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Can the use of multiple stop signals reduce the disinhibiting effects of alcohol?

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Abstract

Background—Research has consistently demonstrated that alcohol impairs the ability to divide attention across two or more stimuli. However, under certain circumstances, the presentation of multiple stimuli can actually facilitate performance. The “redundant signal effect” (RSE) refers to the phenomenon by which individuals respond more quickly and accurately when information is presented as redundant, bimodal stimuli (e.g., visually and aurally), rather than as a single stimulus presented to either modality alone. Recent work has shown that response time (RT) to redundant signals is hastened under alcohol, ameliorating the slowing effects of the drug. However, no research has examined whether RSE can reduce the impairing effects of alcohol on the ability to inhibit behavior.

Methods—This study examined whether the impairing effects of alcohol on inhibitory control might be altered by the presentation of redundant inhibitory signals. Inhibitory control was assessed by a go/no-go task which included single and redundant inhibitory signals. Performance was tested following placebo (0.0 g/kg) and alcohol (0.65 g/kg). The effect of redundant activation signals on alcohol impairment of response activation was also measured.

Results—The results showed evidence for RSE on the activation of behavior, but not for inhibitory control. Compared with placebo, alcohol slowed RT and reduced response inhibition. Redundant signals had a robust speeding effect on RT, even following alcohol. By contrast, redundant signals failed to improve inhibitory control following placebo or alcohol.

Conclusions—These findings have important implications for understanding how drinkers respond to multimodal signals in their everyday environments and highlight the vulnerability of inhibitory control to alcohol’s impairing effects.

Keywords

alcohol; redundant signal effect; inhibitory control; behavioral impairment

Introduction

Considerable laboratory research indicates that alcohol impairs a range of skills relevant for everyday activities. Alcohol has been shown to slow simple and complex reaction time, decrease steadiness, impair motor coordination, and reduce the ability to inhibit action

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Disclosure

Dr. Fillmore and Melissa Miller designed the study, and Melissa Miller wrote the protocol. Melissa Miller oversaw the data collection and coding. Dr. Fillmore and Melissa Miller managed the literature searches and summaries of previous related work, and Melissa Miller undertook the statistical analysis. Dr. Fillmore and Melissa Miller wrote the manuscript. Both authors contributed to and have approved the final manuscript.

(Fillmore, 2007; Holloway, 1995; Laberg and Loberg, 1989). A general determinant of the magnitude of alcohol impairment is the drinker's blood alcohol concentration (BAC) at the time of testing, with impairment increasing as a function of BAC (Holloway, 1995). However, other factors also contribute to the degree of alcohol impairment observed. Intensity of alcohol impairment also depends on the demands of the behavioral functions being examined. Indeed, alcohol impairment intensifies as a function of task demand and complexity (Maylor et al., 1992). Activities that are the most sensitive to the impairing effects of alcohol are those that require drinkers to divide their attention across multiple stimuli (e.g., Fillmore, 2007; Steele and Josephs, 1990). For example, some divided attention tasks require individuals to respond to information presented visually while engaged in a simultaneous listening task. It has been shown that performance on such tasks is more sensitive to the disruptive effects of alcohol compared with performance on simpler tasks with fewer demands. In fact, divided attention performance can be significantly impaired at BACs as low as 20 mg/100 ml (Holloway, 1995; Moskowitz and Robinson, 1998).

