Binge Drinking’s Cognitive and Emotional Correlates:
A Multi-Definitional Investigation

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DISsertation
Submitted as partial fulfillment of the requirements
For the degree of Doctor of Philosophy in Psychology
in the Graduate College of the
University of Illinois at Chicago, 2014

Chicago, Illinois

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This dissertation is dedicated to my family and friends, without whom my Ph.D. could not have been accomplished. Thank you for getting me through the past six years.
ACKNOWLEDGEMENTS

I would like to thank my dissertation committee – Jon Kassell, Robin Mermelstein, Neil Pliskin, Colleen Corte, and Raul Gonzalez – for their support and assistance. They provided valuable guidance that helped me accomplish my research goals. Thank you to the members of the Substance Use Research Lab at UIC – Megan Conrad, Grace Giedgowd, and Natania Crane – for their help in data collection and database preparation.

ARB
# TABLE OF CONTENTS

## I. INTRODUCTION

A. Background ............................................................. 1
B. What is Binge Drinking? ............................................. 2
C. Kindling Theory of Alcohol Withdrawal .......................... 4
D. Binge Drinking and Brain Damage ............................... 7
E. Negative Affect .......................................................... 12
F. Alcohol Expectancies ............................................... 14

## II. THE PRESENT STUDY

A. Aims ................................................................. 15
B. Hypotheses ............................................................ 18

## III. METHODS

A. Participants .......................................................... 19
B. Procedures ............................................................ 20
   1. Screening ......................................................... 20
   2. Study Visit ....................................................... 20
      2.1 Questionnaires ............................................... 21
      2.2 Cognitive Measures ......................................... 24
C. Data Analysis ........................................................ 27
   1. Preliminary Analyses ............................................ 27
   2. Primary Analyses ............................................... 28

## IV. RESULTS

A. Preliminary Analyses .............................................. 30
   1. Demographics .................................................... 30
   2. Drinking Habits and Other Drug Use .......................... 31
   3. Correlational Analyses .......................................... 34
B. Primary Analyses ................................................... 36
   1. Hypothesis 1. Executive Function and Self-Report Measures .... 36
   2. Hypothesis 2. National Institutes of Alcohol Abuse and Alcoholism Definitions .............................................. 41
      2.1 Hypothesis 2a. National Institutes of Alcohol Abuse and Alcoholism Dichotomous Definition of Binge Drinking ... 41
      2.2 Hypothesis 2b. National Institutes of Alcohol Abuse and Alcoholism Continuous Definition of Binge Drinking ..... 43

## V. DISCUSSION

A. Binge Drinking and Executive Function .......................... 46
B. Impulsivity ............................................................ 48
C. Iowa Gambling Task ................................................. 49
TABLE OF CONTENTS (continued)

<table>
<thead>
<tr>
<th>CHAPTER</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. Expectancies</td>
<td>50</td>
</tr>
<tr>
<td>E. Negative Affect</td>
<td>52</td>
</tr>
<tr>
<td>F. Binge Drinking Definitions</td>
<td>53</td>
</tr>
<tr>
<td>G. Binge Drinking and Withdrawal</td>
<td>54</td>
</tr>
<tr>
<td>H. Limitations</td>
<td>55</td>
</tr>
<tr>
<td>VI. CONCLUSIONS</td>
<td>57</td>
</tr>
<tr>
<td>CITED LITERATURE</td>
<td>58</td>
</tr>
<tr>
<td>VITA</td>
<td>69</td>
</tr>
</tbody>
</table>
LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>DESCRIPTION</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.</td>
<td>DEMOGRAPHIC AND ALCOHOL USE CHARACTERISTICS OF THE SAMPLE</td>
<td>30</td>
</tr>
<tr>
<td>II.</td>
<td>CORRELATIONAL ANALYSES AMONG PRIMARY VARIABLES</td>
<td>35</td>
</tr>
<tr>
<td>III.</td>
<td>RESULTS OF ANOVA SUMMARY TABLE USING AUQ DEFINITION</td>
<td>37</td>
</tr>
<tr>
<td>IV.</td>
<td>STRUCTURE MATRIX RESULTS FROM DISCRIMINANT FUNCTION ANALYSIS USING AUQ DEFINITION</td>
<td>39</td>
</tr>
<tr>
<td>V.</td>
<td>RESULTS OF ANOVA SUMMARY TABLE USING THE DICHOTOMOUS NIAAA DEFINITION</td>
<td>42</td>
</tr>
<tr>
<td>VI.</td>
<td>CORRELATION MATRIX OF BINGE AND WITHDRAWAL VARIABLES</td>
<td>44</td>
</tr>
<tr>
<td>FIGURE</td>
<td>PAGE</td>
<td></td>
</tr>
<tr>
<td>--------</td>
<td>------</td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>40</td>
<td></td>
</tr>
</tbody>
</table>

1. Drinking Habits of the Sample
2. Canonical Discriminant Functions
**LIST OF ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>AEQ</td>
<td>Alcohol Expectancy Questionnaire</td>
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<td>ANOVA</td>
<td>Analysis of Variance</td>
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<td>ARPS</td>
<td>Alcohol-Related Problem Scale</td>
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<td>AUQ</td>
<td>Alcohol Use Questionnaire</td>
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<tr>
<td>BAC</td>
<td>Blood Alcohol Content</td>
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<tr>
<td>BMI</td>
<td>Body Mass Index</td>
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<tr>
<td>CI</td>
<td>Confidence Interval</td>
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<tr>
<td>CNS</td>
<td>Central Nervous System</td>
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<td>df</td>
<td>Degrees of Freedom</td>
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<td>fMRI</td>
<td>Functional Magnetic Resonance Imaging</td>
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<td>GABA</td>
<td>Gamma-Aminobutyric Acid</td>
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<td>GPA</td>
<td>Grade Point Average</td>
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<td>IGT</td>
<td>Iowa Gambling Task</td>
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<tr>
<td>IQ</td>
<td>Intelligence Quotient</td>
</tr>
<tr>
<td>M</td>
<td>Mean</td>
</tr>
<tr>
<td>MANCOVA</td>
<td>Multivariate Analysis of Covariance</td>
</tr>
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<td>NA</td>
<td>Negative Affect</td>
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<tr>
<td>NAART</td>
<td>North American Adult Reading Test</td>
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<td>NIAAA</td>
<td>National Institute on Alcohol Abuse and Alcoholism</td>
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<tr>
<td>NMDA</td>
<td>N-methyl-D-aspartic acid</td>
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<td>oz</td>
<td>Ounce</td>
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<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
<td>PA</td>
<td>Positive Affect</td>
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<td>PANAS</td>
<td>Positive and Negative Affect Schedule</td>
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<tr>
<td>SAMHSA</td>
<td>Substance Abuse and Mental Health Services Administration</td>
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<tr>
<td>SAWS</td>
<td>Short Alcohol Withdrawal Scale</td>
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<tr>
<td>SD</td>
<td>Standard Deviation</td>
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<td>UPPS-P</td>
<td>Impulsive Behavior Scale</td>
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<td>WCT</td>
<td>Wisconsin Card Sort Test</td>
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<td>WTAR</td>
<td>Wechsler Test of Adult Reading</td>
</tr>
</tbody>
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SUMMARY

Binge drinking is associated with physical, social, and emotional consequences, rendering it a serious public health concern. The highest rates of binge drinking are consistently found to be among young adults, ages 18 – 26. This age range also coincides with a time, developmentally, that the frontal lobe is not yet fully developed, and thus highly vulnerable to changes. Because the frontal lobe is responsible for executive functioning, it seems then, that binge drinking and executive functioning may be related, especially among this young adult cohort. Other aspects too, such as alcohol expectancies and mood have been found to be related to alcohol use, and binge drinking specifically. Complicating the picture further, is the fact that there is no clear, universal definition of binge drinking in the literature, which inherently leads to researchers using different definitions of this phenomenon.

Therefore, the present study had two main, overarching aims. First, we set out to investigate the cognitive and emotional correlates of binge drinking among young adults. Our second aim was to further examine commonly used definitions of binge drinking in the literature. Results indicated that different definitions of binge drinking yielded different results, and that executive function was only related to binge drinking when using one definition of binge drinking. Using a different definition, positive alcohol expectancies was found to be highly related to binge drinking. Complete results are discussed in greater detail. These mixed results lend themselves to future research directions, which are discussed.
I. INTRODUCTION

A. Background

Binge drinking poses a serious public health concern in the United States, especially among young adults. In 2009, 35% of 18-20 year olds reported binge drinking (SAMHSA, 2010). The highest binge rates were among 21 – 25 year olds, with almost half of this age group reporting current binge drinking. Among high school seniors, 20% reported binge drinking at least once in the past two weeks (Patrick, Schulenberg, Martz, Maggs, O’Malley, & Johnston, 2013). These results are consistent with the Behavioral Risk Factor Surveillance System that also found the highest rates of binge drinking to be among young adults, ages 18-25 (Naimi, Brewer, Mokdad, Denny, Serdula, & Marks, 2003). Despite the fact that the highest rates of binge drinking are found in young adults, many college students do not view their own binge drinking as problematic (Eshbaugh, 2008), increasing the likelihood that they will continue to engage in this behavior.

Binge drinking is associated with a variety of harmful behaviors including tobacco use and illicit drug use (SAMHSA, 2010). Binge drinking is also associated with an array of social problems such as economic losses, disruption of family and social relationships, emotional problems, violence, and problems with the law (NIAAA, 2000). Sadly, a recent study found binge drinking to be one risk factor associated with suicide among current and former United Stated military personnel (Leardmann, Powell, Smith, Bell, Smith, Boyko et al., 2013). Further, approximately 40% of all fatal traffic crashes in the United States in 2004 were alcohol-related (Yi, Chen, & Williams, 2006). Alcohol use is also associated with several health consequences including various cancers, cardiovascular disease, and brain damage (de Lange, Hijmering,

Combined, these data render binge drinking a cause for major concern.

B. **What Is Binge Drinking?**

To date, there is no one agreed upon definition of binge drinking. According to the National Institute of Alcohol Abuse and Alcoholism (NIAAA; 2004) a “binge” is defined as:

a pattern of drinking alcohol that brings blood alcohol content (BAC) to 0.08 gram percent or above. For the typical adult, this pattern corresponds to consuming five or more drinks (male), or four or more drinks (female) in about two hours. (p. 3)

NIAAA defines a standard drink as 0.5 ounces of alcohol as is found in one 12-oz beer, one 5-oz glass of wine, or one 1.5-oz shot of distilled spirits (NIAAA, 2004).

There are several issues that make the NIAAA definition of binge drinking incomplete. First, what defines a “typical adult”? This vague terminology does not lend itself to scientific scrutiny. Second, body weight is not taken into consideration, a factor that inevitably influences the number of drinks necessary to raise one’s BAC to 0.08. Next, this definition defines one binge episode, but fails to provide a definition for a pattern of binge drinking (i.e., how many times must a person binge in what period of time?) that is likely more important when considering the social and health consequences of binge drinking outlined above. Finally, in addition to the absolute number of drinks consumed, there are other social, emotional, and cognitive factors that affect the “drunkenness” of an individual including alcohol expectancies, subjective intoxication, drinking environment, and tolerance to alcohol (see Courtney & Polich, 2009 for review). All of these issues should be considered if the essential concern regarding binge drinking is raising one’s BAC to a dangerously high level in a short period of time, and the associated risks and dangers associated with that, rather than the absolute number of drinks consumed.
Even the cut-off of 5 drinks for men, and 4 drinks for women, ultimately proves arbitrary. Some experimental studies have concluded that the NIAAA definition of binge drinking actually tends to capture BACs lower than 0.08 and therefore suggest that the 5/4 definition does not reflect true binge episodes (Lange & Voas, 2000). Instead, they propose that 6 (males)/5 (females) drinks per episode better captures BACs of 0.08 or higher. Likewise, another study examining BACs among those who would be classified as “binge drinkers” using the 5/4 definition, found that 48% of “binge drinkers” remained below .08% BAC. Moreover, a total of 30% of “binge drinkers” engaged in a drinking episode that resulted in a BAC below .06% (Perkins, DeJong, & Linkenbach, 2001). Again, these findings have led some researchers to question labeling 5/4 drinks as binge drinking (Perkins et al., 2001).

