Effects of Alcohol on Person Perception: A Social Cognitive Neuroscience Approach

Bruce D. Bartholow University of North Carolina at Chapel Hill Melanie A. Pearson University of Missouri—Columbia and University of Illinois at Urbana–Champaign

Gabriele Gratton and Monica Fabiani University of Illinois at Urbana–Champaign

The acute effects of alcohol on cognitive processing of expectancy violations were investigated using event-related brain potentials and a cued recall task to index attentional and working memory processes associated with inconsistency resolution. As predicted, expectancy-violating behaviors elicited larger late positive potentials (LPP) and were recalled better than expectancy-consistent behaviors. These effects were moderated by alcohol and the valence of initial expectancies. For placebo group participants, positive targets performing negative behaviors elicited the largest LPP responses and were recalled best. For those in the alcohol groups, negative targets behaving positively elicited the largest LPP and recall responses. These findings suggest that alcohol does not globally impair working memory processes in person perception but instead changes the nature of valenced information processing. Findings are discussed in the context of alcohol's effects on working memory processes, reward sensitivity, and the prefrontal cortical structures thought to mediate them.

Alcohol is a complex drug, producing both desirable and undesirable effects for the user. For example, alcohol is known to activate neural reward systems associated with positive affect (e.g., Fromme & D'Amico, 1999), and in the realm of person perception, alcohol is credited with making potential mates appear more attractive than they otherwise would. However, these positive effects are offset by alcohol-related impairments in cognitive function that can lead to negative consequences for the drinker. Basic information-processing operations including attention (see Jääskeläinen, Näätänen, & Sillanaukee, 1996), learning and encoding of information (e.g., Birnbaum, Johnson, Hartley, & Taylor, 1980), retrieval of information from memory (e.g., Nelson, McSpadden, Fromme, & Marlatt, 1986), and the overall rate of information processing (e.g., Fillmore, Carscadden, & Vogel-Sprott, 1998) are known to be adversely affected by alcohol consumption.

A number of theories (e.g., Sayette, 1999; Steele & Josephs, 1990) posit that alcohol-related changes in social behavior are mediated by impairment of higher order cognitive functions, leading to incomplete stimulus evaluation and short-sighted decision making. Such information-processing deficits have been used to explain alcohol's effects on aggression (e.g., Hoaken, Giancola, & Pihl, 1998), likelihood of risky sexual contact (e.g., Cooper, 1992; MacDonald, Zanna, & Fong, 1996), and other risky behaviors more generally (MacDonald, Zanna, & Fong, 1995). Given the evidence of alcohol's influence on attention and cognition, processes involved in person perception (e.g., impression formation, stereotyping, categorization) may differ in important ways following alcohol consumption. Furthermore, the fact that many of the negative consequences associated with alcohol consumption are interpersonal in nature (e.g., fights, risky sex, etc.) suggests that basic dynamics of person perception are important to understanding how consumption can lead to problems. Some initial evidence has suggested that alcohol affects causal inferences, leading to exaggeration of either situational or dispositional causes for behavior depending on which factors are most salient (Herzog, 1999). However, the influence of alcohol in person perception has not been systematically studied, and the precise nature of alcohol's effects on the encoding and interpretation of others' behavior remains largely unknown.

The ability to deal with the unexpected behavior of others is one aspect of person perception that could prove particularly suscep-

Bruce D. Bartholow, Department of Psychology, University of North Carolina at Chapel Hill; Melanie A. Pearson, Department of Psychological Sciences, University of Missouri—Columbia, and Beckman Institute, University of Illinois at Urbana–Champaign; Gabriele Gratton and Monica Fabiani, Department of Psychology and Beckman Institute, University of Illinois at Urbana–Champaign.

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Correspondence concerning this article should be addressed to Bruce D. Bartholow, Department of Psychology, CB 3270, Davie Hall, University of North Carolina, Chapel Hill, North Carolina 27599. E-mail: bartholow@unc.edu

tible to alcohol's acute effects. Predicting others' behavior-and detecting when behavior is inconsistent with predictions-is a basic and fundamental aspect of social perception (e.g., Jones, 1990; Olson, Roese, & Zanna, 1996). A large number of studies have indicated that encountering behavior that violates previously established expectations results in more elaborative and effortful processing as perceivers attempt to integrate new, discrepant information with existing beliefs (e.g., Bartholow, Fabiani, Gratton, & Bettencourt, 2001; Hastie & Kumar, 1979; Srull, Lichtenstein, & Rothbart, 1985; Stangor & Duan, 1991), a process sometimes referred to as inconsistency resolution (Srull & Wyer, 1989). The elaborative processing associated with inconsistency resolution often is reflected in a recall advantage for expectancy-violating (EV) information (e.g., Bartholow et al., 2001; Stangor & Duan, 1991; see also Higgins & Bargh, 1987; Stangor & McMillan, 1992).

Such findings have led researchers to conclude that the process of inconsistency resolution involves working memory (see, e.g., Macrae, Bodenhausen, Schloersheidt, & Milne, 1999). Working memory can be described as a set of cognitive processes that enable temporary storage, manipulation, and movement of information between short-term and long-term stores in the service of behavioral or self-regulatory goals (see Baddeley, 1986, 1992). The working memory system is generally divided into two components: short-term storage and a set of "executive processes," including (among others) the focusing of attention on relevant task demands as well as the monitoring and updating of working memory content (see Smith & Jonides, 1999). Brain imaging data have indicated that these functions are primarily mediated by activity in the dorsolateral prefrontal cortex (DLPFC) and areas of the parietal cortex (Cohen et al., 1994; McCarthy et al., 1996; Petrides, 2000).

A number of studies have provided support for the idea that executive working memory is required to carry out some or all of the steps involved in inconsistency resolution. Specifically, when aspects of executive function are impaired (e.g., by having participants perform a concurrent mental task), the typical recall advantage for inconsistent information is lost (e.g., Dijksterhuis & van Knippenberg, 1995; Macrae et al., 1999; Macrae, Hewstone, & Griffiths, 1993). However, not all reductions of cognitive resources impair inconsistency resolution. In a compelling demonstration of this notion, Macrae et al. (1999) found that when participants were asked to generate random digits (an activity known to impair executive function) during an impressionformation task, they later recalled more schema-consistent information about targets. In contrast, participants asked to concurrently repeat a single word while forming impressions (a "nonexecutive" cognitive task) recalled more schema-inconsistent information about the targets.

Some evidence has suggested that alcohol impairs the functioning of executive working memory, at least under some conditions (e.g., Finn, Justus, Mazas, & Steinmetz, 1999). For example, alcohol consumption has been shown to limit one's ability to engage in controlled, effortful processing of verbal information (e.g., Lister, Eckardt, & Weingartner, 1987; Tracy & Bates, 1999), particularly that related to integration of new information with previously stored knowledge (e.g., Birnbaum & Parker, 1977; Craik, 1977). These findings are consistent with a recent review suggesting that alcohol impairs many processes mediated by the prefrontal cortex (Lyvers, 2000). As such, alcohol consumption might disrupt the process of inconsistency resolution, presumably eliminating the typical recall advantage for unexpected information.

However, the inconsistency-resolution process also has been shown to depend on the valence of initial expectancy information (Ybarra, 2002). A number of studies have indicated that encountering negative information unexpectedly is more likely to engage the inconsistency-resolution process than encountering positive information unexpectedly. For example, Ybarra, Schaberg, and Keiper (1999) found that participants who held positive expectancies about a target later recalled more EV (i.e., negative) than expectancy-consistent (EC) behaviors, whereas participants who held negative expectancies of the same individual recalled both types of behaviors equally well. Other researchers have reported similar results (e.g., Sherman & Frost, 2000; Trafimow & Finlay, 2001; Vonk, 1993). Such findings are consistent with a large body of research indicating a more general tendency for negative information about others to receive more processing and have a greater impact than positive information (e.g., Peeters & Czapinski, 1990; Reeder & Coovert, 1986).

