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Alcohol Responses, Cognitive Impairment, and Alcohol-Related Negative Consequences

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Alcohol Responses, Cognitive Impairment, and Alcohol-Related Negative Consequences

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Alcohol Responses, Cognitive Impairment, and Alcohol-Related Negative Consequences

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Under frameworks such as Alcohol Myopia Theory, a body of literature has developed demonstrating how alcohol intoxication can increase behavioral risk-taking, potentially via impaired inhibition of prepotent behavioral responses. A separate area of research has shown that responses to alcohol intoxication are not homogenous across the population. Whereas most previous research has considered alcohol responses in relation to risk for alcohol use disorders, the present investigation tested whether they may additionally contribute to the *acute* effects of alcohol on drinking-episode-specific cognitive and behavioral consequences. We recruited 82 moderate-to-heavy drinking emerging adults to each complete 2 research protocols: a placebo-controlled, within-subject, counterbalanced alcohol challenge in a simulated bar laboratory and a 21-day, event-level self-monitoring follow-up. Replicating previous research, the alcohol challenge increased heart rate and subjective stimulant-like and sedative-like responses and impaired psychomotor performance and response inhibition. Individual differences in subjective stimulation but not sedation were significantly associated with inhibitory impairment. In the event-level follow-up, we found little evidence that alcohol responses elevated risk for adverse behavioral outcomes, although evidence was stronger that alcohol responses were associated with alcohol-induced memory blackout. Whether and

how alcohol responses relate to the physiological, cognitive, and behavioral consequences of alcohol intoxication may depend on a) the quality of the response (e.g., stimulation vs. sedation), b) the type of outcome (e.g., response inhibition vs. blackout vs. behavioral risk-taking), and c) whether perceptions of alcohol-induced effects may contribute to emerging adults' evaluations of risk (e.g., driving after drinking and riding with a drinking driver).

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Chapter 1: Background and Introduction

Although the traditional conceptualization of alcoholism as a chronic condition of adulthood has informed treatment research on alcohol use disorders (AUDs; e.g., Anton et al., 2006), theoretical accounts now recognize that the prevalence of alcohol consumption and many of its adverse consequences peaks during the developmental period termed emerging adulthood (approximately ages 18-25; Arnett, 2000; Sher & Gotham, 1999). Notably, alcohol dependence onset risk declines dramatically after age 25 (Li, Hewitt, & Grant, 2004), and AUDs are more common during the third decade of life than at any later time. The 12-month prevalence of alcohol dependence in the U.S. over the past 2 decades has been approximately 4% overall but more than 9% among those aged 18 – 29, and alcohol abuse has shown similar age-trends (B. F. Grant et al., 2004).

It is important to note, moreover, that the public health cost of emerging adult alcohol use includes more than AUDs. Population-mean levels of heavy episodic drinking rise to a peak of approximately 40% during the early 20s (Johnston, O'Malley, Bachman, & Schulenberg, 2009). Dangerous in its own right (O'Neill, Parra, & Sher, 2001), heavy episodic drinking is also linked to a wide range of other potentially harmful activities, which, for the purpose of this proposal, will be grouped under the term "behavioral risks." These behaviors include aggression, unsafe sexual behavior, and intoxicated driving, each of which can result in social, physical, psychological, and legal consequences for the intoxicated individual and those around her. College students comprise a substantial segment of the emerging adult population, and alcohol contributes annually to nearly 600,000 unintentional injuries and 1,800 injury deaths among students (Hingson, Zha, & Weitzman, 2009). This high—and increasing—mortality rate is largely

attributable to intoxicated driving, which factors into nearly half of all traffic deaths among those aged 18 – 24 (Hingson et al., 2009). As recent research has made clear, driving after drinking—like many other consequences of alcohol use—does not exclusively occur among those with an AUD (Martin, Sher, & Chung, 2011).

ALCOHOL USE AND BEHAVIORAL RISKS

Understanding the link between alcohol use and its consequences has been a focus of research using a variety of methodologies. Survey research consistently demonstrates global associations between alcohol use and other behavioral risks, with individuals who drink more heavily at risk for numerous other harmful behaviors (e.g., Cooper, Wood, Orcutt, & Albino, 2003; Flowers et al., 2008; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Shope & Bingham, 2002). Global association studies cannot, of course, generate conclusions about the causal role of alcohol use in aggression, unsafe sex, or other outcomes. There is, in fact, a strong alternative explanation for these associations beyond the direct role of alcohol intoxication: Common underlying factors may give rise to heavy alcohol use and to other behavioral risks. Indeed, behavioral genetic research has demonstrated that a highly heritable predisposition can explain a great deal of why some individuals are at risk for problematic alcohol use and a range of other problem or externalizing behaviors (Krueger et al., 2002; Young et al., 2009).

Even in the presence of dispositions toward alcohol use and other externalizing behaviors, however, alcohol intoxication may also increase the likelihood of behavioral risk-taking. Randomized, placebo-controlled studies in which an alcohol dose is administered under controlled laboratory conditions have tested this possibility. Many of these studies have focused on three of the behavioral risks that have been most commonly attributed to intoxication: aggression, driving after drinking, and unsafe sexual behavior.

A rich experimental literature has supported the role of alcohol intoxication in aggressive responses to provocation, particularly among men (Bushman & Cooper, 1990; Chermack & Giancola, 1997; Giancola, Josephs, Parrott, & Duke, 2010). Although the experimental literature on alcohol's influence on risky sexual behavior is more complicated relative to aggression (Cooper, 2006), it generally supports the notion that, under certain conditions, alcohol intoxication can increase sexual risk-taking (Cooper, 2002; Davis et al., 2009; Davis, Hendershot, George, Norris, & Heiman, 2007; MacDonald, Fong, Zanna, & Martineau, 2000). Similarly, several laboratory-based studies have implicated intoxication in decisions to drive after drinking (MacDonald, Zanna, & Fong, 1995; Marczinski & Fillmore, 2009; Marczinski, Harrison, & Fillmore, 2008).

Alcohol Use and Behavioral Risks in the Natural Environment

Laboratory-based experimental studies have important strengths for isolating the causal influence of alcohol intoxication. They are not, however, without limitations. Given ethical considerations, these studies must rely on behavioral analogues or reported intentions to engage in behavioral risks rather than directly measuring outcomes. Further, laboratory studies cannot exceed ethically permissible alcohol doses. Many studies target blood alcohol concentrations (BACs) at or near the legal limit for driving (.08 g%), which emerging adults greatly exceed in their real-world drinking (Rutledge, Park, & Sher, 2008; White, Kraus, & Swartzwelder, 2006). In sum, experimental studies cannot conclusively establish relations between alcohol intoxication and behavioral risks *as they actually occur*. As an alternative, event-level methodologies can maximize ecological validity by capturing both alcohol use and behavioral risks in real-world drinking contexts. These approaches involve the assessment of behavior as it occurs in daily life, either in the moment or via retrospective self-monitoring. Neal and Fromme (2007), for

example, found that college students became more likely to engage in both aggression and unsafe sexual behavior on occasions when their alcohol intoxication (as measured by estimated blood alcohol concentrations; eBACs) was higher. Although event-level approaches lack the experimental control and randomization to beverage condition of experimental research, they can provide complementary evidence in support of alcohol's influence on other behavioral risks.

The example of intoxicated aggression illustrates this interplay well. Laboratory-based, experimental studies of aggression using the Taylor Aggression Paradigm have consistently found that alcohol increases aggression more among men than among women (Giancola, 2006; Giancola et al., 2009; Giancola & Zeichner, 1995; Gussler-Burkhardt & Giancola, 2005; Taylor, 1967), with few exceptions (but see Duke, Giancola, Morris, Holt, & Gunn, 2011). Whether this pattern reflects a true gender difference or a lack of task validity among women, however, has been a source of debate (Giancola & Parrott, 2008). Event-level research can offer an opportunity to test this finding in a different methodology, although limitations of assessment and sample size had until recently rendered conclusions about any gender differences in event-level intoxicated aggression premature (Neal & Fromme, 2007; Wells, Mihic, Tremblay, Graham, & Demers, 2008). We extended Neal and Fromme's (2007) single-year, 30-day online self-monitoring study using 3 additional years of event-level data and found that, although within-person increases in alcohol intoxication were associated with aggression in both genders, this event-level association was significantly stronger among men (Quinn, Stappenbeck, & Fromme, 2013). We concluded that the gender difference found in the laboratory may not have been entirely attributable to lack of task validity.

Although it is important to recognize that neither methodology can provide definitive evidence of real-world causal relations, conclusions can gain in strength

through replication across approaches. Taken together, random-assignment experiments and event-level studies of real-world behavior provide consistent evidence that alcohol intoxication increases the likelihood that emerging adults will experience adverse consequences. Evidence to date appears strongest for some behavioral risks in particular (i.e., aggression and unsafe sex).

Alcohol Myopia Theory

Perhaps the most widely accepted pharmacological (as opposed to expectancy-based) model of the intoxication-behavior relation is Alcohol Myopia Theory (Giancola et al., 2010; Moss & Albery, 2009; Steele & Josephs, 1990). Alcohol Myopia Theory proposes that alcohol impairs controlled cognitive processing (Casbon, Curtin, Lang, & Patrick, 2003; Curtin & Fairchild, 2003; Moss & Albery, 2009). A consequence of this impairment is that intoxicated individuals' attention becomes limited to only the most salient environmental, mental, or physiological cues (Davis et al., 2007; Gallagher & Parrott, 2011). Although in many circumstances these cues may do little to alter behavior, the model proposes that, under conditions of response conflict (i.e., when roughly equivalent pressures both promote *and* inhibit a behavior), the most salient environmental, social, or internal cues should exert a strong influence over intoxicated behavior.

An important feature of Alcohol Myopia Theory is that the model does not predict that alcohol intoxication will universally lead to behavioral risks or other disinhibited behavior. Although cues impelling behavioral risks may be more common in the bars and parties where alcohol is typically consumed, the model also generates the prediction that, in the presence of inhibiting cues, alcohol might actually protect against behaviors under response conflict. Indeed, studies of alcohol and unsafe sex have demonstrated that, in the

presence of inhibitory cues, intoxicated individuals may be *less* likely to engage in unsafe sex (Cooper, 2006; MacDonald et al., 2000). Similarly, distraction during the Taylor Aggression Paradigm can reduce aggressive responding to a greater extent among intoxicated relative to sober individuals (Giancola & Corman, 2007), and alcohol intoxication can increase the influence of situational pressures on reported attitudes toward driving after drinking (MacDonald et al., 1995)

Factor analytic research has distinguished among three components of cognitive control: set shifting, working memory uploading, and the inhibition of prepotent but inappropriate behavioral responses (Miyake et al., 2000). Although intoxicated individuals demonstrate impairment across a wide range of cognitive processes, the pharmacological effects of alcohol appear to most strongly impair response inhibition (Fillmore, 2003). Cued Go/No-Go Tasks of response inhibition, in which pre-response cues provide information about the likelihood of a "go" (i.e., key-press response) trial, have shown particular sensitivity to alcohol's effects (Fillmore, Blackburn, & Harrison, 2008; Marczinski & Fillmore, 2003; Schweizer & Vogel-Sprott, 2008).

ALCOHOL RESPONSES

Largely separate from research on alcohol's acute effects on behavior, a body of research has developed examining individual differences in responses to the effects of alcohol (for recent reviews, see Morean & Corbin, 2010; Quinn & Fromme, 2011b; Ray, MacKillop, & Monti, 2010). This research has primarily focused on the potential roles of alcohol responses in the development of AUDs, and several theories have been developed proposing alcohol responses as components in a genetically influenced pathway toward AUDs. The first such theory, the Low Level of Response Model (LLRM), proposes that a lower general sensitivity to the effects of alcohol serves as an inherited endophenotype

(Gottesman & Gould, 2003) for AUDs. Specifically, if alcohol responses are genetically influenced and "if individuals drink for effects and more alcohol is required to achieve the feelings they want, [low responders] are more likely to drink more heavily," which will ultimately lead to a greater likelihood of developing dependence among those at genetic risk (Schuckit, 2009, p. S7).

Evidence in support of this model originated in a series of studies conducted by Schuckit and colleagues demonstrating a lower level of response to alcohol challenge among men with a positive family history of AUDs relative to those without. This reduced sensitivity has been found for subjective ratings of intoxication (Schuckit, 1980; Schuckit & Gold, 1988), in addition to physiological indices such as body sway (Schuckit, 1985) and levels of cortisol and other hormones (Schuckit, Gold, & Risch, 1987a, 1987b; Schuckit, Risch, & Gold, 1988). Moreover, responses to alcohol are relatively stable over time, even among heavier drinkers (Schuckit & Smith, 2004; Schuckit, Smith, & Tipp, 1997), and twin studies in the U.S. and Australia have found that alcohol responses are moderately heritable (Heath et al., 1999; Viken, Rose, Morzorati, Christian, & Li, 2003). Longitudinal studies show that lower responses predict the development of AUDs (Schuckit, 1994; Schuckit & Smith, 1996, 2000, 2001; Trim, Schuckit, & Smith, 2009).

In contrast to the LLRM, Newlin and Thomson's (1990) Differentiator Model (DM) emphasizes the role of motivation in its account of how individual differences in alcohol responses might confer risk. Alcohol intoxication's subjective effects are biphasic, with the drug producing more stimulant-like effects early in a drinking episode—while alcohol is absorbed—and more sedative-like effects later—while alcohol is metabolized and expelled (Martin, Earleywine, Musty, & Perrine, 1993). Indeed, Ray and colleagues (2009) have found that subjective measures of alcohol responses can be

distinguished into positively valenced, stimulant-like effects and negatively valenced, sedative-like effects (in addition to effects that reflect melioration of negative affect; i.e., tension reduction) and that the Subjective High Assessment Scale, which was used in many of the original alcohol challenge studies, captures sedative-like effects. The DM proposes that the *quality* of the alcohol response (positive vs. negative) may dictate the extent to which it motivates heavier drinking and AUDs. That is, those at risk may be "more sensitive to the drug during the rising blood alcohol curve, when euphoria is greatest, and less sensitive during the falling curve, when anxiety and depression are greatest" (Newlin & Thomson, 1990, p. 399).

Perhaps the best evidence in support of the DM comes from a recent study in which heavier drinkers demonstrated lower response to alcohol on measures of subjective sedation but *greater* response on measures of subjective stimulation (King, de Wit, McNamara, & Cao, 2011). Moreover, heavier drinkers who experienced more rewarding and less sedative subjective alcohol effects (at the peak of the breath alcohol curve) drank more through two-year follow-up. These findings are reinforced by our quantitative review of older alcohol challenge studies, which found some support for the LLRM from family history studies but also indicated that heavier drinkers reported greater stimulant-like and lower sedative-like responses (Quinn & Fromme, 2011b). Moreover, studies of specific drinking episodes show that greater stimulant-like effects are associated with subsequently greater alcohol consumption (Corbin, Gearhardt, & Fromme, 2008; Ray, Miranda et al., 2010; Wetherill & Fromme, 2009).

ALCOHOL RESPONSES AND BEHAVIORAL RISKS

The current investigation represented an attempt to expand current understandings of the public health relevance of alcohol responses. Rather than examining the

contribution of responses to AUD etiology, we tested the roles of alcohol responses in the relation between alcohol intoxication and its acute consequences, notably including other behavioral risks. Previous research on alcohol intoxication's influence over behavior has typically examined either a main effect of alcohol intoxication or a moderation of that effect by contextual or personality factors (e.g., Moss & Albery, 2009; Neal & Fromme, 2007). These approaches assume that alcohol intoxication itself is a largely homogenous condition. Evidence of meaningful inter-individual variability in alcohol responses demonstrates that this assumption may not always be justified.

We tested whether alcohol responses may affect the degree to which an acute dose of alcohol increases an individual's propensity to engage in behavioral risks. Several pieces of preliminary evidence support this proposition. Responses to alcohol have been most often assessed with self-report measures of subjective experiences. However, subjective responses correspond to physiological indices (e.g., body sway, cortisol release, heart rate reactivity) and cannot be entirely explained by expectancies (i.e., they are pharmacological, rather than placebo, effects; Corbin et al., 2008; Morean & Corbin, 2010; Pollock, 1992). This evidence suggests that alcohol responses are not merely capturing epiphenomenal perceptions or evaluations of alcohol's effects but may rather involve individual differences in pharmacological response.

Further, one study has explicitly tested whether alcohol responses can amplify alcohol's effects on a particular behavioral risk, aggression. In a placebo-controlled, laboratory-based paradigm, Assaad and colleagues (2006a) tested whether individuals who experienced greater heart rate reactivity to alcohol engaged in more aggressive responding on the Taylor Aggression Paradigm. Consistent with the hypotheses of this investigation, higher heart-rate responders engaged in greater intoxicated aggression relative to lower heart-rate responders. Subjective stimulation and sedation, however,

were not assessed, precluding any inferences about the specificity of the effects to heart rate reactivity.

Previous research in our laboratory has begun examining alcohol responses and their effects in the natural environment, and a third piece of preliminary evidence comes from a study of 21st birthday celebrations. Using structured interviews conducted shortly following celebrations, Wetherill and Fromme (2009) found that individuals who experienced greater subjective stimulation and sedation were more likely to experience memory impairment (i.e., alcohol blackout) and hangover, controlling for eBACs reached. That is, holding "objective" levels of intoxication constant, greater subjective response was associated with more cognitive and physiological consequences.