To address the issue of binge drinking pattern versus one binge drinking episode, a revised definition of binge drinking pattern has been offered as

a pattern of drinking alcohol that raises one’s blood alcohol content (BAC) to 0.08 gram percent or higher (which corresponds to 5 drinks for men and 4 drinks for women in a 2 hour period) on more than one occasion in the past 6 months (Courtney & Polich, 2009).

Nonetheless, this definition does not help to scientifically define a binge episode either. It merely gives a conservative frequency that defines binge drinking pattern. The debate as to what exactly constitutes binge drinking and binge drinking pattern thus remains open. Further studies are needed to help shed light on and define these constructs with the goal of creating a unified, comprehensive definition.

What is clear is that binge drinking pattern consists of heavy drinking episodes followed by periods of sobriety. Some take this notion one step further and assert that binge drinking may be conceptualized as resulting in repeated withdrawals from alcohol (Duka, Gentry, Malcom, Ripley, Borlikova, Stephens, et al., 2004). Evidence suggests that withdrawal from alcohol,
rather than the actual alcohol exposure, plays a large role in the occurrence of brain damage (Hunt, 1993). Thus, it is these multiple withdrawal episodes that contribute to brain damage among binge drinkers.

C. **Kindling Theory of Alcohol Withdrawal**

The idea of alcohol withdrawal leading to brain damage forms the basis of what is known as the *kindling theory*. The kindling phenomenon was first observed by Goddard et al. (1969). Goddard and colleagues electrically stimulated the brains of rats daily. Initially, these stimulations had no effect. After one week of daily stimulation, rats began to exhibit small tremors. After two weeks, rats began to experience seizures. Once the animal had experienced one seizure, that animal then experienced a seizure on each successive stimulation. The authors concluded that the repeated electrical stimulations led to permanent brain changes, particularly in the limbic system, that resulted in a lower seizure threshold. Later studies revealed that chemical stimuli produced the same effect as Goddard and colleagues’ electrical stimulations (Becker, 1998). As such, this study elucidated that the critical component of kindling was not the type of stimulation per se (i.e., electrical or chemical), but rather that the stimulus is administered repeatedly and intermittently (i.e., repeated administrations, each separated by a period of time) (Becker, 1998).

Interestingly, and analogous to the epileptic kindling syndrome, there is evidence to suggest that as episodes of withdrawal from alcohol increase, so too does the severity of the withdrawal symptoms. Kindling as a model for alcohol withdrawal was first theorized by Ballenger and Post (1978). They retrospectively examined the hospital charts of 200 alcoholic men and observed that only 9% of the men who had been drinking for less than three years experienced withdrawal symptoms more severe than a mild tremor. In contrast, among those
who had abused alcohol for at least ten years, 67% experienced severe symptoms of withdrawal including “shakes” and fever. This patterning of findings held true, irrespective of age. The authors concluded that repeated episodes of withdrawal served as stimuli for kindling, particularly in the limbic, hypothalamic, and thalamic nuclei (Balenger & Post, 1978).

Kindling as a model for alcohol withdrawal has been observed in both human and non-human animals. For example, compared to animals that have undergone one withdrawal episode, animals that experience multiple episodes of withdrawal from ethanol exhibit a lower seizure threshold and, thus, are more likely to experience seizures during withdrawal from ethanol (Becker, 1994; Becker, Diaz-Granados, & Hale, 1997; Becker, Diaz-Granados, & Weathersby, 1997; Pinel, Oot, & Mucha, 1975; Stephens, Brown, Duka, & Ripley, 2001; Ulrichsen, Clemmesen, & Hemmingsen, 1992). Becker and Hale (1993) further demonstrated that an intermittent schedule of alcohol administration was an imperative factor in the kindling effect. Specifically, they administered equivalent amounts of ethanol to one group of mice that received the ethanol in one dose, and another group that were administered the same dose of ethanol, but over several administrations, with breaks in between. Withdrawal severity was measured by assessing convulsions every hour for the first 10 hours and then at 24 hours post-withdrawal. Increased seizure activity was observed only among those in the multiple administration group, suggesting that it is not the absolute amount of alcohol that is important, but rather that multiple withdrawals appear to be a significant contributor to this observed kindling effect. Later, Veatch and colleagues (2007) found the same kindling effect for ethanol withdrawal-related seizures among male mice. Further, among non-human animals, alcohol withdrawal produced electroencephalography (EEG) abnormalities that became progressively
more severe as episodes of withdrawal increased (Guerrero-Figueroa, Rye, Gallant, & Bishop, 1970; Walker & Zornetzer, 1974).

There is also ample evidence for the kindling effect of alcohol withdrawal among humans. The first example of this phenomenon among humans was observed in a retrospective study of patients’ charts who were admitted to a hospital for alcohol withdrawal (Gross, Rosenblatt, Malenowski, Broman, & Lewis, 1972). In this study, 75% of patients who experienced a seizure during withdrawal had had at least one previous admission to the hospital for alcohol withdrawal. Later, it was suggested that, just as with the epileptic kindling effect, as alcohol abuse becomes more severe, so too do symptoms of alcohol withdrawal among humans. (Ballenger & Post, 1978). Further support for the kindling model came from a retrospective study of hospital charts of alcohol dependent men who had experienced alcohol withdrawal seizures and those who had not (Brown, Anton, Malcom, & Ballenger, 1988). Among the seizure group, 48% of patients had previously experienced at least five prior detoxifications. Only 12% of the non-seizure group fell into this category. Finally, a large-scale, multi-site sample of 6818 men hospitalized in 172 different U.S. Department of Veterans Affairs medical centers also lent support to the kindling hypothesis, revealing that seizures were more common among men who had greater numbers of previous hospitalizations for withdrawal or alcohol-related events (Booth & Blow, 1993). Several similar studies have been conducted, yielding results that followed this pattern, revealing that as the number of withdrawal episodes increases, so too does the likelihood of experiencing a seizure during withdrawal (Lechtenberg & Worner, 1990; Lechtenberg & Worner, 1991; Lechtenberg & Worner, 1992; Worner 1996; cf. Wojnar, 1999). In addition to seizures, there is ample evidence that multiple withdrawals from alcohol correlate with more severe withdrawal symptomatology generally as well, including symptoms
such as nausea, sweating, anxiety, agitation, and perceptual disturbances (Malcolm, Roberts, Wang, Myrick, & Anton, 2000; Shaw, Waller, Latham, Dunn, & Thomson, 1998).

In sum, evidence from both animal and human studies support the kindling model of alcohol withdrawal, which stresses that it is not the absolute amount of alcohol that contributes to more severe withdrawal symptoms, but rather, the repeated, intermittent nature of alcohol administration (as opposed to alcohol dependent individuals whose BACs remain consistently high) that leads to a heightened withdrawal syndrome. Heavy binge drinking clearly falls under the category of repeated and intermittent administrations of alcohol, and thus may be conceptualized and perhaps better understood within the framework of the kindling theory of alcohol withdrawal.

D. Binge Drinking and Brain Damage

In addition to seizure risk and withdrawal symptom severity, binge drinking is often linked to more general brain damage as well. Alcohol consumption, and binge drinking in particular, affects the brain in several ways. Glutamate and gamma-aminobutyric acid (GABA) are the primary excitatory and inhibitory neurons, respectively. Alcohol, a CNS depressant, acts to potentiate GABA and inhibit glutamate (Zeigler, Wang, Yoast, Dickinson, McCaffree, Robinowitz, et al., 2005). As such, repeated ethanol binges leads to an imbalance of inhibitory and excitatory neurotransmission, particularly in the nucleus accumbens (Szuminski, Diab, Friedman, Henze, Lominac, & Bowers, 2007). The release of glutamate, which binds to several different receptors including N-methyl-D-aspartic acid (NMDA), is increased as a result of binge alcohol consumption. Interestingly, during actual alcohol intoxication, there does not appear to be a change in glutamate levels. In fact, while alcohol is present in the body, it actually appears to be protective against neurodegeneration and apoptosis (Hunt, 1993). In contrast,
approximately 3-5 hours after detoxification begins, changes in NMDA receptor sensitivity occur, along with a large increase in glutamate (Chandler, Sumners, & Crews, 1993). Excess glutamate leads to neuronal death (Ward, Lallemand, & de Witte, 2009). Additionally, NMDA receptors allow increased calcium to permeate neurons, a process that proves toxic to the cells (Hunt, 1993).

Thus, it is clear that binge alcohol consumption negatively impacts the brain, frequently resulting in neuronal damage and cell death. Of course, these observed effects do not, and in and of themselves, “disprove” the notion that certain individuals may possess inherent predispositions towards alcohol abuse and/or binge drinking behavior. However, this evidence certainly suggests that harmful brain alterations do indeed follow binge drinking.

Animal studies provide further insight into the detrimental effects of binge ethanol exposure on the brain. Binge ethanol consumption alters levels of neuropeptide Y, which affects both consummatory behavior as well as seizure activity (Bison & Crews, 2012). Additionally, abstinence from alcohol after intermittent ethanol exposure (i.e., binge drinking) is associated with a dysfunctional relationship between the medial prefrontal cortex and the central nucleus of the amygdala, an important connection for executive function (George, Sanders, Freiling, Grigoryan, Vu, Allen, et al., 2012). For example, animals in binge ethanol conditions exhibit learning deficits (Coleman, He, Lee, Styner, & Crews, 2011; Stephens, Brown, & Duka, 2001; Robinson, Khurana, Kuperman, & Atkinson, 2012) that have also been observed in humans (Stephens, Ripley, Borlikova, Schubert, Albrecht, Hogarth, et al., 2005), as well as impairment in spatial memory and non-spatial object recognition, and working memory (Cippitelli, Zook, Bell, Damadzic, Eskay, Schwandt et al., 2010; George et al., 2012). Binge drinking has also been linked to decreased neuronal development and cell death in the hippocampus, neocortex, and
cerebellum (Pascual, Blanco, Cauli, Miatarro, & Guerri, 2007; Taffe, Kotzebue, Crean, Crawford, Edwards, & Mandyam, 2010). Moreover, not only is brain damage observed soon after binge drinking exposure, but such brain damage is also evident months after the binge exposure as well (Cippitelli et al., 2010; Coleman et al., 2011; Ehlers & Criado, 2010; Pascual et al., 2007; Taffe et al., 2010).

In humans, the frontal lobe continues to develop into young adulthood (Romine & Reynolds, 2005). Therefore, this area of the brain may be particularly vulnerable towards damage from binge drinking in adolescents and young adults, the precise groups that manifest the highest levels of binge drinking (Naimi et al., 2003; SAMHSA, 2010). The frontal lobe is responsible for executive function, and imperative for many abilities such as decision making, planning ability, cognitive flexibility, sustained attention, and inhibition. Indeed, compared to non-binge drinkers, binge drinkers displayed lower sustained attention, worse episodic memory, and slower planning ability (Hartley, Elsabagh, & File, 2004). Female binge drinkers exhibited greater impairment in tasks linked to the dorsolateral prefrontal cortex and orbitofrontal cortex compared to non-binge drinkers (Scaife & Duka, 2009). Likewise, compared to non-binge drinkers, female binge drinkers in particular, exhibited significantly worse performance on a task in which appropriate inhibition of response is required, implicating frontal lobe dysfunction (Townshend & Duka, 2005). Binge drinkers also exhibited decreased activation in the prefrontal cortex for matching stimuli, as well as deficient electrophysiological differentiation between correct and incorrect responses on a task measuring working memory (Crego, Rodriguez-Holguin, Parada, Mota, Corral, & Cadaveira, 2009; 2010). The Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994), which reliably measures decision making capacity, differentiated adolescents who were binge drinkers and those who exhibited few
alcohol problems such that those who performed poorly on this task were more likely to binge drink, compared to those who displayed better performance, who were less likely to experience alcohol problems (Xiao, Bechara, Grenard, Stacy, Palmer, Wei et al., 2009). Finally, number of withdrawal episodes was inversely related to ability to follow goals and display appropriate inhibition on this task (Duka, Townshend, Collier, & Stephens, 2003).

