzke, 1998; Stritzke, Patrick, & Lang, 1995) suggest that alcohol may instead alter emotional response by impairing higher cognitive functions needed to process affective cues in complex, naturalistic contexts. Here, we present the first direct evidence that alcohol attenuates fear and impairs response inhibition via its effects on cognitive processing. This work illustrates a model of cognition-emotion interactions and a methodology that can advance understanding of clinical disorders involving defective inhibition (Patrick & Lang, 1999).

The well-established phenomenon of fear-potentiated startle (FPS; Davis, 1989) provides a noninvasive methodology for examining the effects of drugs on subcortical emotion systems in humans. Numerous studies have shown that the startle response to an abrupt, intense stimulus (e.g., loud noise) increases above baseline when elicited in the presence of a cue that has been paired with shock. Through association with shock, the cue acquires fear-eliciting properties, and in its presence the startle response is potentiated because fear entails a priming of protective reflexes (P.J. Lang, Bradley, & Cuthbert, 1990). Animal research indicates that this effect is mediated by the amygdala, a subcortical structure implicated in fear behavior (Hitchcock & Davis, 1986). Its central nucleus projects to the nucleus reticularis pontis caudalis (nRPC), a component of the primary brain-stem startle circuit, and lesions of this amygdala-nRPC pathway abolish FPS without eliminating the primary reflex. In animals, FPS is blocked by anxiolytic drugs (Davis, 1979; Davis, Redmond, & Baraban, 1979), and enhanced by anxiogenics (Davis et al., 1979).

In humans, FPS (measured as an increase in the magnitude of blink response to an abrupt noise) occurs during exposure to unpleasant pictures as well as shock cues, and is blocked by administration of diazepam (Patrick, Berthot, & Moore, 1996)—but not by moderate doses of ethanol (Stritzke et al., 1995). The latter finding challenges the idea that alcohol directly suppresses fear. A competing perspective is that alcohol impairs cognitive capacity, so that emotional features of a stimulus context escape detection or are weakly processed (A.R. Lang et al., 1999; Sayette, 1993; Steele & Josephs, 1990). Consequently, attention is restricted to, and behavior governed by, immediate, explicit contingencies. This "alcohol myopia" model is supported by several lines of evidence. One is that alcohol produces marked impairments on divided-attention tasks involving simultaneous processing of competing stimuli. Another is that intoxication attenuates reports of negative affect primarily when attention is directed away from an impending stressor (Curtin et al., 1998; Steele & Josephs, 1988).

The present experiment assessed subcortical-emotional, cortical-attentional, and overt behavioral responses to a threat cue under two different conditions: (a) divided attention, when the threat cue was presented as an incidental stimulus in the context of a primary visual-motor task, and (b) threat focus, when the threat cue was presented in isolation. Fear was assessed by FPS. Behavioral response inhibition was measured via task reaction time (RT), with slower RT expected on threat-cue trials (Kida, 1983). Attentional processing of threat cues was assessed using the P3 component of the cortical event-related potential (ERP), a positive, parietal-focused component that covaries in magnitude with the degree of attention devoted to stimuli in a processing stream (Halgren, Squires, & Wilson, 1982; Knight, 1996; Kramer

Address correspondence to John J. Curtin, Department of Psychology, University of Wisconsin, 1202 West Johnson St., Madison, WI 53706; e-mail: jjcurtin@facstaff.wisc.edu.

& Spinks, 1991). In dual-task paradigms, reciprocity is observed in the magnitude of P3 responses to primary- and secondary-task stimuli: As one task is prioritized or made more difficult, P3 responses to concurrent competing task stimuli become smaller, reflecting reduced attentional processing. The P3 provides specificity in that its magnitude is influenced by attention allocation but not response parameters in perceptual-motor tasks (Isreal, Chesney, Wickens, & Donchin, 1980; Kutas, McCarthy, & Donchin, 1977).