Despite evidence that the impairing effects of alcohol can be especially pronounced in environments that require dividing attention across multiple stimuli, some circumstances have been identified whereby the presentation of multiple stimuli can actually facilitate performance. The "redundant signal effect" (RSE) refers to the phenomenon by which individuals respond more quickly and accurately when information is presented as redundant stimuli (e.g., stimuli simultaneously presented aurally and visually), rather than as a single stimulus presented to either modality alone (Todd, 1912). RSE has most commonly been examined in studies of reaction time (RT) and response accuracy (e.g., Gondan et al., 2005; Kim et al., 2012; Miller, 1982). In these studies, participants are required to respond quickly to information presented as either a visual (e.g., a color) or an auditory stimulus (e.g., a tone). RTs to these individual stimuli are then compared with RT to the stimuli when presented simultaneously, as bimodal, redundant signals. Findings indicate that RT to bimodal, redundant signals is faster than RT to either of the single-modal signals. These findings are somewhat counterintuitive given that performance is typically hindered when attention is divided across two modalities. However, the ability to detect and respond to features of a stimulus is markedly enhanced when information about the stimulus is derived simultaneously from more than one sensory input. Although the neurophysiological mechanisms underlying the redundant signal effect are not entirely clear, the effect appears to involve specialized multisensory neurons in the superior colliculus and association cortex that allow for intersensory activation between the visual and auditory channels at some level of processing prior to responding (e.g., Cavina-Pratesi et al., 2001; Miller, 1986; Mordkoff and Yantis, 1991; Schroger and Widmann, 1998; Stein, 1998).

Given evidence that responses are facilitated by redundant signals, it might be the case that redundant signals can also ameliorate the slowing effects of alcohol on RT. To explore this possibility, we examined the extent to which redundant signals reduced the slowing effects of alcohol on RT in healthy adults (Fillmore, 2010). Participants performed a two-choice RT 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 three alcohol doses: 0.65 g/kg, 0.45 g/kg and placebo (0.0 g/kg). Redundant signals produced faster RT compared with either of the unimodal signals. Alcohol slowed RT to all stimuli. However, the speed advantage produced by the redundant stimuli was maintained at BACs above 80 mg/100 ml. Evidence for an RT advantage to bimodal stimuli under alcohol is important because it challenges the assumption that alcohol impairment is intensified in multi-stimulus environments.

To date, the ability of redundant signals to reduce impairment under alcohol has only been examined with regard to behaviors that involve the execution of actions (e.g., RT, response accuracy). However, alcohol is also well-recognized for its impairing effects on response inhibition. Stop-signal and cued-go/no-go tasks have been used to examine the ability to inhibit prepotent (i.e., instigated) responses (Logan, 1994; Logan and Cowan, 1984; Miller et al., 1991). These tasks require participants to quickly respond to a go signal and to inhibit a response when a stop or no-go signal is presented. Alcohol studies using these tasks find that the drug reliably increases failures to inhibit responses to stop-signals in a dose-dependent manner (Fillmore et al., 2005; Marczinski and Fillmore, 2003). Moreover, alcohol-induced disruptions of inhibitory control have been linked to risky behaviors such as excessive binge drinking in humans and laboratory animals (Poulos et al., 1998; Weafer and Fillmore, 2008). As such, it is important to determine whether redundant signals might potentially reduce the impairing effects of alcohol on the ability to inhibit action.

There is some evidence that redundant inhibitory signals can enhance inhibitory control in the sober state (i.e., Cavina-Pratesi et al., 2001; Gondan et al., 2005; 2010). However, the possibility that redundant signals can ameliorate the impairing effects of alcohol on inhibitory control is uncertain. Inhibitory control appears especially vulnerable to the disruptive effects of alcohol. For instance, studies show that inhibitory control is impaired at low BACs that do not slow RT (de Wit et al., 2000; Fillmore and Vogel-Sprott, 1999). Alcohol-induced impairments of inhibition also persist longer following a dose than other behaviorally impairing effects, and drinkers show little tolerance to the disinhibiting effects of the drug despite a history of heavy drinking (Fillmore and Vogel-Sprott, 1995; Fillmore et al., 2005; Miller et al., 2012). Such vulnerability to alcohol impairment raises questions about whether redundant signals can improve response inhibition under alcohol to the same extent that they enhance response activation under the drug.

