Journal of Abnormal Psychology
2003, Vol. 112, No. 3, 424–436

0021-843X/03/$12.00 DOI: 10.1037/0021-843X.112.3.424

Alcohol and Cognitive Control: Implications for Regulation of Behavior During Response Conflict

John J. Curtin and Bradley A. Fairchild
University of Wisconsin—Madison

Alcohol intoxication often leads to dysregulated behavior in contexts characterized by conflict between prepotent response tendencies and incompatible alternative responses. Recent research has identified 2 components of an anterior executive attention system that are essential for adaptive behavior when response conflict exists. Event-related potential (ERP) measures of evaluative and regulative cognitive control were collected to determine if impaired executive attention was responsible for observed behavior deficits when intoxicated. Intoxicated participants displayed task performance deficits on incongruent color-naming trials relative to sober controls. Alcohol did not affect P3 magnitude/latency, indicating that timing and integrity of stimulus evaluation remained intact. In contrast, alcohol did reduce frontal components of ERP that index evaluative and regulative cognitive control processes.

Links between alcohol use and "real-world" dysregulated responding have been clearly established across multiple response domains. For example, acute alcohol intoxication has been observed to lead to aggression (Bushman & Cooper, 1990), sexual and other risk-taking behaviors (Burian, Liguori, & Robinson, 2002; Morris & Albery, 2001), increased self-disclosure (Caudill, Wilson, & Abrams, 1987; Rohrberg & Sousa-Poza, 1976), and alterations in emotional response (Curtin, Lang, Patrick, & Stritzke, 1998; Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001). Moreover, intoxicated individuals display behavioral deficits in experimental paradigms requiring response inhibition such as the go–stop (Mulvihill, Skilling, & Vogel-Sprott, 1997) and go/no-go (Finn, Justus, Mazas, & Steinmetz, 1999) tasks.

Many examples in which alcohol intoxication results in dysregulated behavioral response are characterized by conflict between prepotent, but inappropriate, response tendencies and incompatible alternative responses that are more adaptive (Casbon, Curtin, Lang, & Patrick, in press; Steele & Southwick, 1985). For instance, unprotected sexual intercourse may occur when a strong and immediate appetitive response tendency conflicts with the inclination to delay or abstain from intercourse until appropriate protection is available. Similarly, aggressive response may result when salient instigatory cues such as physical or verbal provocation overcome competing environmental (e.g., provoker is significantly larger than self) or internal (e.g., associative knowledge about potential adverse consequences of aggressive response) cues that suggest an alternative nonaggressive response. Recent cognitive neuroscience research has identified two components of an anterior executive attention system (evaluative and regulative cognitive control) that are essential for adaptive behavior when response conflict exists (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Braver, Barch, & Cohen, 1999; Carter, Botvinick, & Cohen, 1999). This article describes results from a project that examined intoxicated behavior in a laboratory analogue conflict paradigm that is well validated, the Stroop task. Event-related potential (ERP) measures that assess evaluative and regulative cognitive control were collected to determine if impaired function in these components of executive attention were responsible for observed behavior deficits when intoxicated.

Alcohol, Response Conflict, and Behavior

The thesis that alcohol will affect behavior when response conflict exists is a relatively longstanding idea in the literature. Steele and Southwick (1985) conducted a meta-analysis to examine the role of conflict in alcohol's effect on social behaviors, including aggression, risk taking, sexual interest, gambling, and other stereotypic intoxicated behaviors. Results provided strong support for the thesis that alcohol affects behavior through its impact on response conflict as alcohol effect sizes were dramatically larger in studies entailing high response conflict in comparison to those with low conflict. In fact, the alcohol effect on behavior was nonsignificant among low-conflict studies.

Building on observations from this meta-analysis and other data sets examining behavioral (Steele, Critchlow, & Liu, 1985) and emotional (Josephs & Steele, 1990; Steele & Josephs, 1988; Steele, Southwick, & Pagano, 1986) responses among intoxicated individuals, Steele and colleagues developed the alcohol myopia (AM) model (Steele & Josephs, 1990) that highlights alcohol's deleterious effects on attentional function to explain intoxicated behavior and emotion. The AM model suggests that alcohol influences behavior when conflicting cues simultaneously activate and inhibit behavior. Because of impaired attentional capacity, the inebriated individual is unable to attend to and encode all relevant information in the environment. Therefore, behavior is guided

Editor's Note. Michael Sayette served as the action editor for this article.—TBB

John J. Curtin and Bradley A. Fairchild, Department of Psychology, University of Wisconsin—Madison.

Correspondence concerning this article should be addressed to John J. Curtin, Department of Psychology, University of Wisconsin—Madison, 1202 West Johnson Street, Madison, Wisconsin 53706. E-mail: jjcurtin@facstaff.wisc.edu

instead by a more limited set of the most salient cues. Disinhibited behavior occurs when important inhibitory cues are not attended to because they are peripheral to more salient activating cues.

More recently, Vogel-Sprott, Fillmore, and their colleagues examined the role of response conflict in alcohol's effect on behavior in a go–stop paradigm that elicits incompatible activating and inhibiting response processes (Fillmore & Vogel-Sprott, 1999, 2000; Mulvihill et al., 1997). Across studies, alcohol intoxication did not alter task performance on trials that included only go signals. However, on trials in which conflicting go and stop signals were both presented, alcohol intoxication led to increased rate of inhibition errors. These researchers interpreted their findings to indicate that alcohol selectively interferes with specific cognitive processes important for inhibitory control of behavior in the presence of response conflict (Logan & Cowan, 1984).

These and other recent theoretical formulations (e.g., appraisal disruption theory; Sayette, 1993) have significant advanced understanding of intoxicated behavior. In particular, they direct researchers to closely examine the cognitive mechanisms, including attentional function, which are responsible for the adaptive resolution of response conflict. Importantly, recent cognitive neuroscience research on behavioral regulation, and on the attentional networks that support this regulation, now provide the necessary foundation to conduct systematic research designed to advance and refine these initial theoretical formulations about the cognitive–attentional mechanism(s) of action for alcohol's effect on behavior.

Attention and Response Conflict

Attention is a broad construct, and cognitive neuroscience research has identified diverse functions of the attentional system, including maintenance of an alert state, sensory orienting, and the executive function responsible for the control of cognitive operations (Posner, 1995; Posner & DiGirolamo, 1998). Research suggests that coordinated, but independent, neural systems are responsible for these different components of attentional function. For example, sensory orienting may result from activity in a posteriorly located attentional system. For visual stimuli, this *posterior sensory orienting system* includes neural structures such as the parietal lobe, the pulvinar, and the superior colliculus. This system is responsible for attentional engagement and switching and the initial processing amplification provided to stimuli in the focus of attention. In contrast, cognitive control processes associated with the recruitment and application of attentional resources to support goal-directed responding are accomplished in an *anterior executive attention system* that includes the prefrontal cortex (PFC), anterior cingulate cortex (ACC), and supplementary motor area (Posner & Petersen, 1990). This anterior attention system biases processing in favor of task-relevant stimuli and responses in order to establish contextually appropriate and adaptive stimulus–response mapping (MacDonald, Cohen, Stenger, & Carter, 2000). This cognitive control is required to address novel, complex, or otherwise difficult tasks. In particular, cognitive control is crucial when response conflict exists and adaptive response requires inhibition of habitual or prepotent responses that are not contextually appropriate (Braver et al., 1999; MacDonald et al., 2000).

Within this anterior executive attention system, further distinctions have been suggested between two main components of cognitive control: (a) an evaluative component, which is responsible for monitoring the need for control and signaling when adjustments in control are necessary, and (b) a regulative component, which is responsible for activation and implementation of control-related processes (Botvinick et al., 2001; Braver et al., 1999; MacDonald et al., 2000). The evaluative component is believed to operate by detecting response conflict, and a number of studies have demonstrated increased activation of ACC under conditions believed to involve such response conflict (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 2000; van Veen & Carter, 2002). Thus, activation of ACC signals the regulative component that more control is needed (Botvinick et al., 2001). Activation of the regulative component is believed to reflect actual implementation of top-down support for task-relevant processes and responses, allowing them to compete effectively against inappropriate ones when response conflict exists. A critical element of the implementation of regulative control is the active maintenance and utilization of context representations (including current goals, task instructions, and previously presented stimuli) in working memory to guide task-appropriate behavior; numerous studies have established a role of PFC in working memory function (Goldman-Rakic, 1992, 1996; Jonides, Schumacher, Smith, & Lauber, 1997). As such, regulative control has been closely associated with activation in PFC (Botvinick, et al., 2001; MacDonald et al., 2000).

