

EFFECTS OF ALCOHOL AND EMOTION REGULATION ON PERFORMANCE
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ABSTRACT

Previous research indicates that alcohol dampens negative affect and is sometimes used in the regulation of psychological distress (see Greeley & Oei, 1999; Sher, 1987). Other work indicates that individuals using less effective emotion regulation strategies are at heightened risk for developing alcohol use problems (Cooper et al., 1995). To date, however, no research has directly tested the extent to which an acute dose of alcohol differentially affects individuals differing in emotion regulation styles. This issue could have important implications for understanding how the potential benefits (decreased negative affect) and costs (impaired cognitive control) associated with alcohol use differ between individuals, and how such differences might increase understanding of risk for developing alcohol use disorders. For example, individuals at particular risk for developing alcohol use problems may benefit the most from the affect regulatory benefits of alcohol while suffering the least from the cognitive impairment properties that accompany intoxication, a combination with the potential to strongly reinforce alcohol use. The primary purpose of the current research was to investigate whether individual differences in emotion regulation modulate alcohol's effects on a task that engages both emotional and cognitive processes, using a combined behavioral and psychophysiological approach. Participants were randomly assigned to one of three beverage groups (alcohol, placebo, or control beverage) and then engaged in a trial-and-error learning task (Holroyd & Coles, 2002) while event-related brain potentials (ERPs) were recorded. Individual differences in emotion regulation were assessed using the emotion regulation questionnaire (ERQ; Gross & John, 2003).

Consistent with hypotheses, learning was less robust for the alcohol group overall in comparison to the placebo and control groups. Data reflecting this difference was found in both behavioral measures of performance (i.e., categorization accuracy) and the neural markers underlying performance monitoring (i.e., ERN amplitude). This main effect was qualified by an Emotion Regulation x Beverage interaction indicating that beverage consumption differentially affected performance as a function of emotion regulation groups. Specifically, Reappraisers were less accurate following alcohol in comparison to placebo and control beverage consumption. In contrast, Suppressors' accuracy was not influenced by the beverage they consumed. A similar pattern was evident in the ERN amplitude data. Specifically, whereas Reappraisers' ERN amplitude was attenuated following alcohol relative to placebo and control beverage consumption, Suppressors showed no effect of beverage on their ERN amplitude. Although amplitude of the feedback ERN (fERN) did not vary as a function of beverage condition, it was affected by emotion regulation. Specifically, whereas Reappraisers showed an increase in fERN amplitude for trials in which learning was more likely compared to trials where learning was less likely, Suppressors showed no such difference.

Taken together, the current results demonstrate that while Reappraisers experience significant cognitive impairment following alcohol consumption (reflected in both behavioral and neural measures), Suppressors experience very little impairment. Given that reappraisal is considered a more effective emotion regulation strategy than suppression, Reappraisers may be resistant to developing alcohol problems as they appear to have less need to use alcohol to aid their emotion regulation efforts. Also, given the degree to which alcohol impairs their cognitive ability, the effects of alcohol may be

particularly aversive for Reappraisers. Suppressors, however, may not be so lucky. Given that suppression is largely ineffective at altering emotional experience at will (Gross & John, 2003), alcohol may be particularly enticing to these individuals given the emotion-regulatory benefits it provides. This fact, coupled with the current finding that Suppressors experience less cognitive cost associated with drinking, may put this group at heightened risk for developing alcohol use disorders. In other words, when alcohol intoxication provides a rewarding affect-regulatory experience with seemingly little cost, virtually every theory of alcohol abuse predicts an increasing pattern of problematic use that, over time, has the potential to lead to drinking-related problems (see Cooper et al., 1995; Lang et al., 1999).

Chapter 1

Introduction

Alcohol abuse and dependence are among the most frequently diagnosed psychiatric disorders in the U.S. (Chen et al., 2006). An important factor in many such diagnoses appears to be difficulty regulating emotion. Drinking to regulate negative emotions has been theorized as the strongest motivational correlate of problematic alcohol involvement (see Sher & Grekin, 2007), and emotion regulation has been an essential component underlying most major theories of drinking behavior (see Blane & Leonard, 1999). Experimental work indicates that alcohol dampens stress responses (see Greely & Oei, 1999), and in large doses has a direct anxiolytic effect specific to negative affect (Donahue et al., 2007; Moberg & Curtin, 2009). Such findings have led to the hypothesis that alterations in affective response play a key role in individual differences in drinking behavior and the likelihood of developing alcohol use disorders (see Koob & Le Moal, 2001).

The empirical investigation of the relationship between alcohol and affect was spurred by Conger's (1951, 1956) "tension reduction hypothesis". The tension-reduction (or drive-reduction as it would later become) hypothesis posited that the primary reinforcing mechanism underlying alcohol consumption was the attenuation of some emotional or physiological state. Empirical work following this theory has found that in the presence of noxious stimuli, alcohol consumption diminishes stress responses, as indicated by physiological measures such as electrodermal response (Carpenter, 1957;

Coopersmith, 1964; Greenberg & Carpenter, 1957, Lienert & Traxel, 1959;), cardiac (Lehrer & Taylor, 1974), and startle magnitude (Moberg & Curtin, 2009), and self-reported anxiety. Since this early work, much research has investigated and supported the stress-response dampening (SRD) properties of alcohol (Levenson, Sher, Grossman, Newman, & Newlin, 1980; Sayette, 1993; Sher et al., 2007; see Sher, 1987). Of particular interest, research has found that due to these SRD properties, some situations inducing negative affect and/or stress motivate alcohol consumption (e.g., Cornelius et al., 2003; Curran et al., 2002; Flynn et al., 2004; Litt et al., 1990; Miller et al., 1996). For example, Litt et al. (1990) demonstrated that alcoholics who received a negative mood induction rated their desire to drink higher than those receiving a neutral mood induction. Similarly, research shows a strong relationship between alcohol relapse and emotional distress (Cornelius et al., 2003; Curran et al., 2002; Flynn et al., 2004; Miller et al., 1996). In sum, alcohol has been shown to reduce stress and negative affect and some stressful or emotional situations motivate alcohol consumption.

Given alcohol's SRD properties, Sher and Levenson (1982) have argued that individuals experiencing higher levels of stress-response dampening may be particularly drawn to alcohol. Along these lines, individuals high on trait impulsivity, a population well-known for seeking out alcohol (see Sher, Bartholow, & Wood, 2000), have been found to experience greater sensitivity to the stress-reducing properties of alcohol (Levenson, Oyama, & Meek, 1987; Sher, Bylund, Walitzer, Hartmann, & Ray-Prenger, 1994; Sher & Levenson, 1982). The SRD properties of alcohol may also be particularly enticing to individuals who experience greater difficulty regulating their emotions. In support of this argument, alcohol use has been found to be highly comorbid with

disorders involving abnormalities in affective response (e.g., anxiety; mood disorders; Reiger et al., 1990). Furthermore, Cooper et al. (1995) found among population-based samples that drinking to cope with negative emotion was strongly associated with heavy drinking.

In spite of the relationship between alcohol and emotion regulation, research to date has failed to directly investigate this issue experimentally, or to test potential mechanisms through which such individual differences might influence affect-related responses following alcohol consumption. The present research seeks to identify whether mechanisms underlying basic self-regulatory processes are differentially affected by alcohol consumption for individuals utilizing differing emotion regulation strategies. Better understanding of these mechanisms has the potential to reveal the factors that put poor emotion regulators at risk for substance use disorders.

Individual Differences in Emotion Regulation

Emotion researchers have identified stable individual differences in emotion regulation strategies associated with the experience of both positive and negative affect (Gross & John, 2003; John & Gross, 2004). Of particular interest here are two commonly used strategies referred to as cognitive reappraisal and expressive suppression. *Cognitive reappraisal* is categorized as an antecedent-focused strategy, meaning it occurs early in the emotion generative process allowing it to intervene before an emotional response has been fully generated. Thus, using cognitive reappraisal allows the ensuing course of emotion to be altered. For example, an individual using reappraisal might view a loss (e.g., in a game) as an opportunity to learn from mistakes and improve. *Expressive suppression*, however, is considered a response-focused strategy because it occurs later in

the emotion-generative process, modifying the behavioral aspect of emotional response. In contrast to cognitive reappraisal, then, expressive suppression is associated with altering the expression of emotion but not the course of subsequent emotion. Continuing the game analogy, individuals using expressive suppression regulate emotions by keeping a “poker face” regardless of the cards they hold, thereby not allowing the emotion they feel to be visibly expressed.

Previous research has established that differences in the use of these emotion-regulation (ER) strategies alter affective experience. For instance, individuals who tend to use reappraisal (i.e., Reappraisers) have been found to report greater positive emotion and diminished negative emotion, whereas those who generally use suppression (i.e., Suppressors) experience decreased positive emotion and greater negative emotion (Gross & John, 2003). Additionally, compared to Suppressors, Reappraisers demonstrate superior social interaction and enhanced well-being (Gross & John, 2003). These strategies also have been associated with differences in physiological stress reactivity. Hageman et al. (2007) found that, compared to Reappraisers, Suppressors exhibit increased sympathetic activation of the cardiovascular system during acoustic startle. Taken together, these findings suggest that reappraisal tends to be a more effective strategy for regulating emotions than suppression, and that ironically suppression can lead to increased distress. Thus, given that their default strategy is not particularly effective, it is possible that Suppressors may be more likely to seek out alcohol as a way to supplement their regulation of negative experience, and that they might derive greater affect-regulatory benefit from alcohol, both of which could put them at increased risk for developing alcohol abuse (see Cooper et al., 1991).

