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EFFECTS OF MULTISENSORY STOP SIGNALS ON SENSITIVITY TO ALCOHOL-INDUCED DISINHIBITION IN DRINKERS WITH ADHD

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EFFECTS OF MULTISENSORY STOP SIGNALS ON SENSITIVITY TO
ALCOHOL-INDUCED DISINHIBITION IN DRINKERS WITH ADHD

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THESIS

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A thesis submitted in partial fulfillment of the
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By

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ABSTRACT OF THESIS

EFFECTS OF MULTISENSORY STOP SIGNALS ON SENSITIVITY TO
ALCOHOL-INDUCED DISINHIBITION IN DRINKERS WITH ADHD

        Multisensory environments facilitate behavioral functioning in humans. The redundant signal effect (RSE) refers to the observation that individuals respond more quickly to stimuli when information is presented as multisensory, redundant stimuli rather than as a single stimulus presented to either modality alone. Our studies show that the disinhibiting effects of alcohol are attenuated when stop signals are multisensory versus unisensory. The present study expanded on this research to test the degree to which multisensory stop signals could also attenuate the disinhibiting effects of alcohol in those with attention-deficit hyperactivity disorder (ADHD), a clinical population characterized by poor impulse control. The study compared young adults with ADHD with healthy controls and examined the acute impairing effect of alcohol on response inhibition to stop signals that were presented as a unisensory stimulus or a multisensory stimulus. For controls, results showed alcohol impaired response inhibition to unisensory stop signals but not to multisensory stop signals. Response inhibition of those with ADHD was impaired by alcohol regardless of whether stop signals were unisensory or multisensory. The failure of multisensory stimuli to attenuate alcohol impairment in those with ADHD highlights a specific vulnerability that could account for heightened sensitivity to the disruptive effects of alcohol.

KEYWORDS: Disinhibition, Alcohol Impairment, Attention-Deficit Hyperactivity
                Disorder, Alcohol, Multisensory

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March 25, 2019

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Introduction

Overview

Alcohol is well known for its disinhibiting effects on behavior. Slower reaction time on laboratory tasks due to disrupted cognitive processes and a weakened ability to inhibit behaviorally inappropriate actions (i.e., impulsivity) are two well-known acute effects of alcohol (Fillmore & Weafer, 2012; Hendershot et al., 2015). However, multisensory environments have been shown to facilitate behavioral functioning in humans. The "redundant signal effect" (RSE) refers to the well-documented observation that individuals respond more quickly to stimuli when the information is presented as multisensory redundant stimuli (e.g., aurally and visually), rather than as a single stimulus presented to either modality alone. RSE appears to be due to specialized multisensory neurons in the superior colliculus and association cortex that allow for intersensory co-activation between the visual and auditory channels. Previous research of response inhibition has shown that disinhibiting effects of alcohol are attenuated when drinkers respond to no-go stimuli that are presented as redundant multisensory signals (e.g., Visual + Auditory no-go signals) versus a single signal to just one modality alone (e.g., Visual no-go signal) (Roberts, Monem, & Fillmore, 2016). However, it is not known if this attenuation of disinhibition occurs in clinical populations, specifically individuals with ADHD. Individuals with ADHD are known for increased risk for alcohol use and heightened disinhibition in response to alcohol (Charach, Yeung, Climans, & Lillie, 2011; Lee, Humphreys, Flory, Liu, & Glass, 2011; Weafer, Fillmore, & Milich, 2009). My thesis compares young adults with ADHD to healthy controls to examine their responses to the acute disinhibiting effects of alcohol versus a placebo.

Inhibition failures, manual reaction time, and saccadic reaction time were tested in response to two different stimulus conditions: a single (visual) no-go stimulus and a multisensory (Visual + Auditory) no-go stimulus. The next sections review background on the construct of impulsivity and its role in alcohol abuse with specific focus on poor inhibitory control as a factor underlying impulsivity.

Impulsivity and Inhibitory Control

  Impulsivity is a pattern of uncontrolled behavior in which an individual lacks the ability to delay gratification and acts without forethought or consideration of potential consequences. As a personality trait and central characteristic of psychopathology, impulsivity is associated with risk of drug abuse (Bjork, Hommer, Grant, & Danube, 2004; Dom, De Wilde, Hulstijn, Van Den Brink, & Sabbe, 2006; Soloff, Lynch, & Moss, 2000*)*. Substance use disorders have high comorbidity with personality disorders such as antisocial, borderline, and histrionic disorder, all of which are characterized by under-controlled, impulsive patterns of behavior (Grekin, Sher, & Wood, 2006; Trull, Waudby, & Sher, 2004). Impulsive individuals tend to drink more frequently, in larger amounts, and are more likely to binge drink (Goudriaan, Grekin, & Sher, 2007; Marczinski, Combs, & Fillmore, 2007). Abusers of illicit drugs and individuals diagnosed with alcoholism tend to score higher on measures of impulsivity, disinhibition, and related traits, such as sensation-seeking (Bergman & Brismar, 1994; Sher, Trull, Bartholow, & Vieth, 1999; Trull et al., 2004). Additionally, impulsive characteristics often precede the onset of problem alcohol use suggesting that trait impulsivity might also play a causal role in alcohol abuse. Longitudinal studies have shown that impulsivity predicts early onset drinking age and the development of heavy drinking and alcohol dependence in

young adults (August et al., 2006; Ernst et al., 2006). This predictive relationship between impulsivity and early onset drinking, heavy drinking, and alcohol dependence provides insight to the involvement of impulsivity and maladaptive drinking behaviors. Heritability studies of substance use disorders also point to the involvement of impulsivity. Studies of individuals with a familial risk for substance use disorder, such as children of alcoholics, find that these individuals also display increased impulsivity (Alterman et al., 1998; Sher, 1991).

Impulsivity is multifaceted. A key component of impulsivity, that is a focus of this thesis, is response inhibition. Poor response inhibition is observed as a failure to suppress prepotent action in the moment, which can result in impulsive action. Studies in neuropharmacology and neuroanatomy have identified distinct neural systems that implicate separate inhibitory and activational mechanisms in the control of behavior (Jentsch & Taylor, 1999; Lyvers, 2000). The mechanism of inhibitory control is thought to involve frontal lobe substrates that exert inhibitory influences over conditioned responses and reflexive behaviors. Physiologically, brain regions implicated in inhibitory control include the anterior cingulate, dorsolateral prefrontal cortex, insula, and parietal regions (Botvinick, Cohen, & Carter, 2004; Seeley et al., 2007). The ability to inhibit or suppress an action enhances an organism's behavioral repertoire by affording some control over when and where responses may be expressed. As such, the inhibition of behavior is an important function that sets the occasion for many other activities that require self-restraint and regulation of behavior. Not surprisingly, deficient or impaired inhibitory control has been implicated in the display of impulsivity and disorders of self-control, such as antisocial personality, obsessive–compulsive, and ADHD (Barkley,

2006; Nigg, 2006).

A number of behavioral tasks have been used to assess deficits of response inhibition. Stop-signal and cued go/no-go models evaluate control as the ability to activate and to inhibit prepotent (i.e., instigated) responses (Logan, 1994; Miller, Schäffer, & Hackley, 1991). The tasks model behavioral control using a reaction time scenario that measures the countervailing influences of inhibitory and activational mechanisms. Individuals are required to quickly activate a response to a go-signal and to inhibit a response when a stop-signal occasionally occurs. Activation is typically measured as the speed of responding to go-signals and inhibition to stop-signals is assessed by the probability of suppressing the response or by the time needed to suppress the response. In these models, inhibition of a response is usually required in a context in which there is a strong tendency to respond to a stimulus (i.e., a prepotency), thus making inhibition difficult. The validity of these models is well documented; however, the models are sensitive to inhibitory deficits characteristic of brain injury (Malloy, Bihrle, Duffy, & Cimino, 1993), trait-based impulsivity (Logan, Schachar, & Tannock, 1997), and self-control disorders, such as ADHD (Tannock, 1998).

