

**ALCOHOL CONSUMPTION, EXECUTIVE FUNCTION AND RISKY DECISION
MAKING**

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ALCOHOL CONSUMPTION, EXECUTIVE FUNCTION AND RISKY DECISION

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DEDICATION

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ABSTRACT

Previous research has shown that alcohol intoxication can adversely affect behavior by impairing higher cognitive function (e.g., Giancola, 2000) and can lead to increased risk-taking (Leigh, 1999) via impaired executive control. The purpose of this project was to assess the degree to which individual differences in interference control, including neural measures, are associated with self-reported risk-taking behaviors and whether these behaviors are moderated by alcohol intoxication. Participants were 96 male and female adults ages 21-35. Ps completed several self-report measures of risky behavior and executive function before being assigned to one of three beverage conditions: a no-alcohol control beverage, an active placebo beverage, or an alcohol beverage (1.0 g/kg ethanol). They then engaged in a laboratory cognitive control (flanker) task while their EEG (electroencephalogram) was recorded. This research suggests that effects of alcohol on the relationship between neural measures of cognitive control, task performance and self-reported real world risk behavior may be influenced more by alcohol use expectancy than by actual alcohol consumption.

CHAPTER 1

INTRODUCTION

Risk-taking behaviors, such as excessive drinking, engaging in physical fights or unprotected sex, result in considerable physical and mental health problems annually. Moreover, the propensity to engage in risk-taking behavior is an important component of many psychiatric conditions, including alcohol and drug abuse, antisociality, and other conditions that include a significant impulsivity or disinhibition component (see Sher et al., 1999). Thus, understanding factors that moderate the degree to which people are likely to engage in risk-taking behavior is an important public health concern.

People vary widely in their likelihood of engaging in risky behaviors (Sitkin & Weingart, 1995), a fact that has been linked, in part, to differences in so-called executive cognitive functioning (e.g. Giancola, 2000). Executive functions (EFs) include a set of higher-order cognitive abilities such as inhibition, attention control, and working memory updating (see Miyake et al., 2000), all of which are important for regulating behavior and guiding effective, goal-directed decision-making. Although EFs are highly heritable (Friedman & Miyake, 2008), their effectiveness in guiding behavior and decision-making also is known to be influenced by environmental factors, including drug and alcohol consumption (see Giancola, 2000).

The primary purpose of this project was to assess the degree to which individual differences in a specific EF component ability – interference control, or the ability to focus attention and resist the influence of distracting, peripheral information – assessed via a standard laboratory task, are associated with self-reported risk-taking behaviors. Also, given that alcohol is believed to significantly impair executive control (e.g.,

Giancola, 2000; Pedersen et al., 1997), and that alcohol intoxication often leads to increased risk-taking (Fromme et al., 1997; Leigh, 1999), another purpose of this work was to test the extent to which the link between interference control and self-reported risk-taking is moderated by alcohol intoxication.

Self-control, Cognitive Control, and Risky Decision-making

Theoretical models (e.g., Block & Block, 1980) suggest that self-regulation facilitates adaptive responses to life's challenges. One way self-regulation can be conceptualized is in terms of self-control (i.e. when a person attempts to change the way he or she would otherwise think, feel or behave; see Muraven & Baumeister, 2000). Risk-taking is a key component of behavioral self-control. Previous research has defined risk-taking as the voluntary participation in any behavior that carries some probability for negative consequences (Boyer, 2006). Self-control deficits are at the heart of many psychiatric disorders, such as those involving anti-social behavior, drug and alcohol abuse, and general risk-taking behavior (Magar, Phillips & Hosie, 2008). The current project aims to understand how risk-taking behaviors, particularly those associated with drug and alcohol use and sexual behavior, relate to performance on a laboratory measure of self-control.

People exert self-control when they follow rules rather than following their internal impulses, or inhibit immediate desires in order to delay gratification (Hayes, Mischel, Shoda & Rodriguez, 1989; Hayes, Gifford, & Ruckstuhl, 1996). Recent theories suggest that engagement in risky activities is importantly determined by individual differences in self-control abilities, with poor self-control competence increasing the likelihood of risk participation (Byrnes, 1998; Steinberg, 2004, 2005). Some research

suggests that poor cognitive control or executive function is linked to greater endorsement of risky activities (Byrnes, 2005), an over-emphasis of the benefits associated with risky activities, and a higher incidence of problems associated with excessive alcohol consumption (Magar et al., 2008). Much empirical work on self-control has focused on young children (Barkley, 1997; Baumeister, Leith, Muraven, & Bratslavsky, 1998), which provides little generalizability to other age groups. However, some research on adolescents suggests that substance use and peer pressure may be processes through which risk proneness and poor self-control lead to, for example, risky sexual behavior (Crockett, Raffaelli & Shen, 2006). Though there are likely multiple domains of risky behavior that are affected by self-regulatory processes in different ways, it is hypothesized that risky sexual behavior is especially relevant with regard to alcohol use because these behaviors often co-occur, and the consequences of poor sexual decisions (Sexually Transmitted Diseases -- STDs -- and unintended pregnancy) are very serious. Thus, the potential effects of alcohol on risky sexual decision processes are particularly important to understand.

Higher-order cognition, mediated by activity in areas of prefrontal cortex (Posner & Raichle, 1994), plays an important role in self-regulatory control. Mental (or cognitive) control is a function of human consciousness that arises from the ability to reflect on our own mental activities and influence their operation (see Wegner, 1994). Although cognitive control is a broad construct that can encompass a number of abilities from control of attention to inhibition of responses, the focus of the current work is on interference control, which is considered a facet of inhibitory control (e.g., Friedman & Miyake, 2004). In a general sense, interference control represents the extent to which an

individual can focus on a target stimulus amidst other, distracting stimuli that compete for attention (Wilson & Kipp, 1998; Dempster & Corkill, 1999; Nigg, 2000). A classic laboratory paradigm used to investigate interference control is the Eriksen flanker task (Eriksen & Eriksen, 1974). Some versions of this task require participants to respond to target letters embedded in a string of other letters that elicit either the same response as the target (i.e., compatible trials: HHHHH) or the opposite response (i.e., incompatible trials; SSHSS). Another common version of the flanker task requires participants to indicate the direction of a central arrow (left or right) shown amid other arrows. This version of the task has the advantage of mapping responses to stimuli that share the same orientation in physical space. Also, this version of the flanker task has been used in previous research on the effects of alcohol on self-control (Ridderinkhof et al., 2002).

Three behavioral indices of cognitive control can be derived from the flanker task data. First, the flanker interference effect (or compatibility effect) is calculated as the difference in either reaction times or accuracy between incompatible trials and compatible trials. Responses tend to be slower and less accurate on incompatible compared to compatible trials (e.g., Bartholow et al., 2005; Coles et al., 1985; Gratton et al., 1992), which reflects the behavioral consequences of response conflict elicited by the interfering flanker stimuli. Previous work suggests that, at least under some conditions alcohol can exacerbate the flanker interference effect (Bartholow et al., 2003), suggesting that alcohol impairs interference control processes. Second, the flanker interference effect typically is modulated by trial sequence, such that the effect is smaller on trials that follow an incompatible trial compared to trials that follow a compatible trial (see Gratton et al., 1992). This effect has been interpreted as evidence that participants use cognitive

control to adjust their behavior following the experience of conflict (see Kerns et al., 2004). To the extent that alcohol impairs the use of cognitive control (e.g., Fillmore & Vogel-Sprott, 1999, 2000), this sequential adjustment effect should be smaller following alcohol consumption relative to placebo or control beverage consumption.

Finally, researchers have long known that responses on trials following an incorrect response (i.e., post-error trials) tend to be slower and more accurate than responses on trials that follow a correct response (i.e., post-correct trials) (see Rabbit, 1966). This so-called post-error slowing (or post-error adjustment) generally is thought to reflect the influence of cognitive control being increased following an error (i.e., a control failure). Previous research (e.g., Ridderinkhof et al., 2002) has shown that post-error adjustment is impaired following alcohol relative to placebo consumption, suggesting that alcohol disrupts typical implementation of increased cognitive control.

Research examining the cognitive factors associated with self-control has the potential to illuminate how multiple factors contribute to risk-taking behavior. For example, cognitive tasks can be structured in such a way as to allow for separate estimates of the contributions of unconscious or automatic processes and controlled processes to behavior and decision-making (Bargh & Chartrand, 1999). The interaction between controlled and automatic processes may be particularly important to understand in relation to risky behaviors because risky decisions are likely to be made quickly and impulsively (i.e., via automatic processing) (Palfai, 2004). By understanding the relationship between an individual's behavioral performance on a laboratory measure of self-control that reflects both automatic and controlled processing and self-reports of

risky behavior, we may be able to understand how the ability to focus attention in the face of distraction contributes to risky behavior.

Alcohol and Self-control

There has been a great deal of research testing the effects of alcohol on self-regulatory control (e.g., Bartholow et al., 2003a, 2003b; 2006; Bartholow, Henry, Lust, & Saults, 2009; Casbon et al., 2003; Curtin & Fairchild, 2003; Fillmore & Vogel-Sprott, 1999, 2000). According to some models, alcohol intoxication is thought to adversely affect behavior by impairing a host of higher cognitive functions (e.g., Giancola, 2000; Steele & Josephs, 1990). Moreover, some models suggest that alcoholism can develop in part from alcohol impairing cognitive processing resources when they are needed most, for example, when one is trying to resist a well-learned behavior sequence that has been activated in memory, such as drinking to excess (Tiffany, 1990; also see Fillmore & Vogel-Sprott, 2006). One example of this process would be a situation in which an individual experiences cognitive impairment from drinking alcohol, which leads them to a downward spiral of decisions that involve more drinking and further cognitive impairment (Weafer & Fillmore, 2008). Other research specifically focused on implementation of self-control has shown that people who must exert self-control prior to consuming alcohol may actually drink more when given the opportunity, even in situations that demand restraint (such as being aware that one must drive a vehicle in the near future) (Muraven, Collins & Nienhaus, 2002). It is suggested that this happens because exertion of control at one point in time depletes resources to exercise control at later points in time. This process may reflect a risk for falling into a pattern of further drinking and self-control failures.

These well-documented effects of alcohol on cognition and self-control can have important implications for understanding intoxicated risk-taking. Though several domains of risky behavior have been studied with regard to the influence of self-control, including truancy, property violation, gambling, physical risk taking and the negative consequences of heavy alcohol use itself (Fromme & Corbin, 2004), sexual risk-taking while intoxicated is especially relevant (George & Stoner, 2000; Weinhardt & Carey, 2000), not only because the negative outcomes of poor sexual decision making (STDs, unintended pregnancy etc.) can be so dire, but also because sexual behavior is inherently arousing and can be associated with impulsive decisions (Ariely & Loewenstein, 2006). Research has shown that alcohol use is related to the decision to have sex and to indiscriminate forms of risky sex, such as having multiple or casual sex partners, but is inconsistently related to protective behaviors such as condom use (Cooper, 2002). Correlational findings suggest that adolescents who use alcohol are more likely to be sexually active than abstainers. In addition, heavy drinkers are more likely than moderate drinkers to have unprotected sexual intercourse. Since alcohol is known to impair decision making it may exacerbate choices made on impulse (Donohew, Zimmerman, Cupp, Novak, Colon & Abell, 2000). For example, when the benefits of more hedonic physical pleasure seem to outweigh the more long-term health risks of STDs, individuals may decide to forego condom use. However, this topic has yet to be studied with a laboratory measure of self-control.