One limitation of that study, however, was that it could not disentangle the effects of individual differences in alcohol responses from the effects of episode-to-episode variation in responses. Wetherill and Fromme's (2009) findings could have resulted from either a greater individual sensitivity to the subjective, cognitive, and physiological effects of alcohol *or* contextual factors elevating subjective responses while also increasing risk for consequences. The present investigation was primarily concerned with between-persons differences in responses, yet it is important to acknowledge that responses may vary across drinking events as a function of, for example, ingestion rate (Conrod, Peterson, Pihl, & Mankowski, 1997; Martin & Earleywine, 1990) and social context (Ray, Miranda et al., 2010).

Distinguishing individual differences in alcohol responses from within-person, between-episode variability is a crucial step in isolating the impact of alcohol responses on episode-specific alcohol outcomes, and our previous event-level research has attempted to do so. Our first study of data from a large-scale daily self-monitoring study of college students used a person-mean-centering approach, in which models included

both person-mean subjective intoxication (assessed with a global measure of perceived intoxication that has been most strongly associated with sedative-like responses in laboratory research) and person-mean-centered subjective intoxication (Quinn & Fromme, 2011a). Person-mean subjective intoxication assessed purely between-persons variation, whereas person-mean-centered subjective intoxicated assessed purely within-person, episode-to-episode variation. This study found that greater responders were more likely to aggress, have unsafe sex, and use illicit drugs relative to lower responders, controlling for episode-average eBAC.

Beyond making this important distinction, however, there were several notable limitations of our first study. Individual differences in subjective intoxication were assessed exclusively via self-report, were subject to potential confounding by other between-persons variables (e.g., typical drinking contexts, personality factors), and were not placebo controlled or differentiated into stimulant-like and sedative-like effects. Moreover, our analyses included both drinking and non-drinking days, which could have confounded subjective intoxication with differences between alcohol consumption and abstention. Subsequent studies of the same dataset, in which we limited analyses to drinking days, found that greater typical responders were more likely to aggress and drive after drinking, although these studies were also subject to the other potential confounds described above (Quinn & Fromme, 2012; Quinn et al., 2013). In sum, these preliminary investigations provided evidence consistent with the possibility that greater alcohol responders are more likely, on average, to engage in at least some behavioral risks.

Related to a secondary goal of this project, our previous research has also found evidence of event-level covariation between within-person increases in subjective intoxication and some behavioral risks. In our most recent event-level study of aggression, for example, participants were more likely to engage in aggression when they

experienced greater-than-their typical subjective intoxication, controlling for eBACs (Quinn et al., 2013). Further, in contrast with the finding that greater between-persons subjective intoxication predicted more driving after drinking, within-person increases in subjective intoxication (i.e., feeling more intoxicated than usual) actually moderated the association between daily eBAC and driving after drinking, protecting students against driving after drinking when they became more impaired (Quinn & Fromme, 2012). In examinations of the relation between individual differences in alcohol responses and behavioral risks, it will therefore be important to continue to account for the potential role that episode-to-episode variation in alcohol responses may play.

Differential Inhibitory Impairment as a Cognitive Mechanism

If greater alcohol responders are more likely to engage in a variety of intoxicated behavioral risks and if this increased likelihood results from an amplification of alcohol's intoxicating effects, it will be important to identify the mechanisms that underlie this relation. Given the central role of impaired cognitive control in Alcohol Myopia Theory and other models of alcohol's pharmacological effects on behavior (Fillmore, 2003; Moss & Albery, 2009), one possibility is that, relative to lower responders, greater responders become more cognitively impaired. That is, greater alcohol responses may result in greater reductions in aspects of cognitive control affected by alcohol intoxication, such as response inhibition. Relatively little research has addressed this potential mechanism. In one study, Assaad and colleagues (2006b) examined the association between heart rate response to alcohol and response inhibition. Controlling for task performance in a placebo condition to account for individual differences in response inhibition not due to alcohol, greater heart rate responders made more errors of commission (i.e., failures of response inhibition). This finding suggests that greater responders, at least as assessed by

heart rate reactivity, may experience greater inhibitory impairment when intoxicated. Moreover, Weafer and Fillmore (2008) found that individual differences in alcohol-induced response disinhibition predicted *ad libitum* drinking during a laboratory session. This finding supports the relevance of between-persons differences in alcohol-induced inhibitory impairment to excessive drinking and additionally raises the possibility that these differences may contribute to behavioral risks in other domains as well.

Given preliminary support for a potential relation between alcohol responses and alcohol-induced inhibitory impairment, one important question is whether this association generalizes across stimulant-like and sedative-like alcohol responses or whether it is specific to one type of response. Although Assaad and colleagues (2006b) examined heart rate reactivity only, the different response types suggest differing cognitive mechanisms through which alcohol might impair response inhibition. If the association between alcohol responses and disinhibition is specific to heart rate reactivity or other measures of the stimulant-like effects of alcohol, it is possible that greater responders are at greater risk because they experience greater inhibitory impairment and attention restriction via increased physiological arousal. This account would be consistent with recent evidence that alcohol's myopic effects on aggression can be replicated through sympathetic nervous system activation (Ward et al., 2008). Considered together, these findings would suggest that individuals who experience greater stimulation following a dose of alcohol may experience greater physiological arousal, which would then in turn impair cognitive control.

In contrast, given the uncertainty about whether other alcohol responses might be associated with differences in response disinhibition, preliminary evidence is also consistent with the possibility that increased propensities to engage in behavioral risks may result from a greater overall sensitivity. That is, greater alcohol responders may

experience a broad-based sensitivity to not only subjective stimulation or sedation but also cognitive impairment. This perspective is supported by the finding that greater stimulation *and* sedation during a drinking episode predicted the likelihood of alcohol-induced memory impairment (i.e., blackout; Wetherill & Fromme, 2009).

ALCOHOL RESPONSES AND DISINHIBITED PERSONALITY

Much of the preliminary evidence supporting the potential contribution of individual differences in alcohol responses to behavioral risks has come from global association studies. It is important to acknowledge, however, that these global associations do not necessarily support a causal relation between alcohol responses and behavioral risks. In our current project, we intended to test the hypothesis that alcohol responses exacerbate alcohol's intoxicating effects, increasing the likelihood of engaging in behavioral risks. It is also possible, however, that previously established global associations reflected a (unmeasured) shared underlying propensity to engage in behavioral risks and to experience alcohol's effects more strongly.

One such propensity may be a disinhibited personality disposition, which is a well-established correlate of adolescent and young adult alcohol use and other problem behaviors (e.g., Cooper et al., 2003; Krueger et al., 2002; Sher & Trull, 1994). Beyond their links to externalizing behaviors, facets of disinhibited personality have also been associated with alcohol responses in several studies. In one study, more disinhibited college students experienced greater subjective stimulation (Erblich & Earleywine, 2003). Similarly, greater heart rate reactivity has been associated with sensation seeking and reward sensitivity (Brunelle et al., 2004) and Zuckerman's impulsive sensation seeking scale (Ray, McGeary, Marshall, & Hutchison, 2006). Thus, alcohol responses may be associated with the facets of personality most strongly linked to externalizing

behaviors. Although the identification of common personality factors that might explain associations between alcohol responses and behavioral risks was not a primary objective of this study, we additionally tested this hypothesis as an alternative explanation.

THE PRESENT STUDY

This study combined a laboratory-based, placebo-controlled, within-subject, counterbalanced alcohol challenge and with a subsequent 21-day, event-level daily self-monitoring methodology to test whether greater alcohol responses may help explain why some intoxicated emerging adults are more likely than others to react aggressively, engage in risky alcohol-related driving behaviors, or act in otherwise dangerous ways. Further, we tested whether alcohol responses increase the propensity to engage in intoxicated behavioral risks via differential impairment of response inhibition among greater responders. In addition to our primary study hypotheses, this investigation also enabled an examination of alternative alcohol response associations. We assessed alcohol responses at the episode level, permitting the examination of event-level associations between responses and outcomes. Moreover, we included a comprehensive battery of self-report measures of disinhibited personality, which has previously been suggested as a potential correlate of at least some alcohol responses. See Figure 1 for a logic tree of study hypotheses and potential patterns of relations.

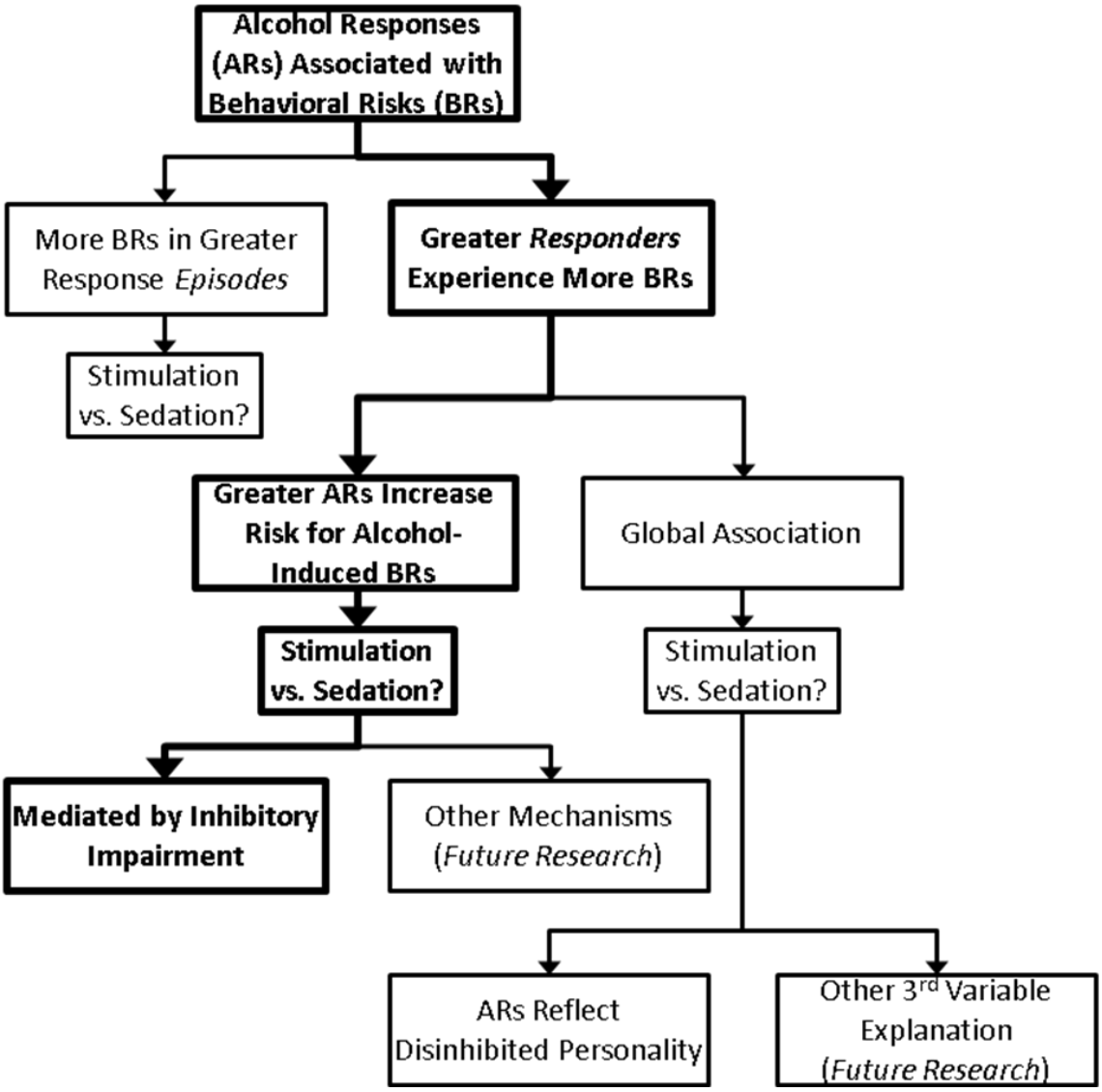

Figure 1: Alcohol responses and behavioral risks logic tree.

Note. Logic tree represents non-mutually exclusive possible explanations for association between individual differences in alcohol responses and behavioral risks. Primary hypothesized relations are bolded.

Chapter 2: Hypotheses

The first research question driving this study was whether alcohol responses are related to alcohol-induced impairment of prepotent response inhibition. We estimated the magnitude and significance of associations between subjective and objective alcohol responses and alcohol-induced inhibitory impairment obtained from a placebo-controlled, within-subject, counterbalanced alcohol challenge conducted in a simulated bar laboratory. Although we did not have any *a priori* hypotheses regarding whether these associations were specific to stimulant-like or sedative-like responses, we examined associations with both responses. The study tested the following specific hypotheses:

1a. An acute dose of alcohol will, on average, impair response inhibition on the Cued Go/No-Go Task relative to performance in a placebo condition.

1b. Measures of alcohol responses will load onto two latent factors: stimulation and sedation.

1c. Greater alcohol responses will be associated with greater alcohol-induced inhibitory impairment on the Cued Go/No-Go Task, controlling for placebo task performance.

The second major objective was to demonstrate that greater alcohol responses predict greater increases in the likelihood of engaging in behavioral risks as a function of alcohol intoxication. We tested the direct and moderating effects of individual differences in laboratory-assessed alcohol responses on the likelihood of engaging in intoxicated behavioral risks during the event-level daily self-monitoring follow-up. Hypotheses were as follows:

2a. Greater alcohol responders will be more likely to engage in a variety of behavioral risks and experience other alcohol-related consequences, controlling for typical alcohol consumption.

2b. Further, alcohol responses will moderate the event-level association between alcohol use and outcomes, such that greater responses (as assessed in the laboratory) will amplify the association between increasing intoxication and increases in the likelihood of outcomes.

2c. Where hypotheses 1c and 2b are supported, we then tested whether this moderation effect was mediated by greater alcohol-induced inhibitory impairment among greater alcohol responders.

Additionally, we tested two secondary hypotheses regarding associations between alcohol responses and outcomes. First, given the expectation of within-person variation in alcohol responses across drinking episodes over and beyond variation in alcohol consumption, we tested whether episode-to-episode alcohol response differences are associated with behavioral risks and other consequences.

3a. Greater within-person increases in alcohol responses, controlling for eBAC, will be associated with increases in the likelihood of engaging in some but not all outcomes during specific drinking events.

3b. Greater within-person increases in alcohol responses, controlling for eBAC, will not, however, be associated with increases in the likelihood of driving. Rather, as in our previous research (Quinn & Fromme, 2012), driving after drinking will be most common during drinking episodes in which eBACs are elevated but alcohol responses are decreased relative to typical levels.

Finally, in the event that we found global associations between alcohol responses and behavioral risks (i.e., hypothesis 2a is supported), we planned to test whether these associations reflected common underlying contributions from disinhibited personality to both alcohol responses and behavioral risks.

4a. Individuals higher in disinhibited personality traits will report greater alcohol responses.

4b. Individuals higher in disinhibited personality traits will also endorse more behavioral risks.

4c. The association between alcohol responses and behavioral risks may reflect, at least in part, shared associations with disinhibited personality.

Chapter 3: Method

PARTICIPANTS AND RECRUITMENT PROCEDURE

Participants (N = 82) were emerging adults aged 21 – 25 from the surrounding community and introductory psychology subject pool at a large, public university. Posted flyers and internet advertisements provided interested emerging adults with contact information to access further study details and complete a brief telephone or online screening questionnaire (SurveyMonkey, Palo Alto, CA). Potential participants were screened for eligibility on demographics, typical alcohol consumption and binge drinking, and contraindications to participation in an alcohol-administration study. We targeted an equal number of male and female participants using the following criteria:

Inclusion Criteria

1) Ages 21 – 25
2) At least moderate drinking frequency, defined as two or more typical-week drinking occasions
3) At least occasional binge drinking, defined as one or more binge drinking episodes (four or more standard drinks in a two-hour period for women, five or more standard drinks in a two-hour period for men) in the two weeks prior to screening

Exclusion Criteria

1) Possible alcohol dependence, defined as a score greater than 15 on the Alcohol Use Disorders Identification Test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001)

2) Other medical, personal, or ethical contraindications to participation in an alcohol-administration study
3) Positive breath alcohol concentration (BrAC) prior to either laboratory session
4) Self-reported pregnancy, possible pregnancy, or positive pregnancy test (for women)[1]
5) Failure to reach BrAC threshold (i.e., BrAC ≥ .05 g% at peak assessment) during alcohol-condition session

Of the 97 eligible participants who completed one laboratory session, 84 returned for a second laboratory session and were enrolled in the daily self-monitoring follow-up. Eight of the remaining 13 participants completed a placebo session only, with the others completing an alcohol session only. Of the 84 returning participants, 2 did not reach the BrAC threshold, resulting in the final sample of 82 participants. This included sample was 49% female, 46% White, 29% Asian or Asian-American, 11% Hispanic or Latino, 4% African-American, and 10% multiethnic or other ethnicities. The mean age was 22.18 years (*SD* = 1.00, range = 21.05 – 25.40), and 36% reported a positive family history of alcohol problems (*n* = 29 of 81 who provided complete family history data).

University undergraduates who participated in the study for course credit were informed that their participation would not affect their class standing. All study procedures were approved by the Institutional Review Board of The University of Texas at Austin and followed NIAAA guidelines for administering alcohol in human subjects research (NIAAA, 2005).

[1] A subset of participants (*n* = 8 included women) were additionally screened for ineligibility on the basis of self-reported nursing in accordance with a change in procedures requested by the Institutional Review Board.

LABORATORY PROCEDURE

Participants completed a placebo-controlled, within-subject, counter-balanced alcohol challenge study across two laboratory sessions. Eligible participants were randomly assigned to complete the alcohol or placebo session first. They were instructed to eat a full meal at least four hours before the start of the session to ensure comparable rates of alcohol absorption across participants. Participants were also instructed to abstain from alcohol for 48 hours and from caffeine and tobacco for 3 hours prior to the session. We attempted to schedule the alcohol and placebo laboratory sessions one week apart for all participants but permitted participants to reschedule their second sessions when necessary. The laboratory sessions were separated by M = 9.23 days (median = 7, SD = 9.06, range = 6 – 70).

