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ALCOHOL AND EMOTION: INSIGHTS AND DIRECTIVES FROM AFFECTIVE SCIENCE

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Among the properties of alcohol that define it as a psychoactive substance are its ability to alter psychological processes that include emotion and cognition. More specifically, mood lability and affect-linked responses such as sexual and aggressive behavior figure prominently in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision; American Psychiatric Association, 2000) description of alcohol intoxication; "clinically significant . . . distress" (pp. 181–182) is a primary dimension on which individuals are expected to be high when they meet diagnostic criteria for alcohol abuse or dependence. Not surprisingly then, virtually all major theories of drinking behavior and its problems include an important role for emotional factors and affect regulation. In this connection, Goldman, Brown, and Christiansen (1987) asserted, "If any characteristic has been seen as a central, defining aspect of alcohol use, it is the presumed capacity of alcohol to alter anxiety, depression, and other moods" (p. 200). Given the pivotal position assigned to emotion in most conceptualizations of drinking behavior and alcohol use disorders, it is useful to examine how modifications of emotional response associated with alcohol intoxication

operate at the different levels of analysis featured in major theories of drinking-related phenomena.

Influential biological models of drinking incorporate stimulant properties of alcohol that are evident while blood-alcohol level (BAL) is low and ascending shortly after consumption begins. For example, early in an episode of intoxication, humans exhibit increased cortical activity that often correlates with self-reported experience of positive emotions such as elation and euphoria as well as increased arousal described as energy and vigor (Lukas, Mendelson, Benedikt, & Jones, 1986; Martin, Earleywine, Musty, Perrine, & Swift, 1993). Such stimulation is also apparent in the initially increased spontaneous motor activity of lower animals exposed to alcohol (Lewis & June, 1990). Furthermore, alcohol-induced psychomotor activation is associated with stimulation of brain reward systems, an effect that is important because alcohol and many other psychoactive drugs are thought to exert their addictive power through actions on dopaminergic fibers projecting into areas of the brain that mediate reinforcement, emotional state, and approach behavior (e.g., Robinson & Berridge, 2003; Wise & Bozarth, 1987). In another vein, several theorists (e.g., Baker et al., 2004; Koob & Le Moal, 2001; Solomon, 1977) have noted that certain neuroendocrine and central nervous systems are organized to oppose or suppress the unconditioned, hedonic effects of alcohol and other addictive drugs. Accordingly, with repeated alcohol or other drug use, neuroplastic adaptations in affect systems designed to support homeostasis may lead to persistent dysphoria and/or other problems with negative affect in the absence of the drug. As a result, drinking or other drug use may eventually come to be motivated more by a desire to relieve dysphoria than by a desire to attain euphoria. In any case, it is clear that modulation of emotional state is a major force driving the indulgence.

At a cognitive level of analysis, Goldman et al. (1987) concluded from their early work on alcohol expectancies that beliefs about the drug's ability to alter mood and affective reaction are both prevalent and pivotal, with arousal (activation-sedation) and affective-social valence (positive-negative) as the primary dimensions organizing alcohol expectancies (for a review, see Goldman, Del Boca, & Darkes, 1999). Furthermore, Cooper, Frone, Russell, and Mudar (1995) have demonstrated that such expectancies and the memory networks presumed to underlie them map onto important motives for drinking, with drinkers reporting that enhancement of positive emotions and/or coping with negative ones are central motives for alcohol use.

The key role of emotion in drinking and drinking problems is also suggested by the high rate of comorbidity of alcohol use disorders with other mental disorders (e.g., anxiety, mood, and antisocial personality disorders) that involve abnormalities in affective response (e.g., Regier et al., 1990) as well as in the observation that negative emotional traits and states are often associated with alcoholic relapse (e.g., Hodgins, el-Guebaly, & Armstrong, 1995). The myriad of such connections suggests that study of the alcohol-

emotion nexus is critical for understanding drinking and its attendant problems. Integration of the diverse and complex array of variables and mechanisms that comprise this connection is a challenging task. In attempting to tackle it, we maintain that psychology is the discipline perhaps best equipped to make such a contribution because of its potential to overcome artificial boundaries between biological, psychological, and social levels of analysis.

We begin with a brief review of prominent contemporary theories that specifically address alcohol-emotion relationships. We then survey the theoretical and basic research literature in affective science to develop a multidimensional, multilevel model of emotion and identify research directives that derive from this model. With this framework in mind, we turn to examination of emerging evidence regarding alcohol's effects on affective response. Because experimental analog investigations of these phenomena afford the greatest opportunity for elucidation of the mechanisms that might underlie connections between alcohol and emotion across different levels of analysis, we place our emphasis there, with special attention to one particular aspect of the relationship: the impact of acute alcohol intoxication on precisely measurable human psychophysiological response to emotional stimuli. We view a better understanding of the interplay between acute intoxication and emotional response as an essential foundation for enlightened application of affective science to the more clinical domain of chronic alcohol use disorders within which experimental administration of alcohol is proscribed for ethical reasons. Accordingly, in the concluding section, we not only seek to integrate research findings within the basic research framework of affective science to plot future research directions there but also to suggest their possible relevance to clinical science applications.

