ABSTRACT

Title of dissertation: IDENTIFYING MECHANISMS UNDERLYING THE ASSOCIATION BETWEEN ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND PROBLEMATIC ALCOHOL USE IN COLLEGE STUDENTS

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Students with ADHD represent a significant population on college campuses who have been found to be at risk for problematic alcohol use. Yet little is known about mechanisms underlying the association between ADHD and alcohol-related problems. We examined patterns and consequences of alcohol use in college students with and without ADHD, as well as three possible mediators of the association between ADHD and alcohol-related impairment: (1) self-reported difficulty stopping a drinking session, (2) cue dependency on a behavioral task of response inhibition, and (3) self-reported trait disinhibition. Participants with ADHD reported higher rates of negative consequences of alcohol use relative to the non-ADHD group, despite equivalent rates of alcohol use. In addition, the ADHD group had higher rates of difficulty stopping a drinking session. Difficulty stopping a drinking session mediated the relationship between ADHD and

negative consequences of alcohol use. Cue dependency and trait disinhibition did not mediate this relationship. These findings indicate that college students with ADHD are experiencing higher rates of negative consequences of alcohol use relative to their peers without ADHD. Difficulty stopping a drinking session may be one mechanism that explains the relationship between ADHD and alcohol-related problems.

# IDENTIFYING MECHANISMS UNDERLYING THE ASSOCIATION BETWEEN ATTENTION-DEFICITY/HYPERACTIVITY DISORDER AND PROBLEMATIC ALCOHOL USE IN COLLEGE STUDENTS

By

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## Introduction

Developmental models of psychopathology identify critical transition periods during which an individual's ongoing trajectory of health and well-being is vulnerable to change (Mash & Dozois, 2003). The college years, occurring during the transition between adolescence and young adulthood, represent a time when the initiation and escalation of heavy drinking may set the stage for lifelong difficulties with alcohol and other drugs (Maggs, 1997). According to developmental models, pathways to the development substance use disorders (SUD) arise from the interaction between an activating environment and within-individual vulnerabilities (Tarter, Vanyukov, & Kirisci, 2008). The college campus, with its unique drinking norms (Wechsler, Kuo, Lee, & Dowdall, 2000), may be an activating environment for students at risk for the development of SUD. Attention-deficit/hyperactivity disorder (ADHD) and behavioral disinhibition, two known risk factors for problematic alcohol use (Wilens, 2004), may represent an intraindividual vulnerabilities that interact with college-specific environmental factors to create a pathway for the development of alcohol-related problems. Drawing upon a behavioral control model of alcohol use (Fillmore, 2003), the current study examines behavioral disinhibition as a specific vulnerability that may contribute to (and partially explain) alcohol-related impairment among college students with ADHD.

ADHD is characterized by developmentally inappropriate inattention and hyperactivity/impulsivity resulting in academic and social impairment (APA, 2000). Recent theories have attributed the core symptoms of ADHD (i.e., inattention, hyperactivity, and impulsivity) to deficits in executive functioning (e.g., difficulties with

behavioral inhibition, planning, goal persistence, and working memory; Barkley, 1997; Nigg, 2001). Many of these deficits have also been identified as risk factors for problematic alcohol use, independent of ADHD (Lyvers, Czerczyk, Follent, & Pheobe, 2009; Martel et al., 2007; Nigg et al., 2004). In addition, the impairments associated with the persistence of ADHD symptoms into adolescence amplify the risk for alcohol-related problems in this population. These impairments include difficulty adapting to increasing academic demands, interpersonal problems, and a persistent reliance on external sources of structure (e.g., parents) during a time when such external support is not considered developmentally normative (Pelham & Fabiano, 2008; Smith, Waschbusch, Willoughby, & Evans, 2000). Many of these impairments have been proposed as mechanisms underlying the association between ADHD and the development of problematic alcohol use. These proposed mechanisms, however, have largely gone unstudied. Alternatively, executive functioning deficits, such as behavioral disinhibition, that are central to theories of both ADHD and problematic alcohol use may represent alternative mechanisms that explains the relationship between ADHD and alcohol-related problems.

Problematic alcohol use tends to peak during late adolescence/early adulthood and declines throughout the mid-to-late twenties (Dawson, Grant, Stinson, & Chou, 2004; Johnston, O'Malley, Bachman, & Schulenberg, 2009). The college years overlap developmentally with both the peak period of alcohol use (Johnston et al., 2009) and the stage of life when personality traits, such as impulsivity or disinhibition, are relatively unstable (Roberts et al., 2006). Thus, the trajectory for the development of problematic alcohol use may be ripe for change during this transitional period. Identifying factors that affect this trajectory for vulnerable college students, such as those with ADHD, is crucial

to understanding which students may likely experience alcohol-related problems during the college years and into adulthood. In addition, the knowledge about underlying causal mechanisms has the potential to contribute to the development of targeted interventions aimed at modifying factors that are amenable to change. The majority of individuals *mature out* of problematic alcohol use during their mid- to late-twenties (Bachman, Freedman-Doan, O'Malley, Schulenberg, & Johnston, 2008; Christo, 1998; Donovan, Jessor, & Jessor, 1983; Fillmore, Carscadden, & Vogel-Sprott, 1998; Jessor, Donovan, & Costa, 1991; Littlefield, Sher, & Wood, 2009). This normative decline in alcohol use has been associated with the assumption of adult roles and responsibilities incompatible with excessive drinking, such as marriage, parenthood, and full-time employment (Bachman et al., 2008; Bachman et al., 2002; Gotham, Sher, & Wood, 2003; Leonard & Rothbard, 1999). This hypothesis has received empirical support, and life transitions and social roles likely influence patterns of alcohol use (Bachman et al., 2008). In addition, an alternative hypothesis focused on changes in personality traits (e.g., impulsivity, neuroticism, extraversion) during the transition between adolescence and young adulthood has recently been examined (Littlefield et al., 2009).

Traditionally, personality traits have been considered unchanging internal characteristics (Costa & McCrae, 1995; Dick et al., 2010; McCrae et al., 1999; McCrae et al., 2000). More recently, however, these traits have come to be viewed as dynamic constructs which change systematically over the course of development (Johnson, Hicks, McGue, & Icano, 2007; Roberts, Walton, & Viechtbauer, 2006). Typically, individuals become more socially dominant, conscientious, and emotionally stable as they age (Caspi, Roberts, & Shiner, 2005; McCrae et al., 1999). In addition, a recent meta-analysis

found that personality traits changed more during late adolescence and young adulthood than during any other period (Roberts et al., 2006). Within the context of a large longitudinal study, which assessed participants 6 times between the ages of 18 and 35, Littlefield and colleagues (2009) examined the relationship between developmentally normative changes in personality and the developmental pattern of *maturing out* of problematic alcohol use. The authors found that changes in the personality traits of impulsivity and neuroticism were associated with the normative decline in alcohol use. Specifically, as impulsivity and neuroticism decreased over time, problematic alcohol use decreased as well. In addition, the authors found that while marriage and becoming a parent both influenced changes in neuroticism and problematic alcohol use, they did not sufficiently explain the association between personality and alcohol use. This study appears to be the first to identify changes in personality traits that partially explain the developmentally normative process of maturing out of alcohol-related problems. While researchers are only beginning to examining mechanisms underlying developmental patterns in alcohol use, a well-established literature identifies late adolescence and early adulthood as the peak periods of alcohol use. These developmental periods overlap with the college years and may represent a key time point for alcohol use remediation and prevention measures.

### *Patterns of Alcohol Use in College Students*

A wealth of information about college student drinking behavior has been collected through several large-scale studies (Johnson et al., 2009; Presley, Cheng, & Pimentel, 2004; Wechsler et al., 2002). Across studies college students have been found to engage in higher rates of heavy episodic drinking than their non-college peers

(Johnson et al., 2009; Wechsler et al., 2002). The Monitoring the Future study (an ongoing study of behaviors, attitudes, and values of American secondary school students, college students, and young adults) provides valuable comparison data on patterns of alcohol use among college students and their same-age peers who are not attending college (Johnston et al., 2009). While alcohol use is prevalent among late adolescents and young adults, results from the Monitoring the Future study generally show riskier patterns of use among college students relative to their non-college peers. Specifically, college students report a significantly higher rate of having used alcohol during the past 30 days relative to their non-college peers (69% vs. 55%) and report a greater incidence of having ever been drunk during the past 30 days (45% vs. 31%). Heavy episodic drinking constitutes a particularly hazardous pattern of alcohol use that is associated with an increased risk for accidental injury, unplanned and unsafe sex, and a host of other social and psychological problems (Wechsler, Davenport, Dowdall, Moeykens, & Castillo, 1994; Wechsler, Lee, Kuo, & Lee, 2000; Wechsler et al., 2002). Across studies, college students have a significantly higher prevalence of heavy episodic drinking (defined as five or more drinks on one occasion) during the past two weeks relative to their non-college peers (40% vs. 30%). In addition, fraternity and sorority members consistently report rates of heavy episodic drinking that are higher than those of the general college student population (Bartholow, Sher, & Krull, 2003; Borsari & Carey, 2006; Borsari & Carey, 1999; Capone, Wood, Borsari, & Laird, 2007; Cashin, Presley, & Meilman, 1998; Larimer, Irvine, Kilmer, & Marlatt, 1997; Lo & Golobetti, 1995; Rabow & Duncan-Schill, 1995; Sher, Bartholow, & Nanda, 2001).

There is evidence to suggest that environmental factors specific to college campuses influence patterns of alcohol use among students. Findings from the Harvard School of Public Health College Alcohol Study, which included more than 100 colleges in four national surveys spanning a 12-year period, revealed that heavy episodic drinking (i.e., binge drinking) varies by college (ranging from 1% of students to 76%; Wechsler et al., 2002). Within colleges, however, rates of heavy episodic drinking have remained stable over time (Wechsler et al., 2002). This suggests that environmental factors (e.g., type of residence, price of alcohol, density of alcohol outlets, and prevailing drinking rates at the college) influence drinking patterns among college students (for a review see Wechsler & Nelson, 2008). The Monitoring the Future research group suggests that the *role incompatibility* hypothesis (Yamaguchi & Kendel, 1985) may partially also explain differences in drinking patterns between college students and their non-college peers (Johnson et al., 2009). Specifically, traditional college students are less likely than their non-college peers to be married and are more likely to move away from home without first obtaining full-time employment, two factors that have repeatedly been shown to decrease the likelihood of heavy drinking (Bachman et al., 2008; Bachman et al., 2002; Leonard & Rothbard, 1999). In summary, patterns of alcohol use among college students differ from those of their non-college peers. College students engage in higher rates of heavy episodic drinking and drink more frequently than their peers who are not attending college. In addition, late adolescence and early adulthood represent developmental time points when hazardous alcohol use is at its peak. For students who may be predisposed to the development of alcohol-related problems, such as those with ADHD, the college

years may represent a key developmental period when alcohol prevention and intervention efforts are needed.

*College Students with ADHD*

Advances in the use and effectiveness of psychosocial and pharmacological interventions and increased legislative support[1] have made higher education more accessible for students with ADHD (Gallagher, Sysko, & Zhang, 2001). While specific data on the prevalence of college students with ADHD is not available, preliminary findings suggest that students with ADHD represent a significant population on college campuses. Specifically, 2 - 8% of college students self-report symptoms consistent with a diagnosis of ADHD (for a review see Weyandt & DuPaul, 2008) and up to 10% of college students report elevated levels of current ADHD symptoms (Garnier-Dykstra, Pinchevsky, Caldeira, Vincent, & Arria, 2011). These rates are comparable to United States general population prevalence estimates, which indicate that approximately 7.8% of children aged 4 – 17 have received a diagnosis of ADHD (Centers for Disease Control and Prevention, 2005). College students with ADHD may represent a unique subgroup of the broader ADHD population given that they have thus far achieved a relatively high level of academic functioning (DuPaul, Weyandt, O'Dell, & Varejao, 2009). Studies suggest that relative to their peers with ADHD who are not attending college, college students with ADHD have greater cognitive abilities, greater past experience with academic success during grade school, and better coping skills (Glutting, Youngstrom, & Watkins, 2005). Despite this relative advantage, college students with ADHD appear to have more academic and interpersonal difficulties than their peers without ADHD (for a

[1] Section 504 of the Rehabilitation Act of 1973 and the Americans with Disabilities Act of 1990

review see DuPaul, Weyandt, O'Dell, & Varejao, 2009). In addition, college students with ADHD are likely at increased risk for problems with alcohol and illicit drugs (Rooney et al., 2011). These impairments, in addition to the core symptoms of ADHD, may make it difficult for these students to successfully meet the challenges associated with college attendance (e.g., moving away from home, making autonomous decisions, establishing a new peer group, managing unstructured social and academic environments; DuPaul, Weyandt, O'Dell, & Varejao, 2009; Weyandt & DuPaul, 2008). In addition, these impairments may exacerbate the vulnerability for alcohol-related problems in students with ADHD.

*Patterns of Alcohol Use in Adolescents, Young Adults, and College Students with ADHD*

Despite the fact that the college years are a developmentally-critical time period, few studies have examined alcohol use among college students with ADHD. In contrast, a substantial literature exists on non-college adolescents and young adults with ADHD. A majority of these studies are longitudinal in design and benefit from having assessed participants during childhood, a time when ADHD is most easily and accurately diagnosed according to DSM criteria (APA, 2000; Pelham, Fabiano, & Massetti, 2005). With regard to the assessment of substance-related problems, however, these studies suffer from a number of weaknesses. Early longitudinal research in this area frequently relied exclusively on a diagnosis of alcohol use disorder (AUD) as an indicator of alcohol-related problems (Molina, Pelham, Gnagy, Thompson, & Marshall, 2007). This practice may have resulted in an underestimation in the rate of alcohol-related problems because the age of participants at the time of assessment (i.e., approximately 15 years)

was often below the age range associated with peak risk for AUD (Grant & Dawson, 1997). Thus, an AUD diagnosis may not have been a developmentally appropriate indicator of problematic alcohol use (Molina et al., 2007). Not surprisingly, these early studies did not find differences in rates of AUD between adolescents with ADHD and without ADHD (Gittelman et al., 1985; Hartsough & Lambert, 1987).

More recently, longitudinal studies have aimed to incorporate more developmentally-sensitive indicators of problematic alcohol use among adolescents (e.g., age of first use, frequency of use, hazardous patterns of use; DeWit, Adalf, Offord, & Ogborne, 2000; Grant & Dawson, 1997; Warner & White, 2003). These recent studies have found that adolescents with ADHD typically begin using alcohol at an earlier age (Molina & Pelham, 2003), use alcohol with greater frequency, and engage in more hazardous patterns of use (e.g., have 5 or more drinks in a sitting) than adolescents without ADHD (Molina & Pelham, 2003; Langley et al, 2010). In addition, when compared to age-matched controls without ADHD, Molina and colleagues (2007) found higher rates of AUD among ADHD participants between the ages of 15-17, but not among ADHD participants ages 13-15. Longitudinal studies have also identified riskier patterns of alcohol use in young adults with confirmed ADHD diagnoses relative to those without ADHD (Weiss & Hechtman, 1993; Wilens et al., 1998; Wilens et al., 2002). Young adults with ADHD were significantly more likely to abuse alcohol or have alcohol use disorder (Smith, Molina, & Pelham, 2003; Weiss & Hechtman, 1993) than their peers without ADHD. In contrast, young adults with ADHD did *not* appear to use alcohol at higher rates than those without ADHD (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman et al., 1997). This finding may be explained by the fact that increased alcohol

use is normative during young adulthood in the United States (Johnston et al., 2009; SHAMSA, 2009). In contrast, behavior patterns that constitute alcohol abuse or AUD are not developmentally normative and are, by definition, associated with impairment in functioning.

In summary, early studies of alcohol use among adolescents with confirmed ADHD diagnoses did not find clear evidence of higher rates of alcohol use disorders in this group. Findings from more recent studies, which incorporated developmentally-sensitive measurements, suggest that adolescents and young adults with ADHD are more likely to engage in patterns of alcohol use indicative of current or future alcohol-related problems. A number of limitations, however, are not addressed in the existing literature. These include (1) a reliance on predominately male samples, (2) a reliance on samples of longitudinal study participants who were diagnosed with ADHD in childhood and who may or may not have continued to meet diagnostic criteria during adolescence, and (3) a failure to consider college enrollment status when examining patterns of alcohol use. As outcomes may differ as a function of gender and current diagnostic status (Barkley, 1998; Monuteaux, Faraone, Gross, & Biederman, 2007), and college students engage in patterns of alcohol use that differ from those of their non-college peers (Johnston et al., 2009; Wechsler & Nelson, 2008), future studies should address these additional limitations. Lastly and perhaps most importantly, while a number of studies have examined differences in patterns of alcohol use between individuals with and without ADHD, few studies have examined mechanisms underlying the association between ADHD and problematic alcohol use.

To the best of our knowledge, our preliminary study was the first to examine patterns of alcohol use in college students with confirmed ADHD diagnoses that were based on a comprehensive assessment using Diagnostic and Statistical Manual – Version 4, Text Revision (DSM-IV-TR; APA, 2000) criteria (Rooney et al., 2011). We found that ADHD was associated with significantly higher scores on a global measure of alcohol-related impairment (the Alcohol Use Disorders Identification Test – AUDIT; Saunders, Aasland, Babor, De La Fuenta, & Grant, 1993), an increased risk for alcohol dependence or emerging dependence, and higher rates of negative consequences associated with alcohol use (i.e., having a hangover, getting into an argument or fight, getting nauseated or having vomited, being criticized by someone due to drinking or drug use, experiencing memory loss, doing something one later regretted, being hurt or injured). In addition, self-reported difficulty stopping a drinking session mediated the association between ADHD diagnosis and the negative consequences of alcohol use, suggesting that behavioral inhibition may represent a causal mechanism that warrants further investigation. Consistent with longitudinal studies examining the frequency of alcohol consumption among young adults with ADHD (Weiss & Hechtman, 1993; Wilens et al., 1998; Wilens et al., 2002), college students with ADHD did not report consuming alcohol with greater frequency or in larger quantities than their peers without ADHD (Rooney et al., 2011). As noted by Smith, Molina, and Pelham (2002), the finding that young adults with ADHD are not consuming alcohol with greater frequency or in larger quantities than their peers should not be interpreted as evidence that this group is not consuming alcohol at high rates. Rather, in the United States, high rates of alcohol consumption are *normative* during this developmental stage (SHAMSA, 2009), and young adults with

ADHD appear to be "keeping up with" their non-disordered peers. Given this finding, it does not appear to be the amount of alcohol consumed by college students with ADHD that puts them at risk for alcohol-related problems. Rather, this population appears to be more vulnerable to developing alcohol dependence or experiencing other negative consequences associated with use.

*Behavioral Disinhibition*

Across the lifespan behavioral disinhibition is associated with both ADHD the development of alcohol-related problems (Homack & Riccio, 2004; Iacano, Carlson, Taylor, Elkins, & McGue, 1999; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Nigg et al., 2004; Romine et al., 2004; Sher, Grekin, & Williams, 2005; Stacy & Newcomb, 1998; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). As a construct that is independently associated with both problematic alcohol use and ADHD, behavioral disinhibition may represent one mechanism which underlies the association between ADHD and alcohol-related problems.

*Behavioral Disinhibition: Construct Definition and Measurement*

As a construct, behavioral disinhibition is loosely defined and is often used interchangeably with impulsivity or behavioral undercontrol (Dick et al., 2010; Sher, Bartholow, & Wood, 2000; Whiteside & Lynam, 2001). In addition, there are both behavioral- and personality trait-based approaches to the definition and measurement of disinhibition (for a review see Dick et al., 2010). According to the behavioral approach, disinhibition is broadly defined as an impaired ability to ignore irrelevant information or suppress inappropriate responses to environmental cues, particularly those that are habitual or ongoing (Barkley, 1997; Quay, 1997). Researchers employing a behavioral

approach frequently utilize laboratory performance tasks that are thought to measure variability in the state-based *cognitive* processes that may underlie disinhibited behavior (Logan & Cowan, 1984; Nigg, 2001; Pennington & Ozonoff, 1996). The trait-based approach defines behavioral disinhibition as a personality trait encompassing both *affective and cognitive* processes that contribute to individual differences in ways of responding to and perceiving the world (Dick et al., 2010). Researchers employing the trait-based approach typically utilize self-report questionnaires to measure behavioral disinhibition. Currently there are a variety of self-report questionnaires available, many of which contain subscales measuring facets of disinhibition (for a review of self-report questionnaires see Reynolds, Ortengren, Richards, & de Wit, 2006).

A growing body of evidence suggests that no single personality trait fully encompasses the construct of behavioral disinhibition (Smith et al., 2007; Whiteside & Lynam, 2001). Recent models have instead incorporated three moderately correlated classes of personality traits: emotion-based traits, conscientiousness-based traits, and sensation seeking-based traits (Cyders et al., 2007; Whiteside & Lynam, 2001). Emotion-based traits include *positive-urgency*, the tendency to act impulsively when experiencing a euphoric mood, and *negative urgency*, the tendency to act impulsively when experiencing a negative mood. Conscientiousness-based traits include *lack of planning* and *lack of perseverance*, which manifests as difficulty tolerating boredom or remaining focused in the face of distraction. Sensation seeking does not include any sub-types, and is described as a tendency to seek out novel or thrilling stimulation. Each of these subtypes has been found to be associated with risky patterns of alcohol use (Magid, MacLean, & Colder, 2007; Nagoshi, 1999; Nagoshi, Wilson, & Rodriguez, 1991).