Inhibitory Control and Effects of Alcohol

Inhibitory control is vulnerable to the acute disruptive effects of alcohol. Laboratory studies of young adults have shown how acute doses of alcohol impair inhibitory control as measured by stop-signal and cued go/no-go models in a laboratory setting. Laboratory studies find that alcohol reliably increases failures to inhibit responses to stop-signals in a dose-dependent manner (Fillmore, 2003; Marczinski & Fillmore, 2003). Previous research has demonstrated that, in healthy men and women, impairment

of inhibitory control is a linear function of dose, and significant impairment has been

shown at blood alcohol concentrations (BACs) as low as 50 mg/100 mL, a BAC

produced by as few as two drinks (Marczinski & Fillmore, 2003). The ability to inhibit an

action also appears more vulnerable to the disruptive effects of alcohol than the ability to

execute that action. Indeed, studies using the cued go/no-go task find that subjects display

increased failures to inhibit responses to stop-signals at BACs that do not impair their

ability to execute those responses to go-signals (Abroms, Fillmore, & Marczinski, 2003;

Fillmore, Marczinski, & Bowman, 2005). In sum, the vulnerability of inhibitory control

to the disruptive effects of alcohol has been corroborated across several laboratory studies

in recent years.

Alcohol Impairment of Inhibitory Control and Drinking Behavior

Acute alcohol impairment of inhibitory control can also directly contribute to

abuse potential by increasing the amount of alcohol people drink in one occasion. Weafer

and Fillmore (2008) measured individual differences in the degree to which subjects'

response inhibition was impaired by 0.65 g/kg alcohol versus a placebo using the cued

go/no-go task. During a follow up session, subjects' ad lib alcohol consumption was

measured. Participants were asked to complete an ostensible beer taste-rating task, with

the knowledge that they can drink as much or as little of the beer as they liked, and the

amount of beer consumed was recorded. The results showed that individual differences in

the degree to which alcohol impaired their inhibitory control during the previous session

significantly predicted individual differences in their alcohol consumption. Specifically,

those who were more disinhibited in response to alcohol on the cued go/no-go task

consumed greater amounts of alcohol when given ad lib access. Additional analyses also

examined the degree to which baseline levels of disinhibition (i.e., those observed in response to placebo), trait impulsivity, and alcohol impairment of response activation predicted ad lib consumption. Results showed that none of these factors were significantly associated with consumption. Thus, the amount of beer consumed was predicted specifically by the degree to which an individual's inhibitory control was impaired by alcohol.

Binge drinkers experience significantly heightened disinhibiting effects in response to alcohol; therefore, binge drinkers as a group display greater sensitivity to the disinhibiting effects of alcohol and alcohol-induced arousal compared to non-binge drinkers (Fillmore & Weafer, 2011; Weafer & Fillmore, 2008). Marczinski et al. (2007) compared sensitivity to alcohol impairment of inhibitory control in a group of binge drinkers and non-binge drinkers. Both groups performed the cued go/no-go task in response to placebo and an active dose of alcohol (0.65 g/kg). Task performance analyses demonstrated that the two groups did not differ in degree of inhibitory control under placebo. However, in response to the alcohol dose, binge drinkers committed significantly more inhibitory failures than did non-binge drinkers. Additionally, participants rated their degree of subjective stimulation in response to both doses. The groups did not differ in the level of arousal in response to placebo; however, binge drinkers reported significantly greater stimulation in response to alcohol than did non-binge drinkers. Thus, binge drinkers displayed a heightened sensitivity to both the disinhibiting effects of alcohol and to alcohol-induced arousal. This is evidence that acute impairment of inhibitory control could be a mechanism in alcohol's abuse potential, leading to binge drinking.

Task-related Factors that Affect Impairment

A common explanation for why alcohol impairs cognitive functions, including response inhibition, is that the drug reduces one's information processing capacity. Analyses of subjects' reaction time and event-related potentials show that alcohol impairs performance on various laboratory tasks by slowing information processing to disrupt late stages of stimulus-response selection (Bartholow et al., 2003; Fillmore & Van Selst, 2002; Lukas, Mendelson, Kouri, Bolduc, & Amass, 1990; Moskowitz & Depry, 1968). A common cause of slowed information processing is reduced information processing capacity (Pashler, 1994). In accord with a "reduced processing capacity" account for alcohol impairment of cognitive functions, there is evidence that alcohol impairment intensifies as a function of task complexity. As greater processing is required to complete a task, drinkers tend to be more impaired, even at doses where no impairment is present for the performance of simpler tasks (Maylor, Rabbitt, James, & Kerr, 1992). However, there are instances where the addition of information can reduce the impairing effects of alcohol and benefit task performance.

The Redundant Signal Effect and Alcohol Impairment

Characteristics of stimuli are important in the facilitation of processing and improving performance. For example, people tend to respond more quickly to environmental signals that are delivered redundantly to more than one sensory modality as opposed to one sensory modality at a time (Diederich & Colonius, 2004; Forster, Cavina-Pratesi, Aglioti, & Berlucchi, 2002; Gondan, Götze, & Greenlee, 2010). This phenomenon has been recognized for some time (Todd, 1912) and is referred to as the "redundant signal effect" (RSE). Studies of the RSE typically require participants to

perform a choice response task with three conditions: one in which participants respond to a visual cue (e.g., an X or O), another where they respond to an auditory cue (e.g., a high or low tone), and one condition where both stimuli are presented simultaneously (Sinnett, Soto-Faraco, & Spence, 2008). Performance in the simultaneous redundant signal condition is superior to performance in both unisensory conditions, both in terms of the speed and accuracy of responses.

This facilitative effect of redundant signals could be used to reduce acute alcohol impairment of inhibitory control. Neuroimaging studies indicate that alcohol decreases activity in the same regions impacted in inhibitory control (Anderson et al., 2011; Marinkovic, Rickenbacher, Azma, & Artsy, 2012), which may explain why the drug reduces inhibitory control. Although the neural processes underlying the RSE are not fully understood, there is evidence that specialized multisensory neurons distributed in key brain regions become active in the presence of multisensory stimuli. Chen and colleagues (2015) reported evidence for multisensory activation in many of the brain regions involved in inhibitory control, including the right anterior insula, dorsal anterior cingulate, and posterior parietal cortices, suggesting that multisensory signals may facilitate response inhibition. Indeed, there is some evidence that multisensory inhibitory signals can enhance inhibitory control on measures, such as stop-signal tasks (Cavina-Pratesi, Bricolo, Prior, & Marzi, 2001; Gondan et al., 2010; Gondan, Niederhaus, Rösler, & Röder, 2005). The finding that redundant multisensory signals could enhance neural activation in the same inhibitory control regions where alcohol reduces activation suggests that multisensory signals could also ameliorate the drug's impairing effects on behavior.

Reducing Alcohol Impairment with Multisensory Signals

Our lab was the first to test how redundant signals can reduce the degree to which alcohol impairs task performance (Fillmore, 2010). Participants performed a two-choice reaction time task in which they were required to press a key in response to a stimulus. Stimuli were presented as visual (i.e., letters), auditory (i.e., tones), or redundant signals (i.e., a letter and a tone presented simultaneously). Performance was tested under 3 alcohol doses: 0.65, 0.45, and 0.0 g/kg (placebo). Multisensory redundant signals produced faster reaction time compared with either of the unimodal signals. Alcohol slowed reaction time to all stimuli. However, the speed advantage produced by the multisensory redundant stimuli was maintained at BACs above 80 mg/100 ml. These early findings suggest that the presence of redundant stimuli can attenuate the degree to which alcohol impairs task performance, raising the possibility that redundant "stop" signals might reduce alcohol impairment of inhibitory control.