CONTEMPORARY THEORIES OF ALCOHOL-EMOTION RELATIONSHIPS: THE ROLE OF COGNITION

Although it continues to hold a central position in key theories of drinking and alcoholism, the thesis that alcohol reliably reduces stress or dampens response to threatening stimuli (e.g., Sher, 1987) is without convincing empirical support, despite more than 50 years of intense scrutiny. Even greater doubt surrounds the purported mechanism(s) that might underlie purported stress response dampening (SRD) effects (Greeley & Oei, 1999). Equivocal evidence pertinent to the SRD thesis has been interpreted to indicate that alcohol intoxication does not invariably reduce distress but rather that it does so only under certain circumstances and that it may involve mechanisms other than direct cause alone (Stritzke, Lang, & Patrick, 1996). Specifically, it has been proposed that alcohol can function as an anxiolytic through its disruption of attention to stressors and/or by altering evaluation of the aversiveness of threatening events. At least two important models consistent with this broad cognitive disruption perspective have emerged:

the attention allocation model (Steele & Josephs, 1988, 1990) and the appraisal disruption model (Sayette, 1993). Although to date the predictive scope of these cognitive mediational models has been limited to the effects of alcohol on negative affect, Sayette (1993) allowed that alcohol may differentially affect response to benign or positive information and suggested that future theorizing and research might profitably consider both phenomena. In any case, a brief overview and evaluation of the basic tenets and status of attention allocation and appraisal disruption approaches is in order.

Attention Allocation

The attention allocation model (Steele & Josephs, 1988, 1990) posits that alcohol intoxication modulates affective experience through its effects on information processing. Alcohol's pharmacological action on the brain is thought to impair cognitive activity that requires controlled, effortful processing and restrict attention to the most salient stimuli impinging on the drinker. Although near-ataxic doses of alcohol could obviously prevent concern about any stressor by disrupting all processing of it (Steele & Josephs, 1990), the means by which lower doses might reduce anxious response is not as clear. Attention allocation theory proposes that at more modest doses, psychological distress can be reduced by a complex interaction between alcohol-modified information processing and certain contextual conditions. More specifically, this interaction is thought to involve the combined effects of intoxication in reducing attentional capacity and the presence and nature of stimuli and task demands that compete for available attentional resources. To the extent that attentional resources are captured by nonaversive distractions, there is enhanced potential for anxiety reduction. In fact, if distracting activities are highly demanding, worry over an impending or concurrent negative event can be diminished even without alcohol intoxication (Josephs & Steele, 1990). Nonetheless, alcohol-induced reduction of attentional capacity is regarded as essential to the effects of intoxication on emotion, and moderate doses of alcohol are not predicted to attenuate stress responding in the absence of concurrent distractions. Indeed, proponents of this approach have speculated that drinking in the absence of stimuli that compete with a stressor for attentional resources may even increase the impact of a stressor by intensifying attention to it. Although Steele and his colleagues have provided some support for their model, unfortunately the data have generally been limited to self-reports of anxiety that do not exactly coincide with the presence of distractors (cf. Josephs & Steele, 1990; Steele, Southwick, & Pagano, 1986).

Appraisal Disruption

The appraisal disruption model shares many of the tenets of the attention allocation model, but concurrent distraction during processing of emo-

tional information is viewed merely as one way in which appraisal of that information can be disrupted (Sayette, 1993). The appraisal disruption model more generally holds that alcohol acts pharmacologically to interfere with the initial appraisal of emotional information by constraining the spread of activation of associated information previously established in long-term memory. A central prediction of this model is that individuals who consume alcohol before they are made aware of an imminent stressor are more likely to show SRD than those already aware of the stressor at the time of drinking. In support of this hypothesis, Sayette's (1993) review of the relevant literature noted that none of the psychophysiological studies providing details of a stressor to participants before they drank found SRD effects and that several such studies actually reported an increase in physiological stress responses. However, he also acknowledged that SRD effects were not observed consistently even when participants gained detailed knowledge of the stressor after drinking. Thus, the explanation that alcohol's anxiolytic effects are mediated by disrupted appraisal of stressors remains tentative.

Despite their differences, these two recently developed cognitive models of alcohol-emotion relations converge on the view that alcohol's influence on affective reactivity, at least at the moderate BALs typically tested, probably does not occur directly at the level of subcortical motivational systems but rather through its effect on higher information processing centers that participate in "top-down" regulation of emotional response (cf. LeDoux, 1995). We believe that that adequate evaluation of this thesis depends on more sophisticated conceptual approaches to the construct of emotion and on research methods that permit exploration of the impact of alcohol on higher associative processes concurrent with refined measurement of affective response. Fortunately, recent advances in theory and methodology guiding basic affective science, including neuroscience, offer just such an opportunity to enhance our understanding of how alterations in emotional reactivity might relate to alcohol use and its associated disorders. Reference to the same literature should also ensure that neurobiologically plausible models of the mechanisms underlying alcohol's actions on emotion response are developed. Following this tack, we now move to an overview of current conceptualizations of emotion, their biological substrates, and issues critical to clarification of the alcohol-emotion nexus.