Alcohol also could influence inconsistency resolution through its effects on the processing of valence information. Findings related to alcohol's effects on valence processing have been somewhat mixed. On the one hand, a number of studies have indicated that automatic emotional responses to affective images, as indexed by physiological measures such as startle eye blink magnitude (e.g., Stritzke, Patrick, & Lang, 1995) and the amplitude of facial electromyogram (EMG) responses (Glautier, O'Brien, & Dixon, 2001; Stritzke et al., 1995) are not moderated by alcohol consumption. On the other hand, some studies have suggested that alcohol leads to a positivity bias in information processing. For instance, Bruce, Shestowsky, Mayerovitch, and Pihl (1999) asked participants to learn a group of depressing and elating statements prior to consuming either placebo or a dose of alcohol. In a subsequent recall test, those in the placebo group recalled more depressing than elating statements, whereas those in the alcohol group recalled more elating than depressing statements. These researchers attributed this finding to alcohol's effects on incentive-reward systems (see also Ingvar et al., 1998) and argued that alcohol enhances memory consolidation for reward-congruent information (see also White, 1996).

The apparent discrepancy in these findings may be attributable to researchers examining alcohol's effects at different levels of processing. Cognitive theories (e.g., Steele & Josephs, 1990) posit that controlled, effortful processes (e.g., integrating new information with stored knowledge) are affected by alcohol consumption, whereas automatic aspects of cognition (e.g., initial trait inferences) are left unaffected (see also Herzog, 1999). Studies in which alcohol has been shown to have no moderating effect on reactions to valenced images (Glautier et al., 2001; Stritzke et al., 1995) have been consistent with the notion that relatively automatic valenced reactions are unaffected by alcohol, in that measures such as startle and EMG responses are thought to index automatic processes (see Hugdahl, 1995). Studies showing alcohol-related moderation of the valence dimension (e.g., Bruce et al., 1999) arguably have assessed more controlled and effortful processes involving maintenance of information in memory. The inconsistency resolution process entails both early and relatively

automatic components (e.g., direction of attention to novel or negative information) as well as later occurring controlled processing components (e.g., comparison of new information with previously formed evaluative concepts). The theory and research we have reviewed has suggested that alcohol may have no effect on early components, whereas more effortful components are likely to be affected. Previous research examining inconsistency resolution (Bartholow et al., 2001) has indicated that processing of valence information generally takes place relatively late in the processing stream, with negative behaviors receiving more processing than positive behaviors (see also Ito, Larsen, Smith, & Cacioppo, 1998). Therefore, alcohol could disrupt valence processing during later stages of the inconsistency resolution process in the current study. However, alcohol should have no effect on valence processing in early components.

The Present Research

The research presented here was designed to directly examine questions concerning alcohol's effects on inconsistency resolution in person perception, using both recall and event-related brain potential (ERP) measures of cognitive processing. Briefly, ERPs are aspects of the electrical activity of the brain elicited by the presentation of a specific stimulus and are regarded as manifestations of information-processing activities (see Fabiani, Gratton, & Coles, 2000). In general, variations in the amplitude of ERP components (i.e., peaks and troughs in the waveform and the latency at which they characteristically occur) represent variations in the engagement of information-processing operations, whereas variations in the latency of particular components indicate the speed with which particular aspects of processing are carried out (e.g., Fabiani et al., 2000; Rugg & Coles, 1995). These characteristics make ERPs an excellent measure for examining specific aspects of the inconsistency-resolution process, providing a relatively direct indication of both the level of engagement of specific processing components and the time course of this processing. Thus, measurement of ERPs in conjunction with recall provides a number of advantages over using recall alone to infer the extent of cognitive processing in studies like this.

Two classes of ERP components are of particular interest in the current study. First, the N100 component, a negative deflection peaking between 100 ms and 200 ms poststimulus, is thought to be related to early, automatic attentional processes (e.g., Hugdahl, 1995). N100 amplitude tends to increase according to the amount of attention implicitly directed at an external stimulus (see Fabiani et al., 2000). As such, the N100 can serve as an indication of automatic aspects of the inconsistency resolution process, such as the direction of attention to novel behavior. Theoretically, perceivers should be especially attuned to novelty in the behavior of others at early stages of processing, because this sensitivity would allow for appropriate action sequences to be set in place very quickly. Similarly, negative (as opposed to positive) behaviors theoretically should capture more attention early in processing to the extent that they index a potential threat to the organism (see Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001).

A second type of ERP component—specifically, the P300 or late positive potential (LPP)—is of particular interest for examining processes related to inconsistency resolution. The LPP is a positive-going deflection in the ERP waveform typically peaking between 300 ms and 600 ms following stimulus onset and is typically largest over central-parietal areas of the scalp. The amplitude of the LPP generally increases as a function of the amount of discrepancy between a given stimulus and a preceding context (see Fabiani et al., 2000) and correlates with later recall of information (e.g., Fabiani & Donchin, 1995; Paller, Kutas, & Mayes, 1987). Stimulus events that activate DLPFC and parietal cortex during working memory tasks as seen with brain imaging also elicit enlarged LPP amplitudes (McCarthy, Luby, Gore, & Goldman-Rakic, 1997). Such findings have led to the view that the LPP reflects online updating of working memory (e.g., Bartholow et al., 2001; Donchin, 1981; Donchin & Coles, 1988; Fabiani & Donchin, 1995; Paller et al., 1987). LPP amplitude also is known to vary as a function of stimulus valence (Bartholow et al., 2001; Ito et al., 1998) and has been shown in previous research to reflect processing associated with expectancy violations (Bartholow et al., 2001; Osterhout, Bersick, & McLaughlin, 1997). Finally, a number of studies have shown that alcohol attenuates the amplitude of the LPP and increases its latency (see Jääskeläinen et al., 1996; Porjesz & Begleiter, 1996), further suggesting that alcohol impairs working memory function.

ERPs were used in one previous study (Bartholow et al., 2001) to track the neural activity associated with inconsistency resolution and to examine how this activity relates to later recall. In that study, ERPs were recorded while participants read behavioral statements that either confirmed or violated previously established target-based expectancies (i.e., person impressions). Recall of these behaviors was assessed later. The results indicated that relative to EC behaviors, expectancy violations elicited larger LPP responses and were more likely to be recalled. This same paradigm was used in the current study, except that participants consumed either alcohol or a placebo prior to the impression-formation task.

Hypotheses

Our review of the literature suggested two primary hypotheses. First, it was predicted that unexpected behavior would increase the amplitude of the N100 component of the ERP, indicating early direction of attention to novelty. To the extent that this processing occurs relatively automatically, it should be robust to the effects of alcohol. Second, to the extent that alcohol leads to impairment of controlled, effortful processes but not to more automatic processes (e.g., Steele & Josephs, 1990), those aspects of the inconsistency resolution process involving working memory function should be affected by alcohol. Specifically, participants who consume alcohol should show diminished inconsistency resolution following expectancy violations resulting in attenuated LPP amplitudes and a lack (or perhaps a reversal) of the recall advantage typically seen for EV behaviors, relative to the placebo condition. This distinction also suggests that the processing of valence information might differ according to dose but only to the extent that this processing occurs at later, more controlled stages.

Method

Participants

Newspaper advertisements and word of mouth were used to recruit individuals for a study of the effects of alcohol on cognition. Potential participants were interviewed via telephone and asked a number of questions concerning their medical history and general health in addition to questions specifically related to their history of substance use and abuse. Individuals who indicated any major medical conditions (including pregnancy) that contraindicate alcohol administration were disqualified from the study, as were individuals with any history of substance abuse treatment. In addition, to ensure that the alcohol dose received in the study would be within participants' normal range of experience, naive drinkers (i.e., individuals reporting an average of less than 2 drinks per week) and very heavy drinkers (individuals reporting an average of 25 or more drinks per week) were excluded from the study sample. The sample used for this study included 39 young adults (21 women) ages 21–30 years, who were paid \$8.00 per hour for their participation.

Participants deemed eligible following the telephone interview were required to adhere to a preexperimental protocol that included refraining from any alcohol or drug use for 24 hr prior to their appointment, eating a light meal 4–6 hr prior to their appointment, and refraining from strenuous physical exercise within 3 hr of their appointment. Compliance with these restrictions was assured via signed affidavits completed on participants' arrival at the lab. Additional affidavits were used to recheck participants' general health, drinking habits, and absence of major medical conditions. No participants were disqualified for failure to comply with preexperimental protocol or discrepancies between interview items and signed affidavits. In addition, female participants were required to take a hormonal pregnancy test in the lab prior to the experiment to verify that they were not pregnant (no positive test results occurred).