Upon arrival at each session, participants screened for a .000 g% BrAC (Intoxilyzer 5000, CMI, Inc., Owensboro, KY). Female participants were also screened for pregnancy with urine hCG pregnancy tests at both sessions, after which eligible participants began the baseline assessment protocol. This protocol included baseline interview, questionnaire, and heart rate measures, in addition to familiarization with the Cued Go/No-Go Task. All baseline measures were administered during the first session only, with the exception of the baseline alcohol responses and heart rate assessment. Participants began the first laboratory session by providing informed consent to the laboratory and follow-up procedures.

Alcohol Challenge

Sessions were conducted in a simulated bar laboratory in groups of three or four participants of variable gender composition. Participants completed their first laboratory sessions in twenty-seven cohorts; all participants in a given laboratory session cohort were randomly assigned to the same condition (alcohol or placebo) prior to the start of

the session, and we attempted to prevent friends and acquaintances from participating in the same cohort. Every effort was made to retain the same cohort of participants through both laboratory sessions, including scheduling both sessions after determining eligibility, providing reminders to participants prior to the sessions, and scheduling the sessions one week apart but on the same day of the week when possible. Doing so helped minimize differences in contextual factors between the two sessions beyond the alcohol or placebo manipulation. If necessary, however, participants were permitted to reschedule in order to minimize attrition. Four participants returned on different occasions from the remainder of their groups. In the event that participants were unable or unwilling to attend their sessions, trained undergraduate research assistants age 18 or older served as confederate participants to maintain a minimum of 3 individuals per laboratory session and enable the other participants to complete their sessions as scheduled. Although all sessions included a minimum of three individuals (participants and confederates), some session cohorts included four individuals in one session and three in the other. Confederates were trained to engage neutrally with the participants to help maintain a similar social milieu to that of the other sessions. They consumed placebo beverages regardless of condition but followed all other procedures during the beverage-administration portion of the protocol.

After completing the baseline measures and procedures, participants were invited into the simulated barroom, in which they were administered either the placebo or alcohol beverage as a group. Participants had 10 minutes to consume each of three drinks containing a 1:3 mixture of 40% alcohol-by-volume vodka or a decarbonated tonic water placebo. Alcohol doses were calculated using gender and weight to target a peak BrAC of .08 g%. First session dosing was double-blinded through the alcohol-administration procedures. When research assistants took post-dosage BrAC assessments, however, they became unblinded to condition. Additionally, when the same research assistants ran

participants on both sessions, the research assistants were no longer blind to condition during the second session because of their awareness of the study design.

We followed standard procedures to ensure an effective placebo manipulation (Rohsenow & Marlatt, 1981). First, because this study used a within-subjects design, participants received instruction that they might or might not receive a dose of alcohol ranging up to .08 g% during each session. During the laboratory sessions, participants were instructed to rinse with alcohol-free mouthwash prior to the baseline breathalyzer test, which helped mask taste. The bar was then wiped with tequila immediately prior to participants' entrance to provide a temporary olfactory cue. Regardless of condition, doses were measured and poured from a sealed vodka bottle in front of the participants, with the bottle containing either vodka or decarbonated tonic water depending on condition. In addition, the rims of the first drink glasses were soaked with vodka, and a squirt of 95% alcohol was added to the top of each drink to provide a taste cue. In the placebo condition, participants therefore consumed a non-zero but minute amount of alcohol (peak BrAC < .001 g%). Finally, after the ascending limb BrAC assessment, all participants were provided with false BrAC feedback ranging from .038 g% to .042 g% to evoke similar outcome expectancies across the alcohol and placebo conditions.

Data Collection

Following the completion of the alcohol-administration protocol, participants were transferred to individual testing rooms adjacent to the simulated bar. They then completed three assessments (corresponding to the ascending limb, peak, and descending limb of the BrAC curve) over the course of 90 minutes. See Figure 2. Subjective alcohol responses and heart rate reactivity (HRR) were assessed at all three assessments, whereas the Cued Go/No-Go Task and Digit Symbol Substitution Task were administered only at

the peak assessment in order to reduce practice effects and allocate assessment time to other measures. BrAC was assessed on the ascending limb (target time = 45 minutes after dose initiation, actual time *M* = 46 minutes), at peak (target and actual time = 60 minutes), post-peak (target and actual time = 90 minutes), and on the descending limb (target and actual time = 120 minutes). We used Intoxilyzer 5000 breathalyzers (CMI, Inc., Owensboro, KY), which produce hard-copy records, to assess BrAC at baseline, on the ascending limb, and at the end of the sessions. All other BrAC testing was conducted using hand-held Alco-Sensor IV breathalyzers (Intoximeters, Inc., St. Louis, MO). All testing occurred outside of the simulated bar laboratory, and all post-administration alcohol response, HRR, Cued Go/No-Go Task, and DSST assessments were conducted individually in the interview rooms to reduce cross-participant contamination[2].

[2] We conducted preliminary analyses to determine whether to adjust for dependency of observations as a function of laboratory session cohort. Excluding the four participants whose first and second session cohorts differed, intraclass correlations for personality, alcohol responses, and Cued Go/No-Go Task inhibition failures ranged from .00 to .32 (*M* = .05, median = .02). Only the placebo-adjusted Cued Go/No-Go Task inhibitory impairment score intraclass correlation exceeded .20. We therefore treated each participant's data as independent of other participants' data.

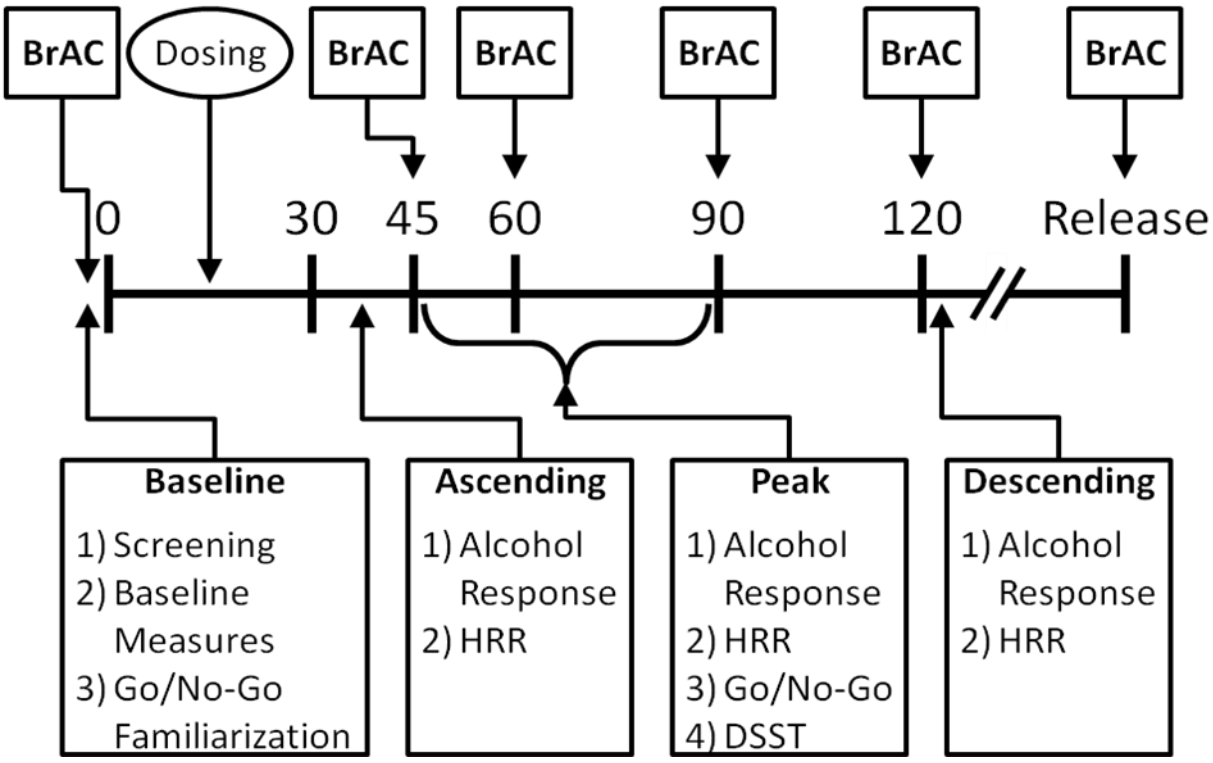

Figure 2: Targeted alcohol challenge protocol timeline in minutes from start of dose.

End of Session

After the completion of the descending limb assessment, participants returned to the simulated bar, where they were permitted to watch movies or television, read, or contact friends or family members. Participants were partially debriefed following the first session and then fully debriefed regarding the laboratory procedures following the second session. BrAC measures were taken approximately every 30 minutes until participants reached .04 g%, at which time they were either driven home by a licensed and insured member of the project staff or picked up by a sober friend or family member. Compensation was $15 for the first laboratory session and $30 for the second, minus any introductory psychology course credit received.

FOLLOW-UP PROCEDURES

At baseline of the second laboratory session, participants were provided with a brief orientation to the daily self-monitoring follow-up procedures. Participants were walked through the self-monitoring survey and provided with information to access the survey. On the second day after the second session, each participant was invited to provide the first of 21 consecutive days of reporting via a secure online survey package (SurveyMonkey). Participants were invited each day via email to log onto the survey website and report any alcohol use, alcohol responses, behavioral risks, and other alcohol-related negative consequences that occurred the previous day and night. Participants were randomly assigned to survey order conditions to counterbalance the presentation of the a) behavioral risk and b) alcohol consumption, responses, and consequences sections.

Participants received their first invitation on the second day after their laboratory session so that the first self-monitoring entry referred to the first day following the completion of the laboratory protocol. Although we encouraged participants to submit surveys each day, we also permitted them to submit reports for the previous seven days to minimize missing data without excessively increasing retrospective bias. Research staff contacted participants via email and phone to ensure the completion of outstanding reports. After the completion of the follow-up period, participants were contacted to return to the laboratory for compensation and final debriefing. They received $5 for each complete week of self-monitoring surveys, with a $5 bonus for completing all 21 surveys (maximum payment = $20).

MEASURES

See the Appendix for the complete self-report questionnaires.

Global Measures Completed at Baseline

Demographics and background characteristics

Participants reported date of birth, gender, ethnicity, family-of-origin income, maternal and paternal occupations and educational attainment, automobile usage and family history of alcohol problems (Mann, Sobell, Sobell, & Pavan, 1985). Parental educational attainment was assessed on a 6-point scale, where 1 = *did not complete high school or obtain GED* and 6 = *post-graduate degree*. Family income prior to high school graduation was assessed on an 8-point scale, where 1 = *under $19,999* and 8 = *$100,000 or over*. Family history was coded such that 0 = *no family history of alcohol problems* and 1 = *definite family history*. Female participants also reported the onset date of their most recent menstrual period.[3]

Age at first drink and first intoxication

Participants reported their age at first drink in years, with first drink defined as *your first drink on your own rather than just a sip from an adult's glass, not including drinking as part of religious ceremonies*. Participants also reported the age at which they first *got drunk after drinking alcohol*. Participants answered both questions on a scale from *5 years old or younger* to *25*.

[3] Although some early research suggested that the effects of alcohol among women differ as a function of changes in menstrual hormones (Linnoila, Erwin, Ramm, Cleveland, & Brendle, 1980), more recent research has failed to find such differences (Holdstock & de Wit, 2000; Terner & de Wit, 2006). Previous alcohol-challenge studies have also been inconsistent in defining menstrual cycle phases, with, for example, Holdstock and de Wit (2000) defining the follicular phase as post-menstrual days 2-6 (early) and 7-11 (late) and the luteal phase as 17-20 (mid) and 25-28 (late), and Terner and de Wit (2006), in a review, defining follicular as the first 14 days and luteal as the last 14 days, with 36 hours of ovulation between the phases. In the current sample, date of period onset was available for 33 women (with the others either not obtained or experiencing inconsistent menstruation). Of these, 7 reported period onsets more than 28 days prior to the alcohol session, rendering their menstrual phases at the session uncertain. Coding the remaining women's cycle phase on the basis of the Terner and de Wit (2006) and Holdstock and de Wit (2000) schemes—with the luteal phase defined as days 17-28 for the latter—we failed to find any differences in the subjective alcohol response factor scores, *t*s (21 or 24) ≤ 1.07, *p*s ≥.30 , *d*s ≤ .45. We therefore did not include menstrual cycle phase in subsequent analyses.

Timeline follow-back interview

Trained undergraduate research assistants administered a version of the widely used *Timeline Follow-Back* interview individually to participants to assess past-30-day alcohol use, alcohol-related negative consequences, and behavioral risks (TLFB; Sobell & Sobell, 1992). The TLFB uses a calendar marked with special dates (e.g., major social occasions, university events) to provide information on daily alcohol consumption. Our TLFB additionally assessed driving after drinking, riding with a drinking driver, gambling, unsafe sex, illicit drug use, property crime, and verbal and physical aggression. For each drinking occasion, participants also reported whether they became subjectively intoxicated ("drunk"), whether they experienced symptoms of hangover or blackout, and whether any behavioral risks occurred before, after, or during alcohol consumption. We used TLFB responses to calculate four indices of alcohol consumption: 1) total standard drinks consumed (i.e., 12 oz of beer, 5 oz of wine, 1.5 oz of liquor); 2) frequency of binge drinking, defined as consuming four or more standard drinks on a day for women or five or more drinks for men (H. Wechsler & Isaac, 1992); 3) frequency of subjective intoxication (Jackson, Sher, Gotham, & Wood, 2001); and 4) most standard drinks consumed in a day (Dawson, 1998). Baseline behavioral risk and alcohol-related consequence data are not presented here. See Table 1.

Alcohol response during early drinking experiences

Using the *Self-Rating of the Effects of Alcohol Scale* (SRE; Schuckit et al., 1997), participants reported the number of standard drinks they required to achieve four alcohol effects (feel an effect, feel dizzy, lose coordination, pass out) during their first five drinking experiences, their heaviest drinking experiences, and the past three months. The *SRE First Five* is a valid measure of early alcohol responses. It has demonstrated moderate associations with responses to alcohol challenge (Schuckit et al., 1997). SRE

scores are typically computed by averaging the number of drinks required across the number of experienced alcohol effects. However, to the extent that, all other things being equal, more drinks will be required to achieve the more severe effects (e.g., passing out), scores may be biased downward for individuals who consumed less during their first five drinking occasions and therefore did not experience the more severe effects. We therefore created an unbiased *SRE First Five* score by standardizing available items and averaging those standard scores.

Variable	Observed Range	Full Sample		Outliers Removed[a]	
		M	*SD*	*M*	*SD*
AUDIT	3 – 15	8.45	3.02		
Self-Rating of the Effects of Alcohol					
First five	1.00 – 10.50	4.09	1.93		
First five from standardized scores	-1.39 – 2.89	0.03	0.95		
30-day Timeline Follow-Back					
Total standard drinks	0.00 – 171.66	49.14	36.35	45.92	30.79
Maximum drinks	0.00 – 31.00	10.28	5.69	9.99	5.23
Frequency of binge	0.00 – 17.00	4.92	3.67	4.72	3.43
Frequency of intoxication	0.00 – 17.00	5.74	3.62	5.62	3.51

Table 1: Baseline alcohol use summary statistics.

Note. $n = 81$ for Self-Rating of the Effects of Alcohol. $n = 78$ for Timeline Follow-Back because dates were calculated incorrectly during one session. AUDIT (Alcohol Use Disorders Identification Test) scores are from screening and include re-tested scores when more than one month elapsed between screening and the first laboratory session.
[a] Timeline follow-back summary statistics after removing 2 participants with improbable values (total standard drinks ≥ 171).

Disinhibited personality

We assessed disinhibited or impulsive personality using a number of validated self-report measures. Participants responded to the *UPPS Impulsivity Scale*, which distinguishes four facets of impulsivity: urgency (12 items), premeditation (11 items), perseverance (10 items), and sensation seeking (12 items) on a 4-point Likert scale

ranging from 1 = *disagree strongly* to 4 = *agree strongly* (Whiteside & Lynam, 2001). The UPPS is validated for administration among college students and substance abusers (Whiteside & Lynam, 2003; Whiteside, Lynam, Miller, & Reynolds, 2005). Positive urgency, a construct defined as impulsivity in response to positive emotions, was assessed on the same 4-point scale with the 14-item *Positive Urgency Measure* (PUM; Cyders et al., 2007). Self-control, the inverse of impulsivity, was assessed with the *Brief Self-Control Scale* (BSCS; Tangney, Baumeister, & Boone, 2004), a 13-item questionnaire answered with a 5-point scale ranging from 1 = *not at all like me* to 5 = *very much like me*. Finally, participants also completed the *Monetary Choice Questionnaire* (MCQ; Kirby, Petry, & Bickel, 1999), a measure of delay discounting (i.e., the capacity to delay gratification). The MCQ consists of 27 questions, each of which provides the respondent with two options: a smaller-but-immediate reward (in dollars) and a larger-but-delayed reward. From these responses, we estimated each participant's delay discounting parameter using the calculations described by Kirby (2000). This parameter serves as an index of the degree to which individuals devalue rewards as a function of delay until receipt.

Risk perceptions

We also assessed participants' attitudes toward behavioral risks as potential covariates in analyses testing associations between alcohol responses and behavioral risks. Risk perceptions have been identified as correlates, for example, of driving after drinking (Bingham, Elliott, & Shope, 2007; Fairlie et al., 2010; McCarthy, Lynch, & Pedersen, 2007). Participants used a 5-point scale where 1 = *not at all likely* and 5 = *very likely* to estimate the likelihood that they *would experience some negative consequences (e.g., become sick, be injured, be embarrassed, suffer legal consequences, or feel bad*

about yourself) if they engaged in each of 13 behavioral risks. Perceived risk ranged from a low of M = 2.22 (SD = 1.46) for gambling to a high of M = 3.67 (SD = 1.50) for potentially unsafe vaginal sex outside a romantic relationship.