This description of cognitive control suggests that these processes are particularly important to guide appropriate behavior under conditions or tasks that involve competition between prepotent and weaker response inclinations. As described earlier, situations characterized by such response conflict are precisely the conditions when alcohol has its most profound behavioral effects. This suggests that selective impairment of cognitive control processes in the anterior attention system may provide a mechanism through which alcohol affects behavior. The current project represents an initial step in a line of research designed to systematically examine alcohol's effect on specific components of anterior executive attention by using electrophysiological indices (e.g., ERPs) within laboratory response conflict paradigms that have advanced researchers' understanding of basic processes in cognitive control.

The Stroop Task

One classic experimental paradigm that involves response conflict is the Stroop task (MacCleod, 1991; Stroop, 1935). In the Stroop task, composite stimuli can vary independently on two stimulus attributes (script color and word meaning), resulting in three types of trials: (a) congruent (script color and word meaning match), (b) incongruent (color and word meaning mismatch), and (c) neutral trials (one attribute does not contain color information). Participants can be instructed to selectively attend to one attribute, resulting in separate color-naming and word-reading tasks. The robust Stroop interference effect refers to the increase in reaction time and error rate observed when participants are required to name the script color, which is incongruent with the word meaning.

Research suggests that the interference observed on incongruent, color-naming trials results from competition between independently processed stimulus–response (S-R) mappings: one initiated

by the task appropriate, script-color attribute and the second by the inappropriate, word-meaning attribute of this composite stimulus (Cohen, Dunbar, & McClelland, 1990; Duncan-Johnson & Kopell, 1981; MacCleod, 1991). Each of these S-R mappings is processed in parallel and converges on the same response output system. The incompatible response activation (i.e., response conflict) from the word-meaning delays (and in some instances overcoming) a correct response to the script color because of its relatively stronger S-R mapping strength for word reading based on previous learning.

Support for the necessary role of cognitive control in successful Stroop task performance has been offered by numerous neuroimaging studies that document increased activity in many of the neural structures composing the anterior executive attention system (Bench, Frith, Grasby, & Friston, 1993; Carter, Mintun, & Cohen, 1995; George et al., 1994; Leung, Skudlarski, Gatenby, Peterson, & Gore, 2000; Pardo, Pardo, Janer, & Raichle, 1990). In particular, these studies consistently observe relative increased ACC activation on incongruent color-naming trials. Moreover, increased Stroop interference, along with concurrent neuroimaging evidence of failures to recruit the neural structures responsible for cognitive control, has been documented among patients with disorders of the executive attention system (Bush et al., 1999; Carter, Mintun, Nichols, & Cohen, 1997).

Event-Related Potentials in the Stroop Task

ERP measures (see Fabiani, Gratton, & Coles, 2000, for a review) have also been invaluable in examining the cognitive mechanism responsible for the Stroop interference effect. For example, P3, a posterior/parietal-focused component of the ERP (Donchin, 1981; Johnson, 1988), has been used to localize the origin of the Stroop interference effect within the overall cognitive processing stream. The latency of the P3 component covaries with the duration of initial attentional orienting and target detection processes involved in stimulus evaluation and categorization, with the peak of this component indicating the termination of these evaluation processes (Duncan-Johnson, 1981; Duncan-Johnson & Donchin, 1982; McCarthy & Donchin, 1981). Moreover, the latency of this component is independent of the duration of response-related motor processes (Kutas, McCarthy, & Donchin, 1977; McCarthy & Donchin, 1981). Therefore, P3 latency can serve as a more specific measure of stimulus evaluation duration that is not contaminated with subsequent response conflict resolution related processes.

Duncan-Johnson and Kopell (1981) utilized P3 to evaluate whether the verbal response slowing during incongruent color-naming trials resulted from delayed stimulus evaluation or subsequent interference that was due to conflict during response selection and production. The typical Stroop interference effect, with response slowing on incongruent trials, was observed. However, P3 latency did not vary among congruent, neutral, and incongruent color-naming conditions, indicating that the source of the interference effect was subsequent to stimulus evaluation. This result has also been replicated using a different response modality (i.e., button press), suggesting that the interference that is due to response conflict is not dependent on any one specific S-R mapping (Ilan & Polich, 1999).

More recently, two frontal components of the ERP waveform have been observed that appear to be directly influenced by cognitive control processes in the anterior attention system that are responsible for adaptive performance of the Stroop task. Specifically, these components include a phasic negativity, which we refer to as *N450*, and a more tonic, negative slow wave, or *NSW*.

N450 is a phasic negative deflection of the ERP waveform with a fronto-central distribution that peaks between 400 and 500-ms poststimulus onset (Liotti, Woldorff, Perez, & Mayberg, 2000; Rebai, Bernard, & Petersen, 1997; West & Alain, 1999, 2000a, 2000b, 2000c). This negative deflection is reliably greater on incongruent trials relative to neutral trials and appears to covary with neural activity associated with the detection of response conflict (i.e., the evaluative component of cognitive control; West & Alain, 1999). Consistent with this interpretation, initial evidence suggests that the source of N450 may be ACC. Specifically, manipulation of the frequency of incongruent trials, which has been demonstrated to increase the activation of ACC (Carter et al., 2000), also affects the magnitude of N450, with N450 augmented concurrently with increased response conflict that results from the higher proportion of congruent trials (West & Alain, 2000b). Dipole source localization techniques have also suggested that this component is generated within ACC (Liotti et al., 2000).

More recently, West and Alain (1999) observed a second frontal ERP component, a NSW, which also appears to covary with processes important for adaptive Stroop task performance. NSW, measured at fronto-central scalp sites, can be most readily observed during the latter part of a trial epoch after the initial, more phasic, ERP components have resolved (West & Alain, 1999, 2000a). NSW is larger during incongruent trials than in neutral trials during the color-naming task. Given its more tonic time course across the trial, West and Alain (1999) have suggested that this component covaries with activation and implementation of conflict resolution processes (i.e., the regulative component of cognitive control). Consistent with theory on the neural substrates of these regulative processes reviewed earlier, West and Alain (2000c) speculated that the reversal of polarity of NSW between the fronto-polar region to the fronto-central region is compatible with activity of a neural generator located within the polar or dorsolateral region of the prefrontal cortex.

The Current Study

Prior research on alcohol and the Stroop task has not revealed consistent behavioral effects (Fillmore, Dixon, & Schweizer, 1999; Gustafson & Kallmen, 1990a, 1990b, 1990c; Lewis, Dustman, & Beck, 1969; Tarter, Jones, Simpson, & Vega, 1971). However, all of these studies involved blocked presentation of primarily or exclusively incongruent trials, and recent research has indicated that the demands placed on certain aspects of cognitive control are reduced when the relative proportion of incongruent trials is high (Carter et al., 2000). In addition, to our knowledge, no study has examined ERP indices of attentional function to investigate the cognitive mechanism of observed behavioral deficits when intoxicated. The current study examined alcohol's effect on task performance in a design that included equiprobable and intermixed congruent, neutral, and incongruent trials designed to significantly tax cognitive control function. Moreover, the measurement of specific ERP components, including parietal P3, frontal N450, and

Table 1
Drinking Behavior and History of Alcohol-Related Problems by Beverage Group

Measure	No alcohol		Alcohol	
	Man	*IR*	*Man*	*IR*
Age	21.5	1.8	22.0	3.0
Drinking frequency (occasions/week)	2.0	1.0	2.0	1.5
Drinking quantity (drinks/occasion)	4.0	2.0	5.0	5.0
Tolerance (drinks until "somewhat intoxicated")	4.0	2.0	3.0	2.5
SMAST score	0.0	1.0	1.0	2.0

Note. N = 48 for age, but 46 for all other measures because of incomplete questionnaire packets for 2 alcohol group participants. Mann-Whitney U tests (performed because parametric assumptions of normality and interval measurement were not satisfied) revealed no significant group differences at $\alpha = .05$. IR = interquartile range; SMAST = Short Michigan Alcohol Screening Test (Selzer, Vinokur, & van Rooijen, 1975).

NSW during Stroop task performance, permitted more direct examination of the underlying function of components of the posterior and anterior attention systems responsible for adaptive task performance. If, as predicted, alcohol intoxication produced task performance deficits, investigation of these sensitive ERP measures of the Stroop cognitive processing stream would allow us to localize the attentional impairment responsible for the dysregulated behavior when intoxicated.

It was predicted that intoxicated participants would evidence task performance deficits (increased response time and error rates) on trials that included conflict between incompatible responses, with adaptive performance requiring intact cognitive control function. Thus, behavioral deficits resulting from intoxication should be most apparent during incongruent color-naming trials. In addition, we suggest that these behavioral deficits result from underlying impairment in cognitive control functions rather than in earlier posterior stimulus-evaluation processes. Thus, we predicted that the frontal (anterior) ERP components, but not parietal (posterior) P3, would be reduced in this paradigm when participants were intoxicated.