Reducing Alcohol Disinhibition with Multisensory Signals

Our group recently tested the possibility that multisensory inhibitory signals can reduce the disinhibiting effects of alcohol in healthy adults (Roberts et al., 2016). Inhibitory control was assessed by a go/no-go task, which included unisensory (visual) and multisensory (Visual + Aural) inhibitory signals. The task measured participants' inhibitory control following a dose of alcohol designed to produce a peak BAC of 80 mg/100 mL and a placebo. Results showed that alcohol reliably impaired inhibitory control when stop signals were unisensory. However, when multisensory stimuli were used as stop signals, alcohol had no impairing effects on inhibitory control. Participants' eye movements (saccades) were also measured to determine how multisensory signals

affected the speed with which they visually located the visual stop and go target stimuli on the computer. Alcohol slowed saccadic reaction time to targets. However, this slowing effect was reduced by the multisensory signals. Additionally, alcohol slowed manual reaction time to targets and the slowing effect was also reduced by multisensory signals. These results suggest that multisensory signals might facilitate the speed with which drinkers gather relevant information from the environment to guide their behavior.

Extending the Evidence to a Clinical Sample

The study by Roberts et al. (2016) is important because the results indicate that multisensory stimuli can serve as a protective factor against the disruptive effects of alcohol. A logical continuation of this research is to determine the degree to which multisensory inhibitory signals can also strengthen inhibitory control in drinkers with deficient inhibitory control and attentional dysfunction, such as those with attention-deficit hyperactivity disorder (ADHD) (Roberts, Milich, & Fillmore, 2013). Individuals with ADHD have heightened impulsivity and increased risk for alcohol and other drug use (Charach et al., 2011; Lee et al., 2011; Weafer, Milich, & Fillmore, 2011). Longitudinal studies have found that childhood ADHD leads to early onset of alcohol use, which can transition to heavy use in young adulthood, especially accompanied by conduct disorder or delinquency (Molina et al., 2014; Sibley, Kuriyan, Evans, Waxmonsky, & Smith, 2014). Studies of adults with ADHD find lifetime rates of alcohol abuse disorders ranging between 21% and 53% (Barkley, Murphy, & Kwasnik, 1996; Biederman, 2004). Moreover, there is growing suspicion that heightened impulsivity might be a key behavioral mechanism that contributes to the elevated risk for alcohol abuse among individuals with ADHD (Fillmore, 2003, 2004). In terms of acute reaction

to alcohol, stop signal and cued go/no-go tasks have demonstrated deficits of response

inhibition in children with ADHD and adults with the disorder (Alderson, Rapport, &

Kofler, 2007; Barkley, 1997; Bekker et al., 2005; Lijffijt, Kenemans, Verbaten, & van

Engeland, 2005; Oosterlaan, Logan, & Sergeant, 1998; Tannock, 1998). Individuals with

ADHD display heightened disinhibition to alcohol similar to other at-risk groups, such as

binge drinkers (Marczinski et al., 2007; Weafer et al., 2009). Given heightened alcohol

sensitivity and elevated risk for alcohol abuse among those with ADHD, it would be

important to determine the degree to which multisensory stimuli could serve as a

protective factor against the disruptive effects of alcohol in this clinical population.

Reducing Alcohol Disinhibition with Multisensory Signals in a Clinical Sample

The purpose of this thesis is to test whether multisensory signals could reduce the

disinhibiting effects of alcohol in individuals with ADHD by facilitating their attention to

inhibitory cues. A group of healthy adults and a group of adults with ADHD received

0.65 g/kg alcohol and placebo and completed a multisensory cued go/no-go task that

measured how multisensory signals affect ability to quickly respond as well as inhibit

responses. I hypothesized that those with ADHD would display generally poorer response

inhibition and heightened disinhibition in response to alcohol compared with controls.

With respect to multisensory facilitation, it was predicted that multisensory signals would

reduce the disinhibiting effects of alcohol in controls. The primary research question

concerns the degree to which multisensory signals would yield a similar reduction in

alcohol-induced disinhibition in those with ADHD.

Methods

Participants

Forty-four adult drinkers, 22 adults with ADHD (11 men and 11 women; age = 23.8, *SD* = 2.1 year) and 22 adults with no history of ADHD (10 men and 12 women; age = 22.7, *SD* = 2.0 year) participated in this study. Recruiting took place through fliers and online advertising seeking adults (Ages 21-29) with and without ADHD for a study of the effects of alcohol. Volunteers were screened via telephone to ensure they were at least 21 years old, had normal or corrected vision and hearing, and consumed alcohol at least once per week. Individuals who reported taking psychotropic medication, other than psychostimulant medication for ADHD, were not invited to participate. Volunteers who reported past or current severe psychiatric diagnoses (e.g., bipolar disorder, schizophrenia) did not participate in this study. Following initial screening, volunteers who met these criteria were contacted via telephone and invited to participate in the study. Urine samples were tested for the presence of metabolites of amphetamine, methamphetamine, barbiturates, benzodiazepines, cocaine, opiates, methadone, phencyclidine, tricyclic antidepressants, and tetrahydrocannabinol (THC; ICUP Drugscreen; Instant Technologies, Norfolk, VA). Positive urine analysis for any substance other than THC or amphetamine, for the ADHD group, resulted in discontinuation from the study. Participants who reported use of marijuana during the 24 hours preceding the session were discontinued. Urine samples were also tested for pregnancy in female participants (Icon25 Hcg Urine Test; Beckman Coulter, Pasadena, CA). No female volunteers who were pregnant or breastfeeding participated in the

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research. All participants were required to abstain from alcohol for 24 hours prior to each session and a breathalyzer confirmed a zero BAC at the outset of each session.

To ensure that members of the ADHD group experienced symptomatology severe enough to necessitate medication, only volunteers who were currently prescribed medication for ADHD were invited to participate. Members of the ADHD group reported several different prescriptions, including amphetamine ($n = 10$), lisdexamfetamine ($n = 8$), methylphenidate ($n = 3$), and dextroamphetamine ($n = 1$). Prescription status was confirmed by the experimenter during the first session. Participants were asked to abstain from taking their medication for at least 24 hours prior to each session to ensure that they were unmedicated during the testing sessions. ADHD diagnosis was confirmed by clinical interview and we required that volunteers met symptoms-based criteria on three measures of ADHD symptomatology, including the Conners' Adult ADHD Rating Scale—Long Form (Conners, Erhardt, & Sparrow, 1999), the Adult ADHD Self-Report Scale Symptoms Checklist (Kessler et al., 2005), and the ADD/H Adolescent Self-Report Scale (Robin & Vandermay, 1996). A similar method of diagnostic confirmation has been successfully used by this research group in other studies (Roberts, Fillmore, & Milich, 2011a, 2011b). Participants completed the 45-item Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behavior Scale (UPPS-P; Lynam, Smith, Whiteside, & Cyders, 2006) as an additional measure of impulsivity. The UPPS-P served to further validate group classification. Rating scale scores for ADHD symptoms and impulsivity measures are reported in Table 1.

Table 1

Group Comparisons on ADHD Symptoms and Impulsivity Measures

	Group					
	Control (*n* = 22)			ADHD (*n* = 22)		
	Mean	SD		Mean	SD	*t*
Diagnostic						
CAARS						
DSM-IA	53.0	9.6		73.1	10.9	-6.5***
DSM-HI	50.3	9.5		59.4	15.8	-2.3
DSM-Tot	52.7	10.3		69.6	13.3	-4.7***
AASRS	1.9	1.4		4.1	1.6	-4.9***
ADDRV	9.3	5.9		21.2	5.8	-6.8***
UPPS						
UPPS-Premed	1.9	0.4		2.4	0.6	-3.1**
UPPS-Urg	2.1	0.6		2.6	0.6	-3.0**
UPPS-Sens	3.2	0.5		3.2	0.5	0.29
UPPS-Presev	1.8	0.5		2.5	0.5	-4.6***

Note. For all comparisons, *N* = 44, degrees of freedom = 42. CAARS scores are T-scores. ADHD = attention-deficit hyperactivity disorder; CAARS = Conners' Adult ADHD Rating Scale; *DSM*-IA = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (*DSM-IV*; American Psychiatric Association, 1994) Inattentive Symptoms; *DSM*-HI = *DSM-IV* Hyperactive-Impulsive Symptoms; *DSM*-Tot = DSM-IV ADHD Symptoms Total; AASRS = score on the first six questions of the Adult ADHD Self-Report Scale; ADDRV = total score on first 18 questions of the ADD/H Adolescent Self-Report Scale; UPPS = scores on the four traits measured by the Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behavior Scale; UPPS-Premed = lack of premeditation; UPPS-Urg = urgency; UPPS-Sens = sensation seeking; and UPPS-Persev = lack of perseverance.
**$p < .01$. ***$p < .001$.