The pattern of brain damage found in binge drinkers who are not diagnosed with an alcohol use disorder is similar to the brain damage found in individuals with an alcohol use disorder. In a study using functional Magnetic Resonance Imaging (fMRI), binge drinking adolescents displayed brain responses similar to adolescents diagnosed with an alcohol use disorder on a verbal encoding task (Schweinsburg, McQueeny, Nagel, Eyler, & Tapert, 2010). Heavy binge drinkers and chronic alcohol users also performed similarly on tasks measuring visuo-motor speed, visuo-spatial organization and planning, learning, proactive interference, retroactive interference, and retrieval efficiency (Kokavec & Crowe, 1999). Binge drinkers only performed better than chronic alcohol users in tasks that required semantic organizational ability.

It appears that binge drinking has a unique effect on the brain. For instance, a study of adolescent binge drinkers, binge drinkers and marijuana users, and non-using control participants revealed that binge drinkers, regardless of marijuana use, evidenced significantly worse white matter integrity in areas of the brain that are important for frontal lobe development and often implicated in neurocognitive function (Jacobus, McQueeny, Bava, Schweinsburg, Frank, Yang et al., 2009). Compared to light drinkers and non-drinkers, adolescent binge drinkers exhibited hypersensitivity to reward on the IGT. At the same time, performance on the IGT significantly predicted binge drinking (Johnson, Xiao, Palmer, Sun, Wang, Wei et al., 2008). Further, the authors found no differences between groups in school performance or in working memory
performance. The binge drinkers’ performance on this task was unique in that they specifically performed worse on the latter trials of the task, which are indicative of decision making under risk, as opposed to the first 50 trials which are indicative of decision making under ambiguity. The authors label this type of performance as a deficit in affective decision making, which was unique to the binge drinking group, and is attributed to the ventromedial prefrontal cortex.

To further explore the link between executive function and binge drinking, a sample of college students was asked whether they intended to binge drink and were then measured again one week later to assess whether they did or not (Mullan, Wong, Allom, & Pack, 2011). These same students completed four tasks measuring different aspects of executive function (planning ability, decision making ability, cognitive flexibility, and inhibition of responses). Inhibition and planning ability moderated the relationship between intention to drink and behavior. Further, binge drinkers displayed worse performance on the decision making task and the planning task compared to non-binge drinkers and moderate drinkers.

Combined, this evidence is compelling and raises an important question: What comes first, the brain damage or the alcohol problems? On one hand, these data may suggest that binge drinkers and individuals with an alcohol use disorder both possess inherent brain differences leaving them vulnerable toward developing alcohol problems. Conversely, another explanation is that binge drinking results in cognitive damage in neural substrates that are integral in protecting the individual from the development of an alcohol use disorder, including planning ability, inhibition, decision making capacity, and cognitive flexibility (Crews, Collins, Dlugos, Littleton, Wilkins, Neafsey, et al., 2004; Duka et al., 2004; Hunt, 1993; Ward et al., 2009; Zeigler et al., 2005). According to the latter theory, then, the developmental trajectory would look as follows: binge drinking → brain damage → deficits in executive function →
vulnerability towards alcohol use disorder. Targeting binge drinking may thus protect against
the development of subsequent brain damage and alcohol use disorders.

In sum, as evidenced across clinical and pre-clinical studies, binge drinking appears to
heighten risk of exhibiting brain damage and/or cognitive deficits. These cognitive deficits are
manifested particularly in the realm of executive function, and include impairments in planning
ability, decision making, cognitive flexibility, working memory, and inhibition. These integral
functions are linked to the frontal lobe, an area of the brain that is not fully developed until the
mid-20s in humans. Further, young adults in their late teens to early 20s exhibit the highest rates
of binge drinking. Thus, young adults who partake in heavy binge drinking are at particularly
high risk for brain damage and impaired executive function.

E. Negative Affect

In addition to brain damage and cognitive dysfunction, there are other factors that
influence the urge and tendency to partake in binge drinking, including negative affect and
alcohol expectancies. Negative affect is defined as a disposition of subjective distress and
unpleasurable engagement (Crawford & Henry, 2004). This includes sadness as well as feelings
of nervousness, tension, and worry (Watson & Clark, 1984). Further, the construct of negative
affect is thought to be situation-independent; that is, although one’s mood certainly changes
across different situations, an individual high in negative affect is thought to be more likely to
respond to a given situation with more subjective distress compared to a person low in negative
affect (Watson & Clark, 1984).

Negative affect is consistently related to alcohol use and alcohol related problems. Binge
drinking specifically has been linked to negative affect, including both current and lifetime
anxiety and depression (Adams, Boscarino, & Galea, 2006; Boscarino, Kirchner, Lester,
Hoffman, Sartorius, & Adams, 2011; Okoro, Brewer, Naimi, Moriarty, Giles, & Mokdad, 2004; Strine, Mokdad, Dube, Balluz, Gonzalez, Berry et al., 2008). Binge drinkers have also been shown to experience less positive mood than non-binge drinkers (Townshend & Duka, 2005). In addition to the association between negative affect and binge drinking, the co-occurrence of binge drinking and depression in a sample of 17,000 Canadian youth was related to increased suicidality, rendering this comorbidity a cause for major concern (Archie, Zangeneh, Azadeh, Akhtar-Danesh, 2011).

It has been posited that the same kindling process of multiple withdrawals that leads to brain damage and cognitive dysfunction, may also lead to emotional disturbances such as anxiety disorders (Lepola, 1994). Compared to non-binge drinkers, both human and animal binge drinkers exhibited impaired fear conditioning and impaired long-term potentiation in the amygdala and hippocampus (Stephens & Duka, 2008; Stephens et al., 2005). Number of withdrawals has also been inversely related to performance on an emotional stroop task, but was not predictive of self-reported anxiety (Duka et al., 2002). However, when comparing problem drinkers to social drinkers, problem drinkers rated themselves higher in trait anxiety, feelings of anger, depression, and confusion and also exhibited exaggerated fear response (Duka et al., 2002; Townshend & Duka, 2003). Depression has also been shown to predict binge drinking, especially in 20-29 year olds (Bonin, McCreary, & Sadava, 2000). Therefore, it is important to consider and measure negative affect in relation to binge drinking in addition to executive function in order to gain a better and more thorough understanding of the motivations and consequences behind binge drinking.
F. **Alcohol Expectancies**

According to social learning theory, cognition plays an important role in learning such that one’s expectations of future rewards or punishments profoundly influences behavior (Bandura, 1971). Accordingly, one’s direct and indirect experiences with alcohol shape one’s expectations and behaviors. Specifically, as cited in Braun et al. (2012),

> according to alcohol expectancy theory, [one’s] beliefs about alcohol and how it will affect [his] mood and/or behavior [yields] a significant impact on the choice to drink alcohol or abstain, how much alcohol to consume (p. 486)

and even one’s feelings and actions after drinking has commenced (Brown, Christiansen, & Goldman, 1987; Jones, Corbin & Fromme, 2001). For instance, one’s prior experiences with alcohol may lead to expectations of being more sociable, feeling “less inhibited, or [decreasing] anxiety” (Koob & Le Moal, 1997; Robinson & Berridge, 2000; Steele & Josephs, 1990).

In line with alcohol expectancy theory, both positive (Biscaro, Broer, & Taylor, 2004; Collins, Koutsky, Morsheimer, & MacLean, 2001; Strahan, Panayiotou, Clements, & Scott, 2011) and negative (Strahan et al., 2011) alcohol expectancies influence binge drinking. Specifically, expectancies for global positive changes predicted both number of drinks as well as binge drinking (Biscaro et al., 2004). Positive alcohol expectancies also predicted number of binge drinking days and alcohol problems (Collins et al., 2001). Further, the tendency for positive and negative alcohol expectancies to predict binge drinking was consistent across cultures in both American and Cypriot college students (Strahan et al., 2011). Thus, measuring positive and negative alcohol expectancies in relation to binge drinking is important.
II THE PRESENT STUDY

A. Aims

Past research indicates that binge drinking is associated with brain damage and impaired executive functioning, as a consequence of multiple withdrawals from alcohol. Further, other factors such as negative affect and alcohol expectancies impact one’s binge drinking intentions and behavior. However, the binge drinking literature lacks one clear, agreed upon definition and measurement of binge drinking. Thus, past studies have employed different definitions and measurements of binge drinking. Furthermore, studies that draw upon the kindling theory of withdrawal assume that binge drinking corresponds to worsening withdrawal symptomatology, but withdrawal episodes and symptoms have rarely, if ever, been measured.

As such, the present study has two main aims: 1) to gain a clearer picture of the cognitive and emotional aspects involved in binge drinking and 2) to investigate two commonly used definitions of binge drinking. Regarding the latter, several definitions of binge drinking have been offered, as outlined above. The central tenant of the kindling theory is that binge drinking is defined as multiple withdrawals from alcohol. However, many studies that rely on this theory fail to actually measure withdrawal symptoms among their participants. Therefore, the present study not only measured binge drinking, but also measured concomitant withdrawal symptomatology.

To compare commonly used definitions of binge drinking, three sets of analyses were conducted, each using a different, commonly used definition of binge drinking. For the first set of analyses, we used the modified Alcohol Use Questionnaire to obtain a binge score for each participant. Based on those binge scores, participants were classified as binge drinkers, non-
binge drinkers, or non-drinkers. After analyses were conducted using these groups, two additional sets of analyses were completed. Using the criteria set forth by the NIAAA, those same 150 individuals were then classified as either a binge drinker or non-binge drinker to form a dichotomous score. Finally, a continuous measure of binge drinking, based on NIAAA criteria, was also analyzed. In other words, groups were initially based on the modified AUQ binge score, but in subsequent analyses, dichotomous and continuous NIAAA definitions of binge drinking were employed to compare the results obtained from these different definitions.

To address the first aim, the present study examined cognitive and emotional correlates of binge drinking including executive function, negative affect, and alcohol expectancies. As noted above, three separate sets of analyses were employed. The first set used the definition of binge drinking provided by the modified AUQ. The second set used a dichotomous definition of binge drinking derived from the NIAAA definition (experienced at least one binge episode vs. experienced zero binge episodes). The third set of analyses employed a continuous measurement of binge drinking derived from the NIAAA definition (number of binge episodes in the past 6 months).

The present study is novel in several ways. First, three groups were studied: binge drinkers, drinkers who are not binge-drinkers, and non-drinkers. Past studies have typically investigated differences between binge drinkers and tee-totalers (Hartley et al., 2004), and between binge drinkers and non-binge drinkers (Scaife & Duka, 2009; Townshend & Duka, 2005). Including all three groups in one study allows us to draw stronger inferences about the specific effects of binge drinking (as opposed to non-binge drinking) and to determine whether drinkers who do not binge are different than those who do not drink at all. In other words, are observed effects associated with drinking in general, or are the effects specific to binge drinking
in particular? Finally, as outlined above, the binge drinking literature has not historically employed a consistent definition or measurement of binge drinking. By conducting three sets of analyses with the present data, using two commonly used definitions of binge drinking, I was able to compare and contrast the results obtained from each definition. The rationale behind conducting several sets of analyses using different definitions was that if the same results were not produced, this study would serve as one example that calls into question our ability to generalize across binge drinking studies that employ different definitions. If, in fact, different definitions did produce differing results, the scientific community at large loses valuable power in the knowledge gained about the phenomenon of binge drinking. Regardless of whether these definitions yield the same or different results, important knowledge will be gained regarding binge drinking definitions.