Alcohol Challenge Measures

Subjective alcohol responses

We followed Ray and colleagues (2009) in developing a measurement model using multiple self-report measures of the subjective effects of alcohol. Participants completed the 14-item *Biphasic Alcohol Effects Scale* (BAES; Martin et al., 1993; Rueger, McNamara, & King, 2009), the 7-item *Subjective High Assessment Scale* (SHAS; Schuckit et al., 2000), a 3-item *Drug Effects Questionnaire* (Evans & Levin, 2003; Johanson & Uhlenhuth, 1980), a 22-item version of the *Subjective Effects of Alcohol Scale* (SEAS; Morean, Corbin, & Treat, 2013, May 6), and 11 items comprising the *Energetic* and *Intoxicated* subscales of the modified *Profile of Mood States* (POMS; Gabrielli, Nagoshi, Rhea, & Wilson, 1991).

From these questionnaires, we selected the following four scales of the euphoric, stimulant-like effects of alcohol: (1) *BAES Stimulation* (7 items), (2) *POMS Energetic* (4 items), (3) *SEAS High Arousal Positive* (4 items), and (4) *DEQ Like* (single visual analogue scale). We additionally selected the following four scales of the sedative-like effects of alcohol: (1) *BAES Sedation* (7 items), (2) *POMS Intoxicated* (7 items), (3) *SEAS Low Arousal Negative* (3 items), and (4) *SHAS* (7 items). Participants completed the full subjective responses battery at the baseline (except the DEQ), ascending limb, peak, and descending limb assessments. Following King and colleagues (King et al., 2011; Rueger & King, 2013), however, the peak assessment will be our primary assessment of interest. Visual analogue scale (*DEQ* and *SHAS*) responses that were

outside the upper (i.e., right-most) bound of the scale were scored as the maximum possible value on the scale.

Heart rate reactivity

Heart rate response to alcohol, which has been found to be positively associated with the subjective stimulant-like effects of alcohol (Brunelle, Barrett, & Pihl, 2007), was assessed using Omron heart rate monitors (Omron Health Care, Bannockburn, IL). These heart rate monitors are strapped across the chest underneath the clothes and transmit heart rate to wristwatch receivers. They are effective between 30 – 240 beats per minute and accurate within 1 beat per minute. Research assistants held the receivers and recorded heart rates at one-minute intervals for five minutes at each assessment. From the means of these heart rates, we computed the percentage change from baseline for each post-dosage assessment (Assaad et al., 2006b).

Psychomotor impairment

Psychomotor performance was assessed with a *Digit Symbol Substitution Task* (DSST) drawn from the WAIS-III (D. Wechsler, 1997). The DSST requires participants to match numbers with symbols provided in a key, with the total number of correct matches made in 120 seconds serving as the task outcome. The DSST is sensitive to alcohol and has been positively associated with perceptions of impairment in previous research (Brumback, Cao, & King, 2007). Participants completed alternate DSST forms during their two sessions to reduce learning effects.

We also assessed perceived impairment with three items based on those used by Brumback and colleagues (2007): (1) *How impaired do you think you are at present?*, (2) *How unsafe do you think it would be to drive an automobile at present?*, and (3) *If you were at work right now, would others think you were intoxicated or behaving unusually?*

on a 10-point scale from 0 = *not at all* to 10 = *extremely*. In addition, participants reported their willingness *to drive an automobile at present* on the same 10-point scale. This item was based on the index of willingness to drive used by Marczinski and Fillmore (2009).

Response inhibition

As a measure of response inhibition, participants completed a Cued Go/No-Go Task (Marczinski & Fillmore, 2003) on E-Prime software (Schneider, Eschman, & Zuccolotto, 2002) at the peak assessment. Performance on this task is sensitive to alcohol, with intoxication impairing inhibition (Fillmore et al., 2008; Marczinski & Fillmore, 2003), particularly among heavier drinkers (Marczinski, Combs, & Fillmore, 2007). Participants were instructed that the objective of the task was to press the forward slash keyboard button (highlighted with a green sticker) as quickly as possible in response to Go targets (green rectangles) but not No-Go targets (blue rectangles). Each trial began with a fixation point (800 ms) and then a blank screen (500 ms), after which the target was preceded by a cue signaling the likelihood of a Go or No-Go target with 80% probability. The Go and No-Go cues were presented as a black, vertical or horizontal rectangle outlines, respectively, displayed for 1 of 5 stimulus onset asynchronies ranging from 150 – 550 ms. Targets were then displayed for 1000 ms or until the participant responded. The task comprised 125 Go and 125 No-Go trials over approximately 15 minutes, with an inter-trial interval of 700 ms. Fast, accurate responding was encouraged by displaying "incorrect" or the reaction time (RT) following each trial. Participants completed 100 trials of the Cued Go/No-Go Task during the baseline period of each session in order to ensure familiarity with the rules governing the cue-target relationships.

Performance on the Cued Go/No-Go Task can be evaluated in terms of failures to respond to Go targets (i.e., errors of omission), incorrect responses to No-Go targets (i.e., errors of commission), and RTs to Go targets. Errors of omission were relatively rare (92 total errors, or 0.45% of all Go trials) and were therefore not considered here. The primary task outcome of interest was the proportion of No-Go trails in which the participant failed to inhibit the prepotent (Go) response following an Invalid Go cue. We also considered the proportion of inhibition failures following Valid No-Go cues, in addition to RTs on correct responses to Go targets. Following Fillmore and colleagues (e.g., Fillmore, Ostling, Martin, & Kelly, 2009; Marczinski & Fillmore, 2003; Weafer, Fillmore, & Milich, 2009), we identified and removed all outlying trials in which RTs were below 100 ms (101 trials, or 0.49% of all non-omission Go trials) or above 1000 ms (0 trials) before calculating participants' average RTs. We did not remove trials with low RTs in calculating proportions of inhibition failures.

Placebo manipulation check

We assessed the efficacy of the placebo manipulation by asking participants to *estimate the number of standard alcoholic drinks [they] were served during this experiment* at the start of the ascending limb assessment.

Follow-Up Daily Self-Monitoring Measures

Participants were invited via email to complete the brief daily self-monitoring survey (regarding behavior from the previous day and night) each morning during their 21-day follow-up periods. The survey assessed relationship status, alcohol use, alcohol responses, and behavioral risks and other alcohol-related consequences.

Alcohol consumption

On each daily survey, participants reported how many standard drinks (i.e., 12 oz of beer, 5 oz of wine, or 1.5 oz of liquor in a shot or mixed drink) they consumed. For surveys in which they reported any alcohol consumption, participants then reported how many discrete drinking occasions they had that day and the duration (in hours) of the heaviest occasion. Drinking episodes of duration *less than 1* hour were coded as 0.5 hours. Using gender, weight (obtained from the dose calculation assessment at the second laboratory session), and drinking quantity and duration, we computed *estimated blood alcohol concentrations* (eBACs; Matthews & Miller, 1979) for each drinking episode using the *balcalc* Stata .ado file. Leeman and colleagues (2010) have recommended the eBAC method for measuring alcohol consumption when BrACs are not available. Though imperfect (S. Grant, LaBrie, Hummer, & Lac, 2012), eBACs are strongly associated with BrACs, particularly at BrACs below .08 g%, and have been used in multiple event-level studies (Hustad & Carey, 2005; Neal & Carey, 2007; Neal & Fromme, 2007; Quinn et al., 2013; Ray, Miranda et al., 2010).

For drinking days, participants also reported how many of the standard drinks they consumed contained *energy drinks (e.g., vodka and red bull, Jagerbomb)*, how many contained *other caffeinated beverages (e.g., rum and coke, Irish coffee)*, and how many energy drinks they consumed independent of their alcohol consumption. They also reported whether they used *any tobacco products (e.g., cigarettes, cigars, pipes, smokeless tobacco)* during drinking episodes. We also asked participants to report where and with whom they drank. Participant selected as many choices as were applicable from lists of drinking locations (i.e., *my own residence*, *my friend(s)' residence*, *my parent(s)' home, a bar or club, a restaurant or café, a recreation event (e.g., sports, music, festival)*, or *other*) and social contexts (i.e., *I was alone, my boyfriend/girlfriend/partner, close*

friends, *acquaintances (people I know who aren't my close friends)*, *people I don't know*, *parent(s)*, *brother(s) or sister(s)*, *other family members*, or *other*). These questions were intended to examine how alcohol responses may vary as a function of social context (Ray, Miranda et al., 2010) and co-ingestion with other substances (Ferreira, De Mello, Pompéia, & De Souza-Formigoni, 2006; Marczinski & Fillmore, 2006; Marczinski, Fillmore, Bardgett, & Howard, 2011; Piasecki et al., 2011) and were beyond the scope of the present hypotheses. Results are not reported here.

Daily alcohol responses

Participants reported their drinking-episode-level alcohol responses using the Brief Biphasic Alcohol Effects Scale (BBAES), which comprises three-item subjective *stimulation* and *sedation* scales drawn from BAES items (*energized*, *excited*, and *up* and *sedated*, *slow thoughts*, and *sluggish*, respectively). The BBAES scales, which use the same response options as the full BAES, have demonstrated strong internal consistency, similar factor structure to the BAES, strong correlations with the full scales, and predictive validity for binge drinking (Rueger & King, 2013; Rueger et al., 2009). To provide consistency with our previous event-level studies, we also asked participants to rate their *subjective intoxication* by responding to the question *how drunk did you feel?* on a scale from 0 = *not drunk at all* to 10 = *extremely drunk* (Quinn & Fromme, 2011a, 2012; Quinn et al., 2013).

Behavioral risks

We used an expanded version of the questionnaire developed by Neal and Fromme (2007) to assess the following behavioral risks: gambling, illicit drug use, driving after drinking, riding with a driver who had been drinking, destroying property or stealing, arguing verbally, physically fighting, and oral, vaginal, or anal sex. When

participants endorsed any sexual behavior, they were asked whether the behavior was with a romantic partner, whether they regretted the behavior, and whether the behavior was potentially unsafe (e.g., for vaginal sex, *you did not use a condom or other protection against STDs and pregnancy*).[4] For each behavioral risk, participants reported whether they a) *did not engage in activity*, b) *engaged in activity when [they] had NOT been drinking*, or c) *engaged in activity when [they] HAD been drinking*. These response options allowed us to establish the temporal precedence of alcohol consumption relative to the behavioral risks.

Alcohol-related consequences

Finally, on surveys in which they reported alcohol consumption, participants also reported whether they experienced any of eight alcohol-related negative consequences, ranging from *social (e.g., felt rejected or hurt your reputation)* to *sickness (e.g., felt sick, vomited)* to *injured someone else* or *injured yourself or were injured*. We aggregated these consequences such that 1 = *endorsement of any consequence* and 0 = *endorsement of no consequences*. Participants also reported whether they experienced consequences related to blackout (*had difficulty remembering things you said or did or events that happened last night*) and hangover (*e.g., the next day, had a headache, felt sick, vomited*).

[4] Of the 111 instances of oral sex observed during follow-up, participants reported that 78 were unsafe but that only 7 occurred with someone other than a romantic partner and 1 was regretted (safety, romantic partner, and regret were not reported for 1 oral sex observation each). Similarly, of the 161 instances of vaginal sex observed during follow-up, participants reported that 60 were unsafe but that only 9 occurred with someone other than a romantic partner and 1 was regretted (safety, romantic partner, and regret were reported for all vaginal sex observations). Finally, only 1 instance of anal sex was observed during follow-up. We therefore analyzed unsafe oral and vaginal sex only.

Chapter 4: Analytic Approach

DATA MANAGEMENT

We examined descriptive statistics and distributions for all study variables: personality, subjective response to early drinking experiences, recent alcohol consumption, laboratory alcohol response and Cued Go/No-Go Task outcomes, and event-level follow-up data. As described elsewhere, when variable distributions deviated from normality, analyses were tested for robustness to transformation or removal of outliers. We found minimal missing data for the laboratory measures, which reduced some analytic sample sizes below the $N = 82$ included participants. For our primary laboratory analyses in M*plus* version 5 (Muthén & Muthén, 1998-2007), however, we used full-information maximum likelihood estimation, which allowed us to analyze all available data, including that from participants with incomplete data on some variables (Schafer & Graham, 2002). Similarly, our Generalized Estimating Equations analyses of the event-level follow-up in Stata version 11.0 permitted the inclusion of participants who did not provide complete follow-up data.

ANALYSES

We used several analytic approaches to test study hypotheses. Prior to testing the primary associations of interest, we conducted a series of repeated measures ANOVAs in SPSS version 15.0 to compare manipulation checks, Cued Go/No-Go Task performance, and alcohol responses across the counterbalanced alcohol and placebo condition sessions. For the placebo manipulation check, DSST performance, and perceived intoxication and willingness to drive—each of which was assessed only once per session—these ANOVAs comprised within-person comparisons between the alcohol and placebo conditions. Cued Go/No-Go Task performance was evaluated by RTs for correct

responses in the Valid Go and Invalid No-Go conditions and proportions of inhibition failures in the Invalid Go and Valid No-Go conditions. We tested for alcohol-induced impairment in RT and inhibition using 2 × 2 (beverage × cue) repeated measures ANOVAs. A final series of beverage × limb repeated measures ANOVAs compared alcohol- and placebo-condition HRR and subjective alcohol responses across the baseline, ascending limb, peak, and descending limb assessments.

Alcohol Responses and Inhibitory Impairment

Our first research objective was to test the associations between alcohol responses and alcohol-induced impairment of response inhibition, as measured by the proportion of inhibition failures in the Cued Go/No Go Task's Invalid Go cue condition. Given the large number of subjective alcohol response measures, we used a measurement model approach based on the factor structure described by Ray and colleagues (Ray et al., 2009). Doing so fit the dual purpose of reducing the number of necessary statistical tests and reducing error in the measurement of alcohol responses. We fit measurement models in M*plus* using placebo-adjusted alcohol response difference scores from the peak of the BAC curve. Subsequent analyses assessed alcohol responses using either the measurement model itself (for analyses in M*plus*) or factor scores obtained from the final measurement model (for analyses in Stata).

In order to test whether individuals who experienced greater alcohol responses also experienced greater alcohol-induced inhibitory failures, we fit a model in which alcohol responses were permitted to covary with alcohol-condition proportions of inhibition failures. We also regressed these inhibition failures on placebo-condition proportions of inhibition failures. Doing so meant that our covariation paths of interest represented alcohol responses' association with the residual variation in alcohol-

condition performance not shared with placebo-condition performance, which effectively controlled for individual differences in task performance not attributable to alcohol intoxication. Prior to fitting this model, we also considered inclusion of other potential covariates (e.g., gender) by examining associations with Cued Go/No-Go Task performance.

Alcohol Responses and Alcohol-Related Negative Consequences

Our second primary research objective was to test whether alcohol responses, as measured in the laboratory-based alcohol challenge, were associated with intoxicated behavioral risk-taking in event-level follow-up of behavior in the natural environment. Because most participants provided multiple drinking occasions during follow-up, we tested these associations using Generalized Estimating Equations (GEE; Hardin & Hilbe, 2003), which permit observations nested within individuals, in Stata 11.0 (StataCorp, 2009). We specified an autoregressive correlation structure to account for event-to-event, within-person dependence (Neal & Carey, 2007; Neal & Fromme, 2007), and we estimated standard errors using Stata's robust option. This correlation structure requires a minimum of two observations per participant, which excluded two participants who provided only one drinking occasion each. Because outcomes were dichotomous indicators of experiencing or not experiencing the behavior or other alcohol-related consequence, we specified the binomial reference distribution and logit link. We estimated models predicting each behavior—in addition to blackouts, hangovers, and other alcohol-related consequences (aggregated)—separately.

In order to isolate within-person, drinking-episode-level associations between changes in (objective) alcohol intoxication and the propensity to engage in behavioral risks, we employed a person-centered approach for eBAC (Enders & Tofighi, 2007;

Raudenbush & Bryk, 2002). We included person-mean-centered *daily eBAC* variables (eBAC – person-mean eBAC), which assessed within-person deviations from typical levels of alcohol consumption. We also included person-mean *average eBAC* variables to assess between-persons individual differences in typical alcohol consumption.

In each model, controlling for daily and average eBAC, we tested whether person-level alcohol responses (as assessed with the factor scores obtained from the measurement model) were associated with outcomes as well as whether they moderated the associations between daily eBAC and outcomes. In cases where alcohol responses amplified associations between daily eBAC and outcomes, we then tested mediation by including placebo-adjusted alcohol-induced inhibitory impairment scores from the Cued Go/No-Go Task as predictors in the models.

We made a number of efforts, consistent with prior research, to ensure quality control of the event-level data. Only 5 participants completed fewer than the maximum of 21 daily self-monitoring surveys. Of these, we excluded data from the 2 participants who provided fewer than 50% of the surveys (10 observations combined). We also excluded 2 observations missing eBAC and 4 with eBAC $\geq$ .40 g%, resulting in a sample of 80 participants with 1,668 total observations, of which participants consumed any alcohol on 598 observations. Because laboratory alcohol responses were our primary independent variables of interest, our models only included days on which eBACs exceeded .00 g% (the 18 drinking-day observations on which participants consumed alcohol but did not reach a measureable eBAC were excluded).