Predictions were that (a) alcohol would attenuate anticipatory fear (i.e., FPS) in the divided-attention condition, but not in the threat-cue-alone condition; (b) reduced fear in the divided-attention condition would be accompanied by a concomitant reduction in attentional processing of the threat cue (i.e., reduced P3); and (c) fear attenuation would be manifested behaviorally as diminished slowing of RT on divided-attention trials involving the threat cue. In sum, we predicted that reduced physiological (FPS) and behavioral (response inhibition) evidence of fear would result from diminished cognitive processing of threat stimuli under complex (divided-attention) conditions, rather than from a direct effect of alcohol on the subcortical fear system.

METHOD

Participants were 48 right-handed undergraduates (24 female) aged 21 or older who reported recent experience with moderate doses of alcohol, and had no alcohol problem or other contraindicating medical condition. Equal numbers of men and women were assigned randomly to alcohol and no-alcohol groups.[1] Participants in the alcohol group received a beverage containing fruit juice mixed with pure ethyl alcohol in a 7:1 ratio. Total alcohol dose was calculated to produce a peak blood alcohol level of 0.080 g/100 ml based on the participant's height, weight, age, and gender, and the length of the drinking period (see Curtin et al., 1998, for details on this algorithm). The alcohol dose was divided into an initial loading dose consumed prior to the start of the experimental task and two booster doses consumed separately during the task procedure to maintain intoxicated participants on the ascending limb of the blood alcohol curve throughout the experimental session. Participants in the no-alcohol group consumed a nonalcoholic drink (fruit juice only) matched in volume to what the alcohol group drank.

The task procedure began 15 min after the initial loading dose. It comprised 24 blocks of 20 trials each. Blocks were of two types: threat focus and divided attention. Each trial consisted of two stimuli (S1 and S2) separated by 2,200 ms (Fig. 1). The S1 varied depending on block type; the S2 was always a blue square. An interval of 2 to 3 s separated the offset of the S2 from the onset of the next trial.

In threat-focus blocks, the S1 was a word from one of two categories, animals or body parts, presented in red script. Participants were advised that a shock could occur 2 s after S1 words from one of the categories (designated *cue+*; this category was counterbalanced across participants). The use of word categories ensured that detection of the threat cue entailed higher-order, semantic processing. Shocks were administered on 50% of cue+ trials. No shocks occurred following words from the other (*cue−*) category. The shock intensities delivered were based on individual tolerance thresholds determined at baseline; four intensities were delivered—threshold and 0.1, 0.2, and 0.3 mA below the threshold. Threshold was adjusted upward by 0.025 mA after each shock to control for habituation. Shocks were delivered for 200 ms through electrodes on adjacent fingers.

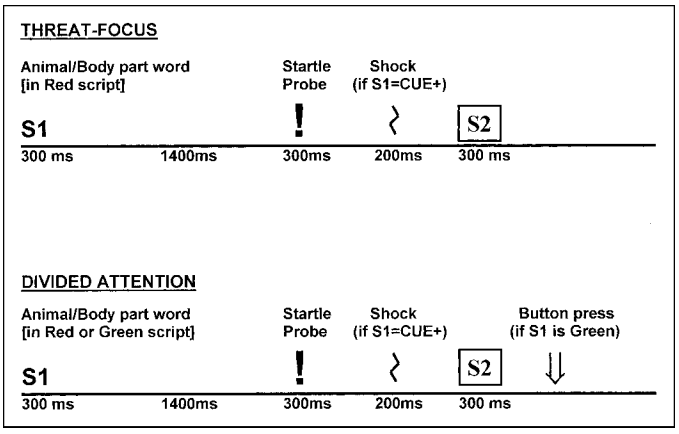

Fig. 1. Trial structure schematic. Cue+ = word category paired with shock; S1 = first stimulus; S2 = second stimulus. Blink response was recorded in a 300-ms window (50-ms baseline, 50-ms startle-eliciting noise probe, 200-ms blink response period) beginning 1,700 ms after S1 onset. See the text for additional details.