The majority of models linking intoxication to disruptions in social behavior focus on impaired inhibitory control as the primary cognitive mechanism (e.g., Eadson & Vogel-Sprott, 2000; Curtin & Fairchild, 2003). According to such models, increased risk-

taking under intoxication results largely from the inability to inhibit responses that one otherwise would control. Other models, though, point to the possibility that interference control abilities might importantly determine alcohol-related risk-taking behavior. For example, a central feature of the alcohol myopia model (e.g., Steele & Josephs, 1990) is that intoxication impairs the control of attention. However, the alcohol myopia model relates to cognitions and a person's attention in their environment more broadly than other tests of interference control typically used in laboratory tasks (e.g. Botvinick, 2001; Lustig, May & Hasher, 2001; Ridderinkhof 2004).

Though interference control is implicated in the alcohol myopia model, very little research using controlled laboratory tasks has tested the effects of alcohol on this specific cognitive process (Ridderinkhof, 2002). There is also no previous research on how alcohol moderates interference control in relation to real-world self-reports of risk behavior. Past research has shown that prepotent response inhibition and the ability to resist distractor interference are closely related, yet theoretically distinct aspects of cognitive control (Friedman & Miyake, 2004). Specifically, Wilson & Kipp (1998) suggest that inhibition is an active suppression process that operates on the contents of working-memory, whereas resistance to interference is a gating mechanism that prevents irrelevant information or distracting stimulation from entering working-memory, though the authors also acknowledge that these types of cognitive control are likely controlled by similar neurological substrates. Friedman & Miyake also suggest both types of tasks share the requirement of actively maintaining task goals. Although inhibitory control is undoubtedly impaired by alcohol intoxication (e.g., Fillmore & Vogel-Sprott, 1999, 2000) and clearly is important for understanding risky decision-making, it could be that

individual differences in interference control, and how such differences interact with effects of alcohol, account for important variability in alcohol-related risk-taking behaviors. Previous research suggests that indeed, drinking alcohol leads to decreases in sustained attention (e.g. Sher, Bartholow, Peuser, Erickson & Wood, 2007; Dougherty, Marsh, Moeller, Chokshi & Rosen). In turn, inability to maintain attention (measured here with the flanker task) can lead to problematic behavior, deficits in self-control and is related to psychopathology (Barkley, 1997; Nigg, 2000). Understanding these individual differences in interference control and how such differences interact with the effects of alcohol is the primary focus of this project.

Electrophysiological Correlates of Cognitive Control

Traditionally, cognitive control has been assessed in the laboratory using some combination of behavioral (e.g., Stroop task; flanker task) and neuropsychological (e.g., Wisconsin Card Sort; Self-ordered Pointing task) measures. Recent advancements in cognitive neuroscience theories of cognitive control have underscored the need to augment such traditional measures with other measures that more directly tap neurocognitive activity online, as participants attempt to engage in cognitive control. Moreover, behavioral responses represent the cumulative output of multiple stages of processing, making inferences derived from behavioral measures unclear as to underlying mechanisms. Event-related brain potentials (ERPs) are very useful in this context, providing millisecond-level temporal resolution of brain activity associated with relevant psychological and behavioral processes (see Fabiani, Gratton, & Federmeier, 2007).

A recent neurocognitive model of cognitive control provides a useful framework for focusing the current research on processes that could play an important role in the effects

of alcohol on risk-taking. Specifically, the neurocognitive model of control proposed by Botvinick et al. (2001) separates control into two complimentary processes believed to be mediated by distinct neural structures. The first is the *evaluative* component, which monitors ongoing behavior for instances of conflict or potential conflict. This component is hypothesized to be associated with activity in the anterior cingulate cortex (ACC), and in particular the dorsal aspect of ACC (dACC). Laboratory tasks involving conflict between competing response tendencies (e.g., flanker, Stroop) have been shown to increase activity in the dACC. A particular component of the ERP, the error-related negativity (ERN), has been linked to this evaluative component of control (see Yeung, Botvinick & Cohen, 2004; Amodio et al., 2004). This link has been suggested because the neural source of the ERN appears to be the dACC, and because the ERN is larger on high-conflict compared to low-conflict trials (see Bartholow et al., 2005), and much larger on error trials than on correct trials. Such findings have led to the hypothesis that dACC activity increases when attempts at control fail (i.e., when self-regulation fails or is inadequate for task performance). One published study to date has linked alcohol-related impairment in cognitive control and behavioral adjustment to impaired error processing, reflected in reduced amplitude of the ERN (Ridderinkhof et al., 2002).

When the evaluative component signals that conflict is high (and, thus, that more control is needed), a second, *regulative* component activates and implements control resources to help ensure adequate performance. This regulative component is thought to be mediated by activity in areas of prefrontal cortex, particularly the dorsolateral prefrontal cortex (DLPFC), which becomes more active following high-conflict trials (see Kerns et al., 2004). Activity of DLPFC associated with the implementation of

control resources has been associated with the amplitude of a negative slow wave (NSW) component of the ERP. For example, West and Alain (1999, 2000) have shown that the NSW is larger on incongruent color-word trials of the Stroop task in which response conflict is successfully resolved. Other research has shown that alcohol-related impairment of cognitive control is reflected in reduced NSW amplitude (see Bartholow, Dickter & Sestir, 2006; Curtin & Fairchild, 2003).

In this study, including measures of the error-related negativity (ERN) and negative slow wave (NSW) will make it possible to determine how neural measures hypothesized to be associated with distinct aspects of cognitive control predict decision making on a trial by trial basis. In turn, these ERPs may predict self-reported real world risky behavior separately from behavioral measures, bolstering the strength and clarity of models of executive function and risky behavior. Indeed, some previous research suggests that ERPs can be more sensitive to the impairing effects of alcohol on cognition than are behavioral measures (e.g., Martin & Siddle, 2003).

To sum up, this research focuses on the interference control component of self-regulation (Miyake et al., 2000) and how individual differences in this cognitive control ability relate to self-reported risk-taking and disinhibited behaviors in a number of domains. In addition, this research investigates how acute alcohol intoxication affects this relationship by testing whether the degree of alcohol-induced cognitive control impairments assessed in the lab is significantly associated with particular domains of risk-taking behavior outside of the lab.

Hypotheses

The objective of this experiment is to assess the degree to which individual differences in interference control are associated with self-reported risk-taking behaviors and to understand how consumption of alcohol moderates this effect.

On the basis of the literature review and on previous research from our lab, a number of hypotheses were formulated. First, it was predicted that alcohol would influence behavioral performance in the flanker task on some but not all indices of performance. Alcohol was expected to slow reaction times overall. However, owing primarily to the fact that participants in all groups underwent an extensive practice period as a way to ensure roughly equivalent error rates across beverage groups, it was anticipated that alcohol would not affect overall accuracy or the size of the compatibility effect in RT. More importantly for the current research, it was predicted that alcohol would reduce post-error performance adjustment (see Ridderinkhof et al., 2002). It was expected that post error slowing and/or post-conflict adjustment would predict risky behavior because these measures represent implementation of my operational definition of self-regulation.

Second, it was predicted that the number of risk-taking behaviors that participants reported were associated with their flanker task behavioral performance. Moreover, it was predicted that beverage contents and flanker task performance interacted to predict risk-taking behavior. That is, the extent to which participants' performance on the flanker task (as a laboratory analog of self-control) is affected by the beverage they consume should predict their reports of risk-taking behaviors outside the lab. Although extensive previous research has shown that self-reported alcohol use is associated with risk-taking

behavior in a number of domains (Derman & Cooper, 2000; Cooper, 2002; Cherpitel, 1993), no previous research has directly tested effects of an acute alcohol challenge on self-reported risk-taking. One experimental study (Fromme, Katz & D'Amico, 1997) has shown that alcohol affects expectations about future negative consequences; however this research did not test behavioral reactions in the lab or self-reports of actual risk taking behavior.

Third, it was predicted that the amplitude of the ERN on error trials would be reduced by alcohol compared to the placebo group (see Bartholow et al., 2008; Ridderinkhof et al., 2002). Whether the ERN also will be reduced in the control group relative to the placebo group is unclear, but has been observed in one previous experiment (Bartholow et al., 2008). Moreover, it was predicted that alcohol would interfere with the typical association between the amplitude of the ERN and behavioral indices of self-regulation, specifically, post-error behavioral adjustment. Whereas larger ERN typically is associated with better self-regulation (i.e., smaller post-error adjustment), alcohol appears to disrupt this association (Bartholow et al., 2008). I also expected that the NSW will be smaller in the alcohol condition, suggesting less successful resolution of response conflict on incongruent trials. There may be a greater ERN and NSW for placebo participants because the expectancy that alcohol will interfere with cognition and motor skills will lead to greater motivation to perform well (Vogel-Sprott, 1992; Marczinski & Fillmore, 2005; Testa et al., 2006.)

Fourth, and most importantly, it was predicted that the strength of the association between ERN amplitude and post-error adjustment would be associated with risk score. This prediction relates most strongly to the notion that the effectiveness of the self-

control system as measured in the lab would be associated with a lack of self-control (i.e., greater risk-taking) outside the lab.

CHAPTER 2

METHOD

Participant recruitment

Participants were 96 male and female adults, ages 21-35, recruited using advertisements in local publications and mass email (MU Info) announcing research on the effects of alcohol. Participants were screened via a structured telephone interview to determine their eligibility for the project. Individuals who indicated any condition that would contraindicate participation in an alcohol challenge (e.g., abstention; history of alcohol or other drug use disorders or other serious mental or physical illness; prescription medication other than oral contraception; pregnancy) were excluded from the sample. Alcohol and drug dependence were assessed using items similar to those developed by Kahler et al. (2005) for comprehensive measurement of alcohol problems in young adults (see Sher, Bartholow, & Wood, 2000). Also, individuals who reported drinking less than an average of 2 or more than an average of 25 standard drinks per week were excluded so that the dose potentially received in the lab would be within participants' normal range of experience. Participants all reported having experienced a binge drinking episode (5 drinks at one occasion for men; 4 for women) at least once within the past year. Individuals younger than 21 cannot legally consume alcohol in Missouri and were excluded. Individuals older than 35 also were excluded to avoid potential confounds between effects of aging and effects of alcohol on neurocognitive function. Finally, the Fagerström Test for Nicotine Dependence (Heatherton, Kozlowski, Frecker, & Fagerström, 1991) was used to test for nicotine dependence. Individuals scoring 3 or above (moderate or more severe dependence) were excluded in order to

avoid the potential for acute nicotine withdrawal during the rather lengthy laboratory session.

Laboratory screening

Upon arrival at the lab appointment, participants were asked to produce 2 forms of identification to verify age eligibility. Their medical and psychiatric history were briefly reassessed using self-report measures based on the interview items used in the initial phone screening and they were asked to sign an affidavit attesting to their adherence to pre-experimental protocols concerning diet (fasting for 5 hours prior to the appointment) and abstention from drugs and alcohol for the previous 24 hours. Female participants were required to self-administer a urine pregnancy test in a private restroom. A female staff member verified test results. A baseline breath alcohol concentration level (BrAC) was measured to ensure participants' initial sobriety. Participants who failed to maintain their eligibility were dismissed at this time.

Self-report Measures

Risk-taking. The propensity to engage in risk-taking behaviors was assessed using 30 items based on a measure developed by Parson, Siegel and Cousins (1997) to assess perceived benefits and risks of a number of risk-related behaviors (see Appendix A). The first part of the questionnaire contained 31 items while the other three sections each contained 29 items. Respondents are asked to indicate how frequently in the past year and past 3 months they have engaged in behaviors in a number of risk-taking domains (i.e. HAVEYOUSCORE; $\alpha = .68$). The domains included problems with alcohol use, drug abuse, sex with unknown partners and using unsafe practices, and participation in sensation-seeking activities such as drag racing. Responses were scored such that “never”

was 0, “yes but not in the past year” was .3, “yes, in the past year but not in the past three months” was .5, “yes in the past three months...” once was 1, twice was 2, three times was 3 and 4+ times was 5. The responses on all items except “have you...drank alcohol, gotten drunk, had sex with a condom, not used a seatbelt and smoked cigarettes” for the past-behavior question were summed to create an overall “risk score”; $\alpha = .67$. This system of scoring was chosen to create a continuous measure for each item in which increasing reported frequency of a behavior would result in a higher score for each item, later making it possible to take an average overall score for all past risk taking behavior. Though this method allowed flexibility with certain analyses, it may not have been ideal for this particular type of data. Another potential option would have been categorical analyses based on whether or not an individual engaged in a particular behavior in their lifetime or in the past three months.

