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Alcohol Effects on Response Inhibition: Variability Across Tasks and Individuals

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Considerable research has investigated the acute effects of alcohol on response inhibition, but a number of issues remain unresolved. Given that most studies use only a single laboratory task to assess inhibition, it is often difficult to determine whether alcohol's effects are task specific or generalize across measures of the same construct. Moreover, relatively few studies have directly compared effects of alcohol under ascending and descending blood alcohol concentrations (BACs), and those that have often failed to disentangle BAC limb effects from the effects of repeated testing. This study was intended to provide a test of alcohol's effects on behavioral inhibition using multiple laboratory measures in a relatively large sample and comparing effects under ascending and descending BAC. Young adults (N = 216) completed three commonly used inhibition tasks (Stroop, antisaccade, and stop-signal) at baseline and again 1–3 weeks later under one of three beverage conditions (alcohol, placebo or control) and one of two BAC limb conditions (ascending/descending or descending only). Findings indicated considerable specificity in alcohol's effects. Relative to control and placebo conditions, antisaccade performance suffered under both ascending and descending BAC and stop-signal reaction time (RT) suffered only under descending BAC. The Stroop RT interference effect was not affected by alcohol, though alcohol did impair response accuracy on incongruent Stroop trials. Baseline performance moderated effects of alcohol on both antisaccade accuracy and Stroop interference, suggesting the importance of individual differences. The current findings suggest that more specificity is required in characterizing acute effects of alcohol on inhibitory control.

Public Health Significance
Drinking alcohol is thought to impair the control of responses. This study demonstrates that alcohol's effects on inhibition, or the ability to stop a response, can depend on the laboratory tasks used to

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measure it. This finding is generally consistent with previous studies that have shown variability in alcohol's effects, and suggests that researchers must do a better job specifying conditions under which alcohol will and will not impair inhibition.

Keywords: alcohol, inhibition, executive functioning, repeated assessment, BAC limb

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Alcohol consumption adversely affects behavior in numerous domains (see Fillmore & Vogel-Sprott, 2006). Several theoretical models (e.g., Giancola, 2000; Lyvers, 2000) posit that such effects are mediated by alcohol's impairment of executive functions (EFs), a set of higher-order cognitive abilities involved in the planning, initiation and regulation of goal-directed behavior (see Banich, 2009; Miyake & Friedman, 2012). But EF is neither a single nor a simple construct; therefore, models positing that alcohol impairs EF are likely oversimplified and tests of this general hypothesis are often underspecified. The purpose of this research was to begin specifying alcohol's effects on EF within the context of a multivariate, correlated factors model (Miyake et al., 2000), focusing in the current study on response inhibition.

Limitations in the Existing Literature Examining Alcohol Effects on Inhibition

Virtually all models agree that inhibition—the ability to withhold a prepotent but goal-irrelevant response—is a critical feature of EF (e.g., see Banich, 2009; Diamond, 2013; Milner & Petrides, 1984; Miyake et al., 2000). Moreover, although considerable variability has been reported in alcohol's effects on EF (e.g., Dougherty, Marsh-Richard, Hatzis, Nouvion, & Mathias, 2008; Dry, Burns, Nettelbeck, Farquharson, & White, 2012; Lyvers & Maltzman, 1991; Lyvers & Tobias-Webb, 2010), the most consistent finding in this literature is that alcohol impairs inhibition (for reviews see Day, Kahler, Ahern, & Clark, 2015; Noël, Tomberg, Verbanck, & Campanella, 2010; Weafer & Fillmore, 2016). Weak inhibitory control in adolescence has been associated with greater substance use (Gustavson et al., 2017; Young et al., 2009). Characterizing alcohol's acute effects on inhibition is important because of its implications for understanding so-called "loss-of-control" drinking (see Field, Wiers, Christiansen, Fillmore, & Verster, 2010), which can facilitate progression from casual to disordered use (Weafer & Fillmore, 2008).

Even so, alcohol's acute effects on inhibition as reported in the scientific literature are far from uniform. Some of this variability can be attributed to differences in the tasks used to assess inhibition in the laboratory (Weafer & Fillmore, 2016), which may tap different and distinguishable types of inhibitory or interference control processes postulated in the literature (e.g., Friedman & Miyake, 2004; Jacob et al., 2010). In addition, existing studies have suffered from a number of limitations. First, most, though not all, studies have used only a single measure of inhibition, such as versions of a go/no-go task (see Marczinski & Fillmore, 2005; Miller & Fillmore, 2014; Miller, Hays, & Fillmore, 2012; Tsujii, Sakatani, Nakashima, Igarashi, & Katayama, 2011; Weafer & Fillmore, 2008), making it difficult to generalize beyond the specific task in question and how it was administered in a given study. This issue is important because laboratory measures of EF—and inhibition measures in particular—often suffer from low reliability (Burgess, 1997; Stuss & Alexander, 2000) and the so-called *task impurity* problem, wherein performance reflects not only the target EF ability but also other task-specific processes (Miyake et al., 2000). Given these concerns and the low intercorrelations among different inhibition tasks often observed in the literature, some scholars have even questioned the viability of "inhibition" as a psychometric construct (see MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003; Rey-Mermet, Gade, & Oberauer, 2017). Thus, acute alcohol effects on a given task cannot be assumed to generalize to other tasks or even to the latent construct of "inhibition" more generally.

Second, sample sizes are often modest, with most studies involving fewer than 20 participants per condition (e.g., Abroms, Gottlob, & Fillmore, 2006; Bombeke, Schouppe, Duthoo, & Notebaert, 2013; Caswell, Morgan, & Duka, 2013; Claus & Hendershot, 2015; Easdon, Izenberg, Armilio, Yu, & Alain, 2005; Easdon & Vogel-Sprott, 2000; Fillmore & Vogel-Sprott, 2000; Marczinski & Fillmore, 2005; Mulvihill, Skilling, & Vogel-Sprott, 1997; Rose & Duka, 2008; for a review, see Day et al., 2015). In recent years, researchers increasingly have raised concerns over the reliability of findings based on small samples, with some noting "samples smaller than 20 per cell simply are not powerful enough to detect most effects" (Simmons, Nelson, & Simonsohn, 2011, p. 1363). Together with the prevalence of publication biases against null results (Kühberger, Fritz, & Schnerndl, 2014), this raises the possibility that findings based on small samples represent false-positive effects (also see Button et al., 2013), or at least that the true magnitude of alcohol effects might be poorly estimated when using small samples.

Third, most previous studies have involved only alcohol and placebo conditions but not a true control condition (or alcohol and control but no placebo; e.g., Claus & Hendershot, 2015; McCarthy, Niculete, Treloar, Morris, & Bartholow, 2012). Failure to include all three conditions prevents testing whether any observed effects are pharmacological, expectancy-related, or both (see Testa et al., 2006).

Finally, a fourth limitation of most prior research is failure to model the dynamic nature of alcohol effects (but see, e.g., Blekher et al., 2002; Miller & Fillmore, 2014; Pihl, Paylan, Gentes-Hawn, & Hoaken, 2003; Schweizer et al., 2006). In particular, the biphasic effects of alcohol, in which blood alcohol concentration (BAC) rises to a peak following consumption (i.e., ascending limb [AL]) and then gradually declines to a sober state (i.e., descending limb [DL]), could change the nature of alcohol's effects on inhibition. Relative to comparable BAC on the AL, the DL tends to be associated with reductions in subjective intoxication (Earleywine

& Martin, 1993) and improvements in some aspects of motor control (Miller & Fillmore, 2014; Schweizer et al., 2006), a phenomenon referred to as *acute tolerance* (Vogel-Sprott, 1997). Recent evidence suggests that inhibition remains impaired on the DL (Fillmore, Ostling, Martin, & Kelly, 2009) even when response time and motor coordination have recovered (Miller & Fillmore, 2014). Determining whether DL performance is attributable to practice (i.e., having previously completed the tasks on the AL) versus other aspects of acute tolerance requires a design in which some participants complete cognitive tasks on both the AL and DL and others complete the tasks only on the DL, a design feature rarely seen in alcohol challenge studies.