In divided-attention blocks, the S1 was an animal or body-part word, colored either red or green. As in threat-focus blocks, S1 words from the cue+ category predicted shock. However, participants were instructed to attend primarily to the color of the S1 word and to press an RT button immediately at onset of the S2 if the S1 was colored green (*go* trial). If the S1 was colored red (*stop* trial), no response was required. Thus, processing of threat cues in divided-attention blocks demanded the division of attentional resources between threat information (i.e., word category) and task information (i.e., word color), with processing of task information prioritized for participants.

Startle-eliciting noise probes (50-ms, 105-dB noise burst with rise time less than 10 μs) were presented 1,750 ms after S1 onset on 48 (24 cue+ and 24 cue−) of the 480 threat-focus and divided-attention trials. Blink response was recorded within a 300-ms window (50-ms baseline, 50-ms noise probe, 200-ms blink-response period) from electrodes positioned under the right eye. The raw electromyogram signal was sampled at 1000 Hz and integrated using an 80-ms time constant. Blinks were scored as the change in response from baseline to peak in microvolts. Fear reactivity to threat cues was indexed by FPS, defined as the difference in blink-response magnitude to probes following cue+ compared with cue− words.

Attentional processing of threat cues was indexed by the P3 component of the cortical ERP. ERP activity was recorded from tin electrodes positioned at Fz, Cz, and Pz according to the 10-20 system (Jasper, 1958) and referenced to linked mastoids, with impedances below 5 Kohm. Raw signal was sampled at 1000 Hz. Off-line processing

1. The decision to use a no-alcohol, as opposed to a placebo, comparison group was a reasoned one. Prior research indicated that alterations in fear response, when observed, have a purely pharmacological basis (Greeley & Oei, 1999). Moreover, the ability to rule out global expectancy effects was strengthened by the prediction of differential alcohol effects across threat-focused versus divided-attention blocks. Although it is likely that participants held the expectancy that alcohol would reduce fear response in general, it is improbable that they expected differences across these two block types. Finally, the extreme-groups approach permitted a maximally powerful test of hypothesized effects.

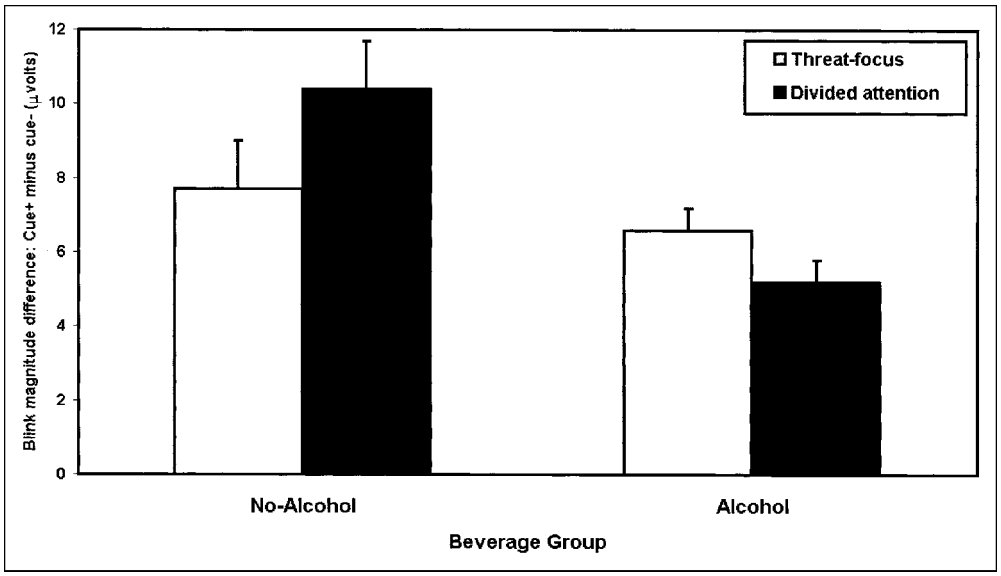

Fig. 2. Fear-potentiated startle by beverage and block type. Error bars represent within-subjects standard error. Cue+ = word category paired with shock; cue− = word category not paired with shock.

included 9-Hz low-pass filtering, baseline and ocular correction, and averaging. P3 in microvolts was scored within a 200-ms window (505–705 ms) following S1 onset, which represented the region of peak activity in the grand average waveform across all participants and trials. Recognition of the threat significance of words from the cue+ category was expected to enhance the P3 response. Accordingly, P3 enhancement was defined as the difference in P3 magnitude for cue+ compared with cue− words. Greater P3 enhancement was interpreted as evidence of greater attentional processing of threat cues. Within divided-attention blocks, task performance on cue+ and cue− go trials was indexed by RT to the S2.

