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FEEDBACK-RELATED NEGATIVITY, DECISION-MAKING, AND COLLEGE BINGE DRINKING

by

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FEEDBACK-RELATED NEGATIVITY, DECISION-MAKING, AND COLLEGE BINGE DRINKING

Laura C. Wahlstrom, Ph.D.

University of Nebraska, 2013

Adviser: Dennis McChargue

Despite increased prevention efforts, binge drinking remains a prevalent issue among college students. Myriad negative consequences are associated with binge drinking (e.g., academic impairment, injury, legal trouble); however, it appears a subset of individuals do not alter future drinking to reduce their risk. This suggests that some binge drinkers are less sensitive to alcohol-related negative outcomes, which may stem from individual differences in neurocognitive functioning. The present study draws from a reinforcement learning theory framework to explain disparities in learning from negative outcomes. Specifically, an electrophysiological measure associated with diminished responsiveness to negative consequences (i.e., feedback-related negativity; FRN) and a decision-making task (Iowa Gambling Task; IGT) were used to index reinforcement learning. The study enrolled thirty college students age 18 to 23 across three categories of drinkers (non-binge, binge, and heavy drinkers) to test study aims. The first aim was to examine FRN differences among non-binge, binge, and heavy drinkers. A dose-response effect was expected with heavy drinkers showing the smallest FRN amplitude (an index of diminished response to consequences) and non-binge drinkers showing the largest FRN amplitude. The second aim was to examine the relationship between FRN and decision-making on the IGT among college binge drinkers. It was hypothesized that there would be a positive linear relationship between FRN amplitude and number of

advantageous decisions on the IGT. The third aim was to examine the relationship between FRN and age of drinking onset, and it was hypothesized that there would be a positive linear relationship between FRN amplitude and age of drinking onset. Study methodology, results, and implications are discussed.

Dedication

To my loving husband, Dustin Wahlstrom, and family, Ray Herschl, Nancy Herschl, and Kimberly Timm. Thank you for all your support and patience throughout the past five years.

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Chapter 1: Introduction

Despite an increase in prevention efforts (Wechsler, Lee, Kuo, Seibring, Nelson, & Lee, 2002), binge drinking remains a prevalent public health issue among college students (Grucza, Norberg, & Bierut, 2009). Myriad negative consequences are associated with binge drinking, including academic impairment, injuries, legal trouble, fighting, blackouts, and sexual assault (Perkins, 2002). Furthermore, college binge drinking accounts for approximately 500,000 injuries and 1,700 deaths each year (Hingson, Heeren, Winter, & Wechsler, 2005). For college students who experience non-lethal negative consequences, a subset do not appear to alter their drinking patterns to reduce their risk of future negative outcomes (Gaher & Simons, 2007; Mallett, Lee, Neighbors, Larimer, & Turrisi, 2006). This suggests that some binge drinkers, presumably those with the heaviest drinking patterns, are less sensitive to alcohol-related negative outcomes. A reduced sensitivity may stem, in part, from disparities in neurocognitive mechanisms associated with the processing of negative outcomes, including brain electrophysiology, prefrontal cortex development, and the dopamine system (Casey, Giedd, & Thomas, 2000; Crone & van der Molen, 2004; Frank & Claus, 2006), and may impact future alcohol-related decision-making (Goudriaan, Grekin, & Sher, 2007). As such, the overarching aim of the present study is to examine neurocognitive mechanisms associated with differential sensitivity to negative outcomes among college students with a wide array of drinking patterns.

Event-related potentials (ERPs) are an optimal index of neurocognitive responses to negative outcomes. ERPs are components extracted from the ongoing electroencephalogram (electrical signals measured through the scalp) that are associated

with specific cognitive, sensory, or motor activities (Luck, 2005). The error- and feedback-related negativities (ERN/FRN) are specific ERP components that reflect neural signals part of the human error-processing system (Gehring, Goss, Coles, & Meyer, 1993). The ERN/FRN occurs when an outcome is worse than expected and is thought to reflect phasic (i.e., rapid and fleeting) dopaminergic activity. When an outcome is worse than expected, there is a dip in midbrain dopamine that signals frontal brain areas (Holroyd & Coles, 2002). This results in the reinforcement of advantageous behaviors and extinction of disadvantageous behaviors. Larger ERN/FRN responses are associated with better learning to avoid negative outcomes (Frank, Woroch, & Curran, 2005). The ERN and FRN are ideal ERP components for examining negative outcome processing among college binge drinkers because that they are: (a) negatively affected by alcohol use (e.g., Ridderinkhof et al., 2002) and (b) integrally involved in reinforcement learning (Holroyd & Coles, 2002).

Reinforcement learning (RL) theory provides a framework in which the interplay among the ERN/FRN, dopamine system, and frontal brain areas explain decision-making and learning. According to RL, the goal of an organism is to maximize rewards and minimize punishments (Sutton & Barto, 1998). However, substance use disrupts this process by increasing phasic dopamine in the midbrain, which results in the brain interpreting all decisional outcomes made during intoxication as advantageous (Redish, 2004). This disruption increases the value of a drug each time it is used and, over time, results in an overvaluation of a drug's reward and devaluation of non-drug, natural rewards (Redish, 2004). Furthermore, this presumably leads to the discounting of negative consequences associated with drug use. Because all decisions are interpreted by

the brain as advantageous, no ERN/FRN is elicited when negative outcomes are experienced (Holroyd & Yeung, 2004). As a result, future drinking behaviors may not be adapted in response to negative outcomes due to the dysfunctional neural error-processing system. ERN/FRN functionality, specifically from a RL perspective, has yet to be examined among college binge drinkers.

In order to assess responses to error, the present study utilized the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994). The IGT is a widely used measure of decision-making and requires participants to maximize rewards and minimize punishments, making it an ideal index of RL. The IGT requires participants to make 100 selections from four decks of cards. Two decks are considered advantageous because they yield small gains on each selection but result in long-term gains; conversely, the two disadvantageous decks yield large gains on each selection but result in long-term losses. Healthy controls tend to sample from all decks during the beginning of the task and then shift their selections to the advantageous decks as the task progresses. However, findings indicate impaired IGT performance across a wide array of substance users, including alcohol-dependent individuals (e.g., Bechara, Dolan, Denburg, Hindes, Anderson, & Nathan, 2001). Poor performance on the IGT among substance users is thought to reflect a preference for immediate rather than delayed reward, hypersensitivity to reward in general, and insensitivity to negative consequences (Bechara et al., 2001).

The present study aimed to advance knowledge of neurocognitive mechanisms that implicate a reduced sensitivity to negative consequences among binge drinkers by testing for differences in FRN response among varying degrees of drinkers, including non-binge drinkers, binge drinkers, and heavy drinkers. The groundwork for the study

aims is laid through a literature review of the primary areas of research that comprise the application the RL theory of the FRN to college binge drinkers. This consists of a review of the binge drinking literature, including the definition of binge drinking, binge drinking prevalence among college students, alcohol-related negative consequences, and neurological development and decision-making during college. This is followed by an introduction to event-related potentials (ERP), and includes a description of what ERPs are, commonly examined ERPs and their associations with alcohol, a review of the ERP and binge drinking literature, a description of the ERPs of interest (the error- and feedback-related negativities), and a review of the literature on ERN/FRN and alcohol use. The following section details reinforcement learning (RL) theory, including its origins in computer science, the role of dopamine in RL, the ERN/FRN theory of RL, the RL theory of addiction, and the interplay among RL, the ERN/FRN, and alcohol. Lastly, the Iowa Gambling Task (IGT) is discussed. This section covers a description of the task, theories of IGT performance, the association between the ventromedial prefrontal cortex and the IGT, IGT performance in substance users, and the IGT and binge drinking literature.

Binge Drinking

Definition

Despite significant advances in research, binge drinking is a greater public health issue than it was for our predecessors (Wechsler et al., 2002). Binge drinking was originally defined as consuming five or more drinks during the same session (Cahalan, Cisin, & Crossley, 1969). Wechsler and colleagues (1994) of the College Alcohol Study altered this definition to account for differences in the metabolism of alcohol between

men and women. As such, the number of drinks necessary to meet criteria for binge drinking was changed to four for women and remained at five for men. However, both the original and modified definitions are somewhat arbitrary, as they do no specify how long a single episode is nor define a standard drink. Contemporary definitions of binge drinking now center on blood alcohol content (BAC) (NIAAA, 2004; Lange & Voas, 2000). Specifically, the National Institute on Alcohol Abuse and Alcoholism (NIAAA) has defined binge drinking as a pattern of alcohol consumption that rapidly raises the BAC to .08 or above. This typically corresponds to five or more drinks for men and four or more drinks for women within a two-hour period of time (NIAAA, 2004).

The change in the definition of "binge" was implemented due to gender differences in alcohol-related risk, stature, and physiological differences in the absorption and distribution of alcohol (Baraona et al., 2001; Wechsler & Austin, 1998; Wechsler et al., 1995; Wechsler & Nelson, 2001). This new definition has proven to be more sensitive to hazardous drinking among women, as the binge drinking prevalence increased 2.6 percentage points using the new definition (Chavez, Nelson, Naimi, & Brewer, 2011). Furthermore, NIAAA (2004) defined standard drinks, which consist of twelve ounces of beer, five ounces of wine, or 1.5 ounces of spirits. These amounts are all equivalent in terms of pure alcohol. Defining a standard drinking has proven important, as college students tend to consume single servings of beer and liquor that are significantly larger than these standards, which leads to inaccuracies in self-reported consumption (White et al., 2005).

The time frame during which drinking need occur to be considered binging has also varied across studies and agencies, ranging from during the past week (Kokavec &

Crowe, 1999) to the past six months (Hartley, Elsabagh, & File, 2004). These various time frames are thought to reflect different aspects of binge drinking patterns given that college students' binge drinking tends to be inconsistent across time (Schulenberg, O'Malley, Bachman, Wadsworth, & Johnson, 1996). However, some data suggest that the most informative time frame to assess binge drinking is during the past six months (Townshend & Duka, 2005; Weingardt, Baer, Kivlahan, Roberts, Miller, & Marlatt, 1998). The Substance Abuse and Mental Health Service Administration (SAMHSA) uses the past thirty days to assess binge drinking (SAMHSA, 2006). Additionally, SAMHSA has differentiated binge drinking from heavy drinking, whereby heavy drinking reflects five or more drinks on the same occasion on five or more days during the past thirty days (SAMHSA, 2006).

Prevalence

Results indicate that among persons aged eighteen to twenty-two, binge drinking rates are significantly higher in college students (Brower, 2002; O'Malley & Johnston, 2002; Slutske, 2005). The 2005 National Survey on Drug Use and Health (SAMHSA, 2006) found that past-month alcohol use was reported by 64.4% of full-time college students compared with 53.2% of persons aged eighteen to twenty-two who were not enrolled in college full-time. Furthermore, forty-four percent of full-time college students also reported binge drinking and 19.5% reporting heavy drinking; whereas comparative eighteen to twenty-two year olds not enrolled in college full-time reported lower rates of binge (38.3%) and heavy drinking (13%). Other data indicate that a surprisingly large proportion of college freshman are drinking at levels two or three-times the binge threshold. White and colleagues (2006) examined alcohol consumption during a two-

week period in 10,424 freshmen at fourteen colleges and universities across the United States. They found that roughly one in five men consumed ten or more drinks during a drinking episode and one in ten women consumed eight or more drinks during a drinking episode.

A body of literature has found that college men engage in binge drinking at higher rates than college women (O'Malley & Johnston, 2002; Wechsler, Dowdall, Maenner, Gledhill-Hoyt, & Lee, 2000; Wechsler et al., 1994; Wechsler, Dowdall, Davenport, & Castillo, 1995; Weitzman, Nelson, & Wechsler, 2003). Men are also more likely to drink at levels far beyond the binge threshold (White, Kraus, & Swartzwelder, 2006), experience alcohol-related negative consequences (Berkowitz & Perkins, 1987a), and to meet criteria for an alcohol use disorder (Clements, 1999; Hill & Chow, 2002) than women. Among some college men, infrequent binge drinking during college leads to heavier drinking after college (Jennison, 2004), which suggests that men may be more vulnerable to not maturing out of binge drinking regardless of how frequently they binge. However, trends indicate that binge drinking among college women is increasing (Mercer & Khavari, 1990; Wechsler et al., 2002).

The College Alcohol Study found that frequent binge drinking among women increased from 17.1% to 20.9% between 1993 and 2001. They also found increases in the number of college women who drank on ten or more occasions during the past thirty days and drinking with the intention of getting drunk (Wechsler et al., 2002). It has been theorized that this convergence of drinking patterns between men and women stems from the notion that "drinking like a guy" is something that college men find attractive in women and symbolizes gender equality. College women who were frequent binge

drinkers throughout college felt that "drinking like a guy" applied to their own drinking behavior (Young, Morales, McCabe, Boyd, & D'Arcy, 2005). Consuming larger quantities of alcohol is a greater risk for college women given that women reach the same blood alcohol level as men after consuming less alcohol due to differences in weight, fat-to-water ratios, and metabolic processing (Perkins, 2002).

Across college men and women, alcohol use greatly increases during the transition from high school to college (Berkowitz & Perkins, 1987b) and continues to increase until age twenty or twenty-one (Johnston, O'Malley, & Bachman, 1998). After age twenty-one, college students' drinking tends to decrease (Johnston et al., 1998; Muthén & Muthén, 2000), which is referred to as the "maturing out" process (Fillmore, 1988; Johnstone, Leino, Ager, Ferrer, & Fillmore, 1996). This process presumably stems from the acquisition of adult roles and responsibilities (Bachman, Schulenberg, Johnston, Bryant, Merline, & O'Malley, 2002), which typically include the end of formal education, full-time employment, financial independence, novel living arrangements, marriage, and children (O'Malley, 2005). However, not all individuals mature out of their college drinking patterns. One factor that has been predictive of maintaining hazardous patterns of alcohol consumption is familial alcoholism (Jackson, Sher, Gotham, & Wood, 2001).

Alcohol abuse and dependence have also been examined within the context of college binge drinking. Clements (1999) found that in a sample of 306 undergraduates, 13% met criteria for alcohol abuse and 11% met criteria for dependence. These rates differed between men and women, with approximately 18% of men meeting criteria for each abuse and dependence, whereas 11% of women met criteria for abuse and 9% for

dependence. Other data from a much larger sample revealed 31% of college students meet criteria for abuse and 9% for dependence (Knight, Wechsler, Kuo, Seibring, Weitzman, & Schuckit, 2002). Across studies, it has been concluded that individuals who binge more frequently and consume larger quantities are at greater risk for meeting criteria for abuse or dependence (Clements, 1999; Knight et al., 2002).

Alcohol-Related Negative Consequences

Myriad negative consequences have been associated with binge drinking among college students (Perkins, 2002). Academic impairment related to binge drinking has been reported by a large number of college students, including poor test performance, missing class, and lower grade point average (Engs, Hanson, & Diebold, 1996; Perkins, 1992; Presley, Meilman, & Cashin, 1996; Wechsler, Dowdall, Maenner, Gledill-Hoyt, & Lee, 1998). Blackouts (i.e., impairment in short- and long-term memory during intoxication while consciousness is retained) have been reported by an upwards of 25% of students during the previous year (Presley et al., 1996; Wechsler et al., 1998).

Injuries, fighting/interpersonal violence, and legal troubles are frequently reported negative consequence of binge drinking (Perkins, 1992; Presley et al., 1996; Wechsler et al., 1994). Studies indicate that between 10% and 30% of college students have sustained an injury while under the influence of alcohol in the past year or gotten into a verbal/physical fight with someone (Perkins, 1992; Presley et al., 1996; Wechsler et al., 1994). Furthermore, individuals who begin drinking before age nineteen are at greater risk for alcohol-related injuries (Hingson, Edwards, Heeren, & Rosenbloom, 2009; Hingson, Heeren, Zakocs, Winter, & Wechsler, 2003). Legal problems, including underage consumption, open container violations, and public intoxication also result from

binge drinking and are experienced by 5% to 12% of students. Specifically, binge drinkers are at significantly greater risk for driving under the influence. Among light to moderate weekly drinkers, 17% of men and 10% of women reported having driven while intoxicated during the past year. These rates are markedly higher for heavy weekly drinkers, with 56% of men and 43% of women having reported drunk driving during the past year (Engs et al., 1996).

Sexual assault is a significant risk among college women. The Department of Justice has estimated that college women have between a 20-25% chance of being raped during college (Fisher, Cullen, & Turner, 2000) and binge drinking further exacerbates this risk (Abbey, 2002; Mohler-Kuo, Dowdall, Koss, & Wechsler, 2004; Parks & Fals-Stewart, 2004). For example, estimates indicate that in a college sample, 74% of perpetrators and 55% of victims of rape had been drinking alcohol at the time of sexual assault (Harrington & Leitenberg, 1994; Howard, Griffin, & Boekeloo, 2008; Mohler-Kuo et al., 2004; Koss, 1988). Consistent with these estimates, one study found that women were nine times more likely to be victims of sexual aggression on heavy drinking days and three times more likely on non-heavy days compared to non-drinking days (Parks & Fals-Stewart, 2004). This disparity in alcohol- and non-alcohol-related sexual victimization is theorized to stem from women's reduced awareness in risky situations, impaired judgment, and inability to resist assault (Larimer, Lydum, Anderson, & Turner, 1999). Moreover, there is a subset of college women who are high risk in terms of drinking and sexual victimization risk due to past sexual victimization. Binge drinking rates among these women tend to be higher than college women who have not been

victimized, which increases their risk for additional sexual assaults (Combs-Lane & Smith, 2002; McCauley, Calhoun, & Gidycz, 2010).