The Current Study

In sum, determining the extent to which alcohol impairs inhibition is more complicated than it might appear. Although some studies have addressed a subset of the limitations outlined above, no previous study has endeavored to address them all. The current study was designed to address these limitations by assigning relatively large numbers of participants to one of three beverage conditions (alcohol, placebo, and control) and by assessing inhibition under both AL and DL conditions using three tasks that have been used to measure response inhibition in previous large-scale multivariate studies (e.g., Friedman et al., 2008; Ito et al., 2015; Miyake et al., 2000): the stop-signal task, the antisaccade task, and the classic color-word Stroop task. The common feature across these tasks is the requirement to withhold or override prepotent, habitual response tendencies. Yet, the tasks differ in requiring withholding of a response (stop-signal task), production of a response conflicting with an innately favored one (antisaccade task), or production of a response conflicting with a learned habitual one (Stroop task).

Versions of these tasks have been used in a number of previous alcohol challenge studies, with inconsistent results. For example, antisaccade accuracy (i.e., the ability to inhibit directing gaze or attention to a cued location) sometimes has been decreased by alcohol (Abroms et al., 2006; Marinkovic, Rickenbacher, Azma, Artsy, & Lee, 2013; AL or peak BAC), but not always (Blekher et al., 2002 [AL and DL]; Vorstius, Radach, Lang, & Riccardi, 2008 [AL]). In some studies accuracy was *increased* by alcohol (Khan, Ford, Timney, & Everling, 2003 [AL and DL]; Roche & King, 2010 [peak BAC]; Vassallo & Abel, 2002 [AL]). Similarly, the classic Stroop interference effect has been increased by alcohol in some studies (e.g., Curtin & Fairchild, 2003; Rose & Duka, 2007, 2008; all AL), but in several of these the effect has been described as "marginal" (i.e., p values $\approx .07$) or was present in error rates but not response latency on correct trials (Curtin & Fairchild, 2003; Rose & Duka, 2007). Other studies have reported no alcohol effects on Stroop performance (Bombeke et al., 2013; Duka & Townshend, 2004; Gustafson & Källmén, 1990; Marinkovic, Rickenbacher, Azma, & Artsy, 2012 [AL]).

The most consistent alcohol-related deficits have been reported for the stop-signal task (e.g., Caswell et al., 2013; de Wit, Crean, & Richards, 2000; Gan et al., 2014; McCarthy et al., 2012; Mulvihill et al., 1997; Reynolds, Richards, & de Wit, 2006; all AL), but even with this task effects have not been uniform. For example, both Dougherty et al. (2008) and Peacock, Cash, and Bruno (2015) reported no effect of alcohol on stop-signal performance on either the AL or the DL (also see Guillot, Fanning, Bullock, McCloskey, & Berman, 2010; Spinola, Maisto, White, & Huddleson, 2017). In addition, in two studies (both using rather small samples) Loeber and Duka (2009a, 2009b; both AL) reported that stop-signal performance decreased from baseline to postdrink in both placebo and alcohol conditions, but that this decline was marginally worse in the alcohol condition.

To some extent, differential effects of alcohol across these tasks likely are attributable to the substantial amount of task-specific variance, as indicated by their low intercorrelations. For example, across two different samples of adolescents, Khng and Lee (2009, 2014) have reported virtually no correlation between stop-signal RT (SSRT) and the Stroop interference effect (see also Kalanthroff, Goldfarb, & Henik, 2013), leading them to conclude, "it seems unlikely that conventional RT measures from the two tasks measure the same effect" (Khng & Lee, 2014, p. 10). Even within models assuming some fundamental similarity in the underlying process measured by the Stroop, stop-signal and antisaccade (Friedman & Miyake, 2017; Miyake & Friedman, 2012), correlations among these tasks are often modest. For example, Friedman and Miyake (2004) reported bivariate correlations among these three tasks ranging from $r = .15$ (Stroop interference effect with SSRT) to $r = .23$ (Stroop interference effect with antisaccade accuracy; also see Friedman et al., 2008, 2016). Thus, testing effects of alcohol on each of these tasks within the same sample can bring needed clarification concerning the generality versus specificity of alcohol's effects on behavioral inhibition.

With these issues in mind, the current study had two primary aims: (a) to determine whether alcohol has similar effects on performance across three tasks purported to measure response inhibition, and (b) to determine whether alcohol effects on specific tasks differ as a function of limb of the BAC curve. A third aim, afforded by the multisession design of the study, was to investigate whether individual differences in baseline inhibitory ability moderate acute effects of alcohol on inhibition. It is generally assumed that people with stronger inhibitory ability might be less susceptible to alcohol impairment, in that their baseline functioning provides a "buffer" against an acute insult (see Peterson & Pihl, 1990; Wood, Sher, & Bartholow, 2002). Finn, Justus, Mazas, and Steinmetz (1999) provided some support for this idea, in that only individuals relatively low in working memory performance showed alcohol-related impairment during a go/no-go inhibition task. Alternatively, it could be that those with better baseline inhibitory ability have "more to lose" from an acute challenge, in terms of regression toward mean levels of ability, and therefore alcohol might make those with stronger inhibitory ability more like their lower-functioning peers. These possibilities were investigated within the context of the three inhibition tasks used here.

Method

All procedures for this study were approved by the University of Missouri Institutional Review Board (protocol number 1135337, "Alcohol and Executive Cognitive Function: MARC Project 8").

Participants

Two-hundred and 41 young adults (aged 21–30) were recruited from the community for a study on the effects of alcohol on

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cognition, using advertisements placed in online classifieds and mass e-mail announcements sent to university employees and students. Interested individuals were interviewed via telephone to determine their study eligibility. Individuals who self-reported conditions contraindicating participation in an alcohol challenge (e.g., abstention; history of alcohol or drug dependence or other serious mental illness; prescription medication other than oral contraception; pregnancy) or that would make completion of laboratory tasks unusually difficult (e.g., color blindness; a primary language other than English) were excluded from the sample. In addition, to ensure that the alcohol dose received in the study would be within participants' normal range of experience, naive drinkers (<2 drinks per week) and very heavy drinkers (>24 drinks per week; see NIH, 2006) were excluded from the sample. Eligible individuals were scheduled for the first of two laboratory sessions. Participants received $35 for completion of the baseline session and were paid $14/hr for participation in the second session. Twenty-five participants completed Session 1 but failed to return for Session 2. Thus, the final sample included 216 individuals (47% women; M_{age} = 22.7 years).

Sample Demographics and Alcohol Use

Participants were asked to estimate their typical quantity and frequency of alcohol use and heavy drinking (number of binge drinking episodes) over the past 30 days and past 3 months (scored in terms of per-week consumption), using items derived from the recommendations of the National Institute on Alcohol Abuse and Alcoholism Task Force on Recommended Alcohol Questions (NIAAA, 2003). Table 1 presents these data, along with other demographics, as a function of experimental group assignments.

Inhibition Measures

The inhibition measures for this study were chosen on the basis of a unity/diversity model of EFs (Friedman & Miyake, 2017; Miyake & Friedman, 2012), in which individual differences in inhibitory ability are characterized by performance on these tasks. The specific versions of these tasks were adapted from those reported in the Friedman et al. (2016) study. In particular, the number of trials in each administration was considerably reduced to fit the time constraints imposed by the BAC curve.

Stop-signal task. On each of 50 trials of the first (prepotent "go") block, participants focused on a central fixation point until a green arrow appeared, pointing to the right or left (direction was pseudorandomly ordered and equiprobable). Participants were instructed to indicate the arrow's direction as quickly as possible via key press. Next, a stop-signal was introduced during two blocks of 80 trials each. In these blocks, the initial green arrow changed to red on 25% of trials, indicating that a response should be withheld. The time before the arrow turned red (i.e., stop-signal delay) was determined by a staircase-tracking algorithm: Each successfully inhibited response resulted in a 50-ms delay in stop-signal onset on the next stop trial, whereas failed stops resulted in the next signal appearing 50 ms earlier, resulting in participants' correctly inhibiting on roughly 50% of stop trials. Additional blocks were completed by any participant whose stopping fell outside a 40%–60% accuracy range for any block (9% of task runs) until two blocks were completed in which stopping accuracy was within this range. The task took 9 min to complete.

