# ALCOHOL PLACEBO EFFECTS ON COGNITIVE CONTROL OF RACE BIAS: INVESTIGATING NEURAL MECHANISMS

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by

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The undersigned, appointed by the Dean of the Graduate School, have examined the thesis entitled

ALCOHOL PLACEBO EFFECTS ON COGNITIVE

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Presented by Joseph Hilgard

A candidate for the degree of Master of Arts in Psychology

And hereby certify that in their opinion it is worthy of acceptance.

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## Abstract

While alcohol is broadly understood to impair control over behavior, the neurocognitive mechanisms of this impairment are still unclear. It is possible that alcohol leads to greater activation of automatic responses, reduced detection of the likelihood of making a mistake, or impaired ability to exercise cognitive control once it is recruited.

In the present study, participants consumed either an alcohol placebo (0.04 g/kg EtOH) or a control beverage (0.00 g/kg EtOH). Participants then performed the Weapons Identification Task (WIT) (Payne, 2004) while brain activity was measured via fMRI. On each trial of the WIT, participants see a White face or Black face followed by a gun or a tool. The activation of racial bias requires participants to exercise control over their responses in order to avoid making biased responses. Participants in the placebo condition compensated for their purported intoxication by exercising increased cognitive control over their responses, making responses less influenced by the racial primes. Additionally, placebo participants, compared to control participants, showed reduced interference from racial cues after errors, suggesting more effective preservation of control in spite of error commission.

In the fMRI data, we observed increased activation of the dorsal anterior cingulate cortex in response to error commission, leading to increased activity of the middle frontal gyrus and middle temporal gyrus on the trial following an error, replicating the model of

conflict detection and the recruitment of control described in Kerns et al., 2004. Finally, placebo participants relative to controls exhibited increased recruitment of a number of regions following an error which include the medial and lateral prefrontal cortex.

*Keywords:* Alcohol, fMRI, automatic associations, cognitive control, executive function

## Introduction

It is well established that alcohol has a number of effects on social behavior. Acute alcohol intoxication has been shown to lead to disinhibition of behavior, leading to increased sexual risk-taking (Cooper, 1992), aggression (Bushman & Cooper, 1990; Chermack & Giancola, 1997), reduced anxiety (Kushner et al, 1996), and increased expression of racial prejudice (Reeves, 1993; Schlauch et al, 2008; Bartholow et al, 2006). It has been proposed that these are mediated by a reduction of self-awareness (Hull, 1983) or by an impairment in attention to inhibitory (Taylor & Leonard, 1983) or less salient (Steele & Josephs, 1990) cues.

Most models (see Giancola, 2000) point to cognitive impairment as the cause of these changes in behavior. More specifically, it is suspected that alcohol's pharmacological effects on cognitive control are responsible for these social and behavioral effects. Alcohol's ability to impair cognitive control is well known and leads to failure to suppress prepotent responses and automatically activated biases (Curtin & Fairchild, 2003). To date, specific mechanisms for alcohol's impairment of cognitive control remain unclear. It is not completely certain whether this effect is due to a narrowing of attentional focus on the most salient and immediate attributes of a stimulus, the anxiolytic effects of alcohol reducing the sensation of conflict (Bartholow et al, 2010), unawareness of the need for control (Ridderinkof et al, 2002), or other psychopharmacological means.

## Cognitive Control

Cognitive control is a higher-order cognitive construct involved in the planning, initiation, and regulation of goal-directed behavior (Luria, 1980; Milner, 1995). It is understood to include attentional control, strategic planning, response sequencing, self-monitoring, and the inhibition of prepotent responses. It is also understood to provide the means by which the human brain is able to account for the *contextual* or *associative* importance of stimuli.

Many psychological models of control have conceptualized behavior as being influenced by some variety of dual-process model marked by (1) an automatic component that is rapid, effortless, and unintentional, composed of some form of automatic association, prepotent response bias, emotion, or heuristic processing, and (2) a controlled component that is slower, effortful, and intentional, involving the conscious processing of stimuli to determine appropriate responses and the inhibition of inappropriate responses (Jacoby, 1991; Lindsay and Jacoby, 1994; see a similar model from Conrey et al., 2005). Much of current psychophysiological research on cognitive control has used these models to provide an estimate of controlled processing and look for associated neural activity (Sherman et al., 2008; Beer et al., 2008; Amodio, 2008).

## Recruiting Control

Theoretically, it is not enough to be able to only execute control – there must also be some cognitive process that detects when control is necessary. Presently, it is hypothesized that there are two separate cognitive processes involved in the execution of cognitive control: one process for determining when control is needed, and one process

for implementing the intended behavior (Botvinick, Braver, Barch, Carter, & Cohen, 2001). The detection of conflicts that signal need for control has been associated with the dorsal anterior cingulate cortex (dACC), while the implementation of controlled behavior has been linked to activity in the dorsolateral prefrontal cortex (dlPFC) (van Veen & Carter, 2002; Kerns, Cohen, MacDonald, Cho, Stenger, & Carter, 2004). To date, conflict has largely been conceptualized as the coactivation of two competing, mutually incompatible representations or responses (Carter & van Veen, 2007), occuring either before a response is made (signaling need for response inhibition) or after an error is made (both responses were activated and the incorrect one was chosen). So, in tasks such as the Stroop, in which participants must name the color of the ink rather than the word itself, or the Weapons Identification Task, in which participants must periodically inhibit knowledge of racial stereotypes to accurately identify objects, conflict is understood to be the mutual incompatibility of the automatic or prepotent response (reading words, associating Blacks with violence) with the proper intended response (naming colors, accurately identifying objects).

**Anterior Cingulate Cortex**

The dorsal anterior cingulated cortex (dACC) is hypothesized to detect and signal the presence of conflicts in information processing. In an fMRI study of the Stroop task, Kerns et al (2004) demonstrated three important properties of the ACC. First, the ACC is sensitive to conflict, showing greater activation on high-conflict (word-color incongruent) trials than on low-conflict (word-color congruent) trials. A similar sensitivity was shown to errors; the ACC was more active on error trials than on correct trials. Second,

activation of the ACC on one trial was associated with greater activity of the PFC on the following trial, as well as a behavioral adjustment in control. Third, the exercise of control on high-conflict trials reduced the activation of ACC relative to high-conflict trials with less exercised control. These properties provide evidence for a theoretical model of cognitive control in which the dACC detects conflict and activates the dorsal lateral PFC. The dlPFC, in turn, enacts inhibitory control over behavior. Finally, the execution of this control reduces the sensitivity of the dACC to conflict, perhaps by inhibiting one of the two incompatible responses or by redirecting attention to the contextually relevant portion of the stimulus.

Activation of the ACC can also be studied using electroencephalography (EEG). The error-related negativity (ERN) is a negative-going frontal EEG component found within 50-150ms following a response error. Dipole modeling (Dehaene et al., 1994) and fMRI studies (Kiehl et al., 2000) have localized this activity to the ACC. The ERN is sensitive to motivation, with increasing motivational salience causing an increase in ERN amplitude (Gehring et al., 1993; Hajcak, Moser, Yeung, & Simons, 2005). Use of Process Dissociation Procedure analysis (PDP) (Jacoby, 1991) estimates a greater controlled (as opposed to automatic) cognitive component on trials with greater ERN amplitude, suggesting an association between ACC activity and controlled behavior (Amodio, 2008). Additionally, in tasks evoking racial prejudice, participants with greater internal motivation for making egalitarian responses show greater ERN amplitude when making racially biased errors, presumably reflecting their elevated concern over these errors (Amodio et al., 2004, 2008). Whether ERN amplitude is associated with greater

post-error adjustments such as response slowing is still the topic of some debate, with some studies finding a positive correlation between ERN amplitude and post-trial adjustments (Alain et al., 2002; Bartholow et al., under review) and others finding no such relationship (Hajcak et al., 2003). Finally, activity of the ACC, thought to be the neural generator of the ERN component, does not vary by the awareness or lack of awareness of errors (Klein et al., 2007), perhaps confirming the theory that ACC activity represents the detection of processing conflict even at levels preceding conscious thought.

The fronto-central N2 ERP component is another conflict-sensitive psychophysiological sign localized to the ACC (van Veen & Carter, 2002). N2, peaking 200-350ms post-stimulus, is similar to the ERN, in that it reflects the response conflict elicited by perception of the stimulus. If conflict is successfully resolved before a response is made, increased activation is found in the N2; if conflict is not resolved and an error is committed, the ERN increases. Among participants with high internal motivations to inhibit prejudice, Amodio et al. (2004) found not only greater ACC activation after race-biased errors but also greater ACC activation as measured by N2 amplitude *before* the successful control of stereotyping. Thus, it seems possible those who are good at regulating stereotypy are more sensitive to the potential for race-biased errors, and detection of this potential may help recruit control over the potential error.