Drinkers encounter rich stimuli in their environments which require them to process multi-sensory signals that direct behavior. Thus, the present study provides a laboratory analysis of how drinkers respond to bimodal stimuli by examining whether the impairing effects of alcohol on inhibitory control might be altered by the presentation of redundant inhibitory signals. The effect of redundant inhibitory signals was tested by comparing response inhibition to a visual no-go signal presented alone or accompanied by a redundant auditory no-go signal. To test the possibility that redundant inhibitory signals could strengthen inhibition and reduce the disinhibiting effects of alcohol, performance was tested following both placebo (0.0 g/kg) and a moderate dose of alcohol (0.65 g/kg alcohol). The effect of redundant activation signals on alcohol impairment of response activation was also assessed in the study.

Materials and Methods

Participants

Fifty-six adults between the ages of 21 and 33 (mean age = 23.1, $SD = 3.0$) participated in this study. Volunteers were recruited by flyers, posters, and newspaper advertisements seeking adults for studies of the effects of alcohol on cognitive functions. Volunteers were screened using health questionnaires and a medical history interview. Those who reported any contraindication to alcohol, impaired cardiovascular functioning, seizure, head trauma, central nervous system (CNS) tumors, or past histories of psychiatric disorder (i.e., Axis I, *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV]*) were excluded from participation. Those who reported alcohol dependence, as determined by a score of 5 or higher on the Short-Michigan Alcoholism Screening Test (S-MAST; Selzer et al., 1975) were also excluded. Any other high-risk indicators of alcohol dependence,

including prior treatment for an alcohol use disorder or conviction for driving under the influence also precluded participation.

Volunteers had to report drinking at least once per month in an amount of at least two drinks to participate. With regard to other drug use, the majority of the sample reported using caffeine ($n = 47$). Thirteen participants reported smoking cigarettes in the amount of less than a pack of cigarettes a day. Nine reported some past month use of marijuana. No other drug use in the past month, including stimulants, opiates, or cocaine, was reported. Participants were in good health with no contraindications to drinking. The University of Kentucky Medical Institutional Review Board approved the study, and participants received \$85.

Cued Response Inhibition Task

Response inhibition was measured using a computerized cued go/no-go model used in previous research (e.g., Fillmore et al., 2005; Marczinski and Fillmore, 2003) and was operated by E-Prime experiment generation software (Schneider et al., 2002). A trial began with a fixation point (+) for 800 ms, followed by a blank screen for 500 ms. A rectangular-shaped cue was then displayed for one of four randomly occurring stimulus onset asynchronies (SOAs = 100, 200, 400, and 800 ms) before a go or no-go target appeared for 1000 ms. If the rectangle turned green (go target) subjects were to make a computer key press as quickly as possible. If the rectangle turned blue (no-go target) they were to inhibit a response. A test consisted of 250 trials with 700 ms inter-trial intervals and required 20 minutes to complete.

The orientation of the rectangular cue signaled the probability that a go or no-go target would appear. A vertically-oriented rectangle (height = 7.5 cm, width = 2.5 cm) turned green on 80% of the trials and turned blue on 20% of the trials. A horizontally-oriented rectangle (height = 2.5 cm, width = 7.5 cm) turned green on 20% of the trials and turned blue on 80% of the trials. Therefore, vertical and horizontal-oriented rectangles operated as go and no-go cues, respectively. The measure of interest was the proportion (p) of inhibition failures to no-go targets in the go cue condition. Presentation of the go cue increases response preparation (i.e., produces a response prepotency), 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 and Fillmore, 2003), and poorer inhibitory control is indicated by greater p -inhibition failures (i.e., disinhibition).

There were two versions of this task containing either single or redundant no-go targets. For the single version, the signal to inhibit a response was the single, visual stimulus (the color blue), as described above. In the redundant version, the no-go target (blue) was always coupled with the simultaneous presentation of a brief 1200 Hz auditory tone.