Although my primary interest was in the overall risk-taking variables noted above (HAVEYOUSCORE and risk score), it also would be possible to parse items on the risk-taking measure into domain-specific subscales. However, some of these subscales produced very poor reliability estimates. For example, 6 items (e.g., “have you taken speed”) comprised the drug-related subscale. However, no participants endorsed the item, “have you used meth,” whereas most participants endorsed “have you used marijuana,” resulting in a very poor reliability coefficient ($\alpha = .14$). Also, 3 items that were expected to form a “truancy” subscale (“Have you missed work or missed a class;” “Have you gotten into trouble at work or school;” and “Have you been fired from a job, or suspended or expelled from school”) did not correlate with one another ($r_s < .09$), and thus could not be combined ($\alpha = .004$)

Respondents also were asked how often they thought they would engage in these activities in the next 3 months. Future predictions of participation in these behaviors were assessed with a 9-point scale ranging from never to weekly (i.e., WILLYOU; $\alpha = .71$). Participants were also asked how risky or dangerous they thought those activities were on 9-point scales ranging from not at all risky to very risky (i.e., DANGER; $\alpha = .88$). In addition respondents were asked how fun they thought participating in those activities would be in general on 9-point scales ranging from not at all fun to very fun (i.e., FUN; $\alpha = .89$). Again, individual items on each scale were summed to create an overall score for the “risky” and “fun” scales respectively. It should also be noted that “riskscore” is technically a subscale of “HAVEYOUSCORE” because it includes all items except those related to alcohol use or the “alc_probs” subscale.

The variables, “sexual, drugs, truancy, sensationseeking and alc_probs” are subscales of the “HAVEYOUSCORE” overall composite variable. The variable “probscore” is an entirely different scale composite based on responses to the problems with alcohol and health behavior questionnaire.

Impulsivity. Participants responded to the Barratt Impulsiveness Scale (Barratt, 1959), which measures motor, cognitive and non-planning traits related to impulsivity. The scale includes 34 items, $\alpha = .83$ (Patton, Stanford & Barratt, 1995). The measure asks participants to respond to statements on a 1 (rarely/never) to 4 (almost always/always) scale, for example, “I plan tasks carefully” or “I act on the spur of the moment.” Several items were reverse coded after which items were summed to create an overall impulsivity score.

Executive functioning. Participants also responded to the Behavior Rating Inventory of Executive Functioning –Adult Version (BRIEF-A) (Roth, Isquith, & Gioia, 2005), which asks respondents to indicate whether they sometimes, never or often do things like talk at the wrong time or forget instructions easily. The measure is a 75-item scale that includes 9 subscales referred to as inhibition, shifting, emotional control, self-monitoring, initiation, working memory, planning and organization, task monitoring and organization of materials. For this study’s analyses I used the General Executive Control (GEC) scale which is a composite sum of all the aforementioned subscales, ($\alpha = .95$).

Other measures of executive function were the Cognitive Failures Questionnaire (CFQ) (Broadbent, Cooper, Fitzgerald, & Parkes, 1982), which includes items such as, “Do you read something and find you haven't been thinking about it and must read it again?” Response options range from 0 to 4, ($\alpha = .915$). Finally, participants completed the Self-Control Scale (SCS) (Grasmick, Tittle, Bursik & Arnekelev, 1993), which asks participants to rate on a 1 (not at all like me) to 5 (very much like me) scale statements such as “I am lazy” or “I have trouble saying no”, ($\alpha = .89$). Composite scores for both the CFQ and SCS were sums of responses to items on each scale.

The SCS and CFQ scales were significantly correlated, $r = .401$, $p < .01$. The CFQ and GEC were scales also significantly correlated, $r = .473$, $p < .01$. The SCS and GEC scales were also significantly correlated, $r = .717$, $p < .01$. Considering all measures were significantly correlated a composite for over all executive functioning (EF) was created which was the average of the composite scores from each of the three scales (SCS, CFQ and GEC). This variable was centered before further analyses were conducted.

Mood. Subjective mood states were assessed throughout the experiment with the Biphasic Alcohol Effects Scale (BAES) (Martin, Earleywine, Musty, Perrine, & Swift, 1993). The BAES contains 7 items designed to tap subjective experiences of stimulation (e.g., elated, energized, excited, stimulated, talkative, up and vigorous) and 7 items related to sedation (e.g., sluggish, slow thoughts, heavy head, down, sleepy, inactive and sedated). In theory, these items are associated with the ascending and descending limbs of the blood alcohol concentration curve, respectively. Participants respond to each item using a scale anchored at 1 (*not at all*) and 10 (*extremely*). This measure was administered 6 times over the course of the experiment, beginning with the first baseline BrAC test. The measure was then administered again after beverage consumption, multiple times during the task, just after completing the task and one final time along with other post-experimental questions.

Post-experimental questions. Once participants completed the experimental task they were asked questions regarding their mood and perceptions of the study. The first set of four questions asked how intoxicated participants felt “right now”, while drinking, just after drinking and during the task. Responses on the 5 point scale ranged from “not at all” to “a lot”. The next set of questions asked participants whether they tried to perform their best on the computer task, how frustrated they were by the task, how much they were upset when they made errors on the task and how much harder they tried on future trials to improve their performance after making an error. Responses on this 5 point scale ranged from “not at all” to “very much”. The third set of questions asked participants how much they thought the drink they had affected their performance, effort and concentration during the task. Once again response options ranged from “not at all” to

“very much” on a 5 point scale. Participants were then asked to circle the number of standard drinks they thought would be equivalent to what they drank in the study that day. Finally participants were asked several open ended questions about their overall impression of the study, whether they thought there was anything problematic with the study, whether they felt any portion of the study was deceptive, what their expectations were concerning the study and whether they had any further comments or questions.

Beverage Conditions and Administration

There were 96 (48 male) participants in this experiment. Participants were assigned to each of three beverage conditions: a no-alcohol control beverage ($n = 30$), an active placebo beverage (0.04 g/kg ethanol) ($n = 33$), or an alcohol beverage (1.0 g/kg ethanol) ($n = 33$). Participants in the control beverage condition were informed that their beverage contained no alcohol, whereas those in the other two conditions were told that their beverage contained alcohol. This combination of conditions permitted examination of true alcohol effects by comparing the alcohol and placebo conditions, and examination of expectancy effects by comparing the control and placebo conditions. Beverage administration procedures closely mimicked those used in previous work in this lab (see Bartholow et al., 2003a, 2003b, 2008). In the alcohol and placebo conditions, an experimenter ostensibly mixed a beverage containing a moderate dose of alcohol mixed in a 5:1, tonic to vodka ratio. The placebo dose was achieved by using diluted vodka (9 parts flattened tonic to 1 part 100 proof vodka mixed in a vodka bottle). Thus, placebo and alcohol group participants all saw the same volume of “alcohol” being mixed into their drinks. Control condition participants consumed a tonic beverage. Total beverage was isovolemic across conditions. Beverage volume and alcohol dose for each participant

was calculated using a computer program based on total body water volume (estimated using age, gender, height, and weight information) and the duration of the drinking period. For the alcohol and placebo conditions, collars were used to indicate the actual contents of the vodka bottles (e.g., “Regular vodka” and “Diluted vodka”), and the lead experimenter removed these collars before bringing the bottles to the second experimenter. Thus, the (second) experimenter who mixed and served the beverage remained unaware of the actual contents of the beverage bottles. The beverage was divided into three equal-size drinks given to the participant one at a time. Participants were allowed 5 minutes to consume each of the three drinks. Following completion of the third and final drink, participants sat idle for 5 min; in the alcohol condition, this allowed alcohol to begin to be absorbed into the blood prior to starting the task.

Laboratory Task

The procedure and stimulus materials in this experiment closely mimicked those used by Ridderinkhof et al. (2002). Participants completed a flanker task in which arrays of right- and left-facing arrows served as stimuli presented inside a horizontal rectangle. Compatible trials were those in which flanker (peripheral) arrows faced in the same direction as the (central) target (i.e., $\rightarrow\rightarrow\rightarrow\rightarrow\rightarrow$ or $\leftarrow\leftarrow\leftarrow\leftarrow\leftarrow$), whereas incompatible trials were those in which the flanker arrows were opposing the direction of the target (i.e., $\rightarrow\rightarrow\leftarrow\rightarrow\rightarrow$ or $\leftarrow\leftarrow\rightarrow\leftarrow\leftarrow$). Compatible and incompatible arrays were presented pseudorandomly, with the constraints that they occur with equal probability and call for left- and right-hand responses equally often. Participants were instructed to respond to left-facing targets by pressing a button with their left index finger and to right-facing targets by pressing a button with their right index finger. The horizontal rectangle

remained on the screen throughout the task, and was shown in black against a light gray (10% black) background. The targets were presented in dark gray (80% black). To strengthen flanker interference, the immediately surrounding arrows were slightly darker (90% black) and 10% larger than the targets, and the outermost flankers were still darker (100% black) and larger (20%) than the targets. Arrow arrays were presented for 100 ms. Participants completed 7 practice blocks of 28 trials each, followed by 10 blocks of experimental trials with 80 trials per block. During the practice blocks, performance was monitored by the experimenter to ensure that each participant attains a speed/accuracy balance that produces approximately 15% errors. Given that the ERN decreases along with decreased response accuracy (see Gehring et al., 1993), this comparability in performance is important in order to ensure that any decrease in ERN amplitude observed in the alcohol group was not due simply to a larger number of errors in that group relative to the other groups.

In addition, and similar to previous studies in which response confidence judgments have been recorded (Bartholow et al., 2008; Scheffers & Coles, 2000; Payne et al., 2005), following the response on each trial a 3-point scale appeared at the bottom of the screen, with anchor points labeled “Sure Correct,” “Don’t Know,” and “Sure Incorrect.” Participants were instructed to indicate their confidence in the correctness of the response they just made by pressing one of three buttons on their button box (the 2 buttons used to make target responses, plus a third button located between them). Participants were told to indicate a confidence rating within 3 seconds. After participants recorded their confidence response on each trial, the next trial began following an inter-trial interval that varied randomly between 1100 and 1500ms.

Electrophysiological Recording

The electroencephalogram (EEG) was recorded continuously throughout the experimental task from 32 electrodes fixed in a stretch-lycra cap (ElectroCap, Eaton, OH) placed on the scalp in standard locations (American Encephalographic Society, 1994) and referenced to the right mastoid; an average mastoid reference was derived offline. This electrode density permitted tests of additional hypotheses concerning the source of scalp-recorded potentials and effects of alcohol on additional components that are not of central interest in this proposal. Epochs were derived offline to permit examination of stimulus- and response-locked ERP components of interest. EEG and EMG signals were amplified with Synamps2 amplifiers (Neuroscan Labs, El Paso, TX) and sampled at 1000 Hz. EEG was filtered on-line at 0.05 to 30 Hz. Electrode locations were cleaned until the measured impedance of the skin was below 5 k Ω . Ocular artifacts (blinks) were removed from the EEG signal off-line using a regression-based procedure (Semlitsch, Anderer, Schuster, & Presslich, 1986). After artifact removal and rejection, EEG data were averaged off-line according to participant, electrode, and stimulus conditions.