In keeping with the theory of the ACC's role in the detection of *evaluative* conflict (for a review, see Botvinick et al., 2004), the extent to which error commission causes ACC activity seems to be mediated by negative affect. In a three-group study (active alcohol, placebo alcohol, and control) using the Weapons Identification Task,

Bartholow et al. (under review) demonstrated that alcohol does not impair error detection, but rather it impairs the evaluative reaction to errors by decreasing negative affect. This decrease of negative affect leads to reduced activation of the ACC as measured by the ERN, leading, in turn, to decreased cognitive control (i.e., reduced post-error adjustment). Despite the task's highly motivating social context, intoxicated participants remained aware of their errors but exerted less behavioral control as measured by post-error slowing. Hypothetically, these intoxicated participants would exhibit less PFC activity, if PFC is recruited by the ACC. The placebo group, on the other hand, showed an increase in negative affect, ERN, and post-error slowing, likely due to a compensatory effort to maintain control in the face of alcohol challenge (as in Saults et al., 2007).

By what processes are intoxicated participants detecting their errors but failing to make behavioral adjustments? The anxiolytic effect of alcohol may play a role here, reducing activation of the amygdala, which has been associated with both negative evaluative biases and an imminent interracial interaction (Amodio, 2008). It is also likely that conflict detection relies on negative affect as a cognitive signal of interrupted processing fluency. Before the ACC's role in conflict detection and cognitive control was known, it was understood to play a crucial role in the sensation of pain (Talbot, 1991), and specifically only when pain is evaluated as being distressing (Rainville et al., 1997). The ACC is also sensitive to distress resulting from social exclusion (Eisenberger et al., 2003). The ACC, then, is likely to be a necessary part of what makes error commission aversive (see Hajcak & Foti, 2008) and motivates the use of control.

**Prefrontal Cortex**

The prefrontal cortex's (PFC's) capacity for associative learning is demonstrated by its rich interconnectivity with higher-order sensory and motor cortex and other connections with the limbic system. In animal studies, neurons in the PFC were demonstrated to be more sensitive to the association between the cue and the reward than to either the cue or the reward (Assad, Rainer, & Miller, 1998). This indicates the role of PFC is likely to be more closely associated with the *construal* of stimuli than with any physical property of the stimuli.

The dorsolateral prefrontal cortex (dlPFC) is thought to be the actual executor of control, recruited by the ACC signal elicited by high-conflict or error trials. The PFC is thought to enact inhibitory control over inappropriate responses or interfering memories, and specific activations have been found during conflict tasks such as the Stroop (1935) and Erikson tasks. Additionally, regions of the PFC also seem to be involved in response inhibition in Go/NoGo paradigms (Ridderinkhof et al., 2004). Activity of the PFC has been demonstrated to be significantly associated with post-conflict and post-error behavioral adjustments such as slower responding (Kerns, Cohen, MacDonald, Cho, Stenger, & Carter, 2004).

It has been hypothesized that the polar or dorsolateral region of the PFC is the generator of the Negative Slow Wave (NSW) component of the ERP (West and Alain, 2000). The NSW is usually observed 600-1200ms post-stimulus and is largest at frontal and central sites. In keeping with the theory that the NSW is a sign of phasic PFC activation and control, inebriated participants made more errors on the incongruent

condition of a Stroop task and demonstrated smaller mean NSW amplitude relative to controls (Curtin and Fairchild, 2003). Furthermore, in a Go-Stop task, the amplitude of NSW was greater on successful inhibition trials than on inhibition error trials, suggesting that the amplitude of NSW seems to reflect the degree to which cognitive control has been successfully activated (Bartholow, Dickter, Sestir, 2006).

Given the combined evidence from fMRI and ERP studies, the PFC would appear to be the final executor of cognitive control, with transient changes in activity predicting successful or unsuccessful suppression of inappropriate responses. It seems likely that this stage of control is compromised by alcohol, although the effect may begin in earlier modules such as at the level of conflict detection in the ACC.

**Controlling Prejudice**

Interactions with outgroup individuals are characterized by a number of difficulties. Foremost among these difficulties is the automatic activation of prejudicial evaluations and stereotypes. This activation of prejudicial thoughts is persistent and automatic, often despite our best efforts and motivations to the contrary (Amodio, 2008). Knowledge and at least some activation of stereotype are demonstrated in even the most egalitarian of individuals (Devine, 1989), even if there may be individual differences in the magnitude and content of stereotype activation (Lepore & Brown 1997; Locke et al 1994; Wittenbrink et al 1997). Despite our best intentions, racial cues are capable of semantic priming and affecting behavior. At best, when these thoughts are successfully inhibited they can still cause anxiety and discomfort; at worst, these automatic

associations may have a role in the accidental shootings of unarmed minorities by policemen (Correll, Park, Judd, & Wittenbrink, 2002).

Since preventing the activation of racially biased thoughts seems impossible, we must self-regulate in order to impose inhibitory control over these automatic thoughts. While the ease and automaticity of this process does vary among individuals, self-regulation of prejudicial thoughts and behaviors is an effortful process. It can be compromised by cognitive load, rapid response deadlines, or even alcohol.

The Quadruple Process model (Conrey et al., 2005) has been applied to understanding previous research involving implicit forms of racial bias and associated activations in paradigms such as the IAT and WIT. This model breaks the automatic and controlled components of decision-making into four parts: the activation of an association (AC), the detection of an appropriate response (D), overcoming a bias or prepotent response (OB), and guessing (G). Using the quad model to estimate the influence of the different automatic and controlled processes, participants understood to be highly egalitarian (high IMS/low EMS) as compared to less egalitarian (low IMS) demonstrated less activation of biased associations (AC), as well as a greater ability to detect appropriate responses (D). Perhaps surprisingly, there was no observed difference in the ability to overcome bias (OB) between participant groups. (Sherman et al, 2008) These results should be taken with a grain of salt, because there seems to be a recurring weakness of this model when it comes to proper estimation and power of the OB parameter – suffice it to say that the egalitarian group demonstrated signs of decreased stereotype activation and some form of increase in controlled processing.

In another study of the IAT, Beer et al. (2008) used fMRI to detect brain activity associated with each of the four processes. They found significant activation in the ACC and dlPFC associated with the detection parameter of the model (D) and on stereotype-incongruent trials relative to stereotype-congruent trials. Significant activation of the insula and operculum was also found in association with D. Unfortunately, while one might hypothesize that the Overcoming Bias (OB) process would be particularly related to the dlPFC, no significant physiological correlations were detected, again due to the poor power associated with the estimate of OB. Regardless, the association of ACC and dlPFC with controlled processing of stereotype remains clear. It would seem that the recruitment and exercise of cognitive control works through similar neurological processes in both classical cognitive control paradigms like the Stroop and social cognitive paradigms like the IAT and WIT.

**Feasibility of alcohol fMRI research**

The current project sidesteps the potential difficulties of alcohol and fMRI by using only an alcohol placebo. Alcohol placebo has not been found to cause vasodilation. However, a brief review of the literature is pertinent, since the inclusion of an active alcohol condition in future projects could lead to powerful and informative research.

To date, research involving active alcohol and fMRI has been limited, but promising. A chief concern of alcohol fMRI paradigms is that alcohol is a vasodilator and causes blood vessels to expand as the smooth muscular linings relax (Gillespie, 1967). This vasodilatory effect is likely the cause of increases in global cerebral blood flow (CBF) following the acute administration of alcohol (Newlin et al., 1982; Mathew

and Wilson, 1986, 1991). Additionally, alcohol has been reported to cause a variety of regionalized increases and even decreases in CBF (Volkow et al., 1988; Sano et al., 1993). Since fMRI records brain activity indirectly by measuring regionalized changes in oxygenated blood flow, it is possible that this could be a source of artifact in the BOLD data. These effects are not tied to alcohol expectancy, and participants dosed with an alcohol placebo show no changes in CBF (Levin et al, 1998).

It is also possible that slow changes in regional CBF, such as those brought about by alcohol, would not affect the BOLD contrast in a meaningful way. Using $H_2^{15}O$ PET, Shimosegawa et al. (1995) examined changes in regional CBF to visual stimulation across different breathing conditions. Participants' levels of blood CO2 were experimentally manipulated during a PET scan of the brain. When blood CO2 was reduced, blood vessels constricted and CBF decreased. When blood CO2 was increased, vessels dilated and CBF increased. Visual stimulation caused brain activity of visual processing areas. This led to transient increases in regional CBF, as it does in BOLD fMRI, and the magnitude of the absolute change in CBF was proportional to the baseline CBF at the time of stimulation. Importantly, the *relative* increase in CBF stayed the same across conditions. Therefore, it seems unlikely that there is an irreconcilable confound between the vasodilatory and psychoactive effects of alcohol, since the relative change in CBF associated with brain activity was the same across vasodilation conditions.

In two studies of fMRI, researchers examined the changes in BOLD contrast following alcohol administration in visual perception paradigms (Levin et al., 1998; Calhoun et al., 2004). Both have claimed to find both global and regional effects of

alcohol on the BOLD signal, suggesting that the chronic changes in bloodflow due to vasodilation could be disentangled from the tonic changes in hemodynamics caused by task-related activity, making fMRI study plausible during active alcohol challenge.

**Summary**

The role of cognitive control in inhibiting stereotypical associations and racial bias is clear. Furthermore, this social-cognitive process relies upon the same structures responsible for executive control in "purely cognitive" tasks such as the Stroop paradigm – the dACC for the detection of conflict, and the dlPFC for the controlled resolution of conflict. It is theorized that individual differences in behavior on tasks such as the IAT and WIT is due to some combination of factors, including stereotype activation, stereotype contents, ability to exercise control, and motivation to exercise control.