Sensation seeking, in contrast with other facts of impulsivity, has been found to differentially predict alcohol related problems rather than rate of alcohol use (Magid et al., 2007). The identification of this three-class model represents significant progress toward an empirically based understanding of the disinhibition construct (Dick et al., 2010). Since this model was developed through an analysis of existing measures of impulsivity, it allows for measures used in previous studies to be placed into one of the three classes. This empirically-based model also provides an enhanced structure for the study of external correlates specific to each of the three classes of traits, as well as differences in the genetic and neurobiological etiology of of disinhibition (Cyders et al., 2007; Dick et al. 2010, Smith 2007; Whiteside et al., 2005). Taken together, recent advancements in our understanding of the disinhibition construct have the potential to refine and expand our understanding of how this personality trait contributes to the development and maintenance of a wide spectrum of problematic behaviors and disorders.

There are both positive and negative aspects to both behavioral and trait-based measurements of disinhibition (Kazdin, 2003). Self-report questionnaires have the benefit of capturing both cognitive and affective responses and can inquire about behavior in real-world settings. They are, however, particularly susceptible to informant bias. In addition, when multiple variables in a single study are measured using self-report questionnaires, correlations among variables may be inflated by shared method variance. Behavioral tasks are performance based and are generally not dependent on participant self-perception. As a result, both informant bias and shared method variance are minimized when comparing behavioral task performance to self-report questionnaire

responses. Laboratory tasks, however, measure behavior in controlled settings, and laboratory task performance may or may not generalize to behavior in real-world settings. In addition, it is often unclear which specific cognitive processes influence performance on any one behavioral task. It is possible, even likely, that cognitive processes other than those of interest are captured by behavioral task performance. This can create challenges when interpreting findings. Until researchers develop a more homogenous definition of behavioral disinhibition and are able to clearly delineate the cognitive processes captured by behavioral tasks performance, it may be prudent to incorporate both behavior-based and trait-based measurements in study designs.

*Behavioral Disinhibition and Alcohol-Related Problems in Normative Samples*

Disinhibition has consistently been associated with problematic alcohol use. Prospective studies have identified behavioral disinhibition during childhood and adolescence as a risk factor for the development of alcohol use disorders (Iacano, Carlson, Taylor, Elkins, & McGue, 1999; King et al., 2009; Nigg et al., 2006; Sher, Bartholow, & Wood, 2000). Recent evidence also suggests that genetic variance in behavioral disinhibition accounts for a significant proportion of the genetic variance in alcohol dependence (Slutske et al., 2002; Schuckit, 2009). In cross-sectional studies, individuals meeting criteria for alcohol use disorder score high on both self-report (Sher, Grekin, & Williams, 2005; Trull, Waudby, & Sher, 2004) and laboratory measures (Hildebrant, Brokate, Eling, & Lanz, 2004; Van der Linden et al., 2007) of disinhibition. These findings must be interpreted with caution, however, since long-term alcohol use can result in changes in cognitive functions, including disinhibition (Bartsch et al., 2007). Alcohol consumption also acutely increases disinhibition in healthy adults (for a review

see Fillmore, 2003). The acute effects of alcohol on behavioral disinhibition have been studied using laboratory tasks based on the stop signal paradigm (de Wit, Crean, & Richards, 2000; Easdon & Vogel-Sprott, 2000; Fillmore & Van Selst, 2002).[2] These studies have consistently found that even moderate doses of alcohol reduced the drinkers' ability to inhibit behavior (Fillmore, 2003; Marczinski, Abroms, Van Selst, & Fillmore, 2005). For those with ADHD, the degree of impairment may be even greater since ADHD is associated with inhibitory deficits in sober individuals. This hypothesis, which has yet to be tested, may explain results from our preliminary study which showed that, despite equivalent rates of self-reported alcohol consumption, college students with ADHD report more negative consequences from alcohol use than college students without ADHD (Rooney et al., 2011).

While studies of stop task performance demonstrate that alcohol impairs the ability to inhibit a behavioral response in healthy participants, these studies do not provide information about the environmental conditions that may exacerbate or attenuate the disinhibiting effects of alcohol (Fillmore, 2003). Fillmore notes that with respect to response inhibition, the environment likely exerts some stimulus control over behavioral responding.

[2] Stop-signal tasks require participants to respond as quickly as possible to a "go" target stimulus. Intermittently the go target is followed by a second target stimulus, the "stop" target. The stop target signals the participant to withhold the prepared "go" response. The stop task assesses two processes: the reaction process ("go") and the inhibitory process ("stop") (Logan, 1994).

Researchers using the cued go/no-go task,[3] a laboratory task based on the stop signal paradigm, have identified a cognitive mechanism termed *cue dependency* that appears to protect individuals against the disinhibiting effects of alcohol. Within controlled laboratory settings the pre-response cues on the cued go/no-go task may mimic the real-world environmental cues that trigger the preparatory cognitive processes necessary for effective behavioral inhibition or behavioral activation of responses to environmental stimuli (Fillmore, 2003). Recent studies have utilized cued go/no-go tasks to assess the influence of preresponse cues on the ability of healthy adults (ages 22 – 29) to activate or inhibit a behavioral response while under the influence of alcohol (Abroms, Marczinski, Fillmore 2003; Marczinski & Fillmore, 2005; Fillmore, 2003; Marczinski & Fillmore 2003; Miller; Fillmore, 2004). These studies have consistently shown that alcohol produces a dose-dependent increase in inhibitory failures on trials with inconsistent cue-target pairings (i.e., a go cue followed by a no-go target). On these trials, participants have begun the preparatory process for *executing* a response based on the information provided by the preresponse cue. Once the no-go target is presented they must act against their preparation, and *withhold* a response. These same studies have also consistently found that alcohol does not produce an increase in inhibitory failures on trials of consistent cue-target pairings (a no-go cue followed by a no-go target). On these

[3] On the cued go/no-go task participants are presented with either a "go" or "no-go" target and are expected to execute or withhold a response. Each target (go or no-go) is preceded by preresponse cue which provides information about the nature of the upcoming target. Go cues indicate that there is an 80% chance the upcoming target will be a go target. Therefore, participants should prepare to *execute* a response to the target. No-go cues indicate that there is an 80% chance that the upcoming target will be a no-go target. Therefore, participants should prepare to *inhibit* or *withhold* a response to the target. On 20% of trials, the cue-target parings are inconsistent. Reliance on preresponse cues, or *cue dependency*, is operationalized as having fewer errors on trials with consistent cue-target pairings and more errors on trials with inconsistent cue-target pairings.

trials, participants have begun the preparatory process for *withholding* a response. Once the no-go target is presented, they act in accordance with their preparation and in fact *withhold* a response. This same pattern of responding was displayed for reaction-time trials, where participants are expected to *execute* a response when the go target is presented. Alcohol slowed reaction time in a dose-dependent manner on trials of inconsistent cue-target parings (a no-go cue followed by a go target), but had no effect on reaction time on trials of consistent cue-target pairings (a go cue followed by a go target).

Participants who respond differentially based on cue condition are displaying a *cue dependency* effect. As the dose of alcohol increases, the participants' level of cue dependency increases. That is, the difference in error rates between trials of consistent cue-target parings and trials of inconsistent cue-target parings increases as a function of alcohol dose. This increase in cue dependency occurs because the participants' behavior becomes increasingly disinhibited on inconsistent cue-target trials, and therefore, the error rate on these trials increases. Simultaneously, participants' behavioral disinhibition remains relatively stable on consistent cue-target trials. Therefore, the degree of difference in the level of behavioral disinhibition displayed between the two cue-target pairing conditions increases as a function of alcohol dose in healthy adults. This suggests that in order to maintain appropriate inhibitory responding under the impairing effects of alcohol, the drinker must increase his/her reliance on the immediate environmental context (Fillmore, 2003; Marczinski & Fillmore, 2005). Increased reliance on the environmental context under alcohol conditions may be a compensatory mechanism that protects against the disinhibiting effects of alcohol in healthy adults (Marczinski & Fillmore, 2005). Given that alcohol slows information processing (Fillmore et al., 1998;

Fillmore & Vogel-Sprott, 1997; Mitchell, 1985; Moskowitz, Burns, & Williams, 1985), it is not surprising that reliance on environmental cues, which increase the amount of time available for responding by enabling early response preparation, improves response inhibition task performance (Marczinski et al., 2005; Posner, Snyder, & Davidson, 1980). In fact, prior research has demonstrated that multiple factors known to slow information processing speed simultaneously increase dependency on environmental cues (Fillmore & Van Selst, 2002). For example, conditions that increase the cognitive load (e.g., increased task complexity, divided attention; for a review, see Pashler & Johnston, 1997) and factors that slow the rate at which information is processed (e.g. age-related cognitive decline, low working memory capacity; Laver, 1993; Finn, Justus, Mazas, & Steinmetz, 1999) also increase reliance on environmental cues. Therefore, alcohol's effect on information processing speed is one of many factors that should be considered in studies examining associations between response inhibition and alcohol-related problems.

*Behavioral Disinhibition and ADHD*

Behavioral disinhibition is central both to theories of problematic alcohol use and theories of ADHD (Barkley, 1997). Thus, when seeking to identify mechanisms that may underlie the impairing effects of alcohol in individuals with ADHD, a model emphasizing the role of behavioral disinhibition represents a viable starting point (Weafer, Fillmore, & Milich, 2009). Researchers have posited a variety of theories to describe the cognitive underpinnings of ADHD symptomatology (for review, see Castellanos & Tannock, 2002). Most recent theories emphasize higher-order cognitive processes controlled by the frontal lobes such as working memory (Rapport, Chung, Shore, & Issacs, 2001), delay aversion (Sonuga-Barke, 2002), and behavioral inhibition (Barkley, 1997). These

processes fall under the umbrella category of executive function. While there is currently no consensus definition of "executive function" (Castellanos & Tannock, 2002), the term is broadly used to describe a wide range of top-down cognitive processes that enable flexible, goal- directed behavior. Examples of these processes include planning, initiating and discontinuing actions, inhibiting habitual or prepotent responses, performance monitoring, and set shifting.

Barkley's (1997) comprehensive theory of ADHD proposes that deficient inhibitory control, or behavioral disinhibition, is the core feature of ADHD that disrupts other secondary executive function processes and leads to downstream effects that result in the characteristic behavioral and academic impairments seen in ADHD. Over the past decade, a substantial body of literature has supported Barkley's theoretical model and has made behavioral disinhibition the most studied of the executive deficits known to be associated with ADHD (Homack & Riccio, 2004; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Romine et al., 2004; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Researchers studying the neurological underpinnings of ADHD use the term "cognitive control" to describe the ability to adjust behavioral responses according to environmental stimuli (Nigg & Casey, 2005). Cognitive control is dependent on the ability to predict temporal and contextual structure in the environment, which allows individuals to appropriately tailor responses based on environmental stimuli (Nigg & Casey, 2005). Behavioral disinhibition may arise when individuals have difficulty predicting the occurrence of events or difficulty detecting violations of expectation, and thus, are unable to adjust the execution or inhibition of a response accordingly (Durston et al., 2007). According to Barkley (1997) the inhibition of behavior is an important

function that “sets the occasion” for many other activities requiring self-restraint and regulation of behavior. Without the ability to inhibit behavioral responses individuals would behave in a highly reactive manner toward environmental stimuli and internal states (e.g., hunger) regardless of conditions that may render these reactions maladaptive.

Support for an inhibitory deficit in ADHD comes from studies utilizing laboratory tasks (Lijffijt et al., 2005; Oosterlaan, Logan, & Sergeant, 1998), specifically the stop task (described in footnote[2] above). A meta-analysis of studies examining stop task performance provides robust evidence for an inhibitory deficit in children and adults with ADHD (Lijffijt et al., 2005; Oosterlaan et al., 1998). While behavioral disinhibition can be observed across the lifespan in those with ADHD, the nature of the cognitive deficit underlying behavioral disinhibition appears to change over the course of development. Specifically, children (ages 18 and under) display deficits associated with processing speed, whereas adults (ages 18 and older) display deficits consistent with the inhibition of a behavioral response (Lijffijt et al., 2005). While performing response inhibition tasks, children with ADHD generally respond to cues more slowly and with greater variability than children without ADHD, but both groups experience equal difficulty when required to quickly execute or withhold a prepotent response (i.e., engaging inhibitory processes). The authors concluded that for children the combination of greater variability in performance and comparable slowing of both executing and stopping a prepared response is not indicative of an underlying deficit in response inhibition per se; but rather, a broader deficit in generalized attention (Lijffijt et al., 2005). In contrast, adults with ADHD were not found to respond to “go” stimuli more slowly than adults without ADHD (Lijffijt et al., 2005). On “stop” trials, however, the mean reaction time was

significantly slower for adults with ADHD relative to adults without ADHD. Moreover, a test for the difference between the adults' mean reaction times on "go" trials and on "stop" trials revealed a moderate effect size, indicating a disproportionately slow response to "stop" signals relative to "go" signals. These findings suggest that: (1) the cognitive underpinnings of performance on the stop task differ as a function of age; (2) the deficit in processing speed and variability in response time observed in the performance of children with ADHD may no longer be impairing once these individuals reach adulthood; and (3) an inhibitory deficit appears to emerge in individuals with ADHD over the course of development, perhaps as processing speed increases and becomes less variable. Given that executive functions continue to develop throughout childhood, adolescence, and young adulthood (Romine & Reynolds, 2005), it is possible and perhaps likely that the cognitive processes underlying an inhibitory deficit in ADHD change over the course of development.

*ADHD and Cued Go/No-Go Task Performance*

The majority studies examining behavioral disinhibition in ADHD have utilized standard response inhibition tasks (e.g., the stop task; Lijffijt et al., 2005; Oosterlaan et al., 1998). With the goal of examining more detailed processes that may influence response inhibition, researchers have recently begun studying cued go/no-go task performance (described in footnote[3] above) in ADHD (Derefinko et al., 2008; Durston et al., 2007). To date, two studies have examined cued go/no-go task performance in children with ADHD (Derefinko et al., 2008; Durston et al., 2007), and one has examined cued go/no-go performance in adults with ADHD who were under the acute influence of alcohol (Weafer et al., 2009). These studies provide preliminary evidence about how

individuals with ADHD may utilize environmental cues to prepare or withhold a stimulus response. As seen in the meta-analyses of stop task performance in individuals with ADHD (described above), there are likely developmental differences in the cognitive processes influencing task performance (Lijffijt et al., 2005). Therefore, while the findings from studies examining cued go/no-go performance in children with ADHD are presented, it cannot be assumed that these findings will apply to adults with ADHD.

The first study using the cued go/no-go task with children (ages 9 – 12) identified differences in cue dependency between the inattentive (ADHD-I) and combined (ADHD-C) subtypes of ADHD (Derefinko et al., 2008).[4] The ADHD – I group responded to all targets requiring the execution of a response (go targets) in a slow and variable manner relative to the ADHD-C and control groups, regardless of whether the cue-target pairings were consistent (go cue followed by a go target) or inconsistent (no-go cue followed by a go target). This pattern of responding is similar to the pattern of slow and variable processing speed observed in the performance of children with ADHD on standard stop tasks (Lijffijt et al. 2005). The ADHD-C and control groups both performed equally better than the ADHD-I group on trials of response execution. Like the ADHD-I group, their performance did not differ as a function of cue condition. Therefore, while the ADHD-C and control groups performed significantly better than the ADHD-I group on trials of response execution, neither the control group nor the ADHD groups relied on

[4] The Diagnostic and Statistical Manual – Fourth Edition – Text Revision (DSM-IV-TR; APA, 2000) describes ADHD as a heterogeneous disorder with three subtypes: inattentive (ADHD-I), hyperactive/impulsive (ADHD-H), and combined (ADHD-C). Diagnostic criteria for all subtypes require that symptoms be present before age 7 and that symptoms are not better accounted for by another DSM-IV-TR disorder or an underlying medical condition. Children with the inattentive subtype must also have a minimum of 6 current DSM-IV-TR inattentive symptoms and fewer than 6 current DSM-IV-TR hyperactive/impulsive symptoms. Children with the combined subtype must have at least 6 current DSM-IV-TR inattentive symptoms and at least 6 current DSM-IV-TR hyperactive/impulsive symptoms.

preresponse cues to prepare their responses to the target stimuli. On trials measuring response inhibition, both the ADHD-I and ADHD-C groups displayed more inhibitory failures on all no-go trials than the control group. However, only the ADHD-C and control groups displayed a cue dependency effect. That is, these two groups performed significantly better on inhibitory trials with consistent cue-target pairings (no-go cue followed by a no-go target) than on inhibitory trials with inconsistent cue-target pairings (go cue followed by a no-go target). The ADHD-I group, in contrast, did not show a cue dependency effect and performed poorly on trials of response inhibition regardless of whether the cue target pairing was consistent or inconsistent. The authors conclude that these findings are indicative of an inability of individuals with ADHD-I to adjust their responses to stimuli based on relevant cues in the environment (Derefinko et al., 2008). It is also possible, however, the difference in cue dependency between groups can be explained by differences in processing speed, and that the cognitive mechanisms underlying behavioral disinhibition differ between the ADHD-I and ADHD-C groups.

Findings related to differences in ADHD subtype in this study should be interpreted with caution because of the atypical diagnostic criteria used to classify the two groups (Derefinko et al., 2008). In an effort to identify a subgroup of ADHD children with only inattentive symptoms the authors followed a unique categorization procedure previously employed by Milich and colleagues (2001). Consistent with DSM-IV-TR criteria, children in the ADHD-I group were required to have 6 or more DSM-IV-TR inattentive symptoms (APA, 2000). In contrast to typical DSM-IV-TR criteria, however, children in the ADHD-I group were permitted to have only 3 or fewer DSM-IV-TR hyperactive/impulsive symptoms rather than the 5 or fewer permitted in the DSM (APA,

200). In fact, the mean number of hyperactive/impulsive symptoms for the ADHD-I group was only 1.5 (SD=1.5). The children in the ADHD-I group were also required to have elevated T-scores on the Conners Cognitive Problems/Inattention scale (Conners, 1997) and T-scores at or below the mean on the Conners Hyperactivity scale (Conners, 1997). Therefore, participants in the ADHD-I group likely had fewer hyperactive/impulsive symptoms than children classified as ADHD-I in most research studies and in clinical practice.

The second study examining response inhibition in children with ADHD utilized a cued go/no-go task to determine whether children with ADHD (ages 7 – 14) experience greater difficulty predicting the occurrence of events based on environmental cues than their peers without ADHD (Durston et al., 2007). The authors hypothesized that if children with ADHD were unable to accurately predict event occurrence, then their reaction to a typically predicable event would be both slower and more variable relative to the reaction of control children to a predictable event. Study results supported this hypothesis. Reaction times on trials where cue-target pairings were consistent, or predictable (i.e., a go cue followed by a go target), were more variable for children and adolescents with ADHD, regardless of subtype, than for children and adolescents without ADHD. In addition, children and adolescents with ADHD showed less of a cue dependency effect than those without ADHD. This finding is *inconsistent* with the results of the study by Derefinko and colleagues (2008), which showed that on response execution trials, neither the ADHD nor the control groups displayed a cue dependency effect. It is possible that differences in sample age may have contributed to the inconsistency across studies. *f*MRI data collected during the task showed that, relative to

the control group, participants with ADHD displayed attenuated activation in the prefrontal regions (i.e., the ventral prefrontal cortex, the anterior cingulated gyrus, and regions in the cerebellum) thought to be associated with the detection of expectancy violations on unpredictable trials (Davidson et al., 2004). The authors conclude that, taken together, these results suggest that children and adolescents with ADHD may not be predicting the occurrence of future events based on environmental cues to the same degree as their non-disordered peers, and are therefore less able to prepare their responses to environmental stimuli (Durston et al., 2007). Durston and colleagues did not, however, report on findings related to response inhibition. While their findings regarding cue dependency on trials of response execution contradict the findings from the Derefinko (2008) study, the *f*MRI findings support the notion that children with ADHD are not utilizing pre-target cues to adjust their expectations about the nature of the upcoming target stimuli.

In summary, studies of children with ADHD provide preliminary evidence for deficient cue-based responding in a least a subset of children with the disorder. While conclusions about ADHD subtype differences and cue-dependency must be interpreted with caution (Derefinko et al., 2008), the findings suggest that the inattentive symptoms may be uniquely associated with a specific pattern of inhibitory responding. Given that prior research has identified differences in response inhibition across age groups, however, knowledge about the performance of adult ADHD participants cannot necessarily be inferred from studies conducted with children. These studies nevertheless provide a model and starting point for research with adult participants.

To date, only one study has examined cue dependency in adults with ADHD (Weafer et al., 2009). This study of 10 adults with ADHD (*M* age = 22.8 years, *SD* = 1.8) and 12 adults without ADHD (*M* age = 22.8 years, *SD* = 1.1) utilized the cued go/no-go task to measure the degree to which cue dependency protected individuals from the disinhibiting effects of alcohol. The authors controlled for factors that may influence response to alcohol such as drinking habits, age, gender, and IQ. Results indicate that, while under the influence of alcohol, the ADHD group performed similarly to the non-ADHD group on trials of response execution. Differences between the two groups emerged however, on trials of response inhibition. The non-ADHD group displayed a cue dependency effect that increased as the dose of alcohol increased. That is, while under the influence of a moderate dose of alcohol, the non-ADHD group performed significantly better on no-go trials with consistent cue-target parings than on no-go trials with inconsistent cue-target parings. In contrast, the ADHD group, while under the influence of alcohol, performed poorly on both cue conditions. Thus, the ADHD group did not appear to rely on the pre-response cues to prepare their responses to the target stimuli on trials of response inhibition. As a result, the pre-response cues consistent with the no-go targets did not protect against the disinhibiting effects of alcohol in the ADHD group as they did in the non-ADHD group. Weafer and colleagues (2009) did not report on the differences in cue dependency between groups for the placebo condition. Graphical representations of the data, however, indicate that task performance in the placebo condition was similar in pattern to the alcohol condition, but lower in the magnitude of difference between groups and between cue conditions.