In line with the old adage, “which came first, the chicken or the egg?” it should be acknowledged upfront that causal mechanisms can only be inferred from longitudinal studies and that directionality cannot be inferred from the present study. Unfortunately, a longitudinal study was beyond the scope of the present project. Therefore, it cannot be known solely from this study whether potential observed differences in executive function, mood, or expectancies are consequences or antecedents of binge drinking. Despite this limitation, the findings gained by this study hold potential to add important information to the binge drinking literature about the relationship between binge drinking and cognitive function, mood, and alcohol expectancies, as well as about the utility of the binge drinking definitions and measurements currently used in the binge drinking literature. Further, the information obtained from the present study may guide the direction of future longitudinal studies.
B. **Hypotheses**

**Hypothesis 1:** Compared to non-binge drinkers and non-drinkers, binge drinkers will display:

- *Hypothesis 1a:* poorer planning ability, measured by the Tower Test.
- *Hypothesis 1b:* poorer cognitive flexibility and set-shifting skills, as measured by Trails B.
- *Hypothesis 1c:* poorer decision making ability, as measured by the Iowa Gambling Task.
- *Hypothesis 1d:* worse working memory ability, as measured by Digit Span Backwards.
- *Hypothesis 1e:* higher levels of negative affect.
- *Hypothesis 1f:* greater positive alcohol expectancies and lower negative alcohol expectancies.
- *Hypothesis 1g:* higher levels of impulsivity.

**Hypothesis 2:** Despite the frequent use of two different definitions of binge drinking (AUQ and NIAAA), using these different definitions to assess the relationship between binge drinking, executive function, and mood will not yield the same results.

**Hypothesis 3:** Number of withdrawal episodes and severity of withdrawal symptomatology will be positively correlated with the AUQ and NIAAA binge scores.
III. METHODS

A. **Participants**

A power analysis was conducted using Power And Precision 4 (Borenstein, Rothstein, & Cohen, 2001) to determine the sample size necessary to achieve a high probability of detecting meaningful changes in the respective dependent variables. In order to detect a medium size effect (f) of .25 with a Type I error protection level set at .05 and power (1-α) of approximately .80, it was estimated that each of the three cells required 50 individuals (N = 150).

Participants were recruited from the community through flyers posted at the University of Illinois at Chicago as well as through online advertisements, list-serves, and word of mouth. All participants were between the ages of 18 – 26, to capture not only the age range of those individuals who report the highest levels of binge drinking, but also to capture a period of time in which frontal lobe development is incomplete, leaving individuals more vulnerable to adverse neural effects from binge drinking. Individuals with a history of neurological disease or traumatic brain injury were excluded from the present study. All participants were instructed to abstain from the use of illicit drugs for at least one week prior to the study visit and from the use of alcohol for at least 12 hours prior to the study visit.

A total of 150 participants were eligible and able to schedule and complete the study visit. A total of 50 binge drinkers, 50 non-binge drinkers, and 50 non-drinkers were included. Binge drinkers and non-binge drinkers were determined based on their AUQ binge scores (see AUQ binge score below). Non-drinkers were defined as those who had not consumed any alcohol in the past 6 months.
B. **Procedures**

1. **Screening**

   Interested individuals were directed to a secure webpage containing the screening questionnaire or could call the laboratory to speak to a research assistant who administered the screening questionnaire. The screening questionnaire was collected and managed using (Research Electronic Data Capture) electronic data capture tools hosted at University of Illinois at Chicago. “REDCap is a secure, web-based application designed to support data capture for research studies” (Harris, Taylor, Thielke, Payne, Gonzalez, & Conde, 2009). The initial screening questions asked the individual to report his age and whether he had experienced a neurological disease or brain injury. Individuals who continued to be eligible after the initial screening questions continued on to complete the revised Alcohol Use Questionnaire and the CAGE online or over the phone. Individuals with eligible binge scores and who answered “yes” to no more than one CAGE item were scheduled for a study visit. Reminder phone calls and/or emails were sent 24 hours prior to the individual’s scheduled appointment.

2. **Study Visit**

   Upon arrival at the laboratory, participants reviewed the study details with a research assistant as part of the informed consent process and written consent was collected. Participants then completed both computerized, and paper and pencil executive function tasks. Following these tasks, participants answered computerized self-report questionnaires (Microsoft Access). Participants were then debriefed and compensated $25 for their time. On average, a typical study visit lasted about 60 minutes.

2.1 **Questionnaires**
**Demographics.** Demographics included age, gender, race/ethnicity, highest level of education, weight, height, whether the individual is presently in college and if so, where that individual lives (e.g., dorm, off-campus, parents’ home), and estimated grade point average (GPA).

*Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978).* A quantity-frequency, beverage-specific index of alcohol consumption for the past 6 months was derived from the AUQ. As described in Townshend and Duka (2002), “The revised questions, by determining brands of liquor, allow the actual alcoholic content (percentage volume) of drinks to be assessed.” Participants estimated “the number of drinking days, the usual quantity consumed, and the pattern of drinking” from which an index was calculated. The AUQ has been found to be a reliable measure of drinking quantity and drinking pattern (Townshend & Duka, 2002). In the present sample, the AUQ was found to be reliable (α=.61)

*AUQ binge drinking score.* As described in Townshend and Duka (2002), “A binge drinking score was calculated on the basis of information given in items 10, 11, and 12 of the AUQ [Speed of drinking (average drinks per hour); number of times being drunk in the previous 6 months; percentage of times getting drunk when drinking” (average)]. The binge score was calculated in the same way as the AUQ score is derived but without items 1-9 that refer to quantity and type of alcohol intake: [4 x (Item 10) + Item 11 + 0.2 x (Item 12); Mehrabian & Russell, 1978]. “This score gives a picture of the drinking patterns of the participants rather than just a measure of alcohol intake. [Participants] who have a high binge score and drink [high quantities] but irregularly may have a similar intake of alcohol to those with a lower binge score who drink on a regular basis.” The cutoff points of the binge score for categorizing binge drinkers and non-binge drinkers were binge score ≤ 16 for non-binge drinkers and binge score ≥
24 for binge drinkers. Individuals whose score fell in the 17 – 23 range were excluded from participation. These cutoffs were used up-front, such that we ended up with 50 participants in each group.

*CAGE* (Mayfield, McLeod, & Hall, 1974). The CAGE is a brief, 4-item questionnaire that was used to screen out individuals who had a current or prior alcohol abuse problem. CAGE stands for attempts to cut back on drinking, being annoyed at criticisms about drinking, feeling guilty about drinking, and using alcohol as an eye opener. The CAGE has been widely used to screen for alcohol abuse in numerous populations and was found to be reliable in the current sample (α=.67).

*NIAAA binge score.* To assess the NIAAA definition of binge drinking the following wording was used: “Think about the past 6 months. On how many occasions did you consume 5 or more (for males)/4 or more (for females) drinks in a 2 hour period or less? A drink is defined as 0.5 ounces of alcohol as is found in one 12-oz beer, one 5-oz glass of wine, or one 1.5-oz shot of distilled spirits”. In accordance with the NIAAA definition of binge drinking, a score of one or higher was classified as a binge drinker.

*Short Alcohol Withdrawal Scale* (SAWS; Gossop et al., 2002). As described in Kassel et al. (2013), The SAWS asks participants to identify [the] intensity of ten different symptoms (anxiety, sleep disturbance, [memory] complaints, nausea, restlessness, tremor/shaking, mental confusion, sweating, heart pounding, and a general sense of misery) on a 4-point scale (0 = None; 3 = Severe). The scale has been independently evaluated for psychometric properties and found to be both reliable and valid. (p. 297) (Elholm, Larsen, Hornnes, Zierau, & Becker, 2010). In the present study, we asked participants to think about the morning after their most recent heavy drinking episode over the past 6 months and to rate the intensity of the above symptoms on that morning (i.e., severity). We then asked
participates to estimate the number of times over the past 6 months that they experienced each symptom (i.e., frequency). In the present study, the SAWS was found to be highly reliable ($\alpha=.83$).

**Alcohol Expectancy Questionnaire (AEQ; Leigh & Stacy, 1993).** The AEQ is a 34-item questionnaire that assesses positive and negative expected effects of alcohol consumption. There are eight expectancy factors, four positive factors (social facilitation, sex, fun, tension reduction), and four negative factors (social, emotional, physical, cognitive/performance). The total AEQ was found to be highly reliable in the current sample ($\alpha=.97$), as were the positive and negative expectancy subscales ($\alpha=.98$, $\alpha=.93$, respectively).

**Alcohol-Related Problem Scale (ARPS; McGee & Kypri, 2004).** The ARPS asks participants to rate 14 problems experienced in the past 6 months. Items include hangover, emotional outbursts, vomiting, heated argument, physically aggressive, blackouts, unable to pay bills, unprotected sex, sex unhappy about at the time, sex later regretted, stole public or private property, committed an act of vandalism, removed from pub/[bar]/club, arrested for drunken [behavior]. (p. 322)

Two response options are given: yes or no. The ARPS was found to be highly reliable in the present sample ($\alpha=.82$).

**Positive and Negative Affect Schedule (PANAS; Watson et al.,1988).** The PANAS is a widely used 20-item questionnaire that assesses both positive and negative affect and provides separate scores for each. The scales have been shown to be highly internally consistent and largely uncorrelated. In the present study, participants were asked to rate items based on how they have felt over the past week. In the present study, the positive affect subscale was found to be moderately reliable ($\alpha=.60$), and the negative affect subscale was found to be highly reliable ($\alpha=.78$).
UPPS-P Impulsive Behavior Scale (UPPS-P; Cyders et al., 2007). The UPPS-P is a revised version of the UPPS Impulsive Behavior Scale (Whiteside & Lynam, 2001). The revised version assesses five components of impulsivity: negative urgency, (lack of) premeditation, sensation seeking, (lack of) perseverance and positive urgency. Items are assessed on a 4-point Likert scale from 1 (agree strongly) to 4 (disagree strongly). The UPPS-P was used in the present study in addition to the behavioral measures to capture impulsivity. The total UPPS-P was found to be highly reliable (α=.75).

2.2 Cognitive Measures

Based on Giancola (2004), a series of tasks were chosen to represent both functional and neuroanatomical aspects of executive function. Tasks were chosen to represent a wide variety of abilities that fall under the rubric of executive function including: planning ability, decision making, cognitive flexibility, speeded set shifting, and the ability to temporarily store information and cognitively manipulate it (i.e., working memory). Further, the executive function tasks that were included in the present study are generally accepted as involving the prefrontal cortex, the primary area in which executive function takes place. Finally, time constraints were also kept in mind when choosing specific tests so as not to have the lab visit be overly burdensome to participants. Thus, only measures that took 15 minutes or less to administer were chosen so as to be able to include multiple facets of executive function in a one hour laboratory visit. Most of the chosen tasks have been used successfully in past studies of social and/or binge drinking populations (Giancola 2004; Hartley et al., 2004; Scaife & Duka, 2009; Townshend & Duka, 2005).