The primary dependent measure derived from the stop-signal task was SSRT (Logan & Cowan, 1984), which estimates the amount of time required to stop an already-initiated response. SSRT was computed as the difference between median RT on go trials (which estimates the time when a response *would have occurred* in the absence of the stop-signal) and the average stop-signal delay (see van den Wildenberg et al., 2006). Larger SSRT values thus indicate that more warning was needed to withhold a response on stop trials.

Antisaccade task. During each trial of the antisaccade task (initially modeled after Roberts et al.'s, 1994 version), a centrally located fixation cross appeared for a variable duration (1,500 ms–3,500 ms, in 250-ms intervals) before being replaced with an initial cue (black square) shown to the left or right of fixation (with equal probability). Next, a numeric target (the digits 1–9) appeared for 150 ms in one hemifield before being masked with gray cross-hatching. Participants' task was to verbally report the target num-

Table 1
Demographic Characteristics and Alcohol Use Variables by Experimental Group

	Experimental group					
	Alcohol		Placebo		Control	
	A/D	D-only	A/D	D-only	A/D	D-only
Participant characteristics	(*n* = 36)	(*n* = 32)	(*n* = 36)	(*n* = 37)	(*n* = 37)	(*n* = 38)
% Male	47.2	59.4	50	58.3	54.1	50
% Caucasian	83.3	84.4	91.9	89.5	83.8	91.9
M age	22.83 (2.00)	23.10 (2.59)	23.07 (1.95)	23.28 (2.50)	23.45 (3.42)	22.73 (2.17)
Drinks per week	8.20 (6.99)	7.09 (6.77)	8.21 (6.87)	7.30 (7.99)	7.88 (6.05)	6.97 (5.63)
Binge drinking	1.31 (1.17)	1.34 (1.26)	1.22 (1.17)	1.11 (1.10)	1.24 (1.06)	1.37 (1.21)

Note. Numbers in parentheses are standard deviations (*SD*s). Drinks per week = average number of drinking occasions per week multiplied by average number of drinks per occasion, estimated over the past 3 months; Binge drinking = mean number of binge episodes (four or more drinks in one occasion for women; five or more for men) per week estimated over the past 3 months; A/D = participants who completed the inhibition tasks twice during Session 2 (during both ascending and descending blood alcohol concentration); D-only = participants who completed the inhibition tasks during Session 2 only during descending blood alcohol concentration. None of the means reported here differed as a function of experimental group.

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ber (recorded by an experimenter). Given the number of possible targets, a vocal response method is preferable because a manual response would require participants to navigate a large number of response buttons, likely interfering with task performance. In the first block (*prosaccade* trials; $n = 25$), targets always appeared in the same hemi-field as the initial visual cue (175-ms postcue presentation). In the subsequent three *antisaccade* blocks ($n = 36$ trials each), participants were instructed to not look at the initial visual cue because the target number would appear on the opposite side of the screen. The cue-to-target interval was fixed within each antisaccade block but decreased by 25 ms across blocks (225 ms, 200 ms, and 175 ms, respectively). The prosaccade and first antisaccade blocks were each preceded by 12 practice trials, and each block contained two "warm up" trials that were not included in the analyses. The task took 11 min to complete. The dependent measure was the proportion of correct responses across the antisaccade blocks.

Stroop task. In the Stroop color-naming task, three types of trials were presented in the following order: (a) one block of 20 neutral trials in which strings of three to five asterisks printed in blue, green, or red were shown; (b) one block of 20 congruent trials where color words were printed in matching font color (e.g., "BLUE" printed in blue); and (c) one block of 60 incongruent trials where color words were printed in nonmatching font color (e.g., "BLUE" printed in green). Based on research showing that the Stroop interference effect occurs primarily in the processing of the verbal response and is considerably larger when using verbal compared with manual responses (MacLeod, 1991; Wheeler, 1977), participants were asked to name the font color aloud into a headset microphone that recorded vocal response latency. This approach maximizes the task's response inhibition requirement; the arbitrariness of response-mapping rules when manual responses are used tends to reduce the task's response inhibition requirement. An experimenter recorded the content of the response to determine accuracy. On each trial, a white fixation cross appeared for 250 ms on a black background followed by the stimulus, which remained until the participant responded. Stimulus lists were pseudorandomly ordered so that three color words (or sets of asterisks) and three stimulus colors occurred with equal probability in each block and no more than three trials in a row would involve the same word or stimulus color. The first two blocks (i.e., asterisk and congruent) were preceded by 10 practice trials each. The task took 6 min to complete.

As in previous studies in which the Stroop has been used to characterize individual differences in inhibitory ability (e.g., Friedman et al., 2008; Ito et al., 2015), the Stroop interference effect was calculated here as the difference between average incongruent trial RT and average asterisk trial RT. In addition, because it has been reported in previous studies that alcohol (relative to placebo or control) increases response errors on incongruent Stroop trials (e.g., Curtin & Fairchild, 2003; Marinkovic et al., 2012; Rose & Duka, 2007, 2008), we also characterized Stroop performance in terms of color-naming accuracy on incongruent trials.

When attempting to characterize individual differences in prepotent response inhibition, presentation of congruent, incongruent, and neutral (asterisk) Stroop trials in separate trial blocks is preferable to mixing trial types within blocks, for three reasons. First, mixing congruent and incongruent trials within trial blocks confounds prepotent response inhibition with a phenomenon known as goal neglect, wherein the color-naming goal is neglected in favor of a word-reading goal on some trials, thereby confounding inhibitory ability with working memory ability (Kane & Engle, 2003). Second, as described by MacLeod (1991) "The presence of congruent trials among the incongruent and control trials will tend to invoke the tactic of splitting attention" (p. 177) between color-naming and word-reading, thereby artificially inflating the interference effect and confounding inhibitory ability with divided-attention ability. Finally, tasks designed to show robust experimental effects across subjects (as in the mixing of trial types in the Stroop) often have very low test–retest reliability, as they were not designed to reflect individual differences (Hedge, Powell, & Sumner, 2017; Strauss, Allen, Jorgensen, & Cramer, 2005). Thus, although congruent and incongruent trials have been mixed in many previous studies testing alcohol effects on Stroop interference (e.g., Christiansen, Rose, Randall-Smith, & Hardman, 2016; Curtin & Fairchild, 2003; Marinkovic et al., 2012), the approach used here is preferable because it allows for greater confidence that the interference effect more directly reflects response inhibition, without being confounded by divided-attention or working memory abilities.

Furthermore, calculating the Stroop interference effect using neutral (asterisk) color-naming trials is preferable to using congruent trials in that the color-naming goal can be entirely neglected on congruent trials, leading to facilitation of responses relative to neutral trials (e.g., Salo, Henik, & Robertson, 2001). Thus, interference effects calculated as the difference between incongruent RT and congruent RT reflect a combination of response inhibition and response facilitation, creating ambiguity in the interpretation of the Stroop effect (MacLeod, 1991).

Procedure

Participants completed two testing sessions 1–3 weeks ($M = 19.1$ days) apart. They were asked to abstain from alcohol and drugs for 24 hr prior to each of their sessions; compliance was assessed by signed affidavits. Upon arrival to the lab, participants provided informed consent and completed a number of self-report measures. Participants then completed the inhibition tasks in a predetermined order (see below), after which they were scheduled for their second session and dismissed. Second sessions began between 12:00 p.m.–1:00 p.m.; the testing portion lasted approximately 4 hr. Participants were asked to eat a light meal 2–4 hr prior to their appointment. After providing informed consent, women were given a hormonal (urine) pregnancy test to self-administer in a private restroom (none tested positive); men were also asked to void the bladder.