Method

Participants

Forty-eight participants (24 women and 24 men) were recruited via research-assistant-initiated phone contact from a database of potential research participants that includes undergraduate introductory psychology students and individuals from the university community who have responded to e-mail or campus flyer advertisements about research opportunities. Inclusion criteria were as follows: at least 21 years old, self-reported normal color vision, recent experience with the dose of alcohol to be administered, and no history of alcohol-related problems[1] or a medical condition that might contraindicate alcohol use. Descriptive information on participants' age, drinking habits, and alcohol-related problems are provided in Table 1. Appropriate volunteers were scheduled and instructed to abstain from all drugs for at least 24 hr, and all food and beverages for at least 4 hr, prior to arrival for appointments. Participants received either monetary compensation ($10/hr) or course extra credit (2 points/hr) for their participation.

Procedure

Consent and screening. Upon arrival for the experiment, all participants were required to provide proof of legal drinking age and sign consent forms, approved by the Institutional Review Board, that included an agreement to remain at the research site until their blood-alcohol levels (BALs) were sufficiently low to permit safe release. They also completed a drinking and medical history questionnaire. All women completed a urine sample pregnancy test (QuickVue One-Step hCG; Quidel Corp., San Diego, CA), with a negative result required for further participation. Qualified participants were then asked to provide a predrink breath sample to verify an initial BAL of 0.00% (Alcosensor IV; Intoximeters, Inc., St. Louis, MO).

Beverage manipulation. Half of the male and half of the female participants were randomly assigned to the alcohol group, the remainder to the no-alcohol group. The alcohol group received a beverage consisting of fruit juice mixed with 100% ethyl alcohol in a 6:1 juice to alcohol ratio. They were accurately informed of their beverage condition and were told that the dose was roughly equivalent to 3–4 drinks in 1 hr for a 150-lb person. The dose required to produce the target peak BAL (0.080%) 30 min after completion of beverage consumption was computed for each participant by using software (Curtin, 2000; see the Appendix) developed for this purpose. Participants assigned to the no-alcohol group were advised as such[2] and received only a mixture of fruit juice in a volume equivalent to the total amount that would have been administered had they been in the alcohol condition. All beverages were evenly divided into two drinks, each consumed in 20 min, for a total drinking period of 40 min. The Stroop task began after a 15-min, postdrinking absorption period. Participants' postdrink BALs were assessed at three points during the experiment: (a) just prior to the start of the Stroop task, (b) during a break at the midpoint of the task (approximately 25 min postdrinking), and (c) immediately after completion of the Stroop task (approximately 35 min postdrinking).

[1] Participants were excluded from participation if they reported any history of substance use–related problems on a medical screening questionnaire. They were specifically queried about (a) formal treatment (including Alchoholics Anonymous) for a substance use disorder; (b) attempts (self-initiated or requested by family, friends, or treatment provider) to reduce substance use; (c) school, occupational, social, or legal problems related to their substance use; and (c) any allergic or other unusual reactions related to their substance use.

[2] The decision to use a no-alcohol, as opposed to a placebo, comparison group was a reasoned one. First, we believed that an initial demonstration of alcohol use impairing specific electrophysiological indices related to cognitive control would significantly advance our knowledge of the cognitive mechanisms responsible for altered behavior among drinkers, regardless of whether the source of the cognitive deficit was expectancy or pharmacology. Obviously, alcohol's expectancy and pharmacological effects are rarely parsed in the real world (i.e., individuals rarely believe they are drinking alcohol without receiving alcohol's pharmacological effects as well). Thus, the results further our understanding of the total effect of "naturalistic" alcohol use on important cognitive processes that may represent the mechanisms that produce intoxicated dysregulated behavior and that justify a more fine grained analysis of expectancy versus pharmacological contributions. Second, our ability to rule out global expectancy effects was strengthened by the prediction of differential alcohol effects across tasks and conditions. Although it was reasonable for participants to hold the expectation that alcohol could impair overall performance, it seems less probable that they would expect differential impairment across variations of each of these separate independent variables. Third, we were concerned about the possible effects of participants' suspicions about imperfect placebo manipulations on cognitive capacity that are necessary for regulation of task performance.

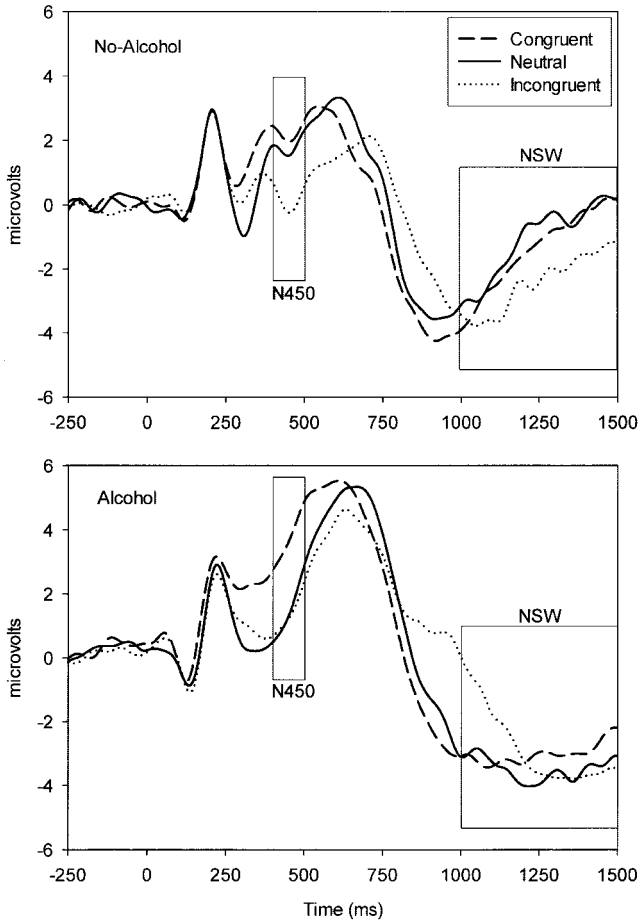

Figure 1. Event-related potential waveforms for the color-naming task from the average frontal/anterior scalp sites (Fz and FCz) by beverage and condition. Stroop stimulus onset is at 0 ms. The magnitude of the N450 component was indexed as the average response within the 100-ms window (indicated with left box) from 400 to 500 ms. The negative slow wave (NSW) was indexed as the average response within the 500-ms window (indicated with right box) from 1,000 ms to the end of the epoch.

Stroop task. Participants completed 432 individual trials of the Stroop task presented on a 21-in. (53.34-cm) monitor, with stimulus display, timing, and behavioral data collection, which were computer controlled, using DMDX software (Forster & Forster, in press). Each trial consisted of a stimulus word presented in colored script on a black background. Stimuli were presented for 500 ms with an intertrial interval varying from 1,500 ms to 2,500 ms. Participants were instructed to either read the stimulus word (word–read task [WRT]) or name the script color (color–name task [CNT]) as quickly as possible, with trials blocked by task type.

Within each task, individual trials were presented in one of three equiprobable conditions (congruent condition [CC], neutral condition [NC], or incongruent condition [IC]) with a random trial order. Color words and script colors included RED, BLUE, and GREEN. For CC trials, the color word and script color were congruent (e.g., the word RED presented in red script). For IC trials, the color word and script color were incongruent (e.g., RED presented in blue script). For NC trials during word reading, the three color words were presented in white script. For NC trials during color naming, the three script colors were presented on noncolor words (TOE, HAND, and WRIST).

Participants completed four contiguous blocks (54 trials per block) of each of the two tasks (i.e., CNT and WRT). Task instructions and a series of 10 practice trials were provided prior to the first CNT and WRT blocks. Brief (30-s) rest periods were provided after every two blocks, with a longer break after Block 4. During this break, the second breath sample for BAL estimation was obtained and new task instructions and practice trials were provided. Task order (CNT first vs. WRT first) was counterbalanced across participants. The entire task required approximately 20 min to complete.

Electroencephalographic (EEG) Recording

EEG activity was recorded using Ag-AgCl electrodes at Fz, Fcz, Cz, and Pz referenced to linked mastoids. Electrooculographic activity was recorded to monitor eye movements by using Ag-AgCl electrodes arranged bipolarly above and below the left eye. All electrodes were positioned according to the International 10-20 System of Electrode Placement (Jasper, 1958), and electrode impedances were kept below 5 K ohms. Neuroscan bioamplifiers were used to continuously digitize (1000 Hz sampling rate), amplify, and bandpass filter (0.15 Hz–200 Hz) the raw EEG signal. Offline data processing included epoching (500-ms prestimulus–1,500-ms poststimulus), lowpass filtering (10 Hz), eyeblink correction (Semlitsch, Anderer, Schuster, & Presslich, 1986), baseline correction, and artifact rejection (epochs with signal that exceeded ± 75 μV were rejected). Average ERP waveforms were calculated for correct trials at each scalp site within task and stimulus conditions.