Consequences of binge drinking may also be lethal, including suicide and death (Hingson et al., 2005; Schaffer, Jeglic, & Stanley, 2008; Powell et al., 2001). Relationships have been found between alcohol use and suicide attempts among college students (Schaffer et al., 2008). Moreover, drinking within three hours of a suicide attempt strongly predicted nearly lethal suicide attempts (Powell et al., 2001). The College Alcohol Study found that between 1998 and 2001, deaths among college students ages eighteen to twenty four increased from 1,600 to 1,700 per year, an increase of 6%. The authors believe these estimates to be conservative, as they exclude suicide, homicide, and traffic-related deaths (Hingson et al., 2005).

Reinforcement learning theory would suggest that drinking behavior is shaped by the previously discussed negative outcomes (Sutton & Barto, 1998). For example, if an individual experienced a blackout and woke up in an unknown place with injuries, they may curtail their alcohol consumption to minimize future potential negative outcomes. However, data indicates that the negative consequences of drinking may have less of an impact of future drinking-related behavior than might be expected. Mallett and colleagues (2006) examined self-reported drinking behavior and resulting negative consequences among college students. Participants also estimated the number of drinks associated with experiencing future negative consequences. Results indicated that students grossly overestimated the number of drinks associated with negative outcomes when compared to their actual number of drinks consumed the last time they experienced the identical negative consequence. The number of drinks associated with actual negative outcomes

and the perceived number were more divergent among heavier drinkers. These findings reveal a disconnect between actual and perceived risk for alcohol-related negative consequences, suggesting that college students are either unaware or consciously discount the risk associated with their degree of consumption. However, a critical gap in the literature relates to the processes associated with how future behaviors are altered (or not altered) in response to negative outcomes.

Neurological Development and Decision Making During College

Though yet to be explicitly tested, persistent binge drinking despite negative consequences may stem, in part, from the fact that brain regions critical for complex cognitive functions have yet to fully mature. College, particularly the first two years, is a key developmental period both neurologically and behaviorally. Neurologically, the prefrontal cortex (PFC), an area of the brain responsible for higher-order cognitive functioning, is not fully developed until young adulthood (Casey et al., 2000; Gogtay et al., 2004; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). Furthermore, findings have specifically revealed improvements in myelination during the first year of college, which equates to improved information transfer throughout the brain (Bennett & Baird, 2006). Evidence also indicates changes in dopaminergic functioning during this period (Wahlstrom, White, & Luciana, 2010), which is integrally involved in the PFC's functionality, other executive functions, and the response to reward-related cues.

Behaviorally, decision-making disparities have been found between younger and older adolescents, suggesting that decision-making skills continue to develop into early adulthood (Hooper, Luciana, Conklin, & Yarger, 2004). These factors may contribute to harmful drinking, as this developmental period is associated with increased risk-taking

(Steinberg, 2007). The scope of this dissertation is not to disentangle whether binge drinking stems from a predisposition to reduced sensitivity to negative outcomes or binge drinking alters neurological and behavioral development. As will be discussed more thoroughly in the next section, the present study will be the first to examine the association between a neural electrophysiology in response to error and the degree to which these responses vary among college students with different patterns of alcohol use. To the extent that electrophysiological responses to error reflect similar processes associated with response to alcohol-related negative consequences, this is a critical first step in clarifying the underlying neural mechanisms associated with the processing of alcohol-related negative outcomes among college students who binge drink because differences have been identified in event-related potential components between binge and non-binge drinkers.

Event-Related Potentials

Event-related potentials (ERPs) are ideal for examining neural disparities in the processing of negative outcomes among college binge drinkers because ERPs provide valuable information related to concomitant behavioral performance and cerebral activity (Campanella, Noël, & Tomberg, 2010). That is, neural activity can be measured throughout performance of cognitive and behavioral tasks, which is important because it provides information that directly links behavior on these tasks to changes in brain activity. For example, specific ERP components reflect neural response to error/punishment (Gehring et al., 1993), are elicited during decision-making tasks, and predict individual differences in decision-making performance (Fein & Chang, 2007; Yu & Zhou, 2008). Further, ERPs are especially useful when examining cognitive and

behavioral correlates of alcohol use and misuse. A body of literature has identified cognitive deficits, including impairment indexed by ERPs, (Weiss & Marksteiner, 2007) among alcohol-dependent individuals (Fein, Bachman, Fisher, & Davenport, 1990). Altered ERPs have also been found after consumption of low doses of alcohol and persist even in the absence of task difficulties or behavioral impairment (Boha et al., 2009). Despite this evidence, few studies have examined ERPs as they relate to binge drinking. The aims of this section are to broadly describe what ERPs are and how they are measured, discuss commonly used ERP components and their associations with alcohol, review the literature on binge drinking and ERPs, describe the ERP component of interest (error- and feedback-related negativities; ERN/FRN), and discuss the relationship between ERN/FRN and alcohol use.

Description

Brain activity was originally measured using the electroencephalogram (EEG), which is the electrical activity that is measured when electrodes are placed on the scalp, the signal is amplified, and changes in voltage are plotted (Niedermeyer & Da Silva, 2005). However, this method is a global measure of brain activity and does not provide information related to specific neural processes. Event-related potentials (ERPs) are associated with specific cognitive, sensory, or motor activities and are extracted from the ongoing EEG (Luck, 2005). Specifically, ERPs detect excitatory and inhibitory postsynaptic potentials that occur primarily in the cerebral cortex and hippocampus (Allison, 1984; Allison, Woods, & McCarthy, 1986). The pyramidal cells that generate an ERP are aligned parallel to each other and perpendicular to the scalp, leading to a summation of currents in the same direction and toward the scalp surface (Allison, 1984).

These signals are a series of positive and negative deflections, which depend on whether the neuron is excited or inhibited (Nelson & Monk, 2001). For example, when a neuron is excited, the input leads to an inflow of positive ions into the cell, which results in an increase in negatively charged ions in the extracellular space (sink). The positively charged ions travel through the cell and back out into extracellular space (source), creating a dipole whose direction can be determined from scalp electrodes (Nelson & Monk, 2001). These ERP signals range between one and thirty millionths of a volt and last only milliseconds.

There are three components of the ERP, including amplitude, latency, and scalp distribution (Johnson, 1992). Amplitude is an index of the degree of neural activation, whereas latency is the timing of the activation. ERP nomenclature typically indicates the direction of the amplitude (e.g., p = positive, n = negative) and latency (e.g., 300 = 300 milliseconds after stimulus onset). Scalp distribution provides information as to which brain areas are activated and in what pattern during an activity. Different combinations of latencies and magnitudes reflect different cognitive processes (e.g., working memory, processing of novel stimuli, visual processing, auditory processing, attention, error processing).

Event-Related Potential Components: Descriptions and Alcohol

P300. Perhaps the most widely studied ERP component is the P300 (also referred to as P3), a positive waveform with latency between 300 and 500 milliseconds. The P300 is elicited in the parietal lobe and is thought to be involved in working memory (i.e., context updating, working memory revisions) (Nelson & Monk, 2001). P300 amplitude is related to task difficulty and latency is influenced by stimulus evaluation and task

complexity (Capanella et al., 2010). Since its discovery, the P300 has been separated into two distinct P3 components, the P3a and P3b. The P3b reflects the traditional P300 wave previously discussed. The P3a occurs sixty to eighty milliseconds before the P3b and is more frontally localized (Squires, Squires, & Hillyard, 1975). Furthermore, the P3a is thought to reflect the processing of novel stimuli, whereas the P3b is elicited during the revision and updating of working memory (Polich & Criado, 2006). Reduced P300 amplitude and delayed latency have been found among alcohol-dependent individual (e.g., Porjesz & Begleiter, 2003) and in the offspring of alcoholic fathers (Hill, Yuan, & Locke, 1999), which map on to cognitive dysfunction and slowed cerebral activity (Picton, 1992). Unlike other ERP components, the P300 does not appear to recover with abstinence from alcohol (Fein & Chang, 2006).

P100. The P100 (also referred to as P1) is a positive waveform that occurs between 80 and 120 milliseconds in the extrastriate cortex and is involved in the processing of visual stimuli and attention (Spehlmann, 1965). One of the earliest methods used to elicit the P100 was flashing geometric objects or shapes (e.g., a checkerboard) very briefly (Spehlmann, 1965). More recently, P100 research has focused on its association with attention, with tasks requiring participants to focus on a target while attending to their entire (or at least part of) visual field to identify stimuli (Van Voorhis & Hillyard, 1977). Some theories suggest that the P100 reflects a "cost of attention" (Luck, Hillyard, Mouloua, Woldorff, Clark, & Hawkins, 1994). That is, if a participant is attending to an area and the target stimulus is presented elsewhere, there is a "cost" of this incorrect attention, which is reflected in reduced P100 amplitude.

Regarding alcohol's effects on the P100, evidence indicates that moderate and high doses of alcohol reduce P100 amplitudes, suggesting sensory-perceptual impairment (Krull, Smith, & Parsons, 1994). Furthermore, the P100 has been implicated in a network of deficient ERP waveforms among alcohol-dependent individuals. Maurage and colleagues (2007) examined P100, N170 (a negative waveform associated with face-processing), and P300 amplitude and latency values among alcohol-dependent individuals and healthy controls. They found reduced latency for P100, N170, and P3b components as well as reduced amplitude for N170 and P3b components among the alcohol-dependent individuals compared to the healthy controls. Furthermore, there were positive correlations among all three components for the alcohol-dependent group. The authors suggest that cognitive impairments found among those with alcohol dependence are exhibited early on in cognitive processing, which may have residual deleterious effects on the P300.

Mismatch Negativity/N200. The mismatch negativity (MMN) is a front-central negative ERP component that is elicited 150 to 250 milliseconds after an odd stimulus is presented in a series of stimuli (e.g., s s s s s s s s s s d s s s) (Näätänen, Gaillard, & Mantysalo, 1978). The MMN is calculated by subtracting the negative deflection during frequent stimuli from the negative deflection during infrequent stimuli. The MMN is elicited both during visual and auditory tasks, thus reflecting either language processing (auditory) (Näätänen, Paavilainen, Rinne, & Alho, 2007) or attentive information processing (visual) (Titinen, May Reinikainen, & Näätänen, 1994). During auditory tasks, the MMN is elicited both during times of attention and inattention (Näätänen et al., 1978). Also falling into this category is the N200 (Sutton, Braren, Zubin, & John, 1965).

Unlike the MMN, which is present during periods of attention and inattention, the N200 requires attention to the stimulus and is thought to map on to response inhibition, response conflict, and error monitoring (Patel & Azzam, 2005).

The evidence related to MMN and alcohol is mixed, with some findings showing enhanced MMN among detoxified alcohol-dependent individuals (Pekkonen, Ahveninen, Jääskeläinen, Seppä, Näätänen, & Sillanaukee, 1998) and among children at high risk for alcohol dependence (Zhang, Cohen, Porjesz, & Begleiter, 2001). However, it has been postulated that enhanced MMN is due to post-withdrawal brain hyperexcitability (Ahveninen, Escera, Polo, Grau, & Jääskeläinen, 2000) and inhibition deficits (Zhang et al., 2001). On the other hand, some evidence indicates delayed MMN latency and reduction in amplitude (upwards of 60%) after an acute low dose of ethanol during an auditory task that required participants to attend to auditory stimuli in one ear and ignore stimuli in the other ear (Jääskeläinen et al., 1995). This finding has been interpreted as disruptions in stimuli processing outside of the scope of attention.

N400 and N450. The N400 is a negative deflection that occurs between 250 and 500 milliseconds after stimulus onset in the centro-parietal brain regions and reflects processing of words, pictures, faces, sounds, and smells (Kutas & Federmeier, 2000; Kutas & Hillyard, 1980). This is typically measured using linguistic tasks, where sentences end with either semantically appropriate (e.g., “I put butter on my toast”) or inappropriate (e.g., “I put butter on my sock”) words. Inappropriate words yield an augmented N400 (Kutas & Hillyard, 1980). This response is thought to reflect the integration of current stimuli into the mental context of the situation (Halgren & Smith, 1987).

Findings have revealed reduced N400 amplitudes among alcohol-dependent individuals, suggesting verbal processing impairments (Ceballos, Houston, Smith, Bauer, & Taylor, 2005). Similarly, the N450 is elicited in the right hemisphere by non-rhyming words compared to rhyming words. During a word-repetition priming task, which required participants to memorize twenty words and identify them in a series of 200 words, social drinkers who had been given alcohol had significant larger N450 amplitudes, suggesting increased difficulty of semantic access and integration (Marinkovic, Halgren, & Maltzman, 2004).

Event-Related Potentials and Binge Drinking

Though many studies have examined the relationship between alcohol and ERPs, few studies to date have investigated ERPs among binge drinkers. Crego et al. (2009) examined differences between binge drinkers and controls on N2 and P3 ERP components using a visual identical-pairs continuous performance task. Traditional continuous performance tasks require participants to maintain focus during a "boring" task consisting of responding to targets (e.g., numbers, symbols, sounds) and inhibiting responses to non-targets. The visual identical pairs continuous performance task increased the working memory load by denoting targets as any stimulus that appears on two successive trials. This requires participants to attend to each stimulus while also maintaining previous stimuli in working memory to detect a "match." Results revealed larger N2 ERPs in the binge drinkers and no differences between the matching and nonmatching conditions on the P3 component among binge drinkers. This suggests that binge drinkers require greater attentional effort to complete the task and possess a deficiency in differentiating between relevant and irrelevant information.

Maurage and colleagues (2009) examined longitudinal differences in P1, N2, and P3b ERPs in first year college students. College students were split into groups based on their expected alcohol consumption during the forthcoming academic year. No differences were found at baseline between the two groups; however, after nine months the binge drinkers had significantly delayed latencies for P1, N2, and P3b ERPs despite no differences in behavioral or psychological measures. This study highlights the importance of examining ERPs among college binge drinkers, as it suggests that cerebral dysfunction is detectable before any behavioral or psychological impairment.

Event-Related Potentials and Alcohol Summary

Taken together, the literature examining alcohol use and ERPs reveal a broad range of cognitive deficits associated with acute and chronic alcohol consumption. These deficits include, but are not limited to, working memory, sensory-perceptual processes, verbal processing, and attention. Although such deficits are associated with decision-making processes, they do not directly examine the degree to which there are neurocognitive mechanisms associated experiencing negative consequences among binge drinkers.

Error- and Feedback-Related Negativities

Because the overall aim of the proposed study is to examine neurocognitive responses to error among college students with varying drinking patterns, the present study employed the feedback-related negativity paradigm to examine study questions. The error- and feedback-related negativities (ERN and FRN, respectively) reflect neural signals that are part of the human error-processing system. These ERP components are ideal for examining the processing of negative outcomes among college binge drinkers

for the following reasons. First, the ERN/FRN is part of a learning process that is disrupted and altered by alcohol use (Holroyd & Yeung, 2003). Second, the ERN signal itself is affected by alcohol consumption (Easdon et al., 2005; Ridderinkhof et al. 2002; Yeung & Cohen, 2006) and family history of alcoholism (Fein & Chang, 2007), whereas other ERP components (e.g., N2, P3) remain intact.

The FRN is a negative deflection approximately 500 milliseconds after feedback in the ongoing EEG that is generated when outcomes from decisions are worse than expected (the ERN is elicited when errors are made but no feedback is given) (Holroyd & Coles, 2002) and is thought to yield information about the functional deficit underlying specific cognitive and behavioral expressions (Nieuwenhuis et al., 2002). Larger ERN amplitudes have been associated with better learning to avoid maladaptive responses or disadvantageous decisions (Frank et al., 2005). However, as noted below, heightened ERN amplitudes are associated with psychopathology, namely the anxiety disorders. This suggests that, similar to other neural factors (e.g., COMT Val^{158}Met; Wahlstrom et al., 2007), the ERN's functionality operates in an inverted U shaped curve, such that it effectively guides future behavior above and below a certain threshold.

Source localization indicates that the ERN/FRN response is elicited in the anterior cingulate cortex (ACC) (Dehane, Posner, & Tucker, 1994; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006; van Veen & Carter, 2002). This has also been supported through lesion studies revealing attenuated ERN responses in ACC lesion patients (Swick & Turken, 2002; Yeung & Cohen, 2006). However, other evidence suggests that a more complex system of frontal brain areas are involved in error-processing in addition to the ACC, including the medial prefrontal cortex, dorsolateral prefrontal cortex, and

orbitofrontal cortex (Brazdil, Roman, Falkenstein, Daniel, Jurak, & Rektor, 2002; Hermann, Rommler, Ehlis, Heidrich, & Fallgatter, 2004).