Participants were randomly assigned to consume either a no-alcohol control beverage ($n = 75$), an active placebo beverage ($n = 73$; 0.04 g/kg alcohol), or an alcohol beverage ($n = 68$; 0.80 g/kg alcohol for men, 0.72 g/kg for women). Participants in the control condition were told that their beverage contained no alcohol; participants in the other conditions were told their beverage contained "a moderate amount of alcohol." In these latter conditions, an experimenter ostensibly prepared (in participants' view) a beverage containing a 5:1, tonic to vodka ratio. The alcohol dose was achieved using 100-proof vodka and tonic, calculated based on total body water volume (estimated using age, sex, height, and weight) and the duration of the drinking period (24 min) using published formulas (see Watson, 1989). The placebo

dose was achieved using 10-proof vodka (one part 100-proof vodka to nine parts de-carbonated tonic water) and tonic. Control group participants consumed a tonic-only beverage. Total beverage was isovolemic across conditions. The beverage was divided into three equal-size drinks and participants were given 8 min to consume each one. After the drinking period, participants sat idle for 5 min to ensure initial alcohol absorption into the blood prior to starting the tasks.

Participants were randomly assigned to one of two task completion conditions. Half of the participants in each beverage group were assigned to an ascending/descending (A/D) group, in which the inhibition tasks were completed twice following beverage consumption—once while breath alcohol concentration (BrAC) was rising and once while it was falling. The other half were assigned to a descending-only (D-only) group, in which task performance was measured only after BrAC had peaked and was falling (or equivalent postconsumption time periods in the control and placebo conditions). After beverage consumption and absorption, participants in the A/D group gave breath samples every 5 min until BrAC was at least 0.065%, at which time the AL task battery (stop-signal, then Stroop, then antisaccade) commenced. BrAC was reassessed following the Stroop task and after completion of the battery. Upon completion of the tasks on the AL, participants' BrAC was assessed every 5 min until BrAC peaked (if it hadn't during the AL tasks) and descended to 0.075%, at which time the task battery was completed in reverse order, with another BrAC assessment following the Stroop.[1] Following previous research (Roehrich & Goldman, 1995), participants assigned to the D-only group watched two episodes of a popular sitcom (*The Office*) while BrAC was rising. To maintain engagement, participants were instructed to press a button whenever a character looked directly at the camera (participants were monitored to ensure compliance). Upon completion of these episodes, D-only participants were monitored every 5 min until their descending BrAC reached 0.075%, at which time they completed the inhibition tasks in the same order as the A/D group's descending limb battery. A/D group participants in the placebo and control groups began the "AL" task battery 5 min after completing their beverages, and began the "DL" task battery 5 min after completing the "AL" battery. D-only participants in the placebo and control groups began the "DL" task battery after watching both episodes of *The Office* (roughly 40 min after completing their beverages). This timing was chosen to mirror the average timing of task completion among participants in the alcohol group.

Upon completion of laboratory tasks, participants completed a series of postexperimental questionnaires, after which those in the placebo and control conditions were debriefed and dismissed. Participants in the alcohol condition were retained until a breathalyzer test indicated sobriety (BrAC $\leq$0.02%; see NIAAA, 2004). Participants in all conditions were provided with transportation home from the lab.

Analytic Approach

Due to equipment errors, data for 12 participants (n = 2 placebo; n = 3 alcohol; n = 7 control) were not recorded for one or more tasks, and therefore degrees of freedom differ across models. Following previous work (e.g., Friedman et al., 2008, 2016; Ito et al., 2015), a number of procedures were used during scoring to make the task data suitable for analyses. For the Stroop task, RTs from error trials (<2% of trials) and RTs <200 ms were eliminated. To obtain the best measure of central tendency for RT difference scores, we applied a within-subject trimming procedure that is robust to nonnormality (Wilcox & Keselman, 2003): For each participant, observations that deviated from the median by >3.32 times the median absolute deviation in each condition were excluded (M = 2.05 observations). Additionally, to improve homogeneity of residuals across conditions, the proportion data for accuracy-based measures were subjected to an angular transform (arcsine of the square root of proportion correct).

Testing effects of alcohol on inhibition. Due to the multigroup nature of the design, in which only half of the participants completed the AL assessments, we examined AL and DL performance in separate hierarchical regression models for each of the three tasks. To permit estimation of both pharmacological effects of alcohol and expectancy-related effects, the regression models included dummy-coded *alcohol* (1 = alcohol, 0 = placebo, 0 = control) and *expectancy* variables (0 = alcohol, 0 = placebo, 1 = control)[2] as predictors in the first step, along with sex and baseline task performance. The interaction of baseline performance and alcohol was added as a predictor in the second step. Inclusion of baseline performance as a main effect ensures that any beverage-related effects in Session 2 are not confounded by variability in baseline performance across the groups.[3] Thus, beverage-related effects can be interpreted as differences across groups in performance change from baseline. Including the Baseline × Alcohol interaction term permits a test of whether postdrinking inhibition performance was moderated by individual differences in baseline ability, and provides a more general statistical model that is not compromised by the strict assumptions of analysis of covariance. Baseline performance was mean-centered prior to creation of the cross-product term to reduce nonessential collinearity between the main effect and the interaction term in the second step of each model (e.g., Cohen, Cohen, West, & Aiken, 2003).

Testing for BAC limb effects. We took two approaches to examining whether task performance differed across the AL and DL. First, in addition to alcohol, expectancy, and baseline performance, models examining DL performance also included a limb condition variable (i.e., whether or not the tasks had been performed on the AL; 0 = yes [A/D group]; 1 = no [D-only group]) in the first step, and an additional Alcohol × Limb condition

[1] Eleven participants in the A/D group achieved peak BrAC values while still completing the AL tasks, and therefore their BrAC values at the beginning of the DL tasks were <.075%. Similarly, seven participants in the D-only group achieved peak BrAC values before the episodes of *The Office* were finished. Thus, the average BrAC value at the beginning of the DL tasks was <.075% for participants in both groups.

[2] Placebo served as the reference level so that both the alcohol condition and the control condition were compared with the placebo condition. This ensures that the *alcohol effect* is purely pharmacological (controls for expectancy) and the *expectancy effect* is purely psychological (controls for pharmacology).

[3] A set of 3 (Beverage group) × 2 (Limb group) factorial ANOVAs testing for potential group differences in baseline performance showed no significant main effects or interactions, Fs < 1.83, ps > .16.

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interaction term in the second step.[4] Testing these effects indicates whether DL performance is affected by having completed the tasks during the AL, and whether any such effects differ as a function of alcohol consumption. Second, to test for potential performance differences across the AL and DL assessments and whether those differences were affected by alcohol, data from the A/D group also were submitted to separate 3 (Beverage group) × 3 (Test; baseline, AL, DL) analyses of variance (ANOVAs) with repeated measures on the latter factor.

Results

Intoxication

For participants in the alcohol beverage, A/D group, BrAC values increased from the start (M = .069, SD = .007) to the end (M = .077, SD = .014) of the AL task battery, $t(35) = 2.85$, p = .007, and decreased over the course of the DL task battery (Ms = .071 to .054, SDs = .007 and .011, respectively), $t(35) = 11.80$, $p < .0001$; a similar magnitude decrease during the DL tasks was observed for D-only participants (Ms = .072 to .057, SDs = .006 and .009, respectively), $t(32) = 11.91$, $p < .0001$. A 2 (Time; DL-start, DL-end) × 2 (Limb group; *A/D, D-only) mixed* analysis of variance (ANOVA) on DL BrAC values indicated no main effect of Limb group and no interaction (Fs < 1), confirming that participants in both groups completed the DL tasks under similar BrAC values, with similar rates of descent. Analyses of subjective intoxication ratings are given in the online supplementary materials.

Associations Across Tasks

Correlations among the inhibition task dependent measures at each assessment are given in Table 2. Means and SDs for each of these measures as a function of experimental group are presented in Table 3. Patterns of these means across measurement occasions are depicted in Figure 1. As indicated in Table 2, performance across the three inhibition tasks was largely uncorrelated at all three assessments. Only antisaccade accuracy and the Stroop interference effect were significantly (modestly) correlated at both baseline and during the DL (rs = −.22, $p < .05$). SSRT was uncorrelated with both antisaccade and Stroop interference at all three assessments (Khng & Lee, 2014). The pattern of correlations within tasks across assessments also suggests differing levels of test–retest reliability across the tasks, with antisaccade performing reasonably well (rs = .67–.86), SSRT performing poorly (rs = .13–.27), and the Stroop interference effect intermediate between the other two (rs = .47–.51). When using data from the placebo and control groups only, these estimates increase: rs = .76–.87 for antisaccade; rs = .52–.59 for Stroop; and rs = .17–.33 for SSRT.