Wechsler Test of Adult Reading (WTAR; Wechsler, 2001). Participants were given the WTAR to provide an estimate of their verbal IQ performance. A list of 50 words that have
atypical grapheme to phoneme translations were presented on a computer screen and the participant was asked to read each word out loud. Each correctly pronounced word received one point, thus scores range from 0 – 50. Another measure, the North American Adult Reading Test (NAART) was considered to capture verbal IQ. In the end, the WTAR was chosen because it presents the same ease of administration as the NAART but has the added advantage of being co-developed and co-normed with the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1997).

_Tower Test._ The Tower Test assesses several key executive functions, including spatial planning, rule learning, inhibition of impulsive responding, and the ability to establish and maintain the instructional set. The objective of this task is to move disks varying in size from small to large across three pegs to build a designated tower in the fewest number of moves possible. The participant is asked to follow two rules: 1) move only one disk at a time and 2) never place a larger disk over a smaller disk. In order to do well in this task, the participant must plan his or her moves to complete the task efficiently (i.e., as few moves as possible in a required amount of time). Dependent variables in this task included the total achievement score which accounts for the total number of moves as well as time limitations. The raw score can range from 0 (failed all items) to 30, which would indicate that all towers were built correctly, using the minimum number of moves, within the given time limit (Delis, Kaplan, & Kramer, 2001). This task has been used successfully in past studies of social drinkers (Giancola, 2004).

_Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994)._ A computerized version of the original IGT was used. As described in Xiao et al. (2008),

Four decks of cards labeled A, B, C, and D are displayed on the computer screen. The backs of the cards all look the same as real decks of cards. The participant starts the with a sum of make-believe money in his or her account ($2,000), represented by a green bar that changes in length as the participant “wins” or “loses” money during the task. The subject is required to select one card at a time from one of the four decks. When the subject selects a card, a message is displayed on the screen indicating the amount of
money the subject has won or lost. The preprogrammed schedules of gain and loss are controlled by the computer. Turning each card can bring an immediate reward of $100 in Decks A and B and $50 in Decks C and D. As the game progresses, there are also unpredictable losses among the card selection. Total losses amount to $1,250 in every 10 cards in Decks A and B compared to $250 in Decks C and D. Decks A and B are equivalent in terms of overall net loss, and Decks C and D are equivalent in terms of overall net gain over the course of the trials. The difference is that in Decks A and C, the punishment is more frequent but of smaller magnitude, whereas in Decks B and D, the punishment is less frequent but of higher magnitude. Thus, Decks A and B are disadvantageous because they yield high immediate gain but a greater loss in the long run (i.e., net loss of $250 for every 10 cards), and Decks C and D are advantageous in that they yield lower immediate gain but a smaller loss in the long run (i.e., net gain of $250 for every 10 cards). In this study, an overall net score of the IGT was calculated by subtracting the total number of selections from disadvantageous decks (A and B) from total number selections from advantageous decks (C and D). (p. 4-5)

**Trails B.** Participants are given a sheet of paper randomly arranged with the numbers 1 through 13 and the letters A through L. This “dot to dot” type task requires the participant to draw a line connecting letters and numbers in alternating sequence (1-A-2-B-3-C etc). If an error is made, the experimenter will quickly inform the participant so that it can be corrected. Performance on this task is measured by the amount of time taken to complete all of the connections (Reitan, 1992). This task is thought to measure cognitive flexibility, (in this case, switching back and forth between letters and numbers). This test has been used successfully to measure cognitive flexibility and speeded set-shifting in social drinking participants (Giancola, 2004). The Wisconsin Card Sort (WCT) was also considered to measure cognitive flexibility and speeded set-shifting. Trails B was chosen because of its ease of administration and accessibility of the test.

**Digit Span Backwards.** In this task, the examiner recites a string of digits and the participant is asked to listen and recite the digits in reverse order. The strings of digits gradually increase in length. This task measures working memory, the ability to temporarily store information and cognitively manipulate it.
C. **Data Analyses**

1. **Preliminary Analyses**

Data were analyzed with SPSS Version 20.0 (SPSS Inc., Chicago IL). All data that were not captured electronically during the test session were double entered by two independent research assistants to ensure accuracy. Frequencies were generated to analyze demographic characteristics such as age, gender, race/ethnicity, and level of education. Descriptive statistics for all measures were generated and plotted on histograms to confirm a normal distribution. Preliminary analyses also included creating a correlation matrix to assess associations between the primary variables of interest including each of the cognitive tasks, the positive and negative affect composite factors from the PANAS, positive and negative alcohol expectancies, ARPS, SAWS, each component factor as well as the total score for the UPPS-P. Finally, drug use was also investigated including lifetime and past 6-month use, as well as family history of drug use.

Means and standard deviations for each substance were created for each group. A series of one-way analyses of variance (ANOVA) were conducted to assess whether groups differed significantly on any of the demographic or drug use variables. In cases where significant differences emerged, those variables were controlled for in subsequent analyses.

The overall drinking behavior of the sample was investigated using independent samples t-tests to compare the two drinking groups (binge and non-binge). Non-drinkers were only included in the analysis of “age of first binge”, because the criterion for non-drinkers was not having had a drink in the past 6 months. They could, however, have had a drink prior to this, and thus could have experienced a binge episode more than 6 months ago. The other variables included in these analyses, in which non-drinkers were not included, were AUQ binge score,
individual items from the AUQ, speed and frequency of drinking, percentage of times getting drunk, age of first binge, and alcohol related problems.

For all analyses, unless otherwise indicated, the critical alpha level was set at 0.05. Effects that were classified as trending towards significance had an alpha level between .051 and .10. Effect sizes are reported as partial eta-squares.

2. **Primary Analyses**

Hypothesis 1: To determine whether the three groups differed on executive function and self-report measures, a Multivariate Analysis of Covariance (MANCOVA) was conducted with group (non-drinker vs. non-binge drinker vs. binge drinker) as the fixed factor, and each executive function task, each component of the UPPS, Negative and Positive Affect, and Negative and Positive Alcohol Expectancies as outcome variables, co-varying for total drug use, age, and education (because as explained below, significant differences emerged between groups in these three variables). Because the sample sizes were equal across groups, homogeneity of variance was accepted as fairly robust. In the event that the assumption of homogeneity was violated, a more conservative critical alpha level was used to determine significance for these variables. Bonferroni corrections were used to control the Type I error rate. Following the MANCOVA, a discriminant function analysis was performed to determine if the executive function tasks and self-report measures significantly predicted group membership.

Hypothesis 2: To assess whether there were differences between binge drink definitions (AUQ binge score vs. NIAAA), two additional sets of analyses were conducted. First, the NIAAA definition was used to create a dichotomous outcome variable (0 = non-binge drinker, 1 = binge drinker). To assess whether executive function and self-report measures differed as a function of binge drinking, according to the dichotomous NIAAA definition, a multivariate
ANOVA was conducted with the NIAAA definition of binge drinking as the fixed variable, and each executive function task and self-report measure as predictor variables, controlling for education, age, and total drug use.

The next set of analyses used the NIAAA definition of binge drinking to create a continuous outcome variable. Linear regression models were created with number of binge drinking episodes over the past 6 months as the dependent variable and each executive function task and self-report measure as the predictor variables.

**Hypothesis 3:** In applying the kindling theory of alcohol withdrawal to binge drinking, one assumes that binge drinking does in fact correspond to multiple withdrawals from alcohol. Thus, to assess this assumption, I conducted Pearson product-moment correlations to measure the strength of the relationship between AUQ binge score, SAWS and NIAAA continuous binge score. Biserial correlations were conducted to assess the strength of the relationship between the NIAAA dichotomous score and the remaining variables of interest.
IV. RESULTS

A. Preliminary Analyses

1. Demographics

A total of 150 participants, evenly distributed among the three groups, completed the study. Participant characteristics can be found in TABLE I.

<table>
<thead>
<tr>
<th>Demographic</th>
<th>Nondrinkers (n = 50)</th>
<th>Non-binge drinkers (n = 50)</th>
<th>Binge drinkers (n = 50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M  SD  n</td>
<td>M  SD  n</td>
<td>M  SD  n</td>
</tr>
<tr>
<td>Age (years)</td>
<td>21.78  2.49  36</td>
<td>23.04  2.28  37</td>
<td>22.62  2.37*  25</td>
</tr>
<tr>
<td>Sex (no. female)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>25.45  7.98  36</td>
<td>24.55  4.79  37</td>
<td>25.24  6.52</td>
</tr>
<tr>
<td>Baseline verbal IQ (WTAR)</td>
<td>35.62  8.30  37</td>
<td>38.76  6.85  26</td>
<td>37.66  6.86</td>
</tr>
<tr>
<td>Currently in college</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education **</td>
<td>5.26  1.65  37</td>
<td>5.98  1.39  26</td>
<td>5.98  1.36*  28</td>
</tr>
<tr>
<td>GPA</td>
<td>3.38  0.48  37</td>
<td>3.39  0.45  26</td>
<td>3.16  0.80</td>
</tr>
<tr>
<td>Alcohol related consequences (for drinkers)</td>
<td>1.84  1.5  26</td>
<td>4.88  2.79*  28</td>
<td></td>
</tr>
<tr>
<td>AUQ binge score</td>
<td>7.74  3.86  37</td>
<td>42.92  20.92*</td>
<td></td>
</tr>
<tr>
<td>Total number drugs ever used</td>
<td>1.54  1.40  16</td>
<td>2.10  1.20  14</td>
<td>3.10  1.72*</td>
</tr>
<tr>
<td>Age of first binge</td>
<td>17.70  1.78  16</td>
<td>18.12  2.59  18.11</td>
<td>18.11  2.47</td>
</tr>
<tr>
<td>Father alcohol problem</td>
<td>16  14  13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother alcohol problem</td>
<td>5  2  5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sibling alcohol problem</td>
<td>14  10  11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>1  1  5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < .05

**Education: (1=K-8, 2=Some high school, 3=Completed high school, 4=One year of college, 5=Two years of college, 6=Three years of college, 7=Completed college)
The sample was primarily Caucasian (60%), followed by 28% African American, 27% Asian, 27% Hispanic/Latino, and 8% “other”. One-way ANOVA revealed that the three groups differed significantly in terms of age, $F(2, 147) = 3.63, p < .05$, education, $F(2, 147) = 3.97, p < .05$, and total drug use, $F(2, 147) = 14.74, p < .001$. Specifically, non-binge drinkers were slightly older than non-drinkers, non-drinkers completed a lower level of education compared to non-binge drinkers and binge drinkers, and binge drinkers reported greater total drug use compared to non-drinkers. Thus, age, education, and total drug use were controlled for in subsequent analyses. One’s Body Mass Index (BMI) may impact subjective and objective intoxication. Therefore, a one-way ANOVA was conducted to determine whether BMI differed significantly across groups. Results indicated that BMI did not differ significantly across groups, $F(2, 147) = 0.33, ns$. Interestingly though, the mean BMI across the full sample was 25.08 ($SD = 6.51$), indicating a sample that was, on average, overweight. Further, correlational analyses were conducted to assess whether BMI was correlated with the AUQ total score, any of the individual AUQ items, or the dichotomous or continuous NIAAA factors. Results indicated that BMI was not significantly correlated with any of these factors. Therefore, BMI was not controlled for in subsequent analyses. Likewise, a one-way ANOVA revealed that groups did not differ on verbal IQ, $F(2, 149) = 2.34, ns$. Therefore, verbal IQ was not controlled for in subsequent analyses.

2. **Drinking Habits and Other Drug Use**

Several interesting findings emerged regarding the overall drinking habits of binge drinkers versus non-binge drinkers in the current sample. The mean AUQ binge scores were 7.74 ($SD = 3.86$) among non-binge drinkers, and 42.92 ($SD = 20.92$) among binge drinkers, $t(98) = -11.70, p < .001$. The two groups did not differ significantly in terms of recency of last binge episode. As seen in Figure 1, independent samples t-tests revealed that compared to non-binge
drinkers, binge drinkers reported drinking significantly greater amounts of wine, $t(85) = -2.82, p < .001$, beer, $t(96) = -4.38, p < .001$ and spirits, $t(93) = -4.98, p < .001$ in a typical week.