Alcohol provides a particularly useful way to investigate cognitive control. First, it is socially relevant, being perhaps the most frequently and socially used drug. Secondly, it compromises specifically inhibitory control processes while leaving intact automatic activation, motivation, interpretation, and implementation (Easdon & Vogel-Sprott, 2000). These properties of alcohol make it particularly suited for research on stereotypy and the processes by which cognitive control is recruited to inhibit automatically activated information and response biases.

In the proposed study, participants will consume either a control beverage or an alcohol placebo. Personal motivations to avoid making prejudiced responses will be measured by the IMS/EMS. Activation of the dACC and dlPFC will be measured during performance of the Weapons Identification Task. Given the importance of dACC and

dlPFC in regulating bias, and given that alcohol placebo appears to modulate recruitment of control, the following hypotheses were advanced for this study: First, high-conflict and error trials will cause activation of the dACC. Activation of the dACC will lead to activation of the dlPFC during the following trial, leading, in turn, to greater behavioral control. Next, alcohol placebo will cause a compensatory increase in control and conflict detection, particularly among participants with high IMS scores. This increase should manifest itself as increased dACC activity specifically in response to high-conflict and error trials, leading to greater dlPFC activity and post-error and post-conflict behavioral adjustments. In this way, we can observe the neurocognitive mechanisms responsible for control over bias and their modulation as a function of motivation and alcohol expectancy.

## Method

### Participants

Thirty-four healthy participants (12 women) ages 21-35 (M = 22.5) participated for $15 an hour. All participants were predominantly right-handed. Only In order to maintain racial homogeneity of the sample and to facilitate comparisons with the extant literature, only White/Caucasian individuals were recruited.

Individuals who indicated conditions that would contraindicate participation in an alcohol challenge (alcohol abstention, symptoms of alcohol or drug dependence, history of serious mental or physical illness, prescription medication other than oral contraception, pregnancy) were excluded from the sample. Participants needed to report drinking an average of 2-24 drinks per week in the past 3 months. Participants abstained from alcohol and drugs for 24 hours to their appointment. Participants were instructed to eat a light meal 4-6 hours prior to their arrival at the lab.

### Self-report Measures

**Demographic information.** Participants reported their age, gender, and race. Participants also reported whether they had consumed alcohol, tobacco, or caffeine in the past four hours, how many hours of sleep they'd had the night before, and how many hours of sleep they get in a typical night. Participants reported the environment they were raised in (urban, suburban, or rural) and a number of Likert-scale items regarding their history of interactions with Blacks (i.e. "Did you know many Blacks when you were growing up?" "Of the Blacks you knew, how many would you consider close friends?").

Participants also reported how aggressive, violent, and dangerous they felt White Americans and African Americans were.

**Positive and Negative Affect Scales.** The Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988) is a brief questionnaire composed of two 10-item mood scales which measure separable factors of positive and negative affect. Using Likert-type ratings, participants report the extent to which they are experiencing a variety of positive (i.e. "Strong", "Active") and negative (i.e. "Guilty", "Nervous") emotions.

**Motivation to Respond Without Prejudice.** Plant and Devine's (1998) Internal (IMS) and External (EMS) Motivation to Respond Without Prejudice Scales measure individual differences in the extent to which people attempt to control racial prejudice when interacting with and making judgments about others, for both internal and external reasons. The IMS measures the degree to which participants attempt to avoid using stereotypes when making social judgments because of internal goals to be egalitarian ($\alpha$=.81). The EMS measures the degree to which participants are motivated by external sources, such as the potential social stigma that results from appearing bigoted ($\alpha$=.80). Together, the scales form a single 10-item questionnaire, with items rated from 1 (strongly disagree) to 9 (strongly agree). This measure was included because individual differences in these motivational factors has been shown in previous research to modulate ACC activation following race-biased errors in the WIT (Amodio et al., 2008) and may affect participants' use of automatic and control-related processes following consumption of an alcohol placebo (Schlauch et al., 2009).

**Weapons Identification Task (WIT)**

The WIT (Payne, 2001) measures racial bias by assessing the extent to which priming with pictures of white versus black men's faces affects the speed and accuracy with which participants can identify handguns (i.e., threat-related objects) and hand tools (i.e., harmless objects). Stimuli used in this task include pictures of four handguns, four tools, four white male faces, and four black male faces. Images are digitized at 228 x 172 pixels. On each trial of the WIT, participants are first primed with either a white face or black face prime for 200ms, then shown a tool or gun target for 200ms, which is subsequently covered by a pattern mask. Participants are asked to identify the object as being either a tool (left hand) or gun (right hand, counterbalanced across participants). The post-target pattern mask remains on the display until either the participant makes a response or 2000ms elapse. On trials which the participant fails to press a button within 500ms, the post-target pattern mask turns red, letting the participant know he or she was too slow on that trial. A schematic of the WIT is given in Figure 1.

Previous research indicates that participants make more errors on Black-tool trials and make faster responses on Black-gun trials, consistent with the idea that implicit biases (i.e., stereotypes linking young Black men with violence) shape performance in the task (e.g., Lambert et al., 2003; Payne, 2001, 2005; Payne, Shimozu, & Jacoby, 2005). As in previous research, participants were informed that errors on black-tool and on white-gun trials are indicative of race bias. These instructions have been demonstrated to increase the accessibility of racial bias and cause more errors on stereotype-incongruent

trials (Payne et al., 2002; Schlauch et al., 2009). This task has been found to be highly socially motivating, and participants invest considerable effort attempting to overcome their innate biases. While the task does not give accuracy feedback, participants have no trouble recognizing their errors.

Participants first performed a single practice block of 24 trials. Participants with an excess of responses slower than 500ms repeated the practice until their reaction times improved. Following the practice, participants performed six blocks of the task while BOLD images were acquired with the fMRI. (FOV: 256mm X 256mm, TR/TE: 1700/28ms, Slice thickness: 4mm, Slice number: 27, Flip angle: 90°, Resolution: 64x64, Voxel size: 4mm x 4mm x 4mm) Each block of the task featured 16 trials of each face-object pairing; each block lasted about six minutes. The inter-trial interval (ITI) was jittered such that there were 0ms, 2000ms, or 4000ms of fixation between trials. This is expected to increase the total variability of the BOLD data, increasing analytical power.

**Beverage Administration**

To ensure that participants abstained from alcohol prior to the study, all participants received a test of breath alcohol concentration (BrAC) immediately prior to receipt of the beverage. Participants with a BrAC above .00 were dismissed (one participant was rescheduled and another was excluded from the study). Female participants were also asked to take a pregnancy test and, in the case of a positive test, excluded from the study (no participants).

Participants were randomly assigned to one of two beverage conditions. Participants in the control group consumed a beverage of tonic water which they knew

contained no alcohol. Participants in the placebo condition consumed a beverage consisting mostly of tonic water but including a trivial amount of alcohol (0.04 g/kg ethanol), and were told that the beverage contained "a moderate amount" of alcohol. Women in the study were asked to self-administer a pregnancy test before receiving the beverage, and in the event of a positive test, excluded from the study.

In both beverage conditions, the beverage was poured in front of the participant. To bolster the placebo cover story, the placebo vodka (90% decarbonated tonic water; 10% 100-proof vodka) was kept in and poured from a Smirnoff 100-proof vodka bottle. To achieve the placebo alcohol dose, the placebo vodka is mixed with tonic water in a 5:1 tonic to placebo vodka ratio, yielding a beverage that is only trivially alcoholic. Participants were invited to add a small amount of cranberry or lime juice to improve the taste of the beverage. The control group consumed an isovolemic beverage of tonic water. In both groups, participants received their beverage in three servings. Participants had ten minutes to consume each serving.

**Functional Magnetic Resonance Imaging (fMRI)**

Brain imaging was carried out using a Siemens Trio 3T scanner. Upon entrance to the scanner, an initial localizer scan made certain that the participant's head was properly oriented in the scanner (TR/TE: 8.6/4.0ms, Flip Angle: 20°, Thickness: 7.0mm, Sagittal alignment). Then, two high-resolution anatomical brain images were taken so as to align the functional brain images across participants. These were one high-resolution T1-weighted image (TR: 2400ms, TE: 3.16ms, Flip Angle: 8°, 176 slices, Thickness: 1.0mm, Sagittal alignment) and one high-resolution T2-weighted image (TR: 3200ms,

TE: 455ms, 176 slices, Thickness: 1.0mm, Sagittal alignment). During this time, participants watched the TV show *Planet Earth* so as to remain alert and reduce fatigue and boredom.

