



Effects of alcohol consumption and alcohol susceptibility on cognition: a psychophysiological examination

Bruce D. Bartholow^{a,*}, Melanie Pearson^b, Kenneth J. Sher^b,
Leighann C. Wieman^b, Monica Fabiani^c,
Gabriele Gratton^c

^a *Department of Psychology, University of North Carolina at Chapel Hill and the Missouri Alcoholism Research Center, CB# 3270, Davie Hall, Chapel Hill, NC 27599, USA*

^b *Department of Psychological Sciences, University of Missouri-Columbia and the Missouri Alcoholism Research Center, USA*

^c *Department of Psychology and Beckman Institute, University of Illinois at Urbana-Champaign and the Missouri Alcoholism Research Center, 603 East Daniel Street, Champaign, IL 61820, USA*

Abstract

The present study sought to examine acute effects of alcohol on cognitive processing and performance within the context of two prominent theories of alcohol's effects; namely, that alcohol restricts the focus of attention (e.g. Steele and Josephs, 1990. *Journal of Abnormal Psychology*, 97, 196–205) and that alcohol impairs response inhibition (e.g. Fillmore and Vogel-Sprott, 1999. *Experimental and Clinical Psychopharmacology*, 7, 49–55; Fillmore and Vogel-Sprott, 2000. *Journal of Studies on Alcohol*, 61, 239–246). Forty-five participants were randomly assigned to receive either a placebo level of alcohol (0.04 g/kg), a moderate dose (0.40 g/kg), or a higher dose (0.80 g/kg). Brain electrical activity (ERPs) and behavioral responses (reaction time and accuracy) were measured while participants performed a modified flanker task, in which a target letter was flanked by response-compatible or response-incompatible letters. Analyses of behavioral data showed that alcohol increased response competition in accuracy but not response times, suggesting that alcohol influences response selection more than attentional processes per se. This finding is in-line with predictions derived from the response inhibition model. ERP latency data provided mixed support for both models. ERP amplitude data showed that the high dose of alcohol primarily influenced a mostly frontal negativity in the ERP, present on both correct and incorrect response trials. Differences in self-reported susceptibility to

* Corresponding author. Tel.: +1-919-843-5486; fax: +1-919-962-2537.

E-mail addresses: bartholow@unc.edu (B.D. Bartholow), grattong@uiuc.edu (G. Gratton).

alcohol were most evident in the amplitude of the P3 component. Findings are discussed in terms of the differential effects of acute dose and susceptibility on information processing.

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1. Introduction

Alcohol is known to impair functioning in a variety of domains including behavior, memory, and judgment (e.g. see Nelson et al., 1998; Sayette, 1999; Steele and Josephs, 1990). Although it has been assumed that these varied effects stem from alcohol's impairment of cognitive functioning, research findings have been unclear with respect to whether these effects result from a global impairment of cognition or rather some specific impairment of certain brain systems.

A number of measures related to cognitive processing have been used to examine the effects of alcohol. For example, measures of response time and response accuracy have been shown to be sensitive to acute alcohol consumption. In addition, some measures of frontal and temporal lobe functioning (e.g. Peterson et al., 1990) and working memory capacity (e.g. Finn et al., 1999; Petros, 1985) show alcohol-related deficits. These data find a counterpart in psychophysiology with the event-related brain potential (ERP), an index of cognitive processing operations. Acute alcohol intoxication has been shown to decrease the amplitude and increase the latency of several components of the ERP, most notably the P3 or P300 (e.g. Lukas et al., 1990; Wall and Ehlers, 1995; for a review see Noldy, 1998). Briefly, the P3 component has been hypothesized to index context updating during information processing (Donchin, 1981; Donchin and Coles, 1988; see also Fabiani et al., 2000). In general, such ERP data indicate that alcohol tends to reduce attention to stimuli, to slow processing and to decrease the efficiency with which people can interpret and properly utilize stimulus-relevant information.

Previous research also has shown that individuals vary in their reactions to alcohol (i.e. ethanol sensitivity). In recent years, research by Schuckit and colleagues has found that level of reactivity to alcohol significantly correlates with subjective intoxication effects and physiological changes during ethanol challenge and predicts the development of alcoholism (Schuckit et al., 1997a,b). However, it is presently unclear whether alcohol sensitivity acts additively or in interaction with acute alcohol effects to determine performance on specific tasks that index attention and cognitive processing.¹

¹ It is important to note that differences in alcohol sensitivity can result from different levels of chronic alcohol exposure, in that individuals with heavier consumption histories can become less sensitive to alcohol's effects due to the development of acquired tolerance. In the current study, we make no assumptions about the relative contributions of dispositional and acquired aspects of sensitivity, recognizing that individual differences in sensitivity represent a combination of innate and acquired characteristics. Moreover, since ethanol sensitivity has been related to constitutional variables such as personality/temperament (e.g. Sher et al., 1999), individual differences in ethanol sensitivity may reflect more fundamental individual differences in temperament and basic adaptive mechanisms.

1.1. *Theoretical models of alcohol's effects*

In the past two decades, researchers have begun to examine specific cognitive mechanisms that may underlie the effects of alcohol. Steele and his colleagues (e.g. Steele and Josephs, 1988, 1990; Steele and Southwick, 1985) proposed a model of alcohol effects that focuses on alcohol's influence on attentional processes, rather than its direct pharmacological effects on motivational systems. According to this attention-allocation model, intoxication restricts one's focus of attention to only the most salient cues in the environment, such that available cues are not fully processed (see Sayette, 1999). This model has been used to account for a diverse range of social behaviors including aggression (e.g. Graham et al., 1998; Bushman, 1997), helping behavior (Steele et al., 1985), and sexual risk-taking among adolescents (Cooper and Orcutt, 1997).

Additional evidence in favor of this theory comes from studies examining alcohol's effects on experimental tasks requiring participants to divide their attention across multiple tasks or spatial locations. For example, although alcohol generally seems to impair performance on divided-attention tasks (e.g. Lex et al., 1994; Maylor et al., 1990), performance is relatively unaffected on those tasks considered to be most important to participants (i.e. their primary task) while performance on secondary tasks is greatly impaired (e.g. Fisk and Scerbo, 1987). Also, studies in which participants are told to attend to stimuli in one modality while ignoring stimuli in a different modality (distracters) show that intoxicated participants perform somewhat better than sober participants (e.g. Erblich and Earleywine, 1995; Patel, 1988), indicating that alcohol actually may improve one's ability to screen out irrelevant information. In addition, alcohol reduces stress associated with threat cues primarily under conditions of divided attention (e.g. Curtin et al., 1998, 2001). All of these findings are consistent with the view that alcohol leads to a narrower focus of attention (or attention span).

A related model proposed by Vogel-Sprout and colleagues posits that, rather than restricting attentional focus, alcohol impairs a form of response inhibition (e.g. Fillmore and Vogel-Sprout, 1999, 2000; Vogel-Sprout, 1992; Vogel-Sprout et al., 2001). This model is based on a theory of cognitive control (Logan and Cowan, 1984) positing that behavioral activation and behavioral inhibition stem from two independent cognitive processes. According to the theory, certain stimuli or events prompt people to activate a given behavior whereas others prompt people to inhibit that behavior. For example, hearing one's favorite music at a party might prompt a person to begin dancing, whereas other cues (e.g. that no one else is dancing) should inhibit that response. Following alcohol consumption, however, this inhibition mechanism may be impaired. Direct support for this model has been provided in studies utilizing a 'go-stop' paradigm, in which participants are engaged in responding to 'go' signals while 'stop' signals occasionally inform them to inhibit the response (e.g. Fillmore and Vogel-Sprout, 1999, 2000; Mulvihill et al., 1997).