Figure 1. Drinking Habits of the Sample.

Consistent with the NIAAA definition of binge drinking which defines a binge episode as consuming 4/5 or more drinks in a 2 hour period, in the current sample, on average, binge drinkers reported drinking 3 drinks per hour, whereas non-binge drinkers reported drinking 1 drink per hour, $t(98) = -8.92, p < .001$. Additionally, binge drinkers reported having been drunk
an average of 19 times in the past 6 months, compared to non-binge drinkers who reported having been drunk an average of 1 time in the past 6 months, an 1800% increase. Consistently, less time passed between binge episodes among binge drinkers compared to non-binge drinkers $t(54) = 2.36, p < .05$. Finally, when asked what percentage of the times that you drink do you get drunk, binge drinkers reported an average of 64% ($SD = 23.63$), compared to non-binge drinkers who reported an average of 9% ($SD = 12.07$), $t(98) = -14.69, p < .001$. Additionally, independent samples t-tests revealed that, as expected, binge drinkers reported suffering significantly more alcohol related consequences, compared to non-binge drinkers, $t(98) = -6.78, p < .001$.

In the total sample, 35% ($n = 52$) reported never having had 5 (4 for females) or more drinks in a 2 hour period. Accordingly, whereas the initial categorization (using the AUQ) resulted in 50 binge drinkers, the criteria set forth by the NIAAA resulted in a total of 98 participants being classified as binge drinkers.

Interesting findings emerged regarding other drug use in the present sample as well. Binge drinkers used significantly more drugs in their lifetime, $F(2,147) = 14.74, p < .001$, and in the past 6 months compared to non-binge drinkers and non-drinkers, $F(2, 147) = 6.81, p < .01$. Specifically, in their lifetime, a significantly larger number of binge drinkers had used marijuana $F(2, 147) = 6.90, p < .01$, cocaine $F(2, 147) = 6.63, p < .01$, amphetamines $F(2, 147) = 9.05, p < .001$, hallucinogens $F(2, 147) = 10.66, p < .001$, and prescription drugs not meant for them $F(2, 147) = 6.22, p < .01$, compared to the other two groups. In the past 6 months, binge drinkers used cigarettes $F(2, 147) = 7.64, p < .01$, marijuana $F(2, 147) = 7.58, p < .01$, hallucinogens $F(2, 147) = 4.88, p < .01$, and prescription drugs not meant for them $F(2, 147) = 3.63, p < .01$, more frequently than both non-binge drinkers and non-drinkers.
3. **Correlational Analyses**

Correlational analyses among the primary variables of interest revealed that, as expected, Tower, Trails, and Digit Span were significantly correlated with one another (see TABLE II).
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<tr>
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<td>.29**</td>
<td>.17*</td>
<td>.13</td>
<td>.08</td>
<td>.07</td>
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<td>.13</td>
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<td>.11</td>
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<td>-.28**</td>
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<td>.03</td>
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<td>-.08</td>
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<td>-.10</td>
<td>-.09</td>
</tr>
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<td>.04</td>
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<td>-.09</td>
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<td>.44**</td>
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<td>.57**</td>
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<td>.06</td>
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<td>.53**</td>
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<td>.75**</td>
<td>.51**</td>
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<td>.48**</td>
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<td>.35**</td>
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<td>.17*</td>
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<td>.08</td>
<td>-.13</td>
<td>.07</td>
<td>.44**</td>
<td>.34**</td>
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<td>-.41**</td>
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<td>.10</td>
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<td>.05</td>
<td>-.09</td>
<td>.06</td>
<td>.57**</td>
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<td>.26**</td>
<td>.05</td>
<td>.48**</td>
<td>.14</td>
<td>.42**</td>
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<td>.05</td>
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<td>14. IGT</td>
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<td>-.04</td>
<td>.04</td>
<td>-.10</td>
<td>.08</td>
<td>-.04</td>
<td>-.13</td>
<td>-.00</td>
<td>.07</td>
<td>-.01</td>
<td>.09</td>
<td>-.03</td>
<td>.05</td>
</tr>
</tbody>
</table>

*Note.* PA = positive affect; NA = negative affect; IGT = Iowa Gambling Task

*p < .05, **p < 0.01
Surprisingly however, the IGT was not correlated with any of the above mentioned executive function tasks. In fact, the IGT was not significantly correlated with any other primary variables, including any of the UPPS subscales. Investigation of the IGT frequencies revealed a normal distribution. Negative urgency and lack of premeditation were moderately correlated with one another. Negative and positive urgency were highly correlated with one another. Finally, negative urgency and negative affect were moderately correlated as well.

B. **Primary Analyses**

1. **Hypothesis 1. Executive Function and Self-Report Measures**

To determine whether the three groups differed on executive function and self-report measures, a Multivariate Analysis of Covariance (MANCOVA) was conducted with group (non-drinker vs. non-binge drinker vs. binge drinker) as the fixed factor, and each executive function task, each component of the UPPS, Negative and Positive Affect, and Negative and Positive Alcohol Expectancies as outcome variables, co-varying for total drug use, age, and education. Evaluation of Levene’s test of homogeneity of error variances indicated that the error variance of the following dependent variables may not be equal across groups ($p < .05$): Tower, Lack of Perseverence, Positive Alcohol Expectancies, Negative Alcohol Expectancies, Positive Affect, and Negative Affect. In this situation, a more conservative critical alpha level is suggested for determining significance for these variables. Therefore, a cutoff of .025 was used in place of .05 when determining significance for these variables (Tabachnick and Fidell, 2001).

Results from the MANCOVA were statistically significant according to Pillai’s, trace, $F(26, 242) = 2.34, p < .001$. Results of the ANOVA summary table for each dependent variable are displayed in **TABLE III**.
TABLE III
RESULTS OF ANOVA SUMMARY TABLE USING AUQ DEFINITION

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>p</th>
<th>ηp²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tower</td>
<td>85.313</td>
<td>2</td>
<td>42.657</td>
<td>2.843</td>
<td>.062</td>
<td>.041</td>
</tr>
<tr>
<td>Trails</td>
<td>4477.514</td>
<td>2</td>
<td>2238.757</td>
<td>2.119</td>
<td>.124</td>
<td>.031</td>
</tr>
<tr>
<td>Digit Span</td>
<td>17.412</td>
<td>2</td>
<td>8.706</td>
<td>1.634</td>
<td>.199</td>
<td>.024</td>
</tr>
<tr>
<td>Negative Urgency</td>
<td>227.771</td>
<td>2</td>
<td>113.885</td>
<td>2.116</td>
<td>.125</td>
<td>.031</td>
</tr>
<tr>
<td>Lack of Premeditation</td>
<td>190.998</td>
<td>2</td>
<td>95.499</td>
<td>3.642</td>
<td>.029</td>
<td>.052</td>
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<tr>
<td>Lack of Perseverence</td>
<td>119.155</td>
<td>2</td>
<td>59.578</td>
<td>2.430</td>
<td>.092</td>
<td>.036</td>
</tr>
<tr>
<td>Sensation Seeking</td>
<td>105.615</td>
<td>2</td>
<td>52.808</td>
<td>1.522</td>
<td>.222</td>
<td>.023</td>
</tr>
<tr>
<td>Positive Urgency</td>
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<td>103.338</td>
<td>2.338</td>
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<td>.034</td>
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<tr>
<td>Positive alcohol expectancies</td>
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<td>.179</td>
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<tr>
<td>Negative alcohol expectancies</td>
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<td>94.938</td>
<td>.506</td>
<td>.604</td>
<td>.008</td>
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<tr>
<td>Positive Affect</td>
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<td>2</td>
<td>2.773</td>
<td>.049</td>
<td>.952</td>
<td>.001</td>
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<tr>
<td>Negative Affect</td>
<td>363.884</td>
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<td>181.942</td>
<td>3.827</td>
<td>.024</td>
<td>.055</td>
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<tr>
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<td>2</td>
<td>8.895</td>
<td>.018</td>
<td>.982</td>
<td>.000</td>
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</tbody>
</table>

Contrary to predictions, executive function did not differ significantly across groups as evidenced by Trails B, Digit Span, and the IGT, but the Tower task trended towards significance.

Planned contrasts revealed that binge drinkers performed better on the Tower task than non-drinkers, $p = .008$, 95% CI [-0.64, -4.11]. Three factors emerged as statistically significant between groups: Positive alcohol expectancies, negative affect, and lack of premeditation.

Planned contrasts revealed that binge drinkers reported significantly greater positive alcohol expectancies compared to non-drinkers, $p < .001$ 95% CI [-33.33, -16.24], and non-binge drinkers reported significantly greater positive alcohol expectancies compared to non-drinkers, $p < .001$, 95% CI [-24.91, -8.98]. The comparison of binge drinkers’ and non-binge drinkers’ positive alcohol expectancies trended towards significance, $p = .055$, 95% CI [-15.85, 0.17]. In
terms of negative affect, non-drinkers reported higher negative affect than both non-binge drinkers, $p = .032$, 95% CI [-5.96, -0.28], and binge drinkers, $p = .046$, 95% CI [-6.16, -0.06]. Finally, binge drinkers reported greater lack of premeditation compared to non-binge drinkers, $p = .008$, 95% CI [-5.15, -0.79]. Lack of premeditation between binge drinkers and non-drinkers did not differ significantly. Lack of Perseverance also trended towards significance, $F(2, 132) = 2.43, p = .09$. Planned contrasts revealed that non-binge drinkers reported more perseverance (i.e., less lack of perseverance) than binge drinkers, $p = .03$, 95% CI [-0.227, -4.44].

Following the significant MANCOVA results, a discriminant function analysis was performed to determine if the executive function tasks and self-report measures significantly predicted group membership. Predictor variables were the Tower, Digit Span, Trails, IGT, each UPPS subscale, Positive Affect, Negative Affect, and Positive and Negative Alcohol Expectancies. A total of 12 cases were missing at least one discriminant variable, and were excluded from analyses.

Two discriminant functions were calculated, with a combined $X^2 (26) = 72.10, p < .001$. However, after removing the first function, there was no longer significant discriminating power, $X^2 (12) = 16.66, ns$, indicating that the group differences seen in the above MANOVA can be explained in terms of one underlying dimension. In fact, the first function accounted for 79.6% of the between group variance, canonical $R^2 = 0.59$, whereas the second function explained only 20.4%, canonical $R^2 = 0.35$. Upon closer inspection, it is apparent that the primary variable in distinguishing between binge, non-binge, and non-drinkers was having higher positive alcohol expectancies (see TABLE IV).
### TABLE IV

**STRUCTURE MATRIX RESULTS FROM DISCRIMINANT FUNCTION ANALYSIS USING AUQ DEFINITION**

<table>
<thead>
<tr>
<th></th>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive alcohol expectancies</td>
<td>0.78</td>
<td>-0.06</td>
</tr>
<tr>
<td>Tower</td>
<td>0.32</td>
<td>-0.16</td>
</tr>
<tr>
<td>Negative alcohol expectancies</td>
<td>0.28</td>
<td>0.01</td>
</tr>
<tr>
<td>Trails</td>
<td>-0.25</td>
<td>0.20</td>
</tr>
<tr>
<td>IGT</td>
<td>-0.12</td>
<td>0.03</td>
</tr>
<tr>
<td>Lack of premeditation</td>
<td>0.35</td>
<td>0.66</td>
</tr>
<tr>
<td>Lack of perseverance</td>
<td>0.19</td>
<td>0.61</td>
</tr>
<tr>
<td>Negative urgency</td>
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<td>Positive urgency</td>
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<tr>
<td>Sensation seeking</td>
<td>0.24</td>
<td>0.39</td>
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<tr>
<td>Digit span</td>
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<tr>
<td>Negative affect</td>
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</tr>
<tr>
<td>Positive affect</td>
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<td>-0.08</td>
</tr>
</tbody>
</table>

*Note*. Pooled within-groups correlations between discriminating variables and standardized canonical discriminant functions.