During task performance, brain activity was measured with a T2*-weighted blood-oxygenation-level-dependent (BOLD) scan. (FOV: 256mm X 256mm, TR/TE: 1700/28ms, Slice thickness: 4mm, Slice number: 27, Flip angle: 90°, Resolution: 64x64, Voxel size: 4mm x 4mm x 4mm)

**Procedure**

Upon arrival to the brain imaging center for their scheduled session, participants were greeted by an experimenter who escorted them to a private room to read the consent form and, following informed consent (no participants declined to give consent), to complete a brief interview to confirm their fMRI safety. Next, participants completed the questionnaire measures, including a baseline PANAS assessment, during which the experimenter randomly assigned the participant to a beverage condition and determined the appropriate amount of beverage. Following completion of these measures, participants were administered their beverages. Following completion of their final drink, participants sat idle for 5 min (ostensibly to allow alcohol absorption for those in the placebo condition), and then were given a second BrAC assessment and a second PANAS.

Next, and following a security procedure to ensure that participants had no ferrous metal on their person, participants were escorted into the scanner room. Next, an experimenter read the instructions for the WIT and participants performed a practice

block on their own. Participants then performed six blocks of the WIT. After participants completed all blocks of the WIT, two additional "resting-state" scans measured spontaneous brain activity while the participant laid at rest with eyes closed.

Participants were then removed from the scanner, and PANAS and BrAC were measured for a third time. Participants then completed a brief set of post-experiment questionnaire items that asked participants how intoxicated they felt at various times throughout the experiment, to what degree they felt their performance on the task was affected by the beverage they consumed, and (for those in the placebo condition) how many standard drinks of alcohol they believe to have consumed at the start of the experiment.

Following the completion of this questionnaire, participants underwent a funneled debriefing. The experimenter asked the participant a series of questions: if they had any questions about the study, if anything about the study had been suspicious, when might the experimenter have lied, and whether they'd thought the experimenter had lied about the beverage. Finally, the participant was fully debriefed, thanked, and dismissed.

## Results

Five participants from the placebo group were removed from analyses: three were not convinced by the placebo manipulation, indicating that they did not feel at all intoxicated during the scan session and that they had consumed no alcohol, and another one had exceptionally poor task performance (overall accuracy 50.1%). The fifth placebo participant fell asleep during two BOLD runs and is excluded from analysis. This left us with a final sample of 15 control participants and 14 placebo participants.

### Manipulation Check

To test the viability of the placebo manipulation, placebo participants' estimates of the number of standard drinks they consumed in the study were examined. On average, placebo participants estimated that they had consumed 3.3 standard drinks ($SD$ = 1.4), which differed significantly from zero, $t(13) = 11.5$, $p < .001$. This estimate is similar to those reported in previous studies in which this placebo method was used (e.g., Bartholow et al., 2003, 2006, 2012). Thus, the placebo manipulation appears to have been effective in creating the expectancy that a moderate amount of alcohol was consumed. [1]

[1] Oddly, 3 participants in the control condition reported believing that they had consumed alcohol. When asked about this response in debriefing, participants could not explain the reason for their responses. It is possible that the novel fMRI environment caused participants to feel strange, which some participants may have attributed to their drink. It is also possible that some control participants presumed themselves to have consumed alcohol, given that BrAC was periodically collected and hidden from them, same as the placebo group. We are reassured to know that both groups suspected the experimenters of deception to similar extents.

**Self-report measures**

Initial analyses examined whether the groups differed on mean levels of variables assessed via questionnaire. A 2 (Condition; placebo, control) x 2 (Scale; IMS, EMS) mixed ANOVA indicated no between-groups differences in motivation to respond without prejudice (*F*s < 1). Separate 2 (Condition) x 3 (Time, baseline, post-drink, post-WIT) mixed ANOVAs were conducted on the PA and NA subscales of the PANAS. The ANOVA on PA scores showed a main effect of Condition, $F(1, 76) = 7.52$, $p < 0.01$, indicating that placebo group participants had significantly higher levels of positive affect (M = 27.6) than control group participants (M = 26.7) across all three time points. There was also a marginally significant effect of time, such that levels of positive affect decreased over time, $F(1, 76) = 3.57$, $p < 0.06$. The ANOVA on NA scores showed no significant main effects or interactions (*F*s < 1), indicating no effects of beverage or time on negative affect.

**WIT Performance**

Prior to analysis, trials were sorted according to prime type and target type and according to correct versus incorrect responses, creating categories of Black-gun, Black-tool, White-gun, and White-tool trials, with correct and incorrect responses to each. Trials were additionally coded for whether the preceding trial had been accurate, whether the preceding trial had been too slow, and the preceding inter-trial interval. Accuracy and response time (for correct response trials) as a function of beverage condition and trial type are given in Table 1.

**Accuracy.** Target response accuracy was submitted to a 2 (Prime: Black, White) X 2 (Target: Gun, Tool) X 2 (Condition: Control, Placebo) mixed-model ANOVA. This analysis showed a significant Prime x Target interaction, $F(1, 27) = 30.587$, $p < 0.001$. Inspection of the means indicated that performance on Black-Gun (M = 0.84) and White-Tool (M = 0.83) trials was significantly more accurate than performance on Black-Tool (M = 0.73) and White-Gun trials (M = 0.73). A significant interaction of Cue X Probe X Beverage was also found, $F(1, 27) = 4.358$, $p < 0.05$, such that accuracy was less affected by the Cue X Probe interaction for participants in the placebo beverage group. As suggested by the pattern shown in Figure 1, the Prime x Target interaction was less pronounced among participants in the placebo group, $F(1, 13) = 8.88$, $p < 0.05$, compared to those in the control group, $F(1, 14) = 21.74$, $p < 0.001$.

**Reaction time (RT).** Reaction times on correct response trials were submitted to a 2 (Prime: Black, White) X 2 (Target: Gun, Tool) X 2 (Condition: Control, Placebo) mixed-model ANOVA. Reaction times were significantly predicted by a main effect of Probe, $F(1, 27) = 61.35$, $p < 0.001$, and an interaction of Cue x Probe, $F(1,27) = 31.8$, $p < .001$. These effects indicate that while responses to Tool trials are slower than (M = 425.8) responses to Gun trials (M = 410.4), this effect is exaggerated by Black cues. (Figure 2)

***Post-error adjustments.*** We wanted to investigate whether responses to current trials were affected by previous-trial accuracy, and whether the variable inter-trial interval moderated this effect. Unfortunately, an insufficient of participants made errors within each cell of the overall design (i.e., previous trial accuracy, current trial type, and

preceding inter-trial interval) to permit estimation of all of these combined effects, so we assessed these effects piecemeal in two separate analyses. Participants with fewer than 4 errors per cell were excluded from each post-error analysis. This removed one participant from each of these two analyses.

First, collapsing across the current trial-type, we computed RT and accuracy differences between trials following errors and trials following correct responses. These differences were then submitted to a 3 (Preceding inter-trial interval: 0ms, 2000ms, or 4000ms) X 2 (Beverage condition: Control, Placebo) mixed-model ANOVA. No significant effects were found for preceding ITI, beverage, or the ITI X Beverage interaction (all $F$s < 2.5).

Next, collapsing across previous ITI, we computed RT and accuracy differences within each cue-probe trial type, taking RT and accuracy for each trial type following an error and subtracting from it the RT and accuracy for each trial type following a correct response. These differences were similarly submitted to a 4 (Trial type: Black-Gun, Black-Tool, White-Gun, White-Tool) X 2 (Beverage condition) mixed-model ANOVA.

We calculated behavioral interference effects [RT on incongruent trials (e.g., Black-Tool) minus RT on congruent trials (e.g., Black-Gun)] and compared their magnitude on trials that followed correct responses to trials that followed errors using a 2 [Beverage] x 2 [Previous Accuracy] mixed-model ANOVA. We found a significant effects of Beverage, $F$ (1, 26) = 5.24, $p$ < 0.03, such that placebo participants exhibited lower levels of interference overall (M = 48.2 ms in the control group, M = 21.9 ms in the placebo group). We also found a Beverage X Previous Accuracy interaction, $F$ (1, 26) =

6.14, $p < .05$. This interaction was such that interference effects for control participants increased after errors (M = 38.4 ms post-correct, M=58.0 post-error, $q(2,26) = 3.59$, $p < 0.05$), while interference effects for placebo participants did not change (M = 26.1 ms post-correct, M = 17.7 ms post-error, $q(2,26) = 1.44$, $p > 0.05$). Placebo participants showed significantly lower interference effects than controls after error trials, $q$ (2,26) = 7.12, $p < .01$, while interference effects did not differ between controls and placebos following correct responses, $q$ (2, 26) = 2.17, $p > 0.10$. (*Figure 3)*

To make sure that these results were not due to a general shifting of responses towards "tool" in order to avoid making racially-biased errors on Black-Gun trials, we calculated an analogous interference effect for White-prime trials [RT on White-Gun minus RT on White-Tool trials]. Interference effects for correct responses to White-prime trials did not vary as a function of Beverage group or previous-trial accuracy, all *F*s < 1.

**Neural Response**

Psychophysiological data were analyzed using the Brainvoyager QX software. Each participant's BOLD data was aligned to a T2 structural image of that participant's brain and then mapped to Talairach space.

BOLD data for each run was preprocessed with slice scan time correction, a Trilinear/sinc interpolation 3D motion correction, and spatially smoothed with a 3D Gaussian filter with a full-width-half-maximum of 4mm.