On trials of response execution both the ADHD and non-ADHD groups showed a cue dependency effect. In both groups, performance remained unaffected by alcohol on trials where the cue-target pairings were consistent (go cue followed by a go target). In contrast, performance was equally impaired on trials where the cue-target pairings were inconsistent (i.e., no-go cue followed by a go target). Since the ADHD and non-ADHD groups showed statistically equivalent decreases in reaction time under alcohol conditions when cue-target pairings were inconsistent, Weafer and colleagues concluded that impairment in inhibitory control seen in ADHD participants could not be attributed solely to alcohol-induced deficits in processing speed. To the best of our knowledge, no studies have examined the cued go/no-go task performance in sober adults with ADHD. The absence of a studies examining cue-dependency in sober adults with ADHD represents a significant gap in the literature. Although one study has found a cue dependency deficit on trials of response inhibition in a subset of children with ADHD, it cannot be assumed that the same pattern of responding exists in adults with the disorder (Lijffijt et al., 2005). Therefore, it is currently unknown whether adults with ADHD display a deficit in cue dependent responding on trials of response inhibition when sober, or if this deficit is only present when alcohol has been consumed. Given that cue-dependency has been found to protect against the disinhibiting effects of alcohol (for a review, see Fillmore, 2003), it may be that a pervasive deficit in cue dependent responding, also present when the individual is sober, becomes particularly impairing when alcohol is consumed.

## Present Study

ADHD has repeatedly been associated with the development of alcohol-related problems in adolescents and young adults (Smith, Molina & Pelham, 2002; Weiss &

Hechtman, 1993; Wilens et al., 1998; Wilens et al., 2002). Few studies, however, have examined mechanisms underlying this association. Behavioral disinhibition, a construct central to theories of both ADHD (e.g., Barkley, 1997; Quay, 1997) and problematic alcohol use (e.g., Fillmore, 2003), may represent one underlying mechanism. The present study is part of a program of research examining substance use behaviors in college students with ADHD. Individuals with ADHD represent a growing population on college campuses, yet few studies have examined the psychosocial functioning of these students. In our prior study we found that ADHD was associated with risky patterns of alcohol use (Rooney et al., 2011). Specifically, we found that ADHD was associated with higher rates of negative consequences from alcohol use relative to those without ADHD, despite equivalent rates of self-reported rates of alcohol consumption. The identification of causal factors underlying the association between ADHD and the development of negative outcomes, such as alcohol-related problems, is crucial to the development of targeted interventions grounded in scientific theory (Sonuga-Barke & Halperin, 2010). The current study evaluated three possible mechanisms (all reflecting aspects of behavioral inhibition) that may, at least in part, explain the association between ADHD and problematic alcohol use: (1) self-reported difficulty stopping a drinking session, (2) behavioral disinhibition (i.e., sober state cue dependency), and (3) trait-disinhibition as mediators of the association between ADHD and alcohol-related problems in college students.

Cue dependency has been identified as a compensatory mechanism that protects against the disinhibiting effects of alcohol on laboratory task performance (for a review, see Fillmore, 2003). The single study that has examined cued dependency in adults with

ADHD found that, when under the influence of alcohol, adults with ADHD did not display the cue dependency effect observed in adults without ADHD. No studies have examined cue dependency in sober adults with ADHD. If individuals with ADHD display a cue dependency deficit under sober conditions, this may indicate the availability of fewer cognitive resources for coping with the disinhibiting effects of alcohol. As a result, individuals with ADHD may experience more negative consequences as a result of alcohol use, despite consuming alcohol at rates equivalent to those without ADHD. The current study is the first to assess sober state cue dependent response inhibition in young adults with and without ADHD. In addition, level of cue dependency under sober conditions and trait-disinhibition were examined as mechanisms by which college students with ADHD experience more negative consequences related to alcohol use than their peers without ADHD. The current study also sought to replicate findings from our preliminary study that identified an association between ADHD and higher rates of negative consequences of alcohol use, as well as difficulty stopping a drinking session as a mediator of this relationship. The current study therefore aims to extend prior research, which sought simply to identify differences in problematic alcohol use between college students with and without ADHD, by identifying mechanisms that may explain the association between ADHD and alcohol-related problems. Specific study aims are as follows:

***Aim 1a:*** To compare participants with and without ADHD on self-report measures of quantity of alcohol use, difficulty stopping a drinking session, and negative consequences associated with alcohol use.

***Hypothesis 1a:*** Consistent with results from our preliminary study (Rooney et al., 2011) and from prior studies of adolescents and young adults with ADHD (Smith, Molina, & Pelham, 2003; Weiss & Hechtman, 1993), it was hypothesized that self-reported quantity of alcohol use will be equivalent between the ADHD and non-ADHD groups. It was expected, however, that the ADHD group would self-report having difficulty stopping a drinking session more frequently than the non-ADHD group. In addition, those with ADHD would report significantly more negative consequences as a result of alcohol use than their peers without ADHD (Rooney et al., 2011).

***Aim 1b:*** To compare the performance of participants with and without ADHD on a laboratory task-based measure of cue dependency.

***Hypothesis 1b:*** It was hypothesized that there would be a significant difference in cue-dependent responding on response inhibition trials between the ADHD and non-ADHD groups. Specifically, on the cued go/no-go task ADHD participants were expected to display a lack of cue dependency on trials measuring response inhibition. The cue dependency deficit associated with ADHD would not be present however, on task trials measuring response execution. In contrast, we hypothesized that the non-ADHD participants would display cue dependency on trials of both response inhibition and response execution. These predictions were based on findings from studies examining cue-dependency in children with ADHD (Derefinko et al., 2008; Durston et al., 2007), and on a study of cue-dependency in adults with ADHD who were under the influence of alcohol (Weafer et al., 2009). We also hypothesized that the ADHD group would display a greater mean proportion of inhibitory failures across both cue conditions. We based this prediction on studies showing that adults with ADHD perform more poorly than those

without ADHD on behavioral tasks measuring response inhibition (Lijffijt et al., 2005). In addition, we hypothesized that there would be no differences between the ADHD and non-ADHD groups in mean reaction time, an indictor of processing speed. This finding was based on studies of adults with and without ADHD on non-cue based behavioral measures of response execution, and on the study examining cued go/no-go task performance in adults with and without ADHD under the influence of alcohol (Lijffijt et al., 2005; Weafer et al., 2009).

***Aim 2a:*** In our preliminary study self-reported "difficulty stopping a drinking session" mediated the relationship between ADHD and negative consequences associated with alcohol use (Rooney et al, 2011). In the proposed study we aim to replicate this finding.

***Hypothesis 2a:*** We hypothesized that results of our prior study would be replicated such that the relationship between ADHD and the negative consequences of alcohol use would be mediated by difficulty stopping a drinking session.

***Aim 2b:*** We aimed to extend our finding that difficulty stopping a drinking session mediated the relationship between ADHD and the negative consequences of alcohol use by identifying a specific inhibitory deficit associated with difficulty stopping a drinking session. Based on prior literature (for a review, see Fillmore 2003), we examined whether sober-state level of cue dependency on trials of response inhibition on the cued go/no-go task was correlated with self-reported difficulty stopping a drinking session. In addition, we examined the degree to which cue dependency on trials of response inhibition mediated the relationship between ADHD and self-reported negative consequences of alcohol use.

***Hypothesis 2b:*** We hypothesized that cue dependency on trials of response inhibition would be significantly correlated with self-reported difficulty stopping a drinking session. In addition, we expected that cue dependency on trials of response inhibition (as described in Aim 1b) would mediate the relationship between ADHD and self-reported negative consequences of alcohol use.

These hypotheses were based on literature identifying difficulty stopping a drinking session as a manifestation of alcohol-induced behavioral disinhibition (Weafer & Fillmore, 2008). Studies have identified a reliance on pre-response cues (i.e., cue dependency) as a factor that protects against the disinhibiting effects of alcohol on task performance. If individuals with ADHD exhibit deficits in cue dependency when in a sober state, then they will likely exhibit this deficit when under the influence of alcohol. Under alcohol conditions this deficit would represent the absence of a cognitive mechanism that protects against disinhibited behavior. Therefore, we proposed that sober state level of cue dependency would be significantly correlated with self-reported difficulty stopping a drinking session, a manifestation of disinhibited behavior when under the influence of alcohol. If these two variables were highly correlated, then we hypothesized that level of cue dependency would also mediate the relationship between ADHD and the negative consequences of alcohol use.

***Aim 3a****:* To examine the degree of correlation between the behavior-based measurement of disinhibition (performance on the cued go/no-go task) and the personality-based measurement of disinhibition (DIS-M).

***Hypothesis 3a:*** Studies have found that behavior-based and personality-based measurements of disinhibition are not highly correlated (Dick et al., 2010; Reynolds et

al., 2006). We therefore hypothesize that the two measures of disinhibition used in the current study will not be significantly correlated. If not significantly correlated, it is likely that these two measures are measuring separate facets of the loosely- defined behavioral disinhibition construct.

***Aim 3b:*** We aimed to extend our finding that difficulty stopping a drinking session mediated the relationship between ADHD and the negative consequences of alcohol use by examining the role of trait-based disinhibition. We examined the degree to which trait-based disinhibition (DIS-M) was correlated with self-reported difficulty stopping a drinking session. In addition, we examined the degree to which trait-based disinhibition mediated the relationship between ADHD and the negative consequences of alcohol use.

***Hypothesis 3b****:* Based on studies suggesting that difficulty stopping a drinking session is a manifestation of behavioral disinhibition (e.g., Weafer & Fillmore, 2008), we hypothesized that trait-based disinhibition would be significantly correlated with difficulty stopping a drinking session. Difficulty stopping a drinking session was found to mediate the relationship between ADHD and the negative consequences of alcohol use in our preliminary study (Rooney et al., under review). If trait-based disinhibition and difficulty stopping a drinking session were significantly correlated, we hypothesized that trait-based disinhibition (DIS-M) would also mediate the relationship between ADHD and the negative consequences of alcohol use.

## Method

*Participants*

Participants included 50 University of Maryland undergraduate students diagnosed with ADHD and 50 University of Maryland undergraduate students without an ADHD diagnosis. Participants in the ADHD sample: (1) met full DSM-IV-TR diagnostic criteria for ADHD during childhood based on self report; (2) had a T-score of 60 or above (1 SD above the mean) on the Conners Adult ADHD Rating Scale - ADHD Index (CAARS; Conners et al., 1999); (3) currently met full diagnostic criteria as specified in the DSM-IV-TR according to self report, with one exception. Given that there are currently no empirically-supported guidelines for diagnosis ADHD in young adulthood, the DSM-IV-TR symptom count specified for a childhood diagnosis was modified to accommodate a young adult symptom presentation. The threshold for diagnosis was set at four current symptoms in either the inattentive or hyperactive-impulsive category rather than the six symptoms specified in the DSM-IV-TR according to the guidelines set forth by McGough and Barkley (2004) and in accordance with procedures used in studies of adult ADHD conducted in our lab (Chronis-Tuscano, Raggi et al., 2008; Chronis-Tuscano, Seymour et al., 2008) and others' research labs (Biederman & Spencer, 2002; McGough & Barkley, 2004); (4) were enrolled as a full-time undergraduate student at the University of Maryland; (5) and lived independently away from their parents. The study included both students who were taking medication to treat ADHD and those who were not. Forty-six percent (n = 23) of the ADHD group was currently taking ADHD medication. In addition, fifty-two percent (n = 26) of participants in the ADHD group reported a previous ADHD diagnosis.

Students in the non-ADHD comparison group: (1) had fewer than three current DSM-IV-TR symptoms of ADHD and no history of the disorder according to self report; (2) had a T-score of 50 or lower (i.e., at or below the mean) on the CAARS ADHD Index; (3) had never been prescribed medication to treat ADHD symptoms; (4) were enrolled as full-time undergraduate students at the University of Maryland; and (5) lived independently away from their parents. Additionally, students with one or more Axis I disorders (not including ADHD) were eligible to participate in either group. Participant characteristics are presented in Table 1.

*Procedures*

Participants in the ADHD and non-ADHD groups were recruited through flyers posted in classroom buildings on campus, through an online system advertising studies offering course credit in exchange for study participation, and through an online system advertising studies offering payment ($25.00) for study participation. Flyers included the following study description:

> Undergraduates who live away from their parents and have been in college for at least six-months may be eligible to participate in our study of behavior and relationships in college students with and without ADHD. Participants attend a single 2-hour appointment in the Biology-Psychology Building. Participants are asked to answer written and verbal questions and complete a computer task. Participants are paid $25. Contact Mary Rooney (rooney@umd.edu or 301-405-4606).

Two versions of the flyer were posted. One with the heading "Study of Students with ADHD" and the second with the heading "Study of Undergraduate Students." Participants were likely drawn to the study because of the financial incentive or because they were seeking extra credit in one of their classes. Some students with ADHD may have been interested in participating because they wanted to contribute to research aimed

at achieving a better understanding of the disorder with which they had been diagnosed.

Participants completed a brief phone survey during which study requirements and procedures were reviewed. Since the study included a computer task that may be sensitive to the effects of stimulant medication (Tannock, Schachar, Carr, & Chzajczyk, 1989; Tannock, Schachar, & Logan, 1995), participants who were currently taking prescribed stimulant medication for ADHD were asked to attend the study appointment without their medication in effect. In accordance with IRB approved procedures, the risks associated with skipping a dose of ADHD medication were discussed (e.g., driving while unmedicated, short-term negative impact on academic performance, etc.). Participants who were comfortable skipping their medication were encouraged to schedule their appointment for a time when withholding medication would not interfere with their academic or occupational performance. In addition, these participants were asked to agree that they would not drive to the study appointment.

Two graduate students under the supervision of a licensed clinical psychologist, Andrea Chronis-Tuscano, Ph.D., conducted diagnostic assessments. Assessments included the administration of the K-SADS (Schedule for Affective Disorders and Schizophrenia for school age children; Kaufman et al., 1997) ADHD module modified for use with adults to assess past and current symptoms (Biederman & Spencer, 2002) and the M.I.N.I. International Neuropsychiatric Interview (MINI; Sheehan, 1998). During the visit, participants also completed electronic versions of five self-report measures related to the current study: the Conners Adult ADHD Rating Scale (CAARS – LV; Conners et al., 1999; Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999a, 1999b), Personal Drinking Habits Questionnaire (PDHQ; Vogel-Sprott, 1992), the CORE

Alcohol and Drug Survey (CORE; Core Institute, 2005), the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuenta, & Grant, 1993), the Zuckerman Sensation Seeking Scale – V (SSS-V; Zuckerman, 1994) and a demographics form (see Appendix for copies of measures). Finally, participants completed the cued go/no-go computer task (Fillmore, 2001). See Figure 2 (p. 83) for participant flow diagram.

Students who received elevated scores on any modules of the MINI or K-SADS or who requested referrals for pharmacological or psychosocial treatment were referred to the University Health Center and the University Counseling Center for treatment.

*Measures*

*ADHD Assessment*

The Conners Adult ADHD Rating Scale – Long Version (CAARS – LV; Conners et al., 1999; Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999a, 1999b) provides a continuous measure of ADHD symptoms specific to adults with the disorder. This measure has excellent psychometric properties and allows for the generation of an ADHD symptom profile that can be compared against established age and gender norms. The CAARS – LV is a 93-item, reliable and valid measure of current ADHD symptoms in a form suitable for adults (Conners et al., 1999; Erhardt et al., 1999a). Students in the ADHD group were required to have a T-score at or above 60 (1 SD above the mean) on the ADHD Index. Participants in the non-ADHD group were required to have a T-score at or below 50 (the mean).

In accordance with the guidelines provided by McGough and Barkley (2004), final group classification was determined through the administration of a semi-structured

interview based on DSM-IV-TR criteria. The investigators administered a modified version of the ADHD module of the K-SADS to the participants. The questions from the K-SADS were modified to be appropriate for retrospective and current self-report by adults in accordance with procedures utilized in previous studies (Biederman & Spencer, 2002; Faraone, Biederman, & Milberger, 1995; Faraone, Biederman, & Monuteaux, 2002). The K-SADS has well-established reliability for the diagnosis of ADHD in children (Ambrosini, 2000).

For the purpose of screening for other disorders that could potentially account for ADHD symptoms and to isolate associations between co-occurring disorders and the variables of interest, Axis I disorders were assessed using a structured diagnostic interview, the revised M.I.N.I. International Neuropsychiatric Interview, version 5.0 (MINI; Sheehan, 1998). The MINI was selected because of its solid psychometric properties and relatively short administration time (approximately 20 minutes). Comparisons of diagnoses made with the clinician-rated MINI and those made with the Structured Clinical Interview for the DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1996) have been characterized by good or very good kappa values (Sheehan et al., 1998). The MINI features a closed-ended question format with optional rater inquires for 23 disorders. The following modules of the MINI were administered in the current study: Major Depressive Episode (Past & Current), Dysthymia (Past & Current), Bipolar Disorder (Past & Current), Panic Disorder (Current & Lifetime), Agoraphobia (Current), Social Phobia (Current), Specific Phobia (Current), Generalized Anxiety Disorder (Current), Obsessive Compulsive Disorder (Current), Alcohol Dependence (Past 12 Months & Lifetime), Alcohol Abuse (Past 12 Months & Lifetime), Substance

Dependence – Non-Alcohol (Past 12 Months & Lifetime), Substance Abuse – Non-Alcohol (Past 12 Months), Antisocial Personality Disorder (Current), Conduct Disorder (Lifetime), Anorexia Nervosa (Current), Bulimia Nervosa (Current), Body Dysmorphic Disorder (Current).

Symptoms associated with Learning Disabilities (LD) may be associated with many of the behaviors under examination in the current study, including alcohol use (Fernandez, 2007; McCrystal, Percy, & Higgins, 2007). The comprehensive neuropsychological assessment required to adequately test for learning disabilities was beyond the scope of this project; however, participant responses to an open-ended question about a previous LD diagnosis were captured. It was intended that LD diagnosis would be included as a covariate in analyses when LD diagnosis was significantly associated with the outcome variable. In the current sample, however, only a small subset of participants in the ADHD group endorsed a prior LD diagnosis (see Table 1). Due to the lack of variability in the comparison group, LD diagnosis was not used as a covariate in analyses.

*Substance Use*

Two questionnaires were included to assess the quantity and frequency of alcohol use. The Personal Drinking Habits Questionnaire (PDHQ; Vogel-Sprott, 1992) was selected because of its use by researchers in previous studies of the effects of alcohol on cued go/no-go task performance. The PDHQ yields quantity and frequency measures of typical drinking habits. Administration of this measure in the current study was problematic as participants reported finding the question format confusing and as a result skipped many items. Form administration was changed from paper and pencil format to

an interview in an effort reduce participant confusion. The large amount of missing data, however, reduced the PDHQ's utility as a primary outcome measure. Exploratory analyses were conducted with this measure and results are presented in Appendix B.

The second measure of alcohol use, the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuenta, & Grant, 1993), also contains questions about the quantity and frequency of alcohol use. This measure was used in our previous study of college students (Rooney et al., 2011) and was developed by the World Health Organization as a simple, brief method of screening for excessive drinking and related impairment (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001). In addition to utilizing the quantity and quantity/frequency measurement included in the AUDIT, the fourth item on the questionnaire was used as a measure of self-reported difficulty stopping a drinking session: "How often during the past year have you found that you were not able to stop drinking once started? (0) Never (1) Less than Monthly (2) Monthly (3) Weekly (4) Almost Daily.

The Core Alcohol and Drug Survey (CORE; Core Institute, 2005) Negative Consequences Subscale is a 19-item scale designed to assess negative consequences associated with alcohol use tailored to the college environment (e.g., "As a consequence of your drinking during the last year how often have you: (a) had a hangover; (b) performed poorly on a test or important project, (c) been in trouble with police, residence hall, or other college authorities; [etc.]"). It was selected specifically for its strong psychometric properties and its relevance to the present study's research questions. Test-retest reliability ranges between .61 - .80 for most items (Biscaro, Broer, & Taylor, 2004; Core Institute, 2005). Responses to items on the Negative Consequences of Alcohol Use

subscale were summed to form a total composite score. The CORE also contains a subscale of polysubstance use during the past 12-months. Responses to questions on the drug use subscale were summed to form a composite score utilized in analyses as a measure of current drug use.

*Disinhibition*

The cued go/no-go task (Fillmore, 2001) was utilized as a measure of cue dependency on trials of response inhibition and response execution, and mean reaction time on trials of response execution. This task has been used with ADHD participants in three previous studies (Derefinko et al., 2008; Durston et al., 2007; Weafer et al., 2009) and in research examining the disinhibiting effects of alcohol (e.g., Fillmore et al., 2005; Marczinski, Abroms, Van Selst, & Fillmore, 2005). This task was selected because, unlike simple reaction time tasks (e.g., the stop task) which provide no information about participant's ability to utilize environmental cues to initiate the process of pre-response preparation, the cued go/no-go task employs pre-stimulus cues that facilitate or impede early response preparation. In studies examining the effects of alcohol on task performance in young adults (ages 22 – 29), appropriate reliance on pre-response cues (i.e., cue-dependency) has been shown to protect against the disinhibiting effects of alcohol (e.g., Fillmore et al., 2005; Marczinski, Abroms, Van Selst, & Fillmore, 2005). Therefore, this measure was appropriate for identifying a sober-state cue dependency deficit in young adults with ADHD.