AFFECTIVE SCIENCE: THEORY, METHODS, AND DIRECTIVES FOR ALCOHOL RESEARCH

There is now considerable agreement in affective science that emotion or affective state involves central activation of "action dispositions" or response tendencies that prepare an organism to act (Davidson, Jackson, & Kalin, 2000; Izard, 1993; Lang, 1995). Theorists (e.g., Lang, 1995; Larsen &

Diener, 1992) suggest that these action dispositions are organized within a two-factor framework, defined by the primary, or what some (Lang, Bradley, & Cuthbert, 1990) have called the "strategic," affective dimensions of arousal (degree of activation) and valence (pleasant or unpleasant). Moreover, emotional response within this two-factor framework is believed to represent activity in two primary brain motivational systems: an aversive system that governs defensive reactions and an appetitive system that governs consummatory and other approach behaviors (Davidson et al., 2000; Lang, 1995).¹ Substantial evidence from both animal and human studies indicates that the amygdala is critically involved in the operations of the aversive motivation system (for a review, see Ledoux, 1996), whereas activation of the ventral striatum (including the caudate, putamen, and the nucleus accumbens) has been observed in studies that involved manipulation of positive affect or administration of primary reinforcers (for a review, see Davidson et al., 2000). Meanwhile, the ventral striatum, which is richly innervated by dopaminergic neurons from the mesolimbic dopamine system, has been implicated in incentive motivation (Berridge & Robinson, 1998) and likely plays an important role in pregoal attainment of positive affect (Davidson, 1994). Substantial evidence from lesioning, neuroimaging, and electrophysiological studies also points to various sectors of prefrontal cortex as critical to both positive and negative affect, with important distinctions between these sectors and left versus right hemispheres for processes related to emotion regulation and goal-directed behavior (Davidson et al., 2000; Harmon-Jones, 2003).

A second important theme evident in contemporary affective science is that emotional reactivity involves the interplay of primitive action mobilization systems and higher cortical processes. In other words, subcortical motivational systems can both influence and be influenced by more complex cognitive processes such as attention, perception, declarative memory, and imagery, through reciprocal connections between subcortical and cortical regions. Thus, the processes involved in emotion cannot be fully understood without reference to potentially interactive cognitive processes. In fact, there is mounting evidence to suggest that emotional phenomena involve a hierarchy of neural, sensorimotor, motivational, and cognitive processes (Izard, 1993; LeDoux, 1996). For example, information-processing theories of emotion (e.g., Bower, 1981) maintain that emotional episodes are represented in associative networks that differ from other memory structures in that they include links between cortical processing regions and the subcortical systems that govern primitive defensive and appetitive reactions (Lang, 1995).

¹An alternative perspective is that positive affect (PA) and negative affect (NA) dimensions represent the primary axes of emotional space (Tellegen, 1985). This viewpoint derives from a somewhat different database involving primarily mood reports, but many contemporary theorists (Lang, 1995; Larsen & Diener, 1992) have been inclined to view it as complementary to rather than incompatible with the arousal-valence model. This view also can be reconciled with Gray's (1987) model, which includes a nonspecific arousal system that is driven by both appetitive and aversive motive systems.

In other research, interesting dissociations have been observed between the fear-conditioning capacities of animals, such that selective reductions in fear responses evoked by complex multimodal, contextual cues—but not simpler unimodal, punctuate cues—can be observed after surgical ablations of the hippocampus (Kim, Rison, & Fanselow, 1993; Phillips & LeDoux, 1992). These findings have been interpreted to mean that connections between sensory processing regions (thalamus, auditory cortex) and the amygdala are sufficient to mediate fear conditioning to simple or explicit cues but that pathways from the hippocampus to the amygdala are required for fear to be elicited by more elaborate or diffuse contextual cues that require information processing. In general, more complex emotion-eliciting stimuli or situations are more likely to entail higher order processing and top-down connections to subcortical emotion centers. Provocative data from emerging research now challenge the assumption that anxiolytic effects of alcohol are exclusively or even typically attributable to direct actions of the drug on the primary, subcortical motivational (i.e., approach or defensive) systems. Instead, evidence points to alcohol's impact on higher level cognitive functions—including attention, appraisal, and declarative memory—that normally participate in and mediate affective processing and expression (Sayette, 1993; Steele & Josephs, 1990; Stritzke et al., 1996).

A third theme highlighted by contemporary affective science concerns the need to consider the temporal nature of emotional processes. Emotions in general, and their psychophysiological manifestations in particular, are dynamic events, and thus the time frame in which they are considered is important. Davidson and his colleagues (Davidson, 1998; Davidson et al., 2000) have been influential in directing attention to the chronometry of emotional response in the study of affective contributions to psychopathology. More specifically, they argued that emotional response is a dynamic phenomenon and that it can be characterized by possibly independent components including tonic level, threshold to respond, initial peak amplitude, risk time to peak, and recovery time (for more detail, see Davidson, 1998). Each of these components of emotional response may represent the action of different underlying neurobiological mechanisms, with variations in some, but perhaps not others, particularly important to understanding acute alcohol challenge effects as well as their relevance to alcohol use disorders. In this connection, Levenson (1987) noted that the grain of physiological measurement in alcohol studies has often been too coarse (20-second to 30-second averages) to allow precise tracking of the effects of alcohol on emotional processes as they unfold over time, a shortcoming that still characterizes much of the alcohol challenge research addressing emotional response.