Brain activity in response to stimuli was measured by a finite impulse response model (FIR). FIR models hemodynamic activity in response to a stimulus at 10 separate

timepoints, starting with the measurement during which the trial is presented and again for each measurement up to and including the 10th post-stimulus measurement. The model also included nuisance factors for head movement and linear and sinusoidal trends over time. Activity in response to stimuli was greatest at 4s-6s post-stimulus, consistent with the traditional hemodynamic response function, so we restricted our analyses to the 2nd and 3rd time points post-stimulus.

Activity within each BOLD run within each participant was entered into a multiple study, multiple participant random effects general linear model. Activation was measured in z-score units. Analysis was restricted to only voxels within the brain using a mask derived from a composite of all participants' Talairach-transformed structural scans. Activation associated with each trial type during the 2nd and 3rd post-stimulus time points was exported to per-subject beta maps. These beta maps were then submitted to ANCOVA random effects analysis. Appropriate contrasts were then conducted and overlaid.

**Task performance**. First, we examined increases in brain activity associated with performing the task compared to baseline activity. We contrasted activation on all correct trials against activation during fixation. This analysis revealed strong, highly significant activations of the dorsal cingulate gyrus (0, 5, 42), bilateral superior frontal gyrus (+/-30, 46, 14), parietal lobule (36, -57, 45), and bilateral insula (+/-38, 14, 6). The task was also associated with significant decreases in activity throughout bilateral middle temporal gyrus (+/-43, -74, 26), posterior cingulate gyrus (-10, -58, 11), bilateral inferior frontal gyrus (+/-49, 27, 3), right post-central gyrus (19, -45, 58), and rostral anterior

cingulate gyrus (0, 45, 2). Deactivated regions are associated with the "default network" of the resting brain and suggest the task causes prominent recruitment of attention and effort. All clusters were significant at $t(112) \geq 2.16$, $p < 0.03$, q(FDR) < 0.05. *(Figure 4.)*

We contrasted activation in response to error trials against activation in response to correct trials. We found significant increases in activation on error trials compared to correct trials in the anterior cingulate cortex (0, 23, 27) and bilateral inferior frontal gyrus (+/- 43, 24, 1), as well as smaller clusters in left superior frontal gyrus (-21, 42, 28) and midbrain (-4, 20, -4). We also found a small cluster of decreased activity in the left postcentral gyrus (-21, -39, 65). All clusters were significant at $t(112) \geq 3.51$, $p < 0.001$, q(FDR) < 0.05. *(Figure 5.)*

We looked for a main effect of beverage, contrasting correct trials between the two groups. Compared to controls, participants in the placebo group demonstrated increased activation in a diffuse network of locations, including rostral cingulate cortex, posterior cingulate cortex, and superior frontal gyrus. Placebo participants also demonstrated reduced activity of bilateral precuneus (+/-28, -66, 34). All clusters were significant at $t(112) \geq 2.91$, $p < .005$, q(FDR) < 0.05. *(Figure 6.)*

No significant differences could be detected between placebo and control participants on error trials at the default significance level of $p < 0.000004$. By relaxing the significance threshold to $t(112) \geq 3.37$, $p < 0.001$, and only examining significant clusters of 4 contiguous voxels or more, we found that placebo participants had significantly lower activations of the bilateral middle frontal gyrus (+/- 38, 38, 25), bilateral precuneus (+/-29, -69, 31), and right supramarginal gyrus (37, -41, 31) in

response to error trials. Placebo participants also demonstrated greater activity of the left postcentral gyrus on error trials (-65, -5, 14). *(Figure 7.)*

To assess activation associated with high-conflict trials, we contrasted correct responses to incongruent trials (Black-Tool, White-Gun) with correct responses to congruent trials (White-Tool, Black-Gun). In this and in other, similar contrasts, we found no significant voxels, even when relaxing the threshold for significance dramatically to $p < 0.02$.

**Post-error adjustments.** To examine possible post-error changes in activity such as the preparation of proactive control for a future trial, we compared all trials with a preceding correct response with all trials with a preceding error response in a 2 [Previous Accuracy] x 2 [Beverage] ANOVA. We found that post-error trials caused significantly greater activations in a broad frontal-dorsal area encompassing the anterior cingulate cortex (0, 30, 27) and the medial superior frontal gyrus (0, 30, 49). Significantly increased clusters were also found in bilateral middle frontal gyrus (+/-45, 12, 33), bilateral superior frontal gyrus (+/-20, 43, 37), bilateral middle temporal gyrus (+/-45, 6, -27), and bilateral insula (+/-40, 17, 5). Activity of the bilateral putamen was significantly decreased (+/-22, 2, -4). All clusters were significant at $t(28) = 2.31$, $p < 0.05$, q(FDR) < 0.05. *(Figure 8.)*

We then inspected the interaction of Previous Accuracy and Beverage. When inspecting the interaction via t-test contrast, a single cluster in right middle temporal gyrus was significant at the default significance level of $t(28) \geq 2.36$, q(FDR)<0.05, $p < 0.03$. However, when inspecting this interaction via F-test, a number of clusters were

significantly affected by a Previous Accuracy X Beverage interaction at the default significance level of $F(1,27) \geq 4.88$, q(FDR)<0.05, $p < 0.01$. These clusters included right middle frontal gyrus (28, 36, -14), rostral anterior cingulate (0, 39, -5), bilateral middle temporal gyrus (+/-50, -12, -8), bilateral superior temporal gyrus (+/-62, -5, 9), and posterior cingulate cortex (0, -52, 19). These areas seem to be more active on trials following errors in the placebo group compared to the control group. *(Figure 9.)*

## Discussion

The primary purpose of this research was to understand the cognitive mechanisms underlying the activation and suppression of racial bias in the Weapons Identification Task. A number of previous studies have shown evidence for increased ACC activation during race-biased errors (i.e., black-tool errors) in the WIT (Amodio et al., 2004; Amodio, Kubota, Harmon-Jones, and Devine, 2006), and recent work suggested that this effect is larger after consumption of an alcohol placebo compared with a control beverage (Bartholow et al., 2012). However, no previous studies have localized this activity using fMRI. This study was designed to address these issues using functional brain imaging in order to specify not only the neural structures involved in the expression and control of race bias as measured by the WIT, but also to further understand alcohol placebo effects on cognitive control more generally.

We predicted that placebo participants would compensate for their supposed inebriation by enacting additional cognitive resources of control in an attempt to make more egalitarian responses. We expected to observe this effect as increased activity of the anterior cingulate cortex associated with detection of conflict and error commission, leading to later increases in activity in the dorsal and lateral prefrontal cortex.

We replicated the effect of alcohol expectancy, such that participants who had consumed placebo alcohol were less influenced by racial cues, presumably due to the compensatory recruitment of cognitive control. Moreover, these participants demonstrated reduced interference [Black-Tool RT minus Black-Gun RT] for post-error trials as compared to control participants. Curiously, the direction of post-error

adjustments in control participants was opposite the typical pattern: control participants demonstrated an increase, rather than a reduction, in interference effect.

This pattern of results is somewhat unusual. In most trial-to-trial analyses of post-error behavior, it has been observed that participants demonstrate reduced amounts of interference on trials following errors (e.g., Bartholow et al., 2012; Rabbitt, 1965; Ridderinkhof et al., 2002) or trials following incongruent trials (Kerns et al., 2004), theoretically because participants make acute recruitments of cognitive control in response to errors or conflict. However, in our data, control participants showed increased interference on trials following errors.

The reason for this atypical pattern of post-error adjustment is unclear. Our best guess is that we are seeing something of a ceiling effect. Participants are given a number of questionnaires about their racial attitudes at the beginning of the study, which many reported made them feel uncomfortable or guilty. Then, when they learn the task, participants are told that Black-Gun errors are indicative of racial bias. Finally, participants are reminded at the start of each BOLD scan not to make any racist mistakes. It is possible that control is chronically recruited throughout the entire session, and that errors are associated with lapses in attention or control due to fatigue rather than insufficient application of control.

It is surprising that brain activity during successful task performance did not seem to vary as a function of trial type. In particular, the anterior cingulate cortex is theorized to be sensitive to the experience of conflict on incongruent trials in tasks like the WIT. We had expected to find increases in anterior cingulate cortex activity associated with

incongruent, as opposed to congruent, trials. It is possible that the task moves too quickly to create a response which can be measured by fMRI – it is unusual to have an fMRI task with such a quick, dramatic RT threshold as ours. It is also possible that the difference in recruited cognitive and physiological resources due to congruency is very small compared to the profound activity associated with task performance.

We have demonstrated the anterior cingulate cortex's sensitivity to errors, and those errors seem to lead to increased activity of middle and superior frontal gyrus, as well as middle temporal gyrus, in a conceptual replication of Kerns 2004. It also seems that post-error adjustments in placebo participants are indeed larger, associated with reduced interference effects on post-error trials and greater activations of the superior frontal gyrus and right middle frontal gyrus, suggesting that these areas are involved in the increased application of control over activated racial stereotyping. Placebo participants also demonstrated greater activity of the superior temporal gyrus and posterior cingulate cortex, the significance of which is yet to be determined.