Dependent Measures

Verbal response was detected and recorded online with a digital voice-activated switch integrated within the DMDX software. Response time (in milliseconds) for correct trials,[3] and response accuracy for all trials, were measured to index performance across task and stimulus conditions.

Analysis of the ERP waveform across scalp sites focused, a priori, on three specific components of the ERP response: (a) the magnitude and latency of the parietal P3, (b) the phasic negative frontal component, N450, and (c) the frontal NSW.

P3 magnitude was indexed as the mean response (in microvolts) at the Pz (midline parietal) scalp site in a 100-ms window surrounding the grand-average waveform peak across beverage conditions (peak at 350 ms, scoring window = 300 ms–400 ms). P3 latency was indexed as time (in milliseconds) to the peak voltage of this parietal ERP component relative to stimulus onset. The phasic frontal N450 component was indexed as the mean response (in microvolts) across Fz and Fcz scalp sites (midline frontal/fronto-central) in a 100-ms window from 400 ms–500 ms (see Figure 1, left dashed box).[4] N450 is a negative component, therefore, lower mean voltages represent a greater contribution of the N450 component to the ERP waveform. The frontal NSW was indexed as the mean response (in microvolts) across Fz and Fcz sites in a 500-ms window at the end of the sampling epoch (1,000 ms–1,500 ms; see Figure 1, right dashed box). Similar to N450, this is a negative component, therefore, lower voltages indicate a greater contribution of this slow wave to the overall ERP

[3] Response times less than 200 ms or greater than 2,000 ms were rejected as artifact (1.8% of trials). All other trials were included in reported analyses. However, results for all analyses are comparable if trials that exceed ± 3 standard deviations are reined in to the fence (i.e., set to ± 3 standard deviation of the mean; 1.2% of trials). Moreover, results for all analyses are comparable if all trials or only correct trials are analyzed.

[4] Analyses of N450 and the frontal NSW, including frontal scalp site (Fz vs. Fcz) as a variable, did not reveal any substantive differences in effects across these two sites. Therefore, reported analyses were collapsed across frontal scale sites. All reported significant effects from these overall analyses were consistent with individual analyses conducted separately at each frontal site.

waveform. Both P3 and N450 are phasic components of the ERP waveform (i.e., brief deflections), and scoring-window durations were chosen to reflect their punctate nature. In contrast, the broader scoring window for NSW was chosen to reflect its more tonic morphology.[5]

Results

Beverage Administration Manipulation Check

All participants registered a BAL of zero upon arrival at the laboratory. A Gender × Time analysis of variance (ANOVA) was performed on BAL for alcohol group participants across the three assessment times subsequent to the beverage manipulation (i.e., immediately pretask, midtask, and immediately posttask). No significant main effect of Gender or Gender × Time interaction was observed, indicating that the effect of the beverage manipulation was consistent across men and women. A main effect of time was observed, $F(2, 44) = 3.35$, $p = .048$, with participants in the alcohol group achieving mean (and standard deviation) BALs of 0.076 (0.018), 0.078 (0.021), and 0.071 (0.018) g/100 ml, respectively, across the three assessment times. Follow-up pairwise contrasts indicated that BAL was significantly lower posttask than midtask, $t(23) = 2.32$, $p = .030$. No other pairwise contrasts were significant.

Behavioral Effects

Analytic strategy. Analysis of behavioral data (error rate and response time) was accomplished within separate repeated measures ANOVAs, with beverage (no alcohol vs. alcohol) as a between-subjects variable and task (CNT vs. WRT) and condition (CC vs. NC vs. IC) as within-subject variables.[6] Huynh–Feldt corrected p values are reported for all within-subject effects involving the condition variable to correct for possible violations of sphericity. Partial eta squared effect size (ES) estimates[7] are reported when appropriate (i.e., when theoretically relevant questions focus on differential magnitude of effect).

Percentage of error rate. Percentage of error rate in the Stroop task is presented in the top panel of Figure 2. As expected, significant task, $F(1, 46) = 51.87$, $p < .001$, condition, $F(2, 92) = 57.73$, $p < .001$, and Task × Condition effects, $F(2, 92) = 48.08$, $p < .001$, were observed, with the interaction indicating that the condition effect was significantly greater during CNT (ES = 0.55) than WRT (ES = 0.07).

As predicted, a significant Beverage × Task × Condition interaction was observed, $F(2, 92) = 6.99$, $p = .007$. Follow-up analysis indicated that the Beverage × Condition effect was significant during CNT, $F(2, 92) = 5.76$, $p = .015$, ES = 0.111, but not during WRT, $F(2, 92) = 1.67$, $p = .197$, ES = 0.035, indicating that the magnitude of the beverage effect on error rate varied across conditions within CNT but not WRT. Further analysis of this Beverage × Condition effect within CNT revealed the predicted significant simple effect of beverage in the incongruent condition, $t(46) = 2.75$, $p = .009$, ES = 0.141, with a higher frequency of errors observed among intoxicated than nonintoxicated individuals. No significant beverage effect was observed in the other two color-naming conditions (mean ES = 0.067).

Response time. Response time for correct trials is presented in the bottom panel of Figure 2. As expected, significant task, $F(1, 46) = 295.25$, $p < .001$, condition, $F(2, 92) = 158.23$, $p < .001$, and Task × Condition effects, $F(2, 92) = 141.37$, $p < .001$, were observed, with the interaction indicating that the condition effect was significantly greater during CNT (ES = 0.78) than WRT (ES = 0.23).

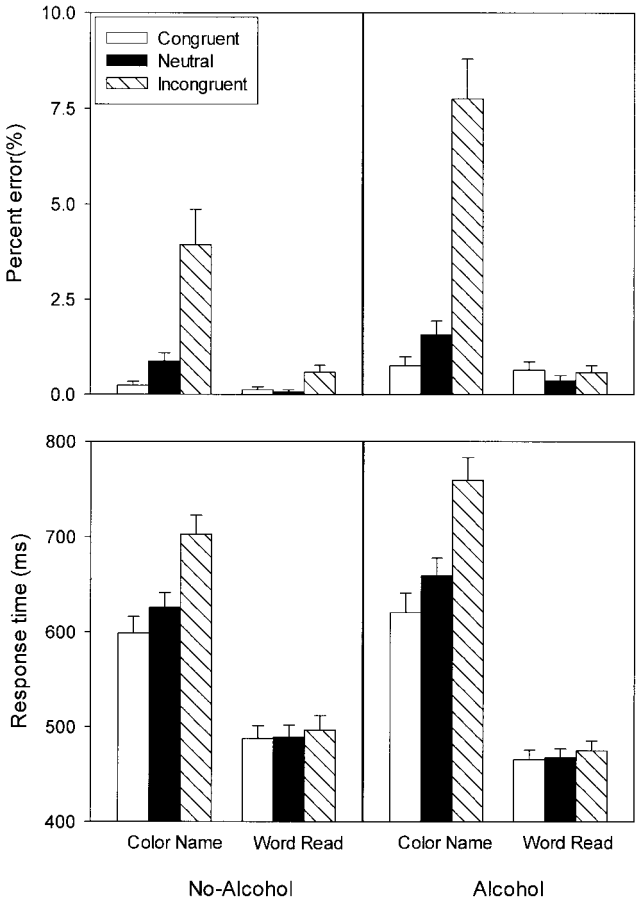

Figure 2. Stroop task accuracy and response time as a function of beverage, task, and condition. Error bars represent standard error.

As predicted, a significant Beverage × Task × Condition interaction was observed, $F(2, 92) = 3.39$, $p = .044$. Follow-up

[5] The broad 500-ms scoring window for NSW was chosen to reflect the tonic nature of this slow wave in comparison to more phasic ERP components such as P3 or N450. However, the effect of beverage on the NSW interference contrast remained significant ($p < .05$) when analysis was performed on mean frontal amplitude within a narrower scoring window (1,200 ms–1,500 ms).

[6] Gender and task order (CNT first vs. WRT first) were included in initial analyses of all behavioral and ERP measures. However, neither gender nor task order moderated (i.e., interacted with) critical significant omnibus interactions involving beverage for any of these dependent measures. Thus, all analyses were collapsed across these two between-subjects variables. With respect to task order, it is important to note that the absence of significant interactions with this variable supports the assertion that observed beverage effects were not the result of differential fatigue (or motivation) across time for intoxicated participants.

[7] Partial eta squared from ANOVA models was equivalent to R^2 from multiple regression models and indicates variance in the dependent variable accounted for by the independent variable.