Materials and Measures

       Multisensory Go/No-Go Task. This task, used in the study by Roberts et al.

(2016), examined the ability of participants to inhibit prepotent responses to stop signals

that were presented either as a visual stimulus or as a multisensory stimulus, comprised

of a visual and auditory signal. The task required individuals to locate the go or no-go

target, which appeared in a different location than where the cue was presented. Figure 1

illustrates a trial sequence on the task. Participants were presented with a cue in the

middle of the screen, which is followed by the presentation of a target in 1 of 8 possible

locations. Participants were instructed to respond to these targets by either pressing a

button (forward slash key) in response to a go target (the word *go*) or inhibit a response

when presented with a no-go target (the word *no*). Each trial consisted of the following

events: (a) presentation of a fixation point for 800 ms; (b) a preresponse cue that was

displayed for 1 of 6 stimulus onset asynchronies preceding the target (SOAs = 100, 200,

300, 400, 500, and 600 ms); (c) a go or no-go target visible at 1 of 8 locations around the

center of the screen, which remained visible until either a response was made or 1 second

elapsed; and (d) a 700 ms intertrial interval.

       The preresponse cue was either green or blue and signaled the probability that a

go or no-go target would be displayed. Green squares preceded the go target on 80% of

the trials and preceded the no-go target on 20% of the trials. Blue squares preceded the

no-go target on 80% of the trials and preceded the go target on 20% of the trials. Green

and blue squares functioned as go and no-go cues, respectively. Presentation of the go-

cue increases response preparation, making it more difficult to inhibit a response when

the no-go target unexpectedly appears. The disinhibiting effects of alcohol are most

evident in this cue condition (Marczinski & Fillmore, 2003). The random SOAs (100,
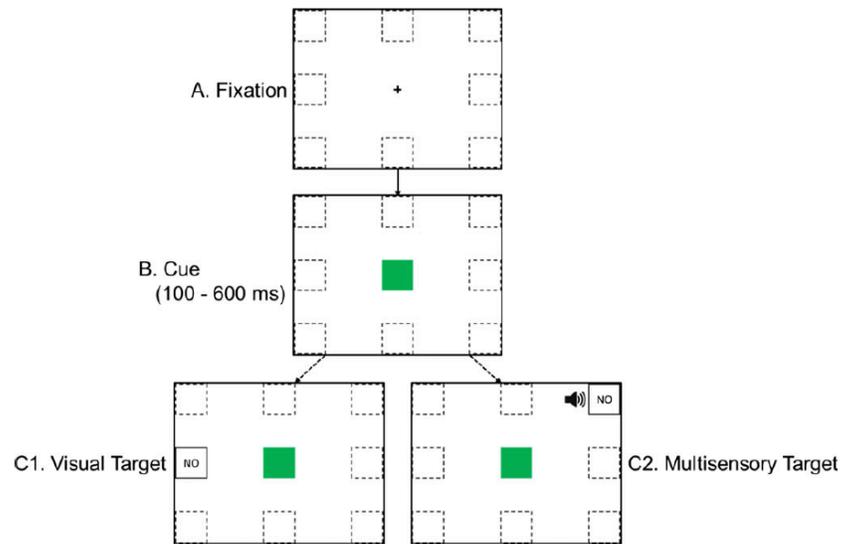
Figure 1. Schematic of a go-cue trial in the multisensory go/no-go task. Following the fixation point (panel **A**), a go-cue is presented (panel **B**). A green square serves as a go-cue, signally that a go target is likely to appear. In this example, a no-go target is then presented (panels **C1** and **C2**). In panel C1, the no-go target is a visual only (unisensory) and is displayed in the central left region. In panel **C2**, the no-go target is presented in the upper right and paired with the no-go (i.e., 125 Hz) tone (multisensory).

200, 300, 400, 500, and 600 ms) between the cues prevented participants from anticipating the exact onset of the targets.

During half of the trials, targets were presented as multisensory signals by presenting an auditory tone in conjunction with the visual "go" and "no" target stimuli. During multisensory target trials, tones were presented concurrently with the go or no-go targets. Go targets were presented with a 1,000 Hz (high) tone and no-go targets were presented with a 125 Hz (low) tone. Participants were told that some trials may include tones, but they were not given specific instruction about the purpose of the tones. Participants were told to locate the visual target before making the appropriate response.

A test consisted of 200 trials that presented all possible cue-target combinations for both visual and multisensory trials and required 15 minutes to complete. Half of the trials presented visual targets, and in the other half of the trials, multisensory signals were presented. The target was presented in each possible radial position at least one time for each cue-target combination for both visual and multisensory trials. SOAs were distributed evenly across the different cue-target conditions in both visual and multisensory trials. The trial order was pseudo-random to avoid clustering of visual or multisensory trials. To encourage quick and accurate responding, feedback was presented to the participant during the intertrial interval by displaying the words *correct* or *incorrect* and their response time in milliseconds.

The task was operated using E-prime software on a PC (Schneider, Eschman, & Zuccolotto, 2002). Participants' eye movements during each trial were measured to assess the speed with which they oriented their attention to the visual target when it was presented. A Tobii T120 Eye Tracking Monitor (Tobii Technology, Stockholm, Sweden)

equipped with dual embedded cameras was used to track eye movements. Participants were seated with their heads approximately 60 cm in front of the computer with a free range of head and neck motion. Gaze locations were sampled at 120 Hz, and fixations were defined as gazes with standard deviations <0.5° of visual angle for durations of 90 ms or longer. All sampled eye locations during a fixation were averaged to determine the location of that fixation. By tracking eye movements, we were able to quantify how quickly participants attended to response targets once they were presented.

Drinking Habits. Participants' drinking habits were assessed using the Timeline Follow-Back (TLFB; Sobell & Sobell, 1992), which assessed daily drinking patterns over the past 3 months, and the Personal Drinking Habits Questionnaire (PDHQ; Vogel-Sprott, 1992), which provided information regarding participants' alcohol consumption. For the TLFB, four measures of drinking habits were obtained: (a) total number of drinking days (drinking days), (b) total number of drinks consumed (total drinks), (c) total number of days characterized by subjective drunkenness (drunk days), and (d) total number of days in which binge drinking occurred (binge days). Binge drinking days were determined by estimating participants BACs on each day according to the participants' weight, the reported number of drinks they consumed, and the amount of time they spent drinking using anthropometric based BAC estimation formulae that assume an average clearance rate of 15 mg/100 ml per hour (Watson, Watson, & Batt, 1981). For the PDHQ, participants recorded both history of alcohol use (number of months of regular drinking), as well as information regarding current, typical drinking habits, including (a) frequency (the typical number of drinking occasions per week), (b) quantity (the number of standard alcoholic drinks [e.g., 1.5 oz of liquor] typically consumed per occasion), and (c) duration

(time span in hours of a typical drinking occasion). Participants also completed the

Alcohol Use Disorders Identification Test (AUDIT; Babor, Kranzler, & Lauerman,

1989). The AUDIT is a screening instrument that was used to assess the occurrence and

severity of alcohol-related problems. The 10-item, self-report questionnaire covers

patterns of drinking, dependence, and other negative consequences of drinking over the

past year and has a total score range from 0 (*no alcohol-related problems*) to 40 (*most

severe alcohol-related problems*).