Behavioral tasks during which the ERN/FRN is elicited include choice reaction time (e.g., flanker task, go-nogo), risk taking (e.g., Balloon Analog Risk Task), and decision-making (e.g., gambling tasks) tasks. ERN/FRN disparities have been identified in a number of clinical populations. Heightened ERN/FRN responses have been identified in individuals with obsessive-compulsive disorder (Gehring, Himle, & Nisenson, 2000; Hajcak, Franklin, Foa, & Simons, 2008; Hajcak & Simons, 2002; Johannes et al., 2001; Ruchsow, Gron, Reuter, Spitzer, Hermle, & Kiefer, 2005) and generalized anxiety symptomology/worry (Hajcak, McDonald, & Simons, 2003). These larger ERN/FRN responses are thought to stem from heightened levels of negative affect (Hajcak, McDonald, & Simons, 2004) and ACC hyperactivity (Hajcak et al., 2003), both cornerstones of anxiety disorders. This results in augmented emotional and electrophysiological responses to error. Conversely, attenuated responses have been found in persons with dorsolateral prefrontal (Gehring & Knight, 2000) and orbitofrontal cortex lesions (Turken & Swick, 2008), depression (Ruchsow et al., 2005), schizophrenia (Bates, Kiehl, Laurens, & Liddle, 2002; Kerns et al. 2005; Polli et al., 2007), attention deficit hyperactivity disorder (Liotti et al., 2005), and substance dependence (Easdon et al., 2005; Franken, van Strien, Franzek, & van de Wetering, 2007). Reduced ERN/FRNs in these populations are theorized to reflect impaired ACC functionality (Forman et al., 2004), reduced emotional responding to errors (Bates et al., 2002), and impulsivity (Ruchsow, Spiter et al., 2005).

Error-/Feedback-Related Negativity and Alcohol Use

To date, few studies have examined the relationship between alcohol use and ERN/FRN. An examination of ERN amplitude during a flanker task (which requires participants to identify the direction of a center arrow that is flanked by either congruent or incongruent arrows) after consuming either alcohol or a placebo drink indicates that alcohol consumption resulted in reduced ERN amplitudes (Easdon et al., 2005; Ridderinkhof et al. 2002; Yeung & Cohen, 2006). Consumption of alcohol was found to have no effect on N2 amplitudes, a negative waveform ERP component associated with attention (Easdon et al., 2005; Ridderinkhof et al., 2002; Yeung & Cohen, 2006). Reduced FRN amplitudes were also found in treatment-naïve alcoholics who had a greater family history density (FHD) of alcohol problems; however, no relationship was found between P300 amplitude and FHD (Fein & Chang, 2007). These results further underscore the deleterious effect of alcohol use on the error processing system.

The chronic effects of alcohol use during college on the FRN have yet to be examined. On the one hand, findings previously discussed would suggest that those with the heaviest drinking patterns would exhibit the smallest FRN amplitudes. This notion is further supported by literature revealing negative neurocognitive effects of binge drinking (e.g., Hartley et al., 2004; Stephens & Duka, 2008; Townshend & Duka, 2005; Zeigler et al., 2005). Though previous studies have examined different ERP components (e.g., P300), the FRN is of particular importance when examining binge drinking among college students because college binge drinking peaks at the tail end of key developmental period when decision-making matures (Crone & van der Molen, 2004; Hooper et al., 2004), frontal brain areas responsible for higher-order cognitive functions have yet to reach full maturity (Casey et al., 2000; Gogtay et al., 2004) and the dopamine

system is in a state of flux (Wahlstrom et al., 2010). Furthermore, ERP deficits are evident among binge drinkers before any psychological or behavioral impairment are exhibited (Maurage et al., 2009). Taken together, college is a key developmental period to assess how neurocognitive mechanisms may explain the link between decision-making and problematic drinking behavior.

Reinforcement Learning

As previously stated, the ERN/FRN responses theoretically stems from reduced levels of dopamine in response to negative outcomes. Reinforcement learning (RL) theory provides a framework in which the interplay between the dopamine system and nearby brain structures explains decision-making and learning. This section will highlight the origins of RL theory, RL theory and dopamine, RL theory and the ERN/FRN, the RL theory of addiction, and the relationships among RL theory, ERN/FRN, and alcohol use.

Origins of Reinforcement Learning Theory

RL theory is a computational approach that differs from other learning theories because it emphasizes direct interaction with the environment without supervision or models of the environment (Sutton & Barto, 1998). In other words, one must discover which actions yield rewards through trial and error. In RL, an agent (e.g., animal, human) observes their environment and then chooses and performs an action. This action changes the state of the environment and results in either a positive (reward) or negative (punishment) payoff. From this payoff the agent learns which actions lead to future payoffs and/or maximizes long term payoffs. These payoffs are subjective in that rewards and punishments are dependent on the agent's evaluation (Dayan & Watkins, 2001). The RL process involves a balance between exploitation and exploration, such that an agent

must exploit what they already know to obtain rewards yet also explore to obtain rewarding future outcomes (Barto, 1994; Sutton & Barto, 1998).

Reinforcement Learning and Dopamine

Early work identified dopamine as the brain's reward signal (Wise, Spindler, de Wit, & Gerberg, 1978; Wise, Spindler, & Legault, 1978); however, contemporary theories of RL have suggested that dopamine firing instead reflects a reward prediction error (Barto, 1995; Bayer & Glimcher, 2005; Hollerman & Schultz, 1998; Schultz, Dayan, & Montague, 1997). The reward prediction error [also referred to as temporal difference error (Sutton & Barto, 1998)] reflects the difference between the predicted reward from an action and the reward actually obtained after the action is complete (Bayer & Glimcher, 2005). If the predicted reward and the reward obtained match (i.e., a monkey expecting juice after pressing a lever obtains juice after pressing a lever), there is no reward prediction error and no firing of dopamine neurons. However, if the predicted reward is incongruent with obtained reward, it produces either a rapid increase (if the predicted reward was lesser than the obtained reward) or decrease (if the predicted reward was greater than the obtained reward) in dopamine (Bayer & Glimcher, 2005). Once the reward is correctly predicted, learning is complete (Redish, 2004).

Reinforcement Learning and the Error-/Feedback-Related Negativity

The ERN/FRN results from the impact of phasic dopamine on the anterior cingulate cortex (ACC), an area of the brain involved in reward prediction, decision-making, and emotion (Bush et al., 2000; Holroyd & Yeung, 2003). When a decision is made, the basal ganglia predict the outcome from that decision. If the outcome is better than the prediction, a positive error signal is produced, whereas if the outcome is worse

than predicted, a negative error signal is produced. These signals are then carried by mesencephalic dopamine system to the ACC, which reinforces behavior, and to the basal ganglia, which strengthens future predictions (Holroyd & Coles, 2002; O'Reilly & Frank, 2006). Positive error predictions map on to phasic increases in mesencephalic dopamine and do not elicit the ERN/FRN. However, negative error predictions are reflected in a phasic dip in mesencephalic dopamine and the ERN/FRN is elicited.

Reinforcement Learning Theory of Addiction

Drug use disrupts normal RL processes. This disruption stems from the pharmacological effects of a substance that increase phasic dopamine, which is interpreted by the brain as a positive prediction error (i.e., outcome is better than expected). Each time a drug is used, its value increases which, over time, results in an overvaluation of a drug's reward and devaluation of non-drug, natural rewards (Redish, 2004). Furthermore, as the drug's value increases, the negative consequences associated with use are increasingly discounted (Dezfouli, Piray, Keramati, Ekhtiari, Lucas, & Mokri, 2009), a cornerstone of addiction (World Health Organization, 1992).

In humans, support has been found for the RL theory, though it has not been widely studied. D2/D3 dopamine agonists have been shown to impair learning of reward-related behaviors in healthy individuals due to an attenuation of the phasic dopamine firing (Pizzagalli et al., 2008). That is, pharmacological alterations of dopaminergic activity disrupt learning via the same mechanisms that substances do. Furthermore, among alcohol-dependent men, functional magnetic resonance imaging (fMRI) has shown that despite unaltered striatal reward prediction errors, the functional connectivity between the striatum and the dorsolateral prefrontal cortex is damaged. The degree of

functional connectivity impairment was associated with greater alcohol craving and learning impairments on a RL task (Park et al., 2010). Faulty fictive (i.e., outcomes that have not been experienced) learning signals have also been found absent among smokers (Chiu, Lohrenz, & Montague, 2008), which suggests that the continued use of substances despite negative consequences may partially stem from an inability to utilize information related to potential (but not actually experienced) negative consequences.

Reinforcement Learning, the Error-/Feedback-Related Negativities, and Alcohol

The mesencephalic dopamine system plays an integral role in alcohol use. The consumption of alcohol activates dopamine neurons in the ventral tegmental area and increases tonic (i.e., sustained) dopamine levels in the nucleus accumbens. Furthermore, reinforcement from alcohol is dependent on D1 and D2 (Dyr, McBride, Lumeng, Li, & Murphy, 1993) dopamine receptor density and affinity in the striatum. Because alcohol increases mesencephalic tonic dopamine activity, the phasic dip required to facilitate RL and elicit the ERN/FRN does not occur. Therefore, when outcomes during alcohol use are worse than expected (e.g., injury, legal problems) future drinking may not be adapted due to disruptions in normal dopamine activity.

As previously discussed, few studies to date have examined the association between the ERN/FRN and alcohol use. Further, no studies have explicitly tested the RL theory of the ERN/FRN. The goal of the present study is to examine FRN functionality among binge drinkers, a population in which the ERN/FRN has yet to be measured. To accomplish the overarching study aim, there is the need to use a behavioral task that parallels real life decision-making. That is, a task where outcomes from decisions are initially unknown and future behavior can be modified based on decisional outcomes.

Iowa Gambling Task

Although originally intended to simulate real-life decision-making (Bechara et al., 1994), the Iowa Gambling Task (IGT) is also an ideal measure of RL within the current framework. First, participants on the IGT are instructed to maximize rewards and minimize punishments but are given limited information regarding task contingencies, which is consistent with the requirements of RL outlined by Sutton and Barto (1998). Furthermore, the maximization of rewards and minimization of punishments changes with each decision; such that one uses the information they have obtained up to that point to guide future choices (Maia & McClelland, 2004). Second, optimal task performance requires a balance of exploration of un-sampled decks and exploitation of already learned task contingencies, a critical component of RL (Maia & McClelland, 2005; Sutton & Barto, 1998). Third, the brain circuitry involved in IGT performance (primarily the ventromedial prefrontal cortex) is also integrally involved in RL in both humans and animals (Newman, Polk, & Preston, 2008). Taken together, this evidence highlights the utility of the IGT as an index of RL. This section will provide a description of the IGT, explain theories of the IGT, discuss the ventromedial prefrontal cortex (the brain area integrally involved in IGT performance), review the literature related to IGT performance among substance users, and discuss IGT performance among binge drinkers.

Task Description

As previously stated, the IGT is a behavioral task that simulates the uncertainty of real-life decisional outcomes by necessitating the balance of rewards and punishments when making decisions. Specifically, individuals are required to make selections from four decks of cards (A, B, C, and D). They are told that the goal of the task is to

maximize winnings and that some decks are more advantageous than others in terms of their ultimate payoffs. Participants are required to make 100 choices on the task and may chose from any deck. After the selection of each card, the participant is informed of the amount of money won and lost (during which the FRN would be elicited) from that selection. Two of the decks (disadvantageous decks) yield high immediate rewards coupled with larger delayed punishments, resulting in long-term losses. On the other hand, two decks (advantageous decks) yield low immediate rewards in conjunction with smaller delayed punishments, resulting in long-term gains (Bechara, Damasio, & Damasio, 2000; Bechara et al., 1994). Each deck is unique with regards to its specific reward and punishment frequency and magnitude and each selection results in a monetary gain and loss that is consistent with the selected deck's reward/punishment schedule. Healthy controls tend to sample from all decks at the beginning of the task and gradually make more selections from advantageous decks and fewer from disadvantageous decks as the task progresses (Bechara et al., 2000).

Theories of the Iowa Gambling Task

Two disparate theories have been proposed to explain IGT performance. The original theory, the somatic marker hypothesis (Damasio, 1994), posits that bodily states (i.e., somatic signals) reflecting an emotional response to a potential decision guide individuals at an implicit level regarding whether or not to choose from a particular deck of cards. Experimentally, skin conductance responses (SCRs) have been utilized as a measure of these somatic markers guiding decision-making. Research has shown that when contemplating a risky decision, healthy controls have heightened SCRs as compared to when contemplating non-risky decision. Presumably, the somatic signals

represented by this heightened SCR activity are generated because advantageous decision makers have properly extracted the contingencies from the decks based on their prior experience (i.e., they are sensitive to the large punishments yielded by the disadvantageous decks). By questioning participants about task contingencies, it has been shown that heightened SCRs occur prior to explicit knowledge of which decks are advantageous and which decks are disadvantageous, which has been interpreted as evidence that participants are relying on somatic markers to guide decisions as opposed to conscious understanding of deck contingencies (Bechara, Damasio, Tranel, & Damasio, 1997).

More recent evidence suggests that participants have more consciously accessible knowledge about task contingencies than posited by the Iowa group. Maia and McClelland (2004) asked participants more sensitive questions to assess their knowledge of IGT contingencies. Specifically, they asked structured questions (including Likert-type items) after the first twenty selections and then subsequently after each subsequent ten selections. They found that when participants behave advantageously on the IGT, they have conscious and accessible knowledge of the good and bad decks. The debate over the implicit versus explicit knowledge about IGT contingencies has yet to be resolved, which has made interpreting the task and understanding the decision-making process more challenging. The proposed study, consistent with Maia and McClelland's (2004) IGT theory, postulates that poor IGT performance results from feedback processing and learning deficits rather than an inability to react to somatic signals.

Ventromedial Prefrontal Cortex

The ventromedial prefrontal cortex (VMPFC) has been widely implicated as the critical brain area in decision-making processes (Bechara et al., 1994; Bechara, Damasio, Damasio, & Lee, 1999; Bechara, Tranel, & Damasio, 2000; Damasio, 1994; Fellows & Farah, 2007; Sanfey, Hastie, Colvin, & Grafman, 2003; Volz, Schubotz, & von Cramon, 2006). Unlike healthy controls, individuals with VMPFC lesions fail to identify and avoid the risky decks despite intact intelligence and neuropsychological functioning (Damasio, 1994; Damasio et al., 1991). Furthermore, VMPFC lesion individuals do not exhibit the SCR changes when contemplating a risky choice that healthy controls do (Bechara et al., 2000). Though early studies indicated that decision-making was localized to the VMPFC, recent evidence suggests that individuals with frontal lobe damage exhibit performance similar to those with VMPFC damage. This does not support the specific association between VMPFC lesions and impaired IGT performance (MacPherson, Phillips, Della Sala, & Cantagallo, 2009).

Iowa Gambling Task Performance and Substance Use

Findings have revealed impaired IGT performance in substance users, including alcohol (Bechara et al., 2001; Gonzalez, Bechara, & Martin, 2007), stimulants (Bechara et al., 2001), heroin (Petry, Bickel, & Arnett, 1998), marijuana (Verdejo-Garcia, Benbrook, Funderburk, David, Cadet, & Bolla, 2007; Whitlow et al., 2004), MDMA (Hanson, Luciana, & Sullwold, 2008), methamphetamine (Gonzalez et al., 2007), and cocaine (Bolla et al., 2003; Verdejo-Garcia et al., 2007). Furthermore, deficits in IGT performance persist even after a sustained period of drug/alcohol abstinence (Fein, Klein, & Finn, 2004). Overall, these findings suggest that drug and alcohol users are more strongly influenced by the large rewards from the disadvantageous decks and have more

difficulty adapting behavior despite long-term losses than healthy controls. This reflects the hypersensitivity to reward and insensitivity to punishment exhibited by substance users (Bechara et al., 2001).

Iowa Gambling Task Performance and Binge Drinking

To date, few studies have examined IGT performance among binge drinkers. Goudriaan and colleagues (2007) followed college students over a two-year time period and classified them based on their binge drinking trajectories. These trajectories included: (1) low binge drinkers, (2) stable moderate binge drinkers, (3) increasing binge drinkers, and (4) stable high binge drinkers. Findings revealed that chronic high binge drinkers performed worse on the IGT than the consistent low binge drinkers, which would theoretically map onto reduced FRN amplitude. Furthermore, results suggest that binge drinking during late adolescence (i.e., eighteen- to nineteen-years) is related to poorer IGT performance compared to binge drinking during young adulthood (i.e., twenty- to twenty-one-years). No differences in IGT performance were found between men and women nor was a significant relationship between impulsivity and IGT performance. Additionally, Xiao and colleagues (2009) examined whether IGT performance would serve as a marker to predict future binge drinking in Chinese adolescents. Results indicated that those adolescents who performed poorer on the IGT were more likely to be current binge drinkers or progress into binge drinking than those adolescents who evidenced better performance on the IGT. Although some data suggest that the IGT may differentiate who is a current binge drinker or one that is progressing towards binge drinking, the purpose of the present study is not to further clarify IGT performance

among binge drinkers. Instead, the task will be utilized to examine FRN responses to making an error on the task.