The task was operated using E-Prime software (Schneider, Eschman, & Zuccolotto, 2002). Trials involved the following sequence of events: (a) presentation of a fixation point (+) for 800ms; (b) a blank white screen for 500 ms; (c) a cue, displayed for

one of five stimulus onset asynchronies (100, 200, 300, 400, and 500 ms); (d) a go or no-go target which remained visible until a response occurred or 1,000 ms had elapsed; and (e) an interval of 700 ms.

The cue was a rectangle (7.5 cm X 2.5 cm) framed in a 0.8 mm black outline that was presented in the center of the computer monitor against a white background  . The cue was presented in either a horizontal (no-go cue)  or vertical (go cue)  orientation. The orientation of the cue signaled the probability of a go or no-go target appearing next on the screen. Cues presented vertically preceded go targets on 80% of the trials and preceded the no go target on only 20% of the trials. Cues presented horizontally preceded the no-go target on 80% of the trials and preceded the go target on only 20% of the trials.

Targets were presented as green (go)  or blue (no-go)  rectangles. Participants were instructed to press the question mark (?) key on the keyboard as soon as the green (go) target appeared on the screen and to withhold a response when a blue (no-go) target appeared. The variability and randomness of the time intervals between the presentation of cues and targets (100, 200, 300, 400, and 500 ms) encouraged participants to pay attention to the cues, and prevented the participants from anticipating the exact temporal display of the targets.

The test consisted of 250 trials that presented the four possible cue-target combinations. An equal number of vertical (125) and horizontal (125) cues were presented, and an equal number of go (125) and no-go (125) target stimuli were presented. Each cue-target combination was randomly presented at each of the five random time intervals. For each trial, the computer recorded whether a response occurred

and, if so, the response time in milliseconds was measured from the onset of the target until the key was pressed. To encourage quick and accurate responding, the program provided feedback to the participant after each response. On response execution trials, the response time in milliseconds was presented on the screen. On response inhibition trials where a keystroke was not withheld (i.e., an participant error occurred) the words "incorrect response" appeared on the screen.

The Sensation Seeking Scale – V (SSS – V; Zuckerman, 1994) is a 40-item, forced choice inventory designed to measure sensation seeking. Sensation seeking is a multifaceted construct comprised of four components: thrill and adventure seeking, experience seeking, disinhibition, and boredom susceptibility (Zuckerman, 1994; Zuckerman, Eysneck, & Eysenck, 1978). Each of these four components is captured in individual subscales of the SSS-V. The reliability and construct validity for the SSS-V has been well established (for a review, see Zuckerman, 1994). The psychometric properties of the scale were reevaluated using a sample of college students at a public university (Roberti, Storch, & Bravata, 2003). Confirmatory factor analysis supported the four-factor structure. Internal consistency was high with Chronbach's alphas reported as follows: Thrill and Adventure Seeking (.80), Experience Seeking (.75), Disinhibition (.80), and Boredom Susceptibility (.76.).

The present study utilizes a modified version of the disinhibition subscale (DIS-M) as a measure of self-reported behavioral disinhibition. Within the context of sensation seeking, disinhibition is characterized by the expression of reduced social constraint (Zuckerman, 1994). Individuals who score high on the disinhibition subscale are generally less constrained by social norms and expectations so they are likely to be more

experimental with regard to their behavior. Of the four SSS – V subscales, disinhibition has repeatedly been found to be most highly correlated with alcohol use (for review see Hittner & Swickert, 2006). The disinhibition subscale contains three alcohol and/or drug use items, which may inflate the association between disinhibition and alcohol use. In a recent meta-analysis, excluding the substance use-related items from the subscale did not significantly diminish the effect size between disinhibition and alcohol use (Hittner & Swickert, 2006). In the present study, however, we chose to act conservatively and, in accordance with procedures used in previous studies (Darkes, Greenbaum, & Goldman, 1998; Henderson, Goldman, Coovert, & Carnevalla, 1994), modified the disinhibition subscale by removing the three items that explicitly endorse alcohol use or involvement ("If feel best after a few drinks," "I often like to get high [drinking alcohol or smoking marijuana]", and "Keeping the drinks full is the key to a good party").

## Evaluation of Aims

### *Preliminary Analyses*

All dependent variables were examined for frequency, variability, and the identification of outliers. One outlier was identified on the cue-dependency task. Analyses were conducted once with the outlier included and once with the outlier excluded. Since results differed greatly when the outlier was removed, final analyses excluded this participant[5]. This participant's data is excluded from the analyses presented in all tables, including the participant characteristics table (Table 1).

[5] The excluded participant is a 19-year-old, white female. She does not meet the study's diagnostic criteria for ADHD and has an estimated IQ in the above average range (standardized WTAR score = 119). The participant's cue dependency score of .40 on trials of response inhibition was 9.5 *SD* above the non-ADHD group mean ($M$ = .017, $SD$ = .041).

Prior to running the main analyses for each outcome variable, correlation analyses were conducted to determine the extent to which the variables of age, gender, race/ethnicity, fraternity/sorority membership, drug use (i.e., composite score of drug use during the past 6 months from CORE), were associated with the mediator and outcome variables. The variables examined in preliminary analyses (i.e., age, gender, race/ethnicity, fraternity/sorority membership, and current drug use) were selected based on studies in the existing literature suggesting that they may differentially affect patterns of substance use.

Regarding age, problematic alcohol use has been found to increase during late adolescence and peak during young adulthood (Littlefield, et al., 2009), and longitudinal studies have found that alcohol consumption varies as a function of age (Johnston et al., 2009).

Regarding gender, research has shown that women generally consume less alcohol per drinking session than men (Johnston et al, 2009), reach higher blood alcohol concentration levels than men when consuming equivalent amounts of alcohol (Baraona et al., 2001), have a later age of first use (Johnston et al, 2009), and a shorter time from first drink to dependence (Johnson, Richter, Kleber, McLellan, & Carise, 2005). In recent cohorts, however, differences between genders in age of first use and time from first drink to dependence were diminished suggesting that gender-based norms may be shifting (Keyes, Martins, Blanco, & Hasin, 2010).

Regarding racial and ethnic groups, differences in patterns of alcohol use and problematic alcohol use have been identified in a in several large scale studies of college student substance use (i.e., College Alcohol Study, Harvard School of Public Health;

Monitoring the Future; The Core Alcohol and Drug Use, and the National College Health Risk Behavior Survey (O'Malley & Johnston, 2002). Consistently across studies White students are highest in heavy drinking, black students are lowest, and Hispanic students are intermediate (O'Malley & Johnston, 2002). Further, in a study examining the interaction of college enrollment and race/ethnicity on patterns of alcohol use, attendance at a four-year college has been found to increase the likelihood of heavy drinking for White young adults, and decrease the likelihood for black and Asian young adults (Paschall, Bersamin, & Flewelling, 2005). Ethnic differences in the ability to metabolize alcohol have also been established (e.g. ALDH2 and ADH1B genotypes disproportionately identified among Asians; Hendershot, et al. 2009).

Regarding fraternity/sorority membership, a host of prior studies have identified increased rates of problematic drinking among fraternity and sorority members relative to non-member college students. Alcohol-related problems among fraternity/sorority members include: higher levels of alcohol use, alcohol-related negative consequences, and dependence symptomatology (Bartholow, Sher, & Krull, 2003; Borsari & Carey, 2006; Borsari & Carey, 1999; Capone, Wood, Borsari, & Laird, 2007; Cashin, Presley, & Meilman, 1998; Larimer, Irvine, Kilmer, & Marlatt, 1997; Lo & Golobetti, 1995; Rabow & Duncan-Schill, 1995; Sher, Bartholow, & Nanda, 2001). In an extensive review of the literature, Borsari & Carey (1999) identified five factors contributing to the heavy drinking observed among fraternity/sorority students: (a) a continuity of heavy alcohol use from high school to college; (b) self-selection into heavy drinking environments; (c) the central role that alcohol plays in fraternity selection; (d) misperceptions of drinking norms; and (e) the enabling environment of the fraternity house. In a recent prospective

study of alcohol use among fraternity/sorority students, researchers obtained findings consistent with these five factors (Capone, Wood, Borsari, & Laird, 2007). In addition, they concluded that heavy drinking within the fraternity/sorority community is a result of a mutually reinforcing system in which initially higher levels of alcohol use and problems by students who self select into the community are exacerbated by the increased affiliation with heavier drinking peers within the fraternity/sorority community. Fraternities and sororities require that members maintain a grade point average of 2.5 or higher (North American Intrafraternity Conference, 2009), suggesting that their members are meeting academic standards despite their heavy drinking. This may suggest that students with ADHD who are members of fraternities or sororities may be a particularly high functioning subset of the ADHD college student population.

Finally, regarding drug use, across multiple large-scale studies drug use has been correlated with alcohol use in young adults and college students in multiple studies (Johnson et al., 2009; Wechsler et al., 2002).

It was proposed that ADHD medication status and LD diagnoses would also be included in preliminary analyses. However, participants taking ADHD medication or with an LD diagnosis were present only in the ADHD group. Due to the lack of variability in the non-ADHD group, these variables were not included in preliminary analyses. Within-group (ADHD) comparisons were conducted for all outcome variables. The means of all outcome variables were not significantly different for participants with and without a self-reported LD diagnosis (see Table 2). Likewise the means of all outcome variables were not significantly different for participants in the ADHD group with a prior ADHD diagnosis and participants within the ADHD group without a prior

ADHD diagnosis (see Table 3). Finally, the means of all outcome variables were not significantly different for participants within the ADHD group who were currently prescribed ADHD medication and those within the ADHD group who were currently not prescribed ADHD medication (see Table 4).

Between group differences in rates of Axis I disorders were also examined. In cases where disorder rates differed, comorbid diagnoses were included in preliminary analyses to examine whether they were associated with outcome variables. Conduct disorder, which occurs in up to 40% of children and adolescents with ADHD (Barkley, 1998; Lahey, McBurnett, & Loeber, 2000) occurred in 20% (n = 10) of the ADHD group and in 0 participants in the non-ADHD group. Of those with comorbid CD, three participants (6%) had symptoms consistent with childhood-onset CD and four participants (8%) had symptoms consistent with adolescent-onset CD. Significantly higher rates of major depressive disorder (MDD) - past episode, panic disorder – current, and social phobia – current were found in the ADHD group relative to the non-ADHD group (see Table 2). No participants in either group endorsed symptoms consistent with a current episode of MDD. Relative to the non-ADHD group, participants in the ADHD group also endorsed significantly higher rates of lifetime alcohol dependence, lifetime alcohol abuse, and current alcohol abuse. Rates of non-alcohol substance use disorders were not significantly different between groups (see Table 5). Correlations between Axis I disorders and outcome variables are presented in Table 6.

Predictor variables associated with the outcome variable at a significance level of $p < .05$ in preliminary analyses were included as covariates in primary analyses. Mean and standard deviation values for all outcome variables are presented in Table 7. A

number of significant correlations were identified during preliminary analyses. First, gender was negatively correlated with the quantity of alcohol consumed, indicating that females consumed less alcohol than males. Second, fraternity/sorority membership was positively correlated with the negative consequences of alcohol use and difficulty stopping a drinking session. Third, panic disorder was significantly correlated with difficulty stopping a drinking session (Table 19). This finding is consistent with studies showing higher rates of alcohol-related problems among individuals with panic disorder (Bystrisky et al., 2010; Otto, Pollack, Sachs, O'Neil, & Rosenbaum, 1992; Swendsen et al., 1998).

Surprisingly higher rates of alcohol use and alcohol related problems were not associated with a history of MDD. This finding is inconsistent with studies showing higher rates of alcohol use and related problems among those with MDD or elevated depression sympatology (Dixit & Crum, 2000; Harrell & Karim, 2008; Nolen-Hoeksema, 2004; Pedrelli et al., 2010). One posibility for the discrepant finding is that only those experiencing a current MDD episode, or currently elevated depression sympomatology, show higher rates of use. No participants in the current sample endorsed symtpoms consistent with a current MDD episode. Only past MDD episodes were reported.

Significant correlations among substance use and alcohol use disorders were also identified. While current alcohol dependence was not significantly associated with any substance use disorders, lifetime alcohol dependence and both current and lifetime alcohol abuse were significantly associated with both current and lifetime substance dependence as well as current substance abuse. The high correlations among these diagnoses are consistent with studies showing high rates of comorbidity among alcohol

and substance use disorders. In addition, these findings are consistent with Krueger's two-factor model of mental disorder classification (Krueger, 1999). According to Krueger's (1999) analyses of the National Comorbidity Survey data, 10 common DSM-IV mental disorders fall into internalizing and externalizing factors, with substance use disorders as well as antisocial personality disorder falling on the externalizing factor. In a replication study examining the consistency of Krueger's two-factor model in a sample of adolescents and young adults, the structure of the externalizing factor was applicable (Wittchen et al., 2009). Additional studies are needed to determine the genetic, neurobiological, or environmental factors underlying the high rate of comorbidity among substance use disorders (Krueger & Markon, 2006).

### *Aim 1a: Analytic Plan*

We evaluated whether differences exist between participants with and without ADHD on self-report measures of quantity of alcohol use (AUDIT item #2), difficulty stopping a drinking session (AUDIT item #4), and negative consequences associated with alcohol use (CORE Negative Consequences Subscale total). One-way ANCOVA analyses (group: ADHD, Non-ADHD) were conducted with each of these outcome variables. Covariates identified in preliminary analyses included fraternity-sorority membership for each of the Aim 1a outcome variables, gender for the quantity and frequency of alcohol use, and panic disorder for difficulty stopping a drinking session. In addition, to account for the possibility that the quantity of alcohol use, the negative consequences of alcohol use, or difficulty stopping a drinking session were a strongly influenced by concurrent drug use, self-reported current drug use (CORE Drug Use

Subscale total) was included as a covariate in analyses that included these outcome variables.

***Aim 1a: Hypotheses Summary***

Quantity of Alcohol Use: Results would not differ significantly between ADHD and non-ADHD groups on self-reported quantity of alcohol use.

Difficulty Stopping a Drinking Session: Participants within the ADHD group would self-report higher rates of difficulty stopping a drinking session than participants in the non-ADHD group.

Negative Consequences of Alcohol Use: Participants within the ADHD group would self-report experiencing more negative consequences of alcohol use than participants in the non-ADHD group.

***Aim 1a: Results***

Our hypotheses were supported for each of the four Aim 1a outcome variables.

Quantity of Alcohol Use: The ADHD and non-ADHD groups did not differ significantly on self-reported quantity of alcohol use ($F$ (4, 96) = .032, $p$ =.859). Results are presented in Table 10.

Difficulty Stopping a Drinking Session: Participants within the ADHD group self-reported significantly higher rates of difficulty stopping a drinking session than those in the non-ADHD group ($F$ (4, 96) =3.229, $p < .01$). Results are presented in Table 11.

Negative Consequences of Alcohol Use: Participants within the ADHD group self-reported significantly more negative consequences as a result of alcohol use than participants in the non-ADHD group when controlling for alcohol quantity,

fraternity/sorority membership, and current drug use ($F$ (4, 96) = 5.544, $p$ < .01). Results are presented in Table 12.

The following specific negative consequences occurred significantly more frequently within the ADHD group than the Non-ADHD group when controlling for alcohol quantity, fraternity/sorority membership, and current drug use: Had a Hangover ($F$ (4, 96) = 6.123, $p$ < .01), Got Nauseated or Vomited ($F$ (4, 96) = 5.865, $p$ < .05), Been Criticized by Someone I Know ($F$ (4, 96) = 7.438, $p$ < .01), Got Into an Argument or Fight ($F$ (4, 96) = 4.582, $p$ < .05), Thought I Might Have a Drinking Problem ($F$ (4, 96) = 5.155, $p$ < .05), Did Something I Later Regretted ($F$ (4, 96) = 5.601, $p$ < .05). Results are presented in Table 13.

The following specific negative consequences did *not* occur significantly more frequently in the ADHD group than the non-ADHD group when controlling for alcohol quantity, fraternity/sorority membership, and current drug use: Performed Poorly on a Test or Important Project ($F$ (4, 96) = .595, $p$ = .442), Missed a Class ($F$ (4, 96) = 2.559, $p$ = .113), Been in Trouble with Police, Residence Hall Staff, or other College Authorities ($F$ (4, 96) = .135, $p$ = .714), Drove a Car While Under the Influence ($F$ (4, 96) = .171, $p$ =.680), Damaged Property, Pulled Fire Alarm, etc. ($F$ (4, 96) = .192, $p$ =.662), Been Hurt or Injured ($F$ (4, 96) = .031, $p$ =.860), Had Memory Loss ($F$ (4, 96) = .1.296, $p$ = .258), Have Been Taken Advantage of Sexually ($F$ (4, 96) = .808, $p$ =.371). Results are presented in Table 13.

***Aim 1b: Analytic Plan***

We evaluated whether differences existed between college students with and without ADHD on cue dependent responding on trials of response inhibition (measured

by performance on the cued go/no-go task). Cue dependency is present when participants perform significantly better on trials with consistent cue-target pairings (no-go cue followed by a no-go target) than on trials with inconsistent cue target pairings (go cue followed by a no-go target). ADHD group classification and cue condition effects on *p*-inhibition failures in the consistent and inconsistent cue conditions were analyzed by a 2(group) x 2(cue condition) ANOVA. To determine whether cue dependency (i.e., differential performance based on cue condition) was unique to performance on trials of response inhibition, a second 2 (group) x 2 (cue condition) ANOVA was conducted on trials of response execution. In addition, we examined group differences in overall mean reaction time and overall *p*-inhibition failures across cue conditions using a one-way ANOVA.

***Aim 1b: Hypotheses Summary***

Cue Dependency – Response Inhibition:

There would be a main effect of group, with the ADHD group performing significantly more poorly on response inhibition trials in both cue conditions.

There would not be a main effect of cue condition. While the non-ADHD group would perform significantly better in the consistent cue condition than the inconsistent cue condition, the ADHD group would not show performance difference between cue conditions.

There would be a significant Group x Cue Condition interaction. Specifically, performance *would* be significantly better in the consistent cue condition than the inconsistent cue condition for the non-ADHD group. In contrast, performance would not

differ significantly between cue conditions in the ADHD group. Thus, the ADHD group would display a deficit in cue dependency.

Cue Dependency – Response Execution:

There would be a main effect of group, with the ADHD group performing significantly more poorly on response inhibition trials in both cue conditions.

There would be a main effect of cue condition. Both the ADHD and non-ADHD groups would perform more poorly in the inconsistent cue condition than the consistent cue condition. Thus, both groups would display a cue dependency effect.

There would not be a significant Group x Cue Condition interaction. Specifically, performance *would* differ significantly based on cue condition for participants in both the ADHD and non-ADHD groups. Thus, members of both groups would display patterns of cue dependent responding on trials of response execution. This would suggest that the cue dependency deficit expected in the ADHD group would be isolated to trials of response inhibition and would not be seen in trials of response execution.

***Aim 1b: Results***

Cue Dependency - Response Inhibition: Contrary to our hypothesis a main effect of group was not observed ($F$ (1, 97) = 3.090, $p$ = .082). Also contrary to our hypothesis, a main effect of cue condition *was* observed ($F$ (1, 97) = 11.575, $p$ <.01), indicating that both the ADHD and non-ADHD groups performed significantly better in the consistent cue condition. Thus, the ADHD group did not display the expected cue dependency deficit. Finally, against expectations, a Group x Cue Condition interaction was not observed ($F$ (1, 97) =.007, $p$ = .936). Results are presented in Table 16.

Cue Dependency - Response Execution:

Contrary to our hypothesis, a main effect of group was not observed ($F$ (1, 97) = 2.922, $p$ = .091). Although the ADHD group had slower reaction times relative to the non-ADHD group, this difference was not statistically significant. Consistent with our hypothesis, a main effect of cue condition was observed ($F$ (1, 97) = 28.128, $p$ <.001). Both groups performed significantly better in the consistent cue condition than the inconsistent cue condition. Also consistent with our hypothesis, a Group x Cue Condition interaction was not observed, as both groups displayed cue dependent responding ($F$ (1, 97) = .081, $p$ =.776). Results presented in Table 17.

ADHD Medication Effects:

Post-hoc analyses were conducted to examine whether current ADHD medication status was significantly correlated with any of the cued go/no-go task outcome variables. Results indicate that ADHD medication status was not correlated with any of the task variables (Table 18).

***Aim 2a: Analytic Plan***

We examined difficulty stopping a drinking session as a mediator of the relationship between ADHD and negative consequences associated with alcohol use when including alcohol quantity as a covariate. Analyses were conducted in accordance with the model of mediation proposed by Baron and Kenny (1986) and reviewed by Kenny (2009). According to Baron and Kenny (1986), a full meditational model is supported when the following four statistical criteria are met: (1) the predictor variable (ADHD diagnosis) is significantly associated with the outcome variable (CORE Negative Consequences Subscale total score); (2) the predictor variable (ADHD diagnosis) is

significantly associated with the mediator (difficulty stopping a drinking session); (3) the mediator (difficulty stopping a drinking session) is significantly associated with the outcome variable (CORE Negative Consequences Subscale total), even after controlling for the predictor (ADHD); and (4) the previously significant predictor (ADHD) - outcome relationship (CORE Negative Consequences Subscale total) is reduced significantly (as determined by the Sobel Test results) when effects of the mediator are controlled.