Table 2
P3 Magnitude and Latency by Beverage Group

Beverage group	P3 magnitude (μvolts)						P3 latency (ms)					
	Congruent		Neutral		Incongruent		Congruent		Neutral		Incongruent	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
No alcohol	7.0	4.0	5.6	3.8	6.5	3.6	332	60	368	85	342	52
Alcohol	7.4	3.3	5.2	3.2	6.4	3.7	367	75	366	97	349	86
Overall	7.2	3.6	5.4	3.5	6.5	3.6	349	70	367	90	345	70

analysis indicated that the Beverage × Condition effect was significant during CNT, $F(2, 92) = 3.29$, $p = .049$, ES = 0.067, but not WRT, $F(2, 92) = 0.03$, $p = .962$, ES = 0.002, indicating that the magnitude of the beverage effect on response time varied across conditions within CNT but not WRT. Further analysis of this Beverage × Condition effect within the color-naming task revealed a trend toward the predicted simple effect of beverage in the incongruent condition, $t(46) = 1.84$, $p = .073$, ES = 0.068, with slower response times observed among intoxicated than nonintoxicated individuals when naming colors presented on incongruent words. No significant beverage effect was observed in the other two color-naming conditions (mean ES = 0.026).

ERP Measures

Analytic strategy. Analysis of ERPs (P3 latency and magnitude, N450, and NSW) during the color-naming task was accomplished in separate repeated measures ANOVAs, with beverage (no-alcohol vs. alcohol) as a between-subjects variable and condition (CC vs. NC vs. IC) as a within-subject variable.[8] Huynh–Feldt corrected *p* values are reported for all within-subject effects involving the condition variable. Main effects and interactions involving condition were decomposed into separate Stroop interference (IC vs. NC) and facilitation (CC vs. NC) contrasts.

Posterior/parietal P3 stimulus evaluation. Means (and standard deviations) for parietal P3 magnitude and latency are presented in Table 2. A significant condition main effect was observed for P3 magnitude, $F(2, 92) = 19.73$, $p < .001$. To follow-up this main effect, it was decomposed into single degree of freedom interference (IC vs. NC) and facilitation (CC vs. NC) contrasts. Both the interference, $F(1, 92) = 27.64$, $p < .001$, and the facilitation contrast, $F(1, 92) = 75.10$, $p < .001$, were significant, indicating that P3 magnitude was significantly greater during IC ($M = 6.5$, $SD = 3.6$) and CC ($M = 7.2$, $SD = 3.6$) trials relative to NC trials ($M = 5.4$, $SD = 3.5$). No significant main effect or interaction with beverage was observed for P3 magnitude. Moreover, no significant main effects or interactions were observed for P3 latency, with grand-average parietal P3 latency observed at 354-ms poststimulus onset. Thus, P3 analyses indicate that alcohol did not affect either the timing (latency) or the integrity (magnitude) of stimulus encoding and evaluation within the Stroop task.

Anterior/frontal N450. The scoring window for the frontal N450 is indicated by the left box over the frontal/anterior ERP waveforms in Figure 1. Consistent with other recent observations of this ERP component, a significant main effect of condition was observed, $F(2, 92) = 16.81$, $p < .001$, decomposition of the condition main effect revealed expected significant interference, $F(1, 92) = 5.37$, $p = .023$, and facilitation contrasts, $F(1, 92) = 11.86$, $p < .001$, with N450 increased during incongruent trials ($M = 0.7$, $SD = 4.5$) and decreased during congruent trials ($M = 3.0$, $SD = 4.9$) relative to the neutral condition ($M = 1.6$, $SD = 4.8$). As indicated previously, both N450 and NSW are negative components, therefore, lower scores represent greater contribution to the ERP waveform.

As predicted, the Beverage × Condition interaction was also significant, $F(2, 92) = 3.28$, $p = .042$. To further examine this interaction, simple effects tests of beverage were conducted on the magnitude of the interference and facilitation contrasts. As indicated previously in the description of the main effect of condition, N450 is increased during the response conflict engendered by Stroop interference on IC relative to NC trials. However, a significant beverage effect was observed on this interference contrast, $F(1, 92) = 4.07$, $p = .046$, which indicated that typical increase in N450 during response conflict on incongruent trials was reduced in intoxicated relative to sober individuals.

In addition, as indicated previously in the description of the main effect of condition, N450 is decreased during CC relative to NC trials. A significant beverage effect was also observed on this facilitation contrast, $F(1, 92) = 5.65$, $p = .020$, such that intoxicated participants evidenced smaller N450 magnitude on congruent trials relative to sober individuals, indicating relatively less activation of the evaluative component of cognitive control on these trials when intoxicated.

Anterior/frontal negative slow wave. The scoring window for the frontal NSW is depicted by the right box in Figure 1. A significant Beverage × Condition interaction was observed, $F(2, 92) = 3.58$, $p = .032$. To examine this interaction, simple beverage effect tests were conducted on interference and facilitation contrasts. A significant beverage effect was observed on the interference contrast, $F(1, 92) = 7.03$, $p = .009$, indicating that the relative size of the NSW for incongruent trials was reduced in intoxicated participants. The beverage effect was not significant for the facilitation contrast.

[8] Analysis of ERP data was limited to the color-naming task for two reasons. First, ERPs were examined to identify potential underlying cognitive deficits responsible for observed behavioral impairment, and no such impairment was exhibited in either sober or intoxicated participants during word reading. More important, the frontal ERP components (N450 and NSW) examined in the current project resulted from cognitive processes specific to the response conflict elicited by the color-naming task. No evidence of these components was observed (or expected) in the ERP waveform when participants performed the word-reading task.

Discussion

Participants in both beverage conditions displayed the typical Stroop interference effect, with increased response time and error rates in incongruent color-naming trials. However, the magnitude of the deficits displayed by intoxicated compared with sober participants is most interesting. Specifically, alcohol produced the largest increase in error rate and response time when color naming of incongruent stimuli was required. Intoxicated individuals did not display overall increased error rate and response slowing relative to sober controls. They did not display impairment of the stronger S-R mapped task (i.e., word reading), even when incompatible color information was presented (i.e., incongruent word-reading trials). Moreover, they did not display overall impairment in the execution of the weaker S-R mapped task (i.e., color naming). Instead, intoxicated individuals exhibited a specific deficit in the execution of the weaker S-R mapping only when it conflicted with the more highly trained mapping involved in word reading. Thus, intoxicated participants displayed selective impairment on the trials for which cognitive control was most critical.

In addition to establishing the specificity of the alcohol-induced dysregulation of behavior, the measurement of ERPs allowed for more direct examination of the underlying cognitive deficits responsible for this impaired behavior. ERP indices that tap function of the posterior and anterior attentional systems (Posner & DiGirolamo, 1998) required for adaptive performance of the Stroop task were measured and analyzed. Specifically, parietal P3 magnitude and latency provided an assessment of the integrity and timing of initial Stroop stimulus encoding and evaluation processes that were dependent on the posterior attention system. Subsequent to the completion of stimulus evaluation, frontal phasic N450 and tonic NSW provided an assessment of two important components of the anterior attentional system (i.e., evaluative and regulative components of cognitive control, respectively) that are necessary for adaptive behavior during response conflict.

Alcohol intoxication did not affect either the magnitude or the latency of parietal P3 during the Stroop color-naming task. Intoxicated participants displayed robust P3 magnitude to color words, which indicates that they were able to extract script-color information from Stroop stimuli in all conditions, including incongruent trials. Grand-average peak P3 latency across beverage and color-naming conditions was observed at 354-ms poststimulus onset, serving as a marker for the completion of the stimulus encoding and evaluation processes. Analysis of P3 latency suggested no delay in execution of these processes in intoxicated participants. Thus, it appears that the behavioral deficits during incongruent color-naming trials when participants were intoxicated were not the result of a delay in or a failure to adequately encode and evaluate the script color.

In addition to parietal P3, two components of the frontal ERP waveform during Stroop task performance, N450 and NSW, were also examined. Current results and other recent empirical studies reviewed earlier strongly suggest that N450 covaries with the neural signal that response conflict has been detected and cognitive control is required. In essence, this phasic ERP component appears sensitive to the signal to switch from an automatic processing strategy and bring online more controlled, executive attentional processes to aid response selection and execution. In addition to the previously reviewed evidence about its sensitivity to Stroop condition, and its possible generation in ACC, the latency of the N450 in the current project suggests that it co-occurs with the detection of response conflict and is observed subsequent to the completion of initial stimulus evaluation (as indicated by parietal P3) and prior to the detection of behavioral response (mean verbal response for Stoop conditions varied from 609 ms to 730 ms for congruent and incongruent trials, respectively).