Stimuli

The stimuli used in this study are identical to those used and described by Bartholow et al. (2001). They are briefly reviewed here.

Establishing expectancies. Participants read 20 randomly ordered impression paragraphs, each describing an individual target person, displayed via computer for 30 s each. The paragraphs described the targets' general behavior in such a way as to lead to a strong trait inference (e.g., "always opens the door for strangers"). Ten of the target individuals were described with positive traits and 10 with negative traits. The sex of the targets was conveyed via masculine and feminine pronouns (half of the targets were depicted as female). All paragraphs were rated by an initial pretest sample (N = 28) to ensure that they conveyed the intended trait inference and that trait inferences could be made easily. To determine whether initial expectancies differed as a function of alcohol, we asked another small sample of participants (N = 9) from a separate alcohol administration study, all of whom received the same dose of alcohol used here for the high-dose group, to rate the paragraphs using the same scales as in the initial pretest. Comparison of these two samples indicated no differences (Fs < 1). That is, positive targets were rated equally positively, negative targets equally negatively, and trait inferences were similarly easy, regardless of alcohol.

Presenting specific target behaviors. Individual target behaviors were described via sentences (all six words in length) presented one word at a time in the center of the computer monitor. Words were presented at a rate of 1 every 350 ms and were displayed for 300 ms (see Bartholow et al., 2001; Osterhout et al., 1997). The final word of each sentence determined whether it described an EC behavior, an EV behavior, or an expectancy-irrelevant behavior and whether the sentence was semantically incongruent. Twelve sentences (trials) were presented for each target person. Of these, the first four were filler trials and always ended with an EC behavior. The remaining eight trials consisted of two each of EC, EV, expectancy-irrelevant, and semantically incongruent sentences, the order of which was randomized within each block of trials.¹

Electrophysiological Recording

The electroencephalogram (EEG) data included in this report were recorded from frontal, central, and parietal midline scalp locations (Fz, Cz, and Pz, respectively), referenced to linked mastoids, using an electrode cap (Electrocap International, Easton, OH).² Vertical and horizontal electrooculograms (EOGs) were recorded bipolarly using Ag/AgCl electrodes placed above and below the right eye and 2 cm external to the outer canthus of each eye, respectively. Ocular artifacts were corrected off-line using a standard procedure (Gratton, Coles, & Donchin, 1983). The EEG and EOG were recorded continuously for the duration of each sentence, including a 100-ms prestimulus baseline prior to the presentation of the final word, at a digitizing rate of 100 Hz. The recording continued for an additional 1,000 ms after the presentation of the last (critical) word in each sentence. Impedance was kept below 10 k Ω . Signals were amplified using Grass amplifiers (Astro-Med, Inc., West Warwick, RI), and a 0.01–30-Hz bandpass was used for the EEG and EOG recording. After artifact removal and rejection, EEG data were averaged off-line according to participant, electrode, and stimulus conditions.

Beverage Administration

Participants were randomly assigned to receive a high dose (0.80 g/kg ethanol for men, 0.72 g/kg ethanol for women), moderate dose (0.40 g/kg ethanol for men, 0.36 g/kg ethanol for women), or placebo (0.04 g/kg ethanol) vodka (Smirnoff 100-proof) and tonic (Schweppes) beverage. Seven men and 6 women were assigned to each dose group. All participants were given the moderate-dose expectancy to reduce the discrepancy between actual and expected doses as much as possible across conditions, thereby enhancing the viability of our cover story (see Sher & Walitzer, 1986). In all three conditions, the experimenter ostensibly mixed a beverage containing a moderate dose of alcohol mixed in a 5:1 tonic-to-vodka ratio. The placebo dose was achieved by using diluted vodka (nine parts flattened tonic to one part 100-proof vodka mixed in a Smirnoff vodka bottle), and the high dose was achieved by using spiked tonic (four parts tonic to one part 100-proof Smirnoff vodka mixed in a tonic bottle). Total beverage was isovolemic across beverage conditions. Collars were used to indicate the actual contents of each bottle (e.g., "Regular tonic," "Spiked tonic," etc.), and the lead experimenter removed these collars before the bottles were brought to the second experimenter. Thus, the second experimenter, who mixed and served the beverage, was unaware of the actual contents of the beverage bottles. The beverage was divided into three equal-size drinks that were given to the participant one at a time. Participants were allowed 5 min to consume each of the three drinks. To improve the taste, lime juice was added according to each participant's preference.

Measurement of Blood Alcohol Concentration (BAC) Levels

BAC was measured throughout the experimental session using an Alco-Sensor IV Breathalyzer (Intoximeters, Inc., St. Louis, MO). Participants were not informed of their actual BAC level during the experimental task.

¹ Although participants read expectancy-irrelevant and semantically incongruent behavioral sentences as well, the current report focuses only on responses to expectancy-relevant behaviors (i.e., EC and EV), and thus data related to expectancy-irrelevant and semantically incongruent behaviors are not presented. Interested readers are referred to an earlier report using this paradigm (Bartholow et al., 2001), in which the comparison of semantic violations and expectancy violations in person perception is discussed in some detail.

² The EEG also was recorded from additional, lateral scalp locations. However, the components of interest in this study (N100 and LPP) are known to be largest at midline locations (see Fabiani et al., 2000), and hypotheses related to the current report focus on overall effects and not on whether or not any effects differ across left and right regions of the scalp. Therefore, only data recorded from midline locations are included here.

To eliminate residual alcohol in the mouth, participants rinsed their mouths with water prior to the first postdrinking BAC measurement. A new disposable mouthpiece was used for each sample taken during a lab session.

Subjective Intoxication Measures

In addition to BAC measurement, we included two measures of participants' subjective experience of intoxication. First, we administered the Biphasic Alcohol Effects Scale (BAES; Martin, Earleywine, Musty, Perrine, & Swift, 1993) at each BAC assessment. The BAES is a self-report measure designed to assess a drinker's experience of the stimulant and sedative effects of alcohol. Participants use a 10-point scale to rate the extent to which they are experiencing seven states associated with stimulation (e.g., elated, excited, stimulated) and seven states associated with sedation (e.g., down, sluggish, sedated). Typically, during the period just after consumption up until BAC reaches its peak (i.e., the *ascending limb*), participants report higher stimulant effects. However, following the peak of the BAC curve (*descending limb*), participants generally report more sedative effects (see Martin et al., 1993).

We also included a short questionnaire at the conclusion of the session designed to assess participants' subjective intoxication level during the study. Five questions asked participants to rate how intoxicated they felt throughout different phases of the experimental task ("right now," "while drinking," "just after drinking," "during the first half of the trials," "during the second half of the trials"). Responses ranged from 0 (*Not at all*) to 4 (*A lot*). Participants also estimated the number of standard alcohol drinks they believed they consumed using a 0-20 scale.

Procedure

On participants' arrival at the lab, an experimenter verified their age by examining two forms of identification and measured their weight using a standard physician's scale. Participants then were seated in an adjacent room where they read and signed the informed consent form and affidavits along with some questionnaire measures not reported here. On completion of these measures, an experimenter read participants the instructions for the experimental task and explained the beverage administration and electrophysiological recording procedures. Participants then were asked to use the restroom in order to void the bladder prior to beverage administration.

Next, participants were led to the experiment room for electrode placement, following which they were seated in the sound-attenuated recording booth in front of a computer monitor. Participants were informed that they would be reading paragraph descriptions of individuals presented on the monitor and were to form impressions of them. They were further told that following each paragraph, sentences depicting the individuals' behavior would be presented one word at a time and that they should read each sentence silently, keeping their initial impression in mind while doing so. Finally, participants were told that they would be given a recall test at the end of the experiment. To familiarize participants with the task prior to beverage consumption, they were shown an impression paragraph and behavioral sentences similar to those used in the actual experimental trials. Following this practice task, an experimenter took a baseline intoxication measurement while a second experimenter measured the appropriate amount of each beverage and mixed the drink in a large pitcher. On completion of the third and final drink, participants sat idle for a 20-min "absorption" period. Following the absorption period, a second intoxication measurement was taken just before participants completed the first half of the experimental trials (10 impression paragraphs and accompanying behavioral sentences), after which a third intoxication measurement was taken. Participants then viewed the paragraphs and sentences for the remaining 10 individuals, after which a fourth intoxication measurement was taken. Following the fourth intoxication measurement, participants were given a sentence-completion task that consisted of a random presentation of each of the 240 behavioral sentences used in the study, each missing the final word. Participants were asked to complete each sentence according to the way it had appeared earlier (no time limit was imposed).