Cued Response Activation Task

RT was measured by a simple cued response task operated by E-prime Experiment Generation software (Schneider, et al., 2002). Participants first saw a rectangular shaped cue that was displayed for one of four randomly occurring SOAs (100, 200, 400, or 800 ms). On half the trials, the cue turned green for 1000 ms, followed by a 700 ms inter-trial interval. Participants were instructed to respond as quickly as possible to the target by pressing the forward slash (/) key. The orientation of the cue (upright vs. flat) signaled the probability that the target would appear on a given trial. An upright cue (valid cue) correctly signaled the onset of the target on 80% of the trials. Thus, valid cues allowed participants to prepare to respond, which speeds RT. The target followed the flat cue on only 20% of the trials (invalid cue). On these trials, participants are unprepared to respond, which slows their RT

to the unexpected appearance of the target. Response activation was measured as the mean RT to targets in this invalid cue condition because RT to non-cued stimuli is more sensitive to alcohol's slowing effects on behavior than responses to the cued stimuli (Marczinski and Fillmore, 2003). A test was comprised of 250 trials and required 20 minutes to complete. Responses less than 100 ms and greater than 1000 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).

There were two versions of this task containing either single or redundant go targets. For the single version, the signal to activate a response was a single, visual stimulus (the color green), as described above. In the redundant version, the signal (green) was always coupled with the simultaneous presentation of a brief 1200 Hz auditory tone.

Timeline follow-back

The timeline follow-back (TLFB; Sobell and Sobell, 1992) assessed the typical quantity and frequency of weekly drinking over the past 3 months. Two measures of drinking habits were obtained: (1) frequency; the average number of drinking occasions per week, and (2) quantity; the average number of standard drinks per drinking occasion.

Subjective Intoxication

Participants rated their degree of subjective intoxication on a visual analog scale by placing a vertical line at the point representing the extent to which they "feel intoxicated" on a 100-mm horizontal line ranging from 0 mm "not at all" to 100 mm "very much."

Procedure

Participants were individually tested in the Behavioral Pharmacology Laboratory of the Department of Psychology between 10 a.m. and 6 p.m. Sessions were scheduled at least 24 hours apart and were completed within two weeks. Participants were instructed to fast for four hours prior to each alcohol session, and to refrain from consuming alcohol or any psychoactive drugs for at least 24 hours before sessions. Prior to each session, subjects provided urine samples that were tested for drug metabolites including amphetamine, barbiturates, benzodiazepines, cocaine, opiates, and tetrahydrocannabinol (On Trak Teststiks, Roche Diagnostics Corporation, Indianapolis, IN, USA) and in women, human chorionic gonadotropin (hCG hormone), to verify that they were not pregnant (Mainline Confirms HGL, Mainline Technology, Ann Arbor, MI, USA). Any participants who tested positive for recent drug use or for pregnancy were excluded from participating. Breath samples were also provided at the beginning of each session to verify a zero BAC.

Familiarization session—After providing informed consent, subjects provided proof of age to verify that they were at least 21 years old. They completed questionnaires concerning health status, drinking habits, and demographic characteristics. Half of the participants ($n = 28$) were assigned to complete the cued go/no-go task and the other half were tested on the cued response activation task. For each of these test conditions, half of the participants ($n = 14$) were tested in the single condition, and the other half were tested in the redundant condition. Assignment to task and condition was random with the constraint that each of the four groups was comprised of an equal number of 7 men and 7 women. Participants performed a familiarization test in their respective conditions.

Test Sessions—Performance was tested under two doses of alcohol: 0.0 g/kg (placebo) and 0.65 g/kg. Doses were administered during separate test sessions, and dose order was counterbalanced across participants. Sessions were separated by a minimum of one day and a maximum of one week. The alcohol dose was calculated on the basis of body weight and

administered as absolute alcohol mixed with three parts carbonated soda. The placebo dose (0.0 g/kg) consisted of a volume of carbonated mix that matched the total volume of the 0.65 g/kg alcohol drink. A small amount (3 ml) of alcohol was floated on the surface of the beverage. It was sprayed with an alcohol mist that resembled condensation and provided a strong alcoholic scent as the beverage was consumed. In similar studies, individuals report that this beverage contains alcohol (Fillmore and Vogel-Sprott, 1998). Drinks were consumed in six minutes. Following 0.65 g/kg alcohol, a peak BAC of 80 mg/100 ml was expected to occur approximately 60 minutes after drinking (Fillmore and Vogel-Sprott, 1998).