It is strange that we see greater activity of the anterior cingulate cortex on post-error trials, since we would typically expect reduced anterior cingulate activation. If control were being elicited by the previous trial's error and acutely prepared for the next trial, we should expect that following trial both to be more accurate and to cause less conflict-related activity in the ACC. Instead, we see increased activity of the anterior cingulate. It is possible that the preparation of control for a post-error trial should occur sometime before, rather than at the time of, the post-error trial. It is also possible that ACC activity in response to an error is lasting, rather than acute – however, ERP data

from previous studies of this paradigm would suggest otherwise. Finally, participants may become more sensitive to conflict after an error, demonstrating increased activation of the ACC.

It is unfortunate that we do not detect increases in activity for incongruent, as compared to congruent, trials. This raises a number of questions about the way in which conflict is detected and control activated in the context of the Weapons Identification Task, and whether we can expect those activations to be detected through the use of fMRI. It is possible that the rapid pace and strict reaction-time deadline of the WIT make it unsuitable for study with fMRI. Since we find no differences between trial types, we cannot determine whether the congruency of errors or the trial following an error is important. Behavioral analyses suggest that the congruency of error trial does not matter.

In addition to the canonical pattern of conflict detection and the exercise of control, we observed a number of novel activations which may be specific to the activation and control of racial bias. For example, placebo participants were less influenced by racial primes after an error, an effect associated with greater activation of the temporal gyrus as compared to control participants. Given the possible role of the temporal gyrus in social cognition (Bigler et al., 2007), it is possible that this activation is associated with attempts at more egalitarian responses.

This study is also notable for the remarkable deadline associated with this task. It is uncommon to see an fMRI task with a response deadline as rapid as 500ms. This is a possible reason for the apparent lack of differences between activation during correct responses to congruent and incongruent trials. However, we are still able to observe a

variety of meaningful activations associated with slower processes such as error detection and the engagement of control.

In the end, little of the questionnaire data was actually used. With the small sample size typical of psychophysiology, there are not many degrees of freedom to go around. As a result, few of the planned covariates were actually used in analysis. These are, however, nonetheless useful in checking that the two groups were not, by chance, significantly different on any potentially important variables.

**Future directions**

There are a number of things yet to be done with this study. First, we intend to look at continuous activation in regions of anterior cingulate cortex and middle frontal gyrus, as was performed in Kerns 2004. We expect to replicate the correlation by which activity of anterior cingulate cortex is associated with later activation of the dorsal lateral frontal cortex. It is also possible that there may be differences between the two groups in the strength of the association, which might tell us more about the means by which the placebo group attempts to recruit additional control over their responses.

In our analyses, we examined activity on trials following error trials, and found that placebo participants had increased activity relative to controls in a number of prefrontal cortical areas. Given that placebo participants also show reduced interference effects (RT difference between incongruent Black-Tool and congruent Black-Gun trials) relative to controls, these areas may be responsible for the execution of control in order to reduce interference effects. Future analyses will investigate the possible correlation between activity in these regions and interference effects.

In the future, we also expect to add an alcohol group. We would expect that task performance in the alcohol group would be more influenced by racial cues, an effect which should be related to reduced activity of the anterior cingulate cortex and dorsal lateral frontal cortex.

In the future, it may also be informative to parameterize responses to the task through a cognitive model such as the Quad Process Model (Conrey et al, 2005). Such a model could make it easier to interpret how participants are performing the task (i.e. are participants avoiding making mistakes on Black-Tool trials through the improved exercise of control, or are they just biasing their responses towards answering "Tool" on all trials?). It would also make it possible to observe which parameters are influenced by alcohol and alcohol expectancy. Previous analyses using the Quad Process Model have been able to observe relationships between parameters (i.e. the probability of detecting the appropriate response) and brain activity (Beer et al., 2008). Such an approach could further explicate whether regions of increased or decreased activity are related to the activation of stereotype or the control over those stereotypes once activated.

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| | Accuracy | | Response Time (ms) | |
|---|---|---|---|---|
| Trial Type | Control | Placebo | Control | Placebo |
| Black-Gun | 0.87 (0.09) | 0.80 (0.18) | 411.6 (71.1) | 423.0 (76.6) |
| Black-Tool | 0.72 (0.12) | 0.75 (0.12) | 454.2 (71.6) | 451.2 (91.2) |
| White-Gun | 0.73 (0.17) | 0.73 (0.16) | 432.5 (69.7) | 434.3 (81.4) |
| White-Tool | 0.85 (0.06) | 0.80 (0.16) | 424.9 (78.4) | 427.7 (82.5) |

*Table 1.* Mean accuracy and response times for each cue-probe pairing within each beverage group.

| Region of Activation | Left/Right Brodmann | *x* | *y* | *z* | *t*-score |
|---|---|---|---|---|---|
| *Error>Correct* | | | | | |
| Anterior Cingulate | L/R 24/32 | +/- 4 | 23 | 27 | 5.02 |
| Medial Frontal Gyrus | L/R 8/9 | +/- 3 | 25 | 47 | 4.90 |
| Insula | L (and R) | -35 | 18 | 2 | 5.15 |
| Superior Temporal Gyrus | L 38 | -34 | 18 | -28 | 4.26 |
| Midbrain / Thalamus | | -2 | -22 | -8 | 3.95 |
| *Placebo Correct > Control Correct* | | | | | |
| Anterior Cingulate | BA 24 | 3 | 38 | 0 | 5.30 |
| Middle Frontal Gyrus | R BA 11 | 27 | 38 | -17 | 4.02 |
| Inferior Frontal Gyrus | R BA 47 | 39 | 25 | -17 | 4.08 |
| *Placebo Error > Control Error* | | | | | |
| Precuneus | L and R | 29 | -67 | 35 | -5.2 |
| Middle Frontal Gyrus | R (and L) | 38 | 38 | 24 | -3.93 |
| Fusiform Gyrus | R BA 37 | 42 | -49 | -6 | -3.86 |

*Table 2.* Talairach coordinates for loci of brain activity.

| Region of Activation | Brodmann Area | *x* | *y* | *z* | *t*-score |
|---|---|---|---|---|---|
| *Previous Miss > Previous Hit* | | | | | |
| Anterior Cingulate | 24, 32 | 0 | 25 | 26 | 5.45 |
| Superior & Medial Frontal Gyrus | 6, 8 | 0 | 26 | 49 | 5.34 |
| Right Middle Frontal Gyrus | R 9 | 39 | 18 | 35 | 2.86 |
| Left Middle Frontal Gyrus | L 9 | -43 | 10 | 39 | 3.65 |
| Right Putamen | | 19 | 3 | 0 | -3.49 |
| Left Putamen | | -19 | 7 | 1 | -4.37 |
| Right Superior Temporal Gyrus | R 38 | 43 | 9 | - | 3.74 |
| Left Middle Temporal Gyrus | L 21 | -44 | 3 | - | 3.14 |
| Right Inferior Frontal Gyrus | 47 | 43 | 19 | -7 | 4.53 |
| Left Inferior Frontal Gyrus | 47 | -44 | 19 | -5 | 5.43 |
| Left Superior Frontal Gyrus | 9 | -23 | 45 | 28 | 5.32 |
| Right Superior Frontal Gyrus | 9 | 25 | 46 | 23 | 3.48 |
| *Placebo Previous Miss > Control Previous* | | | | | |
| Right Medial Frontal Gyrus | 10 | 15 | 57 | 4 | 2.37 |
| Left Superior Frontal Gyrus | 10 | -22 | 57 | 5 | 2.50 |
| Lingual Gyrus | 18 | -1 | -78 | 5 | 3.48 |
| Posterior Cingulate | 31 | -2 | -53 | 20 | 2.82 |
| Right Cuneus | 18 | 15 | -75 | 20 | 2.76 |
| Superior Temporal Gyrus | 22 | -53 | -48 | 13 | 4.08 |

*Table 3.* Talairach coordinates for loci of brain activity on trials following errors.

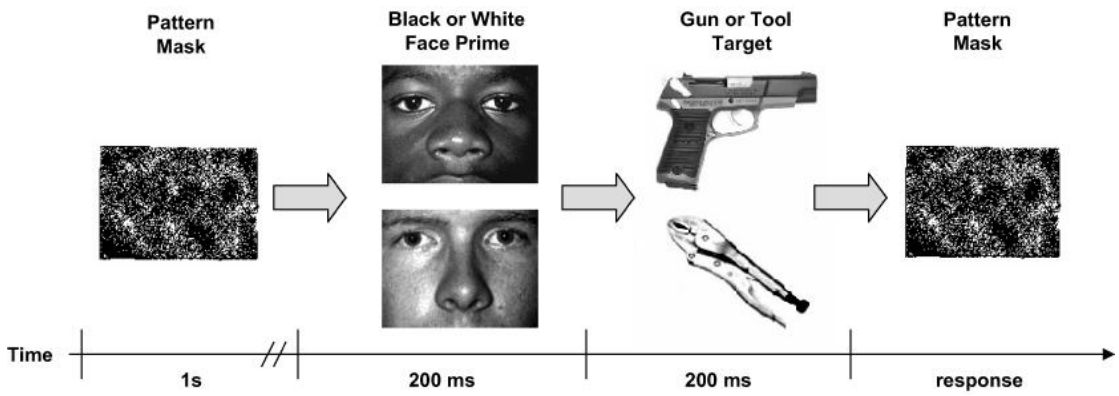


*Figure 1.* The Weapons Identification Task.