Electrodes were then removed and participants were led to another nearby room to complete the postexperimental questionnaire items, including some items intended to probe for suspicion (none was revealed), following which participants were debriefed about the true nature of the study. Participants in the high-dose condition were retained in the lab until a breathalyzer test indicated that their BAC was 0.04% or less.³ These participants were given snacks and water during this time and were encouraged to consume them. All participants, regardless of beverage condition, were driven home after the session by a friend or by taxi provided by the experimenters.

Results

Manipulation Checks

Alcohol dose. BAC levels attained during the experimental task (i.e., not including baseline) by the moderate- and high-dose participants were analyzed using a 2 (dose) \times 3 (assessment time) analysis of variance (ANOVA), with repeated measures on the latter factor.⁴ This analysis showed that participants in the high-dose condition experienced significantly higher BAC levels during the task (M = 0.085%, SD = 0.01) than those in the moderate-dose condition (M = 0.045%, SD = 0.01), F(1, 24) = 107.03, p < .001. Importantly, the assessment time main effect and Dose \times Time interaction were both nonsignificant, indicating that dose levels generally remained stable throughout the task.

Subjective intoxication. BAES Stimulation and Sedation subscale scores were calculated for each participant by summing their ratings of the respective subscale items at each postconsumption assessment period (pretask, midtask, and posttask). These scores were examined using a 3 (dose) \times 2 (subscale) \times 3 (assessment time) mixed ANOVA. The predicted Dose \times Subscale interaction was significant, F(2, 36) = 3.40, p < .05. Inspection of the means indicated that those in the alcohol groups reported higher stimulation (M = 39.2) and lower sedation (M = 19.5) effects compared with those in the placebo group (Ms = 31.3 and 22.1, respectively).

Participants' posttask ratings of how intoxicated they felt over the course of the study were averaged to create a subjective intoxication index ($\alpha = .86$). Scores on this measure were analyzed using a one-way ANOVA, with alcohol dose as the predictor. Not surprisingly, ratings of subjective intoxication increased as a function of alcohol dose, F(2, 33) = 30.08, p < .001(Ms = 0.55, 1.43, and 2.18; SDs = 0.50, 0.54, and 0.63 for placebo, moderate, and high dose, respectively). Planned comparisons indicated that these means all differed from each other (ps <.05). A similar ANOVA was used to examine participants' estimates of the number of standard drinks they consumed during the

³ Because of a change in the procedure that took effect during the final month of data collection for the current study, the last several participants in the high- and moderate-dose groups were retained in the lab until their BAC was at or below 0.02%.

⁴ Placebo group means were not included in this analysis because there was zero variability in these values (i.e., all BAC values were 0.00% in the placebo group). As such, inclusion of these data would violate assumptions of homogeneity of variance in the ANOVA.

study, which also differed according to dose group, F(2, 33) = 13.41, p < .001. Post hoc comparisons indicated that participants in the placebo group believed they had consumed significantly fewer drinks (M = 1.64) than those in the moderate-(M = 3.46) and high-dose groups (M = 4.20, p < .001), but that the moderate- and high-dose group estimates did not differ significantly (p > .20). The fact that those in the placebo group believed, on average, that they had consumed between one and two standard drinks suggests that our cover story (that participants were all receiving a moderate dose of alcohol) was viable.

ERP Data

Analytic approach. ERP data from 5 participants (2 high dose, 1 moderate dose, 2 placebo) were not usable because of a high proportion of artifacts. Thus, analyses of the ERP data were based on 34 participants. Visual inspection of the waveforms obtained from the participants used in the analyses revealed a deflection with latency consistent with the N100 component. Preliminary analyses indicated that the latency of the N100 was approximately 110 ms. Hence, the amplitude of the N100 was quantified as the mean negative-going activity between 50 ms and 150 ms poststimulus for each participant within each of the experimental conditions. The waveforms also showed later positive deflections consistent with the LPP component. Initial inspection of the waveforms indicated that alcohol affected the latency of the LPP, consistent with previous research (see Jääskeläinen et al., 1996). An ANOVA confirmed that the peak of the LPP was significantly delayed in the moderate- (M = 795 ms) and highdose (M = 834 ms) conditions, relative to placebo (M = 650 ms), F(2, 31) = 5.76, p < .01. Follow-up contrasts indicated that the moderate- and high-dose group means did not differ (F < 2), but the placebo group mean differed from the other two, Fs(1,31) = 5.44 and 10.95, respectively, ps < .05. On the basis of this finding, we constructed separate epochs for examining the effects of our manipulations on LPP activity in the placebo and alcohol groups. The placebo group LPP was defined as the average amplitude 500-700 ms poststimulus, and the LPP for those in the alcohol conditions was defined as the average amplitude 700-900 ms poststimulus. Preliminary analyses of LPP amplitudes in these epochs indicated that the LPP was largest at Pz. Therefore, and consistent with prior research, we defined the LPP as the largest positive amplitude at Pz within the epochs just described, and our analyses of the LPP were restricted to the Pz electrode. Figure 1 depicts ERP waveforms as a function of dose, valence of expectancy (context), and consistency of behavior with expectancies.

Early attention component (N100). Recall that N100 amplitudes generally are thought to reflect automatic allocation of attention to external stimuli. To test whether alcohol differentially influenced attention to behaviors as a function of valence or consistency, ERP amplitudes associated with the N100 component were examined using a 3 (dose: placebo, moderate, high) \times 2 (consistency: EC, EV) \times 2 (behavior valence: positive, nega-

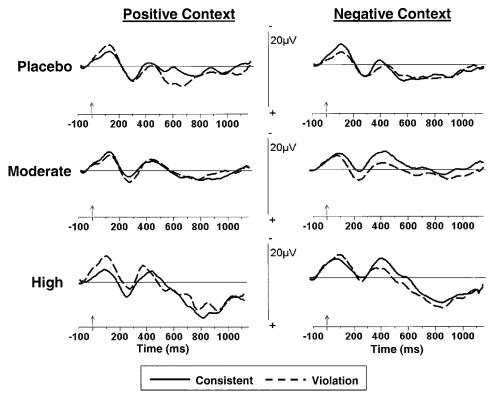


Figure 1. Event-related brain potential waveforms measured at the Pz (midline parietal) electrode site as a function of alcohol dose (rows), expectancy context (columns), and consistency with expectancies (solid vs. dashed lines). The arrow on the timeline represents stimulus onset time.

tive) × 3 (electrode site: Fz, Cz, Pz) ANOVA, with repeated measures on all but the first factor. The analysis showed a significant main effect of valence, F(1, 31) = 5.28, p < .05. As shown in Figure 2, N100 amplitudes were larger in response to negative behaviors ($M = -5.21 \ \mu$ V) than to positive behaviors ($M = -4.21 \ \mu$ V). This effect did not differ significantly across dose conditions, F(2, 31) = 2.19, p > .10. The analysis also revealed a marginal Dose × Consistency interaction, F(2, 31) = 3.14, p < .07. EV behaviors elicited a somewhat larger N100 than EC behaviors, but only in the high-dose group, t(31) = 2.53, p < .01. Overall, the N100 was marginally larger at Cz ($M = -4.97 \ \mu$ V) than at Fz ($M = -4.63 \ \mu$ V) or Pz ($M = -4.43 \ \mu$ V), but these differences were not significant, F(2, 62) = 2.24, p > .10.

Late positivity (LPP). As reviewed previously, the inconsistency resolution process is thought to differ as a function of the valence of initial expectancies (see Ybarra, 2002). In addition, studies using the LPP as an index of evaluative categorization generally have described findings in terms of responses to stimuli presented within a valenced context (see, e.g., Ito et al., 1998). Therefore, we examined the LPP data from this study using separate 2 (valence of context: positive expectancies, negative expectancies) \times 2 (consistency: EC, EV) repeated-measures ANOVAs. The first ANOVA focused on the 500–700-ms epoch (LPP for the placebo group), and the second focused on the 700–900-ms epoch (LPP for the alcohol groups) and so included an additional two-level factor for dose (moderate vs. high).