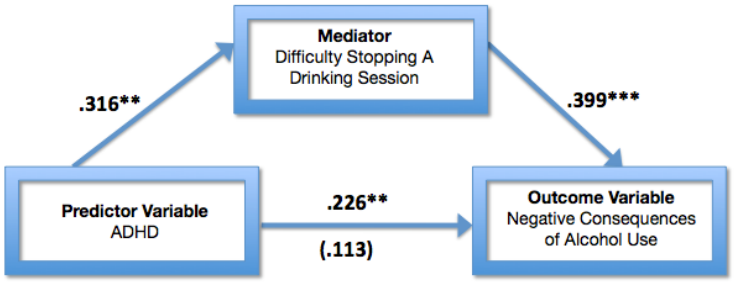
This model was tested using the three regression analyses (with conditions 3 and 4 being tested with a single regression equation). First, linear regression models were executed to examine whether ADHD diagnosis was significantly associated with negative consequences of alcohol use. Second, a linear regression analysis was conducted to examine whether ADHD diagnosis was significantly associated with difficulty stopping a drinking session. Third, in a linear regression equation ADHD diagnosis and the mediator variable, difficulty stopping a drinking session, were entered simultaneously on the same step to examine whether the variance accounted for by the relationship between ADHD diagnosis an the negative consequences of alcohol use was substantially reduced. Finally, the Sobel Test was applied to evaluate the statistical significance of the mediation effect (Sobel, 1982). Fraternity or sorority membership current drug use (CORE Drug Use Subscale total), alcohol quantity (AUDIT Item 2), and panic disorder were included as covariates in these analyses.

### *Aim 2a: Hypothesis Summary*

On the basis of the findings from our preliminary study, we hypothesized that self-reported frequency of difficulty stopping a drinking session would mediate the relationship between ADHD and self-reported negative consequences of alcohol use.

### *Aim 2a: Results*

Consistent with our hypothesis, the relationship between ADHD and negative consequences of alcohol use was mediated by difficulty stopping a drinking session. As Figure 1 illustrates, the standardized regression coefficients between ADHD and negative outcomes of alcohol use decreased substantially when controlling for difficulty stopping a drinking session, rendering the relationship between the predictor and outcome variables insignificant. Sobel Test results confirmed that the reduction in the association between ADHD and negative consequences of alcohol use was indeed significant (Sobel Test Statistic = 2.629, $p < .01$). The other conditions of mediation were also met: ADHD was a significant predictor of difficulty stopping a drinking session, and difficulty stopping a drinking session was a significant predictor of negative consequences of alcohol use while controlling for ADHD. Results are presented in Table 19.




*Note:* ADHD = Attention-Deficit/Hyperactivity Disorder. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 1. Standardized regression coefficients for the relationship between ADHD and negative consequences of alcohol use as mediated by difficulty stopping a drinking session. The standardized regression coefficient between ADHD and negative consequences of alcohol use controlling for difficulty stopping a drinking session is in parentheses.

### *Aim 2b: Analytic Plan*

First we examined the degree to which cue dependency and difficulty stopping a drinking session were correlated. Consistent with methods utilized in prior studies (e.g., Derefinko et al., 2007; Marczinski & Fillmore, 2003), a cue dependency score was calculated by subtracting the proportion of inhibitory failures on trials with consistent cue-target pairings from the proportion of inhibitory failures on trials with inconsistent cue-target pairings. Second, we examined cue-dependency on trials of response inhibition as a mediator of the relationship between ADHD and the negative consequences of alcohol use. The mediation analyses were conducted following Baron and Kenny's (1986) model for mediation (described in Aim 2a).

### *Aim 2b: Hypothesis Summary*

Correlations: Level of cue dependency and self-reported frequency of difficulty stopping a drinking session would be significantly correlated.

Mediation: Level of cue dependency would mediate the relationship between self-reported frequency of difficulty stopping a drinking session and self-reported negative consequences of alcohol use.

***Aim 2b: Results***

Correlations: Contrary to our hypothesis, cue dependency and difficulty stopping a drinking session were not significantly correlated ($r$ =.096, $p$ = .342). Results are presented in Table 9.

Mediation: Contrary to our hypothesis, the relationship between ADHD and negative consequences of alcohol use was *not* mediated by cue dependency. Specifically, the association between ADHD and cue dependency ($\beta$ =-.008, $p$ = .455) was not significant. Results are presented in Table 20.

***Aim 3a: Analytic Plan***

We examined whether the mean overall proportion of failures on trials of response inhibition was correlated with trait disinhibition as measured by the disinhibition subscale of the Sensation Seeking Scale – V (DIS-M; Zuckerman, 1994). If these two measures were significantly correlated, this would indicate that they are likely measuring a similar facet of disinhibition. If not correlated, it is likely that they are measuring separate aspects of the loosely defined behavioral disinhibition construct.

***Aim 3a: Hypothesis Summary***

We hypothesized that these two measures of disinhibition would not be significantly correlated.

### *Aim 3a: Results*

Behavior-based disinhibition and trait-based disinhibition were not significantly correlated ($r$ = .024, $p$ = .836). Results presented in Table 3.

### *Aim 3b: Analytic Plan*

Previous studies have shown that individuals with ADHD report higher levels of trait behavioral disinhibition than those without ADHD (for a review see Faraone, Kunwar, Adamson, & Biederman, 2009). Therefore, we explored group differences in self-reported trait disinhibition. We also evaluated whether trait disinhibition (DIS-M) is a mechanism by which ADHD and the negative consequences of alcohol use are associated. As per procedures used in previous studies (e.g., Darkes et al., 1998; Magid, MacLean, & Colder, 2007), we modified the disinhibition subscale (DIS-M) by removing items that included questions about alcohol and drug use prior to conducting exploratory analyses.

First, one-way ANOVA analyses were conducted to examine whether differences in self-reported behavioral disinhibition on the DIS-M existed between the ADHD and non-ADHD groups. Next we examined the degree to which trait disinhibition (DIS-M) was correlated with self-reported difficulty stopping a drinking session. Finally, we then examined self-reported behavioral disinhibition as a mediator of the relationship between ADHD and the negative consequences of alcohol use by employing Baron and Kenny's (1986) model for mediation (described in Aim 2a). Fraternity or sorority membership and current drug use (CORE Drug Use Subscale total) were included as covariates in these analyses.

***Aim 3b: Hypothesis Summary***

Between Group Comparisons: The ADHD group would have significantly higher levels of trait disinhibition than the non-ADHD group.

Correlation: Trait disinhibition would be significantly correlated with difficulty stopping a drinking session.

Mediation: Trait disinhibition would mediate the relationship between ADHD and the negative consequences of alcohol use.

***Aim 3b: Results***

Between Group Comparisons: Participants in the ADHD group had significantly higher scores on the DIS-M than participants in the non-ADHD group ($F(2, 98) = 5.411$, $p < .05$). Results are presented in Table 21.

Correlation: Self-reported behavioral disinhibition (DIS-M) was significantly correlated with difficulty stopping a drinking session ($r = .330$, $p < .01$). Results are presented in Table 6.

Mediation: Self-reported trait disinhibition (DIS-M) did not mediate the relationship between ADHD and the negative consequences of alcohol use when controlling for alcohol quantity as well as gender and fraternity/sorority status. Specifically, the associations between ADHD and the negative consequences of alcohol use remained significant ($\beta = .202$, $p < .05$) after DIS-M was added to the equation. DIS-M was not significant when ADHD was included in the model. ADHD, however, remained significant throughout all steps of the equation. Results are presented in Table 22.

## Discussion

The present study examined mechanisms underlying the association between ADHD and self-reported negative consequences of alcohol use in college students. While patterns of alcohol use have been studied in adolescents and young adults with confirmed ADHD diagnoses, to the best of our knowledge, only one published study, conducted in our laboratory, has focused exclusively on college students with confirmed DSM-IV-TR ADHD diagnoses (Rooney et al., 2011). Furthermore, to the best of our knowledge, no studies have examined potential mechanisms underlying the association between ADHD and alcohol-related problems. The existing literature on adolescents and young adults with ADHD provides a useful foundation for studies of college students with the disorder. Characteristics unique to college students and the college environment, however, limit the generalizability of findings derived from studies of individuals with ADHD who are not enrolled in college.

The present study focused on both personality trait- and behavior-based aspects of disinhibition, as well as reported difficulty stopping a drinking session, as mediators of the relationship between ADHD and negative consequences of alcohol use. A number of interesting findings were generated. When the ADHD and non-ADHD groups were compared on rates of comorbid psychopathology and substance use disorders, the ADHD group was found to have higher rates of past and current comorbid psychological disorders including alcohol and substance use disorders. This suggests that the study included a clinically representative sample, and that college students with ADHD have comorbidity profiles similar to their peers with ADHD who are not attending college. A detailed discussion of these findings is provided below. Regarding patterns of alcohol use

and related problems, findings are consistent with results from our prior research. Specifically, the ADHD group reported more negative consequences of their alcohol use and higher rates of difficulty stopping a drinking session despite reporting rates of alcohol consumption equivalent to those of the non-ADHD group. Possible explanations for these findings are presented below. In our examination of mediators of the relationship between ADHD and negative consequences of alcohol use, only difficulty stopping a drinking session mediated the relationship when controlling for alcohol quantity. Difficulty stopping a drinking session and trait disinhibition were significantly correlated with each other, suggesting that difficulty stopping a drinking session may be a manifestation of disinhibition. Trait disinhibition itself however, did not mediate the relationship between ADHD and the negative consequences of use when controlling for quantity. Possible explanations for the null findings are described below.

*Discussion of Preliminary Analyses*

A number of significant group differences and correlations were identified through preliminary analyses. When the ADHD and non-ADHD groups were compared on rates of DSM-IV Axis I disorders, the ADHD group has singificantly higher rates of past MDD episodes, higher rates of current social phobia, current specific phobia, and current panic disorder. These findings are generally consistent with patterns of comorbidity found among adolecents and young adults with ADHD (Bagwell, Molina, Kashdan, Pelham, & Hoza, 2006; Barkley, Murphy, & Kwasnik, 1996; Biederman et al., 2008; Biederman et al., 1993; Shekim, Asarnow, Hess, Zaucha, & Wheeler, 1990) . However, given the high rates of depression identified in numerous studies of adolescents and young adults with ADHD (Bagwell, Molina, Kashdan, Pelham, & Hoza, 2006;

Biederman et al., 2008; Chronis-Tuscano et al., 2010; Fischer, Barkley, Smallish, & Fletcher, 2002), it is surprising that in the current study no participants with ADHD endorsed symptoms consistent with a current MDD episode. Evidence suggests that when MDD co-occurs with ADHD, MDD has a longer durartion and results in greater impairment (Biederman et al., 2008). Thus, it is possible that participants with the impairment associated with comorbid ADHD and MDD interfered with students' ability to intiate the process of participating in a research study, resulting in a lack of representation in our sample. It is also possible that students with this pattern of comorbidity are less likely to successfully complete the transition from high school to college, and are therefore not represented among the majority of college stduents with ADHD. Additional studies are needed to better understand patterns of comorbidity among college students with ADHD.

Regarding substance use disorders, findings were consistent with studies of young adults with ADHD (Smith, Molina, & Pelham, 2003; Weiss & Hechtman, 1993), with participants in the ADHD group having higher rates of alcohol use disorders and non-alcohol substance use disorders. This finding suggests that college students with ADHD are experiencing higher rates of clinically significant impairment as a result of their substance use than their peers without ADHD. Surprisingly, higher rates of alcohol use and alcohol related problems were not associated with a history of MDD. This finding is inconsistent with studies showing higher rates of alcohol use and related problems among those with MDD or elevated depression sympatology (Dixit & Crum, 2000; Harrell & Karim, 2008; Nolen-Hoeksema, 2004; Pedrelli et al., 2010). One posibility for the discrepant finding is that only those experiencing a current MDD episode, or currently

elevated depression sympomatology, show higher rates of use. No participants in the current sample endorsed symtpoms consistent with a current MDD episode. Only past MDD episodes were reported. Significant correlations among substance use and alcohol use disorders were also identified. While current alcohol dependence was not significantly associated with any substance use disorders, lifetime alcohol dependence and both current and lifetime alcohol abuse were significantly associated with both current and lifetime substance dependence as well as current substance abuse. The high correlations among these diagnoses are consistent with studies showing high rates of comorbidity among alcohol and substance use disorders. In addition, these findings are consistent with Krueger's two-factor model of mental disorder classification (Krueger, 1999). According to Krueger's (1999) analyses of the National Comorbidity Survey data, 10 common DSM-IV mental disorders fall onto internalizing and externalizing factors, with substance use disorders as well as antisocial personality disorder falling on the externalizing factor. In a replication study examining the consistency of Krueger's two-factor model in a sample of adolescents and young adults, the structure of the externalizing factor was applicable (Wittchen et al., 2009). Additional studies are needed to determine the genetic, neurobiological, or environmental factors underlying the high rate of comorbidity among substance use disorders (Krueger & Markon, 2006).

*Discussion of Primary Aims*

Prior to examining mechanisms that may underlie the association between ADHD and alcohol-related problems, we sought to replicate findings from our preliminary study (Rooney et al., 2011) which found significant associations between ADHD and difficulty stopping a drinking session as well as associations between ADHD and the negative

consequences of alcohol use. These findings were replicated in the current sample. In all cases, the associations were in a positive direction, such that college students with ADHD reported higher rates of both difficulty stopping drinking and negative consequences of alcohol use. In the current study, trait disinhibition (as measured by the DIS-M) was higher in the ADHD group than the non-ADHD group (see Table 7). In addition, trait disinhibition was moderately correlated with difficulty stopping a drinking session (see Table 9). Therefore, it is likely that trait disinhibition is accounting for a proportion of the variance in difficulty stopping a drinking session. There are a number of additional factors not measured in the current study that may explain why those with ADHD report higher rates of difficulty stopping drinking than those without ADHD. First, individuals with ADHD have been shown repeatedly to have a decreased sensitivity to delayed rewards, which frequently presents as a preference for smaller immediate rewards over larger long-term rewards (Castellanos, 2009; Paloyelis, Asherson, & Kuntsi, 2009). It is possible that for those with ADHD the delayed reward of stopping drinking (e.g., preventing nausea or a hangover the next day) is not sufficiently motivating to stop the individual from drinking in the moment. Second, it is possible that one of the facets of disinhibition identified in recent models of disinhibition (Cyders et al., 2007; Dick et al., 2010; Whiteside & Lynam, 2001) but not measured in the current study (i.e., positive urgency, negative urgency, lack of planning, and lack of perseverance), are elevated in individuals with ADHD and contribute to the higher rates of difficulty stopping drinking identified in the current ADHD sample. Additional studies are needed to examine these factors and their association with ADHD and difficulty stopping a drinking session.

Consistent with studies of adolescents and young adults with ADHD (Weiss & Hechtman, 1993; Wilens et al., 1998; Wilens et al., 2002) and with our preliminary findings (Rooney et al., 2011), college students with ADHD did not report consuming alcohol in greater quantities than their non-ADHD peers. Thus, it does not appear to be the *amount* of alcohol consumed by college students with ADHD that puts them at risk for alcohol-related problems. Rather, this population appears to be more vulnerable to experiencing negative consequences during the past year as a result of their alcohol use. In the current study, we did not specifically examine factors explaining why students with ADHD report more negative consequences of alcohol use relative to the non-ADHD group despite reporting a statistically equivalent amount of alcohol consumed.

A more in depth evaluation of the specific negative consequences endorsed more frequently by the ADHD group may aid in forming hypotheses regarding this phenomenon. Of the 16 negative consequences measured, 6 were endorsed more frequently in the ADHD group than the non-ADHD group (i.e., doing something s/he later regretted, having a hangover, becoming nauseated or vomited, getting into an argument or fight, being criticized by someone he/she knows, thinking [he/she] may have a drinking problem). One consequence, being arrested for a DUI/DWI occurred for only one participant (non-ADHD) in the full sample and was excluded from analyses. The remaining 9 negative consequences did not occur at significantly different rates between the two groups (i.e., performing poorly on a test or important project, missing a class, being in trouble with police or college authorities, driving a car while under the influence, damaging property or pulling fire alarm etc., being hurt or injured, having memory loss, being taken advantage of sexually).

Notably, the negative consequences occurring with greater frequencies in the ADHD group included the 2 items related to interpersonal problems (i.e., getting into an argument or fight, being criticized by someone [he/she] knows). This finding is consistent with studies that have identified social skill deficits in children with ADHD (Hoza et al., 2005). It is possible that college students with ADHD are compromised in their interpersonal interactions even when they are not consuming alcohol. Thus, when they consume alcohol their weakness in interpersonal interactions is exacerbated, leading to behavior patterns that elicit criticism from friends and concern on the part of the ADHD student that [he/she] may have a drinking problem.

A third item endorsed more significantly more frequently in the ADHD group, "thinking I may have a drinking problem," (i.e., concern on the part of the student that they, themselves, may have a drinking problem) is likely related to "being criticized by someone I know" since two items are significantly correlated within the ADHD group (see Table 15). Furthermore, "thinking I may have a drinking problem" is correlated with only one of the 6 negative consequences that occurred with greater frequency in the ADHD group: "being criticized by someone I know." This is striking since *all* 5 of the other negative consequences were significantly correlated with each other (see Table 16). Interestingly, no participants in the non-ADHD group reported that they ever thought they might have a drinking problem during the past year. Therefore, endorsement of this item is unique to the ADHD sample and shows a unique pattern of correlation with other items.

It is possible that a fourth item endorsed with greater frequency by the ADHD group, "doing something [he/she] later regretted," is also related to interpersonal

problems. This cannot be confirmed, however, since participants were not asked to provide a description of the "something" that was later regretted. The final 2 items that were endorsed more frequently by the ADHD group were related to the physical effects of alcohol (i.e., having a hangover and being nauseated or vomiting). It is surprising that these were endorsed with greater frequency by the ADHD group since they did not report consuming higher quantities of alcohol. It is possible that individuals with ADHD are paying less attention to the timing and spacing of their alcohol consumption, and are therefore experiencing more negative physical effects than those without ADHD. It is also possible that students are minimizing the quantity of alcohol consumed. A study examining ad-lib alcohol consumption in a laboratory setting would be helpful in exploring this association.

The nine negative consequences not endorsed with greater frequency by the ADHD group included two items related to academic performance, "performing poorly on a test or important project" and "missing a class." Given that individuals with ADHD tend to experience more academic problems than their peers without ADHD (DuPaul et al., 2009; Raggi & Chronis, 2006), it is surprising that this vulnerability would not be exacerbated by alcohol use. It is possible that participants with ADHD did not attribute any poor performance or missing a class to alcohol use, but instead attributed this to their ADHD symptoms, their tendency toward unpredictable academic performance, or external factors such as having unfair questions on a test. Alternatively, it is possible that there were actually no differences between groups on this variable.

Three of the items not endorsed with greater frequency by those with ADHD included negative consequences associated with potential legal consequences, "being in

trouble with police or college authorities," "driving a car while under the influence," and "damaging property, pulling a fire alarm, etc." In exploratory analyses, these three items were found to be significantly associated with childhood- and adolescent-onset CD. In fact, only 4 of the 15 negative consequences were significantly correlated with CD. The fourth item, "being hurt or injured" was also not indorsed with greater frequency by the ADHD group. The rate of CD in this sample was relatively low (Moffit, 2003). Additional studies are needed to determine whether this rate of CD is typical of college students with ADHD in general or if it is specific to the current sample. Regardless, it is possible that in a sample with a higher rate of CD, these items would have been endorsed more frequently by the ADHD group but would have been attributed to the comorbid CD. The remaining two items not endorsed with greater frequency by the ADHD group are "having memory loss" and "being taken advantage of sexually." It is surprising that the rate of reported memory loss was not endorsed with greater frequency by the ADHD group since the two additional items related to the physical effects of alcohol were reported more frequently in this group. It is also surprising that being taken advantage of sexually was not endorsed at a higher rate by this group since young adults with ADHD have been found to engage in higher rates of risky sexual behavior (Flory, Molina, Pelham, Gnagy, & Smith, 2006). It is possible however, that the low base-rate of endorsement and small sample size lead to an absence of between-group differences.

In our preliminary study we found that difficulty stopping a drinking session, a possible manifestation of behavioral disinhibition while under the influence of alcohol (Weafer & Fillmore, 2008), mediated the relationship between ADHD and the negative consequences of alcohol use. This finding was replicated with our current sample and

represents a mechanism underlying the association between ADHD and alcohol-related problems. The identification of mechanisms is necessary for the development of targeted interventions (Sonuga-Barke & Halperin, 2010). While it is likely challenging to develop an intervention that targets problems associated with stopping a drinking session, students with ADHD can at the very least be informed of their vulnerability for experiencing this problem. With this awareness students can proactively implement conditions that my help to limit their alcohol consumption. For example, they can purchase smaller quantities of alcohol or limit their access to alcohol. Alternatively, they may choose to rely on peers who can help them limit the number of drinks they consume in one sitting. In addition, students (possibly with the assistance of peers) may choose not to join friends for "one or two drinks" if they recognize that they may have difficulty stopping. If therapists and counselors are made aware of the need to assess for difficulty stopping a drinking session in their clients with ADHD, they can assist in the development and implementation of these strategies. Additionally, in recent years there has been a growing interest in the development of single-item screeners able to identify high-risk drinkers who present to university health center and primary care settings (Dawson, Pulay, & Grant, 2010; Foote, Wilkens, & Vavagiakis, 2004). Although highly speculative until further evaluation, the single question of whether a student ever experiences difficulty stopping a drinking session may be a candidate for a single-item screener, particularly for students with ADHD who visit university health centers regularly for medication management.