Finally, affective scientists have highlighted the need to differentiate initial emotional response from the processes involved in subsequent regulation of emotion (Davidson, 1998; Gross, 1999). The latter includes a broad array of both automatic and volitional processes that are designed to en-

hance, suppress, or maintain the strength of an initial emotional response. Influences of these regulatory processes may be observed in one or more temporal components of emotional response (e.g., duration-recovery, but not initial response intensity). Given that affect regulation is held by many addiction theorists (e.g., Baker et al., 2004; Cooper et al., 1995; Koob & Le Moal, 2001) to be a primary motive for the use of drugs, including alcohol, measurement with sufficient temporal resolution to parse these components is important.

In sum, affective science directs us to (a) examine the entire range of the emotional valence dimension (i.e., both negative and positive emotions) as well as emotional arousal, with explicit reference to their neurological substrates; (b) consider the dynamic interplay between cognitive and affective processes; and (c) use measures with adequate precision and temporal resolution to parse emotional response into its constituent components and effectively separate emotional response from processes that regulate emotion. The following section reviews an emerging database that shows promising signs of progress toward embracing these directives.

ALCOHOL AND THE MULTIDIMENSIONAL-MULTILEVEL FRAMEWORK OF EMOTION

Alcohol and Arousal

Almost a century ago, Smith (1922) used the "psychogalvanic reflex" to measure the intensity of emotional arousal in the investigation of the effects of alcohol on reactions to emotional stimuli. He reported that alcohol diminished electrodermal response to affect-laden words in an association test but hastened to add that this observation revealed little about the specific valence of emotion involved. In other words, the observed changes were understood to reflect primarily the intensity of nonspecific arousal that might be associated with any kind of emotional excitement, positive or negative. In the ensuing decades, Smith's finding of alcohol-attenuated electrodermal response to affective stimuli was often replicated, using both verbal (e.g., Lienert & Traxel, 1959) and sensory (e.g., Carpenter, 1957) stimuli, but his cautions about their interpretation have gone largely unheeded.

The classic critique of activation theory by Lacey (1967) was important in further exposing the liabilities of heavy reliance on the arousal construct in descriptions of complex physiological response patterns, including those pertinent to emotion. He derived this conclusion from evidence that (a) somatic and behavioral arousal do not necessarily covary, particularly after pharmacological manipulations; (b) different physiological indices of activation are dissociable; and (c) specific situations prompt unique patterns of physiological response, depending on the nature of the stimuli, their con-

text, and task requirements. The implications of Lacey's theoretical advances for the study of alcohol-emotion relationships were recognized by Naitoh (1972), who urged researchers to move beyond simplistic, activation-based studies of stress reduction, toward development of a perspective permitting exploration of possible connections between alcohol-induced physiological arousal and the experience of "pleasant drunkenness." Naitoh proposed that interpretation of alcohol-mediated changes in the relatively ambiguous, altered state of arousal occasioned by intoxication may be either pleasant or unpleasant depending on the context.

The best alcohol-emotion experiments of the 1980s (e.g., Levenson, Sher, Grossman, Newman, & Newlin, 1980) advanced the assessment of the arousal dimension of emotion through the simultaneous and continuous psychophysiological measurement of multiple aspects of autonomic reactivity (e.g., heart rate, blood pressure, and skin conductance), thereby allowing for control of baseline differences and finer grained analyses of response patterns and changes over time. Although the initial enthusiasm these experiments generated by suggesting the possibility that systematic individual variation in alcohol-induced SRD represented a risk factor for problem drinking (e.g., Levenson, Oyama, & Meek, 1987; Peterson & Pihl, 1990; Sher & Levenson, 1982) was later tempered by failures to replicate (e.g., Sayette, Breslin, Wilson, & Rosenblum, 1994; Sher & Walitzer, 1986), they left their mark on measurement strategy.

Despite these methodological improvements, however, alcohol-emotion research stagnated in its maintenance of a relatively narrow focus on the conditions under which alcohol intoxication influences the impact of anxiety-provoking stimuli or stressful situations on nonspecific psychophysiological measures of autonomic arousal and self-reports (cf. Sayette, 1993; Sher, 1987). Persistence in this approach precluded capitalization on important advances in the conceptualization of emotion, including recognition and measurement of its valence dimension and attention to how alcohol effects may be mediated by cortical-subcortical interactions and/or cognitive processes that change as a function of acute intoxication. Fortunately, recent work illustrates the potential benefits of application of more sophisticated conceptualization and measurement of emotion for understanding how alcohol affects it.

Alcohol and Emotional Valence

Although self-report measures of emotional valence have been a staple of emotion research for years, these measures are suboptimal because they are subject to distortion by experimental demand and intentional dissimulation. Such limitations provided a major impetus for the development of psychophysiological indices of emotional valence tied closely to underlying action dispositions and having limited vulnerability to spontaneous efforts at voli-

tional control (see chap. 3, this volume). In particular, alcohol research has begun to profit from the application of such methods that include evaluation of facial expression via assessment of electromyographic activity and inference of affective disposition from startle response patterns. Increases in electromyogram (EMG) activity over the corrugator supercilium muscle that produces frowning and increases over the zygomaticus major muscle involved in smiling have consistently covaried with the unpleasantness and pleasantness of emotions, respectively (for a review, see Tassinari & Cacioppo, 1992). In addition, the magnitude of the startle response to a sudden, intense stimulus "probe" (e.g., a burst of white noise) has been shown reliably to reflect the valence of emotional state in that startle reactivity is augmented if the probe occurs in the context of an unpleasant stimulus and attenuated if the probe coincides with a pleasant stimulus (Lang, 1995; Lang et al., 1990).