On the basis of the findings of Bartholow et al. (2001), we predicted that participants in the placebo group would show enhanced LPP activity to EVs, particularly when those behaviors

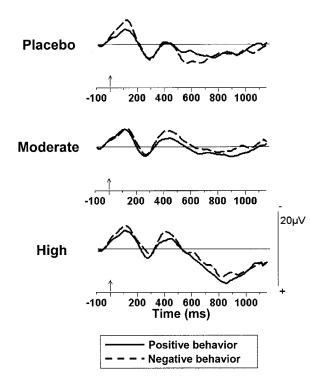


Figure 2. Effects of behavior valence on event-related brain potential waveforms as a function of alcohol dose. The arrow on the timeline represents stimulus onset time.

were negative. The ANOVA examining placebo group means (500-700 ms) showed a marginal main effect of consistency, F(1, 10) = 4.55, p < .06, indicating larger LPP amplitude to EV behavior ($M = 4.98 \mu$ V) than to EC behavior ($M = 3.01 \mu$ V). This effect was qualified by a predicted Valence of Context × Consistency interaction, F(1, 10) = 9.20, p < .01. As illustrated in the upper panel of Figure 1, for placebo group participants, the EV behaviors) was significant in the context of positive expectancies (and thus EV behaviors, respectively), t(10) = 2.94, p < .01 (one-tailed), but not in the context of negative expectancies (when EV behaviors were positive; Ms = 3.40 and 4.70 μ V, respectively), t(10) = 1.35, p > .10 (one-tailed).

For the alcohol groups, it was predicted that if alcohol broadly disrupts executive working memory processes, LPP amplitudes to EC and EV behavior should not differ. The ANOVA examining LPP amplitudes in the alcohol groups (700-900 ms) showed no main effect of consistency (F < 1), but the Valence of Context \times Consistency interaction was significant, F(1, 21) = 4.37, p < .05. The form of this interaction was precisely the opposite of that seen in the placebo group, however (see Figure 1). Simple effects tests showed that the EV effect was significant in the context of negative expectancies (when EV behaviors were positive; Ms = 3.42and 5.57 μ V for EC and EV behaviors, respectively), t(22) = 2.01, p < .05 (one-tailed), but not in the context of positive expectancies $(Ms = 5.43 \text{ and } 3.53 \ \mu\text{V}, \text{ respectively}), t(22) = 1.48, p > .05$ (one-tailed). In fact, this pattern of means suggests that if anything, consistent (positive) behaviors elicited larger LPP amplitude in the positive expectancy context for those in the alcohol groups. This pattern can be seen in Figure 2, where waveforms are collapsed across consistency conditions to illustrate the effects of behavior valence.5

This analysis also showed a main effect of dose, F(1, 21) = 12.67, p < .01. Inspection of the means indicated that the LPP was larger among those in the high-dose group ($M = 7.21 \mu$ V) than those in the moderate-dose group ($M = 1.89 \mu$ V). However, dose did not significantly interact with any other factors in this analysis (Fs < 1), indicating that the pattern of EV effects was similar in both dose groups.

Recall Data

Two independent raters, both unaware of the dose conditions to which participants were assigned, coded participants' cued recall responses for accuracy. Sentences completed with the correct word or synonym were coded as accurate. Interrater agreement was good ($\alpha = .90$), and discrepancies were reconciled through discussion between the coders and Bruce D. Bartholow. Separate proportions were calculated for each condition. Figure 3 displays the mean proportions of words recalled as a function of sentence type and alcohol dose condition.

Recall of behaviors was examined using a 3 (dose) \times 2 (consistency) \times 2 (behavior valence) mixed ANOVA with repeated

⁵ The waveforms presented in Figure 2 can be analyzed using valence of behavior as a factor. When the data are structured in this way, the Valence of Context \times Consistency interaction reported here is represented by a valence main effect, with the same *F* value and degrees of freedom.

Figure 3. Proportion of behaviors correctly recalled as a function of behavior valence, consistency with expectancies, and alcohol dose group. The valence of expectancy-violating (EV) behaviors was opposite that of the impression formed about the individual performing them (e.g., positive EV = negative target individuals' positive behaviors, and vice-versa for negative EV). EC = expectancy-consistent behaviors.

measures on the latter factors. This analysis showed a predicted main effect of consistency, F(1, 37) = 58.16, p < .001, wherein EV behaviors (M = 0.263) were recalled better than EC behaviors (M = 0.181). This main effect was qualified by a significant Dose × Consistency × Behavior Valence interaction, F(2, 37) = 5.33, p < .01. Simple effects tests showed that across dose groups, recall of EC behaviors did not differ as a function of valence (ps > .10). Recall of EV behaviors, on the other hand, differed as a function of valence and dose. Whereas negative EV behaviors were recalled better than positive EV behaviors by those in the placebo group, t(37) = 2.19, p < .05, those in the two alcohol groups recalled more positive EV behaviors than negative EV behaviors. This pattern was significant for those in the high-dose group, t(37) = 2.39, p < .05.

Discussion

Overview of Current Findings

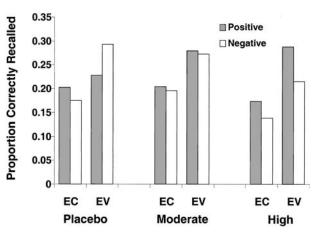
Our review of the literature related to alcohol's effects on cognitive function suggested that alcohol would likely affect more controlled, effortful aspects of the inconsistency resolution process while leaving more automatic aspects relatively unaffected. In general, this hypothesis was supported. Alcohol did not generally affect the amplitude of the N100 component, which served as an index of early and relatively automatic direction of attention to behavioral information. Instead, alcohol's effects were primarily limited to the later, presumably more effortful processing stages associated with working memory updating. In the sections that follow, we examine the major findings in detail.

Despite an apparent lack of alcohol effects at early stages of processing, our specific hypothesis concerning the N100 component generally did not receive support. We predicted that participants would be sensitive to novelty at early processing stages and so expected N100 amplitude to increase in response to EV behavior. Instead, this early component was larger for negative than

positive behaviors overall, suggesting that at early processing stages, person perception is dominated more by processing the valenced implications of others' behavior. Although previous research did not show valence processing at this early stage (Bartholow et al., 2001), these findings generally are consistent with the large literature on valence processing in person perception showing that negative information about others receives more processing than positive information (e.g., Peeters & Czapinski, 1990; Ybarra, 2002). This negativity bias (e.g., Cacioppo, Gardner, & Berntson, 1997; Ito et al., 1998) in social information processing has important adaptive significance, ensuring that people learn to avoid potentially dangerous people and situations (e.g., Ito et al., 1998; Peeters & Czapinski, 1990). The N100 results reported here suggest that the negativity bias in social perception is evident not only at later evaluative stages of processing (Bartholow et al., 2001; Ito et al., 1998) but also affects the early allocation of attention to valenced information about others. From an ERP perspective, this finding is novel in that researchers generally do not report effects of word meaning as early as the N100 (see Fabiani et al., 2000). However, this finding is consistent with the results of an earlier report using this paradigm (Bartholow et al., 2001), in which words depicting negative behaviors were shown to increase the amplitude of the corrugator EMG response within 150 ms of stimulus presentation.

For those in the placebo group, this bias was apparently carried forward to the somewhat later processing stage represented by the LPP. This finding is consistent with previous research using this paradigm (Bartholow et al., 2001). EV behaviors were more likely to prompt efforts at inconsistency resolution when expectancies were positive as opposed to negative for placebo group participants (see Figure 1). The recall findings also reflected this difference, in that negative expectancy violations (i.e., negative behaviors performed by positive individuals) were recalled better than positive expectancy violations. This pattern of effects is consistent with other research in person memory indicating that people tend to be less certain about positive impressions and thus more open to information that leads the initial impression to be updated (e.g., Sherman & Frost, 2000; Trafimow & Finlay, 2001; Vonk, 1993; Ybarra et al., 1999). Similarly, if new information indicates that a person is more potentially threatening than first assumed, it would be considered adaptive to update one's initial impression accordingly (Peeters, 1991).