Testing occurred on the ascending limb of the BAC curve. Task performance was tested 40 minutes after drinking began, and concluded at 60 minutes post-drinking (i.e., near the peak BAC following the active dose), at which point participants completed the subjective intoxication ratings. BACs were measured at 40 and 60 minutes post-drinking (i.e., before and after testing and completion of subjective intoxication ratings). Breath samples were also obtained at these times during the placebo session ostensibly to measure BAC. After testing, participants received a meal and were released once their BAC fell below 20 mg/100 ml.

Data Analyses

For the two groups tested on the cued response inhibition task, a 2 Dose (0.0 g/kg vs. 0.65 g/kg) X 2 Target Condition (single vs. redundant) ANOVA of p-inhibition failures tested the effects of alcohol and target condition on their inhibitory control. For the two groups tested on the cued activation task, the effects of dose and target condition were examined by a 2 (Dose) X 2 (Target Condition) ANOVA of their RT scores. For both behavioral inhibition and activation, the effect of alcohol in each target condition was tested by planned comparison *t* tests contrasting performance under alcohol to performance following placebo. Initially, all analyses were conducted with gender as a factor. There were no significant main effects or interactions involving gender for either inhibitory failures or RT. Therefore, all analyses excluded gender as a factor.

Results

Demographics and drinking habits

Table 1 presents the ages and drinking habits for participants in each of the four target condition groups. A one-way ANOVA revealed no significant differences in age among the four groups, $F(3, 52) = 1.15$, $p = 0.34$. The mean age for the entire sample was 23.1 ($SD = 3.02$) years. With regard to drinking habits, a one-way ANOVA showed no target condition group differences in the weekly frequency, $F(3, 52) = 0.60$, $p = 0.62$, or quantity of consumption, $F(3, 52) = 1.14$, $p = 0.34$. For the entire sample, mean weekly frequency of drinking was 1.99 ($SD = 1.22$), and the mean typical quantity per occasion was 4.67 drinks ($SD = 2.28$).

Blood Alcohol Concentrations

A 4 (target condition group) X 2 (time) ANOVA of BACs following 0.65 g/kg alcohol revealed no significant main effect of group, or a group X time interaction ($ps > 0.14$). A main effect of time was obtained due to the rise of BACs during the session, $F(1, 52) = 45.39$, $p < 0.001$. The entire sample's mean BACs at 40 and 60 min post-drinking were 83.1 mg/100 ml ($SD = 18.94$) and 93.7 mg/100 ml ($SD = 20.33$), respectively. No detectable BAC was observed following placebo administrations.

Cued Response Inhibition

Figure 1 shows the mean p-inhibition failures to the single and redundant targets on the cued go/no-go task. The 2 (dose) X 2 (target condition) ANOVA revealed a significant main effect of dose, $F(1, 26) = 14.04, p < 0.01, \eta^2 = 0.33$. There was no significant effect of target condition, $F(1, 26) = 0.08, p = 0.93, \eta^2 = 0.01$, or a dose X condition interaction, $F(1, 26) = 2.95, p = 0.10, \eta^2 = 0.07$. Planned comparison tests showed that compared with placebo, alcohol significantly increased p-inhibition failures in the redundant target condition, $t(13) = 3.11, p < 0.01, d = 0.83$, and in the single target condition, $t(13) = 2.12, p < 0.05, d = 0.56$.

Cued Response Activation

Figure 2 shows the mean RTs in the single and redundant target conditions in the cued response activation task. A 2 (dose) X 2 (target condition) ANOVA revealed significant main effects of dose, $F(1, 26) = 15.24, p < 0.01, \eta^2 = 0.21$, and target condition, $F(1, 26) = 59.92, p < 0.001, \eta^2 = 0.72$. No significant interaction was observed, $F(1, 26) = 2.59, p = 0.12, \eta^2 = 0.01$. Planned comparisons showed that alcohol significantly slowed RT compared with placebo in both the single, $t(13) = 3.03, p < 0.01, d = 0.34$, and the redundant conditions, $t(13) = 2.78, p < 0.05, d = 0.74$. Two sample t tests comparing the target conditions revealed significantly faster RTs in the redundant condition following both placebo, $t(26) = 8.03, p < 0.001, d = 3.00$ and alcohol, $t(26) = 6.66, p < 0.001, d = 2.52$.