Researchers have identified a compensatory mechanism, cue dependency, which protects healthy adults against the disinhibiting effects of alcohol on behavioral tasks (for a review see Fillmore, 2003). Since no studies have examined cue dependency in sober

adults with ADHD, our first task was to establish the presence of a cue dependency deficit under sober conditions. To this end, we employed the cued go/no-go task (Fillmore, 2001) which has been used frequently in studies of healthy adults under the influence of alcohol (for a review see Fillmore, 2003) and in the single study examining cue dependency in adults with ADHD under the influence of alcohol (Weafer et al., 2009). Contrary to our predictions, ADHD participants did not display a cue dependency deficit on trials of response inhibition in the current study. Instead, ADHD participants appear to have relied on the cues to prepare their responses to targets on trials of response inhibition. Cue dependency in the ADHD group mirrored that of the non-ADHD group, with participants performing significantly better on trials where cue-target pairings were consistent (no-go cue followed by a no-go target) than on trials where cue-target pairings were inconsistent (go cue followed by a no-go target). The ADHD and non-ADHD groups differed however, on the mean overall proportion of response inhibition failures. Across cue conditions, the ADHD group had a significantly higher proportion of inhibitory failures than the non-ADHD group. This is consistent with findings from the study of adults with ADHD under the influence of alcohol (Weafer et al., 2009) and studies of children with ADHD (Derefinko et al., 2008; Durston et al., 2007). Moreover, the ADHD and non-ADHD groups did not differ significantly in mean reaction time on trials of response execution. Therefore, the higher rate of inhibitory failures in the ADHD group cannot be attributed to differences in processing speed.

There are a number of possible reasons why the ADHD group did not display a cue dependency deficit as predicted. First, the cued go/no-go task may not have been sufficiently challenging to allow for the detection of differences in cue dependency

magnitude, either because participant performance was not impaired by alcohol or because members of the ADHD group were derived from a high functioning subset of those with the disorder (i.e., college students). Supporting this explanation is the small mean proportion of inhibitory failures (*p*-inhibition failures) observed in each group (see Table 7). Second, it is possible that the cue dependency deficit observed in adults with ADHD when under the influence of alcohol (Weafer et al., 2009) is simply not present in adults with ADHD when they are in a sober state. Alcohol may acutely affect cognitive functions underlying cue dependency in individuals with ADHD differently than it does in those without ADHD, making them more susceptible to negative consequences of alcohol use. Third, it is possible that the simple cue dependency score used in this study was insufficient; despite the fact that others have previously used this method when analyzing cued go/no-go task performance (Marczinski et al., 2005; Marczinski & Fillmore, 2003; Weafer & Fillmore, 2008). The cue dependency score, like all basic difference scores, does not account for differences in baseline level of performance (Cohen, Cohen, West, & Aiken, 2002). Cue dependency scores increase as failures of response inhibition increase (Fillmore, 2003). Without utilizing a cue dependency value that accounts for differences in the proportion of inhibitory failures, one cannot differentiate effects due to cue dependency from those due to basic response inhibition. In addition, just as the cue dependency score is confounded with the proportion of response inhibition failures, the mean overall response inhibition value derived from the cued go/no-go task is not independent of cue dependency. Since all response inhibition targets were preceded by preresponse cues, the degree to which participants engaged in early response preparation in response to cues influenced their overall task performance. Future

studies should include a measure of response inhibition independent of cue dependency (e.g., the stop task; Logan, 1984) to isolate these cognitive mechanisms.

In addition to studying disinhibition as defined from a behavior-based perspective, we examined associations between personality-based disinhibition as measured by the disinhibition subscale (DIS-M) of the Sensation Seeking Scale – V (Zuckerman, 1994), modified by removal of the three alcohol and drug items. The disinhibition subscale was selected because it is highly correlated with alcohol use in studies of healthy adults (Hittner & Swickert, 2006). Individuals who score high on this subscale tend to be less constrained by social norms and are generally more experimental in regard to their behavior. As discussed previously, questionnaires measuring trait disinhibition have the benefit of capturing both cognitive and affective aspects of disinhibition as they relate to real-world behavior (Reynolds et al., 2006). Personality trait-based measures of disinhibition, however, are typically not highly correlated with behavioral measures of disinhibition (for a review, see Dick et al., 2010). This suggests that trait- and behavior-based measures may be capturing separate facets of disinhibition. Consistent with the existing literature, in the current study, trait disinhibition was not significantly correlated with task-based disinhibition (i.e., overall performance on cued go/no-go response inhibition trials; see Table 9). While the behavioral measure of response inhibition used in the current study was confounded by cue dependency, it appears that trait disinhibition and response inhibition as captured by the cued go/no-go task likely measures two separate facets of disinhibition.

Trait disinhibition has consistently been found to be associated with the development of alcohol-related problems in healthy adults (e.g., Hittner & Swickert,

2006; Magid, MacLean, & Colder, 2007; Sher et al., 2000). To the extent that difficulty stopping a drinking session is a manifestation of trait disinhibition, we predicted that trait disinhibition would mediate the relationship between ADHD and the negative consequences of alcohol use. Contrary to our prediction, trait disinhibition did not mediate this relationship. There are a number of possible reasons why our hypothesis was not supported. First, trait disinhibition was found to mediate the association between ADHD and the negative consequences of alcohol use when alcohol quantity was not included as a covariate (see Table 12). Since alcohol quantity and trait disinhibition are moderately correlated ($r$ =.394, $p$ < .01), it is possible that the positive association between trait disinhibition and the negative consequences of alcohol use is driven heavily by the quantity of alcohol consumed. Therefore, the relationship between trait disinhibition and negative consequences of alcohol use, while significant, is not strong enough to account for the association between ADHD and the negative consequences of alcohol use when controlling for alcohol quantity. Second, it is possible that with a larger sample and greater statistical power, the influence of trait disinhibition on the relationship between ADHD and the negative consequences of use could be detected.

Trait disinhibition has been found *not* to predict *ad lib* alcohol consumption in a laboratory setting (Weafer & Fillmore 2008). This finding is inconsistent with the current study where there was a significant association between trait disinhibition and the self-reported quantity of alcohol typically consumed. This inconsistency may be due to the fact that the trait-based measure of disinhibition differed between the two studies. Since behavioral disinhibition is a loosely defined construct, trait-based questionnaires often

differ greatly in item selection. Therefore, each disinhibition scale differs in the degree to which it is correlated with alcohol use measures (Dick et al., 2010). In addition, while *ad lib* alcohol consumption in a laboratory setting has been found to be a valid indicator of alcohol consumption in real-world settings (Collins, Gollnisch, & Izzo, 1996; Marczinski, Bryant, & Fillmore, 2005), it differs significantly from self-report in its measurement of alcohol consumption. This difference in measurement of alcohol quantity may also explain the inconsistency in findings across studies.

These findings and the questions they generate highlight the need for multi-method multi-trait study designs when examining factors related to alcohol consumption and complex, multifaceted traits such as disinhibition. Shared method variance (i.e., variance attributable to the measurement rather than to the constructs the measures represent) is a potential problem in all behavioral research that relies on a single measurement modality (e.g., self-report forms), and/or a single informant, and/or a single time point (Campbell & Fiske, 1959; Kazdin, 2003). A number of meta-analyses of multitrait-multimethod studies (MTMM) have examined the extent to which shared method variance is present in measures used in behavioral studies. In the most comprehensive of these meta-analyses the amount of common method variance present in measures was examined across 70 MTMM studies (Podaskoff, MacKenzie, Lee, & Podaskoff, 2003). The authors found that shared method variance accounted for 26.3% of the variance in a typical research measure. Using an example from the present study, shared method variance could partially explain the significant correlation between self-reported alcohol quantity and self-reported disinhibition as measured by the DIS-M and the absence of a significant correlation between self-reported alcohol quantity and

behavioral disinhibition on the cued-go/no-go task. It is also possible that the strength of the correlation between alcohol quantity and disinhibition differed between the two measurement methods because the two measures of disinhibition are tapping two different constructs. As described previously, disinhibition is a multifaceted construct, and behavioral measures of disinhibition are typically not significantly correlated with self-report measures of disinhibition (Dick et al. 2010). In fact, in the current study self-reported disinhibition and behavioral disinhibition were not significantly correlated. This example highlights the need for multiple measures, particularly when a multi-faceted, complex trait is being examined. In addition, alcohol use should be measured using both self-report and observation of *ad lib* alcohol consumption in a laboratory setting across multiple time points.

*Limitations and Future Directions*

Several limitations in the current study should be noted. First, the cross-sectional nature of the study limits our ability to draw any inferences about causality. Since heavy alcohol use can lead to higher levels of disinhibition over time (Bartsch et al., 2007), it is possible that higher levels of trait-disinhibition were a result of heavy alcohol use history. Second, the study relied solely on self-report data for information related to alcohol use. Objective measures of use (e.g., hair follicle testing) could potentially provide more accurate information and minimize the inflation of results from shared method variance across measures (Harrison & Huges, 1997). In addition, the use of a laboratory task to measure ad-lib alcohol consumption would be a valuable addition as an observable measure quantity of alcohol consumed and difficulty stopping a drinking session.

Third, the study did not include a non-college student ADHD comparison group.

The absence of this comparison group limits our ability to interpret differences between current study findings and those of previous studies examining non-college young adults with ADHD. In addition, participants in the ADHD group were, on average, in their sophomore year of college. Thus, they may represent a particularly high functioning group since they have already succeeded in managing the adjustment to college that occurs during freshman year. Future studies should aim to enroll those students who enter college but drop out during or after their freshman year. In addition, 24% of the ADHD group and 22% of the non-ADHD group were members of fraternities or sororities. Since these organizations have GPA requirements for membership it is possible that participants who were members of these organizations are also higher functioning than the general college population. Fourth, since ADHD is a heterogeneous diagnostic category (APA, 2000), examining differences in patterns of alcohol use and disinhibition on the basis of ADHD subtype may be of value (Derefinko et al., 2008). The study's small sample size and large number of analyses did not allow for an examination of ADHD subtype (i.e., ADHD-I, ADHD-C) differences. In fact, our relatively small sample limited our ability to conduct a number of important analyses. These include examining the moderating effects of factors such as gender, ADHD subtype, and ADHD medication status. Since each of these factors is accompanied by different correlates and comorbidity profiles, they should be examined in future studies with larger samples. Fifth, the current study was conducted at a large, public, Mid-Atlantic university with stringent admissions criteria and included only "traditional" college students (i.e., those attending college full-time immediately or shortly after completing high school). Therefore, results can only be generalized to similar students with ADHD attending universities with similar

characteristics. Additional studies using larger, more diverse samples at multiple universities are needed to address these limitations. Sixth, as described in detail previously, the cued go/no-go task may not be appropriate (e.g., sufficiently challenging, etc.) for assessing cue dependency in sober adults and fails to independently assess the constructs of cue dependency and response inhibition. Lastly, behavioral disinhibition is a multi-faceted construct that is loosely defined (Dick et al., 2010). In our study we incorporated one of many personality trait-based measures of disinhibition. It is possible, or even likely, that other personality trait-based measures of disinhibition would produce different results. Therefore, results must be interpreted such that they apply only to disinhibition *as measured by* the modified version of the Sensation Seeking Scale – V disinhibition subscale. Future studies should seek to further refine the definition of the disinhibition construct and incorporate multiple scales measuring different facets of the trait.

A variety of factors not examined in the current study may explain group differences in patterns of problematic alcohol use. Family history of alcoholism is one widely recognized risk factor for the development of alcohol related problems. Studies have shown that individuals with a family history of alcoholism are at increased risk for developing alcohol use disorders themselves (Chassin, Pitts, & Prost, 2002; Dawson, 2000; Dawson, Hartford, & Grant, 1992; Schuckit, 1998; Sher, Walitzer, Wood, & Brent, 1991; Weitzman & Weschler, 2000). Those with a greater familial density of alcohol use disorders, as measured by the number of family members with an alcohol use disorder, appear to be at the highest risk for the development of alcohol-related problems (Capone & Wood, 2008). Family history of alcoholism may be a particularly important factor to

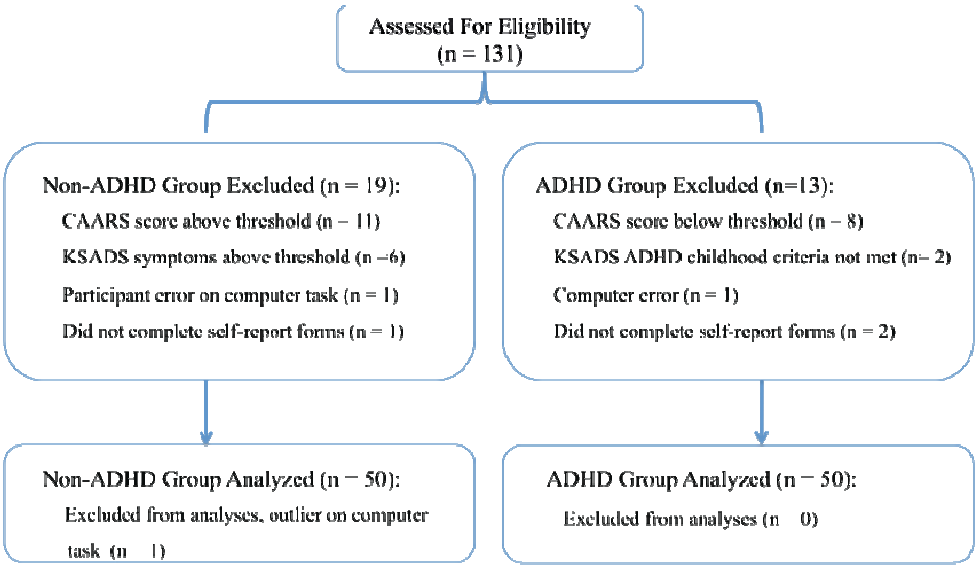
consider in future studies of individuals with ADHD given than those with ADHD are more likely to have a family history of alcoholism than those without ADHD (Knopik et al., 2005; Roizen et al., 1996).

Patterns of alcohol use during high school predict patterns of alcohol use during college. Specifically, college students who engage in heavy episodic drinking during high school have higher rates of heavy episodic drinking and alcohol-related problems during college (Harford, Weschler, & Muthen, 2002; Johnston et al., 2009; Wechsler, Dowdall, Davenport, & Castillo, 1995; Wechsler, Lee, Kuo, & Lee, 2000). Therefore, intra-individual and environmental factors present before and during college should be considered when studying the eitology, prevention, and treatment of problematic alcohol use in college students. Pro-alcohol peer influence has also been found to be one of the strongest predictors of alcohol consumption patterns in college students (Baer, 2002; Borsari & Carey, 2001; Jackson, Sher, Gotham, & Wood, 2001; White, Fleming, Kim, Catalano, & McMorris, 2008). This influence results from both selection and socialization effects (Leibsohn, 1994). The selection effects may be particularly salient when considering students with ADHD. ADHD is associated with higher levels of sensation seeking traits (Downey, Pomerleau, & Pomerleau, 1996), which in turn, are associated with higher rates of risky patterns of alcohol use (Watten & Watten, 2010). To the extent that students with ADHD are seeking out peers similar to themselves, they may select peers who model and promote risky patterns of alcohol use. In addition, research suggests that for students with a family history of alcohol problems, pro-alcohol peer influence is more strongly linked to alcohol use during the college years relative to those without a family history of alcohol problems (White & Jackson, 2004). This finding

highlights the interconnectedness of many of the variables examined in studies of college student alcohol use and the difficulty associated with isolating specific pathways to the development of alcohol-related problems.

Despite these limitations, findings from the current study provide additional evidence that college students with ADHD are a population at risk for the development of alcohol-related problems. In addition, this study is the first to identify a mechanism, difficulty stopping a drinking session, which may partially explain the relationship between ADHD and the negative consequences of alcohol use. This information can help to further elucidate the underlying causes of alcohol-related problems in this group, and may represent a target for prevention and intervention initiatives with this population.

## Tables and Figures




*Note.* ADHD = Attention Deficit Hyperactivity Disorder. CAARS = Conners Adult ADHD Rating Scale. KSADS = Schedule for Affective Disorders and Schizophrenia for school age children ADHD module modified for use with adults to assess past and current symptoms.
Figure 2. Flow diagram of participant assessment and inclusion in analyses.

Table 1

*Demographic and Diagnostic Characteristics*

| Characteristic | Non-ADHD *n = 50* | | ADHD *n = 50* | | Group Contrasts |
|---|---|---|---|---|---|
| | *N* | *(%)* | *N* | *(%)* | |
| Age (*M, SD*) | 19.02 | (1.040) | 20.14 | (1.457) | $t(98) = 4.424$, $p = .045$* |
| Year in School (*M, SD*) | 13.76 | (.8703) | 14.58 | (1.071) | $t(98) = 4.202$, $p = .031$* |
| Fraternity/Sorority | 11 | (22) | 12 | (24) | $\chi(1,N=100) = .056$, $p = .500$ |
| Gender | | | | | |
| Male | 22 | (44) | 22 | (44) | $\chi(1, N=100) = 0.000$, $p = .580$ |
| Female | 28 | (56) | 28 | (56) | |
| Ethnicity | | | | | |
| Hispanic or Latino | 4 | (8) | 2 | (4) | $\chi(1, N=100) = 0.049$, $p = .339$ |
| Not Hispanic or Latino | 46 | (92) | 48 | (96) | |
| Race | | | | | |
| White | 36 | (72) | 35 | (70) | $\chi(1, N=100) = 0.049$, $p = .500$ |
| Black/ African American | 8 | (16) | 2 | (4) | $\chi(1, N=100) = 4.000$, $p = .047$* |
| Asian | 5 | (10) | 6 | (12) | $\chi(1, N=100) = 0.102$, $p = .500$ |
| More than one race | 0 | (0) | 5 | (10) | $\chi(1, N=100) = 5.263$, $p = .028$* |
| Other | 1 | (2) | 2 | (4) | $\chi(1, N=100) = 0.334$, $p = .500$ |
| DSM-IV-TR Diagnoses | | | | | |
| ADHD | | | | | |
| Predominately Inattentive | 0 | (0) | 19 | (38) | --- |

| | | | | | |
|---|---|---|---|---|---|
| Hyperactive/Impulsive | 0 | (0) | 1 | (2) | --- |
| Combined | 0 | (0) | 30 | (60) | --- |
| CD: Childhood Onset | 0 | (0) | 3 | (6) | --- |
| CD: Adolescent-Onset | 0 | (0) | 7 | (14) | --- |
| Learning Disability | 0 | (0) | 9 | (18) | --- |
| ADHD Medication - Current | 0 | (0) | 23 | (46) | --- |
| WTAR Score *(M, SD)* | 113.12 | (10.440) | 112.19 | (10.191) | $t(96) = .447$, $p = .760$ |
| Previous ADHD Diagnosis | 0 | (0) | 26 | (52) | --- |

*Note*. DSM-IV-TR = Diagnostic and Statistical Manual, Fourth Edition, Text Revision. ADHD = Attention-Deficit/Hyperactivity Disorder. CD = Conduct Disorder. WTAR = Wechsler Test of Adult Reading.

* Differences between groups at $p < .05$.

Table 2

*Within ADHD Group Comparison on Outcome Variables Between Participants With a Self-Reported Learning Disability and those without a Self-Reported Learning Disability*

| Variable | No Learning Disability *n = 41* | | Learning Disability *n = 9* | | Group Contrasts |
|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | |
| Difficulty Stopping Drinking | .51 | .746 | .33 | .707 | $t(48) = .657$, $p = .514$ |
| Alcohol Quantity | .151 | 1.165 | 1.22 | .833 | $t(48) = .706$, $p = .484$ |
| DIS-M | 4.170 | 1.986 | 4.444 | 2.068 | $t(48) = -.372$, $p = .712$ |
| Negative Consequences | 12.024 | 9.456 | 12.889 | 10.959 | $t(48) = -.242$, $p = .810$ |
| Cue Dependency | .017 | .0561 | .010 | .0567 | $t(48) = .377$, $p = .708$ |

*Note.* ADHD = Attention-Deficit/Hyperactivity Disorder.
DIS-M = Sensation Seeking Scale – V Disinhibition Subscale Modified.

Table 3

*Within ADHD Group Comparison on Outcome Variables Between Participants Currently Taking ADHD Medication and Participants Not Currently Taking ADHD Medication.*

| Variable | No Medication *n = 27* | | Medication *n = 23* | | Group Contrasts |
|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | |
| Difficulty Stopping Drinking | .48 | (.700) | .48 | (.790) | $t(48) = .015$, $p = .998$ |
| Alcohol Quantity | 1.44 | 1.188 | 1.48 | 1.039 | $t(48) = -.106$, $p = .916$ |
| DIS-M | 3.9529 | 2.335 | 4.565 | 1.440 | $t(48) = -1.140$, $p = .260$ |
| Negative Consequences | 10.593 | 9.249 | 14.043 | 9.934 | $t(48) = -1.271$, $p = .210$ |
| Cue Dependency | 00.78 | .050 | .026 | .0611 | $t(48) = -1.189$, $p = .240$ |

*Note.* ADHD = Attention-Deficit/Hyperactivity Disorder. DIS-M = Sensation Seeking Scale – V Disinhibition Subscale Modified.