For participants in the alcohol groups, however, the pattern of effects was rather different. As with the placebo group, participants in the alcohol groups appeared to direct early attention to processing negative behaviors. This finding is consistent with other studies showing that alcohol does not moderate automatic emotional responses to negatively valenced images (Glautier et al., 2001; Stritzke et al., 1995). However, in contrast to the placebo group, preferential processing of negative information was not apparent later in the processing stream. This finding is generally consistent with our prediction that alcohol would influence valence processing at later, more effortful processing stages. However, rather than simply eliminating the negativity bias, alcohol appeared to produce a positivity bias, evident both in LPP amplitudes and recall performance. Taken together, these findings indicate that alcohol did not simply impair working memory (e.g., Birnbaum & Parker, 1977; Lister et al., 1987; Peterson, Rothfleisch, Zelazo, & Pihl, 1990) but rather that alcohol changed the condi-



tions under which working memory updating occurred, as a function of valence.

What might account for this finding? We argue that alcohol likely influences working memory function via its effects on the cerebral reward system. A number of lines of evidence appear to support this possibility. First, although alcohol acts as a sedative at high doses (e.g., > 1 g/kg body weight) and while BAC is falling (e.g., Martin et al., 1993), at low to moderate doses such as those used here, alcohol produces stimulant effects, mediated in part by increased dopamine levels (see Fromme & D'Amico, 1999), which have been linked to feelings of euphoria and increased arousal (see, e.g., Lang, Patrick, & Stritzke, 1999; Martin et al., 1993). The analyses of BAES data in the current study support the notion that alcohol had such an effect in our participants. A large number of studies have indicated that positive affect can improve working memory and that this effect is mediated by dopamine (see Ashby, Isen, & Turken, 1999). In part, then, our results may reflect enhanced elaboration of information that is evaluatively consistent with the present internal state of the drinker and perhaps inhibition of evaluatively inconsistent information (see also Sayette, 1994). This interpretation is consistent with that offered by Bruce et al. (1999) to explain the pattern of increased recall of elating versus depressing statements following alcohol consumption in their study, and more generally with the larger literature on moodcongruency effects (see Fiedler, 2001).

However, our data suggest that the biased processing of positive information seen in the alcohol groups is dependent on involvement of working memory. Three findings from the current study support this view. First, alcohol did not appear to affect initial impression formation. The ratings provided by our pilot samples indicated that the impressions of positive targets were no more positive and impressions of negative targets were no less negative after alcohol consumption. Second, participants in the alcohol groups preferentially recalled positive behaviors that were unexpected—and thus engaged the inconsistency resolution process but did not simply recall more positive behaviors overall. Third, as already noted, alcohol did not significantly reduce negative valence effects at the earliest stages of processing measured here (i.e., N100 amplitude).

Other evidence in favor of this interpretation comes from studies examining the cortical areas involved in working memory and reward sensitivity. A number of studies have pointed to two areas of prefrontal cortex-orbitofrontal cortex (OFC) and the ventromedial prefrontal cortex (VMPFC)-as sensitive to reward (see Pochon et al., 2002; Rolls, 2000), important in determining the expectation of positive and negative outcomes (Hikosaka & Watanabe, 2000), and more generally determining the motivational significance of stimuli (e.g., Schultz, Tremblay, & Hollerman, 2000; see also Fuster, 1997). Alcohol is known to act on neurons in the OFC and VMPFC, among other areas, findings that have been used to explain the rewarding and reinforcing properties of alcohol and other drugs of abuse (e.g., London, Ernst, Grant, Bonson, & Weinstein, 2000) and alcohol's effects on positive affect (Fromme & D'Amico, 1999). Brain imaging data have indicated that these areas moderate working memory operations in the DLPFC. For example, Perlstein, Elbert, and Stenger (2002) found that DLPFC activation (and working memory performance) was increased under experimental conditions that elicited positive affect but only in tasks for which working memory demands were high. Pochon et al. (2002) similarly found evidence of a neural pathway mediated by VMPFC that responds to rewarding stimulus input during a working memory task. When considered together, these results suggest that activation of reward systems has the potential to increase working-memory-related activity, particularly for reward-congruent information. However, our data are clearly limited in terms of testing this hypothesis directly. Future research should be directed at further specifying which cortical areas or processing systems are affected by alcohol, how these systems interact with working memory processes during person perception, and how this processing changes over time (e.g., Fromme & D'Amico, 1999). It would also be informative to examine the difference in alcohol's effects in person perception as a function of ascending BAC (as was the case here) versus descending BAC. According to the logic we have presented, we would predict that the alcohol-induced positivity bias seen here would be eliminated on the descending limb of the BAC curve, when alcohol's sedative effects dominate (see Martin et al., 1993).

The differential pattern of effects as a function of alcohol seen here has important implications for social behavior. In contrast to the findings from the placebo group, those in the alcohol groups showed evidence of greater inconsistency resolution following positive EV behaviors than negative EV behaviors in terms of both LPP amplitude and recall performance. Stated another way, participants in the alcohol groups were less likely to update positive impressions with new, inconsistent information and were more likely to update negative impressions. This finding suggests that alcohol consumption might put people at risk in interpersonal situations by (a) preventing changes to positive impressions when negative behaviors indicate that doing so would be adaptive and (b) promoting changes in negative impressions such that potentially threatening people are deemed less dangerous.

The apparent inconsistency between alcohol's effects at early and later processing stages suggests biased evaluative processing as a possible mechanism for understanding the reinforcing properties of alcohol consumption. Research indicates that although most drinkers tend to experience both positive (e.g., increased sociability, elevated mood) and negative (e.g., sluggishness, nausea, hangover) effects of alcohol, positive effects are more predictive of future alcohol consumption. For example, alcohol-related expectancies—beliefs or predictions concerning the likely positive or negative effects of drinking alcohol-have been shown to significantly correlate with concurrent drinking and to predict future alcohol use (e.g., Bartholow, Sher, & Strathman, 2000; Goldman, Del Boca, & Darkes, 1999). Importantly though, negative expectancies typically account for far less variance in consumption than do positive expectancies (e.g., Leigh & Stacy, 1993; Rather & Goldman, 1994). The current data suggest that this positivity bias is not a result of motivated processes or selective forgetting of negative information but rather that it reflects insufficient processing of negative information and, to some extent, an increase in processing of positive information under the influence of alcohol. Such findings also have implications for understanding why chronic heavy drinkers repeat patterns of heavy use despite experiencing negative interpersonal consequences (e.g., Vogel-Sprott & Fillmore, 1999).

Other apparent effects of alcohol on the inconsistencyresolution process deserve comment. First, the latency of the late positivity increased as a function of alcohol. As reviewed above, the latency of ERP components reflects the time required to carry out various information-processing steps (e.g., Rugg & Coles, 1995). In general, then, it appears that alcohol increased the time required to process behavioral information, though this finding was not restricted to EV behaviors. Also, in contrast to a large number of studies showing alcohol-related reductions in LPP amplitude (e.g., Jääskeläinen et al., 1996; Porjesz & Begleiter, 1996), our data show that the high dose of alcohol increased the amplitude of the LPP. This finding might reflect the fact that the task used in the current study (interpreting behaviors) was cognitively more complex than the oddball paradigms used in much of the previous research examining acute alcohol effects on LPP amplitude.

Broader Implications

It has been suggested that examining processes at the social, cognitive, and neural levels of analysis can provide a more comprehensive understanding of social psychological phenomena than can be achieved by examining processes at one or two levels alone (Cacioppo, Berntson, & Crites, 1996; Ochsner & Lieberman, 2001). The current findings underscore the benefits of using tools and borrowing insights from the literature in cognitive neuroscience to inform understanding of person perception (see also Macrae et al., 1999). The combination of ERP and recall measures used here allowed us to disentangle early attentional processes from later elaborative processing stages and subsequent recall in inconsistency resolution, something that recall or response time measures alone cannot readily accomplish. Specifically, in the absence of ERPs, it would be more difficult to determine whether the pattern of recall results obtained here was attributable to participants in the alcohol groups simply having paid more attention early in processing to positive information. The N100 amplitude findings suggest that this was not the case; instead, the recall results reflect increased elaboration of positive information at a later processing stage.