Study Aims

Because not all adolescents engage in substance use or binge drinking during college, examining FRN differences among college students with varying patterns of alcohol consumption will provide important insights into the neurocognitive mechanisms underlying reduced sensitivity to binge drinking consequences. As such, the first aim is to examine FRN amplitude differences among non-binge, binge, and heavy drinking college students. It is hypothesized that non-binge drinkers will exhibit the largest FRN amplitudes, whereas heavy drinkers will exhibit the smallest FRN amplitudes. The second aim relates to the explicit relationship between FRN and decision-making. It is hypothesized that there will be a positive linear relationship between FRN amplitude and number of advantageous decisions on the IGT. The third aim makes the first attempt to disentangle how the FRN may be altered in response to dopamine/prefrontal cortex neuroadaptation resulting from the onset of substance abuse before the brain has finished developing, which will be accomplished by examining the relationship between age of drinking onset and FRN amplitude. It is hypothesized that there will be a positive linear relationship between age of drinking onset and FRN amplitude.

Chapter 2: Method

Participants

The sample consisted of thirty college men between the ages of eighteen and twenty-three (M = 19.36, SD = 1.37). Ten participants were included who endorsed at least five binge drinking episodes during each month assessed (heavy drinkers), ten

participants were included who endorsed at least one binge drinking episodes during each month assessed (binge drinkers), and ten participants were included that endorsed fewer than one binge drinking episode each month (non-binge drinkers). To estimate the sample size needed to detect the hypothesized effects, power analyses were conducted using Cohen's (1988) guidelines for effect sizes in conjunction with G*Power Version 3.1.2 (Faul et al., 2007; 2009). G*Power is a computer software program designed to compute necessary sample size for common statistical analyses. Based on prior research reporting a large between group effect size averaging .70 (examining P300 and disinhibitory disorders in adolescent males), as described by Iacono et al. (2002), this corresponds to a $F^2 = .35$. Using this effect size with 80% power and a 5% chance of Type I error, G*Power 3.1.2 indicated that an overall sample size of $N = 24$ (across 3 groups) would be needed to detect the hypothesized effect.

Participants were recruited through Experimetrix, a website used for undergraduate students to enroll in research studies. Consistent with the criteria established by SAMHSA (2006), binge drinking episodes were defined as consuming five or more drinks on the same occasion. A 90-day timeline followback (TLFB; Sobell, Maisto, Sobell, & Cooper, 1979; Sobell & Sobell, 1995) of number of drinks and number of hours spent drinking was given to each participant to assess binge drinking status. Further, blood alcohol content was calculated based on gender, weight, hours spent drinking, and number of standard drinks consumed to ensure that binge drinkers' blood alcohol levels reached at least .08 during reported binge drinking episodes. Episodes were not considered a binge if BAC did not reach .08. Exclusionary criteria were the following: (a) current nicotine and/or drug use; (b) history of traumatic brain injury,

neurological disorder, or loss of consciousness for twenty minutes or longer; (c) currently taking prescription medication; (d) score >90 on the global severity index or two symptomatic dimensions on the Symptom Checklist-90-R; or (e) older than twenty-three years. No participants were excluded based on these criteria. All participants gave written informed consent. The institutional review board at the University of Nebraska-Lincoln approved this study.

Measures

Demographics Questionnaire. Participants completed a demographics questionnaire that included questions on age, gender, ethnicity, history of emotional/mental problems, medications, year in school, grade point average, hometown population, family income, fraternity/sorority affiliation, employment status, family history of alcohol/drug problems, age of drinking onset, and drug use.

Wechsler Abbreviated Scale of Intelligence (WASI). The WASI (Wechsler, 1999) is a brief, reliable measure of intelligence and has been nationally standardized. The WASI yields Verbal, Performance, and Full Scale IQ scores. A full-scale IQ estimate can be obtained by administering only the Vocabulary and Matrix Reasoning subtests.

Symptom Checklist – 90 – Revised (SCL-90-R). The SCL-90-R (Derogatis, 2002) is a 90-item self-report screener used to assess a broad range of psychological symptoms. Each question is answered on a 5-point Likert-type scale, with response options ranging from “not at all” (0) to “extremely” (4). The 90-items comprise nine symptom dimensions, including somatization, depression, anxiety, obsessive-compulsive, interpersonal sensitivity, hostility, paranoid ideation, phobic anxiety, and psychoticism.

Additional global indices of psychological distress include the global severity index, positive symptom total, and positive symptom distress index.

Alcohol Use Disorders Identification Test (AUDIT). The AUDIT (Saunders, Aasland, Babor, De La Fuente, & Grant, 1993) was developed by the World Health Organization and consists of 10 Likert-type items used to assess harmful and hazardous alcohol use. The AUDIT assesses alcohol consumption (quantity and frequency), binge behavior, and alcohol-related consequences. Scores range from 0 to 40, with a score of 8 or higher indicating the likelihood of harmful alcohol consumption. The AUDIT has shown good internal consistency (Fleming, Barry, & MacDonald, 1991) and discriminant validity (Saunders et al., 1993).

Rutgers Alcohol Problem Index (RAPI). The RAPI (White and Labouvie 1989) is used to assess problem drinking in adolescents and young adults. The RAPI includes 26-items related to problems experienced due to alcohol use in the past 6 months, each of which is answered on a Likert-type scale ranging from 0 (never) to 4 (more than 10 times). The RAPI has shown good reliability, internal consistency, and validity with young adults (White and Labouvie 1989).

Iowa Gambling Task (IGT). Participants completed a computerized version of the Iowa Gambling Task (IGT; Bechara et al., 1994; Hooper et al., 2004). Participants were instructed to make choices from four decks of cards (labeled A, B, C, and D) until the computer instructed them to stop. They were told that after each selection they would be awarded money; however, some selections would also result in a loss of money. They were told they could switch among decks are often as they liked, but that some decks were better than others in terms of ultimate payoffs, and that the deck positions and

contingencies were fixed and would not change throughout the task. They were told that their goal was to win as much money as possible, and, if they found that too difficult, to avoid losing money as much as possible. Instructions given were standard for the IGT as reported by Bechara et al. (1994).

Participants made 100 selections during the task and each of the four decks differed in terms of their short- and long-term payoffs. The disadvantageous decks (A and B) yielded $.25 in reward per choice and had either frequent smaller losses (A; 50% of choices) or infrequent larger losses (B; 10% of choices). If a participant chose solely from these decks, it would result in a net loss of $1.25 over the course of 20 selections. The advantageous decks (C and D) yielded $.10 or $.15 in reward per choice and had either frequent (C; 50% of choices) or infrequent (D; 10% of choices) losses, though the losses were smaller than those from decks A and B. If participants chose solely from decks A and B, it would result in a net gain of $1.25 over the course of 20 selections.

The task used in the present study differed from the original Iowa group task in several ways. First, the present task yielded smaller amounts of money that were proportionately consistent with the original task and participants were paid their winnings. Other studies have found results with the altered reward magnitude among adults to be consistent with those from the Iowa group (Ernst et al., 2002; Mazas, Finn, & Steinmetz, 2000; Petry, 2001; Petry et al., 1998; Reavis & Overman, 2001). Second, this task did not limit choices from each deck, resulting in a task that is more representative of real-life decision-making and yielding additional opportunities to make disadvantageous decisions (during which the FRN is elicited).

Procedure

Participants were tested individually across two test sessions that took place at least one day apart. During the first session, participants gave written informed consent followed by completion of the 90-day TLFB questionnaire and the SCL-90-R to ascertain eligibility. They were also administered the Vocabulary and Matrix Reasoning subtests form the Wechsler Abbreviated Scales of Intelligence (WASI) to obtain an intelligence quotient (IQ) estimate to be used as a covariate and completed a demographics questionnaire, the AUDIT, and the RAPI. The second session (ERP session) occurred between one and four days following the first session. Participants were instructed not to consume caffeine, nicotine, alcohol, or processed sugar the morning prior to testing. Compliance with these instructions was confirmed prior to ERP session. During the second session participants completed the IGT, during which ERP data was collected.

During ERP data collection, participants were seated comfortably and their head was measured to determine the appropriate electrode net size by identifying the mid-central position on the top of the head (Cz) and the position central at the top of the bridge of the nose (nasion; Fz). Prior to positioning of the net on the participant, the electrode net was soaked in a mixture of distilled water and saline. After application of the electrode net, participants were positioned in front of a high-resolution computer monitor positioned one meter in front of the participant.

Presentation of the IGT stimuli was controlled by the Electrophysiological Graphical Imaging System (EGIS), v. 2.2. During completion of the task, participants' EEG and behavioral observations were continuously monitored. Stimulus presentation

was suspended during periods of motor activity or inattention and resumed once alertness has resumed and/or motor activity has subsided.

A MacBook Pro computer running NetStation© 4.1.1 (EGI, Inc.) was used to record the electrophysiological data with a sampling rate of 250 samples/sec. The brainwaves were recorded using high-density array of 256 HydroCel electrodes embedded in soft sponges and arranged into a net (Geodesic Sensor Net, EGI Inc.; see Figure 2.1). During recording, all electrodes were referenced to Cz and later transformed to an average-reference. All impedances remained at or below 40 kOhms throughout the testing session as indicated by measures taken before and after the task. The filter setting for high-pass was set to .1 hertz and the low-pass was set to 100 hertz. No notch filter was used. The FRN typically occurs 250 milliseconds after feedback is given, and thus was measured starting 100 milliseconds prior to feedback and lasting 1000 milliseconds after feedback. This time was identified based on previous work that has examined the FRN (e.g., Frank et al., 2005; Holroyd & Coles, 2002).

Data Analysis Plan

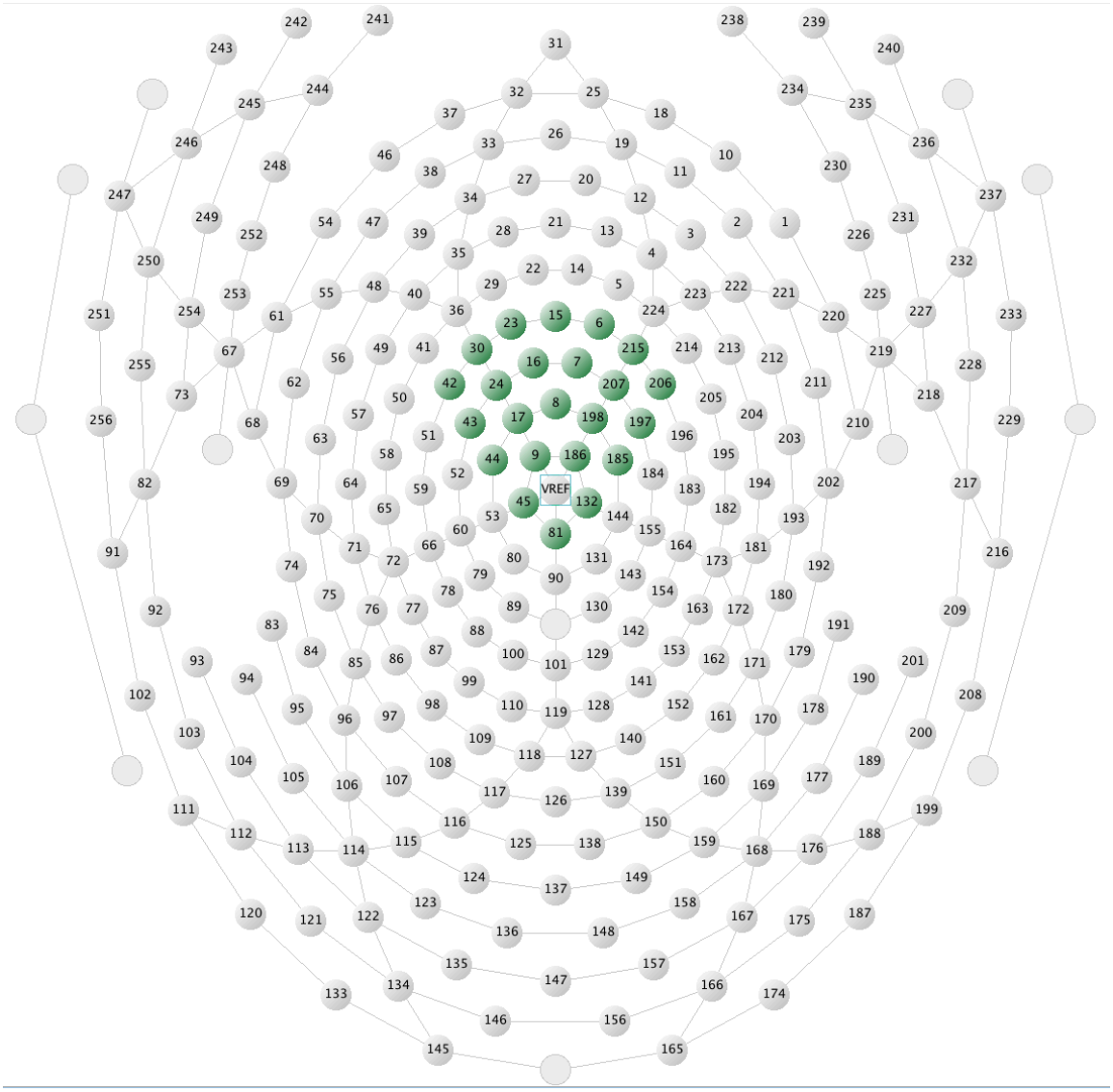
Data Preprocessing. First, data were filtered using a 3.0 hz highpass and 30.0 hz lowpass filter. No notch filter was used. Next, data was segmented to a range of 100 ms pre feedback and 1000 ms post feedback. Because the number of wins was greater than the number of losses, only wins were examined that preceded a loss, allowing for an equivalent number of win and loss segments for the grand averages. Next, bad channels were replaced and artifact detection was conducted for bad channels, eye blinks and eye movements. The max-min microvolt was 150 for each with a window size of 1000 ms and a moving average of 80 ms. Channels were marked bad if they were bad on greater

than twenty percent of targeted trials, and a segment was marked bad if a) it contained more than ten bad channels; b) it contained an eye blink; or c) contained an eye movement. Two participants were excluded from analyses due to high levels of artifact in FRN data. Subsequent to artifact detection, bad channel replacement was conducted again. Montage operations were then run for a 256 1.0 HyrdroCel net to an average reference. Baseline correction was then conducted, selecting from a portion of the segment beginning 200 ms before the feedback and lasting for 200 ms.

Files were averaged by group (non-binge, binge, and heavy drinkers) and also to a grand average including all participants. Data was extracted separately for wins (red line) and losses (blue line). For each, the maximum and minimum amplitudes were measured in a window of 120 ms to 210 ms after feedback. This window was selected based on visual inspection of the occurrence of the FRN in the grand average. Channels were selected based on previous literature (Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003; Figure 1) examining the FRN. The final FRN was determined to be the difference between the most positive peak prior to the FRN and the most negative peak (i.e., peak-to-peak; Frank, Woroch, & Curran, 2005) in a 120 to 210 ms time window. Latency for the negative peak was extracted and occurred between 120 and 210 ms after feedback. Bad segments were excluded from statistical extraction. Electrode 23 was specifically chosen to examine study aims due to its statistical relationship with study variables.

Figure 2.1

Electrodes Originally Selected for Analysis



Primary Analyses. The study aims were addressed using one-way Analysis of Covariance to examine group difference and stepwise linear regression to examine the linear relationship between variables of interest.

A one-way Analysis of Covariance (ANCOVA) was used to examine the FRN peak-to-peak amplitude differences among non-binge, binge, and heavy drinking college

students with WASI FSIQ as a covariate. It was expected that college students classified as heavy drinkers would exhibit the smallest FRN peak-to-peak amplitudes, followed by binge drinkers, and with non-binge drinkers exhibiting the largest FRNs.

Stepwise linear regression was used to examine the relationship between IGT performance and FRN amplitude in the total sample. IGT performance served as the dependent variable. In step 1, WASI FSIQ was entered. In step 2, FRN peak-to-peak amplitude was entered. It was hypothesized that a positive linear relationship would exist between FRN peak-to-peak amplitude and number of advantageous decisions on the IGT.

Stepwise linear regression was used to examine the relationship between age of drinking onset and FRN peak-to-peak amplitude in the total sample. FRN peak-to-peak amplitude served as the dependent variable. In step 1, WASI FSIQ was entered. In step 2, age of drinking onset was entered. It was expected that a positive linear relationship would exist between age of drinking onset and FRN amplitude.

Chapter 3: Results

Preliminary Analyses

Sample characteristics are presented in Table 3.1. The mean age for the total sample was 19.36 (*SD* = 1.37) and the majority of participants were White (89.3%). The mean age of first drink (i.e., age of onset) was 16.89 (*SD* = 1.31), AUDIT total was 12.00 (*SD* = 6.26), WASI FSIQ was 108.86 (*SD* = 8.76), number of advantageous decisions on the IGT was 50.82 (*SD* = 17.88), and FRN peak-to-peak amplitude was 5.91 μv (*SD* = 2.20).

