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Stereotype Activation and Control of Race Bias: Cognitive Control of Inhibition and Its Impairment by Alcohol

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Two experiments tested the hypothesis that alcohol increases race-biased responding via impairment of self-regulatory cognitive control. Participants consumed either a placebo or alcohol and then made speeded responses to stereotypic trait words presented after White and Black face primes while behavioral and event-related brain potential (ERP) data were recorded. Alcohol did not affect stereotype activation in either experiment. Experiment 2 showed that alcohol significantly impaired the ability to inhibit race-biased responses but did not reliably influence control of counterstereotypic responses. This disinhibition appears driven by impairment of regulative cognitive control, as indexed by amplitude of the negative slow wave ERP component. These findings suggest that controlling racial bias can be a function of effective implementation of basic self-regulatory processes in addition to the motivational processes identified in other research.

Keywords: alcohol, stereotypes, response inhibition, cognitive control, event-related potentials

Within the social cognition literature, few topics have received as much attention in the past 20 years as stereotyping and prejudice (e.g., see Bodenhausen & Macrae, 1998; Fiske, 1998). Recently, research efforts have been focused on explicating the processes associated with control of prejudice-related responses. Much of this work has centered on motivational (e.g., Monteith, 1993; Monteith, Sherman, & Devine, 1998; Thompson, Roman, Moskowitz, Chaiken, & Bargh, 1994) and individual difference factors (e.g., Devine, 1989; Monteith & Walters, 1998) assumed to determine whether stereotype activation will result in biased behavior (see Devine & Monteith, 1999; Monteith & Voils, 2001). This article takes a different approach by focusing on cognitive control processes that relate to successful behavioral regulation more generally and examining their role in controlling race bias (see also Amodio et al., 2004; Payne, 2001). Given alcohol's theorized impairment of behavioral inhibition (e.g., Fillmore & Vogel-Sprott, 1999, 2000), alcohol can serve as a useful tool for examining the role of inhibitory processes in this context. The purpose of this article, therefore, is to test a model of alcohol-related impairment of regulatory cognitive control and its effects on stereotype activation and inhibition of race bias.

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Racial Bias, Cognitive Control, and Behavioral Regulation

Activated stereotypes are known to bias judgment and behavior in a number of stereotype-consistent ways (see Wheeler & Petty, 2001). For example, activation of the stereotype for Blacks has been shown to increase ratings of the hostility of others' behavior (e.g., Devine, 1989), to provoke more hostile reactions among participants themselves (Bargh, Chen, & Burrows, 1996), and to elicit more hostile responses from interaction partners (Chen & Bargh, 1997). Current models of prejudice control stress that such biased responses can be resisted given sufficient motivation to respond without prejudice (e.g., Dunton & Fazio, 1997; Monteith & Voils, 2001).

In addition to these conscious motivational processes, specific neural processes associated with behavioral regulation that unfold extremely quickly and operate largely outside of conscious awareness also may determine whether activated stereotypes will result in biased behaviors. Inhibition of biased behavior can be considered part of a more general skill set associated with effective self-regulation, often requiring implementation of top-down control over well-learned responses in favor of other, context-appropriate responses (e.g., MacDonald, Cohen, Stenger, & Carter, 2000). This process, generally known as response conflict, is exemplified by interactions with members of stereotyped groups in which biased responses must be replaced with unbiased ones (Lambert et al., 2003).

Current theories in cognitive neuroscience posit two independent components of cognitive control that work in concert to ensure adaptive responding. First, an evaluative *conflict-detection system* monitors ongoing responses and identifies instances of response conflict, signaling the need for adjustments in control (e.g., Carter et al., 1998; Gehring & Fencsik, 2001; van Veen &

Carter, 2002). Conflict detection by this system alerts the second, *regulatory system*, which implements top-down control-related processes in the service of activating the intended response while inhibiting unintended responses (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter et al., 2000; Kerns et al., 2004). This two-component structure is similar in many ways to the model of mental control proposed by Wegner (e.g., Wegner, 1994), which also contains monitoring and control functions. This model has been used to account for ironic rebound effects that occur when people are attempting to control prejudice by trying not to think in stereotypic ways about others (e.g., Macrae, Bodenhausen, Milne, & Jetten, 1994). However, whereas this model is focused on the consequences of attempts at thought control, it appears limited with respect to identifying the particular cognitive control mechanisms responsible for inhibiting racially biased behaviors when individuals are not specifically attempting to banish stereotypic thoughts.

Some investigators recently have begun to examine this issue. For example, Payne and colleagues have shown that constraints on controlled processing (e.g., requiring quick responses) increase racially biased response tendencies without affecting automatic stereotyping processes (Lambert et al., 2003; Payne, 2001) and that this bias likely results from failures of cognitive control (Payne, Shimizu, & Jacoby, 2005). However, these studies are limited with respect to specifying components of cognitive control that might be involved. Amodio et al. (2004) extended this work by showing that neural conflict detection processes are sensitive to the response conflict inherent in race-biased responses, although detection of conflict does not ensure unbiased responses. However, this study did not directly address the potential role of the regulative component of cognitive control in the inhibition of biased responses (see Amodio et al.).

Despite these recent advances, understanding of the role of cognitive control in regulating racial bias is far from complete. It is important to note that demonstrating the role of regulatory control in the inhibition of bias would seem to require dissociating activating from inhibitory processes. In some recent studies, inhibition of race-biased responses has been conceptualized in terms of slower response times on stereotype-congruent trials among individuals who are motivated to avoid prejudice (e.g., Maddux, Barden, Brewer, & Petty, 2005; Monteith, Ashburn-Nardo, Voils, & Czopp, 2002). This interpretation is questionable, in that slower response times in this context likely reflect weaker response activation (see Lepore & Brown, 1997) rather than inhibition per se. Cognitive theories of behavioral control posit that response activation and inhibition are governed by distinct processing systems (e.g., Logan & Cowan, 1984). Within this framework, the speed of a response is indicative of the relative strength of the response activation system, whereas behavioral inhibition generally is conceptualized as withholding or terminating a response (e.g., Logan, Cowan, & Davis, 1984). The extent to which race-biased responding may represent a failure of control processes underlying behavioral inhibition has never been specifically examined.

In addition, links between neural processes known to index regulative cognitive control and inhibition of race bias have yet to be established. Neural measures are important in this context because they can specify which cognitive processes mediate a particular outcome associated with stereotype activation or control of bias as well as the temporal ordering of those processes. Stereotype activation often is inferred from faster reaction times to stereotype-consistent versus stereotype-inconsistent information (e.g., Dovidio, Evans, & Tyler, 1986; Dovidio, Kawakami, Johnson, Johnson, & Howard, 1997; Fazio, Jackson, Dunton, & Williams, 1995). However, reaction time reflects both relevant cognitive processes associated with stereotype activation and less relevant response-related motor processes (see Ito & Cacioppo, in press).

In contrast, event-related brain potentials (ERPs) can more purely index cognitive operations independently of response generation processes. ERPs are scalp-recorded voltage deflections in the electroencephalogram (EEG) reflecting information-processing operations associated with specific stimulus events (see Fabiani, Gratton, & Federmeier, in press). The P300 is a positive-going ERP component typically peaking between 300 and 800 ms following stimulus onset. Its peak latency has been described as a neural indicator of the speed of categorization and evaluation (see Coles, 1989; Rugg & Coles, 1995) that is not dependent on the duration of response-related motor processes or response selection requirements (Kutas, McCarthy, & Donchin, 1977; McCarthy & Donchin, 1981; Smid, Mulder, Mulder, & Brands, 1992). Thus, P300 latency should be longer when stereotype-incongruent information is processed, relative to stereotype-congruent information. The amplitude of the P300 provides an additional, independent index of construct activation. P300 amplitude increases as the subjective probability of a stimulus decreases, and thus it is thought to reflect online updating of working memory (e.g., Donchin & Coles, 1988; Fabiani & Donchin, 1995). For example, P300 amplitude increases when trait information violates previously established expectancies (Bartholow, Fabiani, Gratton, & Bettencourt, 2001; Bartholow, Pearson, Gratton, & Fabiani, 2003). Thus, to the extent that stereotypes are activated in a given context, stereotype-violating trait information should increase P300 amplitude, relative to stereotype-consistent traits. These characteristics make P300 latency and amplitude useful as measures of implicit stereotype activation that can be used to supplement traditional behavioral measures (see Ito & Cacioppo, 2000, in press).

Other ERP components reflect processes associated with cognitive control. The N2 component tends to be very pronounced on tasks requiring inhibition, leading to the hypothesis that the N2 reflects neural inhibitory mechanisms (e.g., Bokura, Yamaguchi, & Kobayashi, 2001; Falkenstein, Hoormann, & Hohnsbein, 1999). However, others have argued that the N2 reflects conflict detection (see Botvinick et al., 2001), including conflict between activation and inhibition of prepotent responses (Bruin, Wijers, & van Staveren, 2001). Thus, trials requiring inhibition of stereotype-consistent information should elicit a large N2. In addition, the amplitude of the negative slow wave (NSW) component recently has been linked with implementation of cognitive control. West and Alain (1999, 2000) showed that the NSW is larger on Stroop task trials (Stroop, 1935) in which cognitive conflict is successfully resolved (see also Curtin & Fairchild, 2003).

Alcohol, Cognitive Control, and Race-Biased Responses

Demonstrating the role of cognitive control in the inhibition of race-biased responses requires manipulating the extent to which control can be implemented. Although this can be accomplished in a number of ways, alcohol administration provides some advantages over other available methods. For example, in some paradigms participants engage in an initial task intended to deplete control-related resources prior to measurement of a relevant outcome (e.g., Richeson et al., 2003; Richeson & Shelton, 2003).