Alcohol Effects on Inhibition Performance[5]

Ascending limb. Table 4 presents the results of regression models in which effects of alcohol and expectancy (and the interaction of alcohol with baseline) on AL task performance were tested. Each of the four models accounted for a significant proportion of the variance in AL performance, but variance explained differed considerably across the tasks (i.e., R^2 values ranged from .60 for antisaccade to .06 for SSRT). Similarly, effects of baseline performance on AL performance also differed a great deal across the tasks (i.e., bs ranging from .77 for antisaccade to .26 for SSRT), suggesting differences in the stability of the measures.

Most importantly for the study's aims, antisaccade performance was significantly affected by alcohol on the AL. Inspection of the means plotted in Figure 1 shows that whereas placebo and control participants showed accuracy improvements from baseline to the AL assessment (M_{change} = .06 and .08, respectively), this did not occur for alcohol participants (M_{change} = −.01). In contrast, alcohol did not affect the Stroop interference effect, incongruent color-naming accuracy, or SSRT on the AL. There were no significant expectancy effects on any of the measures during the AL, indicating that change from baseline to AL performance did not differ for participants in the placebo and control groups. AL performance also did not differ by sex.

The effect of alcohol on AL antisaccade performance depended upon baseline antisaccade performance, as indicated by the significant Alcohol × Baseline interaction term in Table 4. This interaction indicates that the slopes of the regression lines linking baseline performance with AL performance differed significantly across the alcohol (b = .58) and no-alcohol (b = .88) groups. That is, relative to participants who did not consume alcohol, participants in the alcohol group whose antisaccade accuracy was relatively high at baseline experienced more impairment from alcohol than participants whose antisaccade accuracy was relatively low at baseline (see Figure 2). The Alcohol × Baseline interactions for the other inhibition task measures were not significant, indicating that variability in baseline performance on those tasks did not moderate the effect of alcohol on AL task performance.

Descending limb. The lower portion of Table 4 presents the results of the regression models for DL task performance. Each of the four models accounted for significant variance in DL performance, but again the proportion of variance explained differed markedly across the tasks (i.e., R^2 values ranged from .53 for antisaccade to .04 for SSRT), and baseline performance was differentially related to DL performance across the tasks (i.e., bs ranging from .79 for antisaccade to .21 for SSRT).

As with the AL model, antisaccade performance was affected by alcohol on the DL, such that alcohol participants on average showed no change in their antisaccade performance from baseline, whereas placebo and control participants improved from baseline (M_{change} = .09 in both groups; see Table 3). SSRT performance also was impaired by alcohol on the DL. Although the general tendency was for SSRT to increase (i.e., for performance to worsen) from baseline to the DL, this change was larger in the alcohol group (M_{change} = 14.6 ms) than in the placebo (M_{change} = 6.9 ms) and control groups (M_{change} = 6.2 ms). Alcohol had no effect on Stroop interference (at a main effect level), but color-naming accuracy on incongruent Stroop trials was impaired by alcohol (see Figure 1). DL performance was not affected by expectancy in any of the tasks. Also, the lack of significant limb condition effects in each model indicates that DL performance did

[4] Additional interaction terms involving the expectancy variable and both baseline performance and limb condition were modeled in ancillary analyses; none of those interactions approached significance (ps > .50).

[5] Additional models of Stroop task RTs are reported in the online supplementary material (see Table S1).

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Table 2
Correlations Among Inhibition Task Dependent Measures at Each Assessment

Inhibition task measures	1	2	3	4	5	6	7	8	9	10	11
Baseline ($N = 216$)											
1. Antisaccade	—										
2. Stroop RT	$-.22^{*}$	—									
3. Stroop Inc. acc.	*.12*	$-.14^{*}$	—								
4. SSRT	*−.09*	*.05*	*−.07*								
AL ($N = 109$)											
5. Antisaccade	**.73****	−.10	.08	−.13	—						
6. Stroop RT	$-.19^{*}$	**.48****	−.09	.11	*−.14*	—					
7. Stroop Inc. acc.	.16	$-.23^{*}$	**.43****	.03	*.10*	$-.31^{**}$	—				
8. SSRT	−.13	.14	−.10	**.27****	*−.13*	*−.03*	*−.02*	—			
DL ($N = 216$)											
9. Antisaccade	**.67****	$-.13^{*}$	$.13^{*}$	−.10	**.86****	$-.18^{*}$	.14	−.12	—		
10. Stroop RT	$-.21^{**}$	**.47****	$-.15^{*}$	.02	$-.23^{*}$	**.51****	−.17	.00	$-.22^{**}$	—	
11. Stroop Inc. acc.	.01	−.10	**.33****	.01	.10	−.17	**.41****	−.11	$.15^{*}$	$-.25^{**}$	—
12. SSRT	$-.17^{*}$	−.03	−.11	**.22****	$-.29^{**}$	−.09	−.14	**.13**	$-.15^{*}$	*.06*	*−.07*

Note. Baseline = first session, prior to beverage condition assignment; AL = ascending limb assessment; DL = descending limb assessment. Antisaccade = accuracy (proportion correct) in identifying a digit presented in a noncued location; Stroop = congruency effect in RT (incongruent trial RT minus asterisk trial RT); SSRT = stop signal reaction time; Stroop Inc. acc. = color-naming accuracy on incongruent Stroop trials. Intercorrelations among task performance measures at each assessment are indicated in italics; test–retest correlations within each task are indicated in boldface.
* $p < .05$. ** $p < .01$.

not differ as a function of whether or not the tasks had been performed during the AL. Moreover, the Alcohol × Limb condition interaction was not significant in any of the models, indicating that alcohol effects on DL performance did not differ between the A/D and D-only groups.

However, DL performance for both the antisaccade and Stroop interference was predicted by the interaction of alcohol with baseline performance (see Figure 2). For antisaccade, the form of this interaction was quite similar to the AL model, in that the slope of the regression line linking baseline and DL performance was smaller among participants in the alcohol group ($b = .58$) than in the other groups ($b = .87$), and that better baseline performance was associated with relatively larger alcohol effects during the DL. Examination of the Baseline × Alcohol interaction in the Stroop interference model also indicated that the slope of the regression line linking baseline and DL performance was smaller among

Table 3
Means (and Standard Deviations) of Inhibition Measures as a Function of Experimental Group

	Experimental group					
	Alcohol		Placebo		Control	
Inhibition task measures	A/D ($N = 36$)	D-only ($N = 33$)	A/D ($N = 36$)	D-only ($N = 37$)	A/D ($N = 37$)	D-only ($N = 37$)
Baseline						
Antisaccade	.65 (.14)	.64 (.13)	.64 (.14)	.65 (.14)	.66 (.13)	.64 (.14)
Stroop RT	129.8 (77.6)	145.1 (84.9)	149.6 (66.1)	130.2 (70.3)	131.3 (85.3)	127.3 (62.6)
Stroop Inc. acc.	.98 (.02)	.98 (.02)	.98 (.02)	.98 (.02)	.97 (.03)	.98 (.02)
SSRT	257.3 (40.9)	247.7 (27.9)	248.5 (35.3)	253.0 (36.0)	245.2 (29.9)	258.3 (41.3)
Ascending limb						
Antisaccade	.64 (.12)	—	.70 (.15)	—	.74 (.13)	—
Stroop RT	112.7 (57.4)	—	131.5 (50.5)	—	111.4 (54.3)	—
Stroop Inc. acc.	.96 (.04)	—	.98 (.03)	—	.97 (.04)	—
SSRT	263.0 (43.8)	—	260.5 (33.0)	—	252.9 (29.1)	—
Descending limb						
Antisaccade	.66 (.15)	.63 (.12)	.74 (.14)	.73 (.17)	.74 (.17)	.74 (.14)
Stroop RT	110.2 (61.8)	123.8 (59.2)	127.5 (67.6)	113.6 (71.5)	119.4 (56.9)	114.4 (71.0)
Stroop Inc. acc.	.96 (.03)	.96 (.04)	.98 (.02)	.97 (.07)	.97 (.03)	.99 (.01)
SSRT	274.0 (36.8)	260.1 (38.8)	253.0 (33.1)	262.4 (36.3)	257.5 (24.3)	258.4 (30.3)

Note. A/D = completed tasks on the ascending and descending limb; D-only = completed tasks on the descending limb only; Antisaccade = accuracy (proportion correct) in identifying a digit presented in a noncued location; Stroop RT = reaction time interference effect (incongruent trial RT minus asterisk trial RT); Stroop Inc. acc. = color-naming accuracy on incongruent Stroop trials; SSRT = stop signal reaction time.