Table 3.1

Sample Characteristics

Variable		*M* (*SD*) or *n* (%)
Age		19.36 (1.37)
Ethnicity	White	25 (89.3%)
	Black	1 (3.6%)
	Hispanic/Latino	2 (7.1%)
Age of Onset		16.89 (1.31)
AUDIT Total		12.00 (6.26)
RAPI Total		14.11 (13.58)
WASI FSIQ Estimate		108.86 (8.76)
IGT Advantageous Decisions		50.82 (17.88)
	Block 1	9.18 (3.39)
	Block 2	10.64 (4.47)
	Block 3	10.29 (4.28)
	Block 4	10.61 (4.66)
	Block 5	10.11 (5.21)
FRN peak-to-peak amplitude (in μV)		5.91 (2.20)

One-way analysis of variances (ANOVAs) were used to examine difference in study variables among non-binge (n = 10) binge (n = 9), and heavy (n = 9) drinkers (Table 3.2). AUDIT (F = 17.82, p < .001) and RAPI (F = 4.72, p = .019) total revealed significant mean differences across groups. Specifically, Tukey's post hoc tests revealed that non-binge, binge, and heavy drinkers all significantly differed from one another on AUDIT total (ps > .05). For RAPI total, non-binge drinkers had significantly lower RAPI total scores than heavy drinkers (p = .015). No other group differences were found on age, age of drinking onset, WASI FSIQ, IGT advantageous decisions, or FRN amplitude.

Table 3.2

Differences Among Drinkers

	Non-Binge Drinkers (n = 10)	Binge Drinkers (n = 9)	Heavy Drinkers (n = 9)	F	p
Age	19.20 (.51)	19.67 (.50)	19.22 (.32)	0.32	.726
Age of Onset	17.30 (.40)	17.00 (.37)	16.33 (.50)	1.36	.275
AUDIT Total	6.80 (.88)	12.00 (1.09)	18.50 (2.13)	17.82	<.001
RAPI Total	7.30 (1.88)	12.44 (2.43)	24.50 (6.94)	4.72	.019
WASI FSIQ Estimate	110.00 (2.24)	108.89 (3.78)	107.56 (2.79)	0.17	.842
IGT Advantageous Decisions	50.60 (4.45)	47.78 (6.35)	54.11 (7.23)	0.27	.767
FRN amplitude (in μV)	6.97 (.67)	5.39 (.64)	5.25 (.78)	1.93	.17

Note. Values represent mean (standard error).

Pearson's correlations (Table 3.3) were used to examine linear relationships among age of drinking onset, AUDIT total, RAPI total, WASI FSIQ, FRN amplitude, and IGT advantageous decisions. Age of onset was negatively correlated with AUDIT total (r = -.58, p < .001), and RAPI total (r = -.59, p > .001). AUDIT total was positively correlated with RAPI total (r = .84, p > .001) and negatively correlated with FRN peak-to-peak amplitude (r = -.38, p = .048). No other significant correlations were found.

Table 3.3

Zero-Order Correlations

	1.	2.	3.	4.	5.	6.
1. Age of Onset	-	-.58**	-.59**	.28	.31	-.06
2. AUDIT Total	-	-	.84**	-.33	-.38*	.00
3. RAPI Total	-	-	-	-.27	-.09	.16
4. WASI FSIQ	-	-	-	-	.02	.30
5. FRN	-	-	-	-	-	.01
6. IGT Advantageous Decisions	-	-	-	-	-	-

*Note. p >.05, *p <.05, **p <.001*

ERP Analyses

Figures 3.1-3.4 show a plot of the averaged segmented EEG wave for all 256 electrodes for each non-binge, binge, and heavy drinkers, as well as the total sample. Red lines indicate win trials and blue lines indicate loss trials. Visual inspection reveals that the amplitudes are most prominent in the region of interest, the anterior cingulate cortex. This is also depicted via topography map (Figures 3.5-3.8). For each non-binge, binge, and heavy drinkers, as well as the total sample, the signal strength, frequency, and location on the head are depicted at 0, 120, 150, and 210 ms post feedback. The color corresponds to the color band next to each circle, whereby red is most positive and purple is most negative. The circle on the right represents the aggregated loss trials and the circle on the left the win trials. As is evidenced in the figures, the negativity peaks around 150 ms post feedback. These topographic representations were used to assist in determining

the window in which the FRN would be measured. Lastly, Figures 3.9-3.12 depict the segmented wave for the selected electrode (23) for each non-binge, binge, and heavy drinkers, as well as the total sample.