Alcohol and Self-Regulation

Although alcohol consumption accompanies a host of affect regulatory benefits to the drinker, these perks often come at a cost. Specifically, as much as alcohol is well-known for attenuating negative affect, it is also known for inducing cognitive impairment (see Finnegan & Hammersly, 1992). Among the most notable situations in which alcohol produces negative effects on cognition, is in operating a vehicle (e.g., Ferrara, Zancaner, & Giorgetti, 1994; Howat & Sleet, 1991). The behavioral adjustments needed to drive a car require adaptive control of ongoing behavior (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Groeger, 2000). Alcohol, however, has been shown to disrupt critical components of performance monitoring and self-regulation processes (Bartholow, Henry, Lust, Sauls, & Wood, in press; Ridderinkhof et al., 2002). Specifically, effective self-regulation involves coordination between an *evaluative* mechanism, which monitors for conflict between intended (i.e., goal-directed) and actual behavior, and a *regulative* mechanism that implements cognitive control when necessary to achieve goals (see Botvinick et al., 2001; Kerns et al., 2004). When current performance is inadequate, the evaluative mechanism is thought to signal the regulative mechanism, instigating increased implementation of control-related processes (Botvinick et al., 2001; Kerns et al., 2004). One mechanism by which alcohol has been found to impair self-regulation is by the attenuation of the distress that accompanies the neural marker of this evaluative process (see Bartholow et al., in press ; Ridderinkhof et al., 2002;).

In spite of the conventional wisdom and empirical evidence indicating that alcohol leads to cognitive impairment, individuals' specific expectations regarding the effect of alcohol on their cognitive abilities do not always align with common knowledge

(Christiansen & Goldman, 1983). Specifically, research has found that one of the six most common expectancies that accompany intoxication is improvements in cognitive functioning and motor control (Christiansen & Goldman, 1983). Along these lines, individuals reporting expectancies for improvements in cognitive control are at greater risk for alcohol problems (Christiansen & Goldman, 1983), while individuals reporting experiencing greater impairments are negatively associated with alcohol problems (Fromme & D'Amico, 2000). Although much work has been conducted on the general cognitive impairments as a product of intoxication, individual differences in the extent to which alcohol causes cognitive impairment is less well known.

Given that, in comparison to Reappraisal, Suppression consumes greater cognitive resources (Gross, 2002), elicits greater levels of stress (Gross, 1998), and is less effective at altering emotional responses (Gross & John, 2003), alcohol may not only be especially reinforcing, it may also be less costly on a cognitive level for individuals who primarily use this emotion regulation strategy. Specifically, due to the affect regulatory and stress-response dampening properties of alcohol, after consuming alcohol, Suppressors are likely to have less emotion in need of regulation, which in turn will free up cognitive resources that would otherwise be dedicated to emotional suppression. Additionally, given the well-known relationship between arousal and learning (Yerkes & Dodson, 1908), alcohol may also decrease the stress that accompanies the process of suppression to a level more conducive to learning. In other words, alcohol may help to “take the edge off,” just enough so that stress levels and negative affect drop to a more manageable level, allowing Suppressors to more comfortably focus on the task at hand. Thus, the aim of the current study is to investigate the interactive effects of alcohol and emotion

regulation on performance monitoring and the underlying neural mechanisms that accompany this process.

ERP measures of performance monitoring.

Research has shown that following alcohol consumption, evaluative markers of performance monitoring are impaired as indicated by the decrease in amplitude of an ERP component referred to as the error-related negativity, (ERN; (Ridderinkhof et al., 2002; Bartholow, et al., in press). The ERN is a negative deflection in the ERP that coincides with an incorrect behavioral response (see Gehring et al., 1993; Yeung et al., 2004). A role for the ERN in behavioral adjustment is supported by studies showing that a large ERN is associated with increased accuracy and response time on post-error trials (Gehring et al., 1993; Scheffers et al., 1996). Initial accounts held that the ERN reflected an error detection mechanism (Falkenstein et al., 1994; Gehring et al., 1993; see also Coles et al., 2000). However, this view recently has been challenged by studies showing ERN-like activity on correct response trials (e.g., Vidal et al., 2000), particularly when an activated response strategy is sub-optimal for determining the correct response (Bartholow et al., 2005), and studies linking the amplitude of the ERN to negative affect or distress related to failed regulatory control (Bush et al. 2000; Hajcak et al., 2004; Luu, et al., 2000). As such, the ERN is thought to represent a kind of neural “distress signal” that indicates current performance is inadequate (Bartholow et al., 2005; Bush et al., 2000).

Substantial evidence from electrocortical source localization (e.g., Dehaene et al., 1994; Luu et al., 2003) and brain imaging studies (e.g., Carter et al., 2000; Kiehl et al., 2000) places the neural generator of the ERN within the ACC, a structure long known to

play a key role in the evaluation of physical and psychological distress (e.g., Rainville et al., 1997; Talbot et al., 1991). Given that the ERN is also found on correct-response trials, Bartholow et al. (2005) posited that while erroneous responses typically elicit greater distress than correct responses (as can be seen by the increased ERN amplitude on error trials), correct response trials may also elicit distress if one is using a maladaptive response strategy.

Initial research examining the link between alcohol and performance monitoring had participants complete a task known to engage the evaluative component of self-regulatory control (the flanker task; Ridderinkhof et al., 2002). Results showed that alcohol reduced ERN amplitude following errors and impaired post-error behavioral adjustments, and interpreted this effect in terms of alcohol impairing the brain's ability to detect errors. Although a provocative finding, this interpretation fails to account for (1) findings linking the ERN to a distress signal (see above), and (2) the voluminous literature showing that alcohol reduces negative affect (see Greely & Oei, 1999). Recent findings in our lab (Bartholow et al., in press) indicating that alcohol-related reduction in ERN amplitude is associated with post-consumption changes in negative affect – and is unrelated to detection of errors -- support the notion that affect regulation plays an important role in alcohol's acute effects on self-regulatory control. A primary goal of the current research is to extend that work by investigating the extent to which individual differences in ER strategies moderate this effect.

Study Overview

The paradigm used to study these issues involved a recently developed trial and error learning task (see Holroyd & Coles, 2002). In the current trial and error learning

task, participants were to infer the correct response mapping to a number of visual stimuli via feedback received after each response. Negative feedback (i.e., that a response was incorrect) typically elicits a large feedback ERN (fERN), the amplitude of which has been linked to negative evaluations indicating that a goal (in this case, to infer the correct response) has not been achieved (see Holroyd, Hajcak, & Larsen, 2006). Once response mapping has been learned, incorrect responses elicit an ERN concurrent with the response, reflecting distress over recognition of a self-regulatory failure, and no fERN to error feedback (Holroyd & Coles, 2002; Nieuwenhuis et al., 2002).

This paradigm is useful for the current research for three main reasons. First, utilizing a trial and error learning paradigm elicits a certain degree of stress or anxiety that participants will need to regulate in order to perform adequately (see Prather, 1971). Second, performance monitoring and learning processes engaged by this task are known to be impaired by alcohol (Bartholow et al., in press; Ridderinkhof et al., 2002). Third, this paradigm permits evaluation of the extent to which distress over self-regulatory failure is associated with individual differences in emotion regulation, and allows linking these differences to known effects of alcohol on self-regulation. The ability to learn from mistakes is crucial in situations such as this, which requires effective management of the distress that errors and negative feedback elicit (see Davidson, 2000). Given that Suppressors typically utilize a less effective emotion regulation strategy, I hypothesize that effects of alcohol on performance and underlying performance monitoring mechanisms will be less severe in Suppressors compared to Reappraisers. This hypothesis is based on the notion that the decrease in distress caused by alcohol

consumption will serve to aid Suppressors' emotion regulation efforts, freeing up resources so that they can more easily focus and direct their efforts to the task at hand.

In summary, research has shown that individual differences in emotion regulation are associated with differences in the experience of negative affect (Gross & John, 2003), and that alcohol (at moderately high doses) is effective in regulating negative affect and is often used for that purpose (see Greely & Oei, 1999). However, no previous research has investigated how individual differences in emotion regulation interact with alcohol to produce differences in negative affective responding or differences in self-regulatory performance monitoring and adjustment. Investigating this issue can have important implications for understanding individual differences in both the effects of alcohol on affect regulation and on motives for using alcohol to regulate emotion. Specifically, by investigating effects of alcohol on basic processes fundamental for self-regulatory control, the current work seeks to identify why the effects of alcohol may be particularly reinforcing for individuals who have difficulty regulating negative emotion. Moreover, investigating the mechanisms responsible for alcohol's impairment of performance monitoring and adjustment, which have been implicated in alcohol-induced risk-taking and perseveration of maladaptive responding (see Ridderinkhof et al., 2002), can help inform efforts aimed at reducing drinking-related harm. Thus, the current study seeks to test how alcohol and individual differences in emotion regulation affect psychophysiological measures of distress during performance monitoring. The current work permits critical tests of the role that individual differences play in the effects of alcohol on the evaluative component of self-regulatory control, potentially suggesting new avenues for understanding the use of alcohol to regulate negative affect.

Hypotheses

The current study has three sets of hypotheses. The first set specifies how performance and the underlying neural markers of performance monitoring are expected to vary over time within the current paradigm. Specifically, performance (i.e., categorization accuracy) is predicted to improve from the first half of trials in each block to the second half, as participants are expected to learn response mappings over time. Secondly, I predict that early in trial blocks, before stimulus-response mapping has been learned, the feedback-ERN should be large and should decrease over the course of a trial block for the 80% and 100% response mapping conditions. Conversely, the response-ERN should be small or nonexistent early in trial blocks, and should become larger later, once response mapping is learned and thus performance information becomes available at the time of the response (see Holroyd & Coles, 2002; Nieuwenhuis et al., 2002).