ERP components were defined according to published conventions (Picton et al., 2000). A baseline, computed as the average signal activity across the 200 ms prior to an event (stimulus or response) was subtracted from all single trials. The amplitude of the response-ERN was defined as the peak negativity at electrode FCz in a window 0-150ms following an incorrect response, relative to pre-response baseline. All component windows and primary electrode sites were verified by visual inspection of the data and adjusted if necessary.

Procedure

When participants arrived at the laboratory they were asked to read and sign a consent form describing all the study procedures in addition to two affidavits related to their eligibility for the study and what they had to eat and drink in the past 24 hours. They were also asked to provide two forms of identification. After completing initial questionnaires, participants were asked to use the restroom and females were specifically asked to take a hormonal pregnancy test to ensure their continued eligibility for the experiment. They were then weighed and their height was measured in order to calculate the amount of beverage they would consume. Participants then were informed of the beverage condition to which they had been assigned. Specifically, placebo and alcohol participants were told that they would consume a beverage containing a moderate amount of alcohol; control participants were informed that their beverage would not contain any alcohol.

After participants received their condition assignment, they were fitted with an electrode cap and then received instructions for the laboratory task. Once the task was explained, participants engaged in 5 blocks of practice trials. After each block of practice trials participants were given explicit feedback to either “go faster” or “slow down and be more careful” depending on their performance accuracy during the block. The goal of this feedback was to ensure that participants in all conditions (alcohol, control and placebo) achieved a similar level of accuracy -- roughly 10-15% errors -- so that conclusions about between-group differences in ERP measures would not be confounded with differences in performance. This feedback/training was important for hypotheses related

to the larger study of which this project was a part, but may have been disadvantageous with regard to testing some of the hypotheses in this project, as noted in the Results.

After completing the first 5 practice blocks participants rested while an experimenter prepared their beverage. After beverage consumption and the 5-min absorption period, BrAC level and BAES responses were measured for the second time (BrAC2 & BAES2). Participants then completed 2 more blocks of practice trials, again with performance feedback provided by an experimenter. Next, participants completed 3 blocks of experimental trials, after which the third BrAC measure and BAES responses were taken (BrAC3 & BAES3). Participants then continued with 4 more blocks of experimental trials followed by BrAC4 and BAES4, after which they completed the last 3 blocks of experimental trials followed by BrAC5 and BAES5.

At the conclusion of the laboratory task, participants were shown to a private restroom where they could clean electrode gel from their face and hair. Afterwards, participants were escorted to a “sober-up” lounge containing comfortable chairs, books and magazines, a television and DVD player (and library of DVD movies). Participants then completed the post-experimental questionnaire measures, after which they were debriefed about the nature of the experiment. Participants also were given snacks and soft drinks to consume. For those in the alcohol group, BrAC was monitored every 30 min by project staff. Once their BrAC was $\leq .02\%$, participants were allowed to leave, either in a taxi paid for by the lab or with a friend or relative who agreed to drive them directly home.

Analytic Approach

The basic design of this experiment was a 3 (Beverage: control, placebo, alcohol) x 2 (Flanker Compatibility; compatible arrays, incompatible arrays) x 2 (Response type; correct, incorrect) mixed factorial with repeated measures on the last two factors. Primary analyses were carried out using mixed general linear models (i.e., ANCOVAs and regressions) on each main dependent variable (ERN amplitude; RT and accuracy; and post-error adjustment scores). Multiple regression analyses with categorical variables were used to assess whether performance on the flanker task predicts self-reported risk-taking behavior, and whether this association differs depending on beverage condition. Risk-taking was examined by using overall scores on the risk-taking measure as an index of general risk-taking (i.e., risky score), scored according to the procedure described previously.

CHAPTER 3

RESULTS

Missing or Problematic Data

Several participants had to be dropped from certain analyses due to missing data. Thirty participants' data could not be used for certain ERN analyses because they did not make enough errors (i.e., at least 5; see Olvett & Hajcak, in press) to create a stable average waveform in one or more conditions. This issue was particularly problematic on compatible trials, on which participants generally were very accurate. In order to retain enough data for reliable statistical analyses, ERN amplitudes were imputed for a number of participants using the mean of other participants of their same sex and in the same beverage condition. One participant's data could not be used for any risky behavior analyses due to technical problems with data logging. Another ten participants' data was not properly logged for the executive function measures (CFQ, SCS and GEC).

Some issues also arose with certain behavioral analyses. For example, two participants completed only 5 blocks of practice trials rather than 7 blocks. Finally, it was obvious that for 3 participants the reaction times for compatible trials in 1 block were outliers due to inattention or misunderstanding procedures. Those particular RTs were imputed at the mean for that particular block of trials (i.e., the mean of all other right arrow compatible trials for that individual).

Analytic Strategy

The primary purpose of this study was to test hypothesized relations between alcohol consumption, flanker task performance (including behavioral measures and associated neural correlates) and self-reported risk-taking behavior that occurs outside the

lab. Thus, primary analyses were focused on comparing flanker performance (RT and accuracy) and ERN amplitude across beverage conditions (alcohol, placebo, and control) and relating these data to self-reported risk-taking. As noted previously, however, for reasons related to the larger project of which this study was a part, participants in all conditions were trained to achieve a similar level of behavioral performance, which necessarily limited the variance in between-groups differences that would provide a basis for some of the hypotheses to be tested here.

Initial analyses focused on variables that served primarily as manipulation checks for the beverage manipulation. Primary analyses related to study hypotheses proceeded in steps, as outlined below, to test: (1) effects of beverage group (alcohol, placebo, control) on flanker task behavioral performance (reaction times, error rates, and post-error performance adjustment); (2) the relationship between flanker task behavioral performance and number of self-reported risk-taking behaviors (i.e., risk score); (3) potential interactive effects of beverage group and flanker performance on risk score; (4) effects of beverage group on the amplitude of the ERN on error trials; (5) the extent to which the strength of the association between ERN amplitude and post-error adjustment is associated with risk score. In all main analyses, individual differences in baseline executive function (EF) abilities and typical alcohol consumption (quantity/frequency) were included as covariates to control for effects of these variables on outcomes of interest.

Manipulation Checks

BAES scores. Differences in mood between groups and across trials were analyzed with 3 (Beverage group; alcohol, placebo, control) x 2 (BAES subscale;

stimulation, sedation) x 6 (Assessment time) mixed factorial Analysis of Variance (ANOVA) with repeated measures on the second and third factors. The analysis revealed a trend such that the alcohol group reported higher scores overall (of both sedating and stimulating effects) than participants in the other beverage groups, $F(2, 93) = 2.99, p = .055$. The analysis also showed a significant overall limb effect, such that higher scores (more endorsement) were reported for stimulating than sedating effects items, $F(1, 93) = 12.45, p < .01$. This main effect was qualified by a significant Subscale by Time interaction, $F(5, 465) = 14.69, p < .01$. Inspection of the means showed that stimulating effects decreased over the course of the experimental trials, bottoming out near the end of the trials and then increasing again after the task was completed. In contrast, sedating effects appeared to increase over the course of the experimental task, dropping off again once the task was completed. Finally, all of these effects were qualified by a higher-order Beverage group x Subscale x Time interaction, $F(10, 465) = 3.81, p < .01$. As illustrated in Figure 1, for the alcohol group sedating effects rose over the course of the experiment, only decreasing after the task was over whereas exactly the opposite happened with stimulating effects in the alcohol group such that stimulating effects decreased over the course of the experiment only increasing upon completion of the task and other post-experimental procedures. In addition the alcohol group appeared to report higher scores for stimulating effects than the other groups overall. The control group appeared to have a different experience in which stimulating effects appeared to be “U” shaped such that they decreased initially but then increased again just after completing the task, whereas reported sedating effects also decreased initially, peaked near the end of the task and then decreased once again after completing the task. The placebo group seemed to experience

a steeper drop in stimulating effects over the course of the experiment but a larger jump in sedating effects followed by another steep drop in sedating effects after the task was complete.

BrAC and subjective intoxication. BrAC rose during most of the task for alcohol participants, $F(3, 93) = 2.90, p = .03$ (see Figure 2). Alcohol participants reported greater subjective intoxication throughout the experiment ($M = 1.98$) than placebo participants ($M = 0.71$), $F(1, 63) = 63.22, p < .001$. However, the pattern of effects across time did not differ between the groups, $F(4, 252) = 0.44, p = .78$. Also, alcohol participants estimated having consumed more standard drink equivalents ($M = 4.18$) than placebo participants ($M = 2.06$). However, the fact that placebo group participants estimated consuming, on average, more than 2 drinks indicates that our placebo manipulation was effective.

Correlations

Correlations among study variables are presented in Table 1. Several variables were correlated with self-reported quantity and frequency of alcohol use, including problems with alcohol-related behavior. However, the correlation between quantity/frequency of alcohol use and risk score was not significant without controlling for other factors in the model (see below). Also of interest (though not very surprising), risky behavior scores were strongly associated with sensation seeking and alcohol problem measures. However, surprisingly impulsivity did not correlate with any of the other variables in the study. The compatibility effect in RT (CE_RT) did not correlate with other variables. Only a handful of subscales on the Risky Behavior measure were correlated with each other. The risky sexual behavior scale was significantly correlated with sensation seeking.

Effects of Beverage on Flanker Performance

Effects of alcohol on the compatibility effect in RT, error rates, and post-error adjustment (i.e., post-error slowing) were tested using separate 3 (Beverage group; alcohol, placebo, control) x 2 (Sex) x 2 (Compatibility) x 2 (Arrow direction) general linear models with EF and alcohol use as covariates. It was predicted that the size of the compatibility effect in RT and errors would not differ across beverage groups (Ridderinkhof et al., 2002), but that post-error adjustment would be reduced by alcohol.

Reaction time. The ANOVA on the RT data showed the predicted Compatibility effect, $F(1, 90) = 38.05, p < .0001$, indicating that participants were faster to correctly categorize target arrows on compatible trials ($M = 392.8$ ms) than incompatible trials ($M = 448.4$ ms). As expected due to the training period, (i.e., feedback during practice trials) this effect did not differ across beverage conditions, $F(2, 90) = 0.05, p = .95$. Unexpectedly, there was a significant main effect of Arrow direction, $F(1, 90) = 7.50, p < .01$, indicating that participants were quicker to respond when the target arrows faced left ($M = 408.8$ ms) than when they faced right ($M = 432.5$ ms). This effect was qualified by a complicated (and unpredicted) Beverage x Arrow direction x Sex interaction, $F(2, 90) = 5.29, p < .01$. Visual inspection of the means suggested that, in the alcohol condition, women were slower to respond to left-facing arrows than were men, but that in the control condition men were slower to respond to right-facing arrows than women. No other effects were significant.

Error rates. Participants in all beverage conditions made very few errors on compatible trials ($M = .03$), which limited variability (i.e., a floor effect). Thus, analysis of error rates was based on the arcsine of the square root of errors, which produces a

distribution better suited for analysis of variance. The transformed error rate data were analyzed with a 2 (Compatibility) x 2 (Arrow direction) x 2 (Sex) x 3 (Beverage group) mixed factorial ANOVA with repeated measures on all but the last 2 factors. Results showed a significant Compatibility effect, $F(1, 90) = 408.98, p < .01$, with error rates being higher on incompatible trials ($M = 0.30$) than compatible trials ($M = 0.12$). This effect was qualified by a Compatibility by Beverage group interaction, $F(2, 90) = 3.96, p < .05$. Means associated with this interaction are shown in Figure 3. Follow-up contrast analyses showed significant linear [$F(1, 90) = 5.56, p < .05$] and quadratic trends [$F(1, 90) = 4.36, p < .05$] across beverage groups for compatible trials. The quadratic trend indicates that placebo group participants made significantly fewer errors ($M = .09$) on compatible trials compared to participants in the alcohol ($M = .14$) and the control groups ($M = .13$), which did not differ from each other. In contrast, follow-up analyses of errors on incompatible trials showed no significant linear or quadratic effects ($F_s < 3.05, p_s > .07$), indicating that error rates on incompatible trials did not differ reliably across beverage groups. No other effects were significant in this analysis.