RESULTS

Each physiological measure was analyzed within a Beverage Group (alcohol vs. no alcohol) × Block Type (threat focus vs. divided attention) repeated measures analysis of variance.[2] The two-way interaction was significant for both FPS and P3 enhancement, Fs(1, 46) = 7.91 and 4.72, ps = .007 and .035. For FPS, simple effects tests revealed no group difference during threat-focus blocks. However, during divided-attention blocks, FPS was attenuated in the alcohol group, $t(46) = 2.36$, $p = .023$ (Fig. 2). Thus, the first prediction was supported.

As was the case for FPS, simple effects tests for P3 enhancement revealed no difference between the beverage groups during threat-focus blocks, but during divided-attention blocks, cortical differentiation (P3 difference between cue+ trials and cue− trials) was reduced for intoxicated participants in comparison to control participants, $t(46) = 3.55$, $p = .001$ (Fig. 3). Thus, alcohol selectively impaired cognitive processing of the threat cue under conditions of divided attention (the second prediction).

A final analysis examined RT to the S2 on go trials within the divided-attention condition as a function of whether the S1 was shock-relevant (cue+) or not (cue−). Overall, RT was markedly longer on cue+ than cue− trials (Ms = 235 ms and 182 ms, respectively; difference = 53 ms), indicating that shock anticipation inhibited subsequent behavioral response, $F(1, 46) = 97.28$, $p < .001$. As predicted, however, this effect was significantly smaller in the alcohol group (Ms for cue+ and cue− = 227 ms and 186 ms; difference = 41 ms) than the no-alcohol group (Ms = 243 ms and 178 ms, respectively; difference = 65 ms), $F(1, 46) = 4.78$, $p = .034$. That is, intoxicated participants showed less response inhibition than control participants on divided-attention go trials involving the possibility of shock (the third prediction).

DISCUSSION

This study provides the first direct demonstration of the role of reduced attentional capacity in accounting for decrements in fear reactivity and behavioral inhibition during intoxication. Impairments in attention to the threat cue and subsequent fear response were observed in the alcohol group during divided-attention trials, but not during threat-focus trials. This dissociation, which is consistent with cognitive-attentional theories of alcohol's effects (A.R. Lang et al., 1999; Sayette, 1993; Steele & Josephs, 1990), can be understood from the perspective of a multilevel theory of emotional processing that emphasizes the interplay of cortical and subcortical processing systems (P.J. Lang, 1995; LeDoux, 1995).

The subcortical amygdala is recognized as central to fear processing because it projects directly to defensive action systems (including the startle reflex circuit; Davis, 1989; Fanselow, 1994). However, the amygdala receives afferent input from various brain regions, including primary sensory structures (i.e., thalamus, sensory cortex) and also higher associative systems such as the hippocampus. Thus, the detec-

2. Initial analysis of gender effects revealed no significant overall differences between men and women on the primary dependent measures (i.e., FPS, P3, or RT). Therefore, gender was not included as a factor in subsequent analyses.