Subjective intoxication

A 4 (target condition) X 2 (dose) ANOVA of subjective intoxication ratings revealed no significant main effect of target condition, or a condition X dose interaction ($ps > 0.25$). A main effect of dose was obtained due to higher ratings of intoxication in response to 0.65 g/kg alcohol compared with placebo, $F(1, 52) = 212.57, p < 0.001, \eta^2 = 0.79$. For the entire sample, the mean ratings of subjective intoxication following placebo and alcohol were 12.0 ($SD = 14.9$) and 57.0 ($SD = 20.9$), respectively.

Discussion

The present study sought to determine whether redundant stimuli might reduce the impairing effects of alcohol on response inhibition and activation. Previous work has shown that redundant activation signals can improve the speed and accuracy of responding and that such facilitation can be observed following alcohol (e.g., Fillmore, 2010). Indeed, in the current study, drinkers responded more quickly to redundant, visual-auditory activation signals compared with single signals. Moreover, although alcohol slowed RT in both redundant and single target conditions, the RT speed-advantage in the redundant condition was maintained even under 0.65 g/kg. In fact, RT in the redundant condition was considerably faster than RT in single target condition, regardless of the dose condition. Thus, redundant signals had a robust facilitating effect on RT even following a dose of alcohol which was sufficient to impair (i.e., slow) RT.

The study also showed that alcohol impaired inhibitory control by increasing inhibitory failures in both the single and redundant target conditions. However, unlike RT, redundant signals did not enhance inhibitory control in either the sober or intoxicated states. In fact, the magnitude of alcohol impairment in the redundant condition was larger than the degree of impairment in the single target condition. Thus, not only did redundant signals fail to improve inhibitory control, but they may possibly contribute to greater alcohol impairment of inhibition compared with single inhibitory signals.

To date, the majority of research on the redundant signal effect has focused on the execution of actions (e.g., speeding RT). Although a few studies of response inhibition have shown facilitating effects of redundant signals in sober adults (i.e., Cavina-Pratesi et al., 2001; Gondan et al., 2005), no research has explored the possibility that redundant signals could ameliorate alcohol-induced deficits of inhibitory control. The current finding that redundant inhibitory signals do not reduce impairment under alcohol is contrary to findings on response execution and raises questions about why redundant inhibitory signals failed to reduce the disinhibiting effects of alcohol.

A possible explanation for this finding concerns alcohol's effects on information processing capacity. Evidence suggests that alcohol impairs behavioral control by reducing the drinker's capacity to process information from multiple sources, particularly when the information signals that behaviors should be inhibited (Bartholow et al., 2003; Fillmore and Van Selst, 2002; Medina, 1970; Moskowitz and De Pre, 1968; Steele and Southwick, 1985). Any alcohol-induced capacity limitation in the present study could have limited the ability to effectively integrate information, especially when the information is presented to two or more modalities (visually and aurally). Such an account raises the possibility that redundant inhibitory signals could actually ameliorate alcohol impairment if presented to the same modality (e.g., two visual signals), thereby placing less demand on information processing. Indeed, RSE in the execution of responses is often demonstrated using redundant signals within the same modality (Marzi et al., 1996; Murray et al., 2001). A logical next step in this new area of research is to test the possibility that such single-modal redundant signals could improve inhibitory control, particularly under alcohol.