Direct Effects of Alcohol on Emotional Valence

Capitalizing on these developments in the measurement of emotional valence, Stritzke, Patrick, and Lang (1995) conducted the first alcohol challenge experiment to manipulate positive as well as negative affect in a design based explicitly on a multidimensional (arousal-valence) model of emotion. Participants received either a moderate dose of alcohol or no alcohol before viewing pleasant, neutral, and unpleasant pictures, with the affect-laden images matched for arousal. Eyeblink startle response was used to index the valence of the emotional response evoked by the slides. Changes in emotional valence were also assessed using EMG recording of corrugator ("frown") activity, and skin conductance responses were assessed to index of physiological arousal. Results indicated that although alcohol diminished the overall magnitude of both startle and phasic skin conductance response—regardless of the valence of the foreground images—it did not disturb the normal affective modulation of startle. In other words, regardless of the general reduction in startle magnitude observed in the alcohol group during picture viewing, intoxicated subjects and no-alcohol control participants showed similar, higher magnitude startle blinks to probes delivered during aversive images and lower magnitude blink reactions to probes during pleasant images, both relative to neutral.² A comparable valence effect (i.e., unpleasant > neutral

²It is worth noting in this context that the general suppressant effect of alcohol on overall startle response reactivity has been known for some time (e.g., Greenberg & Carpenter, 1957). In addition to the recent human eyeblink studies cited in the following sections (Curtin, Lang, Patrick, & Stritzke, 1998; Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001; Donohue et al., in press; Stritzke et al., 1995), alcohol challenge has also been shown to substantially reduce general startle reactivity, as indexed by whole-body motor reactions, in lower animals (e.g., Pohorecky, Cagan, Brick, & Jaffe, 1976; Rassnick, Koob, & Geyer, 1992). However, the startle response is a reflex, and care must be taken not to confuse this effect on overall startle response reactivity with either the affective modulation of startle or traditional sympathetic nervous system indices (e.g., skin conductance response) typically linked to the arousal component of emotional state. Although it is possible that a general decrease in startle associated with rising BAL reflects a reduction in activity at the level of the

> pleasant) was also observed for corrugator EMG reactions in both beverage groups. These results run counter to the SRD perspective (Sher, 1987), which would predict that alcohol should selectively block negative reactions to aversive images.

The startle response was also used in two more recent studies (Curtin et al., 1998, 2001) examining the effect of moderate doses of alcohol on negative affective reactions during exposure to explicit threat. In Curtin et al. (1998), sober and moderately intoxicated participants were presented with a series of 2-minute light cues denoting either (a) the possibility that an electric shock could be delivered at any instant (red light = threat), or (b) that no shock would be administered (green light = safe). Startle response was assessed intermittently during these cueing periods. In addition, autonomic activity (skin conductance and heart rate), and corrugator EMG were monitored throughout the experiment. Results indicated that the presence (vs. absence) of shock threat evoked predictable increases in autonomic arousal, in EMG indices of facial frowning, and in startle response magnitude. However, there was no main effect of alcohol on magnitude of negative affective response across these various indices of affective valence. Similarly, in a conceptual replication that included numerous brief (approximately 2-second) exposures to periods of electric shock threat, Curtin et al. (2001) demonstrated that *fear-potentiated startle* (FPS; i.e., the elevation of startle responses observed during the presence of a fear or threat cue vs. during its absence) was not altered by a moderate dose of alcohol when the threat cue was the only focus of attention.

Taken together, these three well-controlled experiments failed to support the notion that alcohol has a direct, suppressive effect on negative affective response. However, one potentially important limitation of this series of studies was neglect of possible dose-response effects. In each of the studies (Curtin et al., 1998, 2001; Stritzke et al., 1995), all participants in the alcohol condition attained only a single, moderate BAL. This was unfortunate because some evidence (e.g., Sher & Walitzer, 1986; Stewart, Finn, & Pihl, 1992) suggests that SRD effects may be more reliably observed at higher alcohol doses.

To address this limitation, Donohue, Curtin, Patrick, and Lang (in press) examined emotional reactivity to the same pleasant, unpleasant, and neutral pictures used in Stritzke et al. (1995) but presented them to participants for whom beverage conditions resulted in zero, low, moderate, or high BALs. The illuminating result emerging from this study was that, as predicted by the SRD model, alcohol selectively diminished the startle response index of negative affective responding to unpleasant images but only when responses

nucleus reticularis pontis caudalis, which, in turn, could reflect an overall reduction in arousal state—that is, nucleus reticularis pontis caudalis activity may indeed covary with general brainstem reticular activation—but this has yet to be confirmed.

in the moderate- and high-BAL groups were contrasted with those in the zero- and low-BAL groups. Startle response inhibition to pleasant images was unaffected by any BAL. To our knowledge, this was the first experiment to demonstrate a selective SRD effect—albeit one confined to higher BALs—using psychophysiological methods specifically designed to index the valence of emotional response.