Alcohol's effects occur concurrently with participants' main task, eliminating the need for a separate resource-depleting task.

Moreover, alcohol consumption has proven very effective in specifically targeting cognitive control resources associated with inhibition. Studies using tasks that separately assess response activation and inhibition have shown that alcohol significantly impairs behavioral inhibition but has no effect on activation and implementation of responses (e.g., Easdon & Vogel-Sprott, 2000; Fillmore & Vogel-Sprott, 1999, 2000; Mulvihill, Skilling, & Vogel-Sprott, 1997) and that these effects are due to alcohol's impairment of cognitive control rather than its potential effects on motivation or information processing more generally (e.g., Abroms, Fillmore, & Marczinski, 2003; Easdon & Vogel-Sprott, 2000). Studies separating automatic from controlled influences on behavior similarly indicate that alcohol's effects are primarily limited to controlled processes (Fillmore, Vogel-Sprott, & Gavrilescu, 1999); alcohol has analogous effects on person perception (e.g., Bartholow et al., 2003; Herzog, 1999). Given these characteristics, alcohol is arguably preferable to some cognitive load manipulations (e.g., imposing short response deadlines; requiring concurrent mental tasks) that likely influence both response activation and inhibition processes. Moreover, alcohol should be expected not to influence stereotype activation, but its impairment of controlled inhibitory processes should produce increased expressions of race bias once stereotypes are activated.

Electrocortical evidence bearing on alcohol's theorized impairment of cognitive control recently was presented by Curtin and Fairchild (2003). These authors reported that alcohol impaired Stroop task performance and reduced the amplitude of the NSW component of the ERP on high conflict trials. These data support the notion that alcohol-related performance decrements on tasks requiring cognitive control can be mapped onto impairment of specific neurocognitive processes.

Finally, research to date simply has not examined the effects of alcohol on race bias. A number of theoretical models posit that alcohol's effects on interpersonal processes, such as aggression and sexual risk taking, are mediated by impairment of just those cognitive processes that are important for proper inhibitory function (e.g., Giancola, 2000). Therefore, using alcohol in the current research permits examination of the possibility that basic processes underlying race bias are similar to those subserving a host of other troubling interpersonal behaviors.

The Current Research

The purpose of the current studies was to test the role of regulative cognitive control in the inhibition of racially biased responses and the influence of alcohol on this process. Given that alcohol is known to affect regulative control (Curtin & Fairchild, 2003), and that such control is critical for effective behavioral inhibition (e.g., MacDonald et al., 2000), studying these processes in both sober and moderately intoxicated individuals provides an effective way to test relevant hypotheses. In two experiments, participants were randomly assigned to consume either a placebo beverage or one of two doses of alcohol and then engaged in a priming reaction time task designed to assess stereotype activation (Experiment 1) and inhibition of race-biased responses (Experiment 2).

We contend that concept activation, response activation, and inhibitory control are associated with distinct mental events reflected in different ERP components. Thus, it is important for the current research to provide independent tests of the manipulations affecting these components. For example, research indicates that tasks designed to measure response inhibition significantly change the distribution, amplitude, and meaning of the P300 component (e.g., Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004). Thus, Experiment 1 was designed to highlight stereotype activation effects in the ERP (focused on the P300), whereas Experiment 2 was focused on demonstrating neural activities associated with conflict detection and inhibition of race bias (N2 and NSW). In this way, these ERP data can inform important issues in current theoretical models of person perception, such as whether regulation of bias is associated with levels of response inhibition rather than activation and whether these processes are mediated by distinct electrocortical events.

Experiment 1

Method

Participants

Newspaper advertisements and posted fliers were used to recruit individuals between the ages of 21 and 30 for a study on the effects of alcohol. Interested persons called the lab and were scheduled for an initial questionnaire screening session. A total of 102 individuals participated in screening sessions (for $5 compensation) during which they completed measures related to alcohol and drug use and general health. Persons who indicated any major medical conditions that contra-indicate alcohol administration (including pregnancy) were disqualified from the later laboratory experiment, as were individuals with any history of substance abuse treatment. In addition, to ensure that the alcohol dose received in the experiment would be within participants' range of experience, individuals who reported an average of less than 2 or more than 24 drinks per week during the past 30 days were excluded from the study sample. From this initial screening sample, 68 individuals were called back and asked to participate in a laboratory study. Of this sample, 48 individuals (24 men) agreed to participate in exchange for $8.00 per hour compensation. All participants were White and right-handed.

Eligible participants agreed to adhere to a preexperimental protocol that included refraining from alcohol and drug use for 24 hr prior to their appointment, eating a light meal 4–6 hr prior to their appointment, and abstaining from strenuous physical exercise for 3 hr prior to their appointment; they were reminded of these requirements via an e-mail sent the day before their appointment. Upon arrival to the lab, participants signed affidavits attesting to their adherence to study protocols; no participants were disqualified for failure to comply with preexperimental instructions. In addition, female participants were required to take a hormonal pregnancy test in the lab prior to beverage administration. One positive test result occurred; the individual was not allowed to participate and was referred to a campus clinic.

Stimuli and Experimental Paradigm

The paradigm used in this experiment was derived from one developed by Dovidio and colleagues to assess stereotype activation (e.g., Dovidio et al., 1986, 1997; Perdue, Dovidio, Gurtman, & Tyler, 1990). Participants were presented with racial category primes (color photos of 2 Black men, 2 Black women, 2 White men, and 2 White women) and control primes (8 photos of houses), each followed by descriptive adjectives. These adjectives consisted of traits that could describe people but not houses (i.e., person words) as well as terms that could describe houses but not people (i.e., house words). On each trial, the participant's task was to decide whether the trait word could ever be true of the specific person or house

that preceded it. Thus, the correct response on any person-prime, person-word trial was "yes." Trait words included 12 person descriptors, 6 of which represented common stereotypes about Blacks (*lazy, violent, ignorant, musical, athletic, humorous*) and 6 of which represented common stereotypes about Whites (e.g., *uptight, boring, stubborn, intelligent, ambitious, educated*). These words were chosen from stereotype-related terms tested in previous research (Wittenbrink, Judd, & Park, 1997). The valence (favorability) of the person descriptors also was varied such that half of the traits in each stereotype condition were positive and half were negative. Twelve house words (e.g., *carpeted, drafty, shingled, furnished*) also were used. Stereotype activation is measured as the extent to which person primes facilitate responses to stereotype-consistent traits relative to stereotype-violating traits (e.g., Dovidio et al., 1986, 1997). Participants indicated their responses by pressing one of two keys on the computer keyboard. Person-prime photos were selected from a larger sample of images on the basis of attractiveness ratings provided by a pretest sample ($N = 20$). An analysis of variance (ANOVA) of pretest attractiveness ratings showed that the eight individuals used as primes in these studies were viewed as average in attractiveness ($M = 4.08$ on a 0–9 scale) and that all were seen as equally attractive regardless of race or sex (Fs < 2, ps $> .19$).

Participants completed four blocks of the priming task, each containing 192 trials, for a total of 768 trials. Of these, 256 were person-prime, person-word trials. Specifically, each person prime was presented 8 times with an instance of each type of person word. Each person prime also was presented with a house word 32 times. The remaining trials involved house primes. On each trial, a priming image was presented for 500 ms, followed by a 400-ms delay prior to the onset of a target word (also presented for 400 ms). An intertrial interval of 2,500 ms was inserted after the participant's response (or after 3 s if no response occurred) prior to the onset of the next trial. The order of picture primes and target words was randomized. Responding hand for "yes" and "no" responses was counterbalanced across participants.

Our choice of an explicit priming procedure for these studies deserves some comment. Some have argued that requiring an explicit link between the primes and targets undermines the argument that the results reflect automatic stereotype activation (e.g., Macrae & Bodenhausen, 2000). Unlike earlier studies using this paradigm in which participants were told to link the target words to the category of the prime (e.g., Dovidio et al., 1986), in the current research participants were instructed to link the target words to individual primes without reference to category. This change in the procedure, and the use of a relatively short interstimulus interval between primes and target words, should reduce the explicit focus on race that limited the interpretation of earlier reports. Moreover, category priming effects generally are identical regardless of whether participants are aware of the priming stimuli (Bargh, 1992). Furthermore, because people typically are aware of the presence of others during real interracial interactions, the use of conscious primes more aptly demonstrates stereotype activation as it usually happens (see Lepore & Brown, 2002).

Electrophysiological Recording

The EEG was recorded from 28 Ag/AgCl electrodes fixed in a stretch-lycra cap (ElectroCap, Eaton, OH) and placed according to an expanded version of the 10–20 system (American Encephalographic Society, 1991). All cap electrodes were referenced online to the right mastoid (an average mastoid reference was derived offline). EEG was recorded continuously throughout the task, and stimulus-locked epochs of 1,400 ms were derived offline (referenced to 200 ms prestimulus baseline). Eye movements were recorded with bipolar electrodes placed just above and below the left eye and 2 cm external to the outer canthus of each eye. A ground electrode was located near the front of the cap, along the midline. EEG and eye movement signals were amplified with a Synamps amplifier (Neuroscan Labs, Sterling, VA) and filtered online at 0.05 to 30 Hz at a sampling rate of 250 Hz. Impedance was kept below 5 kΩ. Ocular artifacts (blinks) were removed from the EEG signal offline by using a regression-based procedure (Semlitsch, Anderer, Schuster, & Presslich, 1986). Trials containing voltage deflections of ±75 microvolts (μV) were rejected prior to averaging. After artifact removal and rejection, EEG data were averaged offline according to participant, electrode, and stimulus conditions and were low-pass filtered at 12 Hz (12-dB roll-off).[1]

Beverage Administration

Equal numbers of men and women 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 active placebo (0.04 g/kg ethanol) vodka and tonic beverage. Two alcohol groups were used to permit tests of whether alcohol effects were dose dependent. To reduce the discrepancy between actual and expected doses across conditions, all participants were told that they would be receiving a moderate dose of alcohol (see Sher & Walitzer, 1986). In all three conditions, the experimenter ostensibly mixed a beverage containing a moderate dose of alcohol in a 5:1 tonic-to-vodka ratio. The placebo dose was achieved by using diluted vodka (9 parts flattened tonic to 1 part 100-proof vodka mixed in a 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). Total beverage was isovolemic across beverage conditions. Collars were used to indicate the actual contents of each bottle (e.g., "regular tonic," "spiked tonic," etc.), and the lead experimenter removed these collars before the bottles were brought to the second experimenter. Thus, the (second) experimenter who mixed and served the beverage was unaware of the actual contents of the bottles. The beverage was divided into three equal-size drinks that were given to the participant one at a time. Lime juice was added for flavor. Participants were allowed 5 min to consume each of the three drinks.