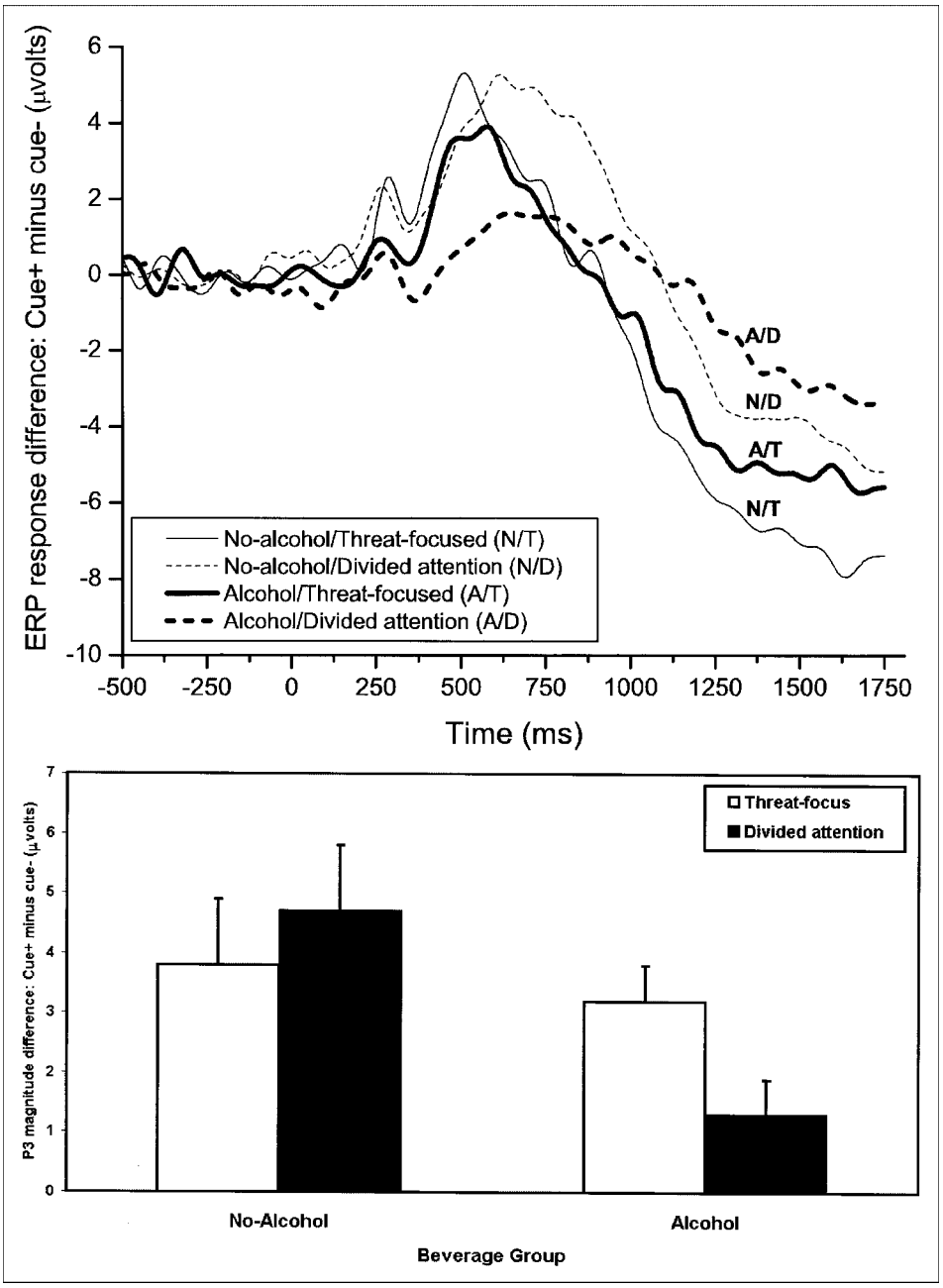

Fig. 3. Event-related potential (ERP) response on trials using the word category paired with shock (cue+) versus trials using the word category not paired with shock (cue−). The top panel shows the difference waveform (cue+ minus cue−) at the Pz scalp site by beverage and block type. The bottom panel shows the mean P3 magnitude difference (cue+ minus cue−) in the scoring window (505–705 ms after onset of the first stimulus) by beverage and block type. Greater P3 difference indicates increased attention to and processing of shock-related information. Error bars represent within-subjects standard error.

tion of affective stimuli of varying complexity entails different levels of cognitive processing, and differing brain systems. As a striking illustration of this, animals with hippocampal lesions do not develop contextual fear, which requires the associative capacities of the hippocampus, although they readily acquire fear to a simple sensory cue (LeDoux, 1995).