It should be noted that this study did not examine responses to "go" and "no-go" signals that were presented as an auditory stimulus alone. There were two reasons for this omission. First, responses to simple auditory stimuli do not differ from responses to simple visual stimuli in these types of tasks. Studies of response activation find that RTs to auditory stimuli are similar to visual stimuli, and that redundant signals show comparable improvement over single signals regardless of their modality (Cheng et al., 2010; Fillmore, 2010). Second, the tasks used in the study examined response inhibition and activation using a cued response model in which an initial stimulus (e.g., a rectangle) provided preliminary information that a specific response would be required on a given trial. This allowed us to examine inhibitory control when there was a response prepotency by first presenting a go cue following by a no-go signal. The cues were visual stimuli (i.e., rectangular shapes in one of two orientations). Visual go cues generally do not increase the pre-potency of responses to auditory signals (Miller et al., 1991). Thus, to ensure the response prepotency effects across target condition, we compared only target conditions that involved visual signals (visual and visual + auditory).

Additionally, this study only examined the effect of redundant signals on alcohol impairment of behavioral effects on the ascending limb of the BAC curve. We know, however, that the magnitude of impairment observed on the descending limb is not always the same as that on the ascending limb, even when BACs are comparable. Indeed, studies examining acute tolerance to the impairing effects of alcohol compare performance on tasks at comparable BACs on the ascending and descending limb. Acute tolerance is observed as a reduction in alcohol impairment on the descending limb of the BAC curve compared with the ascending limb, and has been shown to develop for several behaviors, including reaction time (i.e., Fillmore et al., 2005; Fillmore and Vogel-Sprott, 1996). However, several studies have failed to observe the development of acute alcohol tolerance for measures of inhibitory control (e.g., Fillmore et al., 2005; Ostling and Fillmore, 2010; Weafer and Fillmore, 2012). Given that drinkers will encounter multi-sensory demands even as BACs decline, it is also

important to consider how redundant signals might affect alcohol impairment of activation and inhibitory mechanisms on the descending limb of the BAC curve.

Finally, it is important to consider the ecological relevance of studying drug effects in the context of redundant environmental signals. Common technologies (e.g., cars, navigation systems, phones, and computers) are becoming increasingly complex in their ability to deliver information to the user. One aspect of this complexity concerns the ability of these devices to provide redundant information to two or more modalities (e.g., visual readouts accompanied by verbal prompts and/or information). The tacit assumption is that such redundant information should have facilitating effects on behavior. However, little is known about how such redundant information affects behaviors in the drugged state, when information processing capacity is compromised in some manner. Indeed, the present findings suggest that acts of control, such of the inhibition of behavior, could be disrupted by such redundant information when an individual is intoxicated. As such, it is important to understand how alcohol and other drugs affect not only simple stimulus-response behaviors, but also the ability to execute behavioral control in contexts where information is presented redundantly to two or more modalities. The present study provides a useful model to begin such research.

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Figure 1.

Mean p-failures on the go/no-go task following 0.0 g/kg (placebo) and 0.65 g/kg alcohol for those in the single and redundant inhibitory signal groups. Capped vertical lines show *SEMs*. Asterisks indicates significant difference in p-failures under alcohol compared with placebo, $p < 0.01$.

Figure 2. Mean RT in milliseconds on the cued response activation task following 0.0 g/kg (placebo) and 0.65 g/kg alcohol for those in the single and redundant activation signal groups. Capped vertical lines show *SEMs*. Asterisks indicate significant difference in RT under alcohol compared with placebo, $ps < 0.001$.

Descriptive statistics and drinking habits for the groups of participants who completed the single and redundant target conditions in the go/no-go and cued activation tasks, n= 14 per group.

Table 1

Target condition	Go/No-Go Task		Cued Activation Task	
	Single	Redundant	Single	Redundant
Men: Women	7:7	7:7	7:7	7:7
	M	(SD)	M	(SD)
Age (years)	22.6	(3.3)	22.1	(1.5)
			24.0	(3.3)
Weekly drinking frequency	1.7	(1.0)	1.8	(0.9)
			2.3	(1.6)
Typical drinking quantity	4.7	(2.3)	3.7	(1.2)
			5.2	(2.8)
SMAST score	0.9	(1.9)	0.1	(0.5)
			0.3	(0.7)
			0.3	(1.1)