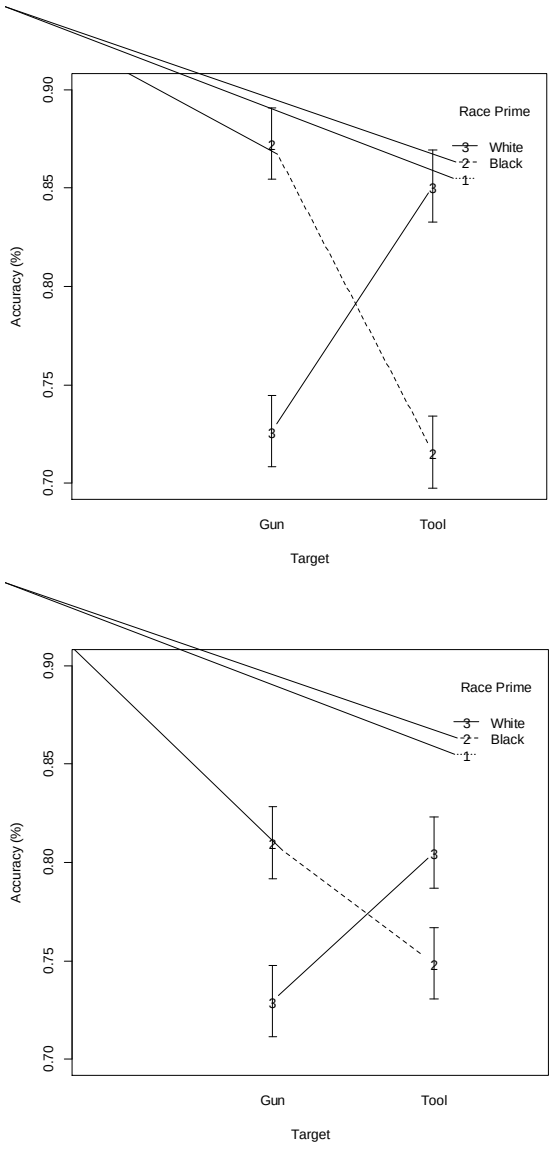


*Figure 2.* Participant accuracy was significantly affected by an interaction of Cue X Probe, $F(1, 27) = 30.587$, $p < 0.001$, and an interaction of Cue X Probe X Beverage, $F(1, 27) = 4.358$, $p < 0.05$.

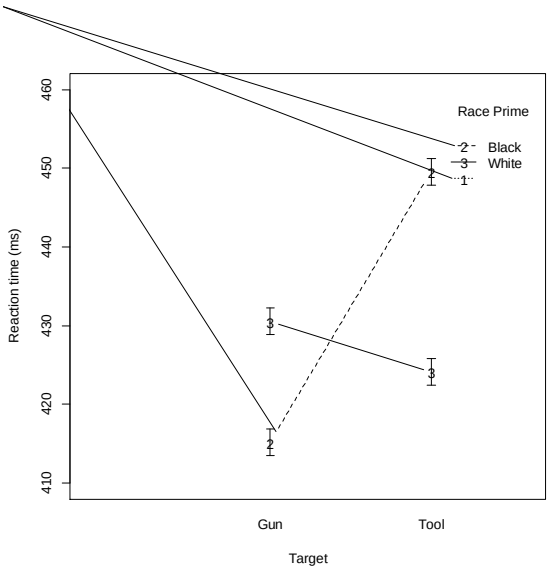


*Figure 3.* Participant reaction times were significantly affected by a main effect of Probe, $F$ (1, 27) = 61.35, $p < 0.001$, and a Cue X Probe interaction, $F$ (1,27) = 31.8, $p < .001$. Responses to Gun trials are faster overall, especially when primed with Black faces.

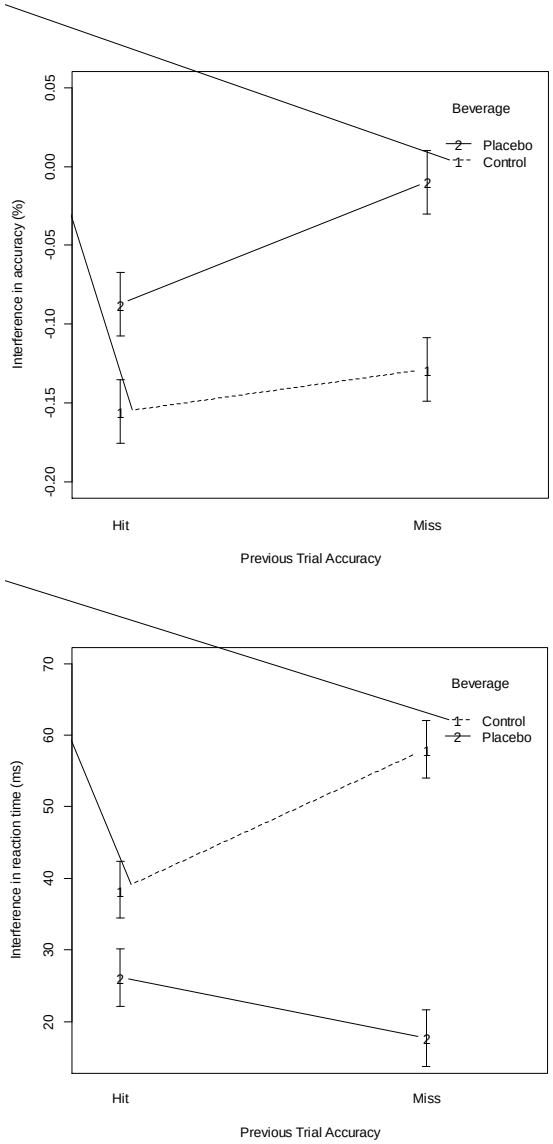


*Figure 4.* Interference effects as a function of previous-trial accuracy and beverage group. Participants in the control group demonstrated reaction times more influenced by interference effects after errors, an effect described by the significant Beverage X Previous Accuracy interaction, $F$ (1, 26) = 6.14, $p < .05$.

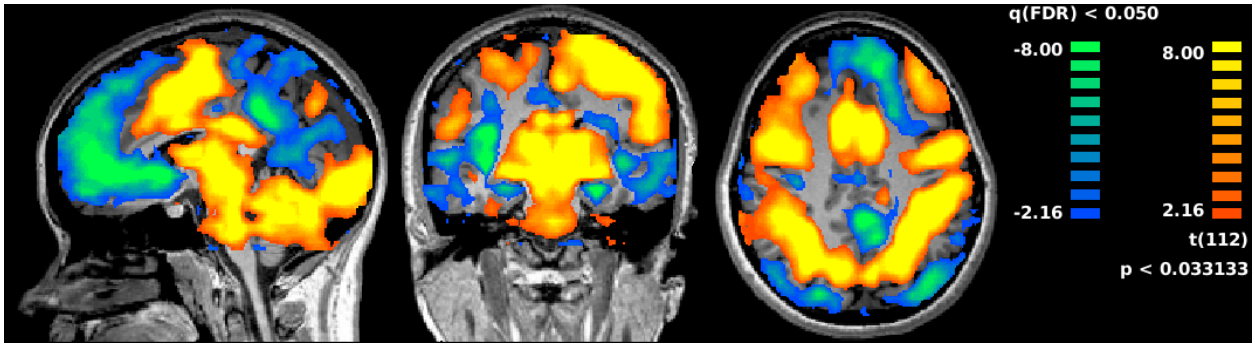


*Figure 5.* Brain activity associated with correct performance of the task. Broad activations include the dorsal cingulate gyrus, middle frontal gyrus, and parietal lobule, while broad deactivations include the medial and inferior frontal gyrus.

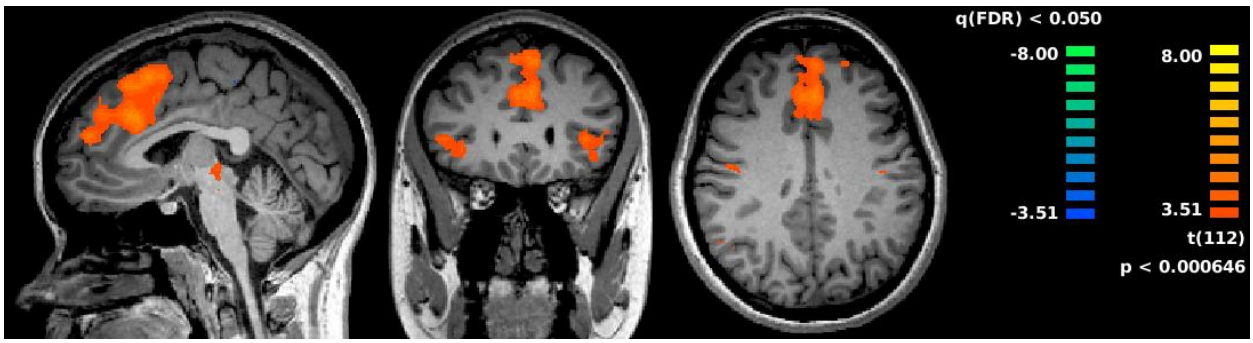


*Figure 6.* Brain activity associated with incorrect, as compared to correct, trials. Increased activity of the anterior cingulate cortex and bilateral insula are observed. Also present but not pictured is decreased activation of the bilateral putamen.