1.2. *The current study*

Although they propose somewhat different mechanisms for alcohol's effects, the attention allocation model (Steele and Josephs, 1990) and the response disinhibition model (e.g. Vogel-Sprout, 1992) are similarly informed by examinations of tasks involving attentional

control and behavioral adjustments. An informative paradigm to study these processes is the Eriksen flanker task (e.g. Eriksen and Eriksen, 1974), in which participants respond to a target letter presented among strings of other letters (noise letters) that are either the same as the target (i.e. compatible with correct response) or different from the target (i.e. incompatible with correct response). The performance impairment typically associated with incompatible trials, relative to compatible trials, is termed the *noise-compatibility effect* (Gratton et al., 1992). Gratton et al. (1992) modified this task by manipulating the probability of compatible and incompatible trials within trial blocks (thereby manipulating participants' implicit expectancies for types of trials) and found that different response strategies were used depending on expectancy. When participants expected compatible trials and thus the noise letters were predicted to facilitate correct responding, they processed and responded to the noise letters. This response strategy, termed *parallel*, provided quicker access to the correct response. However, when incompatible trials appeared during the parallel processing mode, the processing of the noise letters impaired performance. Conversely, when incompatible trials were expected, a *focused* response strategy was used, in which responses were based on the target letter while attempting to inhibit response activation to the noise letters. In general, variations in the size of the noise-compatibility effect as a function of expectancy condition are thought to index the occurrence of strategic control processes (Gratton et al., 1992).

1.2.1. Hypotheses

Based on Steele and Josephs' (1990) theory suggesting that alcohol restricts one's focus of attention, in the Eriksen flanker task, intoxicated participants could be expected to show a smaller noise-compatibility effect as the restricted focus of attention induced by alcohol should correspond with the focused strategy of response. This pattern could come about if alcohol focuses attention on task-relevant information (i.e. the target letter), and/or if response-relevant information provided by the peripheral flankers is not fully processed. If so, our analyses should yield an interaction between dose and compatibility in response time and response accuracy. Furthermore, to the extent that intoxicated participants are more focused on the target, they should be less influenced by manipulation of the probability of compatible and incompatible flanker letters than sober participants (i.e. modulation of the noise-compatibility effect should be reduced). In contrast, if alcohol impairs response inhibition (e.g. Vogel-Sprott, 1992), intoxicated participants should be less able to inhibit response activation associated with the noise letters, leading to a relatively larger noise-compatibility effect (i.e. more response competition), and more modulation of the effect by probability manipulations, relative to sober participants.

ERPs should provide additional data pertaining to the influence of alcohol on processing and performance, in at least two ways. First, under normal (i.e. non-intoxicated) processing conditions, compatible and incompatible arrays should be categorized differently, and thus elicit different P3 amplitudes. However, to the extent that alcohol restricts the focus of attention to the central target letter, intoxicated participants should show little evidence of differences between compatible and incompatible noise trials in P3 amplitude. If so, our analyses of P3 amplitude should reveal an interaction between dose and compatibility. Alternatively, if alcohol impairs response inhibition and thus processing of flankers is increased, the form of the interaction would be expected to differ such that placebo participants might

show a smaller noise-compatibility effect in P3 amplitude than those who consume alcohol. Second, the latency of the P3 component should reflect the extent to which participants are influenced by flankers. P3 latency is thought to reflect the completion of processes of stimulus evaluation and categorization (see Fabiani et al., 2000; Rugg and Coles, 1995). Gratton and colleagues (1992) showed that when compatible trials are expected, the presence of incompatible trials delays processing of the stimulus array, presumably because participants must shift from the parallel to the focused mode of processing. Thus, in the placebo condition, the latency of the P3 component should be longer following incompatible trials in the expect-compatible condition. However, if alcohol focuses attention on the target letter, P3 latencies should be similar for compatible and incompatible arrays. If so, the noise-compatibility effect should be larger among placebo participants compared with those who have consumed alcohol.

In addition, we were interested in directly comparing the effects of acute intoxication and the effects of differences in susceptibility to alcohol on cognition and behavior. At least three possibilities with respect to potential relations require examination. First, these effects may interact, such that acute intoxication effects are more pronounced among relatively more susceptible individuals. Second, the effects of susceptibility may be additive to acute intoxication effects (i.e. two main effects may be obtained). Third, acute intoxication may influence some aspects of cognition, whereas differences in susceptibility may influence other aspects, resulting in different patterns of effects for these variables. In any event, examination of differences in susceptibility may provide further resolution to both of the acute effects models we investigated. That is, predictions of either model may be more or less applicable to particular individuals depending upon their level of susceptibility to alcohol's effects.

2. Method

2.1. Participants

Forty-five healthy young adults (21 females) ages 21–30 were paid \$8.00 per hour for participation in this study. Participants were recruited using newspaper advertisements and by word-of-mouth. In order to be eligible for the study, 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. Potential participants who indicated any major medical conditions (including pregnancy) that contra-indicate alcohol administration were disqualified from the study, as were individuals with any history of substance abuse treatment. In addition, in order to ensure that the alcohol dose received in the study would be within participants' normal range of experience, naïve drinkers (i.e. individuals reporting an average of less than 2 drinks/week) and very heavy drinkers (individuals reporting an average of 25 or more drinks/week) were excluded from the study sample.

Participants deemed eligible following the telephone interview were required to adhere to a pre-experimental protocol in order to maintain their eligibility for the study. Participants were asked to refrain from any alcohol or drug use for 24 h prior to their appointment, to eat

a light meal 4–6 h prior to their appointment, and to refrain from strenuous physical exercise within 3 h of their appointment. Participants' compliance with these restrictions was assured via signed affidavits completed upon arrival at the laboratory. Additional affidavits were used to re-check participants' general health, drinking habits, and absence of major medical conditions. No participants were disqualified for failure to comply with pre-experimental protocol or discrepancies between interview items and signed affidavits. In addition, female participants were required to take a hormonal pregnancy test in the laboratory prior to the experiment to verify that they were not pregnant (no positive test results occurred).