Intoxication Measures

Blood alcohol concentration (BAC) was measured throughout the experimental session by using an Alco-Sensor IV Breathalyzer (Intoximeters, St. Louis, MO). Participants were not informed of their actual BAC level during the experimental task. To eliminate residual alcohol in the mouth, participants rinsed their mouths with water prior to the first postdrinking BAC measurement. A new disposable mouthpiece was used for each sample taken during a lab session. In addition to BAC measurement, we included a short questionnaire at the conclusion of the session designed to assess participants' subjective intoxication level during the study. Five questions asked participants to rate how intoxicated they felt throughout different phases of the experimental task. Responses ranged from 0 (*not at all*) to 4 (*a lot*). Three additional items assessed how much the alcohol participants drank affected their performance, effort, and concentration during the task, by using the same scale. Participants also rated how much they tried to perform their best, as well as how frustrated they were by the task, also by using this scale. Finally, participants estimated the number of standard alcohol drinks they believed they had consumed by using a scale of 0 to 20.

Procedure

Upon participants' arrival at the lab, an experimenter verified their age and measured their weight. Participants then read and signed the informed consent form and affidavits, after which an experimenter read them specific instructions for the experimental task and explained the beverage administration and electrophysiological recording procedures. Female partici-

[1] Although we prefer filtered waveforms for both empirical (Fabiani, Gratton, Karis, & Donchin, 1987) and aesthetic reasons, a set of analyses on the P300 data that used unfiltered waveforms produced findings identical to those we report.

pants then were given the pregnancy test to self-administer in the restroom; male participants were asked to use the restroom at this time to void the bladder prior to drinking.

Upon returning from the restroom (and, for women, verification of a negative pregnancy test result), participants were led to an adjacent room for electrode placement and testing, after which they were seated in a sound-attenuated recording booth 60 cm in front of a computer monitor. The experimenter then read a set of instructions explaining the computer task. Participants were told to respond as quickly as possible on each trial, but also to be accurate. To familiarize them with the task prior to beverage consumption, participants then completed 40 practice trials in which pictures of pets (e.g., dogs, cats) and houses were used as prime stimuli, and words that could describe pets or houses were used as targets. An experimenter monitored practice trial performance to ensure quick and accurate responses and normal EEG signals. Electrode impedance was reverified after the practice trials and again after beverage consumption and absorption.

Following the practice trials, the lead experimenter took a baseline intoxication measurement while the second experimenter measured the appropriate amount of each beverage and mixed the drink in a large pitcher. Upon completion of the third and final drink, participants sat idle for 15 min to allow the alcohol to absorb. Following the absorption period, a second intoxication measurement was taken just before participants started the first half of the experimental trials, after which a third intoxication measurement was taken. Participants were given a 2-min break between blocks of trials and encouraged to rest their eyes during this time. Participants then completed the final two blocks of trials, after which a fourth intoxication measurement was taken.

Following the fourth intoxication measurement, electrodes were removed and participants were allowed to clean up at the sink in an adjacent room. Participants then completed the postexperimental questionnaire items and some questionnaire items intended to probe for suspicion (none was revealed), following which participants were debriefed about the true nature of the study. Participants in the alcohol conditions were retained in the lab until a breathalyzer indicated that their BAC was .02% or less. These participants were given snacks and water or soft drinks and allowed to watch DVD movies or read during this time. All participants, regardless of beverage condition, were driven home after the session by a friend or were taken home by city bus. Considering all phases of the experiment, sessions for each participant lasted between 3.5 and 9 hr, with longer sessions reflecting time for sobering up.

Dependent Variables and Hypotheses

Two behavioral and two electrocortical indicators of stereotype activation served as primary dependent variables in this study. First, reaction times (RTs) to correctly categorized stereotype-consistent and stereotype-violating trials were computed, as in previous research (e.g., Dovidio et al., 1986, 1997). On the basis of prior findings (e.g., Dovidio et al., 1986, 1997; Gaertner & McLaughlin, 1983; Gilbert & Hixon, 1991), we predicted that responses to stereotype-consistent traits would be facilitated relative to stereotype-violating traits, resulting in a two-way interaction between race of prime (Black, White) and stereotypicality of trait words (Black stereotypic, White stereotypic). We also measured the accuracy with which trait descriptors for person primes were categorized. An error was scored on a given trial if a person word was incorrectly classified as an inappropriate descriptor of a person prime or if a participant failed to respond to the target word within 3 s (only a tiny proportion of errors were the result of slow but correct responses).

The latency and amplitude of the P300 component of the ERP elicited on correct trials served as separate electrocortical measures of stereotype activation. The P300 component typically is most pronounced at the midline parietal (Pz) electrode, and initial inspection of the data from this study confirmed this pattern. Therefore, P300 latency was quantified for each participant as the latency (in ms) of the largest positive-going component of the ERP waveform at Pz between 300 and 900 ms poststimulus in each condition, and P300 amplitude was quantified as the amplitude of the largest positive-going component at Pz in this same epoch (see Ito & Urland, 2003).[2] As with the RT data, we predicted that P300 latency would be slower on stereotype-violating than on stereotype-consistent trials. Similarly, we expected P300 amplitude to be largest for stereotype-violating trait words, reflecting working memory updating associated with stereotype violations.

Results

Data from 5 participants were unusable (3 because of equipment failure; 2 due to excessive EEG artifacts), leaving the final sample on which all analyses were based at 42 participants (21 men; $n = 14$ in each dose group). Initial analyses indicated that the effects of interest did not differ as a function of participant sex, so we collapsed across sex in all analyses for both experiments. The effects of the experimental manipulations on each of the dependent variables listed above were examined by using separate 3 (dose: placebo, moderate, high) $\times$ 2 (race of prime: Black, White) $\times$ 2 (sex of prime) $\times$ 2 (valence of trait words: positive, negative) $\times$ 2 (stereotypicality of trait words: Black stereotypic, White stereotypic) mixed factorial ANOVAs with repeated measures on all but the first factor. Planned comparisons for all predicted effects were carried out by using one-tailed tests.

Manipulation Checks

Alcohol dose. Baseline BAC values for all participants were zero, as were values for placebo group participants throughout the study. Postdrinking BAC levels in the moderate- and high-dose groups, measured before, during, and after the priming task, were analyzed with a 2 (dose) $\times$ 3 (assessment time) ANOVA, with repeated measures on the latter factor. This analysis showed that, overall, BACs were higher in the high-dose group ($M = 0.071\%$, $SD = 0.01$) than in the moderate-dose group ($M = 0.037\%$, $SD = 0.01$), $F(1, 23) = 65.91$, $p < .01$. This effect was qualified by a significant Dose $\times$ Time interaction, $F(2, 46) = 4.13$, $p < .05$. Simple effect tests showed that BAC decreased from pretask to posttask assessments in the moderate-dose group (Ms = 0.042, 0.039, and 0.030, respectively), $F(2, 26) = 12.80$, $p < .01$, but did not change significantly in the high-dose group (Ms = 0.069, 0.074, and 0.069, respectively), $F(2, 26) = 1.51$, $p > .20$.

Subjective intoxication. Posttask intoxication ratings were averaged to create a subjective intoxication index ($\alpha = .90$). Ratings of how much participants' performance, effort, and concentration were affected by alcohol were averaged to create a subjective impairment index ($\alpha = .76$). These composites were analyzed by using separate one-way ANOVAs. Ratings of subjective intoxication increased as a function of dose, $F(2, 39) = 12.64$, $p < .001$ (Ms = 0.56, 1.46, and 1.89 for placebo, moderate, and high dose, respectively), as did subjective impairment ratings (Ms = 1.23,

[2] Ancillary analyses carried out with data from all scalp electrodes produced findings essentially identical to those we report, but the latency analyses included some extremely complex five- and six-way interactions, suggesting that some effects differed slightly as a function of lateral (e.g., left vs. right hemisphere) and coronal (anterior vs. posterior) electrode locations. These effects are very difficult to interpret and are not important for the predictions of this study, so they will not be discussed. Amplitude analyses including all electrode locations produced interactions indicating that the reported effects were largest at midline scalp locations.

2.09, and 2.48, respectively), $F(2, 39) = 8.06$, $p < .01$. Participants' estimates of the number of standard drinks they consumed during the study also differed according to dose group, $F(2, 39) = 13.32$, $p < .001$. Tukey follow-up comparisons showed that participants in the placebo group ($M = 1.64$) believed that they had consumed fewer drinks than those in the moderate- ($M = 3.07$) and high-dose groups ($M = 4.07$), ps $< .01$, whose estimates did not differ significantly ($p = .10$). The fact that those in the placebo group generally believed that they had consumed close to two drinks suggests that our cover story was viable. Finally, participants' ratings of how much they tried to perform their best during the task ($M = 3.64$) and how frustrated they were by the task ($M = 2.0$) did not differ significantly as a function of alcohol dose (Fs < 1.50, ps $> .20$).