When participants reported that drinking-day behavioral risks occurred when they had *not* been drinking, we recoded eBAC values to .00, meaning that these observations were excluded as well. Of the analyzed behavioral risks that occurred on drinking days, participants reported that 33.80% of illicit drug use, 7.29% of riding with a drinking

driver, 52.38% of unsafe oral sex, 62.50% of unsafe vaginal sex, 33.33% of verbal aggression, and 7.29% of driving after drinking occurred when they had *not* been drinking. Actual analytic sample sizes therefore varied as a function of drinking day definitions, in addition to missingness on other variables. Models additionally controlled for gender and person-mean-centered monitoring day to test for assessment reactivity. Other potential between-persons covariates, including socio-economic status, family history of alcohol problems, age at first drink, and risk perceptions, were considered on the basis of their bivariate associations with outcomes.

Finally, we also estimated models that included person-mean-centered daily subjective alcohol response measures (i.e., BBAES Stimulation and Sedation) taken from the daily surveys. These models tested whether within-person, episode-to-episode variation in stimulant-like and sedative-like alcohol responses—controlling for episode-to-episode variation in eBAC—was associated with behavioral risks and other alcohol-related negative consequences.

Alcohol Responses and Disinhibited Personality

We then tested whether global associations between alcohol responses and behavioral risks reflected common underlying contributions from disinhibited personality facets to both alcohol responses *and* behavioral risks. We covaried alcohol responses (using the measurement model) with measures of disinhibited personality using structural equation modeling. We then examined between-persons correlations between disinhibited personality and the behavioral risks. Where these associations were significant, we then tested GEE models in which the global association between alcohol responses and behavioral risks was explained by personality facets as third-variable confounds.

Chapter 5: Results

ALCOHOL CHALLENGE

Placebo Manipulation Check

Complete data for the placebo manipulation check were available for 81 participants. One participant did not estimate the number of standard drinks consumed during the alcohol session. As expected, participants estimated that they consumed more drinks in the alcohol condition than in the placebo session. See Table 2, which presents results for all beverage condition comparisons, below. In the alcohol condition, participants estimated that they consumed 3.44 standard drinks (*SD* = 1.33, range: 1 – 8, 95% Confidence Interval [CI]: 3.15, 3.74). In the placebo condition, participants estimated that they consumed 2.09 standard drinks (*SD* = 1.05, range: 0 – 4, 95% CI: 1.86, 2.32). It is important to note that the CI for the placebo condition did not include zero, and only four participants estimated that they consumed zero drinks in placebo sessions. In sum, the placebo manipulation was efficacious. See Figure 3.

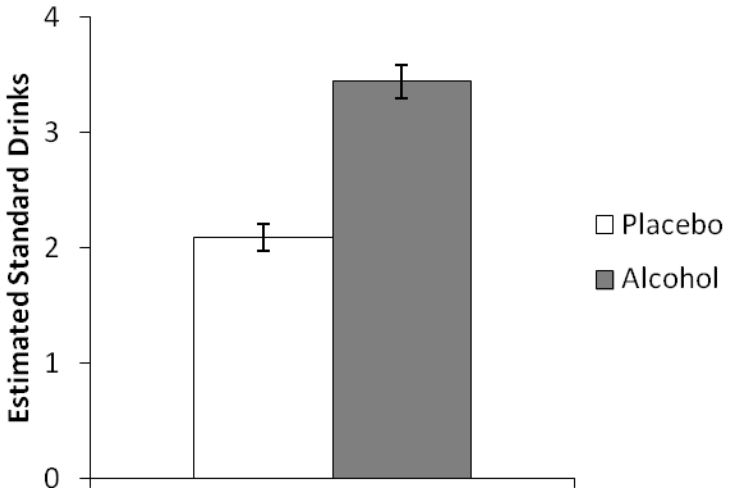

Figure 3: Estimated standard drinks consumed in alcohol and placebo condition sessions (*n* = 81). Bars represent standard errors.

Variable	Comparison	Degrees of Freedom	*F*	η_p^2
Beverage Condition Comparisons				
Placebo manipulation check	**Beverage**	**1, 80**	**79.45**	**.50**
Digit symbol substitution task	**Beverage**	**1, 81**	**44.23**	**.35**
Perceived impairment	**Beverage**	**1, 81**	**181.24**	**.69**
Willingness to drive	**Beverage**	**1, 81**	**40.23**	**.33**
Cued Go/No-Go Task Comparisons				
Reaction time	**Beverage**	**1, 81**	**18.40**	**.19**
	Cue (Valid vs. Invalid)	**1, 81**	**111.65**	**.58**
	Beverage × Cue	1, 81	3.74	.04
Proportion of inhibitory failures	**Beverage**	**1, 81**	**13.16**	**.14**
	Cue (Valid vs. Invalid)	**1, 81**	**52.17**	**.39**
	Beverage × Cue	**1, 81**	**6.45**	**.07**
Natural log transformed proportion of inhibitory failures	**Beverage**	**1, 81**	**12.52**	**.13**
	Cue (Valid vs. Invalid)	**1, 81**	**39.15**	**.33**
	Beverage × Cue	1, 81	1.28	.02
Limb-Varying Response Comparisons				
Heart rate reactivity (HRR)	**Beverage**	**1, 80**	**48.30**	**.38**
	Limb	**1.55, 124.16**	**13.39**	**.14**
	Beverage × Limb	1.78, 142.06	2.25	.03
BAES Stimulation	**Beverage × Limb**	**2.24, 181.36**	**19.23**	**.19**
POMS Energetic	**Beverage × Limb**	**2.64, 211.23**	**7.31**	**.08**
SEAS High Arousal Positive	**Beverage × Limb**	**2.26, 183.38**	**21.02**	**.21**
DEQ Like Drug	**Beverage × Limb**	**1.54, 124.42**	**24.54**	**.23**
BAES Sedation	**Beverage × Limb**	**1.84, 149.41**	**16.35**	**.17**
SHAS	**Beverage × Limb**	**1.81, 146.71**	**60.60**	**.43**
POMS Intoxicated	**Beverage × Limb**	**2.23, 178.13**	**30.66**	**.28**
SEAS Low Arousal Negative	**Beverage × Limb**	**2.15, 174.14**	**28.49**	**.26**

Table 2: Summary of comparisons between alcohol and placebo condition responses.

Note. Bolded comparisons were significant, $p < .05$. DEQ-Like not assessed at baseline, and HRR assessed as change from baseline at ascending, peak, and descending limbs. Where beverage × limb comparisons were significant, main effects are not shown. *n*s = 81 for placebo manipulation check, HRR, and POMS. All limb and beverage × limb comparisons employed a Greenhouse-Geisser correction to degrees of freedom.

Breath Alcohol Concentration

As expected, breath alcohol concentrations (BrACs) increased during alcohol condition sessions from baseline through the peak assessment before beginning to decline. The average BrAC was close to but slightly exceeded the target dose (.08 g%). Beyond the mean-trend displayed in Table 3, there was substantial variation in BrACs. Figure 4 presents each individual's BrAC trajectory. No placebo BrAC exceeded .000.

Assessment Point	Observed Range	*M*	*SD*
Baseline	--	.000	.000
Ascending limb	.041 – .102	.064	.015
Peak	.054 – .129	.083	.015
Post-peak	.052 – .118	.079	.013
Descending limb	.041 – .109	.070	.012

Table 3: Breath alcohol concentration (g%) summary statistics from alcohol sessions.

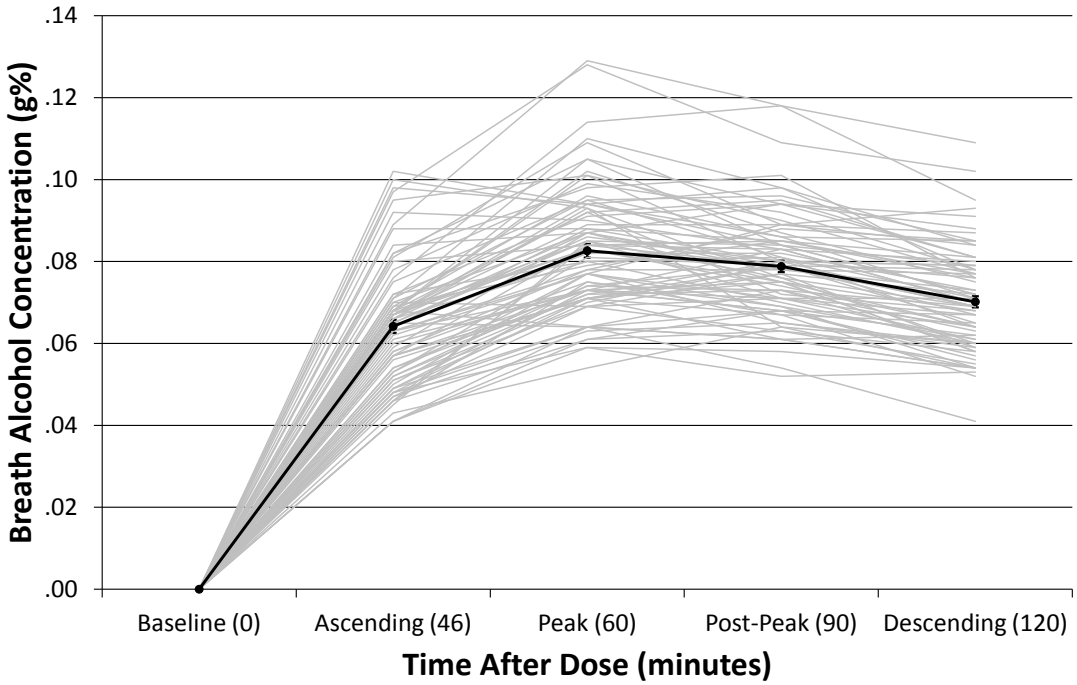

Figure 4: Individual (grey) and mean (black) breath alcohol concentrations as a function of time from onset of dose in alcohol condition. Bars represent standard errors.

Alcohol-Induced Inhibitory Impairment

The Cued Go/No-Go Task, which was used as a measure of alcohol-induced impairment of response inhibition, produced reaction times and inhibitory failures as outcome measures. Reaction times were available in response to targets preceded by Valid Go cues and Invalid No-Go cues, whereas inhibitory failures are available in response to targets preceded by Invalid Go cues and Valid No-Go cues. In a 2 × 2 repeated measures ANOVA, reaction times were slower in the alcohol condition and following Invalid No-Go cues. The alcohol-induced increase in reaction times did not differ as a function of cue condition. Removing one case with outlying alcohol condition reaction times resolved high skew (≥ 2.30) and kurtosis (≥ 10.86) without substantively affecting the findings; results from the whole sample are therefore presented in Figure 5.

Of particular interest for the present analyses was the primary outcome used in previous studies to assess alcohol-induced inhibitory impairment, the proportion of responses that were incorrect (i.e., inhibitory failures) following Invalid Go cues. This outcome assessed the extent to which individuals were unable to inhibit a prepotent (i.e., cued) response. As expected, participants made more inhibitory failures in the Invalid Go condition relative to the Valid No-Go condition, and alcohol increased inhibitory failures. Moreover, replicating previous research, a significant beverage × cue interaction indicated that the significant placebo-alcohol difference in inhibitory failures was stronger in the Invalid Go condition. However, an examination of the distributions suggested that the proportion of inhibitory failures was not normally distributed, skew ≥ 1.76, kurtosis ≥ 2.94. We therefore repeated this 2 × 2 repeated measures ANOVA using log-transformed inhibitory failure scores. We again found significant main effects of beverage and cue, with more inhibitory failures in the alcohol and Invalid Go conditions. However, the beverage × cue interaction was no longer significant. In sum, as shown in

Figure 5, although alcohol-induced failures of response inhibition appeared more prevalent in the Invalid Go condition, this difference may have been driven by a small number of participants with a large number of inhibition failures, meaning that the interaction should be interpreted with caution.

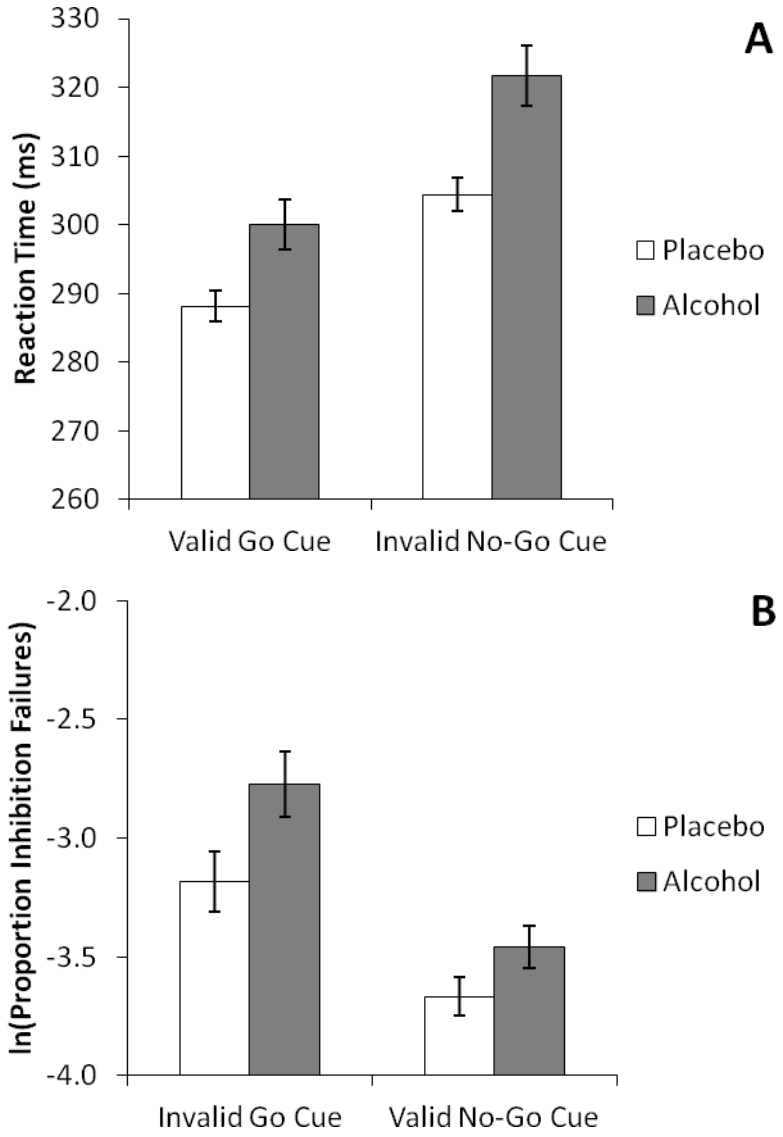

Figure 5: Cued Go/No-Go Task reaction times (Panel A) and natural-log transformed proportions of inhibition failures (Panel B). Bars represent standard errors.

Objective and Subjective Responses to Alcohol

Psychomotor impairment

The Digit Symbol Substitution Task (DSST) was included at the peak assessment as a measure of alcohol-induced psychomotor impairment. As expected, participants provided fewer correct responses to the DSST in the alcohol condition relative to the placebo condition. See Figure 6.

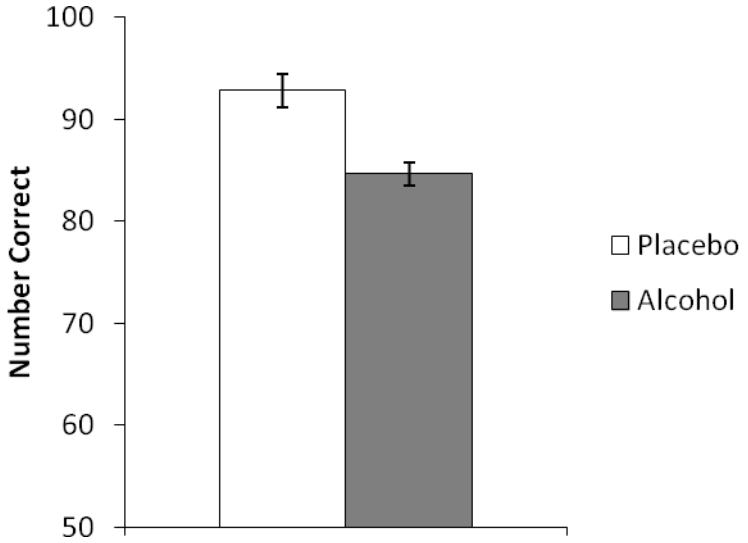

Figure 6: Correct responses on the Digit Symbol Substitution Task. Bars represent standard errors.

Heart rate reactivity

Heart Rate Reactivity (HRR) was greater in the alcohol condition relative to the placebo condition across all three post-dosage assessments (Ascending Limb, Peak, and Descending Limb). A non-significant beverage × limb interaction indicated that the alcohol-induced increase in HRR was relatively constant across limbs relative to placebo. See Figure 7. It should be noted, however, that deviations from normality were detected in HRR and the subjective alcohol response measures presented below. Because, unless otherwise noted, these deviations were not present in the difference scores used in the

primary analyses described below, the raw data are presented here for descriptive purposes.

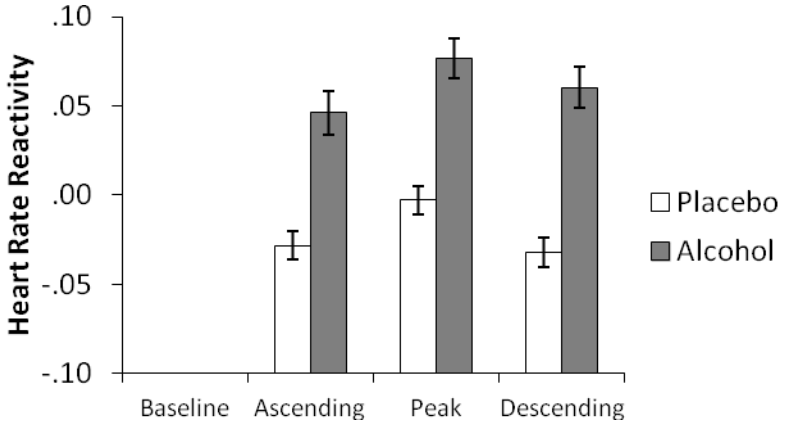

Figure 7: Heart rate reactivity from baseline in response to alcohol and placebo. Bars represent standard errors.