2.2. Pre-experimental measures

2.2.1. Susceptibility to the effects of alcohol

We measured individual differences in susceptibility to the acute effects of alcohol using a composite measure recently developed by O'Neill et al. (2002). This measure consists of 19 items designed to assess subjective effects of drinking alcohol, and is believed to reflect aspects of consumption history and alcohol sensitivity. The items are of three types. The first type (10 items) are related to effects associated with the ascending limb of the blood alcohol curve; for example, becoming more talkative, more flirtatious, feeling high or 'buzzed', feeling more relaxed, etc. (i.e. positive, stimulating effects). These items are structured such that respondents indicate with 'yes' or 'no' whether they ever experience a given effect (e.g. 'Do you ever become more talkative after drinking alcohol'), and then estimate the *minimum* number of drinks that could be consumed before experiencing the given effect. The second type (6 items) are related to effects associated with the descending limb of the blood alcohol curve; for example, passing out, feeling nauseated, throwing up or vomiting, feeling dizzy (i.e. negative, sedative effects). These items are structured such that respondents estimate the *maximum* number of drinks they could consume before experiencing a given effect. For both of these types of items, a response is only included in a participant's score if he or she reports having experienced the effect in question. For instance, if a participant has never passed out from drinking, he or she is not asked to estimate the number of drinks it would take to experience that effect, and such items are not considered when determining a 'susceptibility score'. The three remaining items are designed to assess sensitivity relative to peers. Using a 5-point scale (1 = much more, 5 = much less), respondents indicate how much alcohol they can consume relative to peers of similar age, build, and sex before feeling an effect (item 1) and before feeling tipsy or drunk (item 2). The last peer item assesses how well respondents can hold their liquor relative to peers (1 = much better than, 5 = much worse than).

In a recent study involving nearly 300 young adult drinkers, O'Neill et al. (2002) factor analyzed the items making up the alcohol susceptibility measure, and compared responses on that measure with responses to the tolerance items used here (described below). Several of their findings are important for the current study. First, O'Neill et al. reported extremely high internal consistency among the susceptibility items ($\alpha = 0.97$), indicating a high degree of association among the items. The factor structure of the measure confirmed that the items appear to tap a single construct, with most relations in the data accounted for by a single factor (eigenvalue > 13). Also, these authors reported correlations ranging from 0.49 to 0.51 between susceptibility scores and alcohol tolerance (representing past-year and

lifetime symptoms, respectively) when tolerance was scored dichotomously, and 0.58–0.65 when tolerance was scored in a continuous manner (i.e. number of tolerance symptoms endorsed), suggesting considerable overlap in the constructs assessed by these measures. Finally, scores on the susceptibility measure were positively correlated with sex in that study: Women consistently reported needing fewer drinks to experience given effects than men ($r = 0.60$). In light of these findings, in the current study, responses to susceptibility and tolerance items were standardized and averaged to create a composite susceptibility score for each participant, with higher scores indicating lower sensitivity to alcohol effects (i.e. it takes more drinks to feel an effect).²

2.2.2. *Tolerance to the effects of alcohol*

Participants also responded to a number of items taken from published diagnostic instruments designed to measure symptoms of alcohol dependence (e.g. the Diagnostic Interview Schedule, version III-A [Robins et al., 1985]; Rutgers Alcohol Problems Index [White and Labouvie, 1989]; the Young Adult Alcohol Problems Screening Test [Hurlbut and Sher, 1992]). These items inquired about phenomena related to alcohol having less of an effect than it once did (e.g. ‘Did you ever find that your usual number of drinks had much less effect on you than it once did?’). These items were structured such that participants indicated whether they had never experienced a given phenomenon, had experienced it but not in the past year, or had experienced it in the past year (coded as 0, 1, or 2, respectively).

2.2.3. *Alcohol use*

Alcohol use was measured using a questionnaire in which participants were asked to estimate their alcohol involvement during the previous 30 days and also the past year. For the current study, a composite alcohol quantity/frequency variable (ALC) was created by summing per week alcohol quantity estimates for beer, wine, liquor, and wine coolers (based on past year) and multiplying by per week frequency estimates.

2.3. *Stimuli and experimental paradigm*

The paradigm employed a version of the Eriksen flanker task, as modified by Gratton et al. (1992). Each trial consisted of a 100 ms pre-stimulus baseline period followed by the presentation of one of four 5-letter arrays (HHHHH, SSHSS, SSSSS, or HSHHH) for 200 ms. The central letter in each array was the target letter, which was surrounded by flanker ‘noise’ letters. Participants were instructed to respond to one of the two target letters (H or S) with one hand (left or right) and to respond to the other letter with the other hand, by pressing one of two buttons on a response box. Thus, in each array, flankers were either compatible or incompatible with the correct response. The association between target letter and responding hand was counterbalanced across participants. Arrays were presented on a computer monitor positioned 60 cm in front of the participant. A fixation cross, placed just below the location of the target letter, remained on the screen throughout the experiment. The interval between

² We also conducted additional analyses in which susceptibility was computed on the basis of the alcohol susceptibility items alone (i.e. without including the tolerance items) and found highly similar results to those we report both in terms of behavioral performance and electrocortical responses.

stimulus arrays was 2500 ms. Although the probability of each target letter was kept at 50% throughout the experiment, the probability of compatible and incompatible noise letters was varied across blocks in order to influence participants' expectations concerning upcoming trial types (see Gratton et al., 1992). Specifically, the following probability levels were used: 50/50 (equal proportions of compatible and incompatible noise trials), 80/20 (80% of the trials had compatible noise, and 20% had incompatible noise), and 20/80 (20% of the trials had compatible noise, and 80% had incompatible noise), resulting in expect-neutral (EN), expect-compatible (EC), and expect-incompatible (EI) conditions, respectively.

2.4. Beverage administration

The beverage administration procedure in the current study closely resembles that used by Sher and Walitzer (1986). 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 dose (actually, 0.04 g/kg ethanol) vodka (100 proof) and tonic beverage. All participants were given the moderate dose expectancy in order to reduce the discrepancy between actual and expected doses as much as possible across conditions, thereby enhancing the viability of our cover story. 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 (9 parts flattened tonic to 1 part vodka mixed in a 100 proof vodka bottle), and the high dose was achieved by using 'spiked' tonic (4 parts tonic to 1 part 100 proof vodka mixed in a tonic bottle). 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 blind to the actual contents of the beverage bottles.

2.5. Measurement of blood alcohol concentration (BAC) levels

BAC was measured throughout the experimental session using an Alco-Sensor IV breath analysis device (Intoximeters, St. Louis, MO). Participants were not informed of their actual BAC level during the experimental task. To ensure that residual alcohol would not build up inside the mouthpiece, a new disposable mouthpiece was used for each sample taken during a laboratory session. To eliminate residual alcohol in the mouth, participants rinsed their mouths with water prior to the first post-drinking BAC measurement.

2.6. Subjective intoxication measures

In addition to BAC measurement, we included two subjective indices of alcohol's effects assessed via a short questionnaire at the conclusion of the session. First, participants indicated how intoxicated they felt throughout the experimental task using a 5-point Likert-type scale (0 = not at all, 4 = a lot). Second, participants estimated how much their performance was affected by the beverage they consumed, using a similar scale (1 = not at all, 5 = extremely). Participants also estimated the number of standard alcohol drinks they believed they consumed using a 0–20 scale.