Sequential effects. To test whether participants' use of cognitive control to adjust their behavior following the experience of conflict (e.g., Kerns et al., 2004) is affected by alcohol, we computed separate RT averages per condition as a function of whether the previous trial was compatible or incompatible. These averages were subjected to a 3 (Beverage) x 2 (Current trial; compatible, incompatible) x 2 (Previous trial; compatible, incompatible) mixed factorial ANOVA with repeated measures on the latter 2 factors.

Results showed a significant main effect of Current trial, such that participants were faster to categorize current compatible trials than current incompatible trials overall,

$F(1, 93) = 935.2, p < .01$. However, this effect was qualified by a Current trial by Previous trial interaction, $F(1, 93) = 33.9, p < .01$. Planned comparisons indicated that participants were faster to categorize compatible trials when they followed compatible trials ($M = 369.77\text{ms}$) than incompatible trials ($M = 377.73\text{ ms}$), $F(1, 93) = 74.46, p < .01$, but there were no differences in RT for incompatible trials regardless of previous trial type ($M_s = 431.2$ and 432.6 ms for previous incompatible and previous compatible, respectively), $F(1, 93) = 1.61, p = .21$. This interaction was not further qualified by Beverage group (3-way interaction $F < 1$). No other effects were significant.

Post-error adjustment. Given the paucity of errors on compatible trials, this analysis was restricted to only incompatible previous trials. This analysis was also limited to participants who made at least 4 correct responses on both compatible and incompatible post-error trials ($n = 88$). As in previous research (Ridderinkhof et al., 2002), performance adjustment as a function of the accuracy of previous trials was conceptualized in terms of the size of the compatibility effect (i.e., incompatible RT – compatible RT) for correct trials that followed correct trials versus those that followed errors. Effects of beverage group on performance adjustment in the compatibility effect were tested using a 3(Beverage) x 2 (Previous trial; error, correct) mixed factorial ANOVA. The main effects of both Beverage condition and Previous trial type were nonsignificant ($F_s < 1$). However, the analysis showed a significant Beverage group x Previous trial interaction, $F(2, 85) = 3.62, p < .05$ (see Figure 4). Follow-up simple effect tests of previous trial type for each beverage group showed no significant differences in the post-error versus post-correct compatibility effect for either the alcohol or control group, ($p_s = .12$ and $.85$ respectively). However, participants in the placebo group

showed a smaller compatibility effect on post-error trials ($M = 41$ ms) than on post-correct trials ($M = 57.7$ ms), $F(1, 85) = 4.87, p < .05$, indicating significant post-error performance adjustment.. Also, a linear contrast analysis showed that the compatibility effect for post-error trials decreased linearly across groups from Alcohol ($M = 63.28$ ms) to Control ($M = 51.75$ ms) to Placebo ($M = 41$ ms), $F(1, 85) = 4.3, p < .05$. There was no such linear trend for post-correct trials $F(1, 85) = 1.68, p = .19$.

Effects of Beverage on ERN Amplitude

Given that very few errors were made on compatible trials ($M = 0.12$), for several participants an average could not be made for the ERN component in conditions where no incorrect responses were made. As such, analyses were restricted to incompatible trials. Initial analyses of the response-locked data showed that, as is typical, ERN amplitude on incorrect trials ($M = -6.21\mu\text{V}$) was considerably larger than on correct trials ($M = -0.92\mu\text{V}$), $F(1, 87) = 167.03, p < .0001$. Thus, remaining analyses focused only on incorrect trial responses.

The neural source of the ERN has been identified as the anterior cingulate cortex (Miltner et al., 2003), which is located along the medial surface of the frontal lobes. Thus, the ERN typically is largest at midline fronto-central electrode locations (Hermann et al., 2004). I verified the scalp topography of the ERN in these data with an initial 3 (Lateral locations; left, midline, right) x 5 (Coronal locations; frontal, fronto-central, central, centro-parietal, parietal) repeated measures ANOVA. This analysis showed a significant Lateral x Coronal interaction, $F(4, 348) = 62.4, p < .0001$. Inspection of the means indicated that the ERN was largest overall at midline locations, but especially at the

midline central electrode Cz. Thus, further analyses were restricted to data recorded from Cz.

It was predicted that ERN amplitude would be least negative for the alcohol group compared to placebo and control groups. Results confirmed this significant beverage group difference in ERN amplitude $F(2, 82) = 4.12, p < .05$ (see figure 6). The ERN was smallest (least negative) in the Alcohol group ($M = -5.88 \mu\text{V}$) compared to the Control group ($M = -7.71 \mu\text{V}$) and Placebo group ($M = -8.91 \mu\text{V}$).

Effects of Beverage on NSW Amplitude

This analysis focused on the NSW mean amplitude between 775ms and 1100ms after target onset. The NSW is also typically is largest at midline fronto-central electrode locations (West & Alain, 2000). Scalp topography of the NSW was verified with an initial 3 (Lateral locations; left, midline, right) x 5 (Coronal locations; frontal, fronto-central, central, centro-parietal, parietal) x 2 (Compatibility; compatible trials, incompatible trials) repeated measures ANOVA. This analysis showed a significant Coronal x Lateral interaction, $F(8, 720) = 19.04, p < .0001$. Inspection of the means indicated that the NSW was largest overall at midline locations, but especially at the midline frontal electrode Fz. Thus, further analyses were restricted to data recorded from Fz.

Effects of beverage on NSW amplitude were tested using a 3 (Beverage) x (Sex) x 2 (Compatibility; compatible, incompatible) mixed factorial ANOVA with repeated measures on the last factor. Analyses were restricted to correct trials. Results showed a significant main effect of Compatibility $F(1, 90) = 10.34, p < .01$ such that NSW

amplitudes were greater (more negative) for incompatible ($M = -2.6\mu\text{V}$) than compatible ($M = -1.8\mu\text{V}$) trials (see figure 7). No other effects were significant.

Relation between Flanker Performance and Risk-taking

The hypothesized relationship between flanker task behavioral performance (compatibility effects in RT and accuracy) and risk score, including potential interactions with beverage group, was tested using separate general linear models including Beverage group and flanker performance variables as factors and EF and quantity/frequency of alcohol use as covariates. The analysis using the compatibility effect in RT (CE_RT) showed only a significant main effect of quantity/frequency of alcohol use, $F(1, 78) = 4.56, p = .036$, indicating that, when controlling for the other terms in the model, participants who tended to drink more also had higher risk score values.¹ Contrary to predictions, CE_RT and risk score were not significantly related, $F(1, 78) = .40, p = .53$. No other effects were significant in this analysis.

The analysis using the compatibility effect in accuracy (CE_acc) showed a significant main effect of Beverage group, $F(2, 78) = 3.92, p < .05$, which was qualified by a significant Beverage group x CE_acc interaction, $F(2, 78) = 5.54, p < .01$. This interaction was probed by computing partial correlations (controlling for covariates) between CE_acc and risk score separately for each beverage group (see figure 5). These analyses revealed that the relationship between CE_acc and risk score was not significant for the control ($r = .26, p = .23$) or alcohol groups ($r = .14, p = .46$), but was significant in the placebo condition, $r = -.48, p = .01$). These patterns of relations suggest that, for participants in the placebo group, an increasing number of risk-taking behaviors was

associated with a decreasing compatibility effect (in accuracy) during the flanker task. However, this relationship was not apparent in the other beverage groups.

Contrary to predictions, the analysis using Post-Error Adjustment (calculated by subtracting the compatibility effect post-correct trials from the compatibility effect post-error trials) in RT (CE_PE) showed no significant relationship between CE_PE and risk score, $F(3, 94) = .47$ $p = .70$. No other effects were significant in this analysis.

Effectiveness of Self-regulation and Risk-taking

To test the extent to which neural indices of self-regulatory control and self-reports of risk-taking are associated, and whether any relationship between those variables would be moderated by alcohol, a set of regression equations were computed. The amplitude of the ERN on incompatible trials serves as an index of evaluative control in this paradigm. Thus, the first model included main effects for ERN amplitude (incompatible trials only) and Beverage group in step 1, followed by their interaction in step 2, predicting risk scores. In models such as this, the interaction between the predictor variables is directly proportional to their correlation (D. Steinley, personal communication, November 2008). Thus, these models allow us to estimate the extent to which the correlation between the predictor variables is associated with the criterion variable.

The NSW serves as an index of regulative control. Since it is hypothesized that more control is needed on incompatible compared to compatible trials a difference score was computed by subtracting the NSW amplitude on compatible trials from the NSW amplitude on incompatible trials. As with the ERN models, the first model included main

effects for the NSW difference score and Beverage group in step 1, followed by their interaction in step 2, predicting risk scores.

Unfortunately, in each model tested none of the main effects or cross-product terms were significant predictors of risk scores (all β s $< .13$, ps $> .23$). Thus, these models are not described in any detail here.

CHAPTER 4

DISCUSSION

This project was intended to assess the degree to which individual differences in interference control, assessed via the flanker task, are associated with self-reported risk-taking behaviors. In addition this work sought to test the extent to which the link between interference control and self-reported risk-taking is moderated by alcohol intoxication.

Quantity and frequency of alcohol use were related to several variables in the study, including self-reported measures of executive function and generally served as a significant covariate. Not surprisingly quantity/frequency of alcohol use was significantly positively correlated with problems with alcohol use (e.g. problems with injury, or personal and professional problems) and drug use. These findings are consistent with the alcohol consumption literature (e.g. Cooper, Frone, Russell & Mudar, 1995; O'neill, Parra & Sher, 2001; Wood, Vinson & Sher, 2001), but are not of central interest to the hypotheses in this study.

Owing largely to the fact that participants in all beverage groups received training in the task, there were few differences across groups in flanker performance. There was no effect of beverage on reaction time. However, participants in the placebo group were significantly more accurate than other participants on compatible trials, suggesting a potential compensation effect among placebo participants (e.g., Fillmore & Blackburn, 2002; Williams, Goldman, & Williams, 1981). Beverage effects may have been stronger if the task had been more difficult. Although flanker tasks such as the one used here have been utilized many times to assess cognitive control (e.g. Eriksen & Eriksen, 1974;

Ridderinkhof, 2002; Bartholow et al., 2003), the ceiling effect for compatible trials indicates that at least half of the trials presented very little difficulty for all participants.

The most interesting finding in this study is likely the interactive effect between the compatibility effect in accuracy and beverage group in predicting risky behavior. In this analysis the placebo group appeared to be the only group in which there was a relationship between any task-related variables and risk score. For participants in the placebo group, an increasing number of risk-taking behaviors was associated with a decreasing compatibility effect (in accuracy) during the flanker task.