The present results indicate that alcohol does not affect fear at a primary subcortical (amygdala) level, but instead influences emotional response via effects on higher cortical systems that participate in the detection and recognition of affective cues embedded within a context. The following lines of evidence suggest that the hippocampal-temporal lobe may be one such system: First, in animals, ethanol blocks the

acquisition of contextual, but not explicit, fear (Melia, Corodiman, Ryabinin, Wilson, & LeDoux, 1994). Second, alcohol impairs spatial learning in animals, a hippocampus-dependent capacity (Matthews, Best, White, Vandergriff, & Simson, 1996). Third, the P3 component of the ERP in humans, which was found to be impaired by alcohol under divided-attention conditions, is generated at least in part by the hippocampus (Kramer & Spinks, 1991). Functional brain-imaging techniques could be used to investigate the role of diminished hippocampal functioning more directly. However, the larger point is that understanding of cognitive-emotional impairments underlying behavior disorders (whether chronic syndromes or acute drug states) is most likely to be advanced by the use of emotional-processing paradigms that tap specified cognitive functions with known neural substrates.

Emotions are vital to an understanding of behavior because they are linked directly to action. The RT data of the present study highlight this point. As a function of their reduced fear response to the threat cue on divided-attention go trials, intoxicated participants showed diminished slowing of RT (i.e., reduced response inhibition) to the S2 when a threat word appeared as the S1. This finding coincides with substantial evidence indicating that the capacity of threat or punishment cues to inhibit behavior is reduced under conditions of intoxication. In the present context, this behavioral effect was adaptive—that is, intoxicated subjects outperformed control subjects on the RT task under threat conditions. However, in real life, impaired inhibition more typically leads to maladaptive outcomes, including sexual risk taking, violence, and vehicular fatalities (Leonard & Blane, 1999).

The present study provides a clear demonstration of how alterations in cognitive-processing capacity can influence responsiveness to an emotional cue, with consequent effects on behavior. It also provides compelling evidence that alcohol dampens emotional responsiveness and reduces inhibitions via its effects on higher cognitive systems. This work highlights potential contributions from a multilevel perspective on emotional processing and the need to develop additional system-specific task paradigms for mapping the cognitive-emotional bases of behavior disorders. Moreover, the general approach outlined here also appears to be applicable to the study of drug effects on other emotion and arousal states, including euphoria and stimulation.

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REFERENCES

Curtin, J.J., Lang, A.R., Patrick, C.J., & Stritzke, W.G.K. (1998). Alcohol and fear-potentiated startle: The role of competing cognitive demands in the stress-reducing effects of intoxication. *Journal of Abnormal Psychology*, *107*, 547–565.

Davis, M. (1979). Diazepam and flurazepam: Effects on conditioned fear as measured with the potentiated startle paradigm. *Psychopharmacology*, *62*, 1–7.

Davis, M. (1989). Neural systems involved in fear-potentiated startle. In M. Davis, B.L. Jacobs, & R.I. Schoenfeld (Eds.), *Annals of the New York Academy of Sciences: Vol. 563. Modulation of defined neural vertebrate circuits* (pp. 165–183). New York: New York Academy of Sciences.

Davis, M., Redmond, D.E., & Baraban, J.M. (1979). Noradrenergic agonists and antagonists: Effects on conditioned fear as measured by the potentiated startle paradigm. *Psychopharmacology*, *65*, 111–118.

Fanselow, M.S. (1994). Neural organization of the defensive behavior system responsible for fear. *Psychonomic Bulletin & Review*, *1*, 429–438.

Gray, J.A. (1987). *The psychology of fear and stress* (2nd ed.). Cambridge, England: Cambridge University Press.

Greeley, J., & Oei, T. (1999). Alcohol and tension reduction. In K.E. Leonard & H.T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 14–53). New York: Guilford Press.

Halgren, E., Squires, N.K., & Wilson, C.L. (1982). Brain generators of evoked potentials: The late (endogenous) components. *Bulletin of the Los Angeles Neurological Society*, *47*, 108–123.