The second set of hypotheses involves how alcohol and individual differences in emotion regulation are expected to independently affect performance. First, performance is predicted to be significantly impaired following alcohol relative to placebo and control beverage consumption. Similarly, performance improvements over time are expected to be significantly smaller for the alcohol group as the result of impaired learning processes. Lastly, ERN and fERN amplitudes are predicted to be significantly attenuated as a function of alcohol (see Bartholow et al., in press; Ridderinkhof et al., 2002). With regard to Emotion Regulation, I predict that Suppressors and Reappraisers will show similar accuracy rates. Also, I predict Suppressors will exhibit larger ERN and fERN amplitudes in comparison to Reappraisers. I predict a main effect of ER strategy, such that the amplitude of the ERN and fERN will be significantly enhanced for Suppressors

compared to Reappraisers, for two main reasons. First, Suppressors have been hypothesized to experience more negative affect/distress than Reappraisers, and the ERN appears to reflect distress related to self-regulatory failure (Luu, 2000). Second, because suppression during performance monitoring requires the regulation of two behaviors (i.e., the regulation of task behavior and the inhibition of emotion), using reappraisal should be less cognitively taxing. Thus, it is likely that the additional effort on Suppressors' cognitive control will be difficult and distressing, reflected in a larger ERN/fERN. In sum, I hypothesize that Suppressors will experience greater levels of distress when self-regulation fails, as indicated by the neural indicators of error processing generated by both internal (i.e., ERN) and external (i.e., fERN) signals, in comparison to individuals using Reappraisal.

The last set of hypotheses are specific to how Beverage and Emotion Regulation are expected to interact as a function of beverage. First, I predict that Alcohol will differentially affect performance as a function of emotion regulation strategy. Specifically, performance among Suppressors will not be as impaired as a result of alcohol. Second, alcohol will differentially affect ERN and fERN amplitudes as a function of emotion regulation. Specifically, ERN and fERN amplitude will not be as attenuated as a function of alcohol for Suppressors in comparison to Reappraisers.

Chapter 2

Method

Participants

Ninety adults (45 women) between the ages of 21 and 35 years participated in exchange for monetary compensation. Individuals older than 35 were excluded to avoid potential confounds between effects of aging and effects of alcohol on neurocognitive function. Participants were recruited using advertisements and posted flyers announcing research on the effects of alcohol. Interested individuals 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 use disorders or other serious mental or physical illness; prescription medication; pregnancy) were excluded from the sample. 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. Eligible participants must have also reported having experienced a binge-drinking episode (5 drinks at one occasion for men; 4 for women) at least once within the past year. Finally, the Fagerström Test for Nicotine Dependence (Heatherton et al., 1991) was used to test for nicotine dependence. Individuals scoring 3 or above (at least moderate dependence) were excluded in order to avoid the potential for acute nicotine withdrawal during the laboratory session. In

addition to these measures of eligibility, the emotion regulation questionnaire (ERQ; Gross & John, 2003; described in the next section) was also administered during the pre-screening interview to ensure adequate distribution of individual differences in ER strategies. Participants scoring above the median on the reappraisal subscale of the ERQ and below the median on the suppression subscale of the ERQ were recruited as ‘Reappraisers’ (n = 44). Similarly, participants scoring above the median on the suppression subscale and below the median on the reappraisal subscale were recruited as ‘Suppressors’ (n = 45).

Measures

Alcohol use. Given that behavioral performance and electrophysiological responses might differ as a function of baseline differences in drinking-related variables, participants completed self-report measures of quantity and frequency of alcohol use (Drinking Habits Questionnaire; Vogel-Sprott, 1992) and sensitivity to the effects of alcohol (SRE; see Schuckit et al., 1997; Bartholow et al., 2003a; Bartholow et al., 2007; O’Neill et al., 2002). The Drinking Habits Questionnaire assesses typical alcohol use by asking participants to estimate the number of drinking occasions typically have in a week (i.e., frequency), the amount of alcohol typically consumed on a single drinking occasion (i.e., dose), and the overall time spent drinking (in hours) during a single drinking occasion (i.e., duration). The SRE is a 16-item measure, assessing the individual’s sensitivity to the ascending and descending limb effects of the blood alcohol concentration curve (BAC). Ten items comprise the ascending limb subscale and ask participants to estimate the minimum number of drinks it takes to experience the ascending limb effects of alcohol (i.e., feeling high, talkative, flirtatious, etc). The

remaining items ask participants to estimate the maximum number of drinks they can consume before feeling the descending limb effects of alcohol (i.e., vomiting, passing out, sleepy, etc). Overall sensitivity score is calculated by averaging the number of drinks across all effects. Internal consistency within this scale has been shown to be very high ($\alpha = .97$, see O'Neill et al., 2002).

Emotion regulation strategies. Differences in emotion regulation strategies were assessed using the Emotion Regulation Questionnaire (ERQ; see Gross & John, 2003). The ERQ is a 10-item measure designed to assess individual differences in reappraisal and suppression. Six items comprise the reappraisal scale and four make up the suppression scale. Items on the reappraisal scale are characterized by the use of cognitive change as a mechanism for controlling emotions (e.g., “I control my emotions by changing the way I think about the situation I’m in”). Items on the suppression scale reflect inhibition as the primary means for controlling emotions (e.g., “I control my emotions by not expressing them”). Items are rated on a scale from 1 (strongly disagree) to 7 (strongly agree). Gross and John (2003) reported adequate internal consistency ($\alpha = .79$ & $.73$ for reappraisal & suppression, respectively) and 3-month test-retest reliability ($r = .69$) for these subscales.

Beverage Conditions and Administration

Participants were randomly assigned to one of three beverage conditions: a no-alcohol control beverage ($n = 30$), an active placebo beverage (0.04 g/kg ethanol; $n = 27$), or an alcohol beverage (1.0 g/kg ethanol; $n = 31$). Participants in the control beverage condition were informed that their beverage contained no alcohol, whereas those in the other conditions were told that their beverage contained alcohol. Beverage administration

procedures were similar to those used by Bartholow, Henry, Lust, Sauls & Wood (in press). 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), whereas the alcohol dose was achieved using undiluted 100-proof vodka. 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. The experimenter who mixed and served the beverage was unaware of the actual contents of the beverage bottles. The beverage was divided into 3 equal-size drinks to be given to the participant one at a time. Participants were allowed 5 minutes to consume each of the 3 drinks. Following completion of the third and final drink, participants sat idle for 5 min; in the alcohol condition, this allowed alcohol to be absorbed into the blood prior to the task.

Electrophysiological Recording

The electroencephalogram (EEG) was recorded continuously throughout the experimental task from 20 tin electrodes fixed in a stretch-lycra cap (ElectroCap, Eaton, OH) and placed on the scalp in standard locations (American Encephalographic Society, 1994). Epochs were derived offline to permit examination of stimulus- and response-locked ERP components of interest. EEG signals were amplified with Synamps2 amplifiers (Neuroscan Labs, El Paso, TX) and sampled at 2000 Hz using online filtering

(.05 Hz high pass; 40 Hz low pass). 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, et al., 1986). After artifact removal and rejection, EEG data were averaged off-line according to participant, electrode, and stimulus conditions.

Task Design

The task for this experiment was similar to tasks used in prior research investigating learning from feedback (Holroyd & Coles, 2002; Nieuwenhuis et al., 2002). Participants attempted to learn – inferring by trial and error – the appropriate response to each of four visual stimuli, based on feedback received shortly after a response was made. Participants simply made a speeded two-choice decision for each stimulus by pressing one of two buttons. Unbeknownst to the participants, one of the two stimuli was mapped to the left button and one to the right button, so that they were rewarded each time they pressed the correct button and penalized each time they pressed the incorrect button; these are termed 100% mappings. For two additional stimuli, feedback was mapped to the left button and one to the right button 80% of the time; these are termed 80% mappings. For the remaining two stimuli, feedback was delivered randomly, irrespective of the response, such that 50% of the trials were rewarded and 50% were penalized; these are termed 50% mappings. The 100% mapping conditions permit examination of learning over the course of each block of trials, while inclusion of the 50% mapping conditions ensures that the task is challenging. A 700 ms response deadline was used to ensure some errors due to premature responding in the 100% mapping condition even after mappings have been learned. Participants began the task with \$5.00

in bonus money, and were instructed to increase their bonus as much as possible via their performance. Correct responses were rewarded with \$.04, whereas incorrect responses resulted in loss of \$.04. To further motivate participants, on 14% of trials, there was opportunity to win or lose \$.25 instead of the usual \$.04. Responses exceeding the deadline were penalized \$.02.

The stimuli were images of musical instruments, vegetables, sports items, and flowers, and differed in each block of trials. There were two types of feedback for each trial. Feedback 1 indicated whether or not the response was correct or incorrect by showing either a green smiling face or red frowning face respectively. Feedback 2 indicated the amount of money won or lost for that particular trial (see Figure 1). On trials in which a participant responded too slowly, an image of a turtle (indicating that deadline was exceeded) accompanied the normal correct or incorrect response feedback. The experiment involved 4 blocks of 160 trials each, plus an additional block (administered prior to beverage consumption) for practice.

Procedure

Upon arrival at the lab, participants were asked to produce identification to verify age eligibility. Following informed consent, their medical and psychiatric history was briefly reassessed using self-report measures. Additionally, participants were asked to sign an affidavit attesting to their adherence to pre-experimental protocols concerning diet (fasting for 4 hours prior to the appointment) and abstention from drugs and alcohol for the previous 24 hours. Female participants were then required to self-administer a urine pregnancy test in a private restroom (female staff member verified test results). A baseline breath alcohol concentration level (BrAC) was measured to ensure participants'

initial sobriety. After ensuring eligibility, participants were then escorted to a sound-proof recording chamber where experimenters placed and tested recording electrodes. Next, participants completed a short block of practice trials (64 trials of an equal number of the 100% and 80% mapping condition only) to ensure familiarity with the trial and error learning task. After completion of the practice blocks, an experimenter, blind to the contents of the beverage in the alcohol and placebo conditions, then prepared and served the beverage in front of the participant. Following beverage consumption and a 15 minute absorption period, a second BrAC was administered. Participants then completed the trial and error learning task, stopping for a short break (around 5 minutes) between blocks. BrACs were taken during each break period. Following the completion of the task, participants completed a post-experimental questionnaire, after which electrodes were removed and participants were shown to a private restroom to clean the electrode gel from their face and hair. Participants were then debriefed, after which control and placebo participants were dismissed. Alcohol group participants were retained in the lab until a breathalyzer test indicated that they were sober ($\text{BrAC} \leq .02\%$).