2.7. Procedure

Upon arrival at the laboratory, an experimenter weighed participants who then read and signed the informed consent form and completed the pre-experimental measures and affidavits. Upon 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. To familiarize them with the task prior to beverage consumption, participants completed a short practice sequence consisting of three blocks of 60 trials each of the flanker task in which all letter arrays were equally probable. Participants were instructed to respond as quickly and accurately as possible, but unlike in some previous studies (e.g. Gratton et al., 1992), participants were not trained (via their practice block performance) to respond with any particular level of speed or accuracy (i.e. neither speed nor accuracy were given particular emphasis in verbal instructions). Following these practice blocks, 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. The beverage was then 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, limejuice was added according to each participant's preference. Upon completion of the final drink, participants sat idle for 20 min to allow the alcohol to absorb. Following the absorption period, a second intoxication measurement was taken just before participants completed the first half of the experimental trials (12 blocks of 60 trials each), after which a third intoxication measurement was taken. Participants then completed the remaining 12 blocks of trials, after which a fourth intoxication measurement was taken. Electrodes were then removed and participants were led to another nearby room to complete a brief packet of post-experimental questionnaires, following which participants were debriefed about the true nature of the study. Participants in the high dose condition were retained in the laboratory until a breath test indicated that their BAC was 0.04% or less. All participants, regardless of beverage condition, were driven home after the session by a friend or by taxi provided by the experimenters.

2.8. Electrophysiological recording

The electroencephalogram (EEG) was recorded from 20 standard scalp locations (referenced to linked mastoids) using an electrode cap (Electrocap, International) according to the 10–20 international electrode placement system. Vertical and horizontal electrooculogram (EOG) was 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 procedure described elsewhere (Gratton et al., 1983). The EEG and EOG were recorded continuously for the duration of each trial (1400 ms), including a 100 ms pre-stimulus baseline, at a digitizing rate of 100 Hz. Impedance was kept below 10 k Ω . The signals were amplified using Grass amplifiers, and a 0.01–30 Hz bandpass filter was used.

3. Results

3.1. Analytic approach

Data from three male participants were discarded due to a high proportion of trials with large measurement artifacts in the ERP, leaving our study sample size at 42 participants. A median split was carried out on the alcohol susceptibility scores of the remaining participants to create a group of high-susceptible (HS) and low-susceptible (LS) participants ($n = 21$ in each group; HS and LS participants were equally represented in all dose groups. Four participants failed to complete the post-experimental questionnaire ($n = 1$ from placebo and moderate dose groups; $n = 2$ from the high dose group), so analyses of the post-experimental subjective intoxication items are based on 38 individuals. Probability levels for all analyses involving within-subjects factors with more than two levels were adjusted using the Greenhouse–Geisser correction for potential violations of sphericity.

3.2. Manipulation checks

3.2.1. Alcohol dose

Analysis of BAC levels attained during the experimental task among participants in the three dose groups indicated that, as expected, our dosing procedure resulted in significantly different BAC levels in the placebo ($M = 0.00\%$, $S.D. = 0.00$), moderate dose ($M = 0.035\%$, $S.D. = 0.01$), and high dose groups ($M = 0.07\%$, $S.D. = 0.01$). The levels in the moderate and high dose groups differed significantly, $F(1, 24) = 59.21$, $P < 0.01$.³ Participants' level of susceptibility and BAC levels were not correlated, $r = 0.01$, $P > 0.50$, indicating that any effects of susceptibility on our other measures are not due to differences in BAC.

3.2.2. Subjective effects of alcohol

Participants' post-experimental ratings of how drunk they felt during the task differed monotonically by dose ($M_s = 0.61, 1.16, \text{ and } 2.02$ for placebo, moderate, and high dose, respectively), $F(2, 32) = 9.87$, $P < 0.01$, as did participants' estimates of the number of standard drinks they believed they consumed ($M_s = 1.95, 2.98, \text{ and } 4.51$), $F(2, 32) = 7.37$, $P < 0.01$. Note that participants in the placebo group estimated that they had consumed nearly two standard drinks on average, suggesting that the induction of a moderate dose expectancy for all participants may have had some effect on their subjective experience; planned comparisons indicated that participants' estimates in the placebo condition were not significantly lower than those made by participants in the moderate dose condition ($P = 0.12$), but that high dose participants' estimates were significantly higher than those in both other conditions ($P_s < 0.05$).

³ Due to zero variability in the BAC levels among those in the placebo group, this ANOVA was restricted to only those in the moderate and high dose groups. It is clear, however, that the mean BAC level in the placebo group also differed from that in the other two groups.

3.3. Self-reported susceptibility and alcohol consumption

The correlation between participants' self-reported susceptibility to alcohol's effects and their self-reported alcohol use was positive and significant, $r = 0.60$, $P < 0.001$, indicating that participants who reported more alcohol use also reported that they require a larger number of drinks before feeling the effects of alcohol (i.e. lower susceptibility). Because alcohol consumption typically varies as a function of gender, we also correlated scores on the susceptibility measure with participants' sex. This correlation also was significant, $r = 0.44$, $P < 0.01$. Given that scores on this measure reflect consumption (i.e. the number of drinks needed to feel a given effect), this finding is not surprising, but suggests that accounting for gender effects in our other analyses may be important. Both of these correlations mirror the findings reported by O'Neill et al. (2002).

3.4. Behavioral performance and ERPs during the flanker task

In order to facilitate a clearer interpretation of our results, the effects of our manipulations on response times were examined for correct response trials only. ERP analyses were conducted separately on correct response and error trials.

3.4.1. Behavioral data

Mean response times (RTs) and response accuracy (proportion of correct responses) for compatible and incompatible noise trials as a function of expectancy condition and dose group are presented in the upper and middle panels of Table 1. To simplify the analyses of dose and susceptibility on behavior, within-subjects difference scores were calculated for accuracy and RTs by subtracting responses to compatible trials from responses to incompatible trials (i.e. the noise-compatibility effect) within each expectancy condition, and analyses were performed on these difference scores using separate 3 (Dose: placebo, moderate, high) \times 2 (Susceptibility: LS, HS) \times 3 (Expectancy; expect-compatible, expect-neutral, expect-incompatible) ANOVAs, with repeated measures on the last factor. Prior to analyses, the response accuracy data were standardized using z -score transformations to normalize their distribution. However, we present raw score means for ease of interpretation.

Analyses of the RT data showed a main effect of Expectancy, $F(2, 72) = 127.45$, $P < 0.001$. Consistent with the results of Gratton et al. (1992), the noise-compatibility effect decreased monotonically between EC ($M = 74$ ms), EN ($M = 50$ ms) and EI ($M = 28$ ms) conditions, indicating that participants adjusted their processing strategies according to the type of noise they expected. Note that this effect is identical to the Expectancy \times Compatibility interaction that would be obtained if raw RTs (rather than difference scores) were used in the analyses. Contrary to our predictions, the expectancy main effect did not differ as a function of alcohol dose ($F < 1$). Inspection of the compatibility effect means in Table 1 illustrates that expectancy modulation of this effect was highly similar across dose groups. Follow-up analyses confirmed that the main effect of Expectancy on the compatibility effect means was highly significant for each dose group, $F_s(1, 36) > 50$, $P_s < 0.01$. Moreover, despite the apparent slowing of responses with increasing doses of alcohol evident in Table 1, the main effect of Dose on reaction time was not reliable, $F(2, 36) = 1.05$,

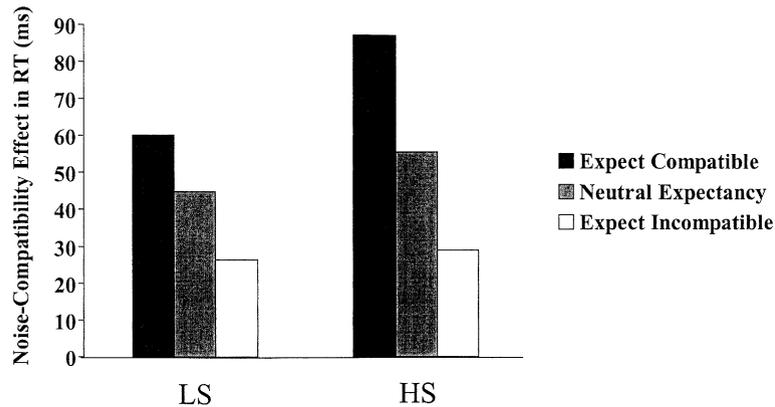


Fig. 1. Noise compatibility effect in response time (RT) as a function of expected trial type and alcohol susceptibility group. The compatibility effect is obtained by subtracting RTs to compatible trials from RTs to incompatible trials.