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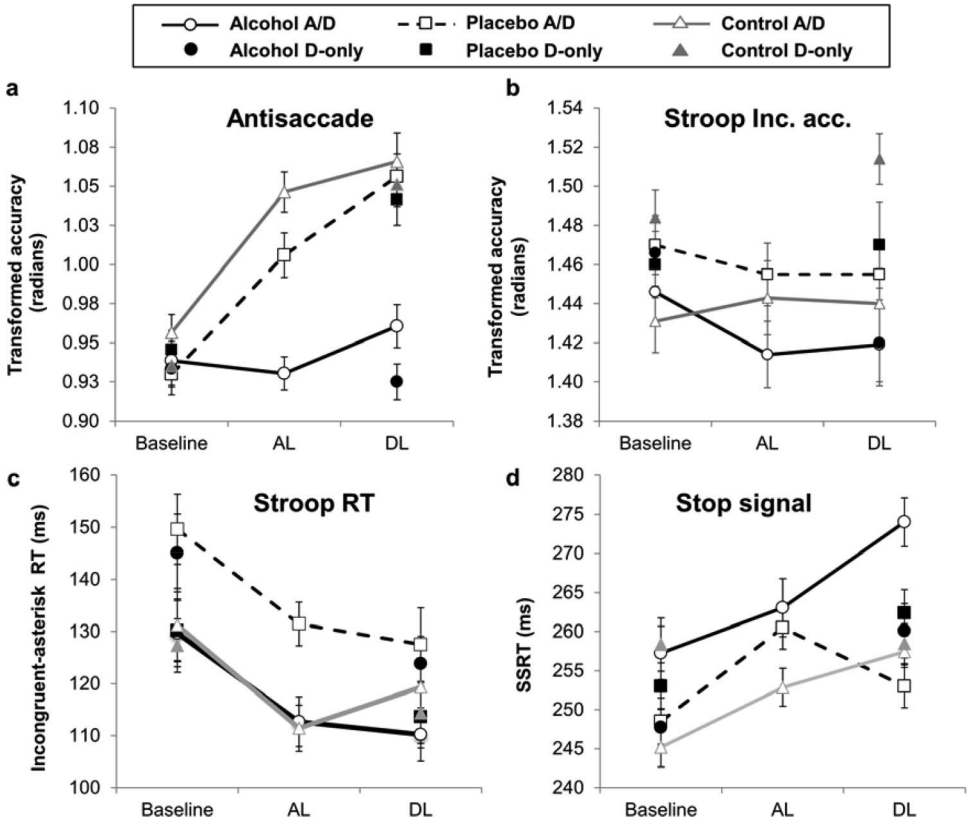

Figure 1. Mean performance on the antisaccade (panel a), Stroop (color-naming accuracy on incongruent trials [panel b] and reaction time interference [panel c]), and stop-signal (panel d) tasks as a function of beverage and limb groups assessed at baseline, ascending limb (AL) and descending limb (DL) measurements. Vertical bars indicate standard errors. Note that participants in the D-only groups were not assessed on the AL. Antisaccade accuracy is depicted in radians, the units of measure that result from subjecting the proportion of correct responses to an angular transformation (arcsine of the square root).

those who consumed alcohol ($b = .25$) than those who did not ($b = .52$). However, given that the Stroop interference effect is calculated as a difference score, and that difference scores are less reliable than the individual test scores comprising them when those individual test scores are highly correlated (e.g., Cronbach, 1990), this effect could simply reflect that the correlation between baseline and DL performance is reduced under alcohol because reliability is compromised. Additional models using incongruent trial RT and asterisk trial RT as the criterion variables showed no significant alcohol effects or interactions with baseline performance ($ts < 1.33$, $ps > .18$).

Effects of repeated testing. The ANOVA examining AL/DL participants' antisaccade performance as a function of beverage group and test showed a significant main effect of test, $F(2, 210) = 35.84$, $p < .0001$, which was qualified by a Test × Beverage Group interaction, $F(4, 210) = 6.54$, $p < .0001$. As evident by the patterns shown in Figure 1, performance did not differ across the groups at baseline ($F < 1$), but control and placebo participants were more accurate than alcohol participants at both AL and DL assessments, $Fs(1, 105) = 9.22$ and 9.63, respectively, $ps < .0035$. Inspection of Figure 1 also suggests that whereas control and placebo participants' performance improved from baseline, no such change occurred for the alcohol participants. A focused contrast specifying increases from baseline in the placebo and control groups but not the alcohol group was significant, $F(1, 105) = 18.56$, $p < .001$, supporting this observation. Finally, the means in Figure 1 suggest that performance in the alcohol group improved from the AL to the DL assessment; a simple effect contrast showed this difference to be significant, $F(1, 105) = 4.29$, $p = .041$.

The ANOVA on the Stroop RT interference effect showed only a main effect of test, $F(2, 210) = 6.09$, $p = .003$, indicating that the Stroop interference effect decreased from baseline ($M = 138.2$ ms) to AL ($M = 119.1$ ms) and DL ($M = 119.0$ ms). The ANOVA on color-naming accuracy during incongruent Stroop trials showed no significant main effects or interaction ($Fs < 1.51$, $ps > .22$).

Finally, the ANOVA on SSRT showed only a main effect of test, $F(2, 200) = 3.72$, $p = .026$, indicating that performance generally declined (i.e., longer SSRT) across assessments. Also, consistent with the regression model (see Table 4), a focused contrast showed that DL performance was worse in the alcohol condition compared to the placebo and control conditions, $F(1, 100) = 7.51$, $p < .001$. However, performance did not differ during the AL and the DL in the AL/DL group, $F(1, 100) = 1.74$, $p = .189$.

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Table 4
Multiple Regression Analyses Predicting Postdrinking Inhibition Performance as a Function of Baseline Performance, Beverage Variables, and Sex and (for Descending Limb) Whether Tasks Were Completed During Ascending BAC

	Antisaccade		Stroop RT		Stroop Inc. acc.		SSRT	
Predictor variables	Adj R^2	*b*	Adj R^2	*b*	Adj R^2	*b*	Adj R^2	*b*
Ascending limb (N = 109)	.60***		.21**		.18***		.06*	
Step 1: Main effects								
Baseline performance		.77***		.33***		.49***		.26**
Sex		.00		−4.41		.01		−7.36
Alcohol		−.08**		−12.34		−.03		−.45
Expectancy		.02		−14.02		.01		−7.58
Step 2: Interaction[1]	.01*		.01		−.01		−.01	
Alcohol × Baseline		−.30*		−.20		−.12		−.08
Descending limb (N = 216)	.53***		.21**		.15***		.04*	
Step 1: Main effects								
Baseline performance		.79***		.42***		.37***		.21**
Sex		−.01		−4.45		.02		−.39
Alcohol		−.10**		−2.07		−.04**		14.83*
Expectancy		.00		.23		.02		−.31
Limb condition		−.02		−.30		.02		−1.78
Step 2: Interactions[1]	.01*		.02*		.00		.01	
Alcohol × Limb condition		−.02		13.42		−.05		−13.78
Alcohol × Baseline		−.28*		−.27*		.05		.19

Note. Antisaccade = accuracy in naming a digit appearing opposite a cued location; Stroop RT = reaction time interference effect (incongruent trial RT minus asterisk trial RT); Stroop Inc. acc. = color-naming accuracy on incongruent Stroop trials; SSRT = stop-signal reaction time; Alcohol = dummy-coded variable testing pharmacological effects of alcohol (alcohol group vs. placebo group); Expectancy = dummy-coded variable testing the expectancy effect (placebo group vs. control group); Limb condition = whether or not tasks were completed on the ascending limb (0 = no [D-only group]; 1 = yes [A/D group]).
[1] The adjusted R^2 value given for Step 2 represents the change resulting from inclusion of the interaction terms.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Discussion