Lack of Premeditation, Tower, Negative Alcohol Expectancies, Trails, Sensation Seeking, and Digit Span, each moderately contributed to the first function. As seen in Figure 2, inspection of the group centroids indicates that the first function discriminates between non-drinkers, and the two groups of drinkers.
Figure 2. Canonical Discriminant Functions.

Note. 0=non-drinker; 1=non-binge drinker; 2=binge drinker.
2. **Hypothesis 2. National Institutes of Alcohol Abuse and Alcoholism Definitions**

2.1 **Hypothesis 2a. National Institutes of Alcohol Abuse and Alcoholism Dichotomous Definition of Binge Drinking**

To assess whether executive function and self-report measures differed as a function of binge drinking, according to the dichotomous NIAAA definition, a multivariate ANOVA was conducted with the NIAAA definition of binge drinking as the fixed variable, and each executive function task and self-report measure as predictor variables, controlling for education, age, and total drug use. As seen in TABLE V, several interesting results emerged.
TABLE V
RESULTS OF ANOVA SUMMARY TABLE USING THE DICHOTOMOUS NIAAA DEFINITION

<table>
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<tr>
<th>Dependent Variable</th>
<th>Sum of Squares</th>
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<th>Mean Square</th>
<th>$F$</th>
<th>$p$</th>
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</tbody>
</table>
Contrary to predictions, binge drinkers performed better on executive function tasks, specifically trails and digit span, compared to non-binge drinkers. Binge drinkers also had higher alcohol expectancies, both positive and negative, as well as better scores on the WTAR, but these results only trended towards significance. Looking back at the results from Hypothesis 1, it is evident that the dichotomous NIAAA definition yielded different results than the AUQ definition.

2.2 Hypothesis 2b. National Institutes of Alcohol Abuse and Alcoholism
Continuous Definition of Binge Drinking

To delve further into the comparison of different binge drinking definitions, I used the NIAAA definition of binge drinking to create a continuous outcome variable. Because the continuous NIAAA binge variable was positively skewed, these data were corrected by taking the logarithm of the NIAAA continuous score plus 1. Analyses were run using the transformed variable. Linear regression models were created with number of binge drinking episodes over the past 6 months as the dependent variable and each executive function task and self-report measure as the predictor variables. To control for education and total drug use, these variables were entered into the first block of the hierarchy, followed by the executive function tasks and self-report measures in the second block. Results indicated that neither the first, $R^2 = .03$, $ns$, or second model was significant, $R^2 = .12$, $ns$. Contrary to predictions, executive function, mood, and alcohol expectancies were not related to number of binge episodes over the past 6 months.


In applying the kindling theory of alcohol withdrawal to binge drinking, one assumes that binge drinking does in fact correspond to multiple withdrawals from alcohol. Thus, to assess this assumption, I conducted Pearson product-moment and biserial correlations to measure the
strength of the relationship between AUQ binge score, SAWS and NIAAA binge score (see TABLE VI).

<table>
<thead>
<tr>
<th>TABLE VI</th>
<th>CORRELATION MATRIX OF BINGE AND WITHDRAWAL VARIABLES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>1. AUQ binge score</td>
<td>1</td>
</tr>
<tr>
<td>2. NIAAA dichotomous</td>
<td>.162</td>
</tr>
<tr>
<td>3. Number of binge occurrences over past 6 months</td>
<td>.275**</td>
</tr>
<tr>
<td>4. Withdrawal symptom frequency</td>
<td>.380**</td>
</tr>
<tr>
<td>5. Withdrawal symptom severity</td>
<td>.311**</td>
</tr>
</tbody>
</table>

Note. * p < .05; ** p < .01.

Results indicated that, as expected, AUQ binge score was significantly correlated with both withdrawal symptom frequency and severity. However, the NIAAA dichotomous binge score was only significantly correlated with withdrawal symptom severity, but not frequency. Contrary to predictions, the NIAAA continuous binge score was not significantly correlated with withdrawal severity or frequency. Surprisingly, the dichotomous NIAAA binge score was not significantly correlated with the AUQ binge score or with the NIAAA continuous binge score.
V. DISCUSSION

Binge drinking is associated with a variety of harmful behaviors, social and legal problems, and serious health conditions. In epidemiological studies, the highest rates of binge drinking are consistently found to be among young adults, ages 18 - 26. A process that coincides with this age range is the final developmental phase of the frontal lobe, responsible for executive function, including decision making, planning ability, cognitive flexibility, and working memory. As binge drinking is associated with such dangerous consequences (e.g., risky behaviors, social and legal problems, etc.), one might wonder whether the underdeveloped executive function processes play a role in this seemingly contradictory behavior. Thus, it is logical to assess the relationship between binge drinking and executive function among young adults. Additionally, mood and alcohol expectancies impact alcohol and drug use as well. Therefore, the first aim of the present study was to examine the cognitive and emotional correlates of binge drinking in young adults.

The second aim of the present study was to investigate different definitions of binge drinking currently used in the scientific literature. Unfortunately, despite its widespread prevalence and associated costs to individuals and society, we lack an empirically supported, sole definition of this phenomenon. Yet, researchers attempt to study this phenomenon without a common, agreed upon definition. This leads to the question of whether all researchers are studying the same phenomenon if they employ different definitions. As such, the second aim of this study was to assess two commonly used definitions of binge drinking by employing these different definitions in different sets of analyses.
The current sample consisted of 50 binge drinkers, 50 non-binge drinkers, and 50 non-drinkers. Binge drinkers reported drinking more wine, beer, and spirits in a typical week, compared to non-binge drinkers. Binge drinkers also reported having been drunk an overwhelmingly greater number of times in the past 6 months compared to non-binge drinkers, as well as a wildly larger percentage of getting drunk in the times they drink. There are huge differences in the drinking habits of binge and non-binge drinkers in the current sample. These large differences between groups render results, or lack of results especially interesting, because the differences in drinking habits between groups are not slight. It is important to look at amounts and frequencies of drinking, rather than using a simplistic and incomplete definition of binge drinking.

A. Binge Drinking and Executive Function

The first hypothesis put forth in the present study was that binge drinkers would perform worse on the executive function tasks compared to non-binge drinkers and non-drinkers. Surprisingly, using both the AUQ and continuous NIAAA binge definitions, there was no significant relationship between executive function and binge drinking (although Tower results trended towards significance using the AUQ definition.) This is contrary to findings from many previous studies (Scaife & Duka, 2009; Townshend & Duka, 2005; Crego, et al., 2009; 2010). However, there is some supporting evidence for these results. Specifically, some studies have found no differences between binge drinkers and non-binge drinkers in reported memory lapses (Heffernan, Clark, Bartholomew, Ling, & Stephens, 2010; Heffernan & O’Neill, 2012), cognitive flexibility, planning and strategy-making skills, and spatial working memory (Parada, Corral, Mota, Crego, Holguin, & Cadaveira, 2012), and reaction time during the visual oddball task (Crego, Cadaveira, Parada, Corral, Caamano-Isorna, & Rodriguez Holguin, 2012). Thus, it
is not without precedence for binge drinkers and non-binge drinkers to perform similarly on executive function tasks. Mixed findings may be due, in part, to different definitions of binge drinking used in previous studies.

As a case in point, using the NIAAA dichotomous definition of binge drinking, markedly different results were revealed compared to using the AUQ definition. Specifically, binge drinkers actually performed better on executive function tasks compared to non-binge drinkers. Although we controlled for participants’ level of education, a recent study found that high school seniors with college-educated parents are more likely to binge drink (Patrick et al., 2013). Parents’ level of education was not assessed in the present study, but perhaps it would be an interesting avenue to pursue in the future.

One possible explanation for the present finding that binge drinkers performed better on executive function tasks compared to non-binge drinkers, may lie in the social contexts of binge drinking in young adulthood. According to social learning theory, social behaviors are learned from one’s environment and peers (Bandura, 1971). As we know, binge drinking is quite prevalent among 18-26 year olds. In order to be classified as a binge drinker according to the NIAAA definition, the participant only needed to have had one binge episode. Further, young adults perceive that their peers are engaging in heavy drinking even more than they actually are (Perkins, Meilman, Leichliter, Cashin, & Presley, 1999). Thus, perhaps engaging in this behavior, during a period of development when binge drinking is pervasive, is normative according to social learning theory, which states that one learns behavior by imitating those around him. Within this framework, participating in a binge-drinking episode during this phase of development would reflect learning by imitating the behavior of those around them. Negative consequences would likely also contribute to learning and future behavior, but because the
present criterion was only one binge episode, it is plausible that some participants classified as binge drinkers had not yet suffered consequences, or might have suffered consequences and then not engaged in this behavior again, but would nonetheless still be classified as a binge drinker under this definition. Further research into binge drinking within the context of social learning theory is needed to substantiate this question, but it presents an interesting starting point to focus on in future studies.

B. **Impulsivity**

As expected, binge drinkers reported the highest levels of lack of premeditation compared to the other two groups. This was true only using the AUQ definition. No differences between groups were seen using the NIAAA definition. Interestingly though, using the AUQ definition, this difference was only significant when comparing binge and non-binge drinkers. In contrast, there was no significant difference in lack of premeditation between binge drinkers and non-drinkers. This result appears curious initially. One feasible explanation is that perhaps non-drinkers and binge drinkers are alike in their amount of premeditation, but that the resulting action goes in opposite directions. In other words, in both groups, there is a lack of premeditation before action, however among non-drinkers, they do not premeditate because they almost automatically go to the conservative end, and know that they are not going to drink. Similarly, perhaps binge drinkers do not premeditate much, but instead go in the other direction, and tend to binge drink, without thinking much about it beforehand. Non-binge drinkers though, are caught in the middle of this spectrum. They are the ones who need to actually think and premeditate and make decisions such as deciding whether or not to drink, how much to drink, how quickly to drink, etc., because for this group of individuals, there is more ambiguity. They are not like the non-drinkers, and they are not like binge drinkers, who get drunk the majority of
the time that they drink. Examining this theory further poses an interesting, and potentially important, future research goal.

C. **Iowa Gambling Task**

There was no relationship between the IGT and binge drinking. Contrary to predictions, the present results suggest that in fact, binge drinkers displayed no impairment in decision making ability compared to those who do not binge drink. The total IGT score, as well as the first and last 50 tasks, did not yield significant results. Although this task has been found to distinguish between adolescent binge and non-binge drinkers (Johnson et al., 2008; Xiao et al., 2009), there is some literature that suggests that even among addicted individuals, there is a subgroup that performs normally on the IGT (Bechara, Dolan, & Hindes, 2002). Thus, our results would suggest that the group of binge drinkers in the present study were similar to this subgroup of substance addicted individuals who showed no impairment on the IGT.