Possible Cognitive Mediation of Alcohol–Emotion Relationships

Our earlier summary of contemporary models of alcohol–emotion relations (i.e., attention allocation and appraisal disruption) indicates a growing consensus that at least under certain circumstances and/or at certain BALs, alcohol might exert its effect on emotional responding indirectly through its alteration of cognitive function. Two of the experiments described earlier (Curtin et al., 1998, 2001) incorporated design features to evaluate this potential role of cognitive mechanisms in the effects of alcohol on emotional response. They provided converging evidence in support of this thesis. Recall that in Curtin et al. (1998) participants were exposed to alternating 2-minute periods involving the presence or absence of a shock threat, with red or green lights, respectively, serving as cues for the two types of blocks. In addition to this threat manipulation, during half of the cue periods of each type, intermittent presentations of pleasant pictures were made via a large monitor positioned immediately in front of participants. These presentations were intended to compete with the threat-cue signal lights for participants' attention. Comparative analysis of the FPS index of emotional response across all possible combinations of beverage, threat, and pleasant picture conditions revealed that FPS was attenuated by alcohol only when pleasant picture presentations competed with threat signals for participant attention. When there were no pleasant pictures distractors, intoxicated participants showed the same level of FPS as sober participants, whose FPS reactions were unaffected by picture presentations. The fact that FPS was significantly reduced only among intoxicated participants distracted from their threatened state by pleasant pictures is consistent with the notion that alcohol's effect on negative emotional response can be mediated by its effect on cognitive functioning because it was the divided attention occasioned by the combination threat–picture condition that involved the greatest cognitive demand, particularly in terms of simultaneous allocation of attention to the processing of competing stimuli. However, without an online measure of alcohol-induced changes in the processing of threat cues, one cannot be altogether confident of just what it was that captured participants' attention. To clarify this, a direct measure the cognitive processing of threat cues concurrent with the measurement of emotional response to them would be needed.

To address this issue, the Curtin et al. (2001) experiment incorporated simultaneous measurement of threat-cue processing, using event-related po-

tentials (ERPs), and of fear responding, using FPS. Sober and moderately intoxicated participants viewed compound stimuli that varied across two dimensions (color and semantic category) in two task conditions. In a threat-focused condition, participants attended only to the semantic category of the word (words came from two semantic categories: animals and body parts), which indicated the threat of administration of electric shock (electric shocks followed a subset of words from one semantic category). Alternatively, in a divided-attention condition, participants were aware of the threat associated with the semantic category of each word but were instructed to attend to the color of the word (red or green) and make a speeded button-press response when words appeared in green type but withhold this response when the word appeared in red. Thus, in the divided-attention condition, the semantic category indicating shock threat was made peripheral or task irrelevant. Results of this study replicated the pattern of affective responding observed in Curtin et al. (1998), with alcohol selectively reducing FPS only in the divided-attention condition in which attention to both the threat parameters and task demands apparently exceeded the cognitive capacity of intoxicated participants. However, the most novel and compelling aspect of this study resided in its online measurement of cognitive processing.

The use of ERPs in the Curtin et al. (2001) experiment afforded an opportunity to evaluate directly the impact of alcohol intoxication on threat-cue processing. In general, the magnitude of the P3 component of the ERP is larger in connection with important stimuli, that is, stimuli that capture or focus attention. Consistent with this principle, P3 was comparably enhanced by the presentation of threat words in the threat-focused condition for both sober and intoxicated participants. Sober participants also displayed comparably enhanced P3 to threat words in the divided-attention condition. However, P3 enhancement to threat words was significantly reduced for intoxicated participants in the divided-attention condition. This last outcome indicates that intoxicated individuals exhibited decrements in threat-cue processing and that the level of FPS associated with threat cues was diminished when participants' alcohol-impaired cognitive capacity was stretched beyond its limits by a task that divided their attention and rendered them less able to process threat cues. In contrast, the responses of sober subjects were not significantly affected by cognitive demands; they apparently had sufficient cognitive capacity to process threat while also performing the assigned reaction-time task. These 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. They also encourage use of the general strategy of simultaneous measurement of cognitive and emotional processes as a promising approach applicable to the broader study of alcohol effects on both positive and negative emotion.

THEORETICAL INTEGRATION, FUTURE RESEARCH, AND POSSIBLE APPLICATIONS

Affective Science Revisited

Our thesis throughout this chapter has been that research on alcohol and emotional response can profit from a conceptualization of emotion that recognizes its multidimensional, multilevel nature. In this connection, we have argued that until recently the valence dimension of emotion has been largely ignored or inadequately represented in alcohol-affect research and noted that this limitation manifests itself in two ways. First, much of the relevant literature has failed to address the possible impact of alcohol on positive emotion altogether, let alone examine its effects across the entire range of valence within the same study. Second, only the rare experiment has moved beyond self-report measures of emotional valence to include specific psychophysiological indices such as startle response, facial EMG, or frontal cortical electroencephalographic (EEG) asymmetries to tap this critical dimension. In addition, although a number of contemporary theories hold that alcohol's effect on emotion, particularly at low to moderate BALs, may depend on its compromise of cognitive capacity and concurrent demands on it, this hypothesis has yet to be subjected to much rigorous study using appropriate designs and measures.