Chapter 3

Results

Analytic approach. ERP components were defined according to published conventions (Picton et al., 2000). The amplitude of the response-ERN was defined as the peak negativity at electrode FCz in a window 0 - 150 ms following an incorrect response, relative to pre-response baseline. The amplitude of the feedback ERN (fERN) was defined as the peak negativity of the at electrode FCz in a window 200-350 ms following feedback onset. All component windows and primary electrode sites were verified by visual inspection of the data and adjusted if necessary.

To isolate the fERN from other overlapping components, the fERN is usually computed as a difference waveform in which the waveform elicited from positive feedback (i.e., the feedback correct-related negativity or fCRN) is subtracted from the response to negative feedback (Miltner et al., 1997; Holroyd & Krigolson, 2007). Utilizing this approach alone, however, may overlook certain between-subject factors, such as such as the attenuation of the fERN as a function of group. For example, alcohol may attenuate both the fERN and the fCRN, however subtraction analysis would not accurately capture this effect if the difference between the two components remained the same across the Placebo and Control group as well. Thus, to best grasp the effect of group differences on the fERN, the primary analyses reported here focus on peak

amplitude of the feedback ERP responses for correct and incorrect feedback (subtraction fERN results can be found in Appendix A).

The basic design was a 2 (ER strategy: Reappraisal, Suppression) x 3 (Beverage: control, placebo, alcohol) x 2 (Mapping; 100%, 80%, 50%) x 2 (Block half; trial 1-80, trial 80-160) mixed factorial with repeated measures on the final 2 factors. Primary analyses were carried out using mixed factorial ANOVAs on each main dependent variable (ERN and fERN amplitude; response accuracy, reaction time). Significant main effects of emotion regulation and beverage were followed-up using Helmert contrast analyses.

Manipulation Checks

Intoxication. Initial assessments of BrAC confirmed that upon arriving in the lab, all participants were sober (BrAC = 0.0). Follow-up assessments among the Placebo group indicated that these participants did not deviate from this initial baseline assessment (Control participants did not receive additional assessments). For alcohol participants, BrAC increased from pre- task ($M = .076$) to mid-task ($M = .09$) to post-task ($M = .13$), $F(2, 27) = 6.38$, $p = .005$, indicating that on average the entire task was completed as participants approached peak BrAC. Post-experiment estimates of the number of standard drinks consumed indicated that the alcohol group's estimate was greater ($M = 4.24$) than the placebo group's estimate ($M = 1.97$), $F(1, 47) = 13.62$, $p = .00$. Given that the Placebo group believed they had consumed almost 2 standard drinks substantiates the effectiveness of the placebo manipulation. Alcohol group participants reported feeling more intoxicated throughout the study ($M = 4.60$) than placebo participants ($M = 2.16$), $F(1, 50) = 39.10$, $p < .00$. However, the pattern of responses from

pre- task to mid-task was consistent, such that participants from both groups reported feeling more intoxicated as the task progressed (Alcohol: $M_{pre} = 4.23$, $M_{mid} = 4.93$; Placebo: $M_{pre} = 2.30$, $M_{mid} = 2.46$). Groups did differ in the pattern of responses from mid-task to post-task, such that while alcohol participants reported that they continued to feel more intoxicated ($M_{post} = 5.10$), participants in the Placebo group reported feeling less intoxicated as the task progressed ($M_{post} = 2.17$).

Behavioral Data

Accuracy. Initial analyses revealed a Response Mapping x Block Half interaction, $F(2, 89) = 3.51$, $p = .03$, indicating that performance improvements between the first and second block-halves varied as a function of response mapping conditions. Specifically, as predicted participants' performance in the 50% mapping condition did not differ between the first ($M = .49$) and second half of trials ($M = .50$; $t(90) = 0.5$, $p = .61$). Conversely, performance in the 100% mapping condition significantly improved from the first half ($M = .67$) to the second half of trial blocks ($M = .71$), $t(90) = 3.78$, $p = .00$. Surprisingly, although feedback was mostly consistent in the 80% mapping condition, performance did not significantly improve across block halves ($M_1 = .56$; $M_2 = .57$; $t(90) = 0.5$, $p = .66$), indicating that participants had a difficult time learning the response mappings for this condition. Given significant improvements in accuracy only occurred on trials in which feedback was always consistent, further analysis of accuracy data was focused on the 100% mapping condition only (for a similar argument see Holroyd & Coles, 2002).

A 3 (Beverage: Alcohol, Placebo, or Control) X 2 (Emotion Regulation: Reappraisal vs. Suppression) X 2 (Block half: First vs. Second) ANOVA on accuracy

rates in the 100% mapping condition showed a main effect of Beverage, $F(2, 85) = 3.47$, $p = .04$. Consistent with hypotheses and past research demonstrating performance decrements following alcohol ingestion (see Finnegan & Hammersly, 1992), inspection of the means indicated that the Alcohol group ($M = .65$) was significantly less accurate than the Placebo group ($M = .72$), $t(57)=2.69$, $p = .009$, and marginally less accurate compared to the Control group ($M = .69$), $t(61)=1.43$, $p = .16$. The Placebo and Control groups did not differ significantly in accuracy, $t(58)=1.28$, $p = .20$.

This main effect was qualified by a significant Block Half x Beverage interaction, $F(2, 84) = 5.04$, $p = .009$, indicating that the Beverage effect differed across block halves. Specifically, performance significantly increased across halves for the Placebo group ($M_1 = .69$; $M_2 = .76$; $t(27) = 4.73$, $p = .00$) and marginally increased for the Control group ($M_1 = .68$; $M_2 = .70$, $t(31) = 1.71$, $p = .09$). In contrast, performance in the Alcohol group did not differ across halves ($M_1 = .65$, $M_2 = .66$; $t(30) = .57$, $p = .57$).

A significant Beverage x Emotion Regulation interaction also emerged, $F(2, 84) = 3.26$, $p = .04$, indicating that the beverage effect differed as a function of emotion regulation group. Planned contrasts showed that, among Reappraisers, performance was significantly worse in the Alcohol group ($M = .63$) compared to the Placebo group ($M = .76$; $t(57) = 3.69$, $p = .00$), and the Control group ($M = .69$), though this difference was not significant, $t(61) = 1.54$, $p = .13$. Performance was also better in the Placebo group than in the Control group, $t(58) = 2.14$, $p = .04$. Notably, Suppressors' performance was unaffected by beverage contents ($M_A = .68$; $M_C = .69$; $M_P = .68$, $t_s < 1$, $p_s > .57$).

This 2-way interaction was further qualified by a significant Block Half x Beverage x Emotion Regulation interaction, $F(2, 84) = 3.26, p = .04$. Inspection of Figure 2 suggests that the two-way interaction between Beverage and Emotion Regulation was driven by performance differences in the second half of trials. This visual pattern was confirmed by a significant Beverage x Emotion Regulation interaction for the second half of trials only, $F(2, 85) = 5.07, p = .01$; the analogous interaction in the first half of the trials was not significant, $F(2, 85) = 1.07, p = .35$. Follow-up comparisons of this interaction's simple effects revealed that among Reappraisers, accuracy in the second half of trials was significantly worse in the Alcohol group ($M = .63$) in comparison to the Placebo group ($M = .82; t(57) = 4.63, p = .00$), and was marginally worse than in the Control group ($M = .70; t(61) = 1.7, p = .09$); placebo and control group means also differed significantly, $t(58) = 2.95, p = .00$. Importantly, accuracy among Suppressors in the second half of trials did not differ as a function of beverage group ($M_A = .68; M_C = .71; M_P = .69, ts < 1, ps > .59$).

Reaction times. Analyses of response latency data showed a main effect of Response Mapping, $F(2, 89) = 81.3, p = .00$, indicating that response times in the 100% mapping condition ($M = 354$ ms) were significantly quicker than the 50% ($M = 371$ ms; $t(89) = 9.02, p = .00$) and 80% mapping conditions ($M = 371$ ms; $t(89) = 9.76, p = .00$). Response latencies between the 80% and 50% mapping conditions did not significantly differ, $t(89) = .16, p = .88$.

Although not significant, there was a notable trend indicating a Response Mapping x Emotion Regulation interaction, $F(2, 89) = 2.96, p = .089$, indicating that

response latencies among Reappraisers and Suppressors differed in the 100% mapping condition. Specifically, Suppressors ($M = 352\text{ ms}$) were quicker to respond than Reappraisers ($M = 356\text{ ms}$). In contrast, response times did not vary between Reappraisers and Suppressors in the 50% ($M_R = 370\text{ ms}$; $M_S = 372\text{ ms}$) and 80% ($M_R = 370\text{ ms}$; $M_S = 372\text{ ms}$) mapping conditions. In spite of these mean differences, follow-up contrasts of these simple effects were not significant ($ts < 1, ps > .20$).

ERP Data

ERN. An initial analysis was conducted to determine the scalp distribution of the ERN. Consistent with past research, a 5 (Coronal; frontal, fronto-central, central, centroparietal, parietal) x 3 (lateral; left, midline, right) repeated measures ANOVA showed a Coronal x Lateral interaction, $F(8, 166) = 9.34, p = .000$ indicating that the ERN was largest at frontal and fronto-central midline locations, especially FCz ($M = -3.78$). Thus, to simplify analyses only data from this location was included in the main analyses reported here. Figure 3 (a and b) shows response-locked ERP waveforms as a function of Beverage Group separately for Suppressors and Reappraisers for the 100% mapping condition at electrode FCz.