$P > 0.30$. A planned contrast of the dose effect, although suggestive, also showed that the linear trend apparent in the data was not significant, $F(1, 36) = 2.10$, $P < 0.16$. The expectancy effect was qualified by susceptibility level, however, $F(2, 72) = 9.56$, $P < 0.001$. As shown in Fig. 1, although both LS and HS participants showed the typical modulation of the noise-compatibility effect as a function of expectancy (e.g. Gratton et al., 1992), this modulation was more pronounced among HS participants. Comparison of standardized effect size estimates confirms this assertion: Cohen's d s (mean differences represented as variance units; see Cohen, 1988) = 2.08 and 3.62 for LS and HS groups, respectively. Also, HS participants showed a larger noise compatibility effect overall ($M = 57$ ms) than LS participants ($M = 44$ ms), $F(1, 36) = 8.45$, $P < 0.01$. Both of these results suggest that HS participants processed (were influenced by) the flankers more than did LS participants.

Mean response accuracy for compatible and incompatible noise trials as a function of expectancy condition and dose group is presented in the middle panel of Table 1. Analysis of response accuracy means showed a predicted main effect of Expectancy, $F(2, 72) = 12.38$, $P < 0.01$. Planned comparisons showed that, as predicted, the noise-compatibility effect became progressively smaller from EC to EN to EI conditions (M s = 0.07, 0.05, 0.03, respectively). This effect was qualified by a significant Dose \times Expectancy interaction, $F(4, 72) = 2.72$, $P < 0.05$. Note that this interaction is identical to the 3-way Dose \times Expectancy \times Compatibility interaction that would be obtained if raw scores were used in the analysis. As shown in Table 1, modulation of the compatibility effect by expectancy was larger among participants in the high dose group ($d = 1.81$) than among those in the placebo ($d = 0.52$) and moderate dose groups ($d = 0.52$). Neither the main effect of Dose nor that of Susceptibility was reliable (F s < 1).

Given the significant correlation between sex and susceptibility scores (reported in the previous section), we conducted an ancillary ANOVAs on the RT and accuracy data

Table 1

Mean response times, proportion of correct responses, and P3 latencies to compatible and incompatible noise trials as a function of expectancy condition and alcohol dose

Dose group	Expect compatible			Neutral expectancy			Expect incompatible		
	Compat	Incompat	CE	Compat	Incompat	CE	Compat	Incompat	CE
<i>Response times (ms)</i>									
Placebo	458 (65)	528 (60)	70	472 (73)	520 (70)	48	482 (69)	508 (66)	26
Moderate	483 (74)	555 (80)	72	495 (80)	544 (87)	49	508 (76)	533 (79)	25
High	494 (70)	572 (87)	78	511 (73)	565 (82)	54	519 (74)	550 (76)	31
<i>Response accuracy</i>									
Placebo	0.97 (0.02)	0.92 (0.09)	0.05	0.98 (0.03)	0.93 (0.07)	0.04	0.95 (0.14)	0.92 (0.14)	0.03
Moderate	0.95 (0.05)	0.90 (0.10)	0.05	0.97 (0.05)	0.93 (0.09)	0.04	0.97 (0.03)	0.94 (0.08)	0.03
High	0.98 (0.02)	0.88 (0.11)	0.10	0.98 (0.01)	0.92 (0.08)	0.06	0.97 (0.03)	0.94 (0.10)	0.03
<i>P3 latency (ms)</i>									
Placebo	501 (112)	547 (105)	46	503 (125)	545 (107)	42	512 (138)	526 (116)	14
Moderate	499 (110)	517 (65)	18	490 (58)	527 (71)	37	506 (85)	518 (52)	12
High	523 (109)	609 (103)	87	556 (125)	586 (98)	30	542 (101)	553 (92)	11

Note: Numbers in parentheses are standard deviations. Compat = compatible noise trials; Incompat = incompatible noise trials; CE = compatibility effect (difference score). For response time and P3 latency, this score was calculated as incompatible-compatible. In order to yield a positive value, for response accuracy, this score was calculated as compatible-incompatible.

in which participants' sex was included as an additional factor in order to account for potential sex effects.⁴ These analysis showed no significant sex main effects or interactions involving sex ($F_s < 2.0$, $P_s > 0.20$), and the other effects remained virtually unchanged.

3.4.2. ERP amplitude data

Prior to analysis of our ERP data, we conducted a principal components analysis (PCA) on the mean ERP amplitudes in order to organize the post-stimulus activity into time intervals with shared variance. Use of this approach provides a way to select time epochs that are not entirely arbitrary, and helps to reduce the effects of overlapping components. Averaged ERP waveforms submitted to PCA with varimax rotation produce component loadings indicating orthogonal sources of variation in the waveform (Chapman and McCrary, 1995; van Boxtel, 1998). One of the resulting matrices contains a single component score for each waveform indicating the degree to which the component varies in that waveform. These scores can be analyzed similarly to peak amplitude measures (see Donchin and Heffley, 1978). Examination of component loadings reveals the point in time at which components of interest are most active. The PCA revealed four post-stimulus time epochs with meaningful loadings: 200–350, 400–500, 600–700 and 900–1300 ms. The first two of these appear to most closely represent the development and peak of the P3 component. Given that our concern was primarily with activity related to the P3, we present here only analyses of component scores in the first two epochs.

The component scores associated with the first two epochs suggested by the PCA as measured at midline scalp locations were analyzed using a 3 (Dose; placebo, moderate, high) \times 2 (Susceptibility; HS, LS) \times 2 (Epoch; 200–350, 400–500 ms) \times 3 (Expectancy; expect-compatible, neutral expectancy, expect-incompatible) \times 2 (Compatibility; compatible trials, incompatible trials) \times 3 (Electrode; Fz, Cz, Pz) mixed-factorial ANOVA, with repeated measures on the last four factors. This analysis showed a main effect of Dose, $F(2, 36) = 3.74$, $P < 0.05$. Inspection of the waveforms presented in Fig. 2 shows that alcohol tended to decrease P3 amplitude. Planned comparisons indicated that when collapsed across all other factors in the analysis, amplitudes in the placebo group ($M = 0.77$) differed from those in the other groups ($M_s = 0.10$ and 0.01 in moderate and high dose groups, respectively, $P_s < 0.01$), but that moderate and high dose group means did not differ ($P > 0.50$). The analysis also showed a main effect of Susceptibility, $F(1, 36) = 5.86$, $P < 0.05$. Fig. 3 depicts the influence of dose and susceptibility on ERP amplitudes elicited by incompatible trials. As shown in Fig. 3, the amplitude of the P3 was markedly smaller among LS participants relative to HS participants, particularly at central and parietal locations. Finally, dose and susceptibility did not significantly interact in their effects on ERP amplitudes, $F(2, 36) = 1.83$, $P > 0.15$. Other significant effects included a main effect of electrode, $F(2, 72) = 32.90$, $P < 0.001$, indicating increasing positivity from frontal to parietal locations; an Epoch \times Compatibility interaction, $F(1, 36) = 22.49$, $P < 0.001$,

⁴ Inspection of the data revealed that sex and susceptibility were completely confounded among participants in the moderate dose group (i.e. all female participants were classified as HS, all males were classified as LS), merely because of random assignment. Thus, analyzing the full model including sex produces empty cells in the design. As such, this analysis includes participants in the placebo and high dose groups only.