Mechanisms of Action for Alcohol

Application of a multidimensional, multilevel conceptualization of emotion provides a useful framework for interpretation of the extant literature on alcohol and affective response and for generation and evaluation of further hypotheses. Our review of available evidence suggests that for intoxication within the range normally experienced by humans, the influence that alcohol exerts on emotional response might involve at least two mechanisms, depending on BAL. When BALs are relatively high, alcohol appears to selectively dampen defensive responding to unpleasant stimuli via a rather direct and specific mechanism, regardless of context (Donohue et al., in press; Sher & Walitzer, 1986; Stewart et al., 1992). Donohue et al. (in press) offered perhaps the most convincing demonstration of this by using affect-modulated startle as a key measure of emotional response, thereby providing an arguably more direct index of activation of the subcortical components of the defensive motivational system, including the amygdala, than is available in the typical SRD study. This is important, considering evidence reviewed by McBride (2002) indicating that intermediate to high doses of ethanol (but not low doses) produce selective activation of GABAergic neurons in the central nucleus of the amygdala (CeA) in rats, as indexed by changes in levels of *c-fos*, a biochemical marker

of neuronal activity (Morales, Criado, Sanna, Henriksen, & Bloom, 1998). Because gamma-aminobutyric acid (GABA) is the major inhibitory neurotransmitter in the central nervous system, this suggests that increased GABA activity in the CeA, occasioned by higher doses of alcohol, constitutes a plausible mechanism underlying the decreased negative emotional response observed for startle and accounts for the depressant and anxiolytic effects associated with elevated BALs (cf. Fromme & D'Amico, 1999). This interpretation also resonates with results from studies showing that animals exposed to classic approach-avoidance conflict manipulations tend to exhibit reduced inhibition by fear and hence resolve conflicts in the approach direction (cf. Gray, 1987).

In contrast to the apparently direct effect of alcohol on negative emotions observed at BALs above a certain threshold, the anxiolytic effects of alcohol at low to moderate BALs appear to depend on cognitive mediation. Curtin et al. (1998, 2001) provided convincing evidence that the ability of modest alcohol intoxication to attenuate FPS was confined to contexts in which attention to threat faced competition from other salient stimuli or prioritized task demands within individuals whose cognitive processing capacity had been compromised by intoxication. In these same studies, a significant level of FPS—equivalent to that observed in sober participants—was evident among intoxicated participants when the context was devoid of stimuli that might compete for attention to threat.

Also relevant here is the work of Melia, Ryabinin, Corodimas, Wilson, and LeDoux (1996) showing "dissociations" in the fear conditioning of animals as a function of alcohol administration. These investigators examined explicit cue versus contextual cue fear conditioning in rats that were administered low and moderate doses of ethanol, with behavioral freezing used as an index of fear. They reported that alcohol did not affect conditioning to tone cues explicitly paired with the shock, but it blocked acquisition of the association between the situational context (novel cage) and aversive stimulation (shock). These findings by Melia et al. (1996) are consistent with the hypothesis that low to moderate doses of alcohol can reduce sensitivity to fear cues by interfering with higher order processing. As described earlier, disruption of hippocampal functioning is one potential mechanism by which alcohol might produce such effects. Although apparently not essential to explicit fear learning, input from the hippocampus to the amygdala is clearly critical for contextual fear learning (LeDoux, 1995). Because alcohol has well-documented effects on a variety of cognitive functions, including vigilance and attention (Casbon, Curtin, Lang, & Patrick, 2003; Curtin & Fairchild, 2003; Holloway, 1994), that reflect its impact on neural systems probably not intimately involved with the hippocampus, it seems likely that other systems are involved as well. For instance, Davis, Walker, and Lee (1999) suggested that the bed nucleus of the stria terminalis comprises the basic substrate of an anxiety system analogous to, but functionally distinct

from, the amygdaloid fear system. This could represent another avenue by which alcohol exerts its effects on emotional response.

Promising Avenues for Further Research

The approach to alcohol–emotion research we have promoted encourages consideration of dimensions, variables, and measures often neglected in prior studies of these phenomena: the joint consideration of the valence and intensity of emotion-eliciting cues, the context of affective stimulation, the brain systems and cognitive processes required to register and process cues of different sorts, and the dynamic nature of affective responding. A few of the particularly salient research questions and promising directions that emanate from a strategy that addresses this neglect are outlined here, together with some brief suggestions about what can be learned from the affective science of alcohol and emotion and how it might be applied to clinical science problems.

Specification of the positive emotional concomitants of drinking has remained an elusive goal, probably in part because of the inadequacies of methods applied to the study of such effects so far. Thus, one high-priority starting point should be development and/or utilization of alternative means to phasically manipulate positive affect. In particular, manipulations of pregoal-attainment positive affect (Davidson et al., 2000) that occurs simultaneously with strong approach motivation have yet to be used in alcohol research and may yield interesting results, especially given observed effects of alcohol on pregoal states surrounding, for example, sexual and aggressive behavior. EEG asymmetry measures that represent affective valence may prove to be particularly useful indices in such studies (for a review, see Harmon-Jones, 2003).