ERN amplitudes measured during incorrect response trials from electrode FCz were submitted to a 3 (Beverage group) x 2 (Emotion regulation) x 3 (Response mapping) Consistent with past research (Holroyd & Coles, 2002), results show a main effect of Response Mapping $F(2, 80) = 72.56, p = .00$, indicating that ERN amplitudes were largest for the 100% mapping condition ($M = -5.31$) and smallest in the 50% mapping condition ($M = -2.96$; $M_{80\%} = -3.01$). Comparison of the simple effects revealed that while the ERN amplitude did not significantly differ between the 50% and 80%

mapping conditions, $t(86) = .32, p = .75$, it was significantly larger in the 100% mapping condition in comparison to both the 50%, $t(86) = 8.70, p = .00$, and 80% mapping conditions $t(86) = 10.03, p = .00$.

As expected, there was also a main effect for Block Half, $F(1, 81) = 12.05, p = .00$, such that ERN amplitudes were larger in the second half of trials ($M = -4.02$) in comparison to the first half of trials ($M = -3.51$). Taken together with the accuracy results, this evidence suggests that by the second half of trials participants had learned response mappings in the 100% condition. As a result, they were able to infer from internal feedback that they had made an error instead of relying completely on external feedback.

There was also a Response Mapping x Half interaction, $F(2, 80) = 5.75, p = .00$, indicating ERN amplitude significantly increased across Block halves for the 100 % mapping condition only ($M_1 = -4.72; M_2 = -5.91; t(87) = 3.96, p = .00$). There was no difference in ERN amplitude as a function of Block Half for the 50%, ($M_1 = -2.95, M_2 = -2.97; t(87) = .08, p = .93$), and 80% ($M_1 = -2.86, M_2 = -3.16; t(87) = 1.33, p = .18$) response mapping conditions.

Although there was not an Emotion Regulation main effect, $F(1, 86) = .63, p = .43$, results did show a Beverage x Emotion Regulation x Block Half interaction, $F(2, 81) = 3.30, p = .04$ (see Figure 4). Follow-up 3 (Beverage) x 2 (Half) ANOVAs within each Block Half showed a significant Beverage x Half interaction for Reappraisers only, $F(2, 42) = 3.42, p = .04$ (Beverage x Half_{Sup} $F(2, 41) = 1.09, p = .35$). Inspection of the means associated with this effect was consistent with the accuracy results such that for Reappraisers, ERN amplitude was largest for the Control group and smallest for the Alcohol Group. Suppressors, however, showed no effect of beverage on their ERN

amplitude. While these means are in the predicted direction, follow-up comparisons of the simple effects were not significant, $t_s < 1$, $p_s > .20$.

fERN. Figure 5 (a and b) illustrates the stimulus-locked waveforms to feedback as a function of Beverage Group averaged across participants separately for Suppressors and Reappraisers for the 100% mapping condition at electrode FCz.

An initial analysis was conducted to determine the scalp distribution of the *fERN*. A 5 (Coronal; frontal, fronto-central, central, centro-parietal, parietal) x 3 (lateral; left, midline, right) repeated measures ANOVA showed a Coronal x Lateral interaction, $F(8, 166) = 2.15$, $p = .03$ indicating that the *fERN* was largest at fronto-central and central midline locations, especially FCz ($M = -6.71$). Thus, to simplify analyses only data from this location was included in the main analyses reported here.

Peak. Analyses revealed a main effect for Response Mapping, $F(2, 162) = 3.55$, $p = .03$, indicating that the *fERN* was significantly larger in the 100% Mapping condition ($M = .46$) in comparison to the 80% ($M = 1.52$) and the 50% mapping condition ($M = 1.45$). There was also a main effect of Block Half, $F(1, 81) = 12.24$, $p = .001$, revealing that the feedback ERN was largest in the second half of a block ($M = .50$) in comparison to the first half ($M = 1.79$). These main effects were qualified by a Response Mapping x Half interaction, $F(2, 162) = 6.32$, $p = .000$, indicating that these main effects were driven by *fERN* amplitude differences across Block halves for the 80% and 100% mapping conditions only. Specifically, *fERN* amplitudes were smaller in the first half of trials for the 80% ($M_1 = 2.11$ and $M_2 = .92$, $t(87) = 2.02$, $p = .03$) and 100% mapping conditions ($M_1 = 1.71$; $M_2 = -.79$; $t(162) = 5.41$, $p = .00$) Feedback ERN amplitude did not differ as a function of Block Half in the 50% condition ($M_1 = 1.53$ and $M_2 = 1.37$, $t(87) = .31$, $p =$

.76). Although this effect was in the opposite direction hypothesized (i.e., fERN amplitudes were expected to be largest in the first half of trials for the 80% and 100% mapping conditions), this could be due to overall task difficulty (see discussion).

Results also show a main effect of Emotion Regulation on fERN amplitude, $F(1, 81) = 3.81, p = .05$, such that Reappraisers exhibited a significantly larger fERN ($M = .13$) in comparison to Suppressors ($M = 2.16$). This effect was qualified by a Response Mapping x Block Half x Emotion Regulation interaction, $F(2, 162) = 3.41, p = .04$ (see Figure 6), indicating that the two-way interaction between Response Mapping and Emotion Regulation was driven by fERN amplitude differences in the second half of trials. That is, follow-up 3 (Response Mapping: 50%, 80%, 100%) x 2 (Emotion Regulation: Reappraiser vs. Suppressor) ANOVAs within each block half showed a significant Response Mapping x Emotion Regulation interaction for Block half 2 only $F(2, 85) = 3.81, p = .02$ (Half 1_{Bev} x EmReg: $F(2, 85) = .36, p = .70$). Inspection of the means indicate that Reappraisers showed a significant linear trend in fERN amplitude as a function of Response Mapping. Specifically, fERN amplitude was largest in the 100% condition and smallest in the 50% condition, $t(43) = 4.03, p = .00$. Feedback ERN amplitude did not, however, differ as a function of response mapping for Suppressors, $t_s < 1, p_s > .05$.

Ancillary Analyses

Although no effects were hypothesized for fERN latency, visual inspection of the fERN waveforms showed a prominent difference between emotion regulation groups. Thus, exploratory analyses were conducted to better understand the nature and significance of this trend. The basic design was a 2 (ER strategy: Reappraisal,

Suppression) x 3 (Beverage: control, placebo, alcohol) x 2 (Mapping; 100%, 80%, 50%) x 2 (Block half; trial 1-80, trial 80-160) mixed factorial with repeated measures on the final 2 factors. Primary analyses were carried out using mixed factorial ANOVA. Significant main effects of emotion regulation and beverage were followed-up using Helmert contrast analyses.

fERN Latency. Analyses of fERN latency showed a main effect of Response Mapping, $F(2, 85) = 3.28, p = .04$, indicating that the fERN was quicker to peak on 100% mapping trials ($M = 263\text{ ms}$; $t(86) = 5.66, p = .02$) in comparison to 50% ($M = 268\text{ ms}$) and 80% ($M = 269\text{ ms}$; $t(81) = 3.94, p = .05$) conditions. Additionally there was a main effect of Block Half, $F(1, 81) = 8.62, p = .004$, indicating that the fERN peaked more quickly in the second half of trials ($M = 264\text{ ms}$) in comparison to the first half of trials ($M = 270\text{ ms}$).

A main effect of Beverage also emerged, $F(2, 85) = 4.03, p = .02$, indicating that the fERN was significantly quicker to peak in the Alcohol group ($M = 259\text{ ms}$) in comparison to the Placebo ($M = 271\text{ ms}$; $t(59) = -1.61, p = .01$) and the Control group ($M = 270$; $t(63) = 1.50, p = .03$). This effect was qualified by an Emotion Regulation x Beverage interaction, $F(2, 81) = 3.04, p = .05$ (see Figure 7). Follow-up contrasts of this interaction revealed that the fERN was quicker to peak for Suppressors who consumed alcohol in comparison to the control beverage, $t(32) = 3.29, p = .002$, and the placebo beverage, $t(29) = 2.11, p = .04$. No such beverage group effects were apparent for Reappraisers, $F_s < 1, p_s > .10$.

Chapter 4

Discussion

The goal of the current study was to investigate the effects of alcohol and individual differences in emotion regulation strategies on performance monitoring, focusing on both the underlying neural correlates of this process and overt behavioral performance. More specifically, I was interested in determining whether alcohol differentially affects performance for individuals who tend to use different emotion regulation strategies. Previous research has found that individuals who experience greater affect regulatory benefit and report experiencing fewer cognitive costs from alcohol use are at increased risk for developing alcohol use problems (Cooper et al., 1995; Christiansen & Goldman, 1983; Fromme & D'Amico, 2000). Given that individuals who tend to use suppression have more difficulty regulating their affective experience (Gross & John, 2003), I hypothesized that after alcohol consumption, they would experience less cognitive cost due to the affect regulatory perks associated with intoxication that are key to implementing cognitive control effectively.

In the current study I tested this proposal by measuring behavioral and neural correlates of the performance monitoring process while participants engaged in a trial and error learning task after consuming a control, placebo, or alcohol beverage. Results show that performance among Reappraisers, or individuals utilizing what is thought to be a more effective emotion regulation strategy, was significantly impaired by alcohol,

relative to the other beverages. Interestingly, Reappraisers also showed a significant increase in performance in the Placebo group in comparison to the control and alcohol group. Taken together with the alcohol-induced impairment Reappraisers experience, this effect may be due to engagement of greater cognitive resources to assist them with their anticipated impairment.