A primary aim of this study was to determine whether alcohol similarly affects performance on each of three tasks identified in multivariate EF models as measures of response inhibition (Friedman et al., 2008, 2016; Ito et al., 2015; Miyake et al., 2000). The current findings provide evidence that the effect of alcohol across these tasks is far from uniform. In fact, only one of the three tasks—the antisaccade—clearly and consistently showed sensitivity to alcohol's effects. This effect took the form of an absence of improvement with repeated testing. That is, consuming alcohol did not worsen antisaccade performance relative to baseline, but unlike participants in the other groups, alcohol group participants failed to show improvement with repeated administrations of the task. Stop-signal performance was impaired by alcohol (relative to baseline and to the other groups' performance) only on the DL. Finally, Stroop interference was unaffected by alcohol on both the AL and the DL; participants in all beverage groups showed similar magnitude improvements across assessments. However, accuracy in color-naming on incongruent Stroop trials was impaired by alcohol on the DL. This suggests that participants who consumed alcohol maintained response speed at the expense of accuracy on incongruent trials, which could be considered a failure of inhibition. Of note, Curtin and Fairchild (2003) also reported that alcohol significantly reduced incongruent color-naming accuracy but only marginally increased incongruent RT during the Stroop task.

Given their relative lack of association, differential effects of alcohol across these tasks are not surprising. As shown in Table 2, although some statistically significant correlations emerged across tasks, none was larger than .22 (between Stroop and antisaccade). This pattern is similar to that seen in previous reports (Friedman & Miyake, 2004; Friedman et al., 2008; Ito et al., 2015; Miyake et al., 2000). For example, Friedman et al. (2016) administered very similar versions of the Stroop, stop-signal and antisaccade tasks to a community-based sample of 786 young-adult twins (M = 22.8-years-old) and reported correlations ranging from .13 to .32 among the tasks. Such patterns suggest that alcohol effects on one purported "inhibition" measure should not be assumed to generalize to other measures, and underscore the need for greater specificity in the constructs measured by these and other commonly used laboratory tasks.

Given the general consensus in the field that alcohol impairs inhibition (Day et al., 2015; Field et al., 2010; Noël et al., 2010), this apparent selectivity is somewhat surprising. However, as reviewed previously, careful examination of previous studies reveals considerable inconsistency in the effects of alcohol on inhibition. To provide some additional context for understanding the current results, we compiled a list of previous studies in which the acute effect of alcohol on inhibition has been tested. This list is summarized in Table 5. The studies included in the summary are those that appeared in recent reviews of this literature (Day et al., 2015; Noël et al., 2010; Weafer & Fillmore, 2016) or that were discovered by entering search terms "alcohol" and "(inhibition OR inhibitory control OR response inhibition)" as Boolean strings in Internet search databases (PsycINFO; Google Scholar).

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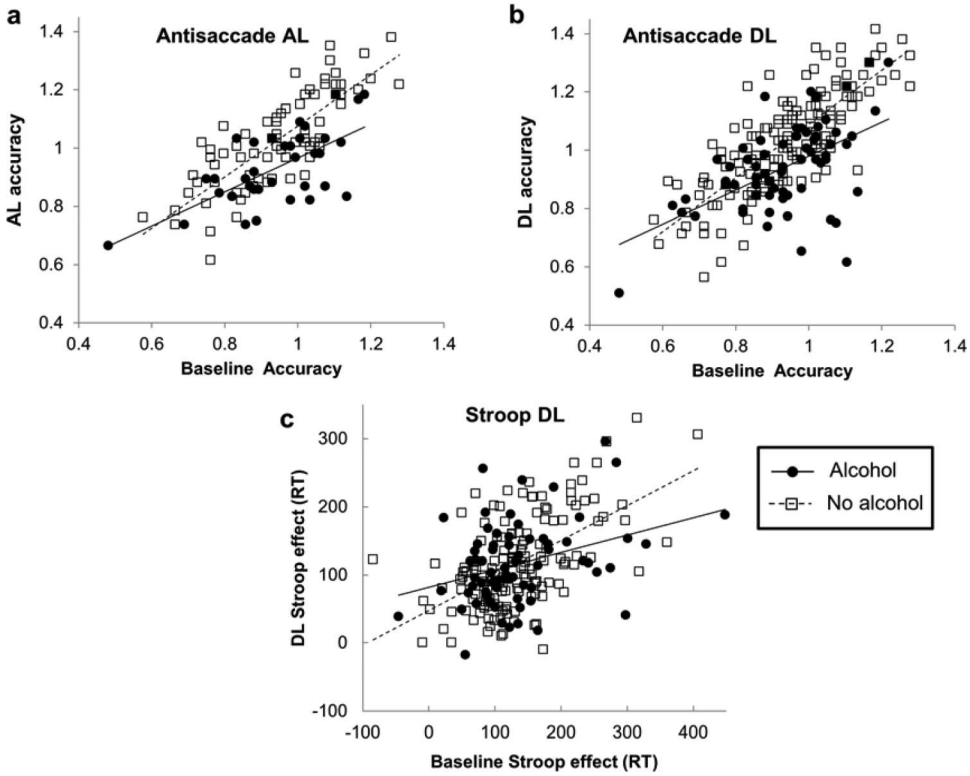

Figure 2. Postdrinking antisaccade performance on the ascending limb (panel a) and descending limb (panel b), and the Stroop RT interference effect on the descending limb (panel c), as a function of beverage group (alcohol vs. no alcohol) and baseline task performance.

Two primary conclusions can be drawn from the information in Table 5. First, and consistent with the current results, acute effects of alcohol on inhibition have varied considerably both across and within the tasks used to measure it. Consider the Stroop task, for example. The published literature includes eight previous studies in which acute effects of alcohol on Stroop performance have been investigated (Bombeke et al., 2013; Christiansen et al., 2016; Curtin & Fairchild, 2003; Duka & Townshend, 2004; Gustafson & Källmén, 1990; Marinkovic et al., 2012; Rose & Duka, 2007, 2008). Of these, four have reported relatively clear (i.e., significant) impairment of either color-naming accuracy or increased RT interference by alcohol (Christiansen et al., 2016; Curtin & Fairchild, 2003; Rose & Duka, 2007, 2008), and four have shown either no alcohol effects on these measures (Bombeke et al., 2013; Duka & Townshend, 2004; Gustafson & Källmén, 1990) or a statistically marginal effect (on color-naming accuracy; Marinkovic et al., 2012). Complicating matters considerably is the fact that the Stroop has been administered in numerous ways across these studies. For example, some have used only incongruent color trials (Christiansen et al., 2016; Duka & Townshend, 2004; Rose & Duka, 2007, 2008) while others mixed congruent and incongruent color trials within trial blocks (Bombeke et al., 2013; Curtin & Fairchild, 2003; Marinkovic et al., 2012); some have measured vocal responses (Christiansen et al., 2016; Curtin & Fairchild, 2003; Gustafson & Källmén, 1990) while in others manual responses were used (i.e., button pressing; Bombeke et al., 2013; Marinkovic et al., 2012). There appears to be no consistent association between a particular method of task administration and a pattern of alcohol effects, although characterizing Stroop performance in terms of color-naming accuracy on incongruent trials, regardless of whether congruent trials are also used, appears to most consistently—though not uniformly—result in impairment by alcohol. This could be because reading aloud produces clearer conflict during incongruent trials than does manually categorizing words by color (MacLeod, 1991). Moreover, in three previous studies the color-naming task was preceded by a block of word-reading trials (Duka & Townshend, 2004; Rose & Duka, 2007, 2008), which arguably strengthens prepotent responding and makes subsequent incongruent color-naming more difficult.