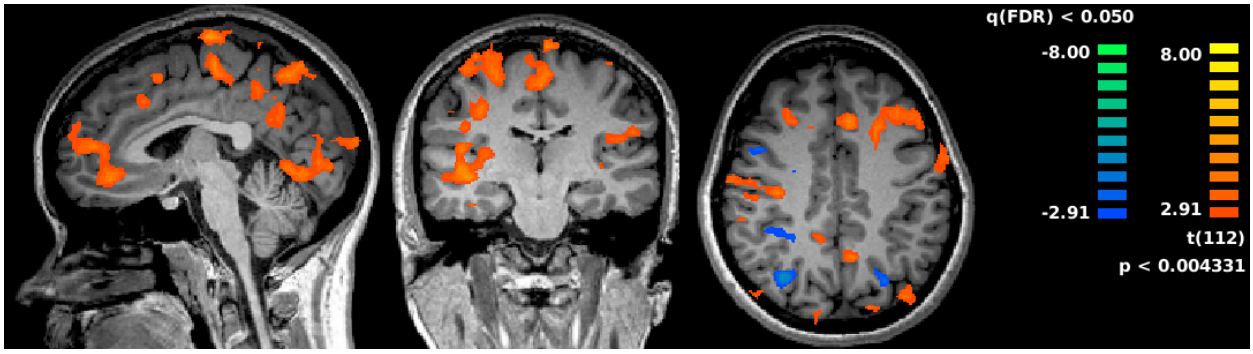


*Figure 7.* Areas with greater activation in placebo participants than controls during successful task performance. Significantly greater activations in left lateral frontal cortex suggest prolonged recruitment of regions responsible for cognitive control, while increased activity of the very anterior cingulate cortex may represent enhanced vigilance for conflict.

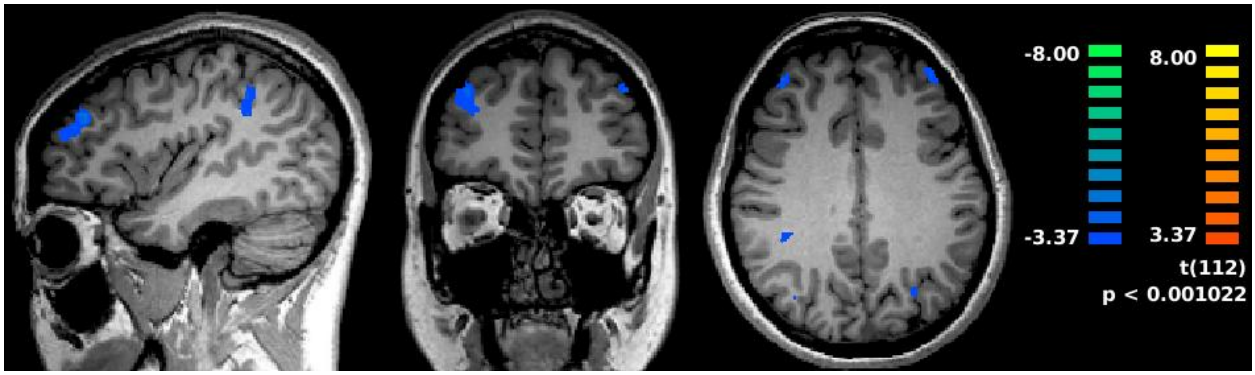


*Figure 8.* When making errors, participants in the placebo group demonstrate reduced activity of the bilateral middle frontal gyrus as compared to their control group counterparts (significance threshold relaxed to $p < 0.001$, minimum cluster of four voxels, no further multiple comparison correction).

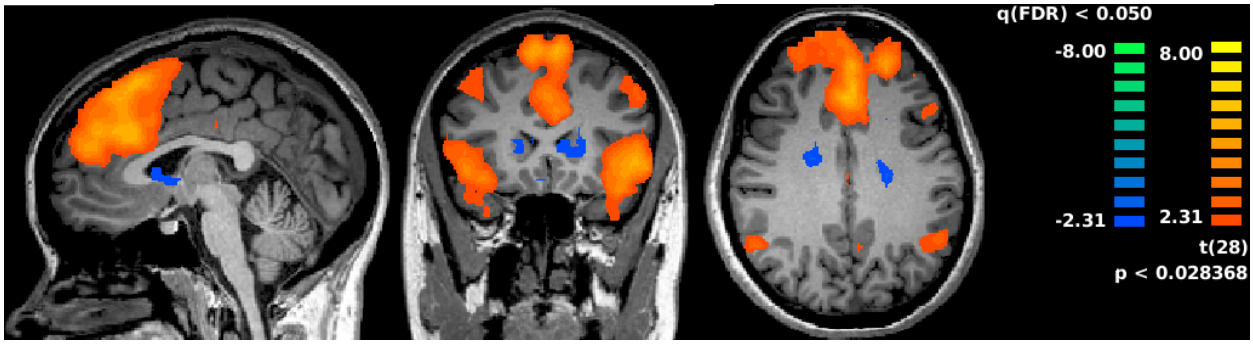


*Figure 9.* Trials after errors, as compared to trials after correct performance, are associated with increased activity of the anterior cingulate, medial frontal gyrus, middle frontal gyrus, insula, and middle temporal gyrus, as well as a decrease in activity of the putamen.

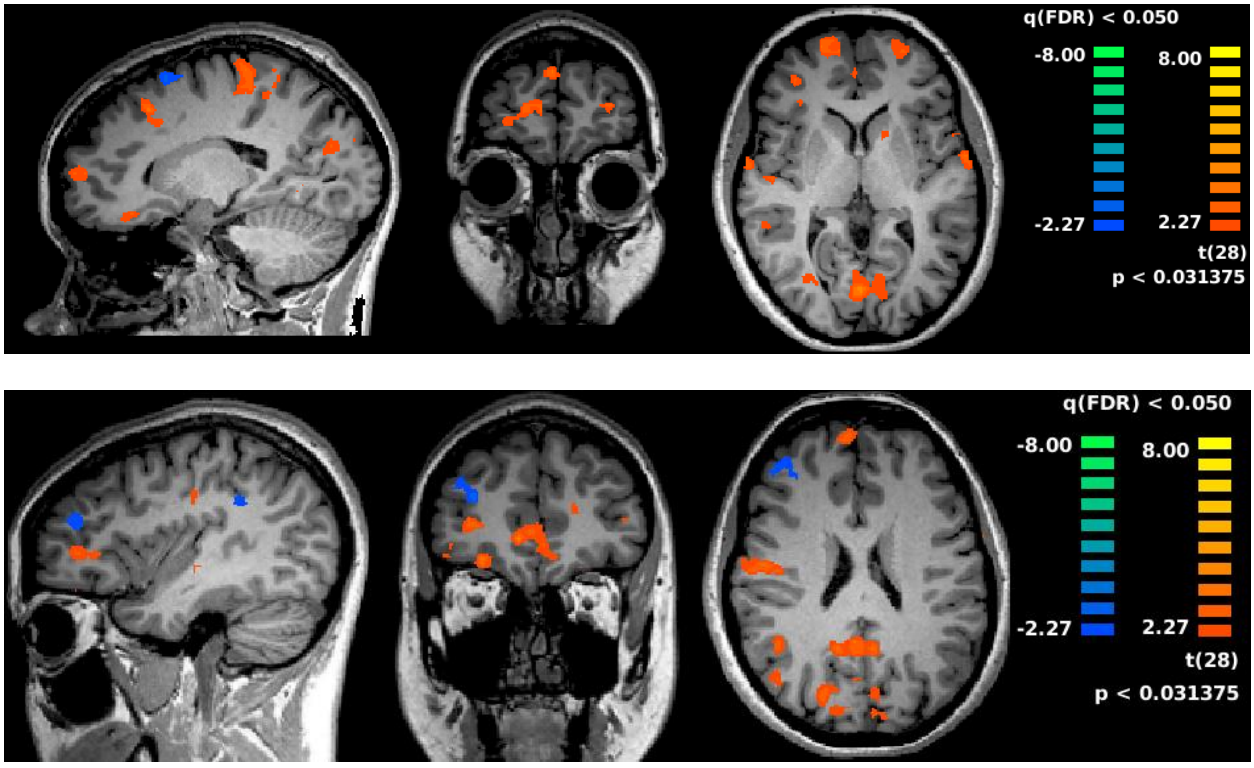


*Figure 10.* Areas of increased and decreased activation on trials following errors of placebo participants relative to controls. Placebo participants demonstrate increased activity of the bilateral superior frontal gyrus (+/- 21, 55, 5), right inferior frontal gyrus (41, 33, -3), and inferior right middle frontal gyrus (34, 38, 4). Curiously, these increases in frontal activation are accompanied by a decreased activation in the superior right middle frontal gyrus (34, 38, 17).