In addition to these behavioral indicators of performance impairment, analyses of the ERN component, an ERP marker of evaluative aspects of performance monitoring (Yeung, Botvinick, & Cohen, 2004), corroborated the pattern of impairment seen in performance. Specifically, ERN amplitude was attenuated for the alcohol condition in comparison to the placebo and control groups, but only among Reappraisers. This finding suggests that after consuming alcohol, Reappraisers experience robust cognitive impairment.

In contrast to Reappraisers, Suppressors showed a striking resiliency to the effects of alcohol. Specifically, neither their neural performance monitoring processes nor their behavioral performance were affected by consuming alcohol relative to either placebo or control beverages. However, at least with respect to their ERN data, it appears that Suppressors experience somewhat blunted performance monitoring overall relative to Reappraisers, in that their ERN amplitudes were less pronounced regardless of the beverage they consumed. In other words, it is not the case that their neural error monitoring was at the same level following alcohol consumption as the Reappraisers' was following control or placebo consumption. It could be, then, that their evaluative control process is already impaired at baseline, such that a dose of alcohol does not further impair it. Interestingly, Suppressors also did not show typical placebo enhancing

effects on their performance, possibly due to their suppression of internal negative feedback following control failures/errors. An alternative possibility is that Suppressors may not have the same expectation regarding cognitive impairment from alcohol as most people (e.g., Reappraisers) and, as a result, are not motivated to try to compensate for such anticipated impairment upon consuming alcohol.

Although the pattern of behavioral and ERN results were generally as predicted, feedback ERN results were less straightforward. Inconsistent with past research (Holroyd & Coles, 2002), fERN amplitude increased as blocks progressed. Although counterintuitive, this could be a result of overall task difficulty. Specifically, given that participants had difficulty learning response mappings for the 80% mapping condition, substantial learning only occurred on 1/3 of trials. Given this level of difficulty, participants may never have relied entirely on their own internal feedback and, as trials progressed, they became increasingly distressed upon making errors likely because they felt like they should have figured out response mappings by that time.

Interestingly, this pattern held for Reappraisers only. Consistent with the effect of beverage on ERN amplitude, Suppressors showed no effect of response mapping on their fERN amplitude. One interpretation of this finding is that Suppressors are “inhibiting” the distress experienced by negative feedback. Similarly, an alternative interpretation of the lack of effect of alcohol on performance or ERN amplitude, may also indicate that Suppressors “brace” themselves for the distress experienced by errors, minimizing the negative affect they experience. If this is indeed what is happening, this may reflect that rather than regulating emotional experiences more generally, Suppression is a strategy best suited for regulating distress or preventing external factors from influencing internal

thought processes. Although an intriguing hypothesis, more research directly testing this idea need be done before conclusions can be made.

Finally, ancillary analyses indicated an Emotion Regulation x Beverage interaction with regard to fERN latency. Although this was not predicted, this effect may shed light on how Suppressors are able to maintain performance levels after intoxication, while Reappraisers are not. Specifically, the latency shift of fERN amplitude seen in Suppressors after alcohol consumption suggests that alcohol eases the extent to which external feedback is processed. If this is the case, this effect may indicate that these two emotion regulation strategies engage different processes to extract information from external error detection. Specifically, while Reappraisers rely on overall sensitivity to external feedback, a method that may be more vulnerable to the cognitive impairments induced by alcohol consumption, Suppressors rely on a method that alters the processing speed of feedback, a method that may be less sensitive to the negative effects of alcohol.

Future Directions

Given that alcohol differentially affects cognitive processing for Reappraisers and Suppressors, this may have implications for alcohol seeking behavior. Specifically, given Reappraisers experience heightened cost associated with drinking, which potentially decreases the benefits associated with drinking and, in turn, discourage this group from consuming alcohol. As Reappraisal tends to be more effective at regulating emotions than suppression, and these individuals experience greater cost associated with alcohol consumption, individuals utilizing this emotion regulation strategy may be especially resilient to developing alcohol use problems. By comparison, individuals utilizing suppression as their primary emotion regulation strategy may not be as fortunate.

Given that Suppressors are thought to use a less effective emotion regulation strategy, in that suppression is not as effective as reappraisal at altering emotional experience, is linked to physiological markers of enhanced stress, results in cognitive impairment in the context of emotional information, and experience more negative affect overall, these individuals may be at greater risk for seeking out alcohol. Coupled with the current study's finding that under the influence, Suppressors do not experience significant cognitive impairment, they may be particularly at risk for developing alcohol use disorders because they may utilize alcohol to help aid their emotion regulation process, and in doing so, experience fewer costs.

Taken together, these results are the first to suggest that alcohol differentially affects individuals utilizing differing emotion regulation strategies. Given that Reappraisers utilize a more effective emotion regulation strategy and experience significant impairment following alcohol consumption, individuals that rely primarily on this emotion regulation strategy may be particularly averse to consuming alcohol. However, given Suppressors' emotion regulation strategy may not meet all their regulatory needs and past research has shown that individuals use alcohol as a tool to help aid regulate their emotional experience, this alone may prompt Suppressors to seek out alcohol more often than individuals utilizing alternative emotion regulation strategies. This coupled with the current finding that performance is not significantly impaired by alcohol consumption for Suppressors, may put them at particular risk for developing substance abuse problems as they are likely to benefit the most from the reinforcing properties of alcohol without as much cost.

APPENDIX 1

Feedback ERN Subtraction Analysis. Analysis revealed a main effect of Beverage, $F(2, 81) = 4.09, p = .02$, indicating that the Placebo group exhibited a significantly greater difference between the fERN and the fCRN ($M = -8.12$) in comparison to both the Control (-6.07) and Alcohol group (-6.25). There was also a main effect of Block Half, $F(1, 81) = 5.64, p = .02$, such that fERN subtraction amplitudes were greatest in the second half ($M = -7.33$) of the block in comparison to the first half ($M = -6.28$).

Additionally, there was a Response Mapping x Emotion Regulation interaction, $F(2, 85) = 3.23, p = .03$. Follow-ups of this interaction revealed that Suppressors exhibited a larger difference wave for the 100% Mapping condition than Reappraisers. Additionally, while reappraisers showed no difference in the amplitude of their difference wave as a result of response mapping, $t_s < 1, p > .2$, Suppressors showed significantly greater differences in subtraction waveform amplitudes for the 80%, $t(42) = 2.79, p = .007$, and 50%, $t(43) = 2.39, p = .02$, mapping condition in comparison to the 100% mapping condition.

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Figure 1

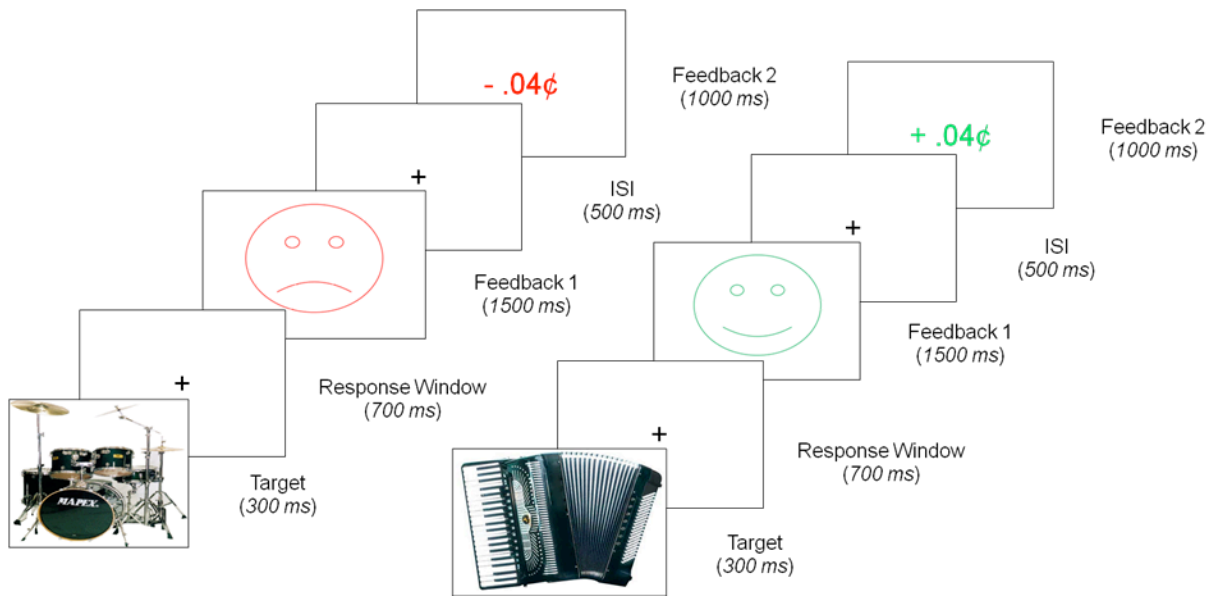


Figure 2.