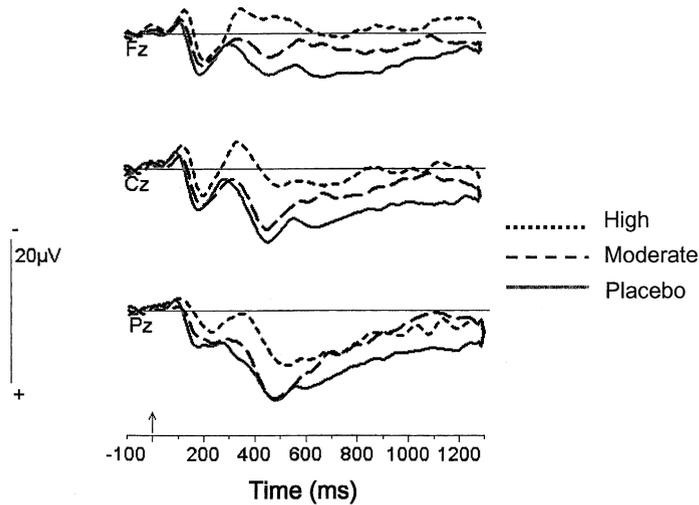


Fig. 2. Grand average ERPs elicited by incompatible trials in the expect-compatible conditions, as a function of alcohol dose. The vertical arrow at 0 ms represents stimulus onset.

indicating that the noise-compatibility effect was only evident in the later epoch; and an Epoch \times Compatibility \times Expectancy interaction, $F(2, 72) = 5.89$, $P < 0.01$. Inspection of the latter interaction revealed that inconsistency between expected and actual trial type (e.g. incompatible trials in the expect-compatible condition) was associated with larger P3 am-

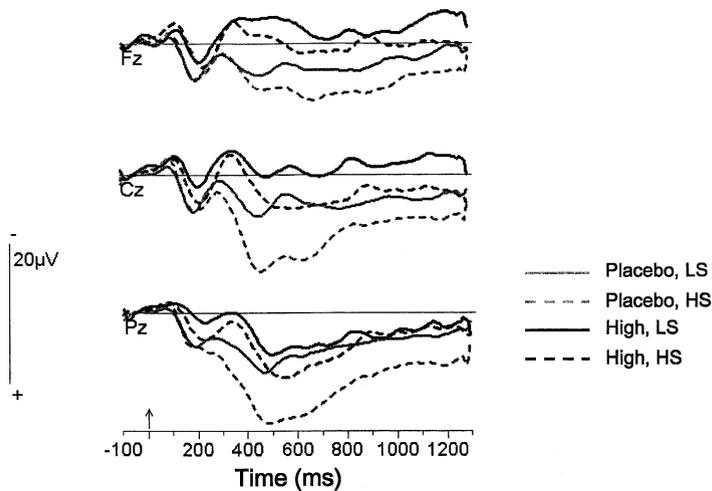


Fig. 3. Grand average ERPs elicited by incompatible trials in expect-compatible conditions as a function of dose and self-reported alcohol susceptibility. To simplify presentation, only high dose and placebo groups are presented. HS = high susceptibility group; LS = low susceptibility group.

plitude in the later epoch. No other main effects or interactions of interest were reliable.⁵ Of note is the lack of a significant Dose \times Compatibility interaction in the later epoch, $F(2, 36) = 0.69$, $P > 0.50$, indicating that participants in all dose groups evidenced a similar difference in P3 amplitude between compatible and incompatible noise trials.⁶

3.4.3. ERP latency data

To examine the hypothesis that alcohol should reduce the compatibility effect in P3 latency, we measured the latency of the positive peak of the ERP occurring between 350 and 800 ms post-stimulus at the Pz electrode location. These data, presented in the lower panel of Table 1, were analyzed using a 3 (Dose) \times 2 (Susceptibility) \times 3 (Expectancy) \times 2 (Compatibility) ANOVA, with repeated measures on the last two factors. This analysis revealed a main effect of Compatibility, $F(1, 36) = 29.87$, $P < 0.01$, which was qualified by a significant Expectancy \times Compatibility interaction, $F(1, 36) = 13.26$, $P < 0.01$. As with the RT data, the compatibility effect in P3 latency decreased between EC ($M = 71$ ms), EN ($M = 32$ ms), and EI conditions ($M = 07$ ms). This interaction was further qualified by a Dose \times Expectancy \times Compatibility interaction, $F(4, 72) = 2.80$, $P < 0.05$. Follow-up contrasts revealed that, as predicted, differences in the compatibility effect as a function of dose were limited to the expect compatible-condition. However, in contrast to predictions, the effects of alcohol were limited to the moderate dose level. Specifically, incompatible trials significantly delayed the latency of the P3 among both placebo ($M = 46$ ms), $F(1, 36) = 7.13$, $P < 0.01$, and high dose participants ($M = 87$ ms), $F(1, 36) = 25.21$, $P < 0.01$, but not among those in the moderate dose group ($M = 18$ ms), $F(1, 36) = 1.32$, $P > 0.20$ (see Fig. 4). In addition, comparison of effect sizes indicates that the effect was larger in the high dose group ($d = 0.94$) than in the placebo group ($d = 0.50$).

3.4.4. Ancillary ERP analyses

Incompatible trials apparently elicited a pronounced frontal-central negativity in the ERP among those in the high dose group (see Fig. 2). The waveforms in Fig. 3 further suggest that this negativity might be more pronounced among LS participants. Although these waveforms are stimulus-related, this component has a similar scalp distribution to the error-related negativity (ERN) that routinely accompanies response errors in choice paradigms (e.g. Falkenstein et al., 1990; Gehring et al., 1993; Gehring and Knight, 2000; Scheffers et al., 1996). To examine this component further, we conducted an exploratory analysis of ERP activity elicited at frontal and central locations on incorrect response trials using a 3 (Dose) \times 2 (Susceptibility) \times 2 (Epoch) \times 2 (Electrode site; Fz, Cz) ANOVA, with repeated measures on the last two factors. Fig. 5 presents ERP waveforms elicited on error trials as a function of dose and susceptibility. The ANOVA showed a main effect of Dose,

⁵ Although the ANOVA revealed several other higher-order interactions involving Expectancy, Epoch, Compatibility, and Electrode, these interactions are not central to the hypotheses of the current study and so will not be discussed.