The second conclusion evident from Table 5 is that only one task—the cued go/no-go task developed by Fillmore and colleagues (Marczinski & Fillmore, 2005)—has consistently shown sensitivity to the acute effects of alcohol. Twenty published studies have reported alcohol-induced disinhibition using this task and none have failed to show an effect, providing strong evidence against the null hypothesis. In contrast, studies employing more traditional (noncued) go/no-go tasks and studies using other inhibition tasks, including the tasks used here, have been only about 1.32 times more likely to find effects than not, which is comparatively weak evidence (Goodman, 2005). A critical element could be that the cued go/no-go task creates a strong prepotency (i.e., to emit a particular response), which is less obviously present in some of the other tasks and varies according to the specific task parameters researchers have used. Importantly, this brief review does not

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Table 5
Studies Reporting Acute Effects of Alcohol on Inhibition as a Function of Type of Task

Alcohol-induced disinhibition?*	Inhibition tasks			
	Cued go/no-go	Other go/no-go	Other inhibition[Ψ]	Other/complex[§]
Yes	20[a]	9[b]	16[d]	2[f]
No	0	5[c]	14[e]	0

* Interpreted as significantly poorer performance or outcome on the primary dependent measure derived from the task in question following alcohol versus nonalcohol (placebo or control) ingestion during ascending and/or descending BAC.

[a] Abroms, Fillmore, and Marczinski (2003); Fillmore (2004, 2009); Fillmore, Blackburn, and Harrison (2008); Fillmore, Marczinski, and Bowman (2005); Fillmore, Ostling, Martin, and Kelly (2009); Fillmore and Weafer (2012); Marczinski (2017); Marczinski, Abroms, Van Selst, and Fillmore (2005); Marczinski, Combs, and Fillmore (2007); Marczinski and Fillmore (2005); Miller and Fillmore, 2014; Miller, Hays, and Fillmore (2012); Ostling and Fillmore (2010); Quinn and Fromme (2016); Roberts, Monem, and Fillmore (2016); Weafer and Fillmore (2008, 2012, 2015); Weafer, Fillmore, and Milich (2009). [b] Birak, Terry, and Higgs (2010); Claus and Hendershot (2015); Dry, Burns, Nettelbeck, Farquharson, and White (2012); Easdon et al. (2005, 2000); Finn, Justus, Mazas, and Steinmetz (1999); Stock, Schulz, Lenhardt, Blaszkewicz, and Beste (2016); Tiplady, Oshinowo, Thomson, and Drummond (2009); Tsujii, Sakatani, Nakashima, Igarashi, and Katayama (2011). [c] Domingues, Mendonça, Laranjeira, and Nakamura-Palacios (2009); Ortner, MacDonald, and Olmstead (2003); Reynolds, Richards, and de Wit (2006); Rose and Duka (2007, 2008).

[Ψ] **Stop-signal task** ([d]Caswell, Morgan, & Duka, 2013; [e]de Wit, Crean, & Richards, 2000; [e]Dougherty, Marsh-Richard, Hatzis, Nouvion, & Mathias, 2008; Dougherty et al., 2015; [d]Fillmore & Vogel-Sprott, 2000; [d]Gan et al., 2014; [e]Guillot, Fanning, Bullock, McCloskey, & Berman, 2010; [d]Kareken et al., 2013; [d]Loeber & Duka, 2009a,b; [d]McCarthy, Niculete, Treloar, Morris, & Bartholow, 2012; [d]Mulvihill, Skilling, & Vogel-Sprott, 1997; [e]Peacock, Cash, & Bruno, 2015; [d]Reynolds et al., 2006; [e]Spinola, Maisto, White, & Huddleson, 2017); **Stroop task** ([e]Bombeke, Schouppe, Duthoo, & Notebaert, 2013; [d]Christiansen, Rose, Randall-Smith, & Hardman, 2016; [d]Curtin & Fairchild, 2003; [e]Duka & Townshend, 2004; [e]Gustafson & Källmén, 1990; [e]Marinkovic, Rickenbacher, Azma, & Artsy, 2012; [d]Rose & Duka, 2007, 2008); **Antisaccade task** ([d]Abroms, Gottlob, & Fillmore, 2006; [e]Blekher et al., 2002; [e]Khan, Ford, Timney, & Everling, 2003; [d]Marinkovic, Rickenbacher, Azma, Artsy, & Lee, 2013; [d]Roche & King, 2010; [e]Vassallo & Abel, 2002; [e]Vorstius, Radach, Lang, & Riccardi, 2008). [§] Wisconsin card-sorting task ([f]Lyvers & Maltzman, 1991); Color-matching task from the ImPACT neuropsychological test battery ([f]Schweizer et al., 2006).

account for publication biases that tend to keep null effects out of the published literature, so the true number of null effects is difficult to determine. In any case, the current results align with this brief review in terms of the inconsistency in alcohol's effects across tasks, despite the prevailing view that alcohol impairs response inhibition.

A second aim of the current research was to determine whether alcohol effects on specific tasks differ as a function of limb of the BAC curve. The current results provide some evidence of differential alcohol effects on the DL compared with the AL. Specifically, whereas only antisaccade accuracy showed an alcohol effect on the AL, three of the four measures showed alcohol effects on the DL. This pattern could reflect fatigue in task performance under intoxication. Antisaccade accuracy improved among those in the alcohol condition between the AL and the DL, but interpretation of this difference is complicated by the fact that average BrACs were also lower during DL compared with AL assessment. Thus, the apparent limb effect could reflect a benefit of practice or of a lower level of intoxication during the DL. The lack of any significant limb condition effects on DL performance suggests that practice effects were not influential in the current study. Moreover, although the current study's design would permit separation of acute tolerance and practice effects, the pattern of findings for each of the tasks provides no evidence for or against possible acute tolerance effects.

A final aim of the study was to examine whether individual differences in response inhibition ability, defined in terms of baseline performance on the inhibition tasks, would moderate effects of alcohol on task performance. Evidence suggesting an influence of individual differences was seen for antisaccade and Stroop. In both cases, baseline performance was a weaker predictor of postdrinking performance for participants who consumed alcohol relative to those who did not. Interpretations focused on individual differences suggest that alcohol tended to have the largest effect among individuals with relatively strong baseline performance, suggesting that such individuals have "more to lose" from an acute cognitive insult. Alternatively, it could be that alcohol simply reduces the reliability of these tasks relative to baseline.

The current study suffered from a number of limitations. First, all participants completed the inhibition tasks in the same order (stop-signal, then Stroop, then antisaccade). A consistent task order was deemed important for assessment of individual differences (at baseline) and for ensuring that alcohol effects on a given task could be tested at comparable BrACs across the AL and the DL, but also meant that antisaccade was completed at higher BrAC levels than were stop-signal and Stroop. This fact could partially explain why antisaccade performance was the most consistently affected by alcohol. The fact that antisaccade was also the most reliable task across sessions cannot be explained by this confound, however, and in fact is difficult to attribute to larger alcohol effects on that task compared with the others.

Second, the versions of the inhibition tasks used here contained fewer trials than in many previous studies. This was necessary to permit participants in the A/D alcohol group to complete all three tasks during both limbs of the BrAC curve, but could have con-

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tributed to reduced reliability of the tasks (especially that of the stop-signal task) and, consequently, their rather poor intercorrelations. This limitation is not unique to the current study, however—numerous theorists have commented on the problematic reliability of various EF tasks, including those used here (e.g., Chan, Shum, Toulopoulou, & Chen, 2008; Hedge et al., 2017; Rabbitt, 1997).

Another limitation of the current study is that we attained less control over BrAC than intended, resulting in some A/D participants reaching peak BrAC prior to completing all AL tasks. In the future, these problems could be addressed by using an alcohol infusion procedure, which allows specific BAC levels to be maintained over long periods of time (see O'Connor, Morzorati, Christian, & Li, 1998). Such a solution could potentially exacerbate other problems, however, such as subject fatigue, which also likely was an issue in the current study.

In sum, the current findings indicate that greater specificity is needed in examining effects of alcohol on EF abilities, particularly inhibition. The fact that measures assumed to tap the same facet of EF (inhibition) evinced differential effects of alcohol suggests that alcohol's effects across EF facets are likely to be even more variable. In future work, researchers should test not only the extent to which acute alcohol effects differ across laboratory tasks but also should carefully deconstruct the neural circuits important for performance of such tasks, their vulnerability to acute impairment by alcohol, and the degree to which impairment on such lab-based measures corresponds to alcohol-related problems outside the lab, such as drunk driving (McCarthy et al., 2012) and serious injury (Cherpitel et al., 2009).

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