Procedure

Volunteers responding to advertisements for this study underwent an intake

screening by telephone. They were told that the purpose of the study was to examine the

effects of alcohol on performance of computer tasks. They then made appointments to

come to the laboratory for three sessions, including one familiarization and two dose-

challenge sessions. The dose-challenge sessions were separated by an average of 4.0 days

(*SD* = 2.5) for the ADHD group and 6.6 days (*SD* = 3.9) for the control group.

Participants were instructed to fast for 4 hours prior to each dose-challenge session. They

were also instructed to abstain from consuming alcohol or using other psychoactive

drugs, including ADHD medication, during the 24 hours preceding each session.

Familiarization Session. All participants completed a familiarization session

during which they became acquainted with laboratory procedures, completed

questionnaires, provided informed consent for participation, completed the Kaufman

Brief Intelligence Test (Kaufman & Kaufman, 2004), and performed a training version of

the multisensory go/no-go task. Volunteers who did not meet criteria for participation in

the study were paid $10 and discontinued.

Dose-Challenge Sessions. Participants were tested under 0.65 g/kg alcohol and placebo. Participants were blinded to dose, and dose order was counterbalanced across the two test sessions. Sessions were separated by no less than 1 day and no more than 1 week. Alcohol doses were calculated on the basis of body weight and administered as absolute alcohol mixed with 3 parts carbonated soda. A peak BAC of 80 mg/100 ml is produced by the 0.65 g/kg dose approximately 65 minutes post administration (Fillmore et al., 2005; Roberts et al., 2013). The placebo dose consisted of an equal volume of carbonated soda mix matching the total volume of the 0.65 g/kg alcohol dose. A small amount (3 ml) of alcohol was floated on the surface of the beverage, and it was sprayed with an alcohol mist that resembled condensation and provided a strong alcoholic scent as the beverage was consumed. All drinks were consumed within 6 minutes.

Participants performed the multisensory go/no-go task 30 minutes after dose administration. BAC levels were recorded throughout the session at 28, 45, 52, 65, and 72 minutes following dose administration for both the 0.0 and 0.65 g/kg dose. BACs were determined from expired air samples measured by an Intoxilyzer Model 400 (CMI, Inc., Owensboro, KY). Following testing, participants remained in a lounge area until their BACs reached 20 mg/100 ml or below. Participants received a meal and were allowed to watch movies and relax. Transportation home was provided if necessary. Participants were paid $80 for completion. Participants were also debriefed upon completion of the final session.

Criterion Variables and Data Analyses

The multisensory go/no-go task measures inhibitory control and response speed to visual and multisensory stimuli. Response inhibition was measured as participants'

failures to inhibit responses to stop targets (i.e., failures of response inhibition). Failure of response inhibition was measured as the proportion (p) of no-go targets in the go-cue condition in which a participant failed to inhibit a response (i.e., p-inhibition failures). Manual reaction time was defined as the mean time taken to make a response during go target trials. Shorter reaction times indicated greater facilitation of response execution. Responses with reaction times <100 and >1,000 ms were excluded. These outliers were infrequent, occurring, on average, less than 0.25% of the trials for which a response was observed (i.e., less than one trial per test). Reaction times and p-inhibition failures were calculated separately for unisensory (i.e., visual) and multisensory trials.

Visual fixations were used to determine saccadic reaction time. Saccadic reaction time was defined as the number of milliseconds that elapsed between the presentation of the target and the beginning of the first visual fixation at the location of the target.

P-inhibition failures, manual reaction time, and saccadic reaction time were each analyzed by 2 (dose: placebo vs. 0.65 g/kg alcohol) by 2 (target condition: unisensory vs. multisensory) by 2 (group: control vs. ADHD) repeated-measures analyses of variance (ANOVAs). A limited number of simple effect tests were performed to test the hypothesis that alcohol-induced disinhibition would be reduced by multisensory stop targets and compare the groups in the magnitude of this effect.

I conducted all analyses of alcohol effects to include sex as a factor. These analyses found no significant effect of sex and did not change the significance level of other main effects or interactions. As such, reported analyses of task performance are collapsed across sex.

Results

Drinking and Demographic Information

Participants' drinking habits and demographic information are presented in Table 2. The table shows no difference between the two groups regarding level of education or IQ scores. With respect to drinking habits, the TLFB and PDHQ show no differences between the two groups. Both groups drank twice a week, on average, with a typical quantity per occasion of approximately 4 standard drinks. In addition to moderate alcohol use, some participants reported past month use of nicotine ($n = 10$), marijuana ($n = 13$), sedatives ($n = 1$), stimulants ($n = 2$), cocaine ($n = 3$), and club drugs ($n = 1$). Seven participants had tetrahydrocannabinol positive urine screens during one or more dose-challenge session. No other drug urine screens were positive.

Blood Alcohol Concentrations

Group differences in BAC under 0.65 g/kg alcohol were examined using a 2 (group) X 4 (time: 25, 45, 65, and 95) mixed-design ANOVA. There was a significant main effect of time, $F (3, 126) = 38.23$, $p < .001$, $\eta_p^2 = .477$, because of the rise and decline of BAC over the time course of the testing session. The mean BACs (mg/100 mL) at 25, 45, 65, and 95 minutes were 68.2 ($SD = 21.4$), 84.2 ($SD = 15.3$), 86.4 ($SD = 17.3$), and 69.6 ($SD = 14.0$), respectively. There was no main effect of group or Group X Time interaction (ps > 0.05). No detectable BACs were observed in the placebo condition.

Table 2

Group Comparisons on Demographic Characteristics and Self-Reported Drinking Habits

	Group					
	Control (*n* = 22)			ADHD (*n* = 22)		
	Mean	SD		Mean	SD	*t*
Demographic						
Age	23.8	2.1		22.7	2.0	1.7
Sex (% male)	50.0			45.5		
Weight (kg)	76.8	15.1		75.8	17.2	0.2
Education	15.5	1.6		15.0	1.6	0.9
IQ: Verbal	102.8	12.8		102.2	7.5	0.2
IQ: Nonverbal	99.2	13.8		103.9	10.9	-1.3
IQ: Composite	101.4	14.0		103.9	9.3	-0.7
Drinking Habits						
TLFB						
Drinking Days	30.4	17.6		23.4	13.5	1.5
Total Drinks	152.0	117.6		113.0	107.8	1.2
Drunk Days	11.8	9.5		8.8	9.4	1.1
Binge Days	12.3	14.3		8.9	10.8	0.9
PDHQ						
Frequency	2.5	1.7		1.6	0.9	2.1
Quantity	3.9	1.8		4.4	1.9	-0.8
AUDIT	8.1	4.2		9.2	4.7	-0.8

Note. For all comparisons, *N* = 44, degrees of freedom = 42. Age is reported in years. ADHD = attention-deficit hyperactivity disorder; TLFB = variables reported on the Timeline Follow-Back Procedure; PDHQ = Personal Drinking Habits Questionnaire; AUDIT = Alcohol Use Disorders Identification Test.