Figure 3.1

Topo Plot Non-Binge Drinkers

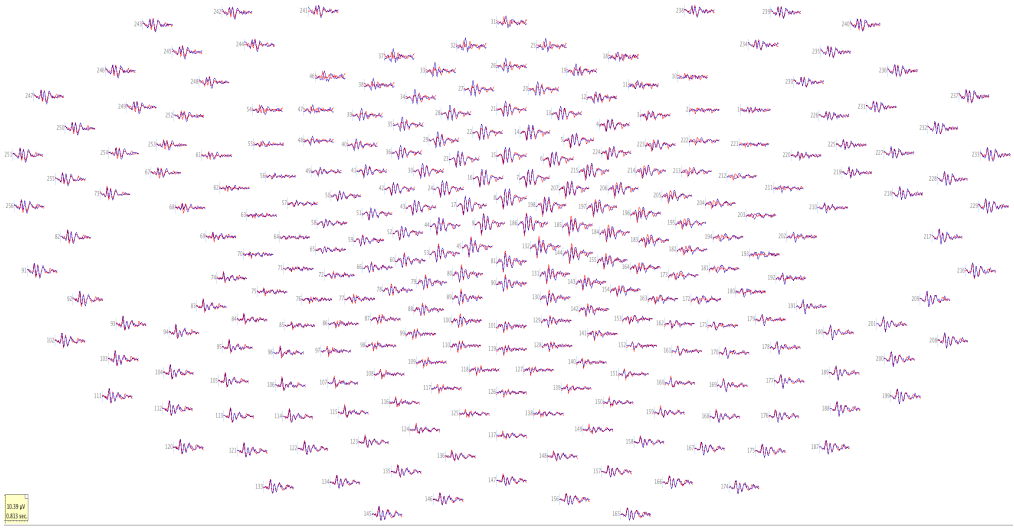


Figure 3.2

Topo Plot Binge Drinkers

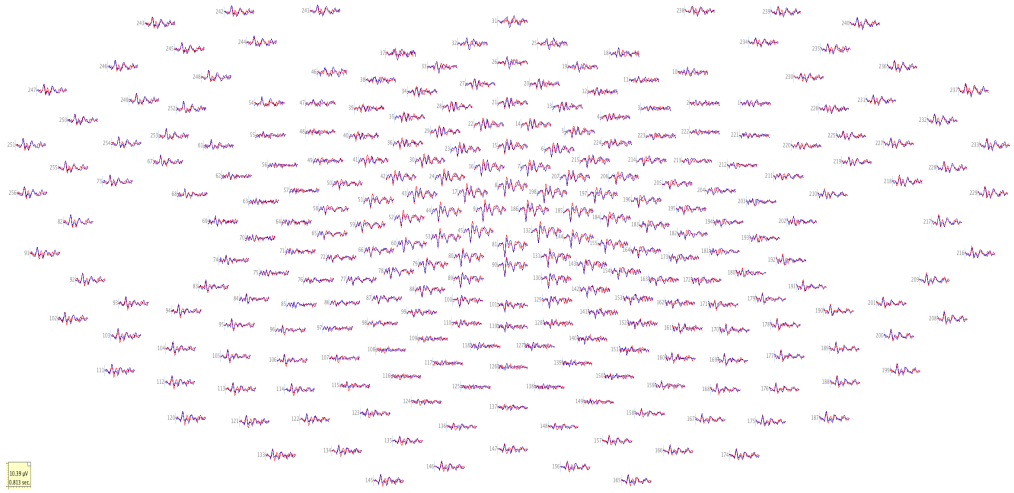


Figure 3.3

Topo Plot Heavy Drinkers

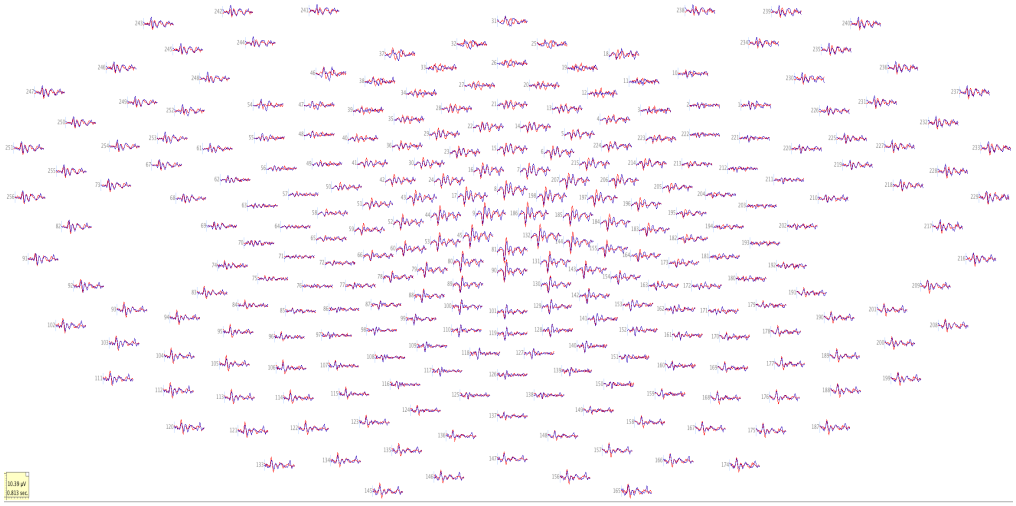


Figure 3.4

Topo Plot Total Sample

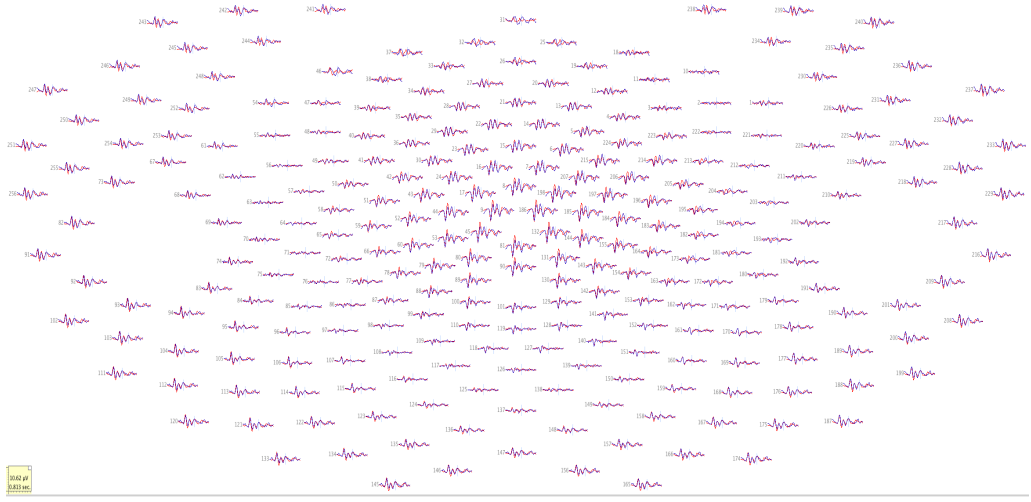


Figure 3.5

Topo Map 0, 120, 150, and 210 ms Post Feedback Non-Binge Drinkers

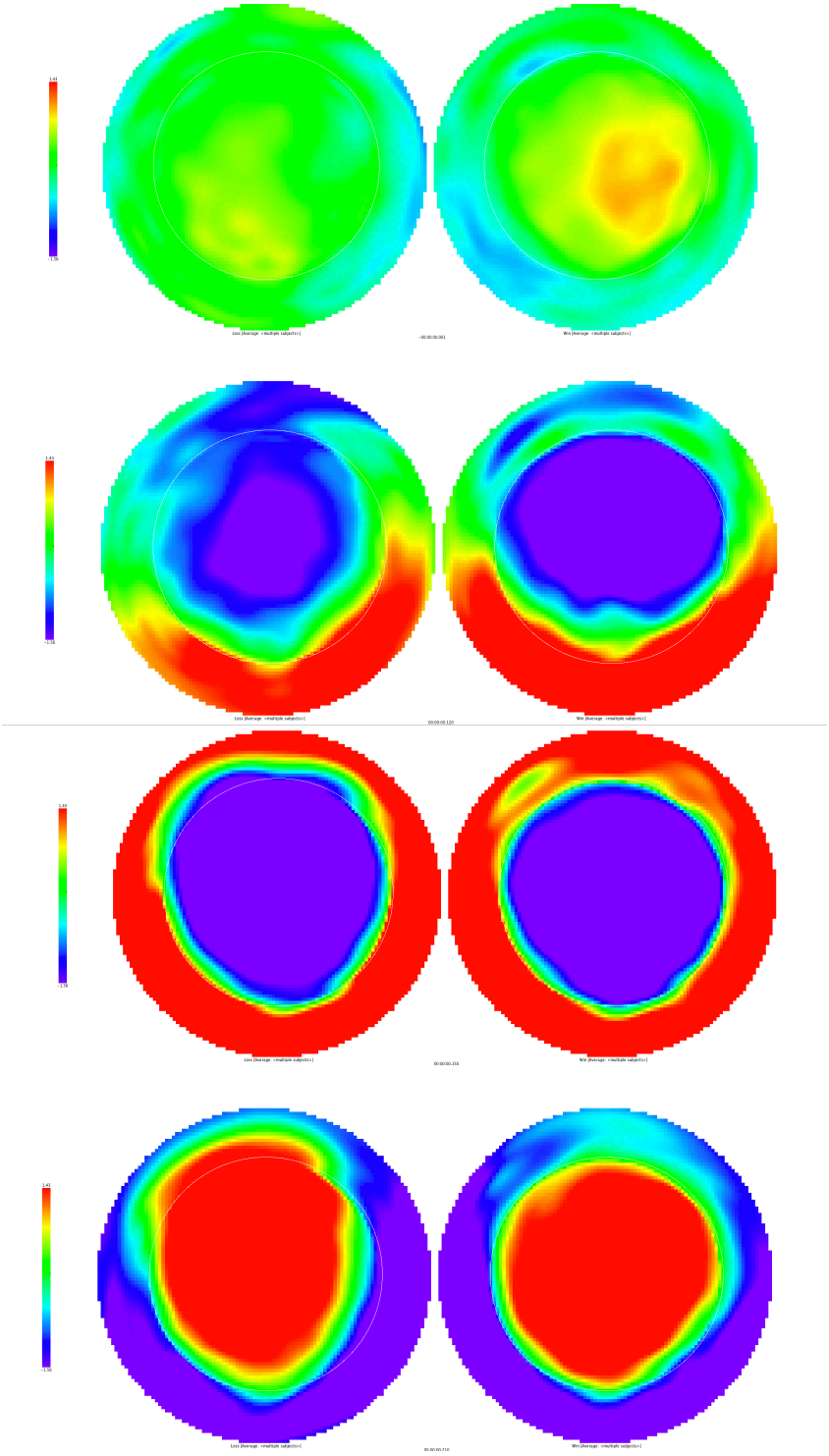


Figure 3.6

Topo Map 0, 120, 150, and 210 ms Post Feedback Binge Drinkers

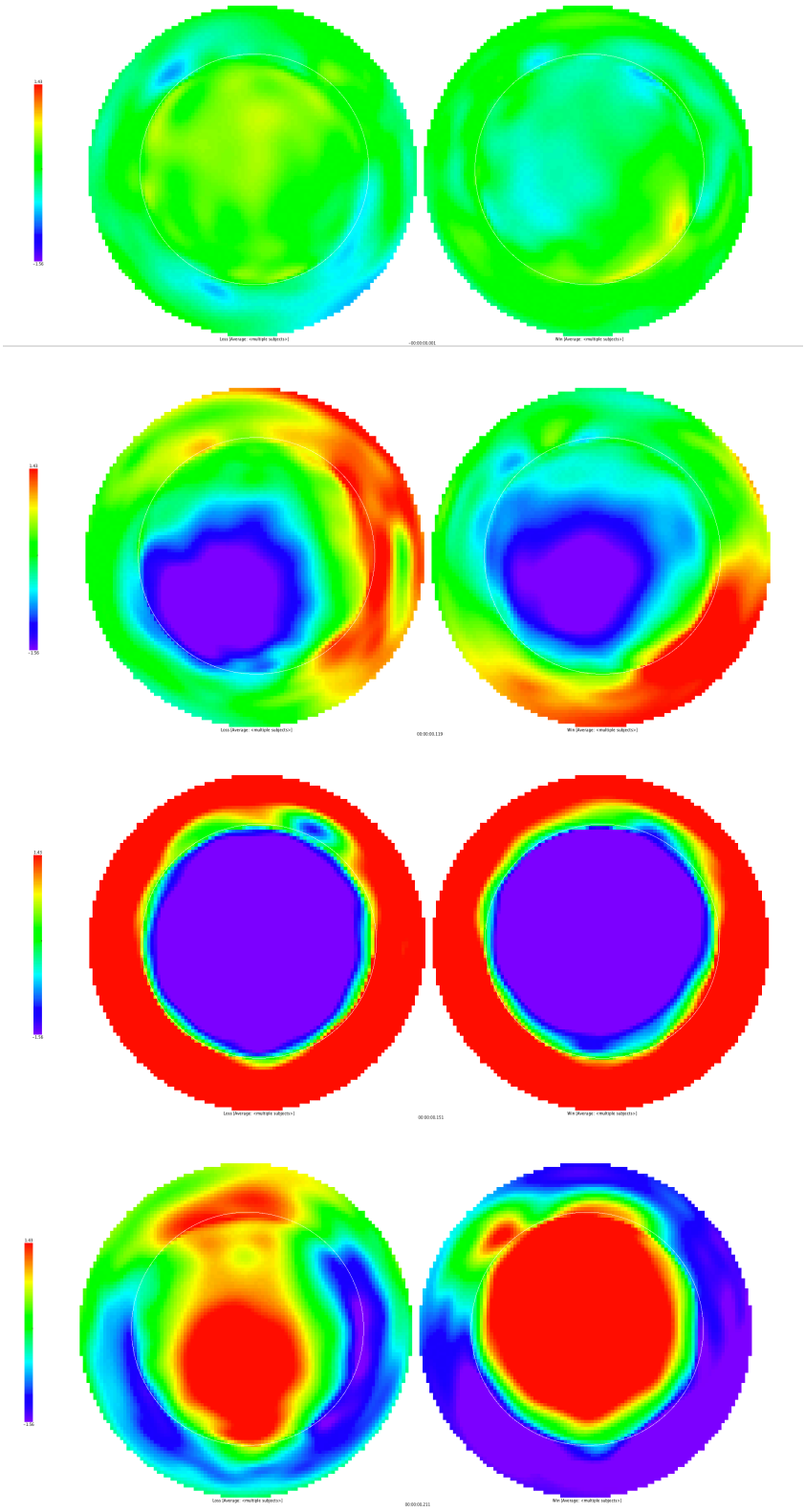


Figure 3.7

Topo Map 0, 120, 150, and 210 ms Post Feedback Heavy Drinkers

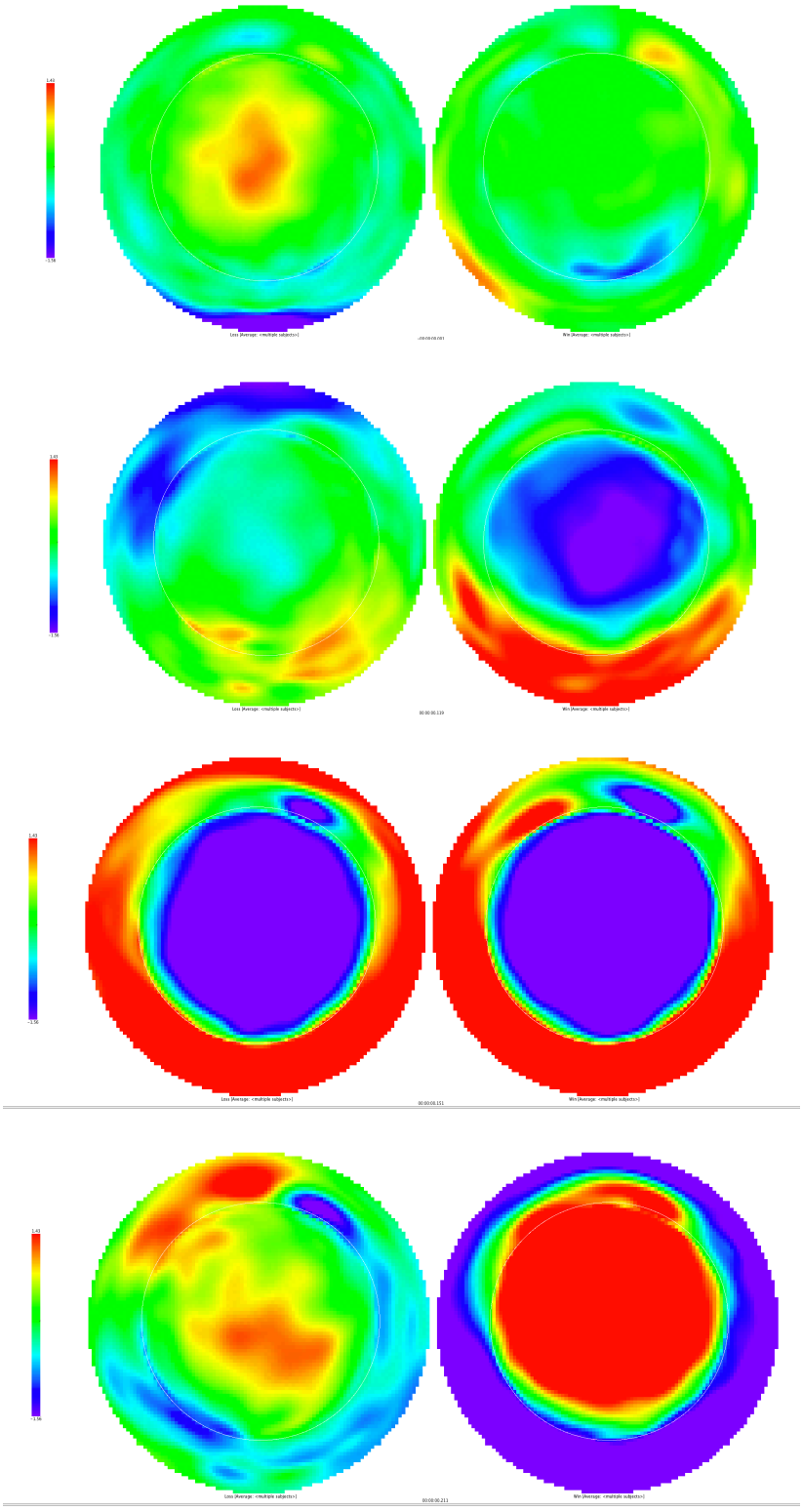


Figure 3.8

Topo Map 0, 120, 150, and 210 ms Post Feedback Total Sample

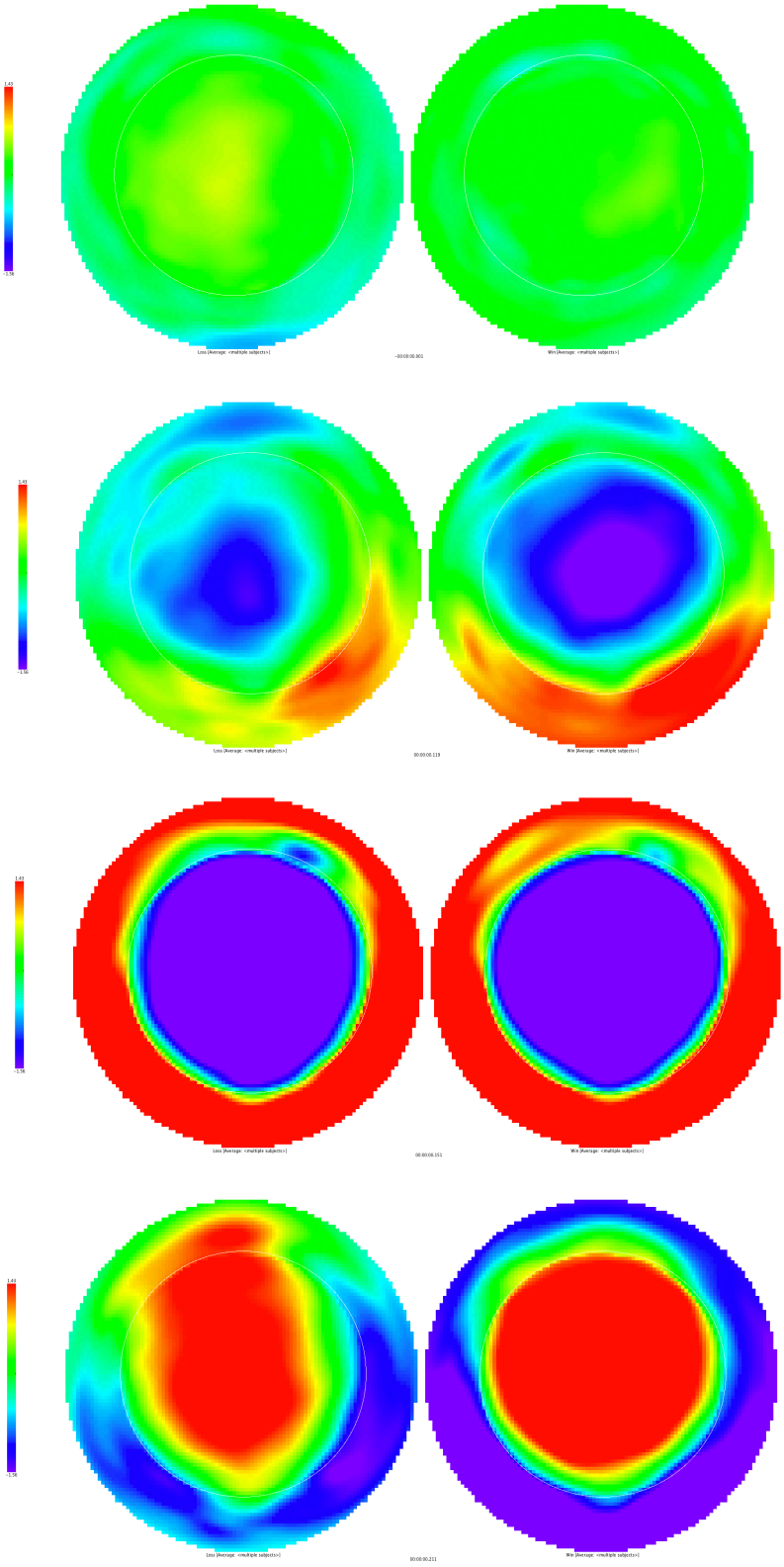


Figure 3.9

FRN Wave (electrode 23) Post Feedback Non-Binge Drinkers

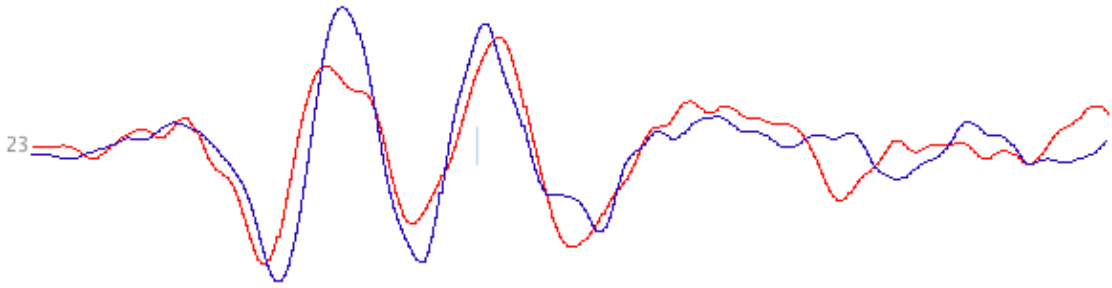


Figure 3.10

FRN Wave (electrode 23) Post Feedback Binge Drinkers

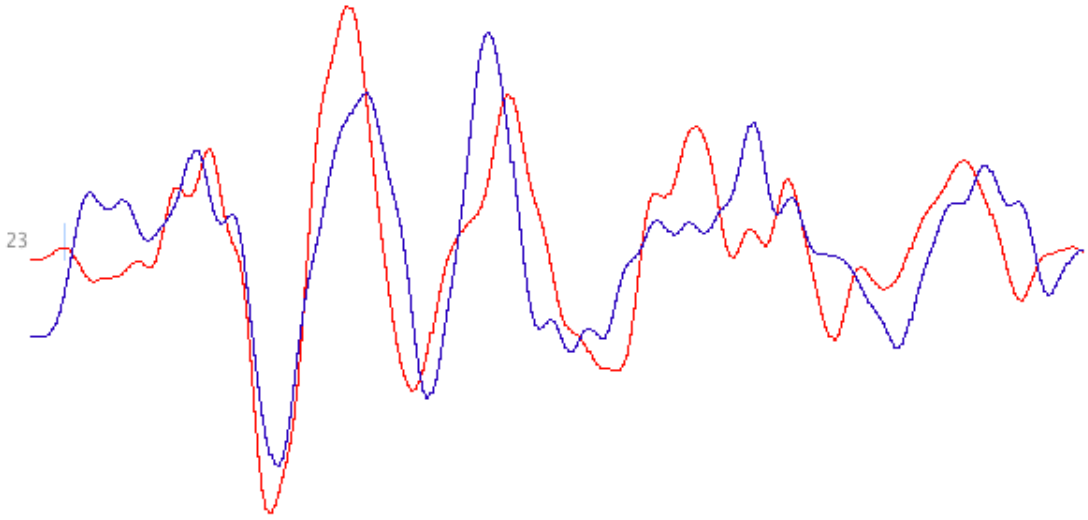


Figure 3.11

FRN Wave (electrode 23) Post Feedback Heavy Drinkers

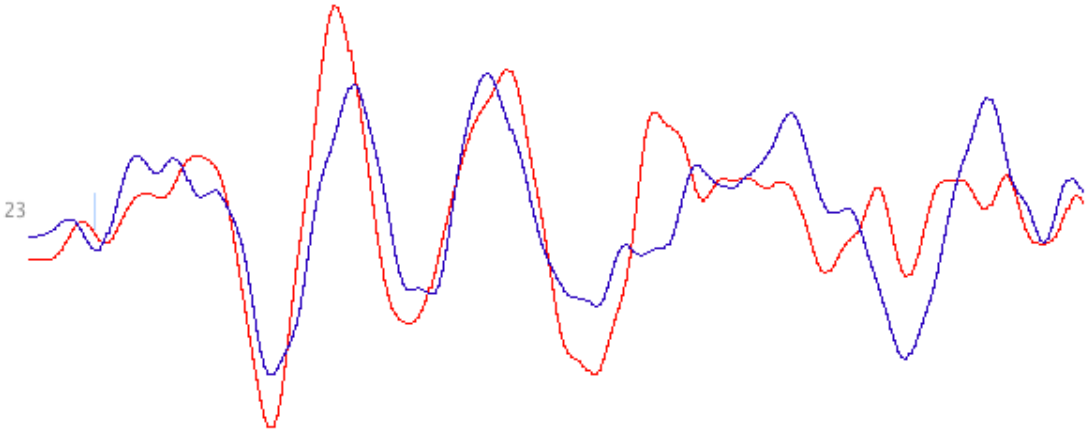
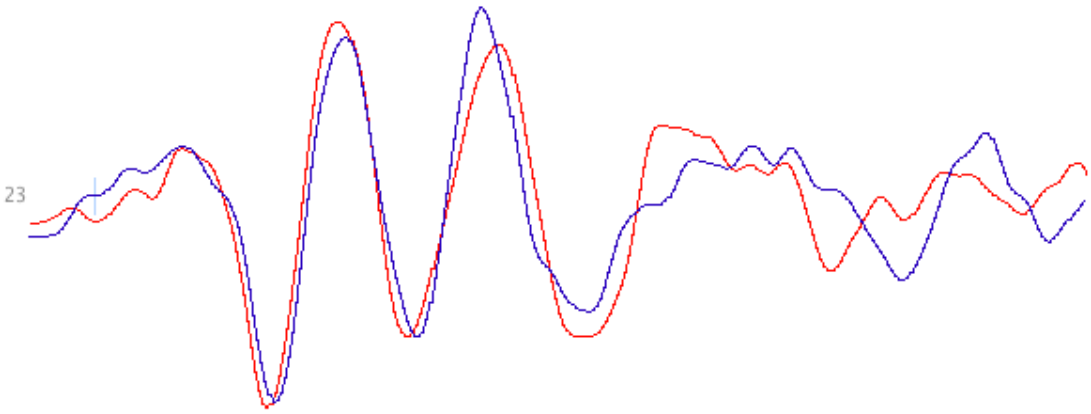


Figure 3.12

FRN Wave (electrode 23) Post Feedback Total Sample



Primary Analyses

Hypothesis 1. A one-way Analysis of Covariance (ANCOVA) was used to examine the FRN peak-to-peak amplitude differences among non-binge, binge, and heavy drinkers controlling for WASI FSIQ. It was hypothesized that college students classified as heavy drinkers would exhibit the smallest FRN amplitudes, followed by binge drinkers, and with non-binge drinkers exhibiting the largest FRN amplitudes. The results indicate no main effect of drinking category on FRN amplitude when controlling for WASI FSIQ, $F(2, 24) = 1.86$, $p = .177$. The adjusted means across the three groups are as follows: 6.98 μV (non-binge drinkers), 5.40 μV (binge drinkers), and 5.24 μV (heavy drinkers). This suggests no difference in neurocognitive response to negative outcomes among different categories of drinkers. Please see Figure 3.13 for the profile plot.

Hypothesis 2. Stepwise linear regression was used to examine the relationship between IGT performance and FRN amplitude in the total sample. IGT performance served as the dependent variable. In step 1, FSIQ was entered. In step 2, FRN amplitude was entered. It was hypothesized that, controlling for WASI FSIQ, a positive linear relationship would exist between FRN amplitude and number of advantageous decisions on the IGT. Contrary to the hypothesis, no relationship (controlling for WASI FSIQ) was found between FRN amplitude and IGT advantageous decisions, $R^2\Delta < .001$, $\beta = .08$, $F\Delta(1, 25) = .02$, $p = .967$. Please see Table 3.4 for regression statistics.