It is interesting though, that the IGT was not correlated with any of the UPPS subscales, in particular, the premeditation subscale. According to Bechara (2013), there are three different types of impulse control that are each linked to different areas of the brain. One type of impulse control, as argued by Bechara, is decision making. This particular type of impulse control is measured by both the IGT and the UPPS premeditation subscale. Thus, it is surprising that the IGT and premeditation were not significantly correlated with one another in the present study, and additionally that premeditation was significantly related to binge drinking, whereas the IGT was not. Perhaps the participants did not fully understand the task or put forth their best effort. The IGT was the last executive function task to be administered. It is possible that participants were fatigued at this point, which impacted their effort on the task and/or performance.
D. **Expectancies**

Positive alcohol expectancies discriminated between binge drinkers, non-binge drinkers, and non-drinkers when using the AUQ definition, and trended towards significance using the dichotomous NIAAA definition. Binge drinkers held higher positive alcohol expectancies than non-binge drinkers and non-drinkers, and non-binge drinkers held higher positive expectancies than non-drinkers (using the AUQ definition; NIAAA has only 2 groups). This is consistent with previous research indicating that positive alcohol expectancies play a major role in the decision to drink, amount to drink, and binge drinking (Karlsson, 2012; Laighi, Balocco, D’Alessio, Bonachina, & Gurrieri, 2009; Morawska & Oei, 2005; Oei & Morawska, 2004; Schulenberg, Maggs, Long, Sher, Gotham, Baier, et al., 2001; Strano, Cuomo, & Venable, 2004). Positive alcohol expectancies have even been shown to predict greater number of binge drinking episodes 3 months later (Blume, Schmaling, & Marlatt, 2003). Specifically, some of the beliefs about alcohol that have been shown to predict binge drinking are those related to negative affect relief, social behavior, and approval from peers (Karlsson, 2012; Turrisi, Wiersma, Hughes, 2000)

Just as binge drinking prevalence decreases into adulthood, the impact of positive alcohol expectancies in binge drinking also decreases among adults compared to adolescents (Rooke & Hine, 2011). As the present study consisted of young adults, it is possible that the role of positive alcohol expectancies seen in the current study, may not be as strong as this sample grows into adulthood. Unfortunately, this supposition cannot be assessed using the present cross-sectional design. Assessing the role of positive alcohol expectancies on binge drinking across development poses an interesting future research goal.

Interestingly, using the dichotomous NIAAA definition, not only did positive alcohol expectancies trend toward association with binge drinking, but negative expectancies did as well.
At first glance, this appears strange. If one expects that alcohol will have negative consequences, then why would he choose to participate in this behavior to such excess? One possible explanation may lie in the fact that binge drinkers, using the NIAAA definition, have had at least one binge episode. Thus, not all individuals in this group are necessarily heavy drinkers. Second, even among frequent or heavy binge drinkers, it is possible that because the non-binge drinkers had such little experience with alcohol, that they really did not have many expectations, be them positive or negative. In other words, they may not necessarily think drinking alcohol leads to particularly good or bad consequences, because they do not have enough experience to draw a conclusion. Indeed, a large proportion of the NIAAA non-binge drinking group consisted of non-drinkers who had not had any alcohol in the past 6 months and who had never had a binge episode. Further, we excluded those individuals who reported problems with alcohol in the past, thus, the non-drinkers are not non-drinkers because of a previous alcohol problem. Future studies should continue to investigate the role of both positive and negative alcohol expectancies on binge drinkers and non-drinkers.

There is a growing body of literature that has found a relationship between binge drinking, expectancies, and impulsivity. Specifically, expectancies moderated the relationship between binge drinking and impulsivity such that impulsivity was positively related with binge drinking among young adults with average or high positive alcohol expectancies, but impulsivity and binge drinking were unrelated among those with low positive expectancies (Carlson & Johnson, 2012). Likewise, researchers found that trait urgency and expectancies were both related to drinking levels and problem drinking as well as another binge behavior, eating. The relationship between urgency and binge eating was moderated by expectancies, but the moderation of urgency and drinking fell short of significance (Fischer, Anderson, & Smith,
A future goal of the present research is to assess whether the relationship seen here between binge drinking and impulsivity, specifically, lack of premeditation, is affected by high or low alcohol expectancies.

E. **Negative Affect**

As expected, negative affect was associated with binge drinking. However, the direction of this effect was unexpected: non-drinkers displayed the highest levels of negative affect compared to both groups of drinkers. The relationship between binge drinking and negative affect was not significant using the NIAAA definition. Numerous studies have linked negative affect with binge drinking (Dawson, Grant, Stinson, & Chou, 2005; Ferriter & Ray, 2011; Fox, Bergquist, Gu, & Sinha, 2010; Hartley et al., 2004; Kuntsche, Knibbe, Gmel, & Engels, 2005; McNamara, Swaim, & Rosen, 2010), whereas others have not (Chassin, Pitts, & Prost, 2002; Randall, Elsabagh, Hartley, & File, 2004). Indeed, studies using different criteria to define binge drinkers, have found differing results with regards to negative affect. Thus, one contributing factor to these mixed results may certainly be the use of different binge drinking definitions.

Randall et al. (2004) found that teetotalers responded to cognitive testing with greater anxiety symptoms than drinkers. In the present study, affect was assessed after the executive function tasks. Possibly, the stress of completing the cognitive tasks impacted the affect ratings of the non-drinkers.

Another interesting possibility relates back to the discussion of social learning theory above (see Expectancies). Remember, drinking is quite prevalent in young adulthood. Setting aside binge drinking, non-binge drinking is even more common among young adults, with epidemiological studies estimating that about 70% of young adults use alcohol (NIAAA, 2006). Most college students view drinking as the norm and do not view their own drinking as
problematic (Eshbaugh, 2008; Piacentini & Banister, 2006). Thus, for those 18-26 year olds who do not participate in drinking at all, going against the norm may be difficult and associated with negative affect (Piacentini & Banister, 2006). Further, many social activities in this age group surround drinking (e.g., going to bars, parties that serve alcohol, “drinking games” among young adults). Perhaps by not drinking, these individuals are isolating themselves from social activities, which may lead to feelings of depression. The converse may also be true: that these individuals feel more depressed, leading to isolation and the absence of opportunities to drink socially.

F. **Binge Drinking Definitions**

It is clear from the present study that using different binge drinking definitions led to different results. Here, the AUQ definition yielded significant results regarding lack of premeditation, positive alcohol expectancies, and negative affect. Specifically, we found the following using the AUQ definition. First, binge drinkers and non-drinkers reported higher levels of (lack of) premeditation compared to non-binge drinkers. Second, binge drinkers and non-binge drinkers reported higher levels of positive alcohol expectancies compared to non-drinkers. And third, non-drinkers reported higher levels of negative affect compared to drinkers. In contrast, the NIAAA dichotomous definition yielded results regarding executive function tasks, and both positive and negative alcohol expectancies such that binge drinkers performed better on the executive function tasks and reported higher positive and negative alcohol expectancies compared to non-binge drinkers. Finally, the continuous NIAAA definition did not yield any significant findings. Adding to the complexity is the fact that the NIAAA dichotomous definition was not significantly correlated with either the AUQ definition or with the continuous NIAAA definition. We have also seen across past studies that using different definitions of
binge drinking leads to different conclusions. The lack of a consistent, unified definition of this phenomenon creates a serious obstacle to drawing conclusions across studies.

As to which definition is most accurate, further studies are needed. However, in the present study, the AUQ definition did provide an accurate portrayal of the drinking habits of binge and non-binge drinkers. According to the AUQ classification, binge drinkers drank significantly more wine, beer, and spirits in a typical week. Binge drinkers drank an average of 3 drinks per hour, compared to non-binge drinkers who drank an average of 1 drink per hour. Binge drinkers reported having been drunk an overwhelmingly greater number of times in the past 6 months compared to non-binge drinkers. Finally, binge drinkers reported less time between binge episodes as well as a much higher percentage of times getting drunk when they drink, compared to non-binge drinkers. Therefore, it does seem that using the AUQ definition provides an accurate picture of binge drinking. On the other hand, several studies have reported that the NIAAA definition of 5/4 drinks is an inaccurate portrayal of BAC, and actually tends to capture BACs lower than .08 (Lange & Voas, 2000; Perkins et al., 2001). Thus, if one were to judge based on these results, then it would seem that the AUQ provides a more accurate definition of binge drinking compared to the NIAAA definition. Further studies are needed to establish consistency with these results.

G. **Binge Drinking and Withdrawal**

The kindling theory assumes that binge drinking results in multiple withdrawals from alcohol, and that these multiple withdrawals cause deficits in executive function. However, withdrawal symptomatology is rarely assessed in past studies that invoke this theory. Thus, the present study assessed withdrawal symptomatology. Consistent with the kindling theory, both the AUQ and dichotomous NIAAA definitions were indeed associated with withdrawal. These
results support the kindling theory, and the notion that binge drinking is associated with multiple withdrawals from alcohol (Hunt, 1993). Nonetheless, it remains important for these results to be replicated, and to continue measuring withdrawal symptomatology in future studies.

H. Limitations

The present study has certain limitations. First, because a cross-sectional design was used, no causal relationships can be inferred. Future studies utilizing longitudinal methods are needed to properly assess the temporal order of the relationships between binge drinking, executive function, mood, and expectancies. Ecological Momentary Assessment would also be a useful tool to evaluate real-time data in terms of drinking speed and frequency, and withdrawal symptomatology.

The present study helped elucidate shortcomings of the current binge drinking literature; namely, the lack of a cohesive operational definition of binge drinking. Analyzing the present data with different definitions led to disparate results. This is an important first step in learning more about the present definitions used. However, it remains imperative to define this phenomenon appropriately, and to have one definition used by the entire scientific community.

Investigating the role of other drug use in binge drinking behavior is an important goal that was outside the scope of the current project. In the current study, we found that binge drinkers used more drugs in their lifetime and in the past 6 months compared to non-binge drinkers and non-drinkers. The present study controlled for other drug use. However, prior studies have found past 30-day drug use to predict binge drinking (Strano et al., 2004). Examining the role of other drug use, more generally, on executive function and mood is also an important task. Delving into the rich relationship between various drug use, binge drinking, and the mechanisms driving these behaviors is a worthy future research goal.
Researchers have found certain correlates of binge drinking among college students specifically, such as living in a fraternity or sorority, viewing parties as an important activity in college, and enjoying an on-campus social life (Wechsler, Dowdall, Davenport, & Castillo, 1995). The present study did not isolate college or non-college students within the 18-26 year age range. Further, of those participants who were in college in the present study, many attended a commuter school, without a large on-campus social presence. This, along with a lack of power, likely played a role in the lack of findings regarding current enrollment in college. Looking at more heterogeneous samples has its pros and cons, but investigating differences and similarities between college student and non-college student young adult binge drinkers may lead to interesting findings.

Finally, potential moderators were not addressed in the current study. One interesting path that has been studied is the potential role of impulsivity in the relationship between alcohol expectancies and binge drinking. Gender is another potential moderator of the relationship between binge drinking, executive function, expectancies, and mood. For the present study, I felt it was important to first gain a solid understanding of the correlates of binge drinking, and to help clarify binge drinking definitions before delving into moderational analyses. However, examining potential moderators of binge drinking in the current sample is a future research goal.
VI. CONCLUSIONS

One main, important conclusion of this study is that different definitions of binge drinking yielded different results, and that remarkably different people were classified as binge drinkers depending on the definition used. This is a serious problem in the field. How can we be sure of our conclusions about binge drinking if different studies may not actually be studying the same thing? It appears that first and foremost, the most important task ahead in the future of the binge drinking literature is to establish a clear, agreed upon definition of binge drinking.

That being said, the present study found that using the NIAAA dichotomous definition, binge drinkers actually performed better on executive function tasks than non-binge drinkers. Also under the NIAAA definition, binge drinkers had higher alcohol expectancies, both positive and negative, but this result did not quite reach significance. Conversely, using the AUQ definition, binge drinking and executive function were unrelated. Rather, using the AUQ definition, non-drinkers reported higher negative affect than both binge and non-binge drinkers, and binge drinkers reported higher lack of premeditation than non-binge drinkers. However, the most important predictor of binge drinking using the AUQ definition, proved to be having greater positive alcohol expectancies.
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