Though it is surprising that the interactions between risk score and behavioral indices were not affected by variability in the alcohol group, it may be that the results in the placebo group are the result of the compensatory effects often seen in other studies in which participants given a placebo appear to try harder because they have an expectancy that their performance may be impaired by intoxication (see Fillmore & Blackburn, 2002; Testa et al., 2006; Williams et al., 1981). On one level it is rather bizarre that increased control in the task (i.e. a smaller compatibility effect) would ever lead to an association with greater risk taking. However it could be that people that tend to take more risks while intoxicated may have a strong expectancy about alcohol's disinhibiting effects. In turn they may try even harder than other individuals in the placebo group to overcome the disruptive effects they believe alcohol has on cognitive functioning and task performance. This finding suggests that alcohol expectancy may actually lead to performance improvement for individuals that are prone to taking risks. Further analyses controlling for alcohol expectancies in this group could help explain this effect

As expected, alcohol dampened the amplitude of the ERN as compared to control and placebo beverages. This is consistent with previous literature (e.g. Bartholow et al., 2008) which suggests that the ERN represents the activity of the conflict-monitoring mechanism associated with the evaluative component of control, which increases when attempts at control fail (i.e., making an error in the task). In other words, alcohol participants are likely less distressed by their mistakes than participants in the control condition or placebo condition (which also suggests this effect is not simply due to alcohol expectancy). Unfortunately, there appeared to be no interactive effect between alcohol consumption, ERN amplitude and risk taking behavior as hypothesized. In addition ERN amplitude did not significantly predict control variables.

Surprisingly no differences were seen between beverage groups for the NSW. The only significant effect was the expected compatibility difference in which NSW amplitudes were greater for incompatible trials. It is difficult to make conclusions about the relationship between flanker performance, neural measures of cognitive control and self-reported risky behavior from the results of this study considering the lack of statistically significant results. While it is possible there may be no true relationship between these variables, it is also possible this experiment simply did not adequately measure these relationships.

There are several issues that could have affected the ability to detect effects. First, it is clear the task may have been too easy for many participants for whom there was a very high accuracy rate overall, but particularly for compatible trials. One simple step that could be taken in future research would be to impose a response deadline that would force participants to make a decision more quickly. However, based on the

experimenter's subjective observation participants generally appeared motivated to perform well and may not actually have been able to respond any more quickly to stimuli, in which case only the accuracy rate would have been affected since participants would be more likely to guess an answer when they were uncertain what the correct response was. A different option would be to use more complex or varied stimuli, for example by using arrows that vary not only by left and right direction but also by facing down or up. Another potential problem could be simply not having enough participants in the study to detect effects since including less than 100 participants provides relatively little power for between-subjects variables (Sex, Beverage Condition and self-report measures). Though this was a disadvantage of the study design, it could not be avoided due to the restrictions of the larger study of which this was a part of. Finally, another important issue was the low reliability of the Risky Behavior Questionnaire measure. Future research would benefit from further scale development of a questionnaire related to risky behaviors which would allow researchers to detect more variability both within and between subjects. On the other hand an intentional decision was made to attempt to keep the Risky Behavior Questionnaire short in this study to not overburden participants for whom it generally took 45 minutes to an hour to complete the entire battery of initial self-report measures. Unfortunately the trade-off for including fewer questions is typically a lower reliability, which makes sense particularly in this domain since some behaviors such as smoking marijuana appear to be very common and potentially bear no relationship with other behaviors such as having sex with multiple partners.

Though the research presented here did not provide support for the hypothesis that alcohol would moderate individual differences in interference control and that those

differences would be associated with self-reported risk-taking behaviors, this research did show that in the placebo group an increasing number of risk-taking behaviors was associated with a decreasing compatibility effect in accuracy during the flanker task. While difficult to interpret, this finding suggests that expectancy plays an important role in the interpretation of one's alcohol experience and may account for an individual's behavior beyond what would be expected from the neurochemical changes produced by ingesting alcohol alone (Hull & Bond, 1986; Fillmore & Blackburn, 2002). If true, then alcohol expectancies may play an important role in risky behavior that is related to alcohol use and therefore should be an important component of future research on this topic (Derman & Cooper, 2000). Indeed, there is much left to explore regarding the relationship between laboratory measures of self-control, executive function, alcohol use and risky behavior. Future research can potentially explore these topics with different cognitive tasks that tap other aspects of self-control, such as inhibitory control, in addition to using more refined questionnaire measures or longitudinal follow-ups that may be more informative than retrospective self-reports.

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FOOTNOTES

1. Quantity/frequency of alcohol use was a significant predictor in each of the models we tested. However, to avoid redundancy, we report this effect only once in the text.

Table 1

Correlations among Study Variables

	1	2	3	4	5	6	7	8
1 EF								
2 quant_freq	0.1127							
3 CE_RT	0.03704	0.06828						
4 CE_acc	-0.13774	-0.05237	0.02233					
5 ERN_incompat	-0.03386	-0.05644	0.00398	0.18427				
6 ERN_compat	0.02374	0.05558	0.04274	0.16703	0.37926*			
7 riskscore	0.14285	<i>0.18316</i>	-0.02047	-0.03536	-0.07848	-0.05526		
8 HAVEYOUSCORE	0.17734	0.23057*	-0.00671	-0.05667	-0.07074	-0.05972	0.98626*	
9 FUN	0.01094	0.2973*	0.04861	0.13775	0.15407	-0.0119	0.30035*	0.34179*
10 sexual	0.03941	-0.00035	-0.05402	0.00727	-0.07904	-0.02958	0.72742*	0.68387*
11 DANGER	-0.05871	-0.00218	-0.09831	-0.13172	-0.01243	0.01001	0.25588*	0.25818*
12 bis11score	-0.02093	0.02903	0.06542	0.00847	0.00114	0.0198	0.06613	0.07016
13 sensationseeking	-0.0289	0.03724	-0.1566	-0.05602	-0.01661	-0.03351	0.60912*	0.58398*
14 alc_probs	0.2408*	0.34752*	0.06907	-0.13593	0.01033	-0.04842	0.35439*	0.504*
15 probscore	0.25679*	0.42127*	0.10149	-0.14003	-0.03033	0.07719	0.36381*	0.44319*

Note: * $p < .05$. *Italics* indicate a potentially interesting (though nonsignificant) trend. EF = executive function composite; quant_freq = quantity/frequency of alcohol use; CE_RT = compatibility effect in reaction time; CE_acc = compatibility effect in accuracy; ERN_incompat = ERN amplitude at the Fcz electrode for incompatible incorrect trials; ERN_compat = ERN amplitude at the Fcz electrode for compatible incorrect trials; riskscore = composite score of endorsed risky behaviors not including alcohol use items; HAVEYOUSCORE = composite score of endorsed risky behaviors including alcohol use items; FUN = composite score of how fun participants felt the risky behaviors were; sexual = composite score of risky sexual behaviors; DANGER = composite score of how dangerous participants felt the risky behaviors were; bis11score = composite of the Barratt Impulsivity Scale (version 11); sensationseeking = composite of sensation seeking related items; alc_problems = composite of alcohol-related items from the risky behaviors questionnaire; probscore = composite of items from the problems with alcohol questionnaire; group = beverage condition.

Table 1 (Cont'd.)

Correlations among Study Variables

Variables

	9	10	11	12	13	14	15
1 EF							
2 quant_freq							
3 CE_RT							
4 CE_acc							
5 ERN_incompat							
6 ERN_compat							
7 riskscore							
8 HAVEYOUSCORE							
9 FUN	0.34179*						
10 sexual	0.68387*	0.11221					
11 DANGER	-0.25818*	-0.54385*	-0.18746				
12 bis11score	0.07016	0.03764	-0.015	-0.0496			
13 sensationseeking	0.58398*	0.15477	0.503*	-0.08183	0.05378		
14 alc_probs	0.504*	0.36436*	0.06783	-0.12358	0.05139	0.12095	
15 probscore	0.44319*	0.16599	0.02738	0.04752	-0.01724	0.05048	0.61787*