Figure 3.13

Hypothesis 1: One-Way ANCOVA

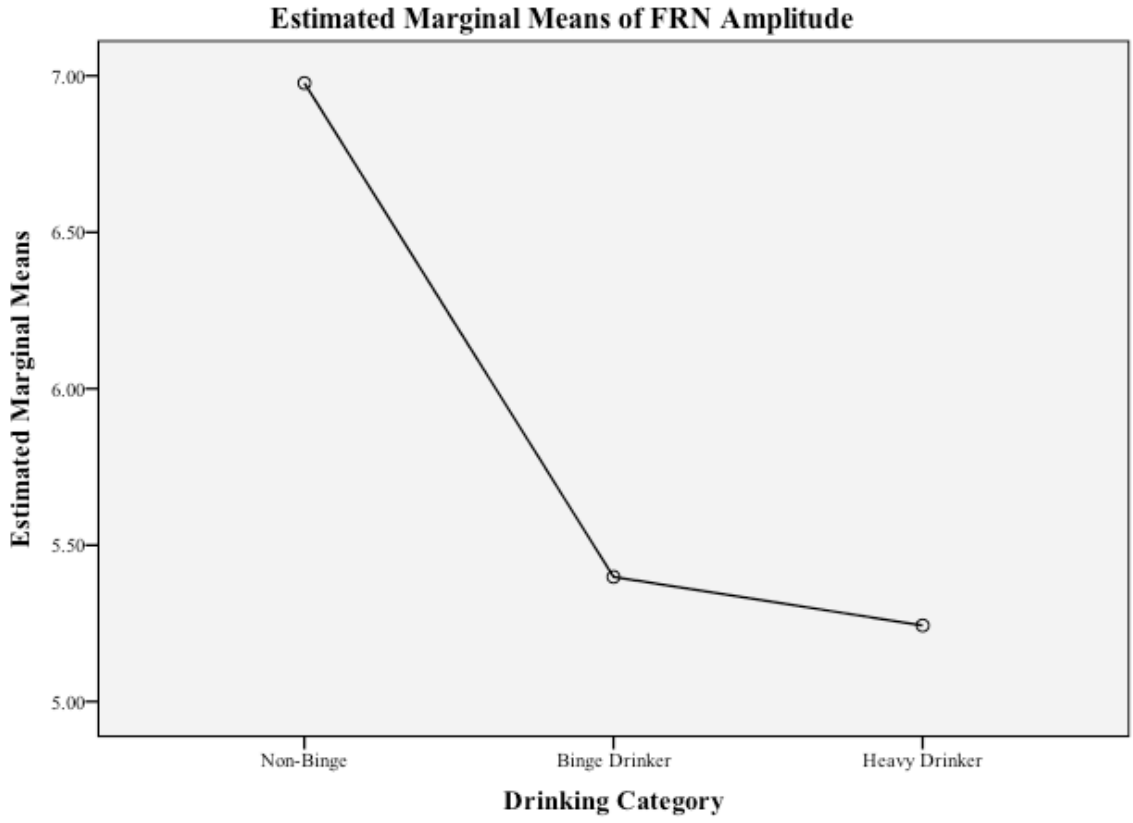


Table 3.4

Hierarchical Regression: Hypothesis 2

Step	Variable	R^2	ΔR^2	ΔF	p
Step 1		.09	.09	2.50	.126
	WASI FSIQ				
Step 2		.09	<.001	.002	.967
	FRN Amplitude (in μV)				

Hypothesis 3. Stepwise linear regression was used to examine the relationship between age of drinking onset and FRN amplitude in the total sample. FRN served as the dependent variable. In step 1, FSIQ was entered. In step 2, age of drinking onset was entered. It was hypothesized that, controlling for WASI FSIQ, a positive linear relationship would exist between FRN peak-to-peak amplitude and age of drinking onset. Contrary to the hypothesis, no relationship (controlling for WASI FSIQ) was found between FRN peak-to-peak amplitude and age of onset, $R^2\Delta = .09$, $\beta = .31$, $F\Delta(1, 25) = 2.82$, $p = .106$. Please see Table 3.5 for regression statistics.

Table 3.5

Hierarchical Regression: Hypothesis 3

Step	Variable	R^2	ΔR^2	ΔF	p
Step 1		.00	.00	.01	.935
	WASI FSIQ				
Step 2		.10	.10	2.82	.106
	Age of Onset				

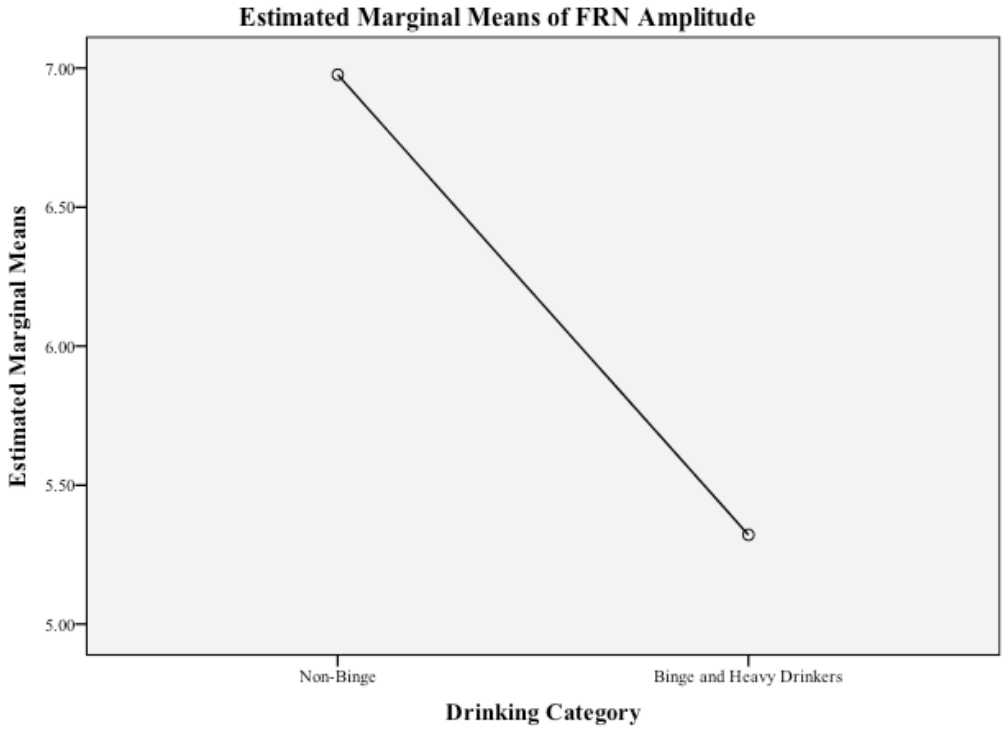
Post-Hoc Analyses

Given the adjusted mean FRN amplitude values, the binge and heavy drinker groups were collapsed ($n = 18$). The results indicate a main effect that approached significance of drinking category on FRN amplitude when controlling for WASI FSIQ, $F(1, 25) = 3.85$, $p = .061$. The adjusted means across the three groups were as follows: 6.97 μV (non-binge drinkers), and 5.32 μV (binge and heavy drinkers). This indicates

that non-binge drinkers have a greater neurocognitive response to negative outcomes than do binge and heavy drinkers. Please see Figure 3.14 for the profile plot.

Figure 3.14

ANCOVA collapsed across Binge and Heavy Drinker Groups



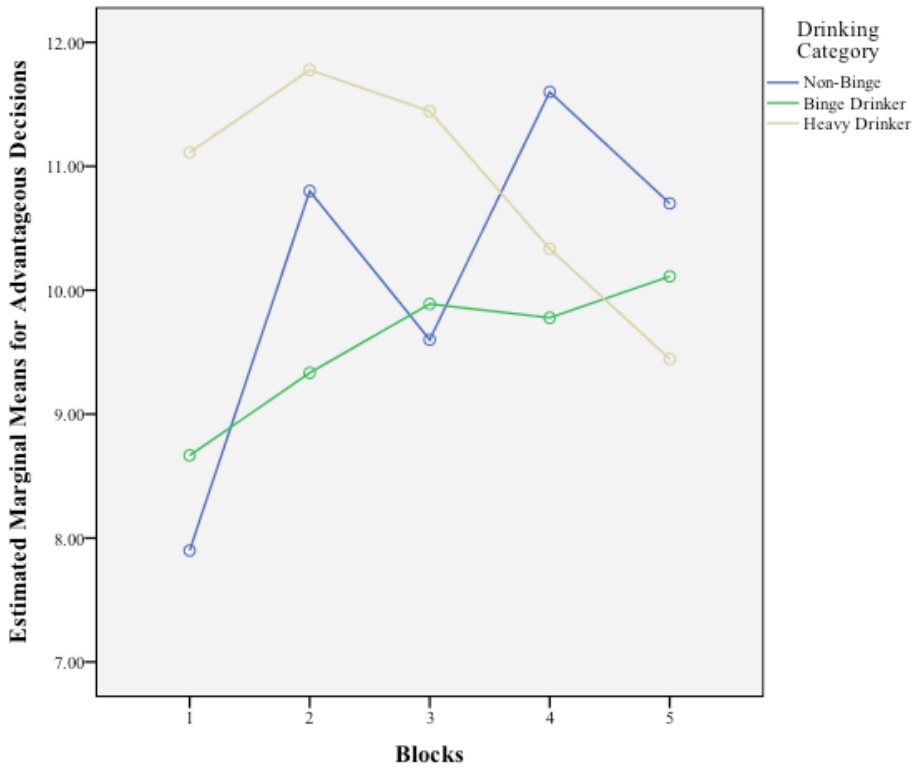
Relationships between additional indices of hazardous drinking (AUDIT and RAPI totals) and FRN amplitude were examined. Zero-order correlations reveal a significant negative linear relationship between AUDIT total and FRN amplitude (r = -.38, .048). This indicates that greater hazardous alcohol consumption is associated with lower neurocognitive response to negative outcomes.

Consistent with how it is typically examined in the literature (e.g., Bechara et al., 2001), IGT performance was broken up into 5 blocks of 20 trials each (Table 3.1; Figure

3.15). A repeated measures ANCOVA (using WASI FSIQ as the covariate) was used to examine individual differences in IGT performance across blocks and determine if performance across blocks differed among drinking groups. No differences were found in advantageous decisions across blocks, $F(4, 96) = .36$, $p = .837$. Furthermore, the IGT block x drinking category x WASI FSIQ interaction was not significant, $F(12, 96) = .99$, $p = .468$. See Figure 3.15 for estimated marginal means. Each block was also entered into its own hierarchical regression model, with FRN peak-to-peak amplitude as the criterion, WASI FSIQ entered into step 1 (covariate), and block performance into step 2. No significant relationships were found (all *p's* > .05).

Figure 3.15

IGT Advantageous Decisions Across Blocks



Post Hoc Power Analyses

Post hoc power analysis revealed adequate power (1.0) to detect an effect for hypothesis 1, and therefore the null findings are not due to inadequate power. However, power analyses for hypothesis 2 and 3 suggest inadequate power (.07 and .48, respectively). The necessary sample size to detect an effect would be $N = 6184$ at 80% power for hypothesis 2 and $N = 71$ at 80% power for hypothesis 3.

Chapter 4: Discussion

To date, few studies have examined the relationship between alcohol use and the ERN/FRN, and no studies have examined it among college binge drinkers. Furthermore, this is the first study to date to test the RL theory of the ERN/FRN in college binge drinkers using the IGT as an index of RL. The overarching aim of the present study was to better understand neurocognitive mechanisms associated with learning from negative outcomes as they relate to binge drinking among college students, and study hypotheses were generated based on the extant literature. First, it was hypothesized that there would be differences in FRN amplitude among three different categories of drinkers: non (fewer than 1 binge drinking episode during each the previous 3 months), binge (at least 1 binge drinking episode during each the previous 3 months), and heavy (at least 5 binge drinking episodes during each of the previous 3 months), such that non-drinkers would have the largest FRN peak-to-peak amplitude and heavy drinkers would have the smallest. Second, it was postulated that (controlling for WASI FSIQ) there would be a positive linear relationship between IGT performance (i.e., number of advantageous decisions on task) and FRN peak-to-peak amplitude. Lastly, it was predicted that, controlling for

WASI FSIQ, there would be a positive linear relationship between FRN peak-to-peak amplitude and age of drinking onset.

Findings revealed no FRN peak-to-peak amplitude differences among non-binge, binge, and heavy drinkers. Based on visual inspection of the marginal mean differences in FRN peak-to-peak amplitude among non-binge, binge, and heavy drinkers (see Figure 14), binge and heavy drinkers were collapsed to form a single group. The marginal mean difference between non-binge and the collapsed binge and heavy drinker groups approached significance ($p = .061$). Additionally, AUDIT and RAPI total data were collected as part of the study, though no a priori hypotheses were generated regarding its relationship with FRN peak-to-peak amplitude or IGT performance. Zero-order correlations revealed a negative linear relationship between AUDIT total and FRN peak-to-peak amplitude. No relationship (controlling for WASI FSIQ) was found between FRN peak-to-peak amplitude and IGT performance or age of drinking onset.

Hypothesis 1

It was hypothesized that non-binge, binge, and heavy drinkers would exhibit differential FRN peak-to-peak amplitudes when FSIQ was controlled for. Specifically, it was expected that non-binge drinkers would exhibit the largest FRN peak-to-peak amplitudes, whereas heavy drinkers would have the smallest FRN peak-to-peak amplitudes. This hypothesis was not supported, such that no group differences in FRN peak-to-peak amplitude were found. However, when binge and heavy drinking groups were collapsed, differences between non-binge and the binge/heavy drinkers approached significance.

As outlined in the introduction, the literature to date has focused on the acute effects of alcohol consumption on the ERN/FRN. To that end, the initial hypothesis is largely inconsistent with the extant data. Acutely, findings have shown that consumption of alcohol relates to a blunted ERN/FRN amplitude (Easdon et al., 2005; Ridderinkhof et al. 2002; Yeung & Cohen, 2006). Chronically, treatment naïve alcoholics with greater family density (FHD) of alcohol problems have also displayed reduced FRN amplitudes (Fein & Chang, 2007). However, recently emerging data are relatively consistent with the present findings. Euser and colleagues (2011) had participants complete a risk-taking task after consuming either a moderate dose of alcohol or a placebo and found no difference in FRN amplitude between groups. Similarly, ERN/FRN amplitudes have been found unrelated to externalizing proneness (Bernat, Nelson, Steele, Gehring, & Patrick, 2011). Externalizing proneness has been related to substance abuse and the behavioral activation system (i.e., BAS) and is believed to reflect dopaminergic responsiveness (Boksem, Tops, Wester, Meijman, & Lorist, 2006). Coupled with the present findings, these data suggest several things. First, the FRN-alcohol literature appears to support the acute effects of alcohol consumption on the FRN, but little (outside of the present study) is known about the chronic effects of alcohol on this signal. It is possible that chronic binge drinking during college, which most students "mature out" of (Fillmore et al., 1988; Johnstone et al., 1996), does not have deleterious effects on the FRN. Second, the previous point draws into question if the FRN serves as an endophenotype as has been postulated (Olvet & Hajcak, 2008).

When the binge and heavy drinking groups were collapsed, differences in FRN peak-to-peak amplitude between non-binge drinkers and the collapsed binge and heavy

drinking group approached significance (p = .061). Interpreting this data with caution, it may indicate that a blunted FRN peak-to-peak amplitude is associated with having at least one binge drinking episode per month (i.e., reaching blood alcohol concentration of at least .08) and is not further effected by an upwards of five binges per month (i.e., heavy drinking). These findings are more consistent with the literature previously discussed (e.g., Easdon et al., 2005; Ridderinkhof et al., 2002; Yeung & Cohen, 2006). That is, the error-processing system is affected by alcohol use, such that greater levels of chronic use map on to a reduced response to error and, in line with RL theory, reduced dopaminergic functioning. When interpreted in conjunction with the null findings of hypothesis 3 (i.e., no linear relationship between age of drinking onset and FRN controlling for WASI FSIQ), it suggests that the blunted FRN amplitude is not likely due to deleterious structural and dopaminergic functioning effects from the onset of alcohol use during adolescence, and that the most affected individuals tend to fall into the binge and heavy drinking groups. Moreover, no differences in IGT performance were found among drinking groups, which suggests that differences in FRN peak-to-peak amplitude do not map on to behavioral differences in this sample. This would be consistent with the notion that neurocognitive deficits are apparent prior to behavioral deficits (Maurage et al., 2009). Two important factors remain to be identified: a) if behavioral deficits emerge at a later point, and b) if the FRN peak-to-peak amplitude in the collapsed binge and heavy group would augment after a sustained period of reduced drinking.

Also of note, FRN peak-to-peak amplitude was negatively correlated with AUDIT total and unrelated to RAPI total. As previously discussed, the AUDIT assesses a broad range of hazardous alcohol criteria, including quantity/frequency, binge behavior, and

alcohol-related negative consequences, whereas the RAPI only assesses negative alcohol-related consequences. It would have been expected that, consistent with RL theory, that FRN peak-to-peak amplitude would relate to both AUDIT and RAPI or, at the very least, only RAPI. Mean RAPI and AUDIT scores were largely consistent with previous studies examining binge drinking in the same population (Wahlstrom, McChargue, & MacKillop, 2012) and greater than other college student populations (e.g., Carey & Correia, 1997; Kokotailo, Egan, Gangnon, Brown, Mundt, & Fleming, 2004; Neighbors, Lee, Lewis, Fossos, & Larimer, 2007). In line with other findings, RAPI may reflect a construct similar to IGT performance (i.e., negative consequences experienced and learning from negative outcomes), even though the two measures are unrelated. AUDIT data may be more consistent with the timeline followback (the method used to determine drinking categories), which is based on quantity and frequency.