In particular, our conclusions have implications for understanding the links between social cognition, cognitive neuroscience, and neuropsychology. Given the apparent interconnections between areas of prefrontal cortex mediating both alcohol's effects and aspects of social cognition, examination of social cognitive processes in otherwise healthy individuals temporarily impaired by alcohol provides a methodology for bridging gaps between diverse research literatures that traditionally have been somewhat segregated. We hope that other researchers will see similar benefits from this kind of research. As more studies of this kind begin to accumulate, it will become possible to compare the findings regarding effects of acute alcohol administration on social cognitive processes with results obtained from studies examining the same processes in patients with specific brain lesions. Interestingly, neuropsychological studies indicate that patients with damage to VMPFC and OFC display the same kinds of disinhibited, socially inappropriate behavioral patterns and insensitivity to future consequences that often are associated with intoxication (e.g., Damasio, 1994), although their working memory remains intact. Rolls (1999) also noted that such patients have difficulty properly recognizing negative but not positive emotional facial expressions. Comparing findings from such studies with those of alcohol challenge studies should improve understanding of both the cortical and subcortical foundations of social cognition and the ways in which brain impairment influences these processes.

References

- Ashby, F. G., Isen, A. M., & Turken, A. U. (1999). A neuropsychological theory of positive affect and its influence on cognition. *Psychological Review*, 106, 529–550.
- Baddeley, A. (1986). Working memory. Oxford, England: Oxford University Press.
- Baddeley, A. (1992, January 31). Working memory. *Science*, 255, 556–559.
- Bartholow, B. D., Fabiani, M., Gratton, G., & Bettencourt, B. A. (2001). A psychophysiological examination of cognitive processing of and affective responses to social expectancy violations. *Psychological Science*, 12, 197–204.
- Bartholow, B. D., Sher, K. J., & Strathman, A. (2000). Moderation of the expectancy–alcohol use relation by private self-consciousness: Data from a longitudinal study. *Personality and Social Psychology Bulletin*, 26, 1409–1420.
- Baumeister, R. F., Bratslavsky, E., Finkenauer, C., & Vohs, K. D. (2001). Bad is stronger than good. *Review of General Psychology*, 5, 323–370.
- Birnbaum, I. M., Johnson, M. K., Hartley, J. T., & Taylor, T. H. (1980). Alcohol and elaborative schemas for sentences. *Journal of Experimental Psychology: Human Learning and Memory*, 6, 293–300.
- Birnbaum, I. M., & Parker, E. S. (1977). Acute effects of alcohol on storage and retrieval. In I. M. Birnbaum & E. S. Parker (Eds.), *Alcohol* and human memory (pp. 99–108). Hillsdale, NJ: Erlbaum.
- Bruce, K. R., Shestowsky, J. S., Mayerovitch, J. I., & Pihl, R. O. (1999). Motivational effects of alcohol on memory consolidation and heart rate in social drinkers. *Alcoholism: Clinical and Experimental Research*, 23, 693–701.
- Cacioppo, J. T., Berntson, G. G., & Crites, S. L., Jr. (1996). Social neuroscience: Principles of psychophysiological arousal and response. In E. T. Higgins & A. W. Kruglanski (Eds.), *Social psychology: Handbook* of basic principles (pp. 72–101). New York: Guilford Press.
- Cacioppo, J. T., Gardner, W. L., & Berntson, G. G. (1997). The affect system has parallel and integrative processing components: Form follows function. *Journal of Personality and Social Psychology*, 76, 839– 855.
- Cohen, J. D., Forman, S. D., Braver, T. S., Casey, B. J., Servan-Schreiber, D., & Noll, D. C. (1994). Activation of prefrontal cortex in a non-spatial working memory task with functional MRI. *Human Brain Mapping*, 1, 293–304.
- Cooper, M. L. (1992). Alcohol and increased behavioral risk for AIDS. Alcohol Health and Research World, 16, 64–72.
- Craik, F. M. I. (1977). Similarities between the effects of ageing and alcoholic intoxication on memory performance, construed within a "levels of processing" framework. In I. M. Birnbaum & E. S. Parker (Eds.), *Alcohol and human memory* (pp. 9–21). Hillsdale, NJ: Erlbaum.
- Damasio, A. R. (1994). Descartes' error: Emotion, reason, and the human brain. New York: Grosset/Putnam.
- Dijksterhuis, A., & van Knippenberg, A. (1995). Memory for stereotypeconsistent and stereotype-inconsistent information as a function of processing pace. *European Journal of Experimental Social Psychology*, 25, 689–693.
- Donchin, E. (1981). Surprise! ... Surprise? *Psychophysiology*, 18, 493–513.
- Donchin, E., & Coles, M. G. H. (1988). Is the P300 component a manifestation of context updating? *Behavioral and Brain Sciences*, 11, 355– 372.
- Fabiani, M., & Donchin, E. (1995). Encoding processes and memory organization: A model of the von Restorff effect. *Journal of Experimen*tal Psychology: Learning, Memory, and Cognition, 21, 224–240.

- Fabiani, M., Gratton, G., & Coles, M. G. H. (2000). Event-related brain potentials: Methods, theory, and applications. In J. T. Cacioppo, L. Tassinary, & G. Berntson (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 53–84). New York: Cambridge University Press.
- Fiedler, K. (2001). Affective influences on social information processing. In J. P. Forgas (Ed.), *Handbook of affect and social cognition* (pp. 163–185). Mahwah, NJ: Erlbaum.
- Fillmore, M. T., Carscadden, J. L., & Vogel-Sprott, M. (1998). Alcohol, cognitive impairment, and expectancies. *Journal of Studies on Alcohol*, 59, 174–179.
- Finn, P. R., Justus, A., Mazas, C., & Steinmetz, J. E. (1999). Working memory, executive processes and the effects of alcohol on go/no-go learning: Testing a model of behavioral regulation and impulsivity. *Psychopharmacology*, 146, 465–472.
- Fromme, K., & D'Amico, E. J. (1999). Neurobiological bases of alcohol's psychological effects. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 422–455). New York: Guilford Press.
- Fuster, J. M. (1997). The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe (3rd ed.). New York: Raven.
- Glautier, S., O'Brien, J., & Dixon J. (2001). Facial electromyographic (EMG) responses to emotionally significant visual images: Differences between light and heavy drinkers. *Drug and Alcohol Dependence*, 64, 337–345.
- Goldman, M. S., Del Boca, F. K., & Darkes, J. (1999). Alcohol expectancy theory: The application of cognitive neuroscience. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 203–246). New York: Guilford Press.
- Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, 55, 468–484.
- Hastie, R., & Kumar, P. (1979). Person memory: Personality traits as organizing principles in memory for behaviors. *Journal of Personality* and Social Psychology, 37, 25–38.
- Herzog, T. A. (1999). Effects of alcohol intoxication on social inferences. Experimental and Clinical Psychopharmacology, 7, 448–453.
- Higgins, E. T., & Bargh, J. A. (1987). Social perception and social cognition. Annual Review of Psychology, 38, 369–425.
- Hikosaka, K., & Watanabe, M. (2000). Delay activity of orbital and lateral prefrontal neurons of the monkey varying with different rewards. *Cerebral Cortex*, 10, 263–271.
- Hoaken, P. N. S., Giancola, P. R., & Pihl, R. O. (1998). Executive cognitive functions as mediators of alcohol-related aggression. *Alcohol* and *Alcoholism*, 33, 47–54.
- Hugdahl, K. (1995). Psychophysiology: The mind-body perspective. Cambridge, MA: Harvard University Press.
- Ingvar, M., Ghatan, P. H., Wirsen-Muerling, A., Risberg, J., von Heijne, G., Stone-Elander, S., & Ingvar, D. H. (1998). Alcohol activates the cerebral reward system in man. *Journal of Studies on Alcohol*, 59, 258–269.
- Ito, T. A., Larsen, J. T., Smith, N. K., & Cacioppo, J. T. (1998). Negative information weighs more heavily on the brain: The negativity bias in evaluative categorizations. *Journal of Personality and Social Psychol*ogy, 75, 887–900.
- Jääskeläinen, I. P., Näätänen, R., & Sillanaukee, P. (1996). Effect of acute ethanol on auditory and visual event-related potentials: A review and reinterpretation. *Biological Psychiatry*, 40, 284–291.
- Jones, E. E. (1990). Interpersonal perception. New York: Freeman.
- 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.
- Leigh, B. C., & Stacy, A. W. (1993). Alcohol outcome expectancies: Scale

construction and predictive utility in higher order confirmatory models. *Psychological Assessment, 5,* 216–229.