Behavioral Data

Response accuracy. Eight data points (representing 1.2% of the data; distributed roughly equally across conditions) were identified as extreme outliers. Following suggestions of others (Tabachnick & Fidell, 1989; Tukey, 1990; Wilcox, 1995), extreme outliers were modified to the value of the next closest nonoutlying value in the distribution (i.e., Winsorizing). This procedure resulted in more normal distributions suitable for analysis of variance.

The predicted Race × Stereotypicality interaction was significant, $F(1, 39) = 14.68$, $p < .001$, but was further qualified by sex of prime, $F(1, 39) = 12.52$, $p < .001$. This three-way interaction was probed by using separate ANOVAs for male prime trials and female prime trials. The Race × Stereotypicality interaction was highly significant for male primes, $F(1, 39) = 23.33$, $p < .001$; participants were more accurate in classifying Black- than White-stereotypic traits following Black primes (Ms = 0.965 and 0.912, respectively), $t(39) = 4.42$, $p < .001$, and more accurate in classifying White- than Black-stereotypic traits following White primes (Ms = 0.980 and 0.964, respectively), $t(39) = 2.17$, $p < .01$ (one-tailed). This interaction was not significant for female primes, $F(1, 39) = 0.25$, $p > .60$. No other effects of interest were significant in this analysis. Accuracy was not significantly influenced by alcohol dose, either as a main effect, $F(2, 39) = 0.20$, $p > .80$, or via interactions with other variables (Fs < 2.50, ps $> .10$).

Response time. Only correct response trials were included in this analysis. Prior to analyses, trials with extremely long RTs (>2.5 standard deviations above each participant's mean value) were excluded (1.8% of all trials). The remaining RTs were subjected to log transformation in order to normalize the distributions (see Fazio, 1990), and analyses were based on these transformed data. For ease of interpretation, the untransformed means (in ms) are presented in the text.

The predicted Race × Stereotypicality interaction was significant, $F(1, 39) = 47.92$, $p < .0001$. Planned contrasts indicated that following White primes, RTs were significantly longer to Black-stereotypic ($M = 697.6$, $SD = 133.1$) than White-stereotypic traits ($M = 654.7$, $SD = 108.3$), $t(39) = 7.04$, $p < .001$; following Black primes, RTs were significantly longer to White-stereotypic ($M = 693.6$, $SD = 136.2$) than Black-stereotypic traits ($M = 663.4$, $SD = 110.8$), $t(39) = 3.89$, $p < .001$, consistent with previous research (e.g., Dovidio et al., 1986, 1997). This interaction was not further qualified by alcohol dose ($F < 1$) nor by any other factors. Other significant effects included a main effect of valence, $F(1, 39) = 53.16$, $p < .001$, indicating that responses generally were quicker to positive traits ($M = 660.1$) than to negative traits ($M = 694.5$), replicating earlier priming studies (e.g., Perdue et al., 1990). No other effects of interest were significant.

ERP Data

P300 latency. As with RTs, the predicted Race × Stereotypicality interaction was significant, $F(1, 39) = 5.43$, $p < .03$. Planned contrasts indicated that P300 latencies were significantly longer to White-stereotypic ($M = 594.5$, $SD = 83.2$) than Black-stereotypic traits ($M = 575.6$, $SD = 66.9$) following Black primes, $t(39) = 1.96$, $p < .05$, and were marginally longer to Black-stereotypic ($M = 595.6$, $SD = 77.5$) than White-stereotypic traits ($M = 580.5$, $SD = 75.5$) following White primes, $t(39) = 1.47$, $p < .07$ (one-tailed). This interaction was not further qualified by alcohol dose ($F < 1$) nor by any other factors. No other effects of interest were significant in this analysis.[3]

P300 amplitude. The ANOVA examining P300 amplitude also showed a significant Race × Stereotypicality interaction, $F(1, 39) = 11.17$, $p < .002$. As predicted, following White primes, Black-stereotypic traits elicited larger P300 amplitudes ($M = 7.72$ μV) than White-stereotypic traits ($M = 6.45$ μV), $t(39) = 3.20$, $p < .01$; and following Black primes, White-stereotypic traits elicited larger P300 amplitudes ($M = 8.05$ μV) than Black-stereotypic traits ($M = 7.05$ μV), $t(39) = 2.00$, $p < .05$. The pattern was not further qualified by alcohol dose, $F(2, 39) = 1.45$, $p > .20$. As shown in Figure 1, stereotype-violating traits elicited larger P300s than stereotype-consistent trials in each dose group.

Discussion

The results of the first experiment convincingly demonstrated stereotype activation, generally replicating earlier reports using similar paradigms (e.g., Dovidio et al., 1986, 1997; Perdue et al., 1990). Just as convincingly, these results indicated that alcohol did not influence the stereotype activation process. A consistent pattern of stereotype activation effects was evident across both behavioral and physiological measures, and alcohol dose did not significantly alter that pattern in any case. Although the response accuracy data suggested that stereotype activation was only apparent following male primes, the patterns for the three other dependent measures indicated a more general phenomenon not limited to men. It is noteworthy that the results for valence did not parallel those for stereotypicality, suggesting that the race primes activated the cognitive contents of racial stereotypes more than the affective tone associated with them.

The apparent lack of any alcohol effect on stereotype activation is consistent with other recent data indicating that alcohol gener-

[3] Other significant effects included a main effect of valence, $F(1, 36) = 7.48$, $p < .01$, indicating that P300 latencies were longer to negative words ($M = 592.7$) than positive words ($M = 578.5$), and a Valence × Stereotypicality interaction, $F(1, 36) = 14.54$, $p < .01$, indicating that latencies to White-stereotypic positive words ($M = 566.8$) and Black-stereotypic negative words ($M = 578.3$) were faster than latencies to White-stereotypic negative words ($M = 607.1$) and Black-stereotypic positive words ($M = 590.3$).

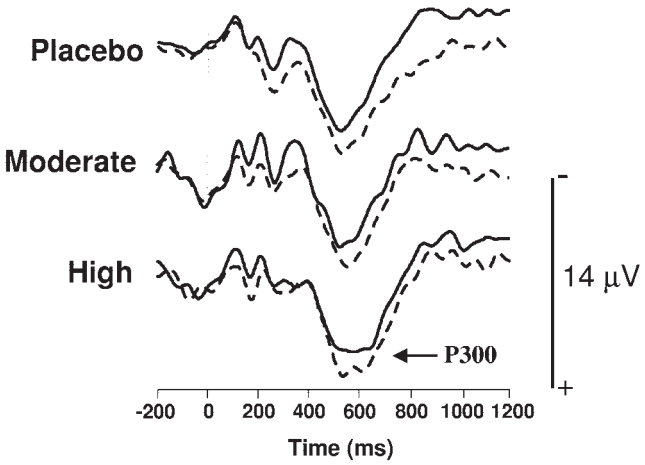

Figure 1. Event-related brain potential waveforms measured at the midline parietal (Pz) scalp location for stereotype-consistent trials (solid line) and stereotype-violation trials (dashed line) as a function of alcohol dose group, Experiment 1. Stimulus onset (i.e., word presentation) occurred at Time 0.

ally does not disrupt processes believed to be automatic (e.g., Bartholow et al., 2003; Fillmore et al., 1999; Herzog, 1999). This finding further supports our contention that responses in our version of this priming task were driven by largely automatic processes, despite the fact that prime stimuli were presented consciously. As noted by Bargh (1996, p. 171), "exercise of conscious control over [a] default automatic process requires an act of will." Had participants attempted to exercise control over their responses, we would expect different patterns in the placebo and alcohol groups, particularly because people generally are motivated to control race-biased responses (e.g., Monteith, 1993). Thus, although it remains possible that participants controlled their responses in this study, there is no evidence that they did so.

The ERP data from this experiment are particularly noteworthy. Both P300 amplitude and latency were sensitive to violations of racial stereotypes. Previous studies have shown similar effects on P300 amplitude for violations of target-based expectancies (e.g., Bartholow et al., 2001, 2003) and for violations of gender stereotypes in sentence comprehension (Osterhout, Bersick, & McLaughlin, 1997). The current results extend this prior work into the domain of racial stereotype violations and suggest that ERPs can provide a sensitive, implicit measure of stereotype activation unconfounded by less relevant response preparation and execution processes (see also Crites, Cacioppo, Gardner, & Berntson, 1995; Ito, Thompson, & Cacioppo, 2004).

Although finding that stereotype activation is robust to moderate doses of alcohol is of interest in itself, this experiment did not directly address questions pertaining to inhibition of race-biased responses. In addition, the task used in Experiment 1 was not well suited to elicit neural activity associated with cognitive control, and thus it did not permit examination of the central aspect of our overarching hypothesis that cognitive control of response inhibition is necessary to withhold race-biased responses following stereotype activation. Experiment 2 was conducted to test these hypotheses.

Experiment 2

Researchers interested in measuring the relative strength of activation and inhibition processes under various circumstances traditionally have used forms of the "go–stop" choice RT task (Logan, 1994). This task engages participants in responding to go signals while stop signals occasionally inform them to withhold their responses. Response activation in the go–stop task is measured by the speed of responses to go signals, and inhibitory control is defined by the frequency of unintended responses to stop signals (i.e., inhibition errors; Logan & Cowan, 1984). Researchers using this paradigm have found that alcohol increases inhibition errors but has no effect on RT to go signals (e.g., Fillmore & Vogel-Sprott, 1999, 2000; Mulvihill et al., 1997).