Perceived impairment and willingness to drive

As described above, alcohol decreased scores on measures requiring psychomotor performance, such as the DSST. This effect persisted when examining levels of *perceived* impairment, with participants reporting greater perceived impairment after consuming alcohol relative to placebo. Moreover, participants reported being more willing to drive, on average, in the placebo condition relative to the alcohol condition. See Figure 8.

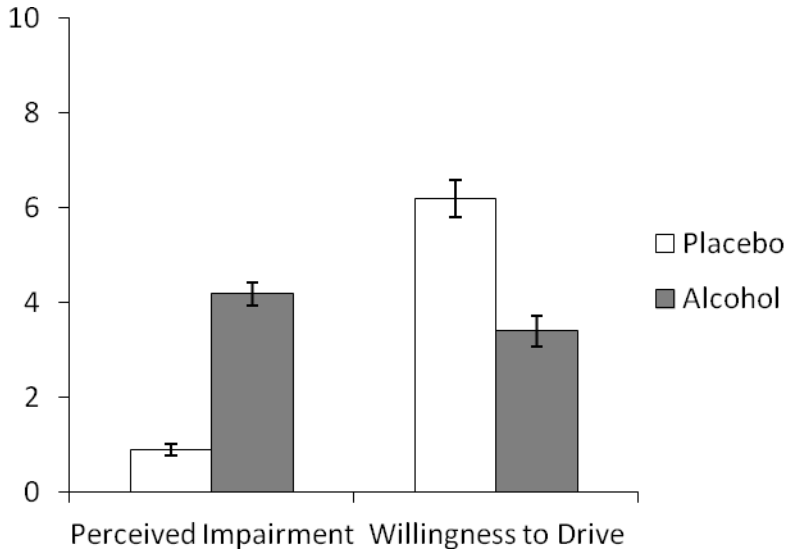

Figure 8: Perceived impairment and willingness to drive after consuming alcohol and placebo. Bars represent standard errors.

Subjective alcohol responses

The next series of comparisons tested whether responses to measures identified *a priori* as indices of alcohol's subjective stimulant-like effects (BAES Stimulation, POMS Energetic, SEAS High Arousal Positive, DEQ Like Drug) were greater in the alcohol condition relative to the placebo condition. There were significant beverage × limb interactions for all four measures. As shown in Figure 9, alcohol responses appeared to exceed placebo responses most strongly on the ascending limb and at peak.

Similarly, there were significant beverage × limb interactions for all four measures identified *a priori* as indices of alcohol's subjective sedative-like effects (BAES Sedation, POMS Intoxicated, SEAS Low Arousal Negative, SHAS). Although participants reported less sedation than stimulation, alcohol-induced sedation appeared to persist through the descending limb when compared with placebo. See Figure 10.

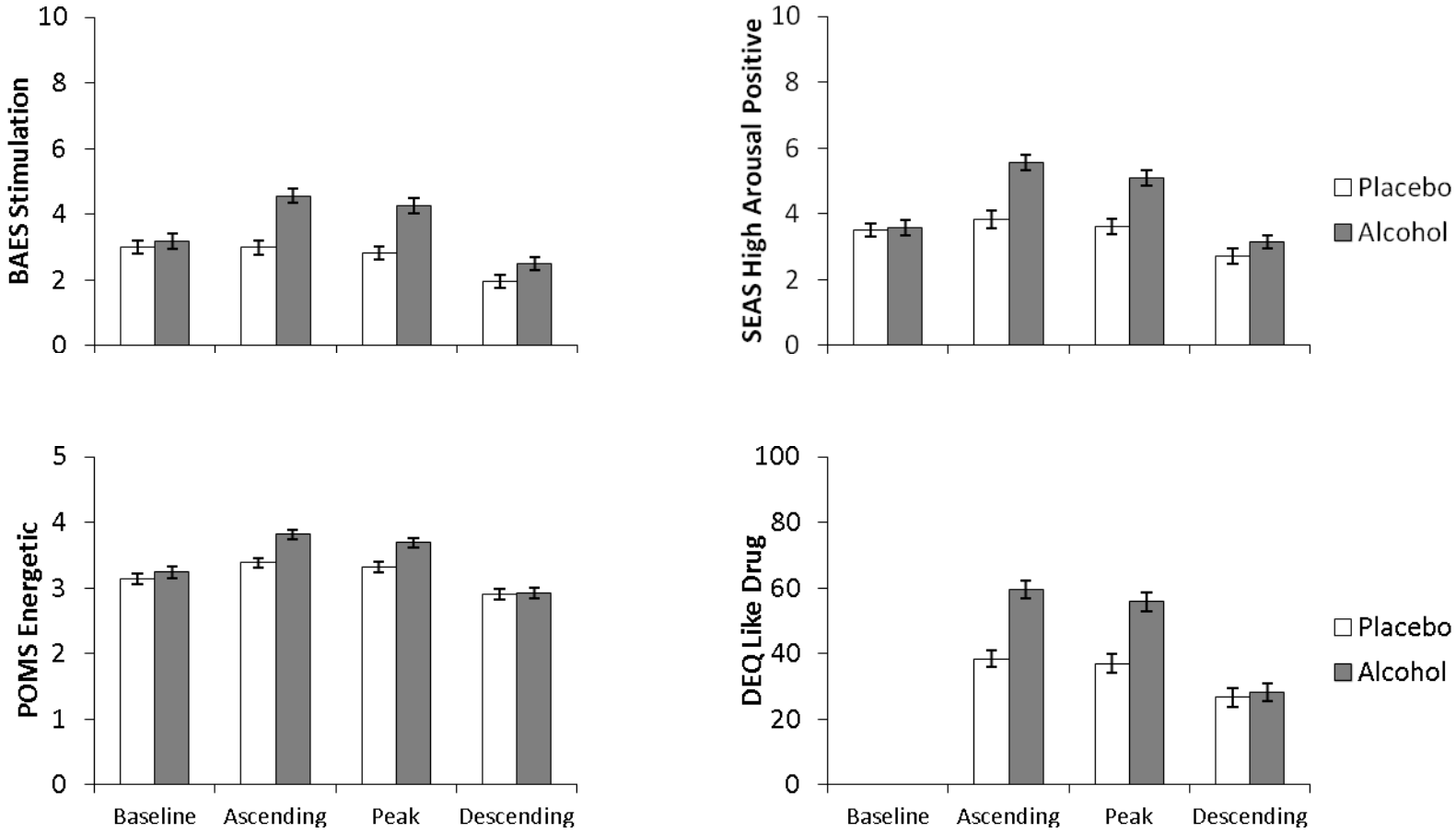

Figure 9: Subjective stimulation in response to alcohol and placebo. Bars represent standard errors.

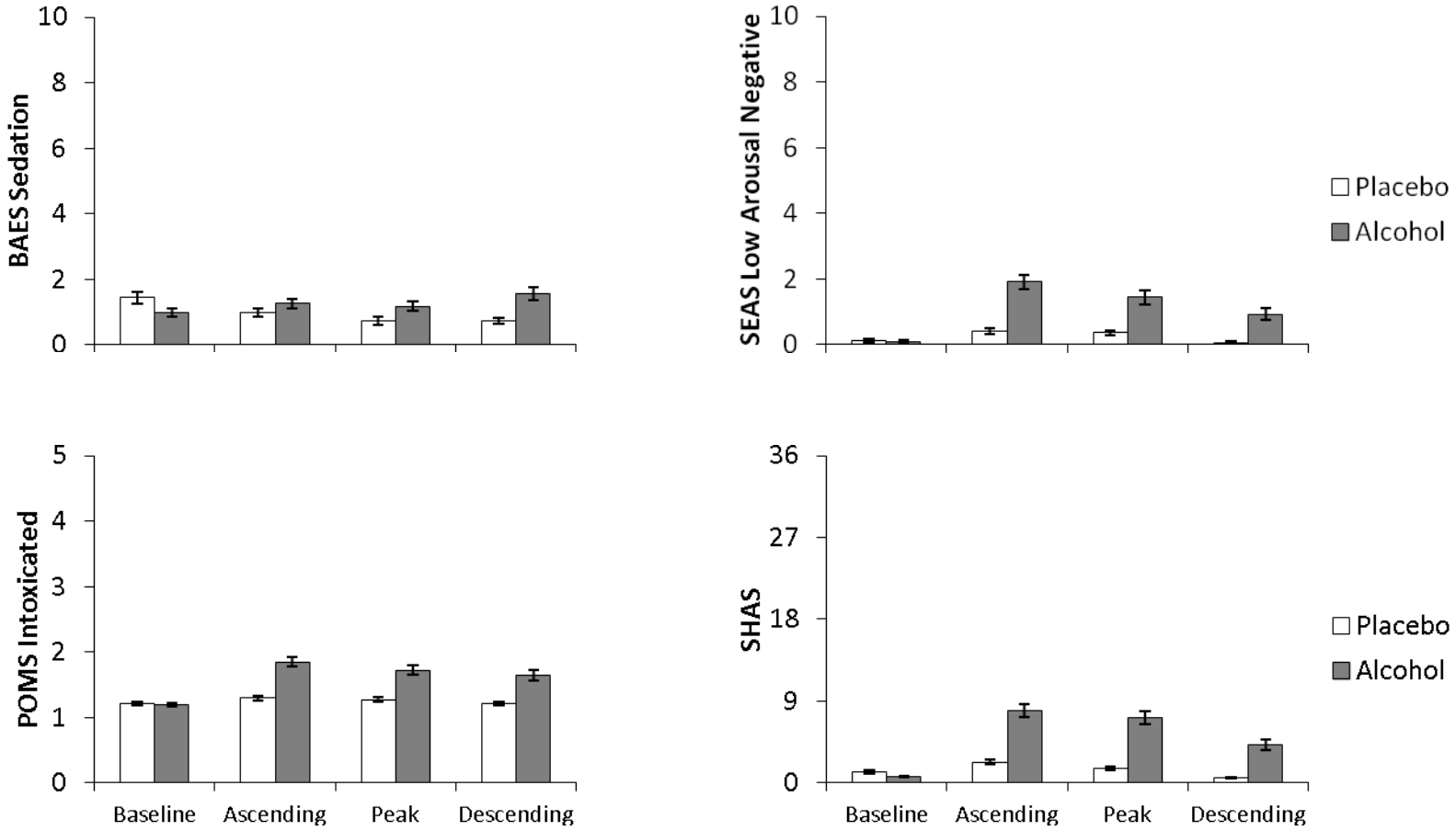

Figure 10: Subjective sedation in response to alcohol and placebo. Bars represent standard errors.

Alcohol Response Measurement Model

After examining beverage condition differences in Cued Go/No-Go Task performance and objective and subjective alcohol responses, our next analytic step was to attempt to fit an alcohol response measurement model similar to that found by Ray and colleagues (2009). In order to prepare alcohol response data for the measurement model, we computed placebo-adjusted Peak change scores for mean scores on each of the measures of interest. For most of the subjective measures, these scores were computed as follows: alcohol response (peak – baseline) – placebo response (peak – baseline). As used by King and colleagues (2011), these values represent the change in alcohol responses from baseline to the Peak assessment that is beyond the change produced by the placebo beverage. Where baseline scores were not available (i.e., DEQ Like Drug, HRR, DSST), we computed change scores as the difference between alcohol and placebo scores. Greater scores on all measures indicated greater alcohol responses, with the exception that greater DSST scores indicated better performance. Summary statistics and bivariate correlations for the placebo-adjusted alcohol response scores are presented in Table 4.

The difference scores were then fit with a series of measurement models. Because Cued Go/No Go Task inhibitory failures and placebo-adjusted SEAS Low Arousal Negative scores exceeded 3 in kurtosis, we fit all models in M*plus* using a robust estimator (Estimator = MLR). We expected, *a priori*, that a two-factor model would fit the data well, with one factor representing stimulant-like effects and the other factor representing sedative-like effects. Although Ray and colleagues (2009) did not include objective alcohol response indices in their measurement model, we also considered models including HRR and DSST scores.

Variable	Observed Range	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11	12
1. Cued Go/No-Go Task inhibitory impairment	-0.32 – 0.48	0.04	0.11	--											
Subjective stimulation															
2. BAES Stimulation	-4.29 – 9.71	1.27	2.42	.12	--										
3. POMS Energetic	-1.75 – 2.75	0.27	0.93	**.27**	**.65**	--									
4. SEAS High Arousal Positive	-3.50 – 8.25	1.41	2.30	.08	**.69**	**.66**	--								
5. DEQ Like	-42.45 – 84.17	18.86	27.36	.04	**.26**	.20	**.32**	--							
Subjective sedation															
6. BAES Sedation	-4.71 – 6.71	0.89	1.78	.02	-.18	**-.43**	**-.31**	-.06	--						
7. POMS Intoxicated	-0.57 – 2.71	0.47	0.61	.06	-.15	**-.39**	-.11	.06	**.59**	--					
8. SEAS Low Arousal Negative	-1.33 – 7.67	1.13	1.88	-.12	.13	-.14	.19	.15	**.50**	**.71**	--				
9. SHAS	-4.14 – 28.35	6.16	6.35	.10	**.22**	-.06	**.24**	**.28**	**.51**	**.70**	**.77**	--			
Objective alcohol responses															
10. Heart rate reactivity	-0.12 – 0.49	0.08	0.11	.20	-.06	-.16	-.12	-.14	.21	**.30**	.21	**.30**	--		
11. Digit symbol substitution task	-38.00 – 34.00	-8.20	11.16	**.36**	.12	.19	.11	.04	-.11	-.20	**-.26**	-.09	.00	--	
12. Perceived impairment	-1.67 – 7.67	3.28	2.21	.02	**.34**	.08	.20	.18	**.29**	**.57**	**.57**	**.74**	**.33**	-.09	--
13. Willingness to drive	-10.00 – 10.00	-2.79	3.99	.18	-.07	.13	.03	-.01	**-.27**	**-.31**	**-.32**	**-.29**	-.11	-.01	**-.39**

Table 4: Placebo-adjusted peak alcohol response summary statistics and bivariate correlations.

Note. Bolded correlations are significantly different from zero, $p < .05$. *n*s = 81 for POMS and heart rate reactivity.

We began with a preliminary model in which all eight subjective and both objective responses loaded onto a single latent factor. As displayed in Table 5 and suggested by the correlations in Table 4, this model did not fit the data well. Indeed, DSST performance and—to a greater extent—HRR did not appear to correspond well with subjective sedation and stimulation, respectively. We therefore repeated the preliminary model without the objective measures. Again, however, this model fit the data poorly. We next fit the hypothesized two-factor model of subjective responses. Although this model fit the data better than did the single-factor model, overall fit was still poor. Modification indices suggested that this poor fit may have been due to untested cross-loadings, and permitting two subjective sedation measures (BAES Sedation and POMS Intoxicated) to cross-load onto the subjective stimulation factor significantly improved model fit. However, the χ^2 and RMSEA both indicated that fit could be improved. We therefore fit a final measurement model retaining the above specifications but also permitting POMS Energetic to cross-load onto the subjective sedation factor.

As shown in Table 5, this final model fit the data well. The subjective stimulation factor was well defined, with loadings of .80 or above for BAES Stimulation, SEAS High Arousal Positive, and POMS Energetic. Similarly, the subjective sedation factor had loadings of .87 or above for SHAS, SEAS Low Arousal Negative, and POMS Intoxicated. The cross-loadings were all negative, indicating that higher scores on BAES Sedation and POMS Intoxicated were associated with lower stimulation, whereas higher scores on POMS Energetic were associated with lower sedation. The two factors were moderately but not significantly correlated, $r = .28$, $p = .08$. See Figure 11.

A model in which the two factors were permitted to covary with HRR and DSST performance also fit well, χ^2 (28) = 23.88, $p = .69$, CFI= 1.00, RMSEA = .00. Neither subjective stimulation ($\beta = -.08$, $p = .52$; $\beta = .13$, $p = .31$) nor sedation ($\beta = .30$, $p = .06$; β

= -.17, p = .12), however, was associated with HRR or the DSST, respectively. This result provided further evidence that the objective indices could be distinguished from subjective stimulation and sedation.

Model	Description	χ^2 (*df*)	CFI	RMSEA	AIC	$\Delta\chi^2$ (*df*)[a]
Preliminary	Single Factor, including HRR and DSST	249.10* (35)	.32	.27	3452.40	--
1	Single factor, Subjective only	262.72* (20)	.16	.39	2946.29	--
2	Stimulation and Sedation factors	75.05* (19)	.81	.19	2837.89	--[b]
3	Model 2 plus POMS Intoxicated and BAES Sedation cross-loadings	29.07* (17)	.96	.09	2798.08	135.30* (2)
4	**Model 3 plus POMS Energetic cross-loading**	**12.29 (16)**	**1.00**	**.00**	**2782.79**	**--[b]**

Table 5: Measurement model fit statistics.

Note. Bolded model was selected for subsequent analyses. * $p < .05$.
[a] Satorra-Bentler scaled chi-square difference score.
[b] Scaled chi-square difference score was negative; statistical significance of model comparison could not be computed.