Figure 1

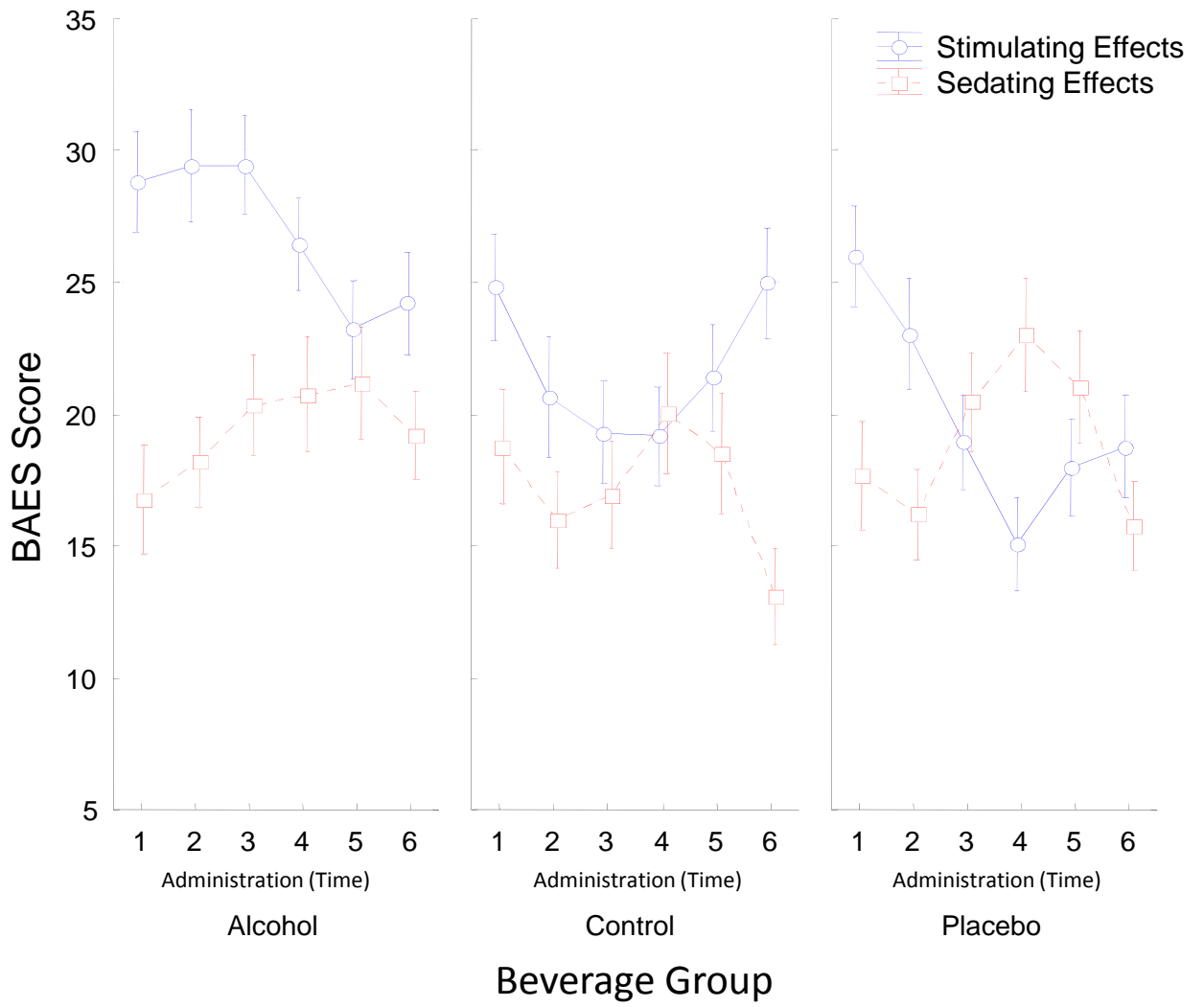


Figure 2

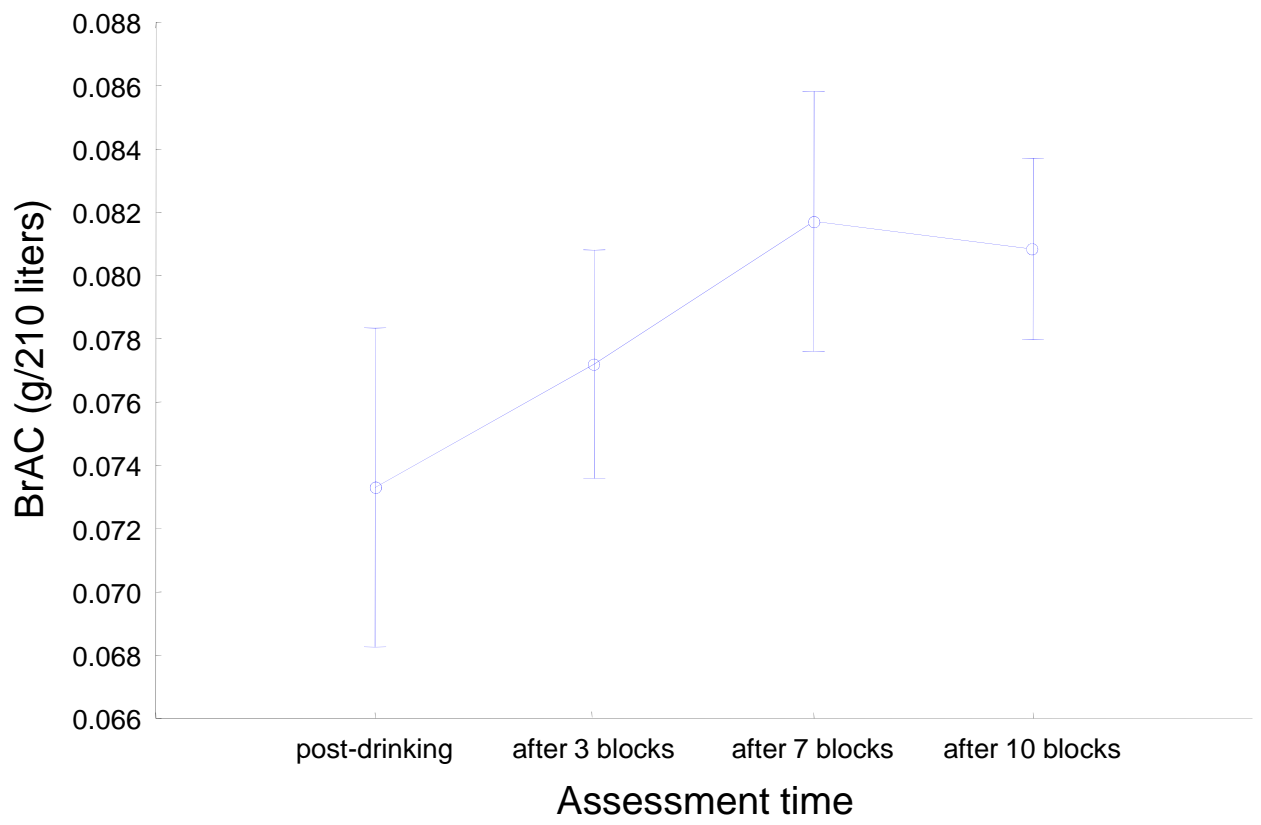


Figure 3

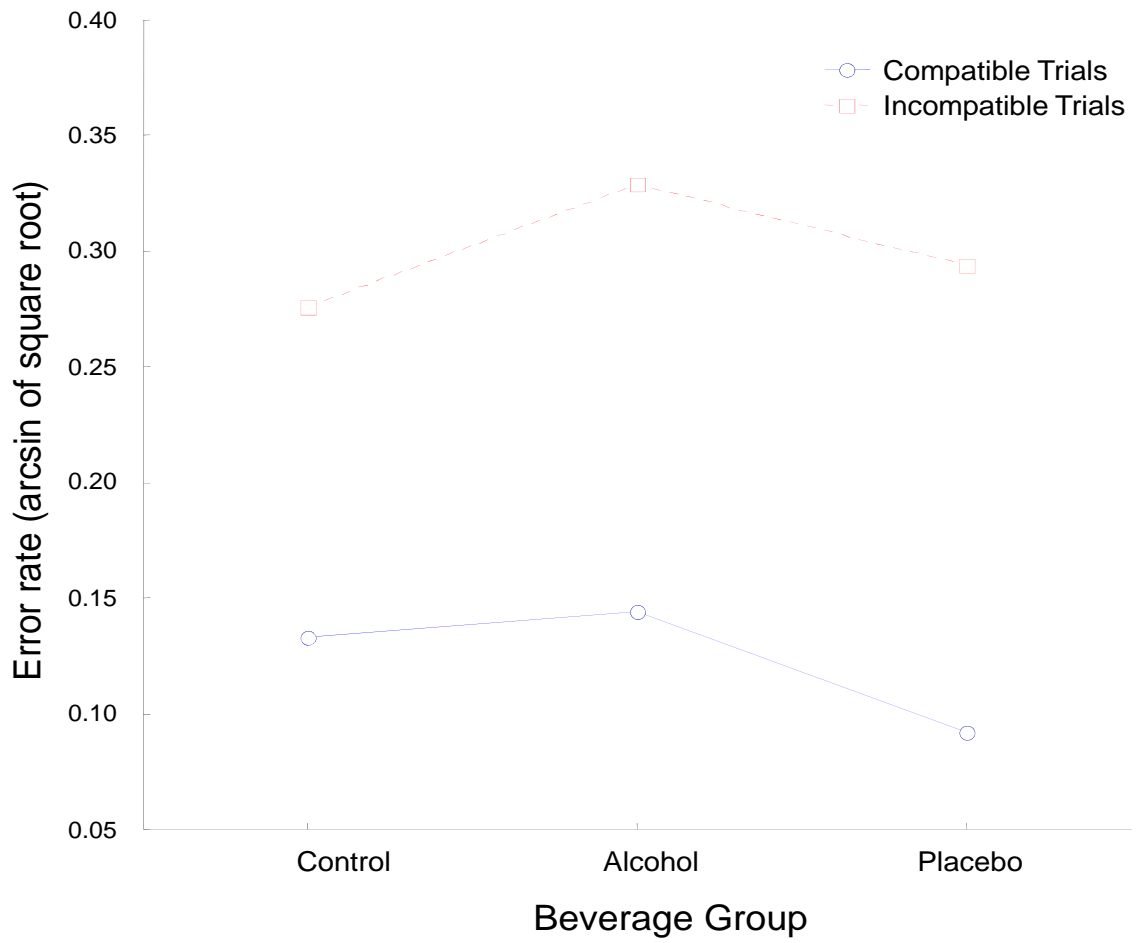


Figure 4

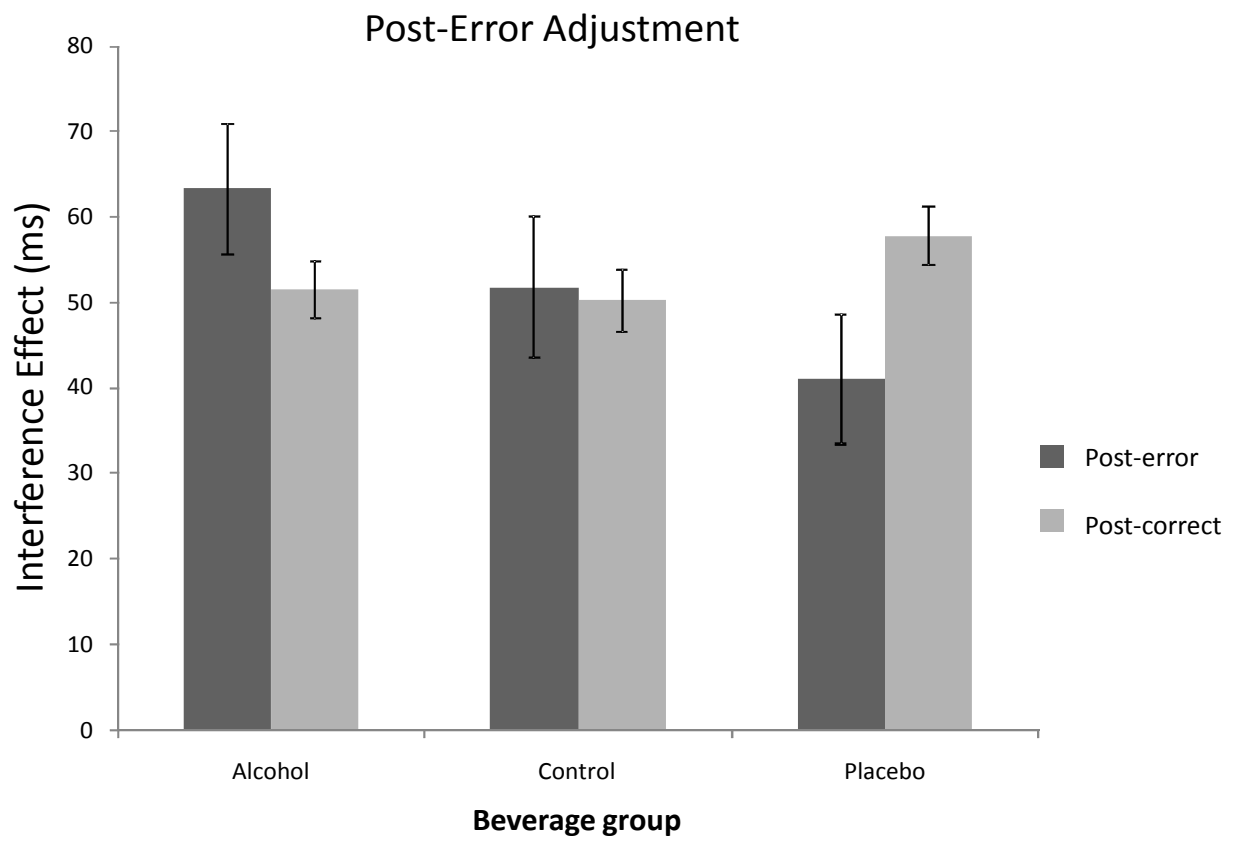


Figure 5

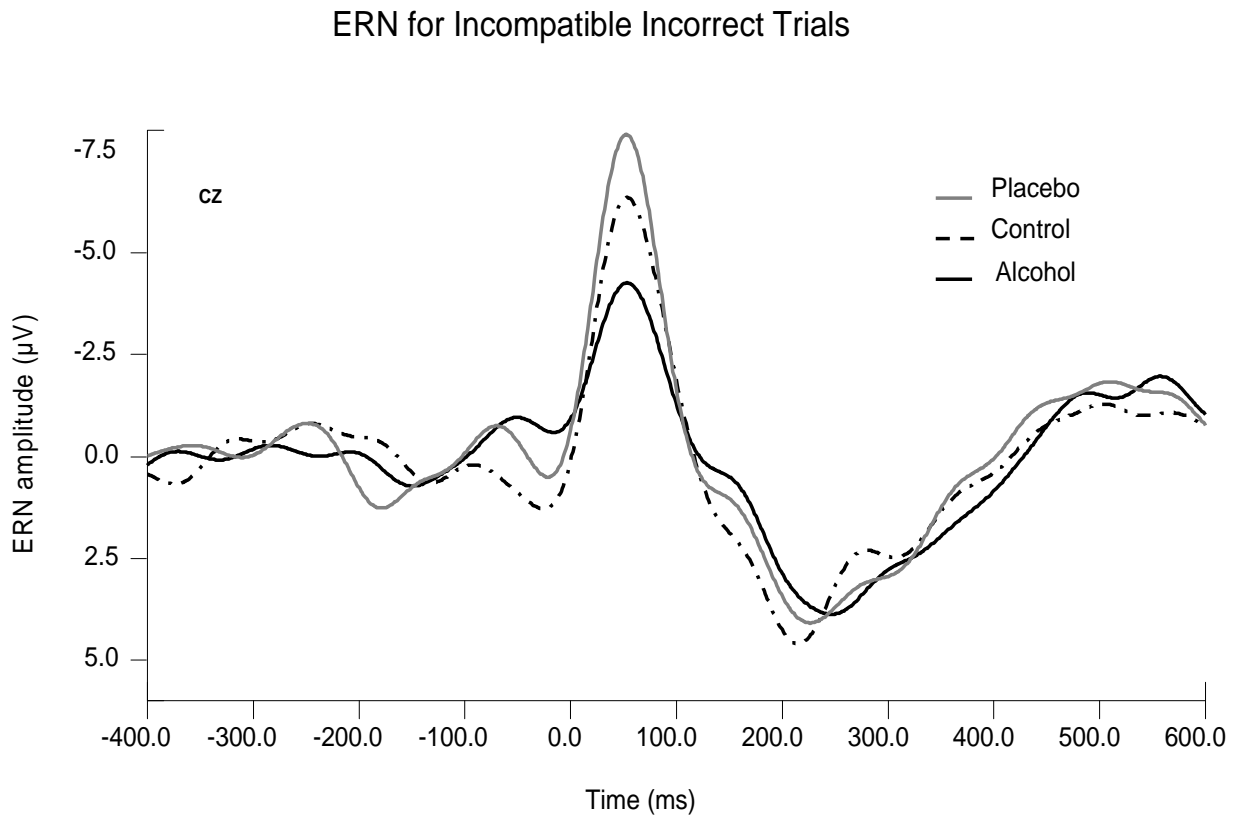


Figure 6

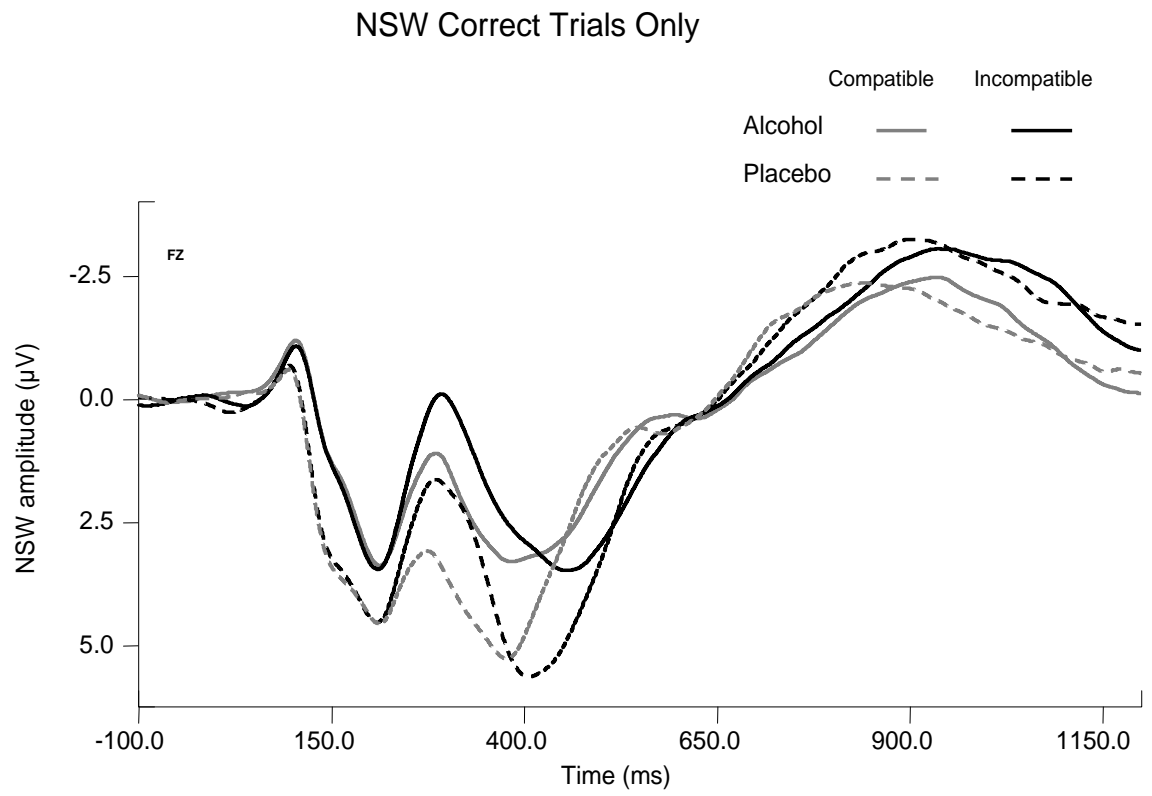
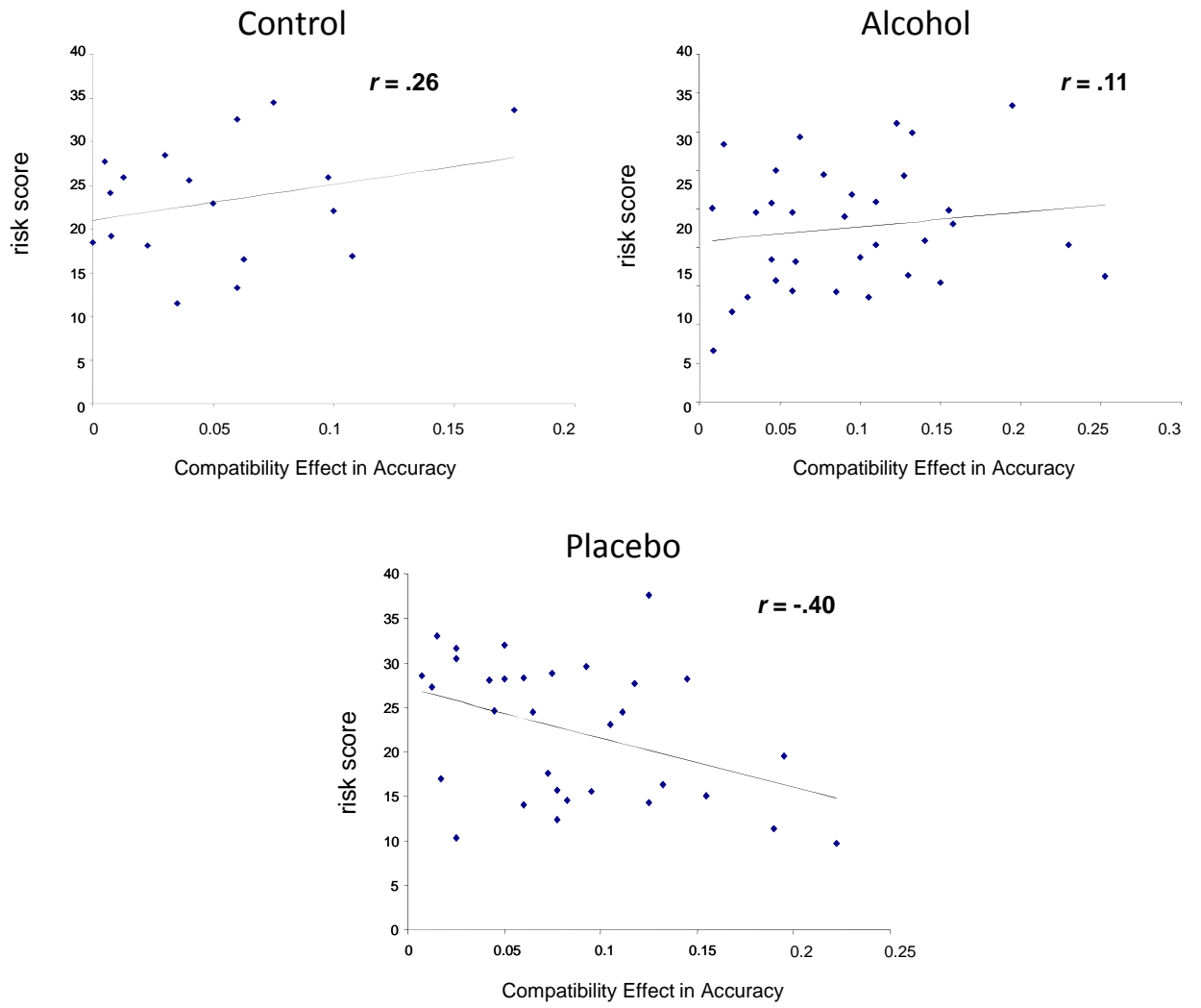


Figure 7



APPENDIX

Risky Behaviors Questionnaire

Have you:	NEVER	Yes, but not in the past year	Yes, in the past year, but not in the past three months	Yes, in the past three months			
				Once	Twice	3 times	4+ times
drank alcohol							
gotten drunk							
rode with a drunk driver							
drove drunk							
passed out in a strange location							
used cocaine/crack							
taken speed							
used marijuana							
used meth							
taken prescribed drugs for recreational use							
abused prescribed drugs							
had sex with a condom							
had sex without a condom							
had anal sex with a condom							
had anal sex without a condom							
had sex with a stranger							
had sex with multiple partners							
had sex while drunk							
had sex while high on drugs							
gotten into a sexual situation you later regretted							
vandalized property							
gambled							
drag raced							
rode a motorcycle without a helmet							
shoplifted							
not used a seatbelt							
smoked cigarettes							
missed work or missed a class							
gotten into trouble at work or school							
been fired from a job or suspended or expelled from school							

In the <i>next</i> 3 months how often do you think you will ___?	1	2	3	4	5	6	7	8	9
	never				weekly				daily
drink alcohol									
get drunk									
ride with a drunk driver									
drive drunk									
pass out in a strange location									
use cocaine/crack									
take speed									
use marijuana									
use meth									
take prescribed drugs for recreational use									
abuse prescribed drugs									
have sex									
have sex without a condom									
have anal sex									
have anal sex without a condom									
have sex with a stranger									
have sex with multiple partners									
have sex while drunk									
have sex while high on drugs									
vandalize property									
gamble									
drag race									
get into a physical fight									