Available evidence suggests that the tonic NSW can serve as a neuroelectric marker of the activation of regulative cognitive control strategies that allow representations of task instructions and the current context in working memory to bias processing in favor of behavior that is most adaptive in this context. Research reviewed earlier indicated that NSW is sensitive to Stroop condition, and its topography suggests a generator in the prefrontal cortex, a neural structure implicated in working memory function. Moreover, the relatively tonic nature of NSW is expected because information such as task instructions must be maintained in working memory over an extended period of time. Its timing, subsequent to detection of response conflict, also fits with a process that is proposed to aid response selection across trials in situations in which stimulus-driven automatic processing based solely on the strength of S-R mappings is not adequate for adaptive behavioral response.

Alcohol significantly reduced the magnitude of both N450 and NSW. With respect to N450, qualitative examination of intoxicated individuals' ERP waveforms failed to reveal any evidence of this phasic (i.e., brief deflection) neuroelectric signal, which covaries with detection of response conflict and results in recruitment of controlled-processing regulative control strategies for adaptive resolution of this conflict. Furthermore, the NSW observed on incongruent color-naming stimuli in sober participants was also significantly attenuated in participants who received alcohol, suggesting a reduced contribution of regulative control strategies to intoxicated participants' task performance. Regulative cognitive control is required to bias processing in favor of adaptive response based on the current context. Within the framework provided by basic cognitive control research, observations of alcohol's effect on these frontal ERP components suggest a parsimonious account of the cognitive mechanism underlying the observed behavior impairment. Because of deficient cognitive control processes resulting from alcohol intoxication, individuals who received alcohol had specific difficulty performing the Stroop task in the condition in which cognitive control was critical; that is, when execution of a weaker S-R mapping that conflicts with a relatively stronger S-R mapping is required. These cognitive control deficits suggest that across situations, intoxicated performance will be stimulus driven, with the context-independent strength of S-R association controlling behavior, regardless of whether that strongest mapped response is consistent with current goals.

Limitations and Future Directions

Pharmacology versus expectancy. Interpretations such as those provided in the preceding paragraphs imply a largely neuropharmacological account of the mechanisms through which alcohol affects cognitive control and behavior. However, because the current study did not include a placebo group, we cannot entirely rule out alcohol expectancy as a potential cause of the observed beverage group differences. In our view, it appears

unlikely that alcohol expectancy would have produced the observed patterns of differential task performance and brain electrophysiology across tasks and conditions, but expectancy effects can produce complex patterns of responding in behavioral tasks (e.g., Linnoila, Stapleton, Lister, Guthrie, & Eckardt, 1986). For example, the expectancy that alcohol will slow responding has led to compensatory speeding of response time at the cost of decreased task accuracy in previous investigations (e.g., Linnoila et al., 1986; Mitchell, 1985). However, response time and task accuracy data from the current study do not support the notion of a speed–accuracy trade-off, even if we allow that intoxicated participants tried to compensate for a highly specific expectancy that alcohol would only slow performance on incongruent color-naming trials. Alcohol group participants displayed decreased task accuracy even as their response time slowed in the critical incongruent color-naming condition. Similarly, the selective effects of alcohol on ERP indices of processes related to response selection (N450 and NSW), but not stimulus evaluation (i.e., P3), are difficult to explain by expectancy set. Regardless, future research in this area should use an additional placebo control group to provide more definitive support for the pharmacological explanation offered here.

Response conflict. Results from the current project provide additional evidence that intoxicated individuals have difficulty with adaptive resolution of response conflict. However, many important questions about the types of conflict that are sensitive to alcohol effects remain. For example, conflict potentially exists at two stages in the processing stream for incongruent stimuli in the Stroop paradigm (Kornblum, Hasbroucq, & Osman, 1990; Zhang, Zhang, & Kornblum, 1999). First, there is early stimulus evaluation conflict at the sensory processing stage, which requires the individual to encode simultaneously script color and incompatible word-meaning information. Subsequent to stimulus encoding and evaluation, response conflict emerges when script-color and word-meaning inputs concurrently activate incompatible response outputs. ERP results from the current study provide initial evidence that early sensory processing stages may remain intact despite stimulus evaluation conflict, with sizable deficits in performance resulting instead from later response conflict at the response selection stage. Future research involving direct manipulation is needed to systematically examine the conflict construct across the various stages of processing and the impact of acute alcohol intoxication on conflict in this processing stream.

It is also worthwhile to consider whether the cognitive mechanisms that control behavior in the presence of behavioral response conflict can also account for alterations in emotional response. First, it is important to note that much of the real-world "behavioral conflict" that has been observed to be altered by alcohol (e.g., aggression and sexual behavior) involves emotional and motivational components that may not have been well represented in the more "cognitive" conflict engendered by Stroop interference. Interestingly, research reviews (Cappell & Greeley, 1987; Greeley & Oei, 1999) on alcohol's effect on emotion have concluded that support for the tension-reducing effects of alcohol is most consistently obtained in studies that examine affective response within approach–avoidance conflict paradigms that simultaneously activate competing appetitive and aversive motivations systems. In fact, Brown and colleagues have generated experimental evidence in animals that suggests that alcohol selectively reduces the weaker motivational drive in these affective conflict paradigms (Brown, Mansfield, & Skurdal, 1980). Moreover, recent theory and research with humans have suggested that alterations in emotional response evident during intoxication may be mediated by alcohol-induced impairments in attentional processes (Curtin et al., 1998; Curtin et al., 2001; A. Lang, Patrick, & Stritzke, 1999; Steele & Josephs, 1988). If neural structures responsible for cognitive control of behavior are also recruited to address conflict between primary motivational–emotion systems (Gray & McNaughton, 2000; P. Lang, Bradley & Cuthbert, 1997), then alcohol-induced deficits in this anterior executive attentional system may underlie both behavioral and emotional consequences of alcohol use. Consistent with this, recent neuroscience evidence suggests that ACC may be divided into subregions that respond differentially to cognitive versus emotional inputs (Bush, Luu, & Posner, 2000).

Attention. Current ERP results suggest the construct of attention, as utilized in theories such as the alcohol myopia model, needs to be updated to incorporate recent basic cognitive and cognitive neuroscience research. Specifically, the contrast of robust P3 magnitude and latency with reduced N450 and NSW for incongruent color-naming trials when intoxicated suggests the need for revision of the singular attention construct. Theory and data that suggest attention is not a unitary construct have existed for many years (Navon & Gopher, 1979; Wickens, 1984). Moreover, current cognitive neuroscience research draws important distinctions between the attentional functions of the posterior sensory orienting system and the anterior executive system responsible for cognitive control, with further distinctions possible among subcomponents of the executive attention system (e.g., evaluative vs. regulative cognitive control). ERP results from the current study suggest that intoxicated dysregulated behavior does not result from a failure to attend to and encode information within the environment (i.e., deficit in posterior sensory orienting), but instead from a disruption of cognitive control within the anterior executive attention system. By anchoring theorizing about the cognitive mechanisms responsible for alcohol effects to current cognitive neuroscience research, researchers can achieve a greater level of specificity about the cognitive processes that are disrupted, the neural systems underlying these effects, and the likely behavioral–emotional consequences of this cognitive impairment. Moreover, researchers can benefit from the rapid advances in cognitive neuroscience resulting from new technologies for noninvasive investigation of brain function (D'Esposito, Zarahn, & Aguirre, 1999).

Specific components of cognitive control. Current cognitive neuroscience theories distinguish between at least two important components of cognitive control: (a) an evaluative component, responsible for monitoring the need for control and signaling when adjustments in control are necessary, and (b) a regulative component, responsible for activation and implementation of control-related processes. The current project measured two ERP indices—N450 and NSW—that recent research has suggested covary with the evaluative and regulative components of cognitive control, respectively. Alcohol attenuated both of these ERP components, suggesting that impairment in both evaluative and regulative cognitive control may underlay the behavioral deficits observed among intoxicated participants in incongruent color-naming trials. However, activation of regulative control processes in the current

paradigm required intact evaluative processes. Thus, these two components were inextricably linked in the current paradigm.

To disentangle alcohol's effect on these two components of cognitive control, future research must utilize experimental paradigms in which the relative contribution of one (or both) of these components can be independently manipulated. Such paradigms have already been developed and utilized in basic research on cognitive control and are readily available to advance researchers' understanding of the specificity of alcohol's effect on individual components of cognitive control (Carter et al., 2000; MacDonald et al., 2000). In addition, future research should examine the specificity of alcohol effects by using other electrophysiological indices with well-established neural generators in structures associated with cognitive control processes (e.g., error-related negativity; Gehring, Goss, Coles, Meyer, & Donchin, 1993). Use of ERP measures with well-validated neural sources will reduce some of the inherent circularity involved in validating an ERP index through its association with behavioral effects, and subsequently, this ERP component will be used to explain beverage effects on behavior. Moreover, research that more directly examines alcohol's effects on neural structures implicated in cognitive control can provide relatively independent and convergent tests of the those offered here.