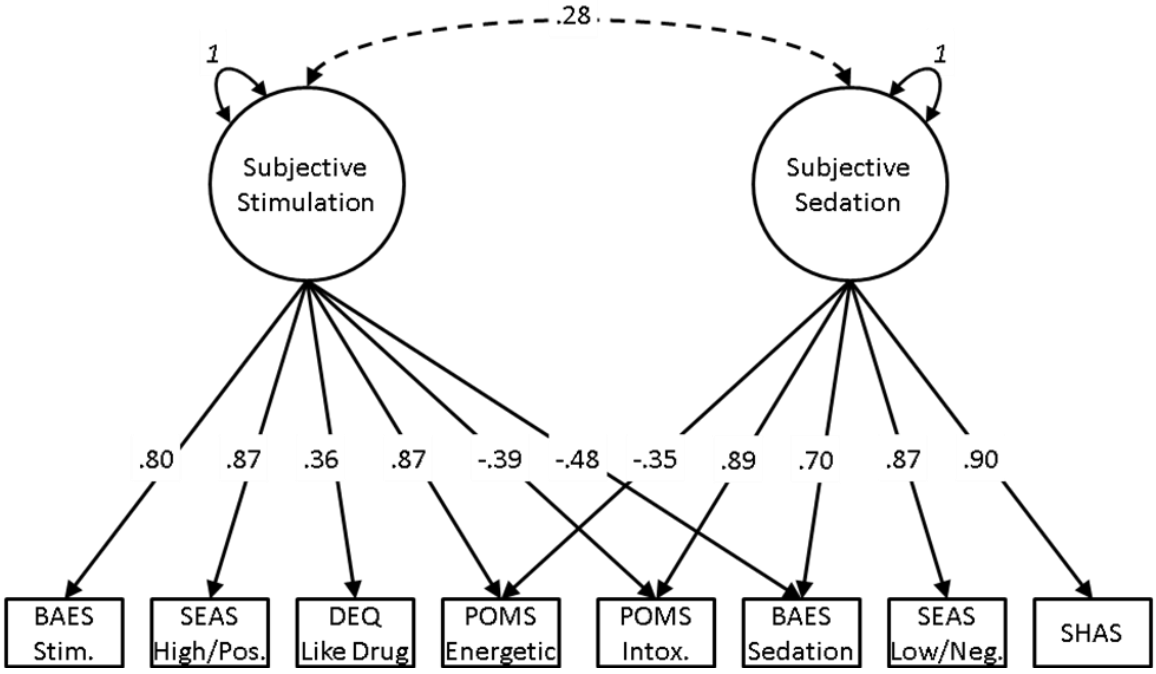

Figure 11: Measurement model of subjective alcohol responses.

Note. Residual variances not shown. Standardized loadings, correlations, and *p*-values were taken from M*plus* STDYX output. Solid paths indicate $p < .05$; dashed path indicates $p = .08$. Italicized values indicate model constraints.

Alcohol Responses and Inhibitory Impairment

The primary goal of the laboratory phase of this project was to evaluate the association between stimulant-like and sedative-like alcohol responses and alcohol-induced inhibitory impairment as measured by the Invalid Go cue condition of the Cued Go/No-Go Task. In order to test these associations, we fit a model in which the factors identified in the measurement model were permitted to covary with inhibitory failures, in addition to HRR and DSST scores. Prior to doing so, we fit a series of models to identify potential background covariates. Given concerns about the number of parameters given the sample size, we first fit models covarying alcohol-condition and placebo-adjusted inhibitory failures with gender, BrAC, alcohol consumption, and family history of

alcohol problems without including the alcohol responses. Cued Go/No-Go Task inhibitory failures were not associated with BrACs at peak, Timeline Follow-Back total drinks consumed (excluding two outliers as described above), or family history of alcohol problems. Inhibitory failures were, however, more common among men. We therefore included gender as an exogenous covariate in all subsequent models.

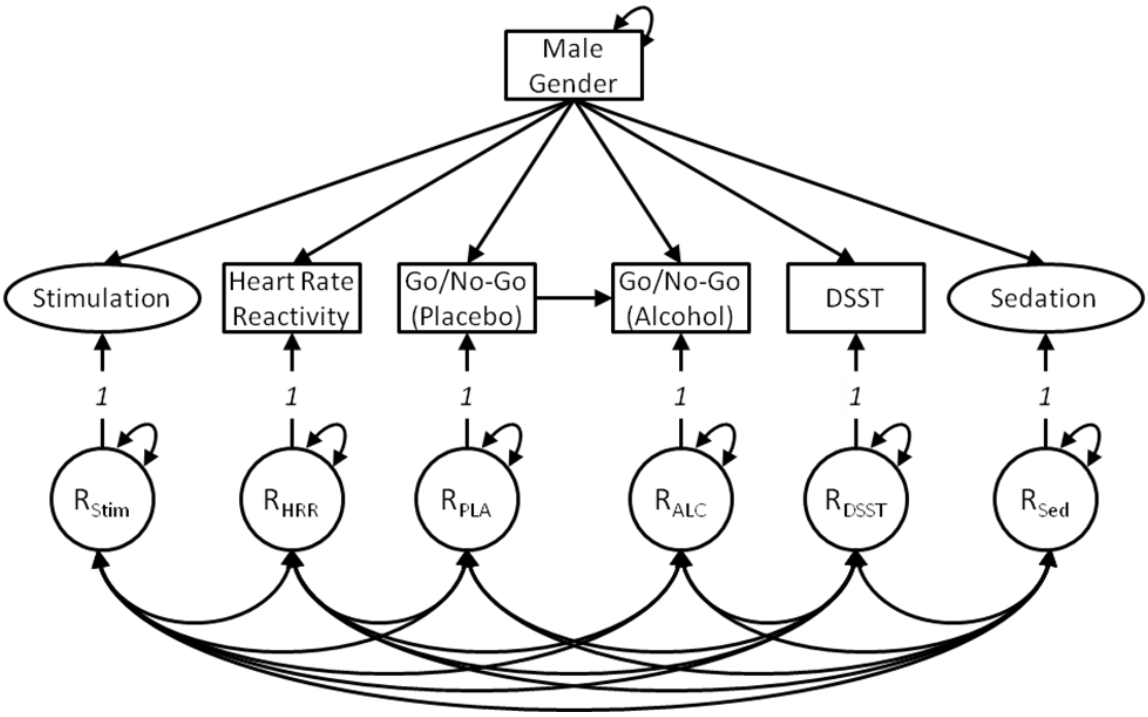

Figure 12: Illustration of model testing associations between alcohol responses and alcohol-induced inhibitory impairment.

Note. Measurement model for subjective stimulation and sedation is not shown.

The final analytic model is illustrated in Figure 12. We regressed subjective stimulation and sedation, HRR, DSST scores, and placebo- and alcohol-condition Cued Go/No-Go Task inhibitory failures on gender. In addition, alcohol-condition inhibitory failures were regressed on placebo-condition inhibitory failures. Residual variation terms for all response indices were then permitted to covary. These covariance paths tested the associations of interest: Were alcohol responses associated with alcohol-condition inhibitory failures after controlling for gender and placebo performance?

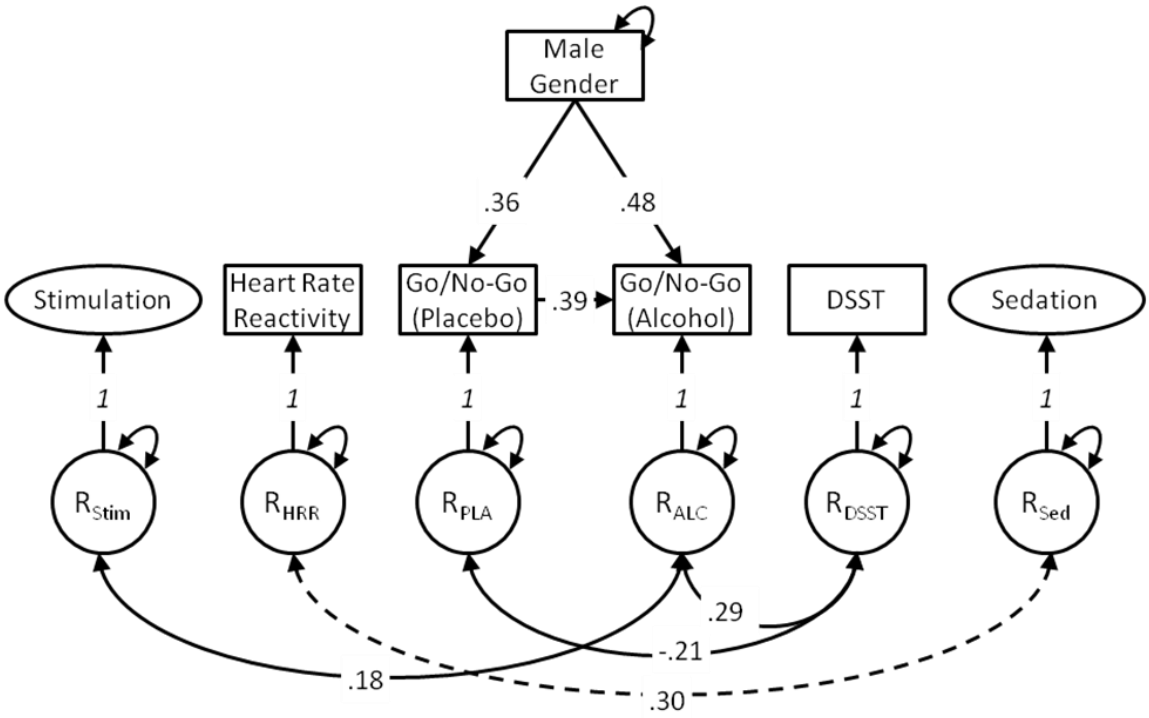

Figure 13: Standardized parameter estimates for associations between alcohol responses and alcohol-induced inhibitory impairment.

Note. Standardized coefficients and *p*-values from M*plus* STDYX output (except STDY for gender, which was binary). Significant paths and covariances shown only, $p < .05$. Measurement model for subjective stimulation and sedation is not shown. Dashed line indicates $p = .054$. Italicized values indicate (unstandardized) model constraints.

Parameter estimates are shown in Figure 13. The model fit the data well, χ^2 (46) = 55.78, p = .15, CFI = .97, RMSEA = .05. Most importantly, subjective stimulation was significantly associated with the variance in alcohol-condition inhibitory failures not explained by placebo-condition failures, r = .18, p = .03 (p = .052 for the unstandardized estimate). This path could not be constrained to equal zero without significant decrement in model fit, Satorra-Bentler scaled $\Delta\chi^2$ (1) = 4.19, p = .04. That is, participants who experienced greater stimulation also experienced greater alcohol-induced inhibitory impairment. In contrast, subjective sedation (r = .12, p = .45) and HRR (r = .22, p = .15) were not associated with inhibitory failures. Participants who were less impaired by alcohol on the DSST made fewer inhibitory failures in the placebo condition (r = -.21, p = .02) but made *more* failures in the alcohol condition, r = .29, p = .001.

In order to test whether subjective stimulation was associated with alcohol-induced inhibitory failures over and beyond the other alcohol responses, we estimated the same model but specified regression paths rather than covariances between alcohol responses and alcohol-condition inhibitory failures. In this model, which fit identically to the covariance model, subjective stimulation was no longer significantly associated with inhibitory impairment (β = .13, p = .14), whereas the DSST was, β = .25, p = .003. Taking into consideration the small sample size relative to the number of regression paths, in addition to the finding that the DSST and subjective stimulation were not significantly associated, this result suggests that any association between subjective stimulation and unique variance in inhibitory impairment was not detectable in the present analyses.

Robustness testing

Some previous examinations of associations with Cued Go/No-Go Task inhibitory impairment (Weafer & Fillmore, 2008) have used the difference between alcohol- and placebo-condition inhibitory failures as the outcome of interest rather than including both alcohol and placebo outcomes in the model. We therefore repeated the above models removing placebo-condition inhibitory failures and replacing alcohol-condition inhibitory failures with the alcohol – placebo inhibitory failures difference score. The results of this models were largely consistent with those of our initial models, χ^2 (40) = 45.31, p = .26, CFI = .99, RMSEA = .04. Subjective stimulation was significantly associated with placebo-adjusted inhibitory failures, r = .19, p = .03 (p = .059 for the unstandardized estimate). Better placebo-adjusted DSST performance was again associated with more inhibitory impairment (r = .34, p < .001), whereas sedation and HRR were not associated with inhibitory impairment. Constraining the stimulation path to zero resulted in a marginal decrement in model fit, scaled $\Delta\chi^2$ (1) = 3.60, p = .06. Replacing the covariance paths with regression paths again rendered the stimulation path (β = .15, p = .12) but not the DSST path (β = .32, p < .001) non-significant.

We finally tested a series of models to examine the robustness of the significant subjective stimulation association to alternative modeling specifications and restrictions. Coding two participants with minor irregularities in Cued Go/No-Go Task procedures as missing inhibitory failures produced results comparable in magnitude and significance. In contrast, dropping the gender covariate rendered the stimulation associations non-significant for both the model covarying placebo performance (r = .15, p = .11; p = .14 for the unstandardized estimate) and the model including a single difference score, r = .16, p = .10; p = .13 for the unstandardized estimate. In sum, participants who experienced greater subjective stimulation were significantly more likely to experience

greater alcohol-induced inhibitory impairment. Men, however, made significantly more inhibitory failures and experienced modestly and non-significantly less subjective stimulation. Without taking these associations into account, the association between stimulation and inhibitory failures appeared weaker and fell short of significance.

EVENT-LEVEL FOLLOW-UP

The primary goal of the event-level follow-up was to determine if individual differences in responses to alcohol were associated with increased risk for alcohol-related negative consequences in natural drinking environments. Each participant provided up to 21 consecutive days of online self-monitoring, from which we extracted individual drinking episodes for analyses. In all, 80 participants provided 1,668 self-monitoring surveys that met quality control criteria, of which 598 included any alcohol use. Table 6 presents person-level alcohol use and outcome summary statistics from these surveys. All participants reported drinking on at least one occasion, with participants providing an average of more than seven drinking days. Average drinking day eBACs reached the NIAAA-defined criterion for a binge drinking episode (.08 g%; NIAAA, 2004). As shown in Table 6, several behavioral risks (physical aggression, gambling, and property crime) were rare enough to preclude further analyses. Other behavioral risks ranged in prevalence from 25% (unsafe vaginal sex) to 59% (riding with a drinking driver). Examining drinking occasions only, 50% of participants reported driving after drinking, whereas 39% and 55% reported symptoms of blackout and hangover, respectively.

	Alcohol Use					
	Entire sample			**Drinking days only**		
Variable	Observed range	*M*	*SD*	Observed Range	*M*	*SD*
Included monitoring days	18 – 21	20.85	0.53	1 – 17	7.48	3.30
Drinks per day	0.10 – 5.14	1.66	1.11	1.00 – 10.80	4.50	2.20
eBAC per day (g%)	.002 – .122	.029	.023	.012 – .257	.080	.052

	Alcohol-Related Negative Consequences				
	Percent days endorsed			**Endorsed at least once**	
Variable	Observed range (%)	*M*	*SD*	%	95% CI
Illicit drug use	0.00 – 100.00	8.00	21.35	26.25	17.04, 37.29
Riding with a drinking driver	0.00 – 28.57	6.20	7.19	58.75	47.18, 69.65
Unsafe oral sex	0.00 – 47.62	4.67	9.10	31.25	21.35, 42.59
Unsafe vaginal sex	0.00 – 52.38	3.62	8.69	25.00	15.99, 35.94
Verbal aggression	0.00 – 38.10	3.21	6.55	30.00	20.26, 41.28
Physical aggression	*0.00 – 9.52*	*0.18*	*1.20*	*2.50*	*0.30, 8.74*
Gambling	*0.00 – 19.05*	*0.60*	*2.56*	*7.50*	*2.80, 15.61*
Property crime	*0.00 – 9.52*	*0.24*	*1.29*	*3.75*	*0.78, 10.57*
Drinking days only					
Driving after drinking	0.00 – 100.00	15.42	21.94	50.00	38.60, 61.40
Blackout	0.00 – 66.67	9.39	16.21	38.75	28.06, 50.30
Hangover	0.00 – 80.00	13.41	16.84	55.00	43.47, 66.15
Other consequences	0.00 – 100.00	30.35	28.16	71.25	60.05, 80.82

Table 6: Aggregate (person-level) summary statistics from daily self-monitoring follow-up.

Note. $N = 80$. Italicized rows indicate variables with base rates too low for subsequent analyses.

We first examined bivariate associations between background characteristics and outcomes in order to identify potential covariates for the GEE models. As shown in Table

7, there were relatively few background correlates of behavioral risks. Significant correlates were included in subsequent analyses.

Potential Covariate	FH+	Age at First Drink	Age at First Intoxication	Family Income	Parental Education	Risk Perceptions[a]
M		16.58	17.63	6.22	4.53	
(*SD*)	35%	(2.61)	(2.10)	(2.04)	(1.27)	--
Observed range		5 – 21	11 – 22	1 – 8	1 – 6	
Correlations with Outcome (Endorsed at Least Once)						
Illicit drug use	.09	-.14	-.17	.12	-.02	**-.23**
Riding with drinking driver	.20	.04	-.11	.10	-.03	**.23**
Unsafe oral sex	.01	-.09	-.12	-.10	.01	-.16
Unsafe vaginal sex	.12	-.17	-.19	.07	-.09	-.14
Verbal aggression	-.20	.09	.07	-.22	-.11	.08
Driving after drinking	.04	.04	-.10	.10	.02	-.05
Blackout	.02	-.02	.01	.18	.05	--
Hangover	.09	.19	.02	-.02	-.06	--
Other consequences	.01	.12	.04	-.12	-.01	--
Correlations with Outcome (Percent Days Endorsed)						
Illicit drug use	.05	-.11	-.16	**.23**	-.07	**-.22**
Riding with drinking driver	.05	-.03	-.19	.14	-.02	.11
Unsafe oral sex	.02	-.08	-.06	.01	.12	-.04
Unsafe vaginal sex	.13	-.09	-.15	.10	-.05	-.07
Verbal aggression	-.18	-.06	-.07	-.10	.00	.15
Driving after drinking	.11	.07	-.03	.01	-.06	-.12
Blackout	.00	.05	.03	.15	-.03	--
Hangover	.04	**.29**	**.25**	-.04	.07	--
Other consequences	-.13	.22	.11	**-.23**	-.01	--

Table 7: Between-persons correlations with potential follow-up covariates.