Therefore, in Experiment 2, we created a go–stop version of the priming paradigm used in Experiment 1. The prime and target word stimuli used in the second study were identical to those used in Experiment 1, with one exception. Because of the added go–stop manipulation, it was deemed necessary to eliminate one factor in the design in order to maintain the same number of trials as used in Experiment 1 while ensuring that sufficient numbers of each trial type would be represented in the stop condition. Doing so helped to ensure that fatigue would not become a significant factor in performance and that those in the alcohol conditions would complete the task before their BAC levels fell markedly. Therefore, we combined trait stereotypicality and trait valence into a single factor, focusing on positive White-stereotypic traits and negative Black-stereotypic traits. Though this approach limited our design, it is consistent with that used in other stereotype-priming studies (e.g., Macrae, Milne, & Bodenhausen, 1994; Macrae, Stangor, & Milne, 1994).

According to the logic of the sequential priming task, and on the basis of the results of Experiment 1, stereotype-consistent trials should result in stronger response activation (i.e., faster RTs on go trials; stronger tendency for inhibition errors on stop trials) than stereotype-violating trials because stereotype-consistent responses should be facilitated by the racial category primes. To the extent that alcohol impairs cognitive control of inhibition, inhibition errors should increase with alcohol, particularly on stereotype-consistent trials. However, alcohol is unlikely to influence inhibition errors on stereotype-violating trials. Also, because alcohol is hypothesized not to impair response activation and implementation (e.g., Fillmore & Vogel-Sprott, 1999, 2000), RTs and accuracy on go trials overall should be unaffected by alcohol.

Regarding the ERP measures, to the extent that the N2 reflects conflict between response activation and inhibition tendencies, and if stereotype-consistent trials elicit stronger response activation, N2 amplitude should be largest on stereotype-consistent stop trials. Moreover, if alcohol does not impair response activation, it should not affect N2 amplitude (Fillmore & Vogel-Sprott, 1999, 2000). To the extent that the NSW reflects the engagement of cognitive control processes subserving inhibition, we expected the NSW to be larger on successfully inhibited stop trials than on go trials. However, given alcohol's theorized impairment of cognitive control (e.g., Curtin & Fairchild, 2003), we expected this pattern only in the placebo group. More important in the present context, we predicted larger NSW in the placebo group on stereotype-consistent trials than on stereotype-violating trials, reflecting stronger engagement of cognitive control necessary to withhold responses that are more strongly mapped to racial categories.

Method

Participants

Participant recruitment and eligibility criteria were very similar to Experiment 1. However, in this experiment, interested persons called the lab and spoke to a research assistant who determined their eligibility by using a computer-guided structured interview. The sample used for this experiment included 51 healthy social drinkers ages 21–30 (all White; all right-handed), who were paid $8.00 per hour for their participation.

Stimuli and Experimental Paradigm

Each trial of the go–stop task consisted of a face prime presented for 500 ms, then a 400-ms delay, then the presentation of a trait word for 400 ms, just like Experiment 1. However, 250 ms after the onset of the trait word on each trial, a go signal (green arrow; 75% of trials) or stop signal (red *X*; 25% of trials) appeared just above the word. Prior research has shown that this proportion of go and stop signals ensures that participants respond as quickly on go trials as they would on similar tasks without stop signals and that they do not simply wait for go signals (see Easdon & Vogel-Sprott, 2000; Logan, 1994). Go and stop signals remained on screen for 250 ms. Stop signals appeared randomly an equal number of times with each prime-trait word pairing. Participants were instructed to respond to trait words as quickly as possible following go signals by using the same decision criteria as in Experiment 1 and to withhold their response on all stop trials. Trials were separated by a 2,500-ms intertrial interval.

Procedure and Physiological Data Collection and Reduction

Beverage administration, intoxication measurement, and physiological recording and data preparation were identical to Experiment 1. Experimental procedures also were virtually identical to the first study, except that during the instructions phase the experimenter stressed the need for participants to respond only on go trials and to withhold responses on stop trials.

Dependent Variables

The primary measure of response inhibition was the frequency of inhibition errors. Numerous researchers have used this measure to index the function of the behavioral inhibition system (see Band, Van Der Molen, & Logan, 2003; Logan, 1994), including those examining alcohol's hypothesized impairment of inhibition (e.g., Fillmore & Vogel-Sprott, 1999, 2000; Mulvihill et al., 1997). The strength of response activation was determined by the mean RT to trait words on go trials; response accuracy also was assessed.

Amplitude of the NSW component of the ERP served as a measure of the neural mechanism underlying cognitive control. The NSW typically develops between 600 and 1,200 ms poststimulus and is generally largest over central or fronto-central scalp locations (West & Alain, 1999, 2000). Therefore, NSW amplitude was quantified here as the mean ERP amplitude between 650 and 1,000 ms following the go and stop signals in each condition at frontal and fronto-central electrodes. The no-go N2 served as a neural indicator of conflict associated with response activation. Initial inspection of the waveforms indicated that the component was most pronounced over frontal and fronto-central electrodes at the midline. Therefore, the no-go N2 was quantified here as the average negative-going amplitude between 250 and 350 ms following go and stop signals for each participant at frontal and fronto-central electrodes.

Results

Data from 9 participants were unusable (4 due to data coding errors; 4 more due to excessive EEG artifacts; and 1 who did not understand the task), leaving the final sample on which all analyses were based at 42 participants (26 women; *n*s = 15 placebo, 14 moderate dose, and 13 high dose). Analyses of all behavioral dependent variables were carried out by using 3 (dose) × 2 (race of prime) × 2 (sex of prime) × 2 (stereotypicality of trait word) mixed ANOVAs with repeated measures on all but the first factor.

Manipulation Checks

Alcohol dose. BAC and subjective intoxication were examined by using ANOVAs as in Experiment 1. BAC was higher overall in the high-dose group ($M = 0.083\%$, $SD = 0.01$) than the moderate dose group ($M = 0.037\%$, $SD = 0.01$), $F(1, 23) = 85.89$, $p < .001$. A significant Dose × Time interaction, $F(2, 46) = 3.59$, $p < .05$, showed that BAC decreased over time in the moderate-dose group (*M*s = .041, .038, and .032), but not in the high-dose group (*M*s = .079, .089, and .082). The fluctuations in the high-dose group resulted in a significant quadratic trend, $F(1, 23) = 17.69$, $p < .01$. However, in both dose groups, only the pretask and midtask means differed from each other significantly ($p < .05$). It should be noted that previous studies using go–stop tasks have shown no performance differences as a function of rising and falling BACs (Easdon & Vogel-Sprott, 2000).

Subjective intoxication. One participant did not complete the posttask questionnaire, so the subjective intoxication data were examined for only 41 individuals. Posttask ratings of intoxication were again averaged to create a subjective intoxication index ($\alpha = .93$), and ratings of performance, effort, and concentration again were averaged to create a subjective impairment index ($\alpha = .74$). Analyses of these composites produced findings highly consistent with those reported for Experiment 1 (i.e., estimates of intoxication and impairment increased with dose). Participants' ratings of how much they tried to perform their best during the task and how frustrated they were by the task again did not differ as a function of dose (*F*s < 1, *p*s > .50). Finally, participants' estimates of the number of standard drinks they consumed differed significantly by dose, $F(2, 38) = 14.29$, $p < .001$. Tukey follow-up comparisons showed that means for the placebo, moderate-, and high-dose groups all differed significantly from one another (*M*s = 1.38, 2.61, and 3.75, respectively; *p*s < .05). Participants in all three groups again believed that they had consumed alcohol.

Response Time on Go Trials

As in Experiment 1, response latencies longer than 2.5 standard deviations above each participant's mean value were discarded (1.6% of trials), and the remaining data were subjected to a log transformation. Replicating Experiment 1, the ANOVA on these log-transformed RTs showed a significant Race × Stereotypicality interaction, $F(1, 39) = 56.22$, $p < .001$. Planned contrasts showed that following White primes, RTs were significantly longer to Black-stereotypic ($M = 780.6$) than White-stereotypic traits ($M = 743.1$), $t(39) = 6.67$, $p < .0001$. Conversely, following Black primes, RTs were significantly longer to White-stereotypic ($M = 777.6$) than Black-stereotypic traits ($M = 753.6$), $t(39) = 3.69$, $p < .001$. As before, this interaction was not further qualified by alcohol dose ($F < 1$) nor by any other factors. Note that despite the 250-ms lag between the onset of the trait words and the onset of the go signal, RTs in this experiment were only approximately 100 ms longer than those in Experiment 1, suggesting that participants

did not simply wait for the go signal before activating responses. No other effects of interest were significant.

Response Accuracy on Go Trials

The predicted Race × Stereotypicality interaction was significant, $F(1, 39) = 7.28$, $p < .01$. Simple effect analyses showed that following White primes, participants responded correctly more often to White-stereotypic traits ($M = .941$) than to Black-stereotypic traits ($M = 0.872$), $t(39) = 2.93$, $p < .01$. Following Black primes, participants responded correctly more often to Black-stereotypic traits ($M = 0.936$) than to White-stereotypic traits ($M = 0.917$), though this difference was only marginally significant, $t(39) = 1.36$, $p < .10$ (one-tailed). This interaction was not further qualified by alcohol dose ($F < 1$), and no other effects involving alcohol approached significance ($ps > .15$).