Individual differences. Sample size and sample composition in the current study preclude potentially interesting examination of individual differences in the cognitive mechanisms highlighted in this project. In other research, for example, individual differences in baseline executive cognitive function (Finn et al., 1999; Giancola, 2000) have been identified as an important moderator of alcohol's effect on various forms of behavioral dysregulation. Similarly, individual differences in the sensitivity of these neural cognitive control systems to alcohol may underlie variation in one motivational pathway leading to the development of alcohol use disorders (Pihl & Peterson, 1995). Future research examining cognitive control processes should systematically sample for heterogeneity on individual differences such as family history of alcoholism, baseline cognitive control function, and disinhibitory personality traits to account for variance in the behavioral effects of alcohol and to advance understanding about the role of these mechanisms in the etiology and maintenance of alcohol use disorders (Sher, Trull, Bartholow, & Vieth, 1999).

Summary

Alcohol intoxication selectively disrupted execution of a relatively weak S-R mapped behavior (color naming) only when it conflicted with a more strongly mapped response (word reading) in the Stroop task. Robust P3 magnitude and latency across beverage groups indicated that alcohol did not interfere with encoding and evaluation of color information. Instead, reduced N450 and NSW among intoxicated participants suggested that their behavioral impairment resulted from failure in cognitive control function necessary to adaptively resolve response conflict. Thus, intoxicated individuals' behavior was stimulus driven rather then influenced by top-down cognitive control that serves to bias processing in favor of task/goal-relevant responses. In general, such stimulus-driven responding will produce inappropriate behavior in situations characterized by response conflict in which adaptive resolution of this conflict requires execution of relatively weaker S-R mapped behavior. Results from the current study demonstrate the utility of incorporating current theory and methods from cognitive neuroscience research when theorizing about mechanisms for intoxicated behavior. Moreover, the results may have important implications for understanding mechanisms underlying the etiology and maintenance of alcohol use disorders. To the degree that drinking behavior is a motivationally prepotent and well-learned response for individuals with alcohol use disorders, and drinking contexts often involve conflict for the dependent user (Breiner, Stritzke, & Lang, 1999), deficits in cognitive control that result from chronic use or that represent premorbid individual differences may interfere with the ability to inhibit drinking behavior in favor of more adaptive alternative responses. Finally, as researchers' understanding of alcohol's effects on these cognitive mechanisms advance, alcohol challenge paradigms may offer experimental psychopathologists an attractive laboratory analogue model to directly manipulate cognitive control processes in order to examine the contribution of these processes to externalizing disorders and other forms of psychopathology (Fillmore & Vogel-Sprott, 1999; Krueger et al., 2002; Patrick & Lang, 1999).

References

Bench, C. J., Frith, C. D., Grasby, P. M., & Friston, K. J. (1993). Investigations of the functional anatomy of attention using the Stroop test. *Neuropsychologia, 31,* 907–922.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108,* 624–652.

Botvinick, M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature, 402,* 179–181.

Braver, T. S., Barch, D. M., & Cohen, J. D. (1999). Cognition and control in schizophrenia: A computational model of dopamine and prefrontal function. *Biological Psychiatry, 46,* 312–328.

Breiner, M. J., Stritzke, W. G. K., & Lang, A. R. (1999). Approaching avoidance: A step essential to the understanding of craving. *Alcohol Research and Health, 23,* 197–206.

Brown, J. S., Mansfield, J. G., & Skurdal, A. J. (1980). An interference–reduction theory of the effects of ethanol on conflict behavior. *Physiological Psychology, 8,* 423–432.

Burian, S. E., Liguori, A., & Robinson, J. H. (2002). Effects of alcohol on risk-taking during simulated driving. *Human Psychopharmacology Clinical and Experimental, 17,* 141–150.

Bush, G., Frazier, J. A., Rauch, S. L., Seidman, L. J., Whalen, P. J., Jenike, M. A., et al. (1999). Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder revealed by *f*MRI and the Counting Stroop. *Biological Psychiatry, 45,* 1542–1552.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4,* 215–222.

Bushman, B. J., & Cooper, H. M. (1990). Effects of alcohol on human aggression: An integrative research review. *Psychological Bulletin, 107,* 341–354.

Cappell, H., & Greeley, J. (1987). Alcohol and tension reduction: An update on research and theory. In H. Blane & K. Leonard (Eds.), *Psychological theories of drinking and alcoholism* (pp. 15–54). New York: Guilford Press.

Carter, C. S., Botvinick, M. M., & Cohen, J. D. (1999). The contribution of the anterior cingulate cortex to executive processes in cognition. *Reviews in the Neurosciences, 10,* 49–57.

Carter, C. S., Macdonald, A. M., Botvinick, M., Ross, L. L., Stenger, V. A.,

Noll, D., & Cohen, J. D. (2000). Parsing executive processes: Strategic vs. evaluative functions of the anterior cingulate cortex. *Proceedings of the National Academy of Sciences of the United States of America, 97,* 1944–1948.

Carter, C. S., Mintun, M., & Cohen, J. (1995). Interference and facilitation effects during selective attention: An H215O PET study of Stroop task performance. *Neuroimage, 2,* 264–272.

Carter, C. S., Mintun, M., Nichols, T., & Cohen, J. D. (1997). Anterior cingulate gyrus dysfunction and selective attention deficits in schizophrenia: [^{15}O]H_2O PET study during single-trial Stroop task performance. *American Journal of Psychiatry, 154,* 1670–1675.

Casbon, T. C., Curtin, J. J., Lang, A. R., & Patrick, C. J. (in press). Deleterious effects of alcohol intoxication: Diminished cognitive control and its behavioral consequences. *Journal of Abnormal Psycyhology.*

Caudill, B. D., Wilson, G. T., & Abrams, D. B. (1987). Alcohol and self-disclosure: Analyses of interpersonal behavior in male and female social drinkers. *Journal of Studies on Alcohol, 48,* 401–409.

Cohen, J. D., Dunbar, K., & McClelland, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review, 97,* 332–361.

Curtin, J. J. (2000). [BAL calculator]. *Unpublished computer program.*

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

Curtin, J. J., Patrick, C. J., Lang, A. R., Cacioppo, J. T., & Birbaumer, N. (2001). Alcohol affects emotion through cognition. *Psychological Science, 12,* 527–531.

D'Esposito, M., Zarahn, E., & Aguirre, G. K. (1999). Event-related functional MRI: Implications for cognitive psychology. *Psychological Bulletin, 125,* 155–164.

Donchin, E. (1981). Surprise . . . Surprise? *Psychophysiology, 18,* 493–513.

Duncan-Johnson, C. C. (1981). P300 latency: A new metric of information processing. *Psychophysiology, 18,* 207–215.

Duncan-Johnson, C. C., & Donchin, E. (1982). The P300 component of the event-related brain potential as an index of information processing. *Biological Psychology, 14,* 1–52.

Duncan-Johnson, C. C., & Kopell, B. S. (1981). The Stroop effect: Brain potentials localize the source of interference. *Science, 214,* 938–940.

Fabiani, M., Gratton, G., & Coles, M. G. H. (2000). Event related brain potentials. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 53–84). New York: Cambridge University Press.

Fillmore, M. T., Dixon, M. J., & Schweizer, T. A. (1999). Alcohol affects processing of ignored stimuli in a negative priming paradigm. *Journal of Studies on Alcohol, 61,* 571–578.

Fillmore, M. T., & Vogel-Sprott, M. (1999). An alcohol model of impaired inhibitory control and its treatment in humans. *Experimental and Clinical Psychopharmacology, 7,* 49–55.

Fillmore, M. T., & Vogel-Sprott, M. (2000). Response inhibition under alcohol: Effects of cognitive and motivational conflict. *Journal of Studies on Alcohol, 61,* 239–246.

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.

Forster, K. I., & Forster, J. C. (2003). DMDX: A Windows display program with millisecond accuracy. *Behavior Research Methods, Instruments, & Computers, 35,* 116–124.

Gehring, W. J., Goss, B., Coles, M. G., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science, 4,* 385–390.

George, M. S., Ketter, T. A., Parekh, P. I., Rosinsky, N., Ring, H., & Casey, B. J. (1994). Regional brain activity when selecting a response despite interference: An H215O PET study of the Stroop and Emotional Stroop. *Human Brain Mapping, 1,* 194–209.

Giancola, P. (2000). Executive functioning: A conceptual framework for alcohol-related aggression. *Experimental and Clinical Psychopharmacology, 8,* 576–597.