Hitchcock, J., & Davis, M. (1986). Lesions of the amygdala, but not of the cerebellum or red nucleus, block conditioned fear as measured with the potentiated startle paradigm. *Behavioral Neuroscience*, *100*, 11–22.

Isreal, J.B., Chesney, G.L., Wickens, C.D., & Donchin, E. (1980). P300 and tracking difficulty: Evidence for multiple resources in dual-task performance. *Psychophysiology*, *17*, 259–273.

Izard, C.E. (1993). Four systems for emotion activation: Cognitive and noncognitive processes. *Psychological Review*, *100*, 68–90.

Jasper, H.H. (1958). The ten-twenty electrode system of the International Federation. *Electroencephalography and Clinical Neurophysiology*, *10*, 371–375.

Kida, M. (1983). Facilitative and suppressive effects of punishment on the reaction time in humans under various conditions. *Psychological Research*, *45*, 187–214.

Knight, R.T. (1996). Contribution of human hippocampal region to novelty detection. *Nature*, *383*, 256–259.

Kramer, A., & Spinks, J. (1991). Capacity views of human information processing. In J.R. Jennings & M.G.H. Coles (Eds.), *Handbook of cognitive psychophysiology: Central and autonomic nervous system approaches* (pp. 179–249). Chichester, England: John Wiley & Sons.

Kutas, M., McCarthy, G., & Donchin, E. (1977). Augmenting mental chronometry: The P300 as a measure of stimulus evaluation time. *Science*, *197*, 792–795.

Lang, A.R., Patrick, C.J., & Stritzke, W.G.K. (1999). Alcohol and emotional response: A multidimensional-multilevel analysis. In K.E. Leonard & H.T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 328–371). New York: Guilford Press.

Lang, P.J. (1995). The emotion probe: Studies of motivation and attention. *American Psychologist*, *50*, 372–385.

Lang, P.J., Bradley, M.M., & Cuthbert, B.N. (1990). Emotion, attention, and the startle reflex. *Psychological Review*, *97*, 377–398.

LeDoux, J.E. (1995). Emotion: Clues from the brain. *Annual Review of Psychology*, *46*, 209–235.

Leonard, K.E., & Blane, H.T. (Eds.). (1999). *Psychological theories of drinking and alcoholism* (2nd ed.). New York: Guilford Press.

Matthews, D.B., Best, P.J., White, A.M., Vandergriff, J.L., & Simson, P.E. (1996). Ethanol impairs spatial cognitive processing: New behavioral and electrophysiological findings. *Current Directions in Psychological Science*, *5*, 111–115.

Melia, K., Corodiman, K., Ryabinin, A., Wilson, M., & LeDoux, J. (1994). Ethanol (ETHO) pre-treatment selectively impairs classical conditioning of contextual cues: Possible involvement of the hippocampus. *Society for Neuroscience Abstracts*, *24*, 1007.

Patrick, C.J., Berthot, B., & Moore, J.D. (1996). Diazepam blocks fear-potentiated startle in humans. *Journal of Abnormal Psychology*, *105*, 89–96.

Patrick, C.J., & Lang, A.R. (1999). Psychopathic traits and intoxicated states: Affective concomitants and conceptual links. In M.E. Dawson, A.M. Schell, & A.H. Boehmelt (Eds.), *Startle modification: Implications for clinical science, cognitive science, and neuroscience* (pp. 209–230). New York: Cambridge University Press.

Sayette, M.A. (1993). An appraisal-disruption model of alcohol's effects on stress responses in social drinkers. *Psychological Bulletin*, *114*, 459–476.

Steele, C., & Josephs, R. (1988). Drinking your troubles away II: An attention allocation model of alcohol's effect on psychological stress. *Journal of Abnormal Psychology*, *97*, 196–205.

Steele, C., & Josephs, R. (1990). Alcohol myopia: Its prized and dangerous effects. *American Psychologist*, *45*, 921–933.

Stritzke, W.G.K., Patrick, C.J., & Lang, A.R. (1995). Alcohol and human emotion: A multidimensional analysis incorporating startle-probe methodology. *Journal of Abnormal Psychology*, *104*, 114–122.

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