Hypothesis 2

The second hypothesis that a positive linear relationship, controlling for WASI FSIQ, would exist between IGT performance and FRN peak-to-peak amplitude was not supported by the data, which is contrary to previous findings (e.g., Frank et al., 2005). In order to best understand the absence of this relationship, IGT performance was examined in greater detail. IGT performance was divided into five blocks of twenty trials each to determine if learning of deck contingencies occurred over the course of the task. No significant differences were found in performance among the five blocks when each of the three drinking groups (non-binge, binge, heavy) were examined separately nor for the total sample. Moreover, mean number of advantageous choices ranged from 9.18 (block 1) to 10.64 (block 2), suggesting approximately equal choices from advantageous and

disadvantageous decks across the task. This is particularly noteworthy for the non-binge drinking group, as previous studies have found that the number of advantageous decisions in later blocks (particularly 4 and 5) should be significantly greater than in earlier blocks (Suhr & Hammers, 2010).

There are several reasons that learning on the IGT may not have occurred within the present sample. First, these null findings map on to other data indicating that participants failed to identify and select from the advantageous decks (Li, Lu, D'Argembeau, Ng, & Bechara, 2010). It could also be that the prefrontal cortex is still developing during college or decision-making skills have not been fully refined (Casey et al., 2000; Crone and van der Molen, 2004; Gogtay et al., 2004; Hooper et al., 2004).

Indeed, other explanations are plausible. One study found that not only did individuals with less than a high school education show greater learning than first and second year university students throughout the IGT, they also outperformed them in terms of selections from advantageous decks during blocks 1, 3, 4, and 5 (Evans, Kemish, & Turnbull, 2004). The authors suggested that low motivation among the university students might have contributed to poor performance. In the present sample, participants were only able to earn up to $5.00 on the task and received their research credit regardless of performance. A second possibility is that explicit knowledge versus emotion-based learning may account for the difference. That is, individuals whose need rely on explicit knowledge more frequently (i.e., college students) may find a task without explicit contingences challenging. Both explanations could potentially explain the absence of learning across participants.

Some data also suggest that college students show preference for disadvantageous decks (Caroselli, Hiscock, Scheibel, & Ingram, 2006), perhaps due higher stakes and thus, greater arousal. Other studies actually show that following a large loss, participants tended to repeat the risky option (Hewig, Trippe, Hecht, Coles, Holroyd, & Miltner, 2007; Yeung & Sanfey, 2004). On the other hand, previous evidence suggests that ERP deficits are detected before psychological or behavioral impairments are exhibited (Maurage et al., 2009). This is consistent with the current data that has found FRN peak-to-peak amplitude differences approach significance between non-binge and binge drinkers (when binge and heavy drinkers are collapsed into one group), such that non-binge drinkers exhibit greater peak-to-peak FRN amplitudes. Thus, this would support the notion that there are neurocognitive (FRN peak-to-peak amplitude) but not behavioral (IGT performance) differences in the present sample.

It has also been suggested that disparate processes modulate prediction of the outcome and behavioral adjustment following feedback. Chase and colleagues (2011) administered a reversal learning task during which contingencies changed on several occasions. Participants were instructed not to alter their behavior until they were sure that the rules had changed. Findings indicated that FRN amplitude was related to the reward prediction error (i.e., outcome from a decision being worse than expected), however, it did not relate to behavioral modifications. Instead, the P3 amplitude was associated with changes in behavior. Thus, it is suggested that the FRN may reflect adjustment of decision values whereas the P3 reflects the actual alteration of future decisions.

There are several additional facets of the IGT that are worthy of discussion, particularly the IGT data as it relates to other study variables. Zero-order correlations and

one-way ANOVAs revealed the absence of relationships with study variables and differences in performance across drinking categories. First, IGT performance did not relate to age of drinking onset, which is consistent with other findings among college students (Goudriaan et al., 2007). Given that the prefrontal cortex, the area of the brain responsible for executive functions (including decision-making) continue to develop into early adulthood, it was expected that earlier onset of alcohol use may impact development of these brain areas and, in turn, result in poorer decision-making. Interestingly, no relationship was found between IGT performance and RAPI total score, a measure of alcohol-related problems, though RAPI total scores did differ significantly between non-binge and heavy drinkers, as well as non-binge and a group that collapsed binge and heavy drinkers. This is certainly supported by a robust body of literature relating binge drinking to negative alcohol-related outcomes (Borsari, Neal, Collins, & Carey, 2001; Wechsler et al., 1998), yet raises questions related to the underlying mechanisms.

Second, IGT performance was not related to WASI FSIQ estimates. Some data reveal a relationship between IGT performance and Shipley FSIQ (Barry & Petry, 2008) and the American College Test (ACT) scores (Goudriaan et al., 2007), a standardized test for high school achievement that has been shown to be strongly related to Wechsler IQ test performance (Baade & Schoenberg, 2004). Correlation values coupled with post hoc power analyses suggest that findings from the present study may reach statistical significance if examined in a larger sample.

Hypothesis 3

It was predicted that, controlling for WASI FSIQ, there would be a positive relationship between age of drinking onset and FRN peak-to-peak amplitude. The data did not support this hypothesis, such that no relationship was found between age of drinking onset and FRN peak-to-peak amplitude. To date, no other studies have examined this relationship; however, the literature examining neurobiological effects of adolescent binge drinking provides a framework in which to understand the present findings. Previous findings indicate that the onset of alcohol use during adolescence can have deleterious effects on brain structural and neurochemical development (Guerri & Pascual, 2010), as well as cognitive functioning (Stephens & Duka, 2008). Animal studies have also revealed that the onset of alcohol use during adolescence results in reduced dopamine gene expression (Coleman, He, Lee, Styner, & Crews, 2011). Other evidence shows that brain areas critically involved in RL continue to develop into late adolescence (Crone, Zanolie, Leijenhorst, Westenberg, & Rombouts, 2008).

Taken together, previous findings suggest that a relationship would have been expected between age of drinking onset and FRN peak-to-peak amplitude. However, several factors may explain the null findings. First, many studies that have examined neurocognitive impairment among adolescent binge drinkers reveal younger ages of drinking onset than were found in the present study. For example, data examining age of onset for low- and high-risk children (based on family history of alcohol dependence) found a mean age of 15.2 (+/- 1.2) for high-risk children and 16.5 (+/- 1.2) for low-risk children (Hill, Shen, Lowers, & Locke, 2000). The mean age of drinking onset in the present sample ranged from 16.3 (*SD* = 0.50) for heavy drinkers to 17.3 (*SD* = 0.40) for

non-binge drinkers. Given the dynamic neurocognitive changes occurring during adolescence, any delay in age of drinking onset may ameliorate the negative neurocognitive effects. Additionally, it's possible that the pre-college drinking patterns of these individuals were of low quantity and frequency compared to other samples.

Limitations

Several limitations are worthy of discussion. First, the sample included only men, and therefore results cannot be generalized to college women, particularly given the identified gender differences in ERN amplitude (Larson, Mikle, & Clayson, 2011), decision-making (Bolla, Eldreth, Matochik, & Cadet, 2004), and binge drinking (O'Malley & Johnston, 2002; Wechsler et al., 2000; Wechsler et al., 1994; Wechsler et al., 1995; Weitzman et al., 2003). Findings also cannot be generalized to cultural groups outside of those of European descent, as 89% self-identified as White. Second, though SCL-90-R scores ensured that the sample was free of severe psychopathology, it does not account for the influence of other personality traits. For example, those high on neuroticism have been found to be able to alter the amplitude of the ERN, whereas those high on consciousness did not show altered ERN amplitudes (Pailing & Segalowitz, 2004). Third, though a priori power analyses were conducted to determine the sample size for the present study, null findings for hypotheses 2 and 3 may be due, in part, to lack of power. In order to better determine which effects are present, a larger sample size would be beneficial. Fourth, given that learning did not occur on the IGT, it is unclear if it is a valid index of RL.

Future Research

Longitudinal examination of the FRN would address critical gaps in the extant literature and build upon the current findings. Previous studies have shown that other ERP components are affected by alcohol use over the course of nine months (Maurage et al., 2009). Most of the students in the present study were in their first or second year of college (mean age = 19.36), and thus the negative effects may occur over a longer period of chronic binge drinking. Furthermore, given that ERN/FRN amplitude is affected by motivation (Hajcak, Holroyd, Moser, & Simons, 2005), providing increased monetary incentives for study completion and success on the IGT may be helpful in addressing both the absence of learning on the IGT and FRN differences across drinking groups. Use of a different index of RL (e.g., Balloon Analog Risk Task) may also be beneficial given that learning did not occur in the present study. Lastly, examining these effects in adolescents with varying degrees of alcohol use may shed light on the developmental effects of alcohol on the FRN. Following these adolescents across time would also help clarify whether the FRN is stable across time (trait) or changes (state).

Summary

Binge drinking and associated negative consequences remain problematic among college students despite increased attention toward the implementation of efficacious interventions (Hingson, 2010). Indeed, this suggests that a subset of college students continue to consume alcohol in a harmful manner despite having experienced negative consequences stemming from their use. This continued use despite negative consequences is supported by the literature (e.g., Jennison, 2004), though the etiology of this behavior has not been fully elucidated. However, data have implicated the role of the

midbrain dopamine system in both binge drinking (Wahlstrom, McChargue, & MacKillop, 2012; Vaughn, Beaver, DeLisi, & Howard, 2009) and alcohol dependence (Berggren et al., 2006). To that end, recent conceptualizations of the role of dopamine in addiction suggest that it reflects outcomes from decisions to either shape future behavior toward or away from certain decisions (Holroyd & Yeung, 2004). As such, the overarching goal of the present study was to better understand neurocognitive mechanisms associated with learning from negative outcomes as they relate to binge drinking among college students.

Event-related potentials (ERPs), specifically the error- and feedback-related negativities (ERN/FRN) serve as an index to objectively measure response to negative outcome. The ERN/FRN occurs when an outcome from a decision is worse than expected, and is believed to reflect midbrain dopamine activity. Furthermore, this neural response is believed to shape behavior away from disadvantageous outcomes, and greater amplitude of this signal relates to better learning. The ERN/FRN is particularly relevant among college binge drinkers because it is negatively affected by alcohol consumption and is involved in reinforcement learning.

Reinforcement learning (RL) is based upon the premise that the goal of any organism is to maximize rewards and minimize punishment, which is modulated via the midbrain dopamine system. When alcohol is consumed, midbrain dopamine activity is altered and all outcomes from decisions are interpreted as advantageous. Across time, this strengthens the perceived reward of the drug and the discounting of negative outcomes associated with consumption. Because the ERN/FRN signal is blunted, or even absent,

future drinking behavior is not altered accordingly. RL theory has yet to be examined among college binge drinkers.

The Iowa Gambling Task (IGT), a widely used measure of decision-making, is an ideal index of RL because it requires participants to use outcomes from decisions to maximize future rewards and minimize punishments. There are 4 decks of cards, 2 "good" decks and 2 "bad" decks, and participants are required to make 100 selections. The "good" decks yield small immediate gains and result in long-term gains, whereas the "bad" decks result in large immediate gains yet result in long-term losses. Healthy controls tend to sample from all decks and then shift their strategy to choose from the "good" decks. However, alcohol-dependent and binge drinking individuals do not shift their strategy as the task progresses. This poor performance is believed to map on to delay discounting (i.e., preference for immediate rather than delayed rewards), hypersensitivity to reward, and insensitivity to negative consequences.

The overall aim of the present study was to examine FRN differences among varying degrees of college binge drinkers. As such, the first aim was to examine FRN amplitude differences among non-binge, binge, and heavy drinkers. It was predicted that non-binge drinkers would have the largest FRN amplitudes and heavy drinkers would have the smallest. The second aim was to examine the relationship between FRN and decision-making. It was hypothesized that there would be a positive linear relationship between advantageous decisions on the IGT and FRN amplitude. Last, the relationship between FRN and age of drinking onset was examined. It was predicted that there would be a positive linear relationship between FRN amplitude and age of drinking onset.

The sample consisted of 30 male college students. Ten participants were non-binge drinkers (< 1 binge each month during past 3 months), ten met criteria for binge drinking (1-4 binges each month during past 3 months), and ten met criteria for heavy drinking (> 5 binges each month during past 3 months). Participants were not eligible to participate if they were currently using nicotine or other drugs, had a history of traumatic brain injury, neurological disorder, or loss of consciousness for twenty minutes or longer, were currently taking prescription medication, or were older than 23. Participation took place across two sessions. The first session included an assessment of alcohol use, administration of the WASI to obtain a FSIQ estimate, and completion or paper-pencil questionnaires. The second session included completion of the IGT with concomitant FRN data collection. All participants gave written informed consent and the study was approved by the University of Nebraska-Lincoln Institutional Review Board.

ERP data was processed using NetStation 4.1.1 (EGI, Inc.). Only wins that preceded a loss were examined so to allow for equivalent number of win and loss segments. Segments were averaged for the total sample and separately for each group (non-binge, binge, and heavy). Based on visual inspection and the literature, the FRN was deemed to occur between 120 and 210 ms post feedback, with the most negative point during this time frame being considered FRN amplitude. Peak-to-peak was determined by subtracting the most positive peak preceding the FRN from the FRN. Study aims were tested using one-way ANCOVAs and stepwise linear regression.

The sample consisted of primarily first and second year college students (mean age = 19.36) and the majority were White (89.3%). AUDIT totals (mean = 12.00) indicate that as a whole, the sample consumes alcohol in a harmful manner. One-way

ANOVAs revealed differences across drinking groups in AUDIT and differences between non-binge and heavy drinkers on RAPI totals. No other group differences were found on study variables. Pearson's correlations revealed correlations between age of drinking onset and AUDIT total, age of onset and RAPI total, AUDIT total and RAPI total, and AUDIT total and FRN total. All correlations were negative except between AUDIT and RAPI total.

WASI FSIQ was controlled for in all primary analyses. Using one-way ANCOVA, no differences were found in FRN peak-to-peak amplitude among non-binge, binge, and heavy drinkers. Furthermore, hierarchical regression revealed no linear relationship between FRN amplitude and number of advantageous decisions on the IGT or age of drinking onset.

Given that none of the primary hypotheses were supported, post hoc analyses were conducted. Based on visual inspection of the adjusted FRN amplitude values, binge and heavy drinking groups were collapsed and differences in FRN peak-to-peak amplitude between non-binge and the collapsed group approached significance (p = .061). Relationships between FRN peak-to-peak amplitude and AUDIT and RAPI totals were also examined. Pearson's correlations revealed a negative linear relationship between AUDIT total and FRN peak-to-peak amplitude, but no relationship between RAPI total and FRN peak-to-peak amplitude. Lastly, IGT performance was examined by block (i.e., 20 trials). No differences were found on advantageous choices across blocks and there was no block x drinking category x WASI FSIQ interaction. No significant relationships were found between each of the five blocks and FRN peak-to-peak amplitude.

The absence of differences among non-binge, binge, and heavy drinkers in FRN peak-to-peak amplitude is contrary to what the extant literature would suggest. Though no study to date has examined FRN differences among college binge drinkers, previous findings have shown that acute consumption and treatment naïve alcoholics exhibit blunted FRN amplitudes (e.g., Easdon et al., 2005; Ridderinkhof et al., 2002; Yeung & Cohen, 2006). On the other hand, other studies have found no difference in FRN amplitude following alcohol administration (Euser at al., 2011). Taken together, it is possible that binge drinking during college does not negatively impact the FRN and dopaminergic functioning. However, when binge and heavy drinking groups were collapsed findings approached significance, suggesting that at least 1 binge per month is the critical point at which this error-processing system is affected and more frequent binging does not exacerbate it. Findings also revealed a relationship between FRN amplitude and AUDIT total but not RAPI total. This is inconsistent with RL theory and suggests that in this population, FRN amplitude may be affected more by drinking quantity/frequency than negative outcomes.

RL theory would suggest that IGT performance (the present study's measure of RL) and FRN amplitude would be related; however, no relationship was found between these variables. Examination of IGT performance in greater detail revealed no differences in relationships across the 5 blocks (20 trials each) and showed, on average, no learning across the task. While this would be expected in the binge and heavy drinking groups, learning was expected to have occurred in the non-binge drinking groups. This may be explained by the relatively immature PFCs in the sample, low motivation on the task, or

preference for disadvantageous decks. Other data suggest that electrophysiological impairments may be detected before behavioral deficits.

No relationship was also found between FRN peak-to-peak amplitude and age of drinking onset. Though no studies have previously examined this relationship, alcohol is known to have negative effects on brain structure, neurochemistry, and cognitive functions, particularly during adolescence. However, other studies have revealed younger ages of drinking onset than were found in the present study, which may temper negative effects.

The findings of the present study are tempered by several limitations. The sample consisted of only college men who were predominantly White (89%), and therefore findings cannot be applied to women or generalized to other cultural groups. Furthermore, findings do not consider other factors that may mediate relationships, including personality traits. Lastly, it is unclear if the IGT was a valid index of RL and the sample size was relatively small which, despite a priori power analyses, may have resulted in the absence of findings. Future research would benefit from examining FRN amplitude longitudinally across adolescence/young adulthood, utilizing different indices of RL, and increasing task performance motivation by increasing the monetary compensation amount.

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