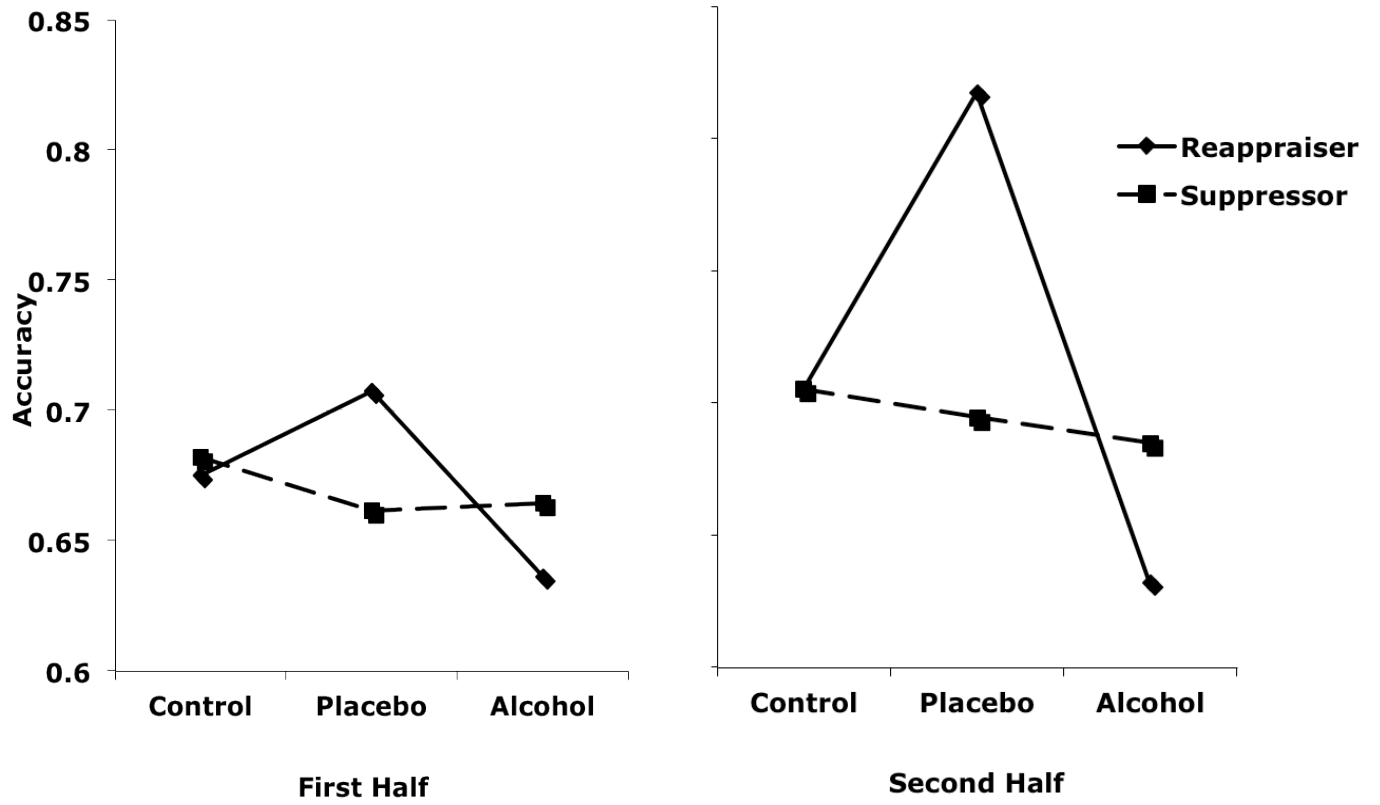


Figure 3.

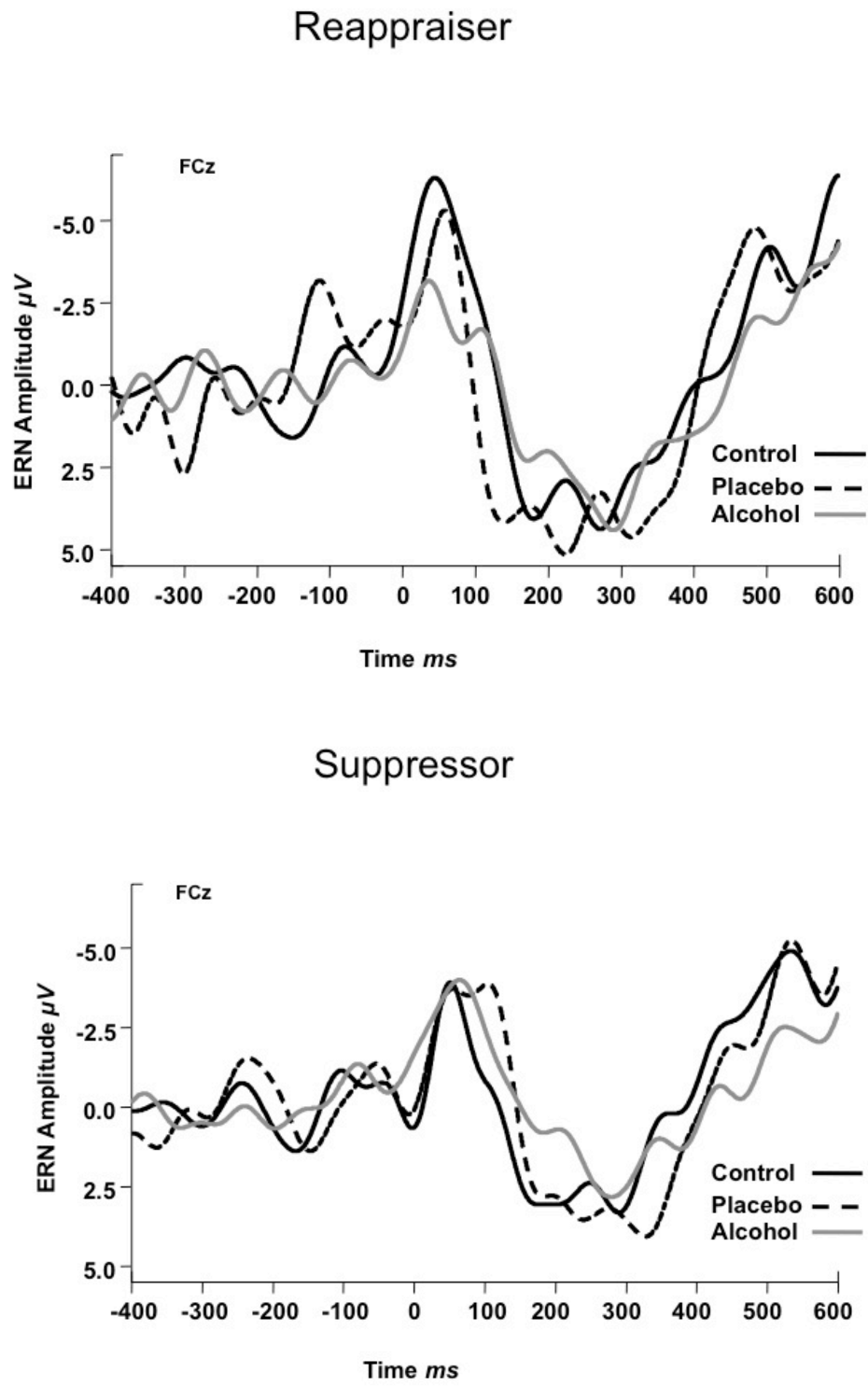


Figure 4.

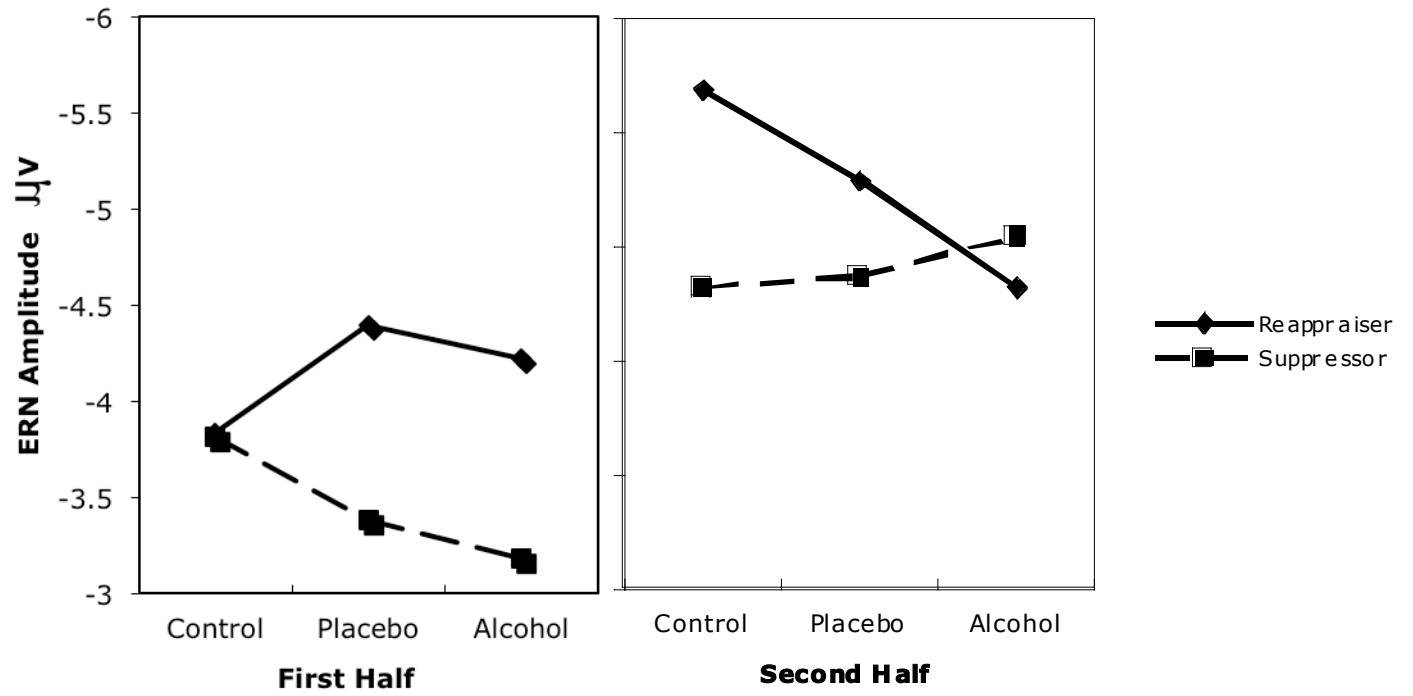


Figure 5.