***P*-inhibition Failures**

Figure 2 presents the mean *p*-inhibition failures following placebo and alcohol in response to multisensory and unisensory signals for the control (left panel) and ADHD group (right panel). For unisensory stop signals, controls displayed increased inhibitory failures in response to alcohol compared with placebo (i.e., disinhibition). However, for multisensory stop signals, alcohol had no disinhibiting effect. By contrast, for those with ADHD, alcohol increased inhibitory failures compared with placebo to both unisensory and multisensory signals. Indeed, Figure 2 shows that the highest degrees of *p*-inhibition failures under alcohol were displayed by those with ADHD regardless of whether signals were unisensory or multisensory. These data were analyzed by a 2 (dose) X 2 (target condition) X 2 (group) ANOVA. The ANOVA showed a significant main effect of dose, $F(1, 42) = 12.28$, $p = .001$, $\eta_p^2 = .226$, and a Target Condition X Group interaction, $F(1, 42) = 4.40$, $p = .042$, $\eta_p^2 = .095$. No additional main effects or interactions were significant. For controls, simple effect comparisons tested the hypothesis that alcohol-induced disinhibition was reduced by multisensory versus unisensory stop targets. This hypothesis was supported as alcohol significantly increased inhibitory failures compared with placebo in the unisensory target condition, $t(21) = -3.0$, $p = .007$, $d_z = -0.64$, but not in the multisensory target condition, $t(21) = -.442$, $p = .663$, $d_z = -0.10$. Indeed, under the active dose, controls made significantly fewer inhibitory failures to multisensory versus unisensory targets, $t(21) = 3.0$, $p = .006$, $d_z = 0.65$. For the ADHD group, who displayed similar increases in *p*-inhibition failures following alcohol regardless of target condition, there were no significant differences in inhibition failures between target conditions
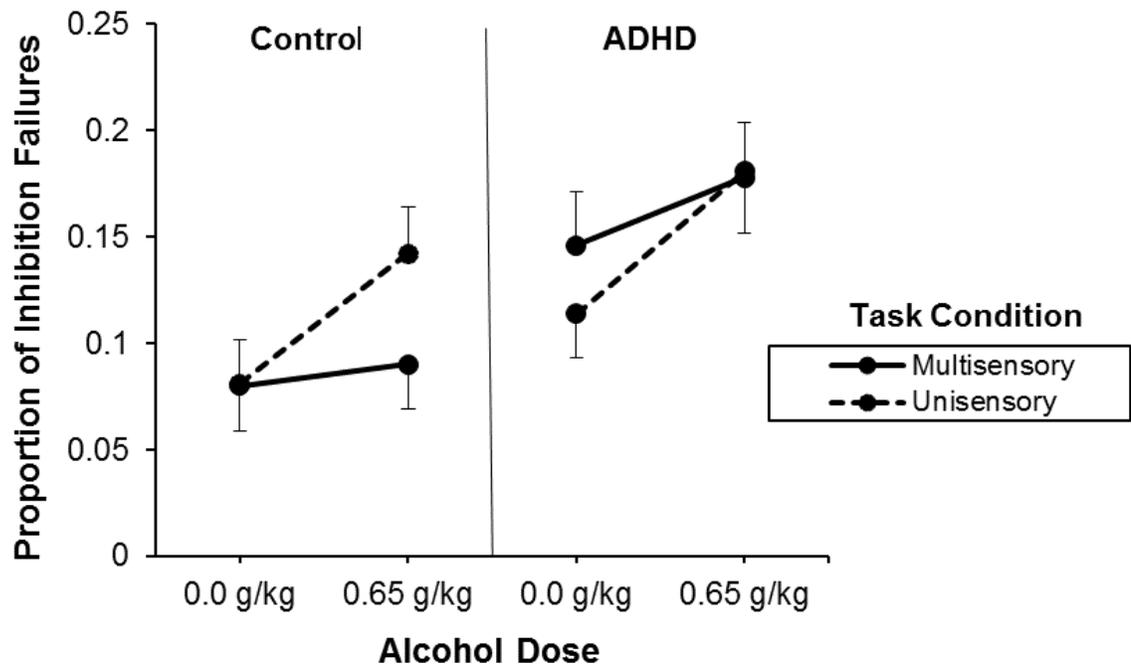
Figure 2. Effects of alcohol dose and target condition on proportion of inhibition failures in the control and attention-deficit hyperactivity disorder (ADHD) groups. Capped vertical lines show standard errors of the means.

following placebo, $t(21) = -1.5$, $p = .143$, $d_z = -0.32$, or alcohol, $t(21) = -0.662$, $p = .515$, $d_z = -0.14$.

Reaction Time

Manual. Figure 3 presents mean manual reaction time following placebo and alcohol in response to multisensory and unisensory signals for the control (left panel) and ADHD group (right panel). The figure shows that alcohol slowed manual reaction time compared with placebo in both groups. Additionally, responses were faster to multisensory go targets versus unisensory go targets for both groups under both placebo and alcohol. The 2 (dose) X 2 (target condition) X 2 (group) ANOVA showed a significant main effect of dose, $F(1, 42) = 19.57$, $p < .001$, $\eta_p^2 = .318$, and target condition, $F(1, 42) = 88.95$, $p < .001$, $\eta_p^2 = .679$. No interactions were significant (ps > 0.05).

Saccadic. Figure 4 plots the mean saccadic reaction time following placebo and alcohol in response to multisensory and unisensory signals for the control (left panel) and ADHD group (right panel). The figure shows that alcohol slowed saccadic reaction time compared with placebo in both groups. Additionally, responses were faster to multisensory go targets versus unisensory go targets for both groups under both placebo and alcohol. The 2 (dose) X 2 (target condition) X 2 (group) ANOVA showed a significant main effect of dose, $F(1, 42) = 69.45$, $p < .001$, $\eta_p^2 = .623$, and target condition, $F(1, 42) = 9.68$, $p = .003$, $\eta_p^2 = .187$. No interactions were significant (ps > 0.05).

Figure 3. Effects of alcohol dose and target condition on manual reaction time in the control and attention-deficit hyperactivity disorder (ADHD) groups. Capped vertical lines show standard errors of the means.
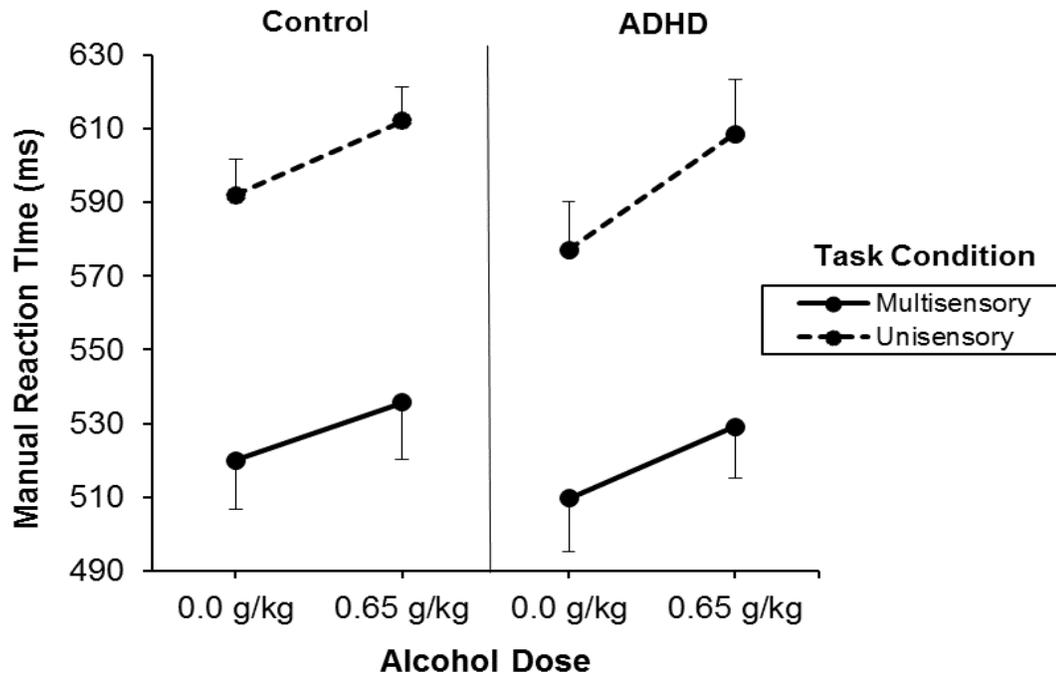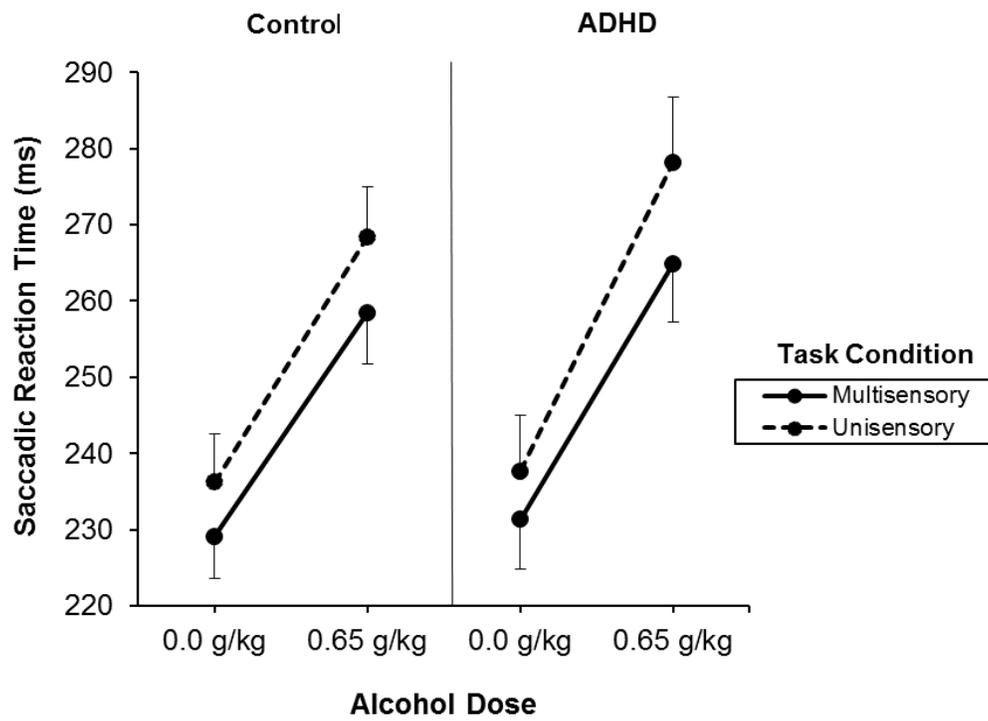
Figure 4. Effects of alcohol dose and target condition on saccadic reaction time in the control and attention-deficit hyperactivity disorder (ADHD) groups. Capped vertical lines show standard errors of the means.