Table 4

*Within ADHD Group Comparison on Outcome Variables Between Participants With and Without a Prior ADHD Diagnosis*

| Variable | No Prior ADHD Diagnosis *n = 24* | | Prior ADHD Diagnosis *n = 26* | | Group Contrasts |
|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | |
| Difficulty Stopping Drinking | .54 | .721 | .42 | .721 | $t(48) = .566$, $p = .574$ |
| Alcohol Quantity | 1.54 | 1.141 | 1.38 | 1.098 | $t(48) = .496$, $p = .622$ |
| DIS-M | 4.21 | 2.084 | 4.23 | 1.924 | $t(48) = -.040$, $p = .969$ |
| Negative Consequences | 11.33 | 10.110 | 12.96 | 9.29 | $t(48) = -.593$, $p = .556$ |
| Cue Dependency | .02 | .061 | .01 | .0498 | $t(48) = .945$, $p = .350$ |

*Note.* ADHD = Attention-Deficit/Hyperactivity Disorder. DIS-M = Sensation Seeking Scale – V Disinhibition Subscale Modified.

Table 5
*DSM-IV Axis I Diagnosis By Group*

| Diagnosis | Non-ADHD *n = 50* | | ADHD *n = 50* | | Group Contrasts |
|---|---|---|---|---|---|
| | *N* | *%* | *N* | *%* | |
| CD: Childhood Onset | 0 | 0 | 3 | 6.0 | N/A |
| CD: Adolescent-Onset | 0 | 0 | 3 | 6.0 | N/A |
| MDD Current | 0 | 0 | 0 | 0 | N/A |
| MDD Past | 6 | 12.0 | 30 | 60.0 | $\chi$(1,N=100) = 25.000, $p$ =.000** |
| Dysthymia Current | 0 | 0 | 1 | 2.0 | $\chi$(1,N=100) = 1.031, $p$ =.495 |
| Dysthymia Past | 0 | 0 | 3 | 6.0 | $\chi$(1,N=100) = 3.093, $p$ =.121 |
| Manic Episode Current | 0 | 0 | 0 | 0 | N/A |
| Manic Episode Past | 0 | 0 | 0 | 0 | N/A |
| Panic Current | 2 | 4.0 | 8 | 16.0 | $\chi$(1,N=100) = 4.00, $p$ =.046* |
| Social Phobia Current | 4 | 8.0 | 13 | 26.0 | $\chi$(1,N=100) = 6.775, $p$ =.009** |
| Specific Phobia Current | 7 | 14.0 | 14 | 28.0 | $\chi$(1,N=100) = 2.954, $p$ =.070 |
| Generalized Anxiety Disorder | 6 | 12.0 | 7 | 14.0 | $\chi$(1,N=100) = .088, $p$ =.500 |
| OCD Current | 0 | 0 | 1 | 2.0 | $\chi$(1,N=100) = 1.010, $p$ =.500 |
| Anorexia Current | 0 | 0 | 0 | 0 | N/A |
| Bulimia Current | 0 | 0 | 1 | 2.0 | $\chi$(1,N=100) = .000, $p$ =.753 |
| Alcohol Dependence Current | 1 | 2.0 | 6 | 12.0 | $\chi$(1,N=100) = 3.840, $p$ =.056 |
| Alcohol Dependence Lifetime | 3 | 6.0 | 11 | 22.0 | $\chi$(1,N=100) = 5.316, $p$ =.020* |
| Alcohol Abuse Current | 7 | 14.0 | 18 | 36.0 | $\chi$(1,N=100) = 6.453, $p$ =.010* |
| Alcohol Abuse Lifetime | 7 | 14.0 | 22 | 44.0 | $\chi$(1,N=100) = 10.928, $p$ =.001** |
| Substance Dependence Current | 2 | 4.0 | 5 | 10.0 | $\chi$(1,N=100) = 1.382, $p$ =.218 |
| Substance Dependence Lifetime | 2 | 4.0 | 5 | 10.0 | $\chi$(1,N=100) = 1.382, $p$ =.218 |

| | | | | | |
|---|---|---|---|---|---|
| Substance Abuse current | 4 | 8.0 | 8 | 16.0 | $\chi(1,N=100) = 2.990$, $p = .074$ |

*Note.* CD = Conduct Disorder. MDD = Major Depressive Disorder. OCD = Obsessive Compulsive Disorder. DSM-IV = Diagnostic and Statistical Manual, 4th revision. ** $p < .01$. *** $p < .001$.

Table 6

*Correlations among DSM-IV Axis I Diagnosis Significantly Associated with ADHD and Substance Use Disorders*

| | MDD Past | Panic Current | Social Phobia Current | Alcohol Dependence Current | Alcohol Dependence Lifetime | Alcohol Abuse Current | Alcohol Abuse Lifetime | Substance Dependence Current | Substance Dependence Lifetime | Substance Abuse Current |
|---|---|---|---|---|---|---|---|---|---|---|
| MDD Past | 1 | | | | | | | | | |
| Panic Current | .003 | 1 | | | | | | | | |
| Social Phobia Current | -.026 | .085 | 1 | | | | | | | |
| Alcohol Dependence Current | .009 | .250* | -.138 | 1 | | | | | | |
| Alcohol Dependence Lifetime | -.023 | .300** | .022 | .702** | 1 | | | | | |
| Alcohol Abuse Current | .018 | -.048 | -.141 | .266** | .354** | 1 | | | | |
| Alcohol Abuse Lifetime | .072 | .057 | -.070 | .299** | .472** | .879** | 1 | | | |
| Substance Dependence Current | -.042 | -.096 | .075 | .064 | .324** | .213** | .324** | 1 | | |
| Substance Dependence Lifetime | -.042 | -.096 | .075 | .064 | .324** | .213** | .324** | 1.000** | 1 | |
| Substance Abuse Current | -.042 | .149 | .051 | .215* | .254* | .201* | .254* | .360** | .360** | 1 |

*Note.* DSM-IV = Diagnostic and Statistical Manual, Fourth Revision. ADHD = Attention Deficit Hyperactivity Disorder. MDD = Major Depressive Disorder. * $p < .05$. ** $p < .01$.

*Table 7*

*Means of Outcome Variables*

| | | | Group | | | | |
|---|---|---|---|---|---|---|---|
| | | | ADHD | | Non-ADHD | | |
| | | | *n = 50* | | *n = 50* | | |
| Variable | Units | Range | *M* | *SD* | *M* | *SD* | Group Contrasts |
| Alcohol & Drug Use | | | | | | | |
| Quantity /Frequency | 0 - 16 | 10 | 5.28 | 2.588 | 4.40 | 2.864 | $F(2, 98) = 1.086$ $p = .100$ |
| Quantity | 0 - 4 | 4 | 1.46 | 1.110 | 1.26 | 1.065 | $F(2,98) = .845$ $p = .360$ |
| Difficulty Stopping Drinking | 0 - 4 | 2 | .48 | .735 | .14 | .351 | $F(2, 98) = 39.673$ $p = .004$** |
| Negative Consequences of Alcohol Use | 0 – 84 | 33 | 12.18 | 9.629 | 7.12 | 7.164 | $F(2, 98) = 5.759$ $p = .018$* |
| Behavior Task Performance: Response Inhibition | | | | | | | |
| *p*-Inhibition Failures – Consistent Cue | 0 – 1 | .16 | .04 | .040 | .02 | .036 | $F(2, 98) = 1.449$ $p = .028$* |
| *p*-Inhibition Failures – Inconsistent Cues | 0 – 1 | .36 | .05 | .068 | .04 | .060 | $F(2, 98) = 0.038$ $p = .217$ |
| *p*-Inhibition Failures – Overall | 0 – 1 | .20 | .04 | .041 | .03 | .04 | $F(2,98) = 4.385$ $p = .039$* |
| Cue Dependency Score – Response Inhibition | 0 – 1 | .29 | .02 | .056 | .02 | .041 | $F(2, 98) = 0.948$ $p = .936$ |
| Behavior Task Performance: Response Execution | | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Mean Reaction Time - Consistent Cues | | 190.48 | 321.30 | 35.947 | 312.07 | 28.944 | $F(2, 98) = 1.786$ $p = .161$ |
| Mean Reaction Time - Inconsistent Cues | | 187.89 | 312.44 | 31.110 | 303.27 | 27.645 | $F(2, 98) = 0.495$ $p = .123$ |
| Mean Reaction Time – Overall | | 182.66 | 319.53 | 34.273 | 310.31 | 27.990 | $F(2, 98) = 2.169$ $p = .144$ |
| Cue Dependency Score - Response Execution | | 36.58 | 8.86 | 18.141 | 8.80 | 15.740 | $F(2, 98) = 1.786$ $p = .161$ |
| Zuckerman Disinhibition Subscale | 0 – 7 | 7 | 4.22 | .198 | 3.26 | 1.79 | $F(2, 98) = 1.667$ $p = .013$* |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. Group contrasts were tested by one-way between-subjects ANOVAs. * $p < .05$. ** $p < .01$.

Table 8

*Correlations among DSM-IV Axis I Diagnosis Significantly Associated with ADHD and Outcome Variables*

| | MDD Past | Panic Current | Social Phobia Current | Alcohol Quantity | Difficulty Stopping Drinking | Negative Cons of Alcohol Use | DIS-M |
|---|---|---|---|---|---|---|---|
| MDD Past | 1 | | | | | | |
| Panic Current | .005 | 1 | | | | | |
| Social Phobia Current | -.022 | .086 | 1 | | | | |
| Alcohol Quantity | -.023 | .064 | .166 | 1 | | | |
| Difficulty Stopping Drinking | .021 | .241* | .143 | .231** | 1 | | |
| Negative Cons of Alcohol Use | .165 | .090 | .103 | .645** | .492** | 1 | |
| DIS-M | -.029 | .113 | .035 | .434** | .330** | .393** | 1 |

*Note.* DSM-IV = Diagnostic and Statistical Manual, Fourth Revision. ADHD = Attention Deficit Hyperactivity Disorder. DIS-M = Zuckerman Sensation Seeking Scale – V Disinhibition Subscale Modified. MDD = Major Depressive Disorder.
* $p < .05$. ** $p < .01$.

Table 9

*Correlations among predictor variables included in preliminary analyses and outcome variables*

| Variable | Age | Gender | Race | Fraternity/ Sorority Member | WTAR | DIS-M | Cue Dependency RI | Drug Use Composite | Negative Consequences of Alcohol Use | Difficulty Stopping Drinking | Alcohol Quantity |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Age | 1.00 | .110 | .185 | .046 | -.032 | -.034 | .136 | -.012 | .033 | .049 | -.127 |
| Gender | | 1.00 | .179 | .101 | -.045 | -.167 | .024 | -.080 | -.047 | -.148 | -.264** |
| Race | | | 1.00 | .009 | .027 | -.085 | -.073 | -.079 | .109 | .095 | -.059 |
| Fraternity Sorority Membership | | | | 1.00 | .030 | .172 | -.008 | .109 | .352** | .234* | .192 |
| WTAR | | | | | 1.00 | -.051 | -.023 | .120 | -.015 | .076 | .030 |
| DIS-M | | | | | | 1.00 | .181 | .320** | .407** | .331** | .394** |
| Cue Dependency: RI | | | | | | | 1.00 | .038 | .115 | .096 | .082 |
| Drug Use Composite | | | | | | | | 1.00 | .367** | .331** | .439** |
| Negative Consequences of Alcohol Use | | | | | | | | | 1.00 | .482** | .552** |
| Difficulty Stopping a Drinking session | | | | | | | | | | 1.00 | .231* |
| Alcohol Quantity | | | | | | | | | | | 1.00 |

**Note:* WTAR = Weschler Test of Adult Reading. DIS-M = Zuckerman Sensation Seeking Scale – V Disinhibition Subscale Modified. RI = Response Inhibition. * $p < .05$. ** $p < .01$.

Table 10

*Between Group Differences (ADHD and Non-ADHD) AUDIT Quantity of Alcohol Use*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | .030 | 1 | .030 | .032 |
| Covariates | | | | |
| (adjusted for all effects) | | | | |
| Fraternity/Sorority | 3.065 | 1 | 3.065 | 3.286 |
| Panic Disorder | .093 | 1 | .093 | .099 |
| Drug Use | 19.789 | 1 | 19.789 | 21.560*** |
| Gender | 6.177 | 1 | 6.177 | 6.279* |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. AUDIT = Alcohol Use Disorders Identification Test. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 11

*Between Group Differences (ADHD and Non-ADHD) AUDIT Difficulty Stopping Drinking*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | 3.229 | 1 | 3.229 | 10.950** |
| Covariates | | | | |
| (adjusted for all effects) | | | | |
| Quantity | 2.045 | 1 | 2.045 | 7.226** |
| Fraternity/Sorority | .749 | 1 | .749 | 2.645 |
| Panic Disorder | 2.086 | 1 | 2.086 | 7.372** |
| Drug Use | 1.817 | 1 | 1.817 | 6.422* |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. AUDIT = Alcohol Use Disorders Identification Test. * $p < .05$. ** $p < .01$.

Table 12

*Between Group Differences (ADHD and Non-ADHD) on Negative Consequences of Alcohol Use*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | 287.529 | 1 | 287.529 | 5.544* |
| Covariates | | | | |
| (adjusted for all effects) | | | | |
| Quantity | 1469.385 | 1 | 1469.385 | 28.33*** |
| Fraternity/Sorority | 391.736 | 1 | 391.736 | 7.553** |
| Panic Disorder | 3.562 | 1 | 3.562 | .794 |
| Drug Use | 69.931 | 1 | 69.931 | 1.348 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 13

*Between Group Differences (ADHD and Non-ADHD) on Specific Negative Consequences of Alcohol Use*

| Source of Variance | Adjusted | df | MS | *F* |
|---|---|---|---|---|
| Had a Hangover | | | | |
| ADHD | 13.188 | 1 | 13.188 | 6.123** |
| Covariates | | | | |
| Quantity | 24.071 | 1 | 24.071 | 11.175** |
| Fraternity/Sorority | 10.419 | 1 | 10.419 | 4.837* |
| Drug Use | .147 | 1 | .147 | .068 |
| Performed Poorly on a Test or Important Project | | | | |
| ADHD | .370 | 1 | .370 | .595 |
| Covariates | | | | |
| Quantity | 3.523 | 13.523 | 3.523 | 5.669* |
| Fraternity/Sorority | 1.536 | 1 | 1.536 | 2.472 |
| Drug Use | 3.401 | 1 | 3.401 | 5.473* |
| Been in Trouble with Police, Residence Hall Staff, or other College Authorities | | | | |
| ADHD | .014 | 1 | .014 | .135 |
| Covariates | | | | |
| Quantity | .090 | 1 | .090 | .855 |
| Fraternity/Sorority | .094 | 1 | .094 | .896 |
| Drug Use | .001 | 1 | .001 | .006 |
| Damaged Property, Pulled Fire | | | | |
| ADHD | .032 | 1 | .032 | .192 |
| Covariates | | | | |
| Quantity | .452 | 1 | .452 | 2.692 |
| Fraternity/Sorority | .512 | 1 | .512 | 3.050 |
| Drug Use | 1.376 | 1 | 1.376 | 8.189** |
| Got Into an Argument or Fight | | | | |
| ADHD | 5.196 | 1 | 5.196 | 4.582* |
| Covariates | | | | |
| Quantity | 11.939 | 1 | 11.939 | 10.528** |
| Fraternity/Sorority | .021 | 1 | .021 | .019 |
| Drug Use | .065 | 1 | .065 | .057 |

| | | | | |
|---|---|---|---|---|
| Had Memory Loss | | | | |
| ADHD | 2.039 | 1 | 2.039 | 1.296 |
| Covariates | | | | |
| Quantity | 37.761 | 1 | 37.761 | 23.997*** |
| Fraternity/Sorority | 34.327 | 1 | 34.327 | 21.814*** |
| Drug Use | .028 | 1 | .028 | .018 |
| Did Something I Later Regretted | | | | |
| ADHD | 5.834 | 1 | 5.834 | 5.601* |
| Covariates | | | | |
| Quantity | 7.043 | 1 | 7.043 | 6.761* |
| Fraternity/Sorority | 10.814 | 1 | 10.814 | 10.382** |
| Drug Use | .569 | 1 | .569 | .546 |
| Been Arrested for a | | | | |
| ADHD | .013 | 1 | .013 | 1.271 |
| Covariates | | | | |
| Quantity | .012 | 1 | .012 | 1.155 |
| Fraternity/Sorority | .001 | 1 | .001 | .097 |
| Drug Use | .000 | 1 | .000 | .027 |
| Have Been Taken Advantage of Sexually | | | | |
| ADHD | .065 | 1 | .065 | .808 |
| Covariates | | | | |
| Quantity | .113 | 1 | .113 | 1.395 |
| Fraternity/Sorority | .065 | 1 | .065 | .803 |
| Drug Use | .349 | 1 | .349 | 4.315* |
| Been Hurt or Injured | | | | |
| ADHD | .012 | 1 | .012 | .031 |
| Covariates | | | | |
| Quantity | 3.186 | 1 | 3.186 | 8.555** |
| Fraternity/Sorority | .046 | 1 | .046 | .125 |
| Drug Use | .039 | 1 | .309 | .104 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. ** $p < .01$. *** $p < .001$.*** $p <$

Table 14

*Correlations Among Negative Consequences of Alcohol Use within the ADHD Group*

| Variable | Had a Hangover | Got Into Fight | Been Nauseated / Vomited | Been Criticized by Someone I Know | Thought I Might Have a Drinking Problem | Did something I later Regretted |
|---|---|---|---|---|---|---|
| Had a Hangover | 1 | | | | | |
| Got Into Argument/ Fight | .396** | 1 | | | | |
| Been Nauseated / Vomited | .616** | .428** | 1 | | | |
| Been Criticized by Someone I Know | .442** | .488** | .453** | 1 | | |
| Thought I Might Have a Drinking Problem | .192 | .209 | -.127 | .415** | 1 | |
| Did something I later Regretted | .303* | .504** | .516** | .467** | .166 | 1 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. * $p < .05$. ** $p < .01$.

Table 15

*Correlations Among Negative Consequences of Alcohol Use within the Non-ADHD Group*

| Variable | Had a Hangover | Got Into Fight | Been Nauseated / Vomited | Been Criticized by Someone I Know | Thought I Might Have a Drinking Problem | Did something I later Regretted |
|---|---|---|---|---|---|---|
| Had a Hangover | 1 | | | | | |
| Got Into Argument/ Fight | .375** | 1 | | | | |
| Been Nauseated / Vomited | .423** | .471** | 1 | | | |
| Been Criticized by Someone I Know | .383** | .113 | .374** | 1 | | |
| Thought I Might Have a Drinking Problem | N/A | N/A | N/A | N/A | N/A | |
| Did something I later Regretted | .550** | .501** | .471** | .402** | N/A | 1 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. N/A = not applicable. Correlation could not be computed because value is constant (0). Item was not endorsed by any participants in the non-ADHD group. ** $p < .01$.

Table 16

*2(Group) x 2(Cue Condition) ANOVA of p-inhibition Failures*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | .013 | 1, 97 | .013 | 3.090 |
| Cue Condition | .014 | 1, 97 | .014 | 11.575** |
| Cue Condition x ADHD | 8.000 | 1, 97 | .001 | .007 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. * $p < .05$. ** $p < .01$.

Table 17

*2(Group) x 2(Cue Condition) ANOVA of Response Execution Reaction Times*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | 5347.548 | 1, 97 | 5347.548 | 2.922 |
| Cue Condition | 4429.154 | 1, 97 | 4429.154 | 28.128*** |
| Cue Condition x ADHD | 12.807 | 1, 97 | 12.807 | .081 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. *** $p < .01$.

Table 18

*Correlations between ADHD Medication (Current) and Cued Go/No-Go Task Performance*

| Variable | ADHD Meds Current | *p*-Inhibition Failures – Consistent Cues | *p*-Inhibition Failures – Inconsistent Cues | *p*-Inhibition Failures – Overall | Cue Dependency: Response Inhibition | Mean RT Overall |
|---|---|---|---|---|---|---|
| ADHD Medication Current | 1.0 | | | | | |
| *p*-Inhibition Failures – Consistent Cues | -.042 | 1.0 | | | | |
| *p*-Inhibition Failures – Inconsistent Cues | .144 | .581** | 1.0 | | | |
| *p*-Inhibition Failures – Overall | .006 | .962** | .782** | 1.0 | | |
| Cue Dependency: Response Inhibition | .169 | .014 | .822** | .287* | 1.0 | |
| Mean RT Overall | .009 | -.218 | -.307* | -.217 | -.267 | 1.0 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. ** $p < .01$. *** $p < .001$.*** $p < .001$

Table 19

*Linear Regression Equations Evaluating Difficulty Stopping Drinking as a Mediator of the Relationship between ADHD and Negative Consequences of Alcohol Use*

| Step and Variable | df | $R^2$ | $R^2\Delta$ | F | SE | β |
|---|---|---|---|---|---|---|
| ADHD → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 4, 95 | .386 | .386 | 14.948 | | |
| Quantity | | | | | .736 | .440*** |
| Fraternity Sorority | | | | | 1.731 | .258** |
| Drug Use | | | | | .154 | .153 |
| Panic Disorder | | | | | 2.335 | -.039 |
| Step 2 | 5, 94 | .436 | .049 | 14.513 | | |
| ADHD | | | | | 1.384 | .226** |
| ADHD → Difficulty Stopping Drinking | | | | | | |
| Step 1 | 4, 95 | .159 | .159 | 4.507 | | |
| Quantity | | | | | .058 | .258* |
| Fraternity | | | | | .137 | .167 |
| Drug Use | | | | | .012 | -.209 |
| Panic Disorder | | | | | .185 | .217* |
| Step 2 | 5, 94 | .256 | .096 | 6.465 | | |
| ADHD | | | | | .108 | .316** |
| Difficulty Stopping Drinking → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 4, 95 | .386 | .386 | 14.948 | | |
| Quantity | | | | | .736 | .440*** |
| Fraternity | | | | | .1731 | .258** |
| Drug Use | | | | | .154 | .153 |
| Panic Disorder | | | | | 2.335 | -.039 |
| Step 2 | 5, 94 | .520 | .134 | 20.366 | | |
| Difficulty Stopping Drinking | | | | | 1.150 | .399*** |

| | | | | | | |
|---|---|---|---|---|---|---|
| ADHD → Difficulty Stopping Drinking → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 4, 95 | .386 | .386 | 14.948 | | |
| Quantity | | | | | .736 | |
| Fraternity | | | | | | |
| Drug Use | | | | | | |
| Panic Disorder | | | | | | |
| Step 2 | 6, 93 | .531 | .145 | 17.543 | | |
| Difficulty Stopping Drinking | | | | | 1.717 | .328*** |
| ADHD | | | | | 1.323 | .134 |

*Note*: ADHD=Attention-Deficit/Hyperactivity Disorder, CD=Conduct Disorder.
* $p$<.05 **. $p$ < .01 *** $p$ < .001.