Further tests of the cognitive mediation of alcohol effects on both positive and negative emotion should be conducted. Such tests can follow from two independent methodological approaches. First, additional studies that simultaneously measure both affective and cognitive processing of emotionally evocative stimuli are clearly warranted, given the strong evidence for cognitive–emotional interactions. Curtin et al. (2001) can serve as a model for how to operationalize simultaneous, online assessment of both constructs to test critical predictions for cognitive models of alcohol effects. Second, there is a need to develop manipulations specifically designed to dissociate various emotion processing pathways, both direct and indirect (LeDoux, 1995). For example, comparisons of explicit versus contextual fear conditioning in humans as a function of alcohol intoxication are of interest, particularly given that surgical ablation of the hippocampus in lower animals reduced fear response to complex multimodal, contextual cues but not to simpler explicit cues (Kim et al., 1993; Phillips & LeDoux, 1992). Such results have obvious implications for the work of Melia et al. (1996), which

revealed that contextual, but not explicit, fear responding in animals was reduced by low to moderate doses of alcohol. Grillon and Davis (1997) developed an experimental paradigm to examine contextual fear responding in humans using FPS. In applying it to alcohol research, a strong prediction would be that moderate doses of alcohol should eliminate the context-related enhancement of startle but should have no effect on startle potentiation associated with simpler explicit threat cues. Such a result would provide convincing evidence of a differential impact of alcohol as a function of the complex cognitive processes involved in context cueing.

Still another promising avenue would be systematic examination of alcohol effects on the constituent components of emotional response (e.g., response threshold, peak intensity, duration, subsequent regulatory processes). To this end, measures with precise temporal resolution, such as the startle response, could be combined with innovative manipulations to probe the dynamic nature of emotional response. For example, in research on the affective concomitants of tobacco withdrawal, Hogle and Curtin (in press) used FPS to examine both the initial intensity of affective responding and the duration of the affective response to stressors in nicotine-deprived smokers. Of special interest here was the fact that tobacco withdrawal did not appear to alter the initial intensity of response but did delay recovery from the stress, suggesting that a disruption of homeostatic processes related to emotion regulation was at work. In other research, Jackson et al. (2003) combined EEG asymmetry and startle response measures to demonstrate that individual differences in frontal cortical asymmetries predict the persistence but not the initial intensity of affective response to emotionally evocative images. Jackson, Malmstadt, Larson, and Davidson (2000) have also developed an experimental task that dissociates emotional response from volitional emotion regulatory processes using the startle response (for application of this task to study emotion regulation during tobacco withdrawal, see Piper & Curtin, 2006). Similar methods could be fruitfully applied to the study of alcohol challenge effects. Moreover, attention to constituent components of emotion might help reduce the equivocation so often associated with the findings from less refined research approaches and thus better inform us of the mechanisms underlying alcohol effects.

Clinical Implications

At first glance, it might not appear that research addressing the acute effects of alcohol on emotional response in relatively normal drinkers could do much to inform clinical science. However, several insights that can be derived from the foregoing review seem worthy of mention. First, there can be little doubt that alcohol expectancies and motives, the psychiatric disorders comorbid with alcohol use disorders, and the antecedents and consequences of nonproblem and problem drinking alike are intimately related to

emotion. This fact alone should make understanding of the nature and mechanisms of alcohol–emotion relationships of interest to clinicians. Appreciation of the role of cognitive processes in the dynamic interplay between drinking and affect seems particularly important. Consider, for example, the forces at work in decision making about initiation and continuation of a drinking episode for the typical alcoholic. Often such individuals start with ambivalence: They are aware from experience that drinking can have immediate and simple reinforcing (euphoric, anxiolytic, etc.) emotional effects, but they also know that drinking often has more delayed and complex punishing effects (impaired role performance, conflicts, illness or injury, etc.) with attendant aversive emotions. Particularly in the absence of alternative coping responses, ongoing or anticipated distress can create pressure to resolve such ambivalence by indulgence in the immediate gratification expected from drinking. As we have seen, however, even modest intoxication compromises cognitive capacity, leading to its association with shallower and more simplistic thinking and a tendency to confine focus to only the most salient aspects of the immediate situation. This concurrently serves to decrease access to more distal concerns about adverse consequences that might otherwise restrain further drinking, hence precipitating an elevated risk for just the sorts of outcomes that define alcohol use disorders. Greater awareness of such processes could facilitate refinement of relapse prevention (Marlatt & Donovan, 2005) and other interventions designed to address drinking problems.

CONCLUSION

The first wave of empirical evidence yielded by psychophysiological studies designed to be sensitive to emotional valence suggests that alcohol can act both directly on subcortical affect systems and indirectly via acute impairment of cognitive functioning. Further, the relative contribution of each of these effects on the emotional experience of the individual is likely interwoven with variations related to BAL, contextual factors, and individual differences. These results and their implications provide ample reason to continue to develop and use a multidimensional, multilevel framework for conceptualizing the alcohol–emotion nexus and pursuing its implications for drinking and alcohol-related problems.

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