Discussion

This study used the go/no-go task to examine the ability of multisensory stop signals to attenuate the disinhibiting effects of alcohol in individuals with ADHD and healthy controls. The results were consistent with previous research that has shown that multisensory stop signals can attenuate the disinhibiting effects of alcohol (Roberts et al., 2016). With the controls, alcohol increased inhibitory failures when the stop signal was unisensory, but when the signal was multisensory, response inhibition was not impaired. In the ADHD group, alcohol increased inhibitory failures regardless of whether the stop signal was unisensory or multisensory. With respect to reaction time, alcohol slowed responses to go targets similarly for controls and ADHD subjects and reaction time was faster to multisensory signals in both groups compared to unisensory signals regardless of dose. Saccadic reaction time to locate the targets showed the same pattern of results. Alcohol slowed saccadic reaction time with reaction times faster to multisensory versus unisensory targets regardless of dose.

This thesis is the first to study the use of multisensory signals with a clinical population. My hypothesis that those with ADHD would display generally poorer response inhibition and heightened disinhibition in response to alcohol compared with controls was supported. Additionally, my hypothesis with respect to multisensory facilitation that multisensory signals would reduce the disinhibiting effects of alcohol in controls was also supported. The primary research question concerning the degree to which multisensory signals yield a similar reduction in alcohol-induced disinhibition in those with ADHD compared with controls has been answered because it was shown that

multisensory signals failed to attenuate the disinhibiting effect of alcohol in those with ADHD.

Overall, this thesis helps provide a better understanding of neural and behavioral mechanisms by which drugs disrupt behavior as well as contribute to the understanding of how multisensory signals effect responses in individuals with ADHD. For the control participants, results were consistent with previous research that has shown that multisensory stop signals can attenuate the disinhibiting effects of alcohol (Roberts et al., 2016). Although it is not entirely clear how multisensory signals protect against alcohol-induced disinhibition, one possible explanation concerns alcohol-induced slowing of information processing speed. Evidence suggests that alcohol impairs behavior by slowing the speed with which drinkers are able to process information (Bartholow et al., 2003; Fillmore & Van Selst, 2002). Presenting multisensory response targets may facilitate the recruitment of additional processing resources, perhaps via activation of multisensory neurons in the superior colliculus involved in saccadic eye movement (Lee, Rohrer, & Sparks, 1988). Saccadic reaction time to target stimuli was hastened by multisensory signals raising the possibility that multisensory stimuli protected against alcohol impairment by increasing the speed with which drinkers can attend to and process the stimuli, including no-go stimuli that signal response inhibition. However, findings from those with ADHD indicate that hastening of saccadic reaction time by multisensory stimuli cannot fully explain their attenuating effects on alcohol-induced disinhibition. Although those with ADHD also demonstrated a hastening of their saccadic reaction time from multisensory signals, these signals had no attenuating effects on the degree to which alcohol impaired their inhibitory control. It is also important to note that for the controls,

multisensory signals did not improve inhibitory control in the sober state (i.e., following placebo). This is possibly due to the fact that while sober, controls' inhibitory control was near optimal, with few inhibitory failures. Therefore, a floor effect could have precluded observing any facilitation of the multisensory signals.

There may be several reasons why multisensory signals failed to attenuate the disinhibiting effect of alcohol in those with ADHD. One possibility is that those with ADHD have an impaired ability to integrate multisensory stop signals that guide behavior. However, those with ADHD showed benefits of multisensory signals in facilitating their reaction time to go stimuli that were comparable to controls. Therefore, any failure of the ADHD group to integrate signals would appear to be specific to signals concerning the inhibition of actions (i.e., stop/no-go signal) rather than signals to execute behavior (go signals). Future studies using functional neuroimaging of these behaviors in those with ADHD in the intoxicated state would be helpful in testing such hypotheses. Any deficit of multisensory integration in those with ADHD could be specific to neural regions involved in the suppression of behavior (e.g., the anterior cingulate insula and dorsolateral prefrontal cortex; Botvinick et al., 2004).

Regardless of the neural basis, the failure of multisensory stimuli to attenuate alcohol-induced impairment of inhibitory control in these individuals highlights a potential vulnerability of the group that could account for their heightened sensitivity to the behaviorally disruptive effects of alcohol. Previous research by our lab has shown that moderate drinkers with ADHD have heightened sensitivity to the disinhibiting effects of alcohol on cued go/no-go tasks and display less acute tolerance to alcohol, prolonging impairment of inhibitory control compared with controls (Roberts, Fillmore, & Milich,

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2012; Weafer et al., 2009; Roberts et al., 2013). Laboratory observations of these impairments could also translate to everyday problems for this population. Outside of the laboratory, cues that signal behavior are often multisensory. It is likely that individuals commonly benefit from redundant multisensory stimuli in the environment to signal whether or not actions should be expressed or withheld. However, those with ADHD might not benefit from this additional information to guide behavior.

The findings of this thesis should be considered in light of some limitations. First, participants were tested under a single active dose of alcohol. Although this dose was selected for its ability to produce considerable behavioral impairment in adult drinkers (Holloway, 1995), it would be informative to examine how multisensory signals affect alcohol impairment under a range of doses. Second, we did not test participants' performance in an auditory only condition. However, prior research using similar paradigms to study the RSE on response activation shows similar reaction times to visual and auditory unisensory targets (Fillmore, 2010). Further, these studies find that multisensory response targets engender comparable improvement in reaction time over both unisensory conditions.