Table 20

*Cue Dependency as a Mediator of the Relationship between ADHD and Negative Consequences of Alcohol Use*

| Step and Variable | df | $R^2$ | $R^2\Delta$ | F | SE | B |
|---|---|---|---|---|---|---|
| ADHD → Negative Consequences | | | | | | |
| Step 1 | 2, 97 | .234 | .234 | 14.789 | | |
| Fraternity/Sorority | | | | | 1.864 | .316** |
| Drug Use | | | | | .152 | .333*** |
| Step 2 | 3, 96 | .292 | .058 | 13.173 | | |
| ADHD | | | | | 1.520 | .243* |
| ADHD → Cue Dependency | | | | | | |
| ADHD | 1, 98 | .000 | .000 | .007 | .010 | -.008 |
| Cue Dependency → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 2, 97 | .234 | .234 | 14.789 | | |
| Fraternity Sorority | | | | | 1.864 | .316** |
| Drug Use | | | | | .152 | .333*** |
| Step 2 | 3, 96 | .245 | .011 | 10.363 | | |
| Cue Dependency | | | | | 15.934 | .105 |
| ADHD → Cue Dependency → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 2, 97 | .234 | .234 | 14.789 | | |
| Fraternity Sorority | | | | | 1.864 | .316** |
| Drug Use | | | | | .152 | .333*** |
| Step 2 | 4, 95 | .303 | .070 | 10.336 | | |
| Cue Dependency | | | | | 15.385 | .108 |
| ADHD | | | | | 1.515 | .244** |

*Note*: ADHD=Attention-Deficit/Hyperactivity Disorder, CD=Conduct Disorder.
* $p$<.05. ** $p$ < .01. *** $p$ < .001.

Table 21

*Differences between ADHD and Non-ADHD Groups on Zuckerman Disinhibition Subscale (modified)*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | 18.490 | 1 | 18.490 | 5.411* |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. * $p < .05$.

Table 22

*Linear Regression Equations Evaluating Self-Reported Behavioral Disinhibition as a Mediator of the Relationship between ADHD and Negative Consequences of Alcohol Use*

| Step and Variable | df | $R^2$ | $R^2\Delta$ | F | SE | B |
|---|---|---|---|---|---|---|
| ADHD → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 3, 96 | .385 | .385 | 20.022 | | |
| Quantity | | | | | .732 | .439*** |
| Fraternity Sorority | | | | | 1.701 | .252** |
| Drug Use | | | | | .152 | .147 |
| Step 2 | 4, 95 | .436 | .051 | 18.329 | | |
| ADHD | | | | | 1.365 | .227** |
| ADHD → SSS-Disinhibition | | | | | | |
| Step 1 | 3, 96 | .191 | .191 | 7.541 | | |
| Quantity | | | | | .185 | .297** |
| Fraternity Sorority | | | | | .430 | .096 |
| Drug Use | | | | | .038 | .179 |
| Step 2 | 4, 95 | .230 | .039 | 7.089 | | |
| ADHD | | | | | .351 | .200* |
| SSS-Disinhibition → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 3, 96 | .385 | .385 | 20.022 | | |
| Quantity | | | | | .732 | .439*** |
| Fraternity Sorority | | | | | 1.701 | .252** |
| Drug Use | | | | | .152 | .147 |
| Step 2 | 4, 95 | .410 | .026 | 16.531 | | |
| SSS-Disinhibition | | | | | .398 | .178* |
| ADHD → Disinhibition → Negative Consequences of Alcohol Use | | | | | | |
| Step 1 | 3, 96 | .385 | .385 | 20.022 | | |
| Quantity | | | | | .732 | .439*** |
| Fraternity Sorority | | | | | 1.701 | .252** |
| Drug Use | | | | | .152 | .147 |
| Step 2 | 5, 94 | .448 | .063 | 15.279 | | |
| SSS-Disinhibition | | | | | .396 | .144 |

| | | |
|---|---|---|
| ADHD | 1.390 | .202* |

*Note*: ADHD = Attention-Deficit/Hyperactivity Disorder. SSS-Disinhibition = Self-reported behavioral disinhibition as measured by the modified version of the disinhibition subscale of the Zuckerman Sensation Seeking Scale – V. * $p < .05$. ** $p < .01$. ** $p < .001$.

Table 23

*Between Group Differences (Measure Completion Method: Paper and Pencil vs. Verbal) PDHQ*

| Variable | Paper and Pencil *n = 18* | | Oral *n =62* | | Group Contrasts |
|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | |
| Frequency of Alcohol Consumption per Month | 6.5278 | 4.075 | 5.669 | 4.144 | $t(78) = .777$, $p = .440$ |
| Alcohol Quantity | 4.889 | 2.948 | 4.723 | 2.949 | $t(78) = .207$, $p = .837$ |

*Note.* PDHQ = Personal Drinking History Questionnaire.

Table 24

*Between Group Differences (ADHD and Non-ADHD) PDHQ Alcohol Quantity*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | 3.507 | 1 | 3.507 | .536 |
| Covariates (adjusted for all effects) | | | | |
| Fraternity Sorority | 3.271 | 1 | 3.271 | .500 |
| Drug Use | 85.958 | 1 | 85.958 | 13.134** |
| Panic Disorder | 1.465 | 1 | 1.465 | .224 |
| Gender | 68.316 | 1 | 68.316 | 10.438** |
| Administration Format | 2.505 | 1 | 2.505 | .383 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. PDHQ = Personal Drinking History Questionnaire. $p < .05$. ** $p < .01$. *** $p < .001$.

Table 25

*Between Group Differences (ADHD and Non-ADHD) PDHQ Alcohol Frequency*

| Source of Variance | Adjusted SS | df | MS | *F* |
|---|---|---|---|---|
| ADHD | 36.283 | 1 | 36.283 | 2.301 |
| Covariates | | | | |
| (adjusted for all effects) | | | | |
| Fraternity Sorority | 19.548 | 1 | 19.548 | 1.240 |
| Drug Use | 95.049 | 1 | 95.049 | 6.028* |
| Panic Disorder | .809 | 1 | .809 | .051 |
| Gender | 5.036 | 1 | 5.036 | .319 |
| Administration Format | 2.750 | 1 | 2.750 | .174 |

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. PDHQ = Personal Drinking History Questionnaire. $p < .05$. ** $p < .01$. *** $p < .001$.

# APPENDIX A

# CONCEPTUAL MODEL

## Theoretical Basis

Despite a substantial literature identifying ADHD as a risk factor for the development of alcohol-related problems and evidence indicating that the college years are a particularly salient time for engagement in hazardous patterns of alcohol use, only a handful of studies have examined the association between ADHD and alcohol use in college students. The majority of information about problematic alcohol use in ADHD comes from several longitudinal studies examining adolescent and young adult outcomes of children diagnosed with the disorder. Findings from these studies show that adolescents with ADHD typically begin using alcohol at an earlier age, use alcohol with greater frequency, and engage in more hazardous patterns of use (e.g., have 5 or more drinks in a sitting) relative to their peers without ADHD (Molina & Pelham, 2003; Langley et al, 2010). Young adults with ADHD do not appear to use alcohol at higher rates than their peers without ADHD (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman et al., 1997), but are significantly more likely to abuse alcohol or have an alcohol use disorder (AUD; Smith, Molina, & Pelham, 2003; Weiss & Hechtman, 1993). Preliminary studies of college students with ADHD reveal patterns of use similar to those identified through longitudinal research. Blase and colleagues (2009) found that students with a self-reported ADHD diagnosis and elevated ADHD symptoms reported consuming alcohol with greater frequency than students without ADHD, but not in greater quantities. In a study conducted in our laboratory, which employed a diagnostic assessment utilizing Diagnostic

and Statistical Manual of Mental Disorders, Version 4, Text Revision criteria (DSM-IV-TR; APA, 2000) to determine group classification, ADHD was associated with higher rates of problematic alcohol using among college students. Specifically, ADHD was associated with: higher scores on a global measure of alcohol related impairment - the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuenta, & Grant, 1993), higher rates of difficulty stopping a drinking session, and higher rates of negative consequences of alcohol use (i.e., having a hangover, getting into an argument or fight, getting nauseated or having vomited, being criticized by someone due to drinking or drug use, experiencing memory loss, doing something they later regretted, being hurt or injured; citation omitted to maintain anonymity). Consistent with studies of young adults with ADHD (Weiss & Hechtman, 1993; Wilens et al., 1998; Wilens et al., 2002), ADHD was not associated with higher rates of alcohol quantity or frequency in our sample of college students. This suggests that college students with ADHD may be experiencing greater impairment as a result of their alcohol use relative to their non-ADHD peers despite equivalent rates of use.

In seeking to understand why college students with ADHD are experiencing higher rates of alcohol related problems, an examination of risk factors common to both ADHD and alcohol related problems provides a viable starting point. Behavioral disinhibition (also referred to as impulsivity or behavioral under control; for a review see Dick et al., 2010) represents one such factor. Barkley's (1997) comprehensive theory of ADHD proposes that deficient

inhibitory control, or behavioral disinhibition, is the core feature of ADHD, which ultimately results in the characteristic behavioral and academic impairments seen in ADHD. Over the past decade, a substantial body of literature has supported Barkley's theoretical model and has made behavioral disinhibition the most studied of the executive deficits known to be associated with ADHD (Homack & Riccio, 2004; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Romine et al., 2004; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Behavioral disinhibition has also consistently been associated with problematic alcohol use. Prospective studies have identified behavioral disinhibition during childhood and adolescence as a risk factor for the development of alcohol use disorders (Iacano, Carlson, Taylor, Elkins, & McGue, 1999; King et al., 2009; Nigg et al., 2006; Sher, Bartholow, & Wood, 2000). In cross sectional studies, individuals meeting criteria for alcohol use disorder score high on both self-report and laboratory measures of disinhibition (Hildebrant, Brokate, Eling, & Lanz, 2004; Sher, Grekin, & Williams, 2005; Trull, Waudby, & Sher, 2004; Van der Linden et al., 2007). In addition, alcohol consumption acutely increases disinhibition in healthy adults (for a review see Fillmore, 2003). Despite the substantial overlap among ADHD, disinhibition, and problematic alcohol use, only one study has examined the interaction among these three constructs. Weafer and Fillmore (2009) studied behavioral task performance on a measure of disinhibition in young adults with and without ADHD while under the influence of alcohol. Results indicate that alcohol differentially affected task performance, with the ADHD group showing significantly greater impairment in behavioral disinhibition than the non-ADHD

group as alcohol dose increased. Thus, when under the influence of alcohol young adults with ADHD may become more disinhibited than their peers without the disorder when consuming equivalent amounts of alcohol.

Theories emphasizing trait-based vulnerabilities may be particularly relevant during late adolescence and young adulthood as this is the time when personality traits are relatively unstable and undergo a significant amount of change (Roberts, Walton, & Viechtbauer, 2006). With regard to alcohol use, changes in the personality traits of impulsivity and neuroticism are associated with the normative decline in alcohol use that occurs during the mid- to late-twenties (Littlefield, Sher, & Wood, 2009). Specifically, as impulsivity and neuroticism decreased over time, problematic alcohol use decreased as well when controlling for other factors known to be associated with this normative decline in use (i.e., marriage, parenthood). In addition, promising results obtained through the delivery of personality-targeted interventions to reduce alcohol-related problems among adolescents (O'Leary, Mackie, Castellanos-Ryan, Al-Khudhairy, & Conrod, 2010; Sloboda et al., 2009) suggest that there is clinical utility in clarifying the role of personality traits in problematic alcohol use.

In summary, alcohol-related problems have consistently been linked independently to both ADHD and disinhibition. In seeking to examine mechanisms that explain the association between ADHD and problematic alcohol use among college students, disinhibition represents a logical starting point given that changes in this trait have been associated with the normative decline in alcohol use that follows the college years and the fact that personality traits are

relatively unstable during this developmental time point, and therefore may be amenable to change. Personality traits, such as disinhibition, can be conceptualized as distal factors that operate through more proximal behaviors (Costa & McCrae, 1994; Hogan & Roberts, 2000). Drawing upon findings from our previous study, which showed that ADHD was associated with higher rates of difficulty stopping a drinking session, we hypothesized that difficulty stopping a drinking session would be a proximal behavior associated with trait disinhibition and behavioral disinhibition as evidenced by it's high correlation with these constructs. In addition, we hypothesized that difficulty stopping a drinking session would mediate the relationship between ADHD and negative consequences of alcohol use, while controlling for quantity of alcohol consumed. We also examined trait disinhibition and behavioral disinhibition as mediators of the relationship between ADHD and the negative consequences of alcohol use, while controlling for alcohol quantity. With these hypotheses we surmise that disinhibition may be the construct that underlies both the experience of feeling unable to control one's drinking as well as decreased of self-control in social interactions, which may explain specific negative consequences being endorsed with greater frequency by the ADHD group, such as: being criticized by a peer, getting into an argument or fight, doing something they later regretted, and ultimately becoming concerned that they may have a drinking problem.

## Measures Overview

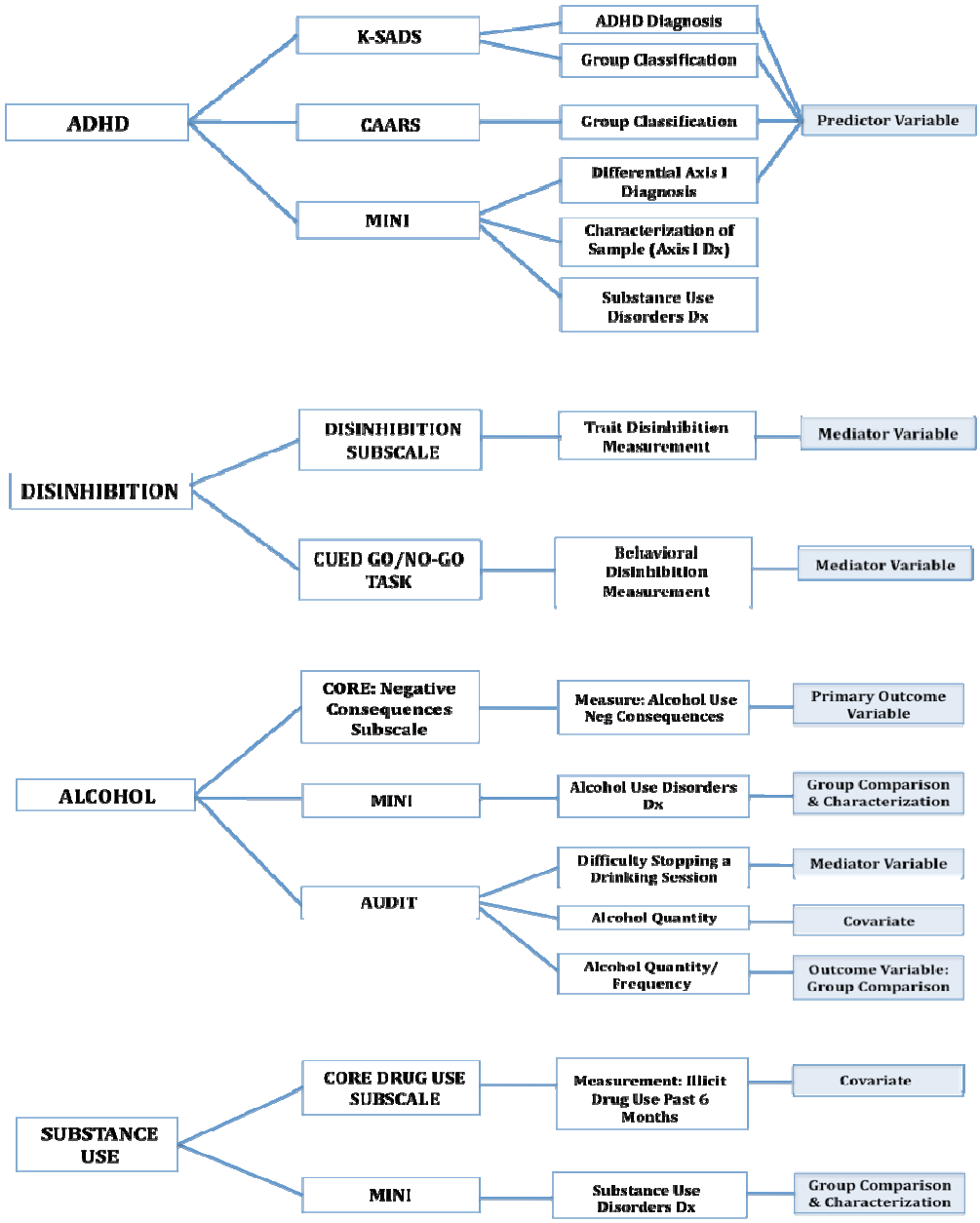



Figure 3. Measures Overview.

*Note.* ADHD = Attention Deficit Hyperactivity Disorder. K-SADS = Schedule for Affective Disorders and Schizophrenia for school age children. CAARS = Conners Adult ADHD Scale. MINI = M.I.N.I. International Neuropsychiatric Interview. AUDIT = Alcohol Use Disorders Identification Test. CORE = CORE Alcohol and Drug Use Survey. Dx = Diagnosis.

## APPENDIX B

EXPLORATORY ANALYSES

*Cued Go/No-Go Task Performance*

In our main analyses, ADHD was not associated with cue dependency on trials of response inhibition or with mean reaction time on trials of response execution. As outlined in the discussion section, there are many possible reasons for the absence of these associations. One possible cause is current use of stimulant medication by members of the ADHD group. Studies have shown that behavioral tasks similar to the cued go/no-go task are sensitive to the effects of stimulant medication (Tannock, et al., 1989; Tannock, et al., 1995). Therefore, we required participants to withhold their medication on the day of the study and obtained verbal confirmation from each participant that they had adhered to this requirement. It is possible, however, that some participants had indeed taken stimulant medication despite their statements to the contrary or that our wash-out period of 24 hours was too short to guarantee that the medication would not be in effect while participants were completing the behavioral task. Forty-six percent (n=23) of the ADHD group reported that they currently take ADHD stimulant medication regularly. Therefore, we explored whether self-reported current ADHD medication may have influenced results despite the fact that medication was reportedly withheld on the day of the study.

We conducted correlational analyses between ADHD current medication status within the ADHD group and each of the cued go/no-go task variables. Results show that there are no significant correlations between current ADHD

medication and performance on any aspect of the cued go/no-go task (i.e., *p*-inhibition failures consistent cues, *p*-inhibition failures inconsistent cues, *p*-inhibition failures across cue conditions, cue dependency on trials of response inhibition, and mean overall reaction time). Results are presented in Table 18. The absence of an association between ADHD stimulant medication status and performance on the cued go/no-go task may indicate that participants withheld their medication on the day of the study as was required and that the washout period of 24-hours was sufficient to prevent stimulant medication from affecting study performance. It is also possible, however, that this particular behavioral task was not sensitive enough to detect the performance-enhancing effects of stimulant medication. Future studies that include larger sample sizes, behavioral tasks of varying degrees of difficulty, and a group of participants taking active stimulant medication while completing the behavioral task are needed to better understand these findings.

*PDHQ*

Consistent with studies conducted by research groups examining the effects of alcohol on cued go/no-go task performance (Fillmore, 2004; Marczinski, Abroms, et al., 2005; Weafer & Fillmore, 2008), it was originally proposed that the variables of alcohol quantity and frequency would be pulled obtained from the PDHQ. Problems with measure administration however, resulted in a significant amount of missing data. Specifically, 13 participants (26%) in the ADHD group and 8 participants in the Non-ADHD group (16%) had unusable PDHQ data. In an effort to correct administration problems, the format

of measure completion was changed from self-report to interview. In post-hoc analyses it was found that the means of outcome variables did not differ as a function of administration method (see Table 23). Administration method was however, included as a covariate in all PDHQ analyses.

Exploratory analyses were conducted with the PDHQ to examine group differences (ADHD vs. Non-ADHD) on the variables of alcohol quantity and alcohol frequency. Between group differences in self-reported alcohol quantity were not statistically significant ($F$ (6, 72) = 2.301, $p$ =.134). Results are presented in Table 24. This finding is consistent with participants self-reported alcohol quantity on the AUDIT in the current study (see Table 10). In addition, PDHQ Quantity was significantly correlated with AUDIT Quantity ($r$ = .637, $p$ < .01). Between-group differences in self-reported frequency of alcohol use were also not significant ($F$ (6, 72) = .536, $p$ = .446). Results are presented in Table 25. PDHQ Frequency and AUDIT Frequency were also significantly correlated ($r$ = .738, $p$ < .01). Both the quantity and frequency findings are consistent with results from our prior study (Rooney et al., 2011) and with studies of young adults with ADHD (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman et al., 1997).

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