Goldman-Rakic, P. S. (1992). Working memory and the mind. *Scientific American, 267,* 110–117.

Goldman-Rakic, P. S. (1996). The prefrontal landscape: Implications of functional architecture for understanding human mentation and the central executive. *Philosophical Transactions of the Royal Society of London, Series B, 351,* 1445–1453.

Gray, J. A., & McNaughton, N. (2000). *The neuropsychology of fear and anxiety.* New York: Oxford University Press.

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

Gustafson, R., & Kallmen, H. (1990a). Alcohol and the compensation hypothesis: A test with cognitive and psychomotor tasks. *Perceptual & Motor Skills, 71,* 1367–1374.

Gustafson, R., & Kallmen, H. (1990b). Effects of alcohol on cognitive performance measured with Stroop's Color Word Test. *Perceptual & Motor Skills, 71,* 99–105.

Gustafson, R., & Kallmen, H. (1990c). Effects of alcohol on prolonged cognitive performance measured with Stroop's Color Word Test. *Psychological Reports, 67,* 643–650.

Ilan, A. B., & Polich, J. (1999). P300 and response time from a manual Stroop task. *Clinical Neurophysiology, 110,* 367.

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

Johnson, R., Jr. (1988). The amplitude of the P300 component of the event-related potential: Review and synthesis. In P. K. Ackles, J. R. Jennings, & M. G. H. Coles (Eds.), *Advances in psychophysiology* (Vol. 3, pp. 69–138). Greenwich, CT: JAI Press.

Jonides, J., Schumacher, E. H., Smith, E. E., & Lauber, E. J. (1997). Verbal working memory load affects regional brain activation as measured by PET. *Journal of Cognitive Neuroscience, 9,* 462–475.

Josephs, R., & Steele, C. (1990). The two faces of alcohol myopia: Attentional mediation of psychological stress. *Journal of Abnormal Psychology, 99,* 115–126.

Kornblum, S., Hasbroucq, T., & Osman, A. (1990). Dimensional overlap: Cognitive basis for stimulus–response compatibility: A model and taxonomy. *Psychological Review, 97,* 253–270.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111,* 411–424.

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

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

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). Motivated attention: Affect, activation, and action. In P. J. Lang & R. F. Simons (Eds.), *Attention and orienting: Sensory and motivational processes* (pp. 97–135). Hillsdale, NJ: Erlbaum.

Leung, H. C., Skudlarski, P., Gatenby, J. C., Peterson, B. S., & Gore, J. C. (2000). An event-related functional MRI study of the Stroop color word interference task. *Cerebral Cortex, 10*(6), 552–560.

Lewis, E. G., Dustman, R. E., & Beck, E. C. (1969). The effect of alcohol

on sensory phenomenon and cognitive and motor tasks. *Quarterly Journal of Studies on Alcohol, 30,* 618–633.

Linnoila, M., Stapleton, J., Lister, R., Guthrie, S., & Eckardt, M. (1986). Effects of alcohol on accident risk. *Pathologist, 40,* 36–41.

Liotti, M., Woldorff, M. G., Perez, R., & Mayberg, H. S. (2000). An ERP study of the temporal course of the Stroop color–word interference effect. *Neuropsychologia, 38,* 701–711.

Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review, 91,* 295–327.

MacCleod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin, 109,* 163–203.

MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000, June 9). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science, 288,* 1835–1838.

McCarthy, G., & Donchin, E. (1981). A metric for thought: A comparison of P300 latency and reaction time. *Science, 211,* 77–80.

Mitchell, M. C. (1985). Alcohol-induced impairment of the central nervous system function: Behavioral skills involved in driving. *Journal of Studies on Alcohol* (Suppl. 10), 109–116.

Morris, A. B., & Albery, I. P. (2001). Alcohol consumption and HIV risk behaviours: Integrating the theories of alcohol myopia and outcome-expectancies. *Addiction Research and Theory, 9,* 73–86.

Mulvihill, L. E., Skilling, T. A., & Vogel-Sprott, M. (1997). Alcohol and the ability to inhibit behavior in men and women. *Journal of Studies on Alcohol, 58,* 600–605.

Navon, D., & Gopher, D. (1979). On the economy of the human-processing system. *Psychological Review, 86,* 214–255.

Pardo, J. V., Pardo, P., Janer, K. W., & Raichle, M. E. (1990). The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proceedings of the National Academy of Sciences, 87,* 256–259.

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

Pihl, R. O., & Peterson, J. B. (1995). Alcoholism: The role of different motivational systems. *Journal of Psychiatry and Neuroscience, 20,* 372–396.

Posner, M. I. (1995). Attention in cognitive neuroscience: An overview. In M. S. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 615–624). Cambridge, MA: MIT Press.

Posner, M. I., & DiGirolamo, G. J. (1998). Executive attention: Conflict, target detection, and cognitive control. In R. Parasuraman (Ed.), *The attentive brain* (pp. 401–423). Cambridge, MA: MIT Press.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13,* 25–42.

Rebai, M., Bernard, C., & Lannou, J. (1997). The Stroop's test evokes a negative brain potential, the N400. *International Journal of Neuroscience, 91,* 85–94.

Rohrberg, R. G., & Sousa-Poza, J. F. (1976). Alcohol, field dependence, and dyadic self-disclosure. *Psychological Reports, 39,* 1151–1161.

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

Selzer, M. L., Vinokur, A., & van Rooijen, L. (1975). A self-administered Short Michigan Alcoholism Screening Test (SMAST). *Journal of Studies on Alcohol, 36,* 117–126.

Semlitsch, H. V., Anderer, P., Schuster, P., & Presslich, O. (1986). A solution for reliable and valid reduction of ocular artifacts applied to the P300 ERP. *Psychophysiology, 23,* 695–703.

Sher, K. J., Trull, T. J., Bartholow, B. D., & Vieth, A. (1999). Personality and alcoholism: Issues, methods, and etiological processes. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism: The Guilford substance abuse series* (2nd ed., pp. 54–105). Guilford Press.

Steele, C. M., Critchlow, B., & Liu, T. J. (1985). Alcohol and social behavior: II. The helpful drunkard. *Journal of Personality and Social Psychology, 48,* 35–46.

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

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

Steele, C. M., & Southwick, L. (1985). Alcohol and social behavior: I. The psychology of drunken excess. *Journal of Personality and Social Psychology, 48,* 18–34.

Steele, C. M., Southwick, L., & Pagano, R. (1986). Drinking your troubles away: The role of activity in mediating alcohol's reduction of psychological stress. *Journal of Abnormal Psychology, 95,* 173–180.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18,* 643–662.

Tarter, R. E., Jones, B. M., Simpson, C. D., & Vega, A. (1971). Effects of task complexity and practice on performance during acute alcohol intoxication. *Perceptual and Motor Skills, 33,* 307–318.

van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 14,* 593–602.

Watson, P. E. (1989). Total body water and blood alcohol levels: Updating the fundamentals. In K. Crow & R. Batt (Eds.), *Human metabolism of alcohol: Vol. 1. Pharmacokinetics, medicolegal aspects, and general interest* (pp. 41–58). Boca Raton, FL: CRC Press.

West, R., & Alain, C. (1999). Event-related neural activity associated with the Stroop task. *Cognitive Brain Research, 8,* 157–164.

West, R., & Alain, C. (2000a). Age-related decline in inhibitory control contributes to the increased Stroop effect observed in older adults. *Psychophysiology, 37,* 179–189.

West, R., & Alain, C. (2000b). Effects of task context and fluctuations of attention on neural activity supporting performance of the Stroop task. *Brain Research, 873,* 102–111.

West, R., & Alain, C. (2000c). Evidence for the transient nature of a neural system supporting goal-directed action. *Cerebral Cortex, 10,* 748–752.

Wickens, C. D. (1984). Processing resources in attention. In R. Parasuraman & D. R. Davies (Eds.), *Varieties of attention* (pp. 63–102). Orlando, FL: Academic Press.

Zhang, H., Zhang, J., & Kornblum, S. (1999). A parallel distributed processing model of stimulus–stimulus and stimulus–response compatibility. *Cognitive Psychology, 38,* 386–432.

Appendix

Procedure Used to Determine Alcohol Dosage

The procedure used to determine alcohol dosage in the present study was developed by using formulae available from Watson (1989). It is predicated on the assumption that in order to reach a target blood-alcohol level (BAL), the alcohol dose to be administered is a function of the participant's height, weight, age, gender, total body water (TBW); duration of the drinking period (DDP); time to peak BAL (TPB); and alcohol metabolism rate (MR). More specifically,

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

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

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

and

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

Received August 15, 2002
Revision received December 20, 2002
Accepted January 30, 2003 ■