⁶ As with the behavioral data, we conducted an ancillary analysis including sex as an additional factor, in order to examine whether our susceptibility (or other) effects are dependent on gender differences. This analysis revealed no significant main effect of sex or any interactions involving sex and other variables. Furthermore, our other effects were unchanged with sex introduced into the model.

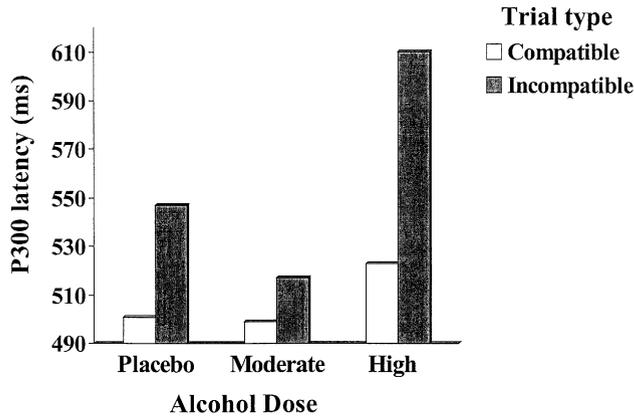


Fig. 4. Mean P3 latencies (300–850 ms) in the expect-compatible condition as a function of compatibility and alcohol dose.

$F(2, 36) = 3.41, P < 0.05$, indicating that this negativity was apparent only among those in the two alcohol groups ($M_s = 0.50, -0.14$, and -0.68 in placebo, moderate, and high dose groups, respectively). This effect was qualified by a marginal a Dose \times Susceptibility interaction, $F(2, 36) = 2.88, P < 0.07$. Specific contrasts of the dose effect among LS and HS groups showed that alcohol significantly enhanced this negativity among HS partici-

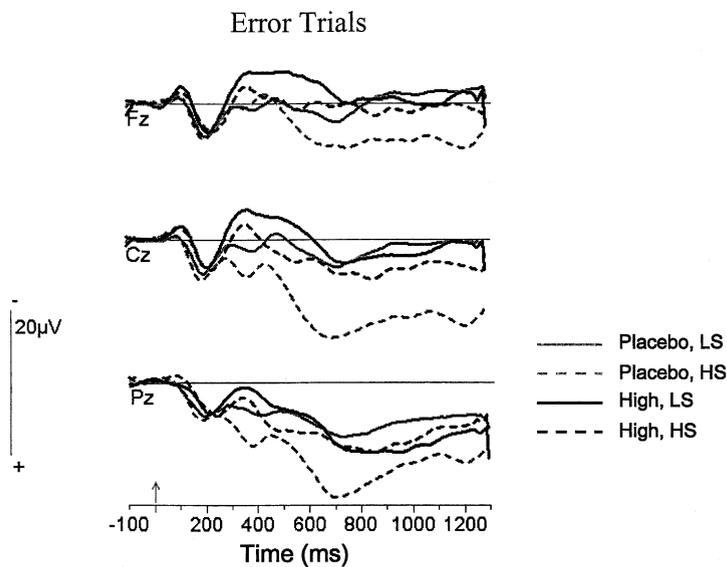


Fig. 5. Grand average ERPs elicited on error trials as a function of alcohol dose and self-reported alcohol susceptibility. To simplify presentation, only high dose and placebo groups are presented. The vertical arrow at 0 ms represents stimulus onset. HS = high susceptibility group; LS = low susceptibility group.

pants ($M_s = 1.40, -0.33, -0.41$, for placebo, moderate, and high dose groups, respectively) $F(1, 36) = 10.16, P < 0.01$, but not among LS participants, $F(1, 36) = 0.75, P > 0.50$, for whom the negativity was apparent regardless of alcohol dose ($M_s = -0.40, -0.03, -0.96$, respectively). However, given the exploratory nature of this analysis, these effects should be interpreted with caution.

4. Discussion

The primary goal of this research was to examine the effects of acute intoxication on attention and strategic control processes, within the context of two theoretical models of alcohol effects; namely, the attention-allocation model (Steele and Josephs, 1990) and the impaired response inhibition model (Vogel-Sprout, 1992; Fillmore and Vogel-Sprout, 1999). We also were interested in whether the acute dose effects would be moderated by self-reported level of susceptibility to alcohol. We tested these models using a response competition paradigm, examining both behavioral and electrocortical measures of attentional control. Although this paradigm does not represent a critical test of either theory, our findings are informative to both models.

The data provided by our behavioral measures provide some support for the response inhibition model of alcohol effects (e.g. Vogel-Sprout, 1992), but appear less consistent with the attention-allocation model (e.g. Steele and Josephs, 1990). The interaction of dose and expectancy in our analysis of response accuracy showed that modulation of the noise-compatibility effect was enhanced in the high dose group, indicating that more or larger adjustments in processing strategy occur under intoxication. Said differently, high dose participants appeared to be more influenced by manipulation of the flanker letters than were participants in the other dose groups, suggesting that alcohol did not restrict attentional focus per se but instead increased the response conflict associated with processing incompatible flanker letters. This finding does not support a strict interpretation of the attention-allocation model, in which intoxication is posited to impair processing of such peripheral information. However, this conclusion should be tempered in light of alternative interpretations of the tenets of the attention-allocation model. That is, if the flanker letters are viewed as more salient to participants under the influence of alcohol, they may direct more attentional resources to these letters or to the entire array, and if so this finding could be interpreted as consistent with the attention-allocation perspective.

It may seem surprising that alcohol did not produce more robust effects on response time in this paradigm. However, the fact that alcohol influenced accuracy but had no reliable effect on response time is consistent with previous findings obtained using other response conflict tasks (e.g. Curtin and Fairchild, 2003; Fillmore and Vogel-Sprout, 2000), and is generally in line with the impaired response-inhibition model of alcohol effects (e.g. Fillmore and Vogel-Sprout, 1999; Vogel-Sprout et al., 2001). Specifically, this finding suggests that processes related to response selection and execution are more sensitive to alcohol's acute effects than are attention control processes per se (see also Curtin and Fairchild, 2003). On the other hand, differences in self-reported susceptibility to alcohol effects did significantly influence response times, but had no reliable effect on response accuracy. That expectancy modulation of the noise-compatibility effect in response time was larger among

HS participants than LS participants indicates that HS participants attended to peripheral letters and therefore processed more information prior to making a response than did LS participants. This pattern is consistent with the use of a parallel processing strategy among HS participants, and suggests that differences in susceptibility may correspond to differences in the initial stages of processing, such as the control of attention or initial attentional filtering. When considered together, the findings from our behavioral measures suggest that differences in self-reported susceptibility do not moderate the effects of acute alcohol consumption, but rather that these variables influence different aspects of processing.