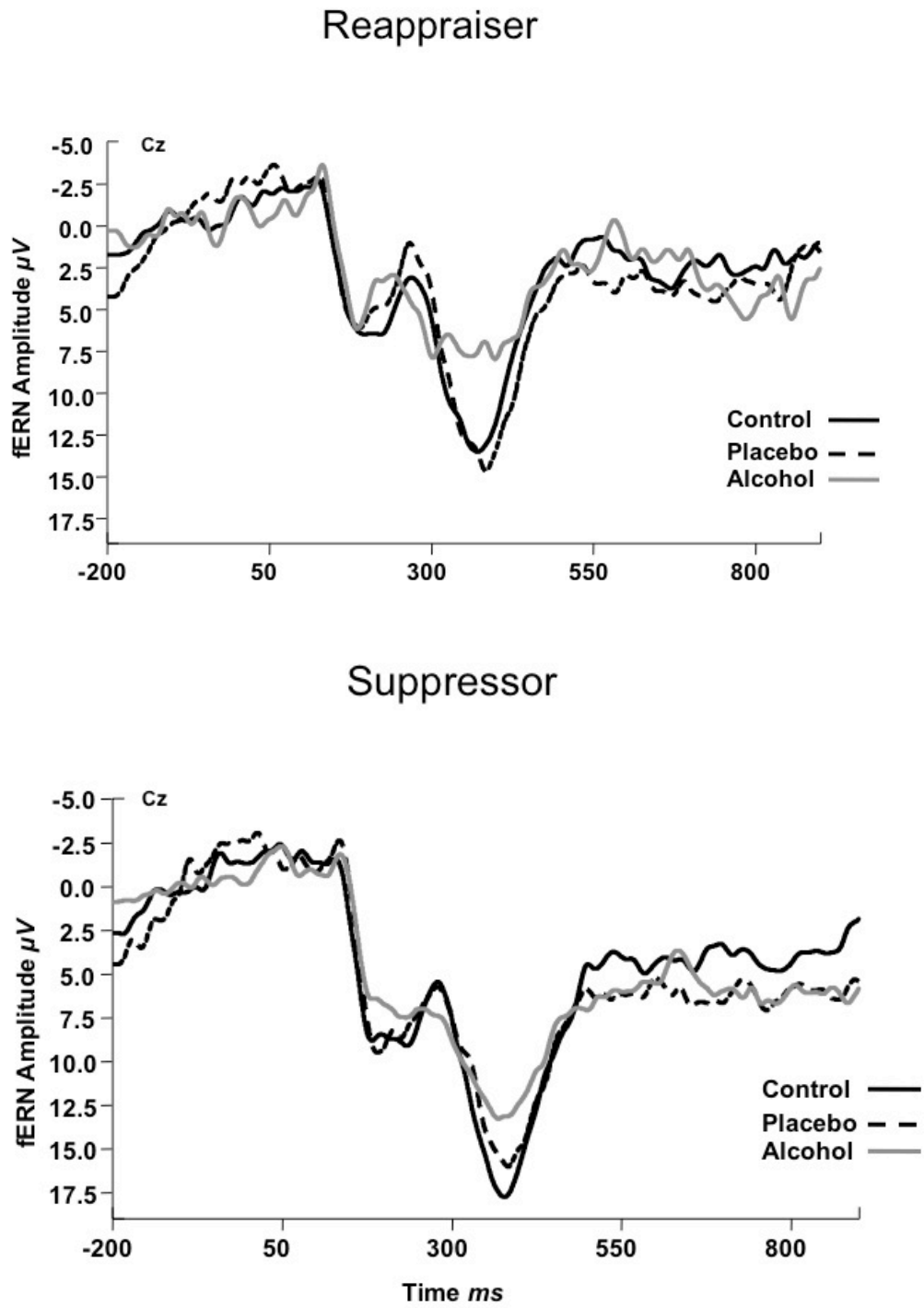


Figure 6.

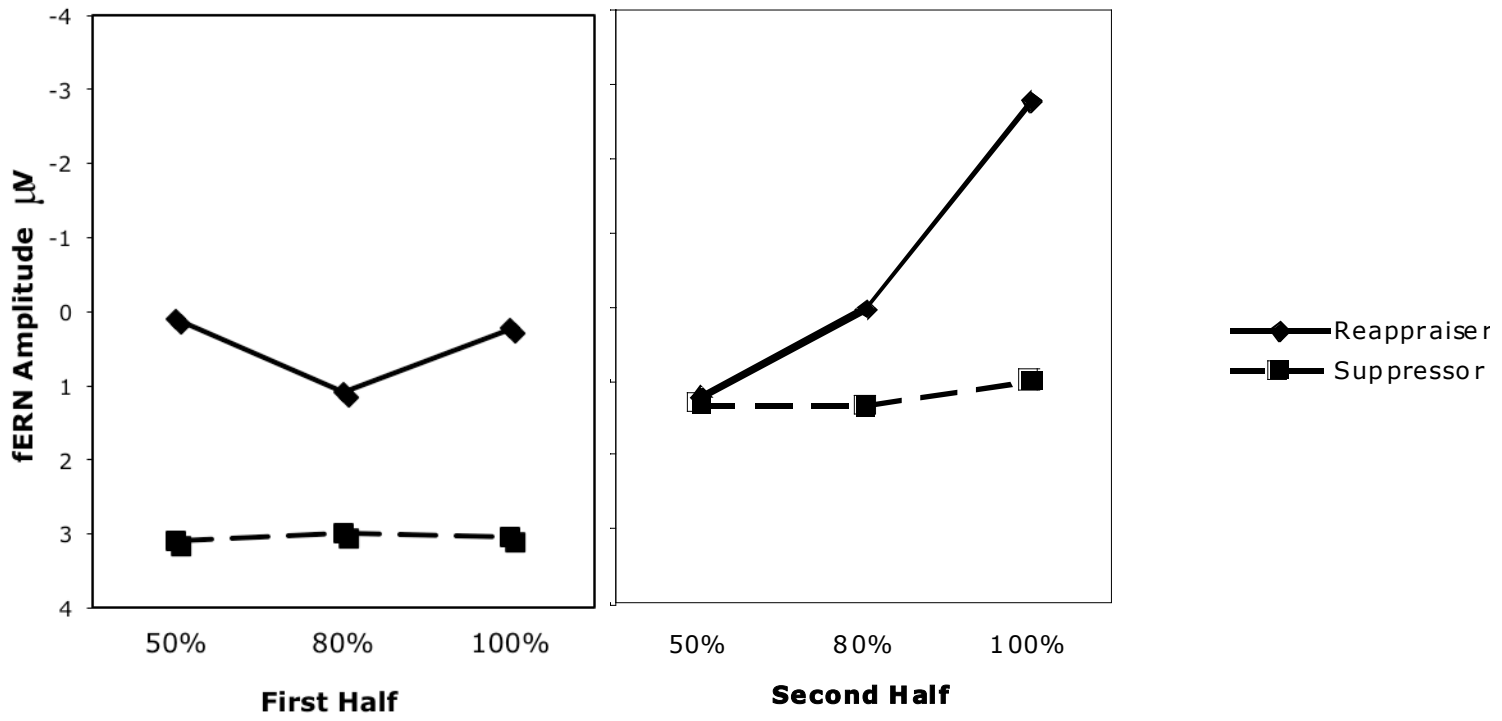
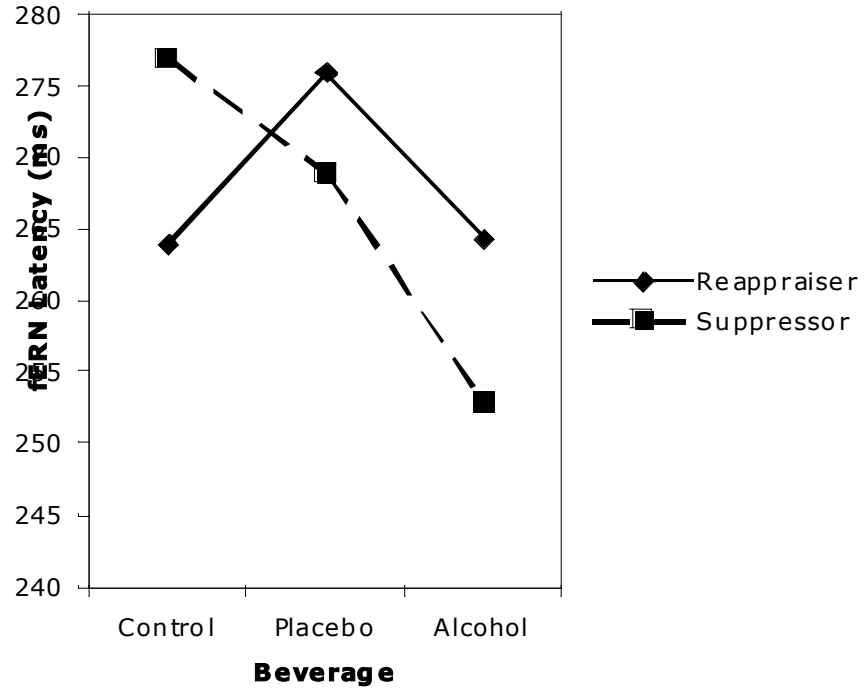


Figure 7.



VITA

Erika Henry was born in New York, however is by no means a New Yorker (although she has nothing against New Yorkers). Beginning when she was one year old, her family began bouncing around the country trying on a variety of locations as their home, none of which stuck for very long. Amidst her family's quest to find the perfect home, they often found themselves returning to Texas. Although Erika calls Texas home, she by no means considers herself a Texan, either (although, again she has nothing against most Texans). Amidst her academic pursuits she has attempted to follow Mr. Twain's advice, not allowing her schooling to get in the way of her education, by picking up a variety of hobbies and activities to keep her view of human behavior fresh and her experience with emotion up to date. Erika currently resides nestled up against the Rocky Mountains in Denver, Colorado with her three ferocious dogs. She continues to (attempt to) unravel the mysteries behind human emotion and self-regulation and, when fresh out of ideas, Erika turns to writing Zombie novels as a means of repletion.