Inhibition Errors

Analysis of the frequency of inhibition errors showed a significant main effect of dose, $F(2, 39) = 6.00$, $p < .01$. Although the overall frequency of inhibition errors was low, inhibition errors increased along with dose ($Ms = 1.0$, 2.2, and 4.38 for placebo, moderate, and high dose, respectively), as predicted. The linear trend apparent in these data was highly significant, $F(1, 39) = 11.31$, $p < .01$, and the quadratic trend was not ($F < 1$). The analysis also revealed a Dose × Race of Prime × Stereotypicality interaction, $F(2, 39) = 3.33$, $p < .05$. Initial inspection of the means suggested that this interaction was driven by greater inhibition errors on stereotype-consistent trials in the high-dose group. To facilitate further examination of this interaction, we therefore combined race and stereotypicality information to create stereotype-consistent and stereotype-violating trials for each participant and then tested for mean differences by dose group and condition. The pattern of means associated with this representation is shown in Figure 2. Post hoc mean comparisons showed that in the placebo and moderate groups, inhibition errors were equally likely on stereotype-consistent and stereotype-violating trials ($ts = 1.30$ and 0.25, $ps > .18$, respectively). In contrast, stereotype-consistent trials elicited more inhibition errors than stereotype-violating trials among those in the high-dose group, $t(12) = 2.28$, $p < .05$. More important, alcohol led to a linear increase in inhibition errors on stereotype-consistent trials, $F(1, 39) = 16.77$, $p < .001$, but did not significantly influence inhibition errors on stereotype-violating trials, $F(1, 39) = 2.20$, $p = .15$.

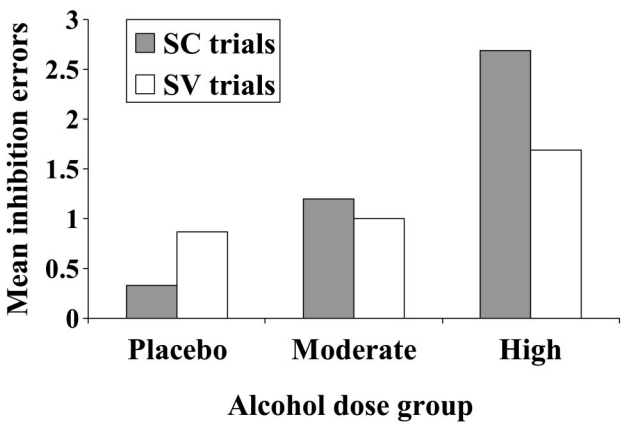

Figure 2. Inhibition errors on stereotype-consistent (SC) trials and stereotype-violation (SV) trials as a function of dose group, Experiment 2.

No-Go N2 Amplitude

Initial analyses confirmed that the N2 was significantly larger (i.e., more negative) on stop trials ($M = -1.91$ μV) than on go trials ($M = 1.70$ μV), $F(1, 39) = 39.39$, $p < .0001$. Therefore, and given that our main interest was in how our manipulations would influence the no-go N2, analyses of this component were restricted to stop trials only, by using a 3 (dose) × 2 (race of prime) × 2 (sex of prime) × 2 (stereotypicality of trait word) × 2 (scalp site: frontal, fronto-central) × 3 (electrode within scalp site: left, midline, right) mixed ANOVA with repeated measures on all but the first factor. The predicted Race × Stereotypicality interaction was significant, $F(1, 39) = 9.26$, $p < .01$. Planned comparisons indicated that following White primes, the no-go N2 was more negative to White-stereotypic traits ($M = -1.77$ μV) than to Black-stereotypic traits ($M = -0.02$ μV), $t(39) = 2.27$, $p < .05$. Following Black primes, the no-go N2 was more negative to Black-stereotypic traits ($M = -1.71$ μV) than to White-stereotypic traits ($M = -0.46$ μV), $t(39) = 1.69$, $p < .05$ (one-tailed). In addition, this interaction was not qualified by alcohol dose ($F < 1$). As shown in the right panel of Figure 3 (in the no-go N2 box), the solid lines differ from the dashed lines (stereotype-consistency effect), but the black lines do not differ from the gray lines (dose effect). The main effect of dose also was not significant, $F(2, 39) = 0.034$, $p > .90$.[4]

The only other significant effect in this analysis was an unpredicted Dose × Race × Scalp Site interaction, $F(2, 39) = 5.58$, $p < .01$. Examination of the means associated with this interaction indicated that the N2 was somewhat larger on White-prime trials than on Black-prime trials in the placebo group, and larger on Black-prime trials than White-prime trials in the high-dose group, but only at frontal electrodes. However, simple effect tests of these mean differences proved nonsignificant ($ts < 1.54$, $ps > .10$).

NSW Amplitude

Our first hypothesis concerning the NSW was that its amplitude should be larger on stop trials than on go trials, but perhaps only in the placebo group. To test this initial hypothesis, we submitted NSW amplitudes to a 3 (dose) × 2 (trial type: go trials, stop trials) × 2 (scalp site: frontal, fronto-central) × 3 (electrode within scalp site: left, midline, right) mixed ANOVA with repeated mea-

[4] Although the no-go N2 is considered a fronto-central component, a notable N2 was evident at more posterior electrodes in the current data (see Figure 3). Thus, we conducted ancillary analyses at posterior electrodes as well, which showed very similar results to those we report (i.e., significant Race × Stereotypicality interaction). As with the P300 in Experiment 1, we also analyzed the amplitude of the N2 by using unfiltered waveforms. In theory, filtering at 12 Hz could have a larger impact on the amplitude of the higher frequency activity associated with the N2 than on P300 amplitude. However, as before, the ANOVA produced effects highly similar to those reported in the main text: Race × Stereotypicality interaction, $F(1, 39) = 8.91$, $p < .01$; Dose × Race × Scalp Site interaction, $F(2, 39) = 5.60$, $p < .01$.

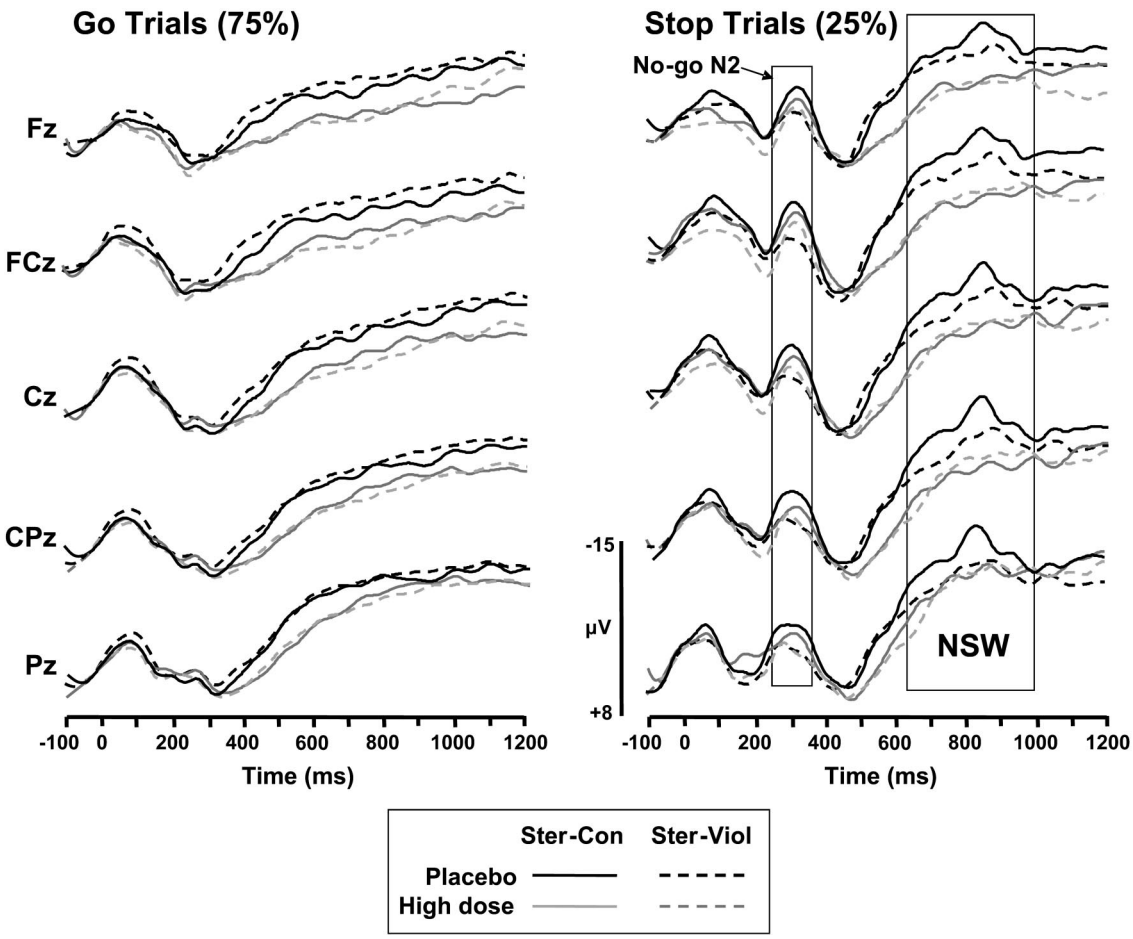

Figure 3. Event-related brain potential waveforms from midline scalp locations on go trials (left panel) and stop trials (right panel) as a function of beverage (placebo and high-dose groups), Experiment 2. Moderate dose waveforms were excluded from the figure in order to simplify presentation. Time 0 represents the onset of the go (left panel) and stop (right panel) signals. Fz = frontal; FCz = fronto-central; Cz = central; CPz = centro-parietal; Pz = parietal; NSW = negative slow wave; Ster-Con = stereotype-consistent trials; Ster-Viol = stereotype-violating trials.

sures on all but the first factor. As predicted, this analysis showed a significant main effect of trial type, $F(1, 39) = 12.56$, $p < .01$, qualified by the predicted Dose × Trial Type interaction, $F(2, 39) = 4.56$, $p < .02$. Planned comparisons indicated that the NSW was larger on stop trials than on go trials in the placebo group (*M*s = −7.20 and −4.40 μV, respectively), $t(14) = 2.98$, $p < .01$, but not in the moderate-dose group (*M*s = −3.50 and −2.32 μV, respectively), $t(13) = 1.25$, $p > .20$, or high-dose group (*M*s = −3.48 and −3.47 μV, respectively), $t(12) = 0.03$, $p > .90$. In addition, the NSW on go trials did not differ significantly as a function of dose (*p*s > .20), but the effect of alcohol on stop trial amplitudes appeared to be dose dependent, $F_{\text{linear}}(1, 39) = 3.97$, $p = .05$. This analysis also showed a significant electrode effect, $F(2, 78) = 6.70$, $p < .01$. Post hoc comparisons showed that the NSW was larger over left hemisphere sites ($M = -4.70$ μV) compared with midline ($M = -3.87$ μV) and right hemisphere sites ($M = -3.65$; *p*s < .02), which did not differ from each other ($p = .73$).