- Lister, R. G., Eckardt, M. J., & Weingartner, H. (1987). Ethanol intoxication and memory. In M. Galanter (Ed.), *Recent developments in alcoholism* (Vol. 5, pp. 111–126). New York: Plenum Press.
- London, E. D., Ernst, M., Grant, S., Bonson, K., & Weinstein, A. (2000). Orbitofrontal cortex and human drug abuse: Functional imaging. *Cerebral Cortex*, 10, 334–342.
- Lyvers, M. (2000). "Loss of control" in alcoholism and drug addiction: A neuroscientific interpretation. *Experimental and Clinical Psychophar*macology, 8, 225–249.
- MacDonald, T. K., Zanna, M. P., & Fong, G. T. (1995). Decision-making in altered states: Effects of alcohol on attitudes toward drinking and driving. *Journal of Personality and Social Psychology*, 68, 973–985.
- MacDonald, T. K., Zanna, M. P., & Fong, G. T. (1996). Why common sense goes out the window: Effects of alcohol on intentions to use condoms. *Personality and Social Psychology Bulletin*, 22, 763–775.
- Macrae, C. N., Bodenhausen, G. V., Schloersheidt, A. M., & Milne, A. B. (1999). Tales of the unexpected: Executive function and person perception. *Journal of Personality and Social Psychology*, 76, 200–213.
- Macrae, C. N., Hewstone, M., & Griffiths, R. J. (1993). Processing load and memory for stereotype-based information. *European Journal of Social Psychology*, 23, 76–87.
- Martin, C. S., Earleywine, M., Musty, R. E., Perrine, M. W., & Swift, R. M. (1993). Development and validation of the Biphasic Alcohol Effects Scale. *Alcoholism: Clinical and Experimental Research*, 17, 140–146.
- McCarthy, G., Luby, J., Gore, J., & Goldman-Rakic, P. (1997). Infrequent events transiently activate human prefrontal and parietal cortex as measured by functional MRI. *Journal of Neurophysiology*, 77, 1630–1634.
- McCarthy, G., Puce, A., Constable, R. T., Krystal, J. H., Gore, J. C., & Goldman-Rakic, P. (1996). Activation of human prefrontal cortex during spatial and object working memory tasks measured by functional MRI. *Cerebral Cortex*, 6, 600–611.
- Nelson, T. O., McSpadden, M., Fromme, K., & Marlatt, G. A. (1986). Effects of alcohol intoxication on metamemory and on retrieval from long-term memory. *Journal of Experimental Psychology: General*, 115, 247–254.
- Ochsner, K. N., & Lieberman, M. D. (2001). The emergence of social cognitive neuroscience. *American Psychologist*, 56, 717–734.
- Olson, J. M., Roese, N. J., & Zanna, M. P. (1996). Expectancies. In E. T. Higgins & A. W. Kruglanski (Eds.), *Social psychology: Handbook of basic principles* (pp. 211–238). New York: Guilford Press.
- Osterhout, L., Bersick, M., & McLaughlin, J. (1997). Brain potentials reflect violations of gender stereotypes. *Memory and Cognition*, 25, 273–285.
- Paller, K. A., Kutas, M., & Mayes, A. R. (1987). Neural correlates of encoding in an incidental learning paradigm. *Electroencephalography* and Clinical Neurophysiology, 67, 360–371.
- Peeters, G. (1991). Evaluative inference in social cognition: The roles of direct versus indirect evaluation and positive-negative asymmetry. *European Journal of Social Psychology*, 21, 131–146.
- Peeters, G., & Czapinski, J. (1990). Positive–negative asymmetry in evaluations: The distinction between affective and informational negativity. *European Review of Social Psychology*, 1, 33–60.
- Perlstein, W. M., Elbert, T., & Stenger, V. A. (2002). Dissociation in human prefrontal cortex of affective influences on working memoryrelated activity. *Proceedings of the National Academy of Sciences*, USA, 99, 1736–1741.
- Peterson, J. B., Rothfleisch, J., Zelazo, P. D., & Pihl, R. O. (1990). Acute alcohol intoxication and cognitive functioning. *Journal of Studies on Alcohol*, 51, 114–122.
- Petrides, M. (2000). The role of the mid-dorsolateral prefrontal cortex in working memory. *Experimental Brain Research*, 133, 44–54.

- Pochon, J. B., Levy, R., Fossati, P., Lehericy, S., Poline, J. B., Pillon, B., et al. (2002). The neural system that bridges reward and cognition in humans: An fMRI study. *Proceedings of the National Academy of Sciences, USA, 99*, 5669–5674.
- Porjesz, B., & Begleiter, H. (1996). Effects of alcohol on electrophysiological activity of the brain. In H. Begleiter & K. Benjamin (Eds.), Alcohol and alcoholism: No. 2. The pharmacology of alcohol and alcohol dependence (pp. 207–247). New York: Oxford University Press.
- Rather, B. C., & Goldman, M. S. (1994). Drinking-related differences in the memory organization of alcohol expectancies. *Experimental and Clinical Psychopharmacology*, 2, 167–183.
- Reeder, G. D., & Coovert, M. D. (1986). Revising an impression of morality. Social Cognition, 4, 1–17.
- Rolls, E. T. (1999). The functions of the orbitofrontal cortex. *Neurocase*, *5*, 301–312.
- Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex*, *10*, 284–294.
- Rugg, M. D., & Coles, M. G. H. (1995). Electrophysiology of mind: Event-related brain potentials and cognition. New York: Oxford University Press.
- Sayette, M. A. (1994). Effects of alcohol on self-appraisal. *International Journal of the Addictions*, 29, 127–133.
- Sayette, M. A. (1999). Cognitive theory and research. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 247–291). New York: Guilford Press.
- Schultz, W., Tremblay, L., & Hollerman, J. R. (2000). Reward processing in primate orbitofrontal cortex and basal ganglia. *Cerebral Cortex*, 10, 272–283.
- Sher, K. J., & Walitzer, K. S. (1986). Individual differences in the stressresponse-dampening effects of alcohol: A dose-response study. *Journal* of Abnormal Psychology, 95, 159–167.
- Sherman, J. W., & Frost, L. A. (2000). On the encoding of stereotyperelevant information under cognitive load. *Personality and Social Psychology Bulletin*, 26, 26–34.
- Smith, E. E., & Jonides, J. (1999, March 12). Storage and executive processes in the frontal lobes. *Science*, 283, 1657–1662.
- Srull, T. K., Lichtenstein, M., & Rothbart, M. (1985). Associative storage and retrieval processes in person memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 11*, 316–345.

- Srull, T. K., & Wyer, R. S., Jr. (1989). Person memory and judgment. *Psychological Review*, 96, 58–83.
- Stangor, C., & Duan, C. (1991). Effects of multiple task demands upon memory for information about social groups. *Journal of Experimental Social Psychology*, 27, 357–378.
- Stangor, C., & McMillan, D. (1992). Memory for expectancy-congruent and expectancy-incongruent information: A review of the social and developmental literatures. *Psychological Bulletin*, 111, 42–61.
- Steele, C. M., & Josephs, R. A. (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.
- Tracy, J. I., & Bates, M. E. (1999). The selective effects of alcohol on automatic and effortful memory processes. *Neuropsychology*, 13, 282– 290.
- Trafimow, D., & Finlay, K. A. (2001). An investigation of three models of multitrait representations. *Personality and Social Psychology Bulletin*, 27, 226–241.
- Vogel-Sprott, M., & Fillmore, M. T. (1999). Learning theory and research. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 292–327). New York: Guilford Press.
- Vonk, R. (1993). The negativity effect in trait ratings and in open-ended descriptions of persons. *Personality and Social Psychology Bulletin*, 19, 269–278.
- White, N. M. (1996). Addictive drugs as reinforcers: Multiple partial actions on memory systems. *Addiction*, 91, 921–949.
- Ybarra, O. (2002). Naive causal understanding of valenced behaviors and its implications for social information processing. *Psychological Bulletin*, 128, 421–441.
- Ybarra, O., Schaberg, L., & Keiper, S. (1999). Favorable and unfavorable target expectancies and social information processing. *Journal of Per*sonality and Social Psychology, 77, 698–709.

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