ride a motorcycle without a helmet									
shoplift									
not use a seatbelt									
smoke cigarettes									
miss work or miss a class									
get into trouble at work or school									

How risky or dangerous would be it for you to ___?	1	2	3	4	5	6	7	8	9
	Not at all risky				Somewhat risky				Very risky
drink alcohol									
get drunk									
ride with a drunk driver									
drive drunk									
pass out in a strange location									
use cocaine/crack									
take speed									

How risky or dangerous would be it for you to ___? (continued from last page)	1	2	3	4	5	6	7	8	9
	Not at all risky				Somewhat risky				Very risky
use marijuana									
use meth									
abuse prescribed drugs									
have sex with a condom									
have sex without a condom									
have anal sex with a condom									
have anal sex without a condom									
have sex with a stranger									
have sex with multiple partners									
have sex while drunk									
have sex while high on drugs									
vandalize property									
gamble									
drag race									
get into a physical fight									
ride a motorcycle without a helmet									
shoplift									
not use a seatbelt									
smoke cigarettes									
miss work or miss a class									
get into trouble at work or school									

How fun would it be for you to ___?	1	2	3	4	5	6	7	8	9
	Not at all fun				Somewhat fun				Very fun
drink alcohol									
get drunk									
ride with a drunk driver									
drive drunk									
pass out in a strange location									
use cocaine/crack									
take speed									
use marijuana									
use meth									
take prescribed drugs for recreational use									
have sex without a condom									
have anal sex with a condom									
have anal sex without a condom									
have sex with a stranger									
have sex with multiple partners									
have sex while drunk									
have sex while high on drugs									

How fun would it be for you to ___ ? (continued from last page)	1	2	3	4	5	6	7	8	9
	Not at all fun				Somewh at fun				Very fun
vandalize property									
get into a physical fight									
ride a motorcycle without a helmet									
shoplift									
not use a seatbelt									
smoke cigarettes									
miss work or miss a class									
get into trouble at work or school									