These results are important because they indicate that multisensory stimuli can serve as a protective factor against the disruptive effects of alcohol. By examining multisensory stimuli, this thesis provides a more ecologically based account of how individuals exercise impulse control in their everyday environments, where information is delivered to multiple sensory channels. A logical continuation of this technique is to use multisensory signals to strengthen inhibitory control in individuals before drinking begins when predrinking alcohol-related cues capture their attention and disinhibit their

behavior. In regard to the ADHD group, those with ADHD were tested in the unmedicated state, which is not the everyday environment for these individuals. It is possible that the inhibitory control of those with ADHD could have benefited from these multisensory signals if they had received their medication treatment (e.g., methylphenidate). Stimulant medications improve inhibitory control (Fillmore, Kelly, & Martin, 2005; Fillmore, Rush, & Hays, 2006; Schachter, Pham, King, Langford, & Moher, 2001; Tannock, Ickowicz, & Schachar, 1995). However, these tests have been based on unisensory models in which the stop signal is a single unimodal stimulus. Future research using the model of multisensory facilitation of behavior could be used to better understand current behavioral and pharmacological treatments for impulse control disorders. It is unknown how stimulant medications would interact with multisensory signals to inhibit action. These types of treatments could have clinical efficacy through improved integration of multisensory signals that guide behavior. This possibility waits to be explored.

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predict alcohol consumption in adults with ADHD and healthy controls. *Drug and
Alcohol Dependence*, *113*(2-3), 139-146.

VITA

ALEXANDRA RENEE D'AGOSTINO
Department of Psychology
University of Kentucky

EDUCATION

University of Kentucky, Lexington KY   August 2017-Present
- Advisor: Mark T. Fillmore, Ph.D. and
  Gregory T. Smith, Ph.D.

Saint Louis University, St. Louis, MO   August 2013-May 2017
- Bachelor of Science: Psychology
- Minor: Biology
- Magna Cum Laude

RESEARCH EXPERIENCE

University of Kentucky, Lexington, KY   August 2017-Present
- Dr. Mark Fillmore's Research Lab
  - Supervisor Mark T. Fillmore, Ph.D.
- Adolescent Risky Behavior Research Laboratory
  - Supervisor: Gregory T. Smith, Ph.D.

Saint Louis University, St. Louis, MO   September 2014-May 2017
- Wellness, Addiction, Gambling and Exercise
  Research Laboratory
  - Supervisor: Jeremiah Weinstock, Ph.D.

Washington University School of Medicine, St. Louis, MO   August 2016
- Health and Behavior Research Center   -May 2017
  - Supervisor: Patricia Cavazos-Rehg, Ph.D.

CLINICAL EXPERIENCE

Harris Psychological Services Center   August 2018-Present
Lexington, KY
- Graduate Student Therapist
- Happy Healthy Kids Class Co-Leader
- Firefighter Assessments

University of Kentucky Counseling Center   August 2018-December 2018
Lexington, KY
- Practicum-Level Individual Therapist
- Interpersonal Process Group Process Observer

University of Kentucky   August 2018-December 2018
Lexington, KY
- Firefighter Integrated Reports
- Personality Assessment Practicum
- IQ Assessment Practicum

TEACHING EXPERIENCE

University of Kentucky   August 2017-May 2018
Lexington, KY
- Graduate Teaching Assistant

HONORS AND AWARDS
- Research Challenge Trust Fund, 2018-2019, University of Kentucky
- Robert Lipmann Research Fellowship, 2018, University of Kentucky
- RSA Student Merit Award for the 41st annual Research Society on Alcoholism Scientific Meeting, 2018
- Sayons Award Recipient, Psi Chi Award, 2017, Saint Louis University
- Dean's List, Spring 2014, Fall 2014, Spring 2016, Fall 2016, Spring 2017, Saint Louis University
- Dean's Scholarship, 2013-2017, Saint Louis University
- Enhanced Merit Scholarship, 2013-2017, Saint Louis University

MEMBERSHIPS
- Association for Psychological Science, Student Member
- Research Society on Alcoholism, Student Member
- Alpha Sigma Nu, Jesuit Honor Society, Saint Louis University
- Omicron Delta Kappa, Leadership Honor Society, Saint Louis University
- Psi Chi, Psychology Honor Society, Saint Louis University

PUBLICATIONS

D'Agostino, A. R., Wesley, M. J., Brown, J., & Fillmore, M. T. (2019). Effects of multisensory stop signals on alcohol-induced disinhibition in adults with ADHD. *Experimental and Clinical Psychopharmacology*. Advance online publication.

D'Agostino, A. R., Peterson, S. J., & Smith, G. T. (2019). A risk model for addictive behaviors in adolescents: From the transdiagnostic to the behavior-specific. *Addiction.* Advance online publication.

D'Agostino, A. R., Optican, A. R., Sowles, S. J., Krauss, M. J., Lee, K. E., & Cavazos-Rehg, P. A. (2017). Social networking online to recover from opioid use disorder: A study of community interactions. *Drug and Alcohol Dependence, 181,* 5-10.

Davis, H.A., Ortiz, A. M., **D'Agostino, A. R.,** & Smith, G. T. (2016). A two-stage risk model for bulimic behavior. In N. Columbus (Ed.), *Eating Disorders: Prevalence, Risk Factors, and Treatment Options.* New York: Nova Science Publishers.

Weinstock, J., Mulhauser, K. R. W., Oremus, E. G., & **D'Agostino, A. R.** (2016). Demand for gambling: Development and assessment of a Gambling Purchase Task. *International Gambling Studies, 16,* 316-327.

PRESENTATIONS

D'Agostino, A., Allen, H., & Fillmore, M. (June, 2019). *Impairing effects of alcohol when responding to redundant signals of the same versus different modalities.* Poster to be presented at the 42nd annual Research Society on Alcoholism Scientific Meeting, Minneapolis, MN.

D'Agostino, A., & Fillmore, M. (June, 2018). *Using redundant, duplicate signals to reduce alcohol impairment.* Poster presented at the 41st annual Research Society on Alcoholism Scientific Meeting, San Diego, CA.

D'Agostino, A., Peterson, S., & Smith, G. (June, 2018). *A risk model for addictive behaviors in adolescents: From the transdiagnostic to the behavior-specific.* Poster presented at the 41st annual Research Society on Alcoholism Scientific Meeting, San Diego, CA.

D'Agostino, A., Sowles, S., Krauss, M., Escobar, K., & Cavazos-Rehg, P. (November, 2016). *Understanding online peer support through OpiatesRecovery: A subreddit for individuals addicted to opiates.* Poster presented at the 9th annual Institute for Public Health Conference, St. Louis, MO.

Escobar, K., Sowles, S., Krauss, M., **D'Agostino, A.,** & Cavazos-Rehg, P. (November, 2016). *The role of OpiatesRecovery, a subreddit online community for individuals with opioid use disorder.* Poster presented at the 9th annual Institute for Public Health Conference, St. Louis, MO.

Baxley, C., **D'Agostino, A.,** Herman, A., & Weinstock, J. (October, 2016). *Exploring anxiety sensitivity in gambling disorder.* Poster presented at the 50th annual meeting of the Association for Behavioral and Cognitive Therapies, New York, NY.

D'Agostino, A., Rice, R., Masson, M., & Bekavac, N. (May, 2016). *The effects of mood induction and personality traits on impulsivity.* Poster presented at the Sigma Xi Research Symposium, St. Louis, MO.

D'Agostino, A., Rice, R., Masson, M., & Bekavac, N. (April, 2016). *The effects of mood induction and personality traits on impulsivity.* Poster presented at the 13th annual Department of Psychology Capstone Symposium, St. Louis, MO.

Oremus, E., Mulhauser, K., **D'Agostino, A.,** & Weinstock, J. (November, 2015). *Expanding alcohol demand construct to gambling: Does it translate?* Poster presented at the 49th annual convention of the Association for Behavioral and Cognitive Therapies, Chicago, IL.