Our second hypothesis for the NSW was that stereotype-consistent stop trials should elicit larger amplitude than stereotype-violating stop trials, but again only in the placebo group. To test this hypothesis, we first combined race and stereotypicality information to create stereotype-consistent and stereotype-violating trials for each participant and then conducted a 3 (dose) × 2 (consistency: stereotype-consistent trials, stereotype-violating trials) ANOVA on the stop trial amplitudes. This analysis showed only the predicted Dose × Consistency interaction, $F(2, 39) = 3.95$, $p < .05$. Figure 3 illustrates the essence of this interaction for the placebo and high-dose groups. Simple effect tests examining the influence of consistency at levels of dose showed that the NSW was significantly larger on stereotype-consistent trials than on stereotype-violating trials in the placebo group (*M*s = −8.34 and −6.04 μV, respectively), $t(14) = 2.32$, $p < .05$, but not in the moderate-dose (*M*s = −2.73 and −4.29 μV, respectively), $t(13) = 1.62$, $p > .10$, and high-dose groups (*M*s = −3.42 and −3.51 μV, respectively), $t(12) = 0.05$, $p > .50$. Moreover, the linear effect of dose on stereotype-consistent trials was significant, $F(1, 39) = 5.00$, $p < .01$, whereas the dose effect on stereotype-violating trials was not, $F(1, 39) = 1.99$, $p > .15$. Although this analysis was restricted to frontal and fronto-central locations,

ancillary analyses using data from all electrodes produced a nearly identical pattern of results: trial type main effect, $F(1, 39) = 10.77$, $p < .01$; Dose × Consistency interaction, $F(2, 39) = 4.08$, $p < .05$).[5]

We have argued that NSW amplitude reflects the extent to which cognitive control processes are brought to bear on inhibition of behavior. If so, NSW amplitude should be larger on successfully inhibited stop trials than on stop trials in which inhibition fails. We tested this idea by using a 3 (dose) × 2 (trial type: successful inhibition trials; inhibition error trials) × 2 (scalp site) × 3 (electrode within scalp site) ANOVA.[6] This analysis produced the predicted main effect of trial type, $F(1, 31) = 4.26$, $p < .05$, indicating that NSW amplitude was larger on successfully inhibited stop trials ($M = -5.01$ μV) than on inhibition error trials ($M = -2.46$ μV), and a Type × Scalp site interaction, $F(1, 31) = 5.57$, $p < .05$, indicating that this effect was largest at frontal scalp locations.

Linking ERPs With Inhibition of Race Bias

The analyses presented thus far have suggested that effective cognitive control, as indexed by NSW amplitude, is necessary for inhibition of race-biased responses. In contrast, the no-go N2 appears to be a neural marker for conflict associated with activation of unintended response tendencies. To further explore links between these ERP components and response activation versus inhibition, we computed a series of zero-order correlations between NSW and no-go N2 amplitudes at all scalp locations and inhibition errors on stereotype-consistent (i.e., race-biased) trials. These correlation coefficients are presented in Table 1.[7] To the extent that amplitude of these negative components reflects inhibitory processes, correlations between their amplitude and the frequency of inhibition errors should be positive. That is, as the amplitude of the component becomes more negative (i.e., larger), disinhibition should decrease. As seen in Table 1, this is the pattern observed for the NSW. In contrast, the pattern for the no-go N2 is negative, suggesting that as this component gets larger, disinhibition increases. To further explore the basis for this negative correlation, we submitted no-go N2 amplitudes on successfully inhibited and inhibition error stop trials to an ANOVA, similar to the one reported previously for NSW amplitudes. This analysis produced a significant effect of trial type, $F(1, 31) = 11.99$, $p < .01$, but the pattern of means was opposite that found for the NSW: Inhibition error trials were associated with larger N2 amplitudes ($M = -2.65$ μV) compared with successful inhibitions ($M = 0.17$ μV). This pattern of results provides further support for our contention that the NSW reflects engagement of cognitive control, whereas the no-go N2 reflects conflict between response activation processes and the need to inhibit responses on stop trials (also see Kok et al., 2004).

To the extent that inhibition of stereotype-based responses depends on cognitive control processes as indexed by the NSW, the significant association between alcohol consumption and inhibition errors on stereotype-consistent trials should be at least partially mediated by the amplitude of the NSW. We tested this assumption by using a series of regression equations (Baron & Kenny, 1986). First, alcohol (BAC) significantly predicted both NSW amplitude (the mediator; $r = .36$, $p < .05$) and inhibition errors ($r = .69$, $p < .01$). To establish mediation, it is necessary to further establish that NSW is significantly associated with inhibition errors (it was; $r = .45$, $p < .01$) and that statistically controlling for NSW reduces the effect of BAC on inhibition errors. The results of this final model indicated that controlling for NSW significantly reduced the association between BAC and inhibition errors, producing a residual (partial) correlation of .40 ($p < .05$). A Sobel test (see MacKinnon, Warsi, & Dwyer, 1995) indicated that the indirect effect of BAC on inhibition errors via NSW was significant ($z = 1.95$, $p = .05$), indicating that NSW amplitude partially mediated the effects of alcohol on inhibition errors.

Table 1
Correlations Between Inhibition Errors and NSW and No-Go N2 Amplitude on Stereotype-Consistent Trials, Experiment 2

	Laterality of scalp locations		
Scalp region	Left	Midline	Right
Frontal	*F3*	*Fz*	*F4*
NSW	.56**	.43**	.39**
No-go N2	−.19	−.39**	−.23
Fronto-central	*FC3*	*FCz*	*FC4*
NSW	.39**	.41**	.29†
No-go N2	−.27†	−.42**	−.28†
Central	*C3*	*Cz*	*C4*
NSW	.35*	.33*	.23
No-go N2	−.23	−.37*	−.24
Centro-parietal	*CP3*	*CPz*	*CP4*
NSW	.24	.46**	.27†
No-go N2	−.16	−.29*	−.10
Parietal	*P3*	*Pz*	*P4*
NSW	.21	.30*	.17
No-go N2	−.06	−.16	−.02

Note. $N = 42$. Numbers in the table represent correlation coefficients between the frequency of inhibition errors and the respective event-related brain potential (ERP) components at each scalp location. Positive correlations indicate that as the component amplitude increases (becomes more negative), the frequency of inhibition errors decreases. Alphanumeric labels in the table represent the nomenclature for specific electrodes (American Electroencephalographic Society, 1991) from which the ERP components were measured. NSW = negative slow wave.
† $p < .10$. * $p < .05$. ** $p < .01$.

[5] We used a hypothesis-testing approach to simplify analyses and reduce the likelihood that significant effects could result simply from Type I errors. It should be noted, however, that analyzing the NSW data with the full ANOVA design including all relevant factors produces the same results. Specifically, a significant Dose × Trial Type × Race of Prime × Stereotypicality interaction is produced, $F(2, 39) = 4.39$, $p < .025$, the essence of which is captured by the Dose × Consistency interaction on stop trial amplitudes presented in the main text and Figure 3.

[6] Only participants who had made inhibition errors were included in this analysis (*n*s = 11 for placebo, 11 for moderate, and 12 for high dose). An additional analysis using data from all electrodes produced the same result, with a somewhat larger F value (5.80, $p < .025$) for the trial type main effect.

[7] We present these correlations collapsing across dose because the processes linking these ERP components to behavior should be the same regardless of potential impairment by alcohol. Secondary analyses in which these correlations were computed separately by dose groups showed a similar pattern across groups, but the coefficients were not significant within each dose group due to small sample sizes.

Discussion

The findings from Experiment 2 provide some of the first evidence directly linking the regulative component of cognitive control to inhibition of race-biased responses. As in the first experiment, participants in all dose groups showed evidence of automatic stereotype activation following exposure to race cues. However, on trials requiring the recruitment of cognitive control to withhold race-biased responses, alcohol produced significant dose-dependent impairment in terms of both behavioral disinhibition and an electrocortical measure reflecting engagement of cognitive control processes (the NSW; e.g., Curtin & Fairchild, 2003; West & Alain, 1999, 2000). This pattern of findings is consistent with the more general notion that alcohol impairs controlled processes while leaving more automatic processes relatively intact (e.g., Fillmore et al., 1999). Moreover, this pattern suggests that alcohol's impairment of behavioral inhibition has implications for stereotype-based intergroup cognitions and behaviors. However, readers are cautioned that because analyses of the ERP data from this experiment involved a rather large number of factors, the possibility that some effects are due to Type I error is increased.