Our ERP data provided mixed support for both theoretical models we examined. The pattern of P3 latencies in the expect-compatible condition was consistent with predictions derived from the attention-allocation model, but only among participants in the moderate dose group. The latency of the P3 component was similar to both compatible and incompatible trials among moderate dose participants. We have argued that participants use a parallel processing strategy when expecting compatible trials, and that they must switch to a focused mode of processing in order to respond correctly when incompatible trials are encountered (see Gratton et al., 1992). Accordingly, these data suggest that moderate dose participants utilized the focused processing mode, or experienced less difficulty switching from parallel to focused mode, relative to sober participants. This pattern would be expected if alcohol focuses attention on the target letter. However, alcohol had the opposite effect at the higher dose level, such that when compatible trials were expected, the latency difference between compatible and incompatible trials was increased relative to placebo. This pattern is more consistent with the impaired response inhibition model, in that the high dose of alcohol led to difficulty in switching to the focused mode and/or increased reliance on flanker information.⁷ Thus, it appears that under low doses of alcohol, the influence of potentially distracting peripheral information may be reduced, a finding consistent with research on divided attention tasks (e.g. Curtin et al., 2001; Erblich and Earleywine, 1995; Patel, 1988). However, under higher alcohol doses, processing of peripheral information was enhanced relative to placebo. That these dose effects were present in P3 latency but not RT suggests that ERP measures may be particularly sensitive to alcohol's effects on cognitive control, or that alcohol produces a disconnect between neural and behavioral manifestations of this process.

Unfortunately, the P3 amplitude data in this study did not clearly support either model. Support for the attention-allocation model would be obtained if the compatibility effect were smaller in the alcohol groups compared to placebo (indicating decreased attention to flankers under alcohol), whereas a larger compatibility effect under alcohol than placebo would be consistent with the response-inhibition model (indicating impaired inhibition of flanker-related response activation). Neither of these patterns was observed. Instead, the compatibility effect appears to have been similar regardless of alcohol dose.

⁷ It also should be noted that P3 latency effects can be caused by variations in motor processes in addition to variations in aspects of stimulus processing. As such, the alcohol effects reported here may reflect alcohol-related impairment of response-related processes. To examine this possibility, we conducted some additional analyses focused on the lateralized readiness potential, a response-related ERP component indexing motor preparation (see Rugg and Coles, 1995). Although this analysis revealed effects of compatibility similar to those reported elsewhere (Gratton et al., 1992), there were no significant main effects or interactions with dose. As such, this alternative explanation seems less compelling than the interpretation we have offered.

However, our ERP amplitude data are informative with respect to understanding the difference influences of dose and susceptibility on attention and response selection processes. Previous studies have suggested that acute alcohol doses decrease the amplitude of the P3 (Noldy, 1998), and that reductions in P3 amplitude may reflect a deficit in cortical inhibitory mechanisms (e.g. Cohen et al., 1997; Ramachandran et al., 1996). Although the pattern of waveforms measured at Pz appears consistent with this notion, the waveforms measured at frontal and central locations suggest that alcohol influences a negative component of the ERP, peaking at around 400 ms. Our examination of error trial activity (i.e. Fig. 5) indicates that this negativity occurred on both correct and incorrect trials in the high dose group, suggesting that alcohol led to the activation of both correct and incorrect responses on each trial (i.e. response competition). Activation of both correct and incorrect response channels on the same trial has been labeled ‘aspecific activation’ (Gratton et al., 1988), and is thought to be the result of a preliminary and incomplete evaluation of the stimulus array, driven primarily by the noise letters. Brain imaging studies point to structures within the prefrontal cortex, especially the anterior cingulate and basal ganglia, as the likely source of scalp-recorded negativity associated with behavioral errors (e.g. Falkenstein et al., 2001; Gehring and Knight, 2000; Kiehl et al., 2000). Furthermore, recent data indicates that individuals with lateral prefrontal impairment show negative components of equal magnitude for correct trials and errors (Gehring and Knight, 2000). To the extent that the stimulus-related negativity seen among high dose participants in the current study is related to response-related ERN activity, these data could also be viewed as evidence that alcohol produced frontal and prefrontal impairment in this paradigm (e.g. Peterson et al., 1990) that is specifically related to response selection and/or execution. Of course, this interpretation should be viewed with caution as this effect was not predicted and is not consistent with previous reports. Future work may help to clarify the significance of this negative component as a function of alcohol consumption.

Differences in alcohol susceptibility, on the other hand, were primarily associated with variations in the amplitude of the P3, with HS participants experiencing more typical (larger) P3s to incompatible trials in the expect-compatible conditions than LS participants. This finding suggests that susceptibility differences are related to the amount of information processing elicited by incompatible trials, and is consistent with the reaction time data indicating that HS participants engage in more or larger strategic adjustments. As argued by Gratton et al. (1992), these strategy adjustments are adaptive in that the goal is to optimize performance by accounting for information conveyed by previous trials. In this context, our data suggest that HS participants are more likely to adjust processing strategies when encountering unexpected information. Although this process appears to slow response times, it is arguably a more adaptive approach.

To our knowledge, these findings are the first to indicate that self-reported differences in reactions to alcohol are related to global differences in information processing and attention. The fact that susceptibility effects were evident in the placebo group is remarkable, and suggests that scores on this measure are an indication of a more general response tendency than differential susceptibility to the acute effects of alcohol. What might underlie scores on this measure? One possibility suggested by our data is that differences in alcohol susceptibility relate to differences in working memory capacity. Working memory capacity (WM) is defined as the number of representations that can be kept in mind at one time and

used to effectively guide behavior (e.g. Baddeley, 1986; see also Engle, 2002; Vogel-Sprott et al., 2001). Considerable research indicates that differences in WM predict performance on a variety of higher-order cognitive tasks, particularly those involving some level of interference (Engle, 2002). In the current study, expectancy modulation of the noise compatibility effect was larger among HS than among LS participants, which could result from HS participants' tendency to hold more information concerning previous trials in working memory during the task. In addition, the larger P3 amplitudes associated with this effect among HS participants are indicative of more extensive updating of WM templates during stimulus processing (e.g. Donchin, 1981; see also Bartholow et al., in press), suggesting basic WM span differences between HS and LS individuals. Ongoing research in our laboratories is currently evaluating this hypothesis further.

Another, related possibility suggested by the correlation between susceptibility scores and recent consumption history is that the information processing differences between HS and LS participants in our data might reflect deficits due to recent consumption. Nichols and Martin (1996) found that P3 amplitudes were significantly reduced among heavy (more than 20 drinks/week) as opposed to light (less than 10 drinks/week) social drinkers during a word presentation task. Typical consumption among participants in the current study ranged from 2 to 25 drinks/week, but the average was less than 10 per week ($M = 7.68$, $S.D. = 6.43$). Therefore, our findings might indicate that even the relatively moderate consumption patterns reported by our heaviest-drinking participants result in potentially long-term processing deficits. This notion is consistent with the WM hypothesis in that increased consumption may determine a decrease in WM capacity that leads to information processing differences such as those we report.

In conclusion, these findings add to the collective understanding of the acute effects of alcohol consumption on cognitive processing and attention. Analyses of the acute effects of alcohol at the higher dose level lend support to the impaired response inhibition model of alcohol effects, whereas effects of the lower alcohol dose were consistent with the attention-allocation model. This dose-response pattern requires further investigation before firm conclusions can be drawn. In addition, our data suggest that alcohol influences a frontal negativity in the ERP, and that this component may reflect response competition processes that are amplified under the influence of alcohol. Also, our findings suggest that self-reported alcohol susceptibility does not generally serve to moderate acute dose effects, but rather that these two variables may influence distinct information processing systems. In the future, researchers should further examine potential correlates of self-reported alcohol susceptibility, both in terms of other self-report measures and in terms of additional cognitive mechanisms that susceptibility may influence.

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