In addition to their implications for theories of race bias, the current findings also bear on current debates concerning the ERP components underlying inhibition of behavior. The current data are consistent with recent interpretations of the no-go N2 as an index of conflict associated with unintentional activation of responses on stop trials (e.g., Bruin et al., 2001; Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003). No-go N2 amplitude was larger on trials where habitual stimulus-response mapping would be expected to produce the strongest response activation (i.e., stereotype-consistent trials) and thus the strongest degree of conflict in withholding a response. Also, amplitude of the no-go N2 was inversely correlated with the ability to withhold a race-biased response, suggesting that this component does not reflect motor-related inhibitory processes. Moreover, the lack of significant alcohol effects on the N2 suggests that response activation conflict (associated here with racial stereotype activation) is not affected by alcohol.

General Discussion

The control of racial discrimination has been a major social policy goal for more than a century (e.g., DuBois, 1903). Only very recently have researchers begun to examine the role of cognitive control in the inhibition of discriminatory behavior (e.g., Amodio et al., 2004; Lambert et al., 2003; Payne, 2001; Payne et al., 2005). Studying factors associated with inhibition of race bias from this perspective is important because doing so can provide evidence for behavioral inhibition that does not depend on conscious control processes (e.g., Monteith & Voils, 2001) or individual differences in levels of motivation to control prejudice (e.g., Plant & Devine, 1998). The current findings show that inhibition of race-biased responding depends on the regulative component of cognitive control. In so doing, this research advances current theories by linking issues of stereotype-based response inhibition to the larger literature on cognitive control and behavioral regulation (e.g., Logan, 1994).

Neural Correlates of Stereotype Activation and Inhibition

Researchers have long proposed cognitive mechanisms to explain stereotyping and prejudice (for reviews, see Bodenhausen & Macrae, 1998; Fiske, 1998). The current studies replicated the basic finding of response facilitation for stereotype-consistent information (e.g., Dovidio et al., 1986, 1997; Gaertner & McLaughlin, 1983; Lepore & Brown, 1997; Macrae, Stangor, & Milne, 1994) but importantly extended this earlier work by demonstrating that P300 latency, arguably a purer index of categorization time (Kutas et al., 1977; McCarthy & Donchin, 1981; Smid et al., 1992), also shows enhanced efficiency of processing stereotype-consistent information. Given the superior temporal resolution of ERP measures, P300 latency also provides an advantage over other brain imaging techniques, such as functional MRI, for examining the timing of neural events associated with stereotype activation. Additionally, P300 amplitude was shown to be sensitive to violations of activated stereotypes. These findings, along with other recent work (e.g., Ito et al., 2004), demonstrate that researchers interested in implicit stereotyping effects who wish to separate relevant information processing from irrelevant response preparation and execution can benefit from adopting electrocortical measures into their paradigms.

The current research also showed that the NSW, a component of the ERP thought to be associated with implementation of cognitive control, significantly covaries with successful inhibition of race-biased responses. Moreover, the NSW was significantly larger when participants successfully inhibited a stereotype-consistent response than when they withheld a stereotype-violating response, but only when cognitive control was intact (i.e., in the placebo group). Taken together, this pattern of results indicates that successful control over prepotent, racially biased responses depends on intact regulative control mechanisms and that these mechanisms specifically—and not response-activation mechanisms—are significantly impaired by a moderate dose of alcohol. These findings extend recent research (Amodio et al., 2004) demonstrating that neural detection of race bias does not ensure unbiased responding. That the pattern of correlations between NSW and inhibition of stereotype-consistent responses was generally strongest over frontal areas of the cortex is consistent with brain-imaging data indicating that the dorsolateral and ventrolateral prefrontal cortex are critically involved in executive cognitive functions subserving control of behavior in general (e.g., Botvinick et al., 2001; Gray & Burgess, 2004; MacDonald et al., 2000; Mecklinger, Weber, Gunter, & Engle, 2003), and racially biased responses (Richeson et al., 2003) and behavioral inhibition (Braver, Barch, Gray, Molfese, & Snyder, 2001) more specifically. In addition, that this activity was partially left lateralized is consistent with brain-imaging data showing greater left prefrontal activation associated with engagement of control processes in go/no-go tasks (Garavan, Ross, Murphy, Roche, & Stein, 2002) and in conflict tasks involving verbal stimuli (MacDonald et al., 2000). However, this potential hemispheric difference should be interpreted with caution in the absence of brain-imaging data.

We have argued that the inverse correlation between no-go N2 amplitude and inhibition of biased responses seen here supports other recent work suggesting that the no-go N2 reflects conflict associated with activation of responses on stop trials (e.g., Nieuwenhuis et al., 2003). When considered together along with the results reported by Amodio et al. (2004), this finding suggests that even though participants appear to have detected the conflict inherent in the need to withhold racially biased responses in this study, they were only able to do so if regulative cognitive control was intact.

Effects of Alcohol on Stereotyping and Prejudice

There are a number of reasons for examining how processes underlying race bias are affected by alcohol. Perhaps the most obvious of these is to increase understanding of intoxicated social behavior. Despite major advances in understanding links between alcohol use and other interpersonal processes (e.g., aggression and sexual risk taking), alcohol's effects on many other social processes are largely unknown. A more general reason for studying alcohol's effects is to increase understanding of the basic mechanisms underlying social processes by examining how they change when aspects of cognition are temporarily impaired by alcohol (see Bartholow et al., 2003).

The current findings indicate that alcohol does not influence stereotype activation but influences the ability to regulate prepotent stereotype-related responses via impairment of cognitive control. Stereotype-consistent responses are considered dominant because of their strong stimulus-response mapping to racial category cues (e.g., Lambert et al., 2003) and are therefore naturally facilitated. In contrast, stereotype-violating responses are naturally inhibited because their association with racial category cues in semantic memory is weaker (e.g., Macrae, Stangor, & Milne, 1994), and therefore, cognitive-control demands to inhibit those responses are not as great. Alcohol specifically impaired the ability to inhibit stereotype-consistent responses, a pattern mirrored in the amplitude of the NSW. Taken together, these findings suggest that alcohol's effects on social cognition are limited to or are expressed most strongly under circumstances in which cognitive control demands are high. The current findings also link the effects of alcohol on racially biased responding to other manipulations intended to weaken cognitive control (e.g., Govorun & Payne, 2004; see also Richeson & Shelton, 2003).

Limitations and Future Directions

Some shortcomings of this work should be noted. First, although the current findings are informative concerning the control of race bias, they do not directly address the control of prejudice (see Monteith & Voils, 2001). In Experiment 1, the effects of race primes were limited to the stereotypicality of trait words (i.e., semantic associations between race primes and targets) and did not generalize to their valence (i.e., evaluative differences associated with race), a finding inconsistent with some other reports (e.g., Dovidio et al., 1986; Fazio et al., 1995).

Second, given the current findings, it would be tempting to conclude that people are necessarily more biased when under the influence of alcohol than when sober. However, it is important to acknowledge that the cognitive control approach taken here may not adequately account for motivational processes, which also are known to play a key role in controlling race bias (e.g., Devine & Monteith, 1999; Monteith & Voils, 2001; Plant & Devine, 1998); research shows that even the impairing effects of alcohol can be largely overcome with sufficient motivation for proper inhibitory control (e.g., Fillmore & Vogel-Sprott, 1999). These data suggest future work examining, for example, whether individuals with strong internal motivation to control prejudice (e.g., Plant & Devine, 1998) would show resilience to alcohol's effects, in terms of both behavioral and electrocortical measures of control.

As noted previously, that priming stimuli were presented within conscious awareness may be considered a limitation in terms of separating automatic and controlled processes. However, we feel that the confluence of evidence from both electrocortical measures (which arguably do not require perceivers' conscious control) and behavioral measures corroborates our claim that conscious, explicit presentation does not necessarily invoke controlled processes (see Bargh, 1996). Nevertheless, future researchers may wish to test the generalizability of the current findings by using a procedure in which prime stimuli are presented outside of awareness (e.g., Bargh et al., 1996; Devine, 1989).

Our findings concerning the no-go N2 and its apparent connection to conflict associated with response activation warrants further study. In the context of the larger behavior regulation system, conflict detection and implementation of cognitive control should work in concert (see Botvinick et al., 2001). However, our data suggest that increased conflict does not necessarily lead to increased control. Rather, when conflict associated with response activation tendencies is too great, efforts at control can fail. More work is needed to better understand the relationship between these two components of control in the context of behavioral inhibition. That alcohol did not appear to influence the N2 is consistent with the interpretation of the N2 as reflecting response activation tendencies (Bruin et al., 2001). Nevertheless, it will be important to replicate this finding before strong conclusions can be drawn.

The current work also suggests future research examining whether, for example, high-prejudice individuals have difficulty regulating cognitive control. That is, in addition to focusing on how prejudice level correlates with differences in stereotype activation processes (Lepore & Brown, 1997, 2002), researchers may wish to examine differences in cognitive control processes among individuals differing in explicit prejudice. Along these lines, Locke, MacLeod, and Walker (1994) found that high-prejudice participants experienced significant interference when naming the color of stereotype-related words relative to stereotype-unrelated words in a Stroop color-naming task; low-prejudice participants' performance was unaffected by word stereotypicality. More research is needed to determine whether high-prejudice individuals show neural evidence of weaker inhibitory control over biased responding compared with low-prejudice individuals (see Amodio, 2004).

In sum, the current research provides some of the first evidence that regulative cognitive control is involved in the inhibition of race bias and identifies a neural mechanism supporting its operation. When considered alongside other recent work (e.g., Amodio et al., 2004; Payne et al., 2005), these studies represent an exciting new direction for research on race bias, with issues pertaining to cognitive control as a central theme.

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