Note. $N = 80$ except $n = 79$ for FH+ and family income. FH+ = Positive family history of alcohol problems. Bolded values are significant, $p < .05$.
[a] Behavioral-risk-specific risk perceptions.

Alcohol Responses and Behavioral Risks

For each behavioral risk, we approached our analyses using the following strategy: 1) we first re-examined behavioral risk prevalence rates for included drinking days to ensure adequate base rates (see Table 8); 2) we then began with a GEE model testing the event-level association between daily (within-person) eBAC and the behavioral risk as it occurred in the natural environment; 3) we next tested for global associations between stimulant-like and sedative-like alcohol responses and drinking-day behavioral risks; and 4) finally, we tested whether alcohol responses amplified the associations between daily eBAC and behavioral risks. Two participants were excluded from all subsequent analyses because they provided only one drinking event each.

Outcome	Included Participants	Included Observations	Prevalence (%)
Illicit drug use	77	546	22.08
Riding with a drinking driver	78	569	53.85
Unsafe oral sex	77	553	18.18
Unsafe vaginal sex	76	554	13.16
Verbal aggression	78	569	14.10
Driving after drinking	78	571	46.15
Blackout	78	577	39.74
Hangover	78	576	56.41
Other consequences	77	570	74.03

Table 8: Prevalence rates for behavioral risks and other alcohol-related consequences on included drinking days.

Event-level alcohol associations

We found evidence of event-level associations between eBAC and riding with a drinking driver (b = .05, OR = 1.05, p < .001) and verbal aggression (b = .13, OR = 1.14,

$p < .001$). A given participant was more likely to engage in either of these behaviors on drinking occasions on which he or she reached higher-than-her-typical eBACs. A .01 g% increase in daily eBAC was associated with a 5% increase in the odds of riding with a drinking driver and a 14% increase in the odds of engaging in verbal aggression. Participants were no more likely to engage in illicit drug use, driving after drinking, or unsafe oral or vaginal sex[5] on drinking days on which they reached higher eBACs, *p*s > .22.

Global alcohol response associations

We next added the placebo-controlled, laboratory-derived stimulation and sedation alcohol response factor scores to the GEE models as between-persons predictors. Controlling for typical alcohol consumption (i.e., average eBAC), participants who experienced stronger stimulation in response to alcohol were marginally more likely to engage in unsafe oral sex ($b = .76$, $OR = 2.15$, $p = .07$, although note that this model was not significant overall, Wald χ^2 [6] = 6.97, $p = .32$). However, they were no more likely to engage in illicit drug use, riding with a drinking driver, verbal aggression, driving after drinking, or unsafe vaginal sex, *p*s > .53. Participants who experienced stronger sedation were marginally more likely to engage in illicit drug use, $b = .51$, $OR = 1.67$, $p = .098$, but marginally *less* likely to engage in unsafe vaginal sex, $b = -.57$, $OR = 0.57$, $p = .06$. Sedation was not globally associated with any other behavioral risk, *p*s > .15.

In sum, we found little evidence that alcohol responses were globally associated with behavioral risk-taking, at least in this sample. It should be noted, however, that low base prevalence rates, particularly for unsafe sex and verbal aggression, may have

[5] In preliminary GEE models, monogamous relationship status was not significantly associated with unsafe oral or vaginal sex (*p*s > .08), nor did monogamy moderate associations between daily eBAC and unsafe oral or vaginal sex, *p*s > .54. Monogamy was therefore not included in models predicting unsafe oral or vaginal sex.

constrained our ability to detect and estimate meaningful associations given our relatively small sample size for these analyses.

Alcohol response moderation

We next tested whether individual differences in responses to alcohol—as measured in the laboratory—moderated the event-level association between eBAC and behavioral risks by adding daily eBAC × stimulation and daily eBAC × sedation terms to the GEE models. The association between daily eBAC and illicit drug use was marginally stronger among participants who experienced greater sedation, b = .02, OR = 1.02, p = .099. See Figure 14. In contrast to study hypotheses, however, the association between daily eBAC and riding with a drinking driver was significantly amplified among participants who experienced *lower* sedation, b = -.03, OR = 0.97, p = .048. See Figure 15. Sedation did not significantly moderate within-person associations for verbal aggression, driving after drinking, and unsafe oral and vaginal sex, ps > .31.

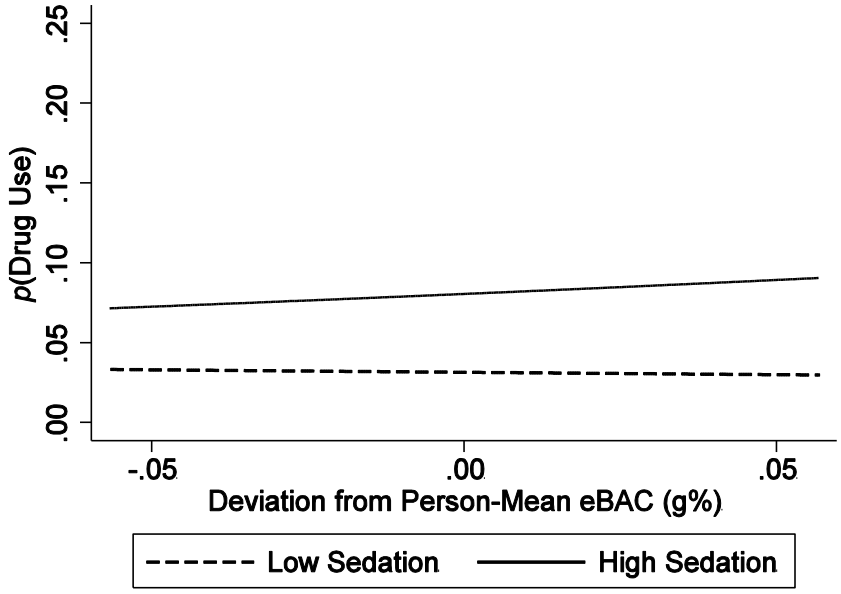

Figure 14: Association between daily eBAC and probability of illicit drug use at one standard deviation above and below the mean of the sedation factor score.

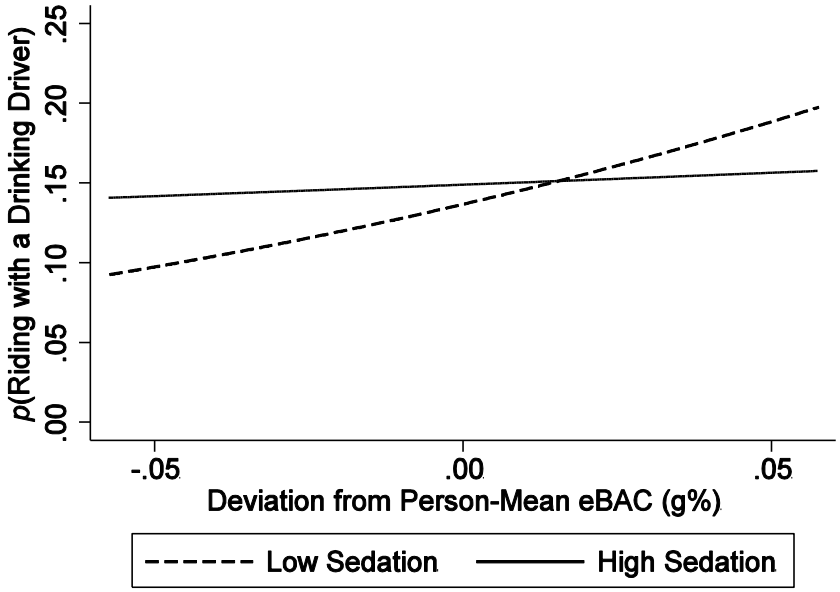

Figure 15: Association between daily eBAC and probability of riding with a drinking driver at one standard deviation above and below the mean of the sedation factor score.

Stimulation did not significantly moderate any within-person associations with behavioral risks, *p*s > .62. Because alcohol-induced inhibitory impairment was associated with subjective stimulation but not sedation, we were unable to test whether individual differences in inhibitory impairment mediated the associations between alcohol responses and behavioral risks. Parameter estimates from final models from which we trimmed sets of non-significant stimulation and sedation response terms are presented in Tables 9 – 11.[6]

[6] We additionally estimated models with a dichotomous aggregate behavioral risk dependent variable (i.e., predicting engagement in any behavioral risk). There were no significant laboratory alcohol response main effects or interactions with daily eBAC in these models.

Variable	Illicit Drug Use			Riding with a Drinking Driver		
	b	*OR*	95% CI *OR*	*b*	*OR*	95% CI *OR*
Intercept	**-2.92**	--		**-1.79**	--	
Between-Persons						
Family income	**0.47**	**1.59**	**1.12, 2.28**	--		
Male gender	0.47	1.60	0.46, 5.61	-0.13	0.88	0.42, 1.82
Risk perceptions[a]	-0.54[b]	0.58	0.33, 1.01	0.06	1.06	0.83, 1.34
Average eBAC	0.08[b]	1.08	0.99, 1.18	**0.06**	**1.06**	**1.003, 1.12**
Stimulation	-0.29	0.75	0.27, 2.08	0.16	1.18	0.70, 1.97
Sedation	0.50	1.66	0.90, 3.06	0.05	1.05	0.70, 1.58
Within-Person						
Monitoring day	0.00	1.00	0.95, 1.06	0.00	1.00	0.96, 1.03
Daily eBAC	0.01	1.01	0.98, 1.03	**0.04**	**1.04**	**1.02, 1.07**
eBAC Moderators						
Stimulation	0.00	1.00	0.96, 1.04	0.00	1.00	0.96, 1.03
Sedation	0.02[b]	1.02	1.00, 1.04	**-0.03**	**0.97**	**0.94, 0.9998**
χ^2 (*df*)	**39.56 (10)**			**44.40 (9)**		

Table 9: Parameter estimates from final GEE models of illicit drug use and riding with a drinking driver.

Note. Bolded values are significant, $p < .05$.
[a] Behavioral-risk-specific risk perceptions.
[b] $p < .10$.

Variable	Unsafe Oral Sex			Unsafe Vaginal Sex		
	b	*OR*	95% CI *OR*	*b*	*OR*	95% CI *OR*
Intercept	**-3.67**	--		**-4.26**	--	
Between-Persons						
Male gender	-0.64	0.53	0.17, 1.59	-0.63	0.53	0.16, 1.73
Average eBAC	**0.07**	**1.08**	**1.003, 1.16**	0.06	1.07	0.98, 1.17
Stimulation	0.76[a]	2.15	0.93, 4.94	-0.05	0.95	0.43, 2.09
Sedation	-0.27	0.76	0.39, 1.47	-0.57[a]	0.57	0.31, 1.03
Within-Person						
Monitoring day	-0.03	0.97	0.89, 1.05	-0.05	0.95	0.86, 1.05
Daily eBAC	-0.03	0.97	0.92, 1.02	-0.04	0.96	0.90, 1.03
χ^2 (*df*)	6.97 (6)			**14.46 (6)**		

Variable	Verbal Aggression			Driving after Drinking		
	b	*OR*	95% CI *OR*	*b*	*OR*	95% CI *OR*
Intercept	**-4.08**	--		**-1.77**	--	
Between-Persons						
Male gender	0.16	1.17	0.32, 4.27	0.17	1.19	0.54, 2.60
Average eBAC	-0.02	0.98	0.88, 1.09	0.00	1.00	0.92, 1.10
Within-Person						
Monitoring day	-0.01	0.99	0.91, 1.07	-0.02	0.98	0.95, 1.02
Daily eBAC	**0.13**	**1.14**	**1.07, 1.21**	0.00	1.00	0.97, 1.03
χ^2 (*df*)	**23.67 (4)**			0.78 (4)		

Table 10: Parameter estimates from final GEE models of unsafe oral and vaginal sex, verbal aggression, and driving after drinking.

Note. Bolded values are significant, $p < .05$.
[a] $p < .10$.

Alcohol Responses and Other Alcohol-Related Consequences

As a complement to our examination of associations between alcohol responses and behavioral risks, we repeated the above modeling steps to test whether alcohol responses were associated with participants' experience of symptoms of alcohol-induced memory impairment (i.e., alcohol blackout) and hangover, in addition to other alcohol-related consequences.

Event-level alcohol associations

Blackout (b = .16, OR = 1.18, $p < .001$), hangover (b = .17, OR = 1.19, $p < .001$), and other consequences (b = .11, OR = 1.11, $p < .001$) were all associated with daily eBAC at the event level. That is, reaching higher eBACs increased risk for all three categories of alcohol-related consequences, with within-person increases of .01 g% in eBAC associated with increases of 18%, 19%, and 11% in the odds of blackout, hangover, and other consequences, respectively.

Variable	**Blackout**			**Other Consequences**		
	b	*OR*	95% CI *OR*	*b*	*OR*	95% CI *OR*
Intercept	**-2.97**	--		**-0.74**	--	
Between-Persons						
Family income	--			-0.14[a]	0.87	0.75, 1.01
Male gender	-0.28	0.75	0.31, 1.84	0.16	1.17	0.63, 2.19
Average eBAC	**0.18**	**1.20**	**1.13, 1.27**	0.05	1.05	0.98, 1.13
Stimulation	-0.19	0.82	0.49, 1.40	0.00	1.00	0.70, 1.44
Sedation	**0.53**	**1.69**	**1.01, 2.82**	**0.52**	**1.68**	**1.16, 2.42**
Within-Person						
Monitoring day	0.02	1.02	0.97, 1.06	-0.02	0.98	0.95, 1.01
Daily eBAC	**0.17**	**1.18**	**1.11, 1.25**	**0.11**	**1.12**	**1.08, 1.15**
χ^2 (*df*)	**79.89 (6)**			**61.00 (7)**		

Variable	**Hangover**		
	b	*OR*	95% CI *OR*
Intercept	**-2.19**	--	
Between-Persons			
Male gender	-0.51	0.60	0.31, 1.17
Age at first drink	**0.30**	**1.34**	**1.09, 1.66**
Age at first intox.	0.02	1.02	0.80, 1.31
Average eBAC	0.04	1.04	0.99, 1.09
Within-Person			
Monitoring day	0.00	1.00	0.94, 1.05
Daily eBAC	**0.17**	**1.19**	**1.13, 1.25**
χ^2 (*df*)	**43.27 (6)**		

Table 11: Parameter estimates from final GEE models of blackout, hangover, and other consequences.

Note. Bolded values are significant, $p < .05$. [a] $p < .10$.

Global alcohol response associations

Adding the laboratory-derived stimulation and sedation factor scores to the GEE models as between-persons predictors, we found evidence of global associations between sedation and blackout (b = .53, OR = 1.69, p = .04) and other consequences (b = .52, OR = 1.68, p = .01) but not hangover, b = .22, OR = 1.24, p = .23. That is, participants who experienced greater subjective sedation were more likely to experience blackout and other consequences, controlling for typical alcohol consumption. In contrast, subjective stimulation was not associated with any alcohol-related consequences, ps > .28. In addition, subjective stimulation and sedation did not significantly moderate daily eBAC associations with blackout, hangover, or other consequences, ps > .39.

Alcohol Responses at the Event Level

An alternative hypothesis regarding links between alcohol responses and outcomes is that episode-to-episode variation in responses over and beyond eBAC might be associated with negative alcohol-related consequences. In order to test this possibility, we added event-level, person-mean-centered BBAES stimulation and sedation scores to the models described above, including background characteristics, gender, monitoring day, average and daily eBAC, and any significant or marginally significant alcohol response main effects or interactions. One participant with two drinking-day observations was excluded from these analyses for missing data on the BBAES. In data from the largest (i.e., blackout) analytic sample, on average, participants reported more drinking-day stimulation (person-mean = 4.19, SD = 2.21, range = 0.00 – 8.00) than sedation (person-mean = 1.29, SD = 1.14, range = 0.00 – 4.90), which is consistent with their laboratory results.

Within-person increases in subjective stimulation were associated with elevated event-level risk for riding with a drinking driver (b = .14, OR = 1.16, p = .03) and unsafe

oral sex (b = .31, OR = 1.37, p = .03) over and beyond daily eBAC. Within-person changes in subjective stimulation were not associated with illicit drug use, verbal aggression, or unsafe vaginal sex (ps > .28), however, and no behavioral risks were associated with BBAES sedation at the event level, ps > .27.

Regarding alcohol-related consequences, within-person increases in subjective stimulation were significantly associated with increases in risk for blackout (b = .40, OR = 1.49, p < .001), hangover (b = .33, OR = 1.39, p < .001), and other consequences (b = .21, OR = 1.23, p = .001) over and beyond daily eBAC. Similarly, within-person increases in subjective sedation were significantly associated with increases in risk for hangover (b = .50, OR = 1.64, p < .001) and marginally associated with increases in risk for blackout (b = .18, OR = 1.20, p = .07) and other consequences, b = .15, OR = 1.16, p = .07. In sum, we found evidence that within-person, episode-to-episode variation in responses to the effects of alcohol may confer risk beyond (objective) alcohol intoxication, particularly for subjective stimulant-like responses and most consistently for blackout, hangover, and other alcohol-related consequences.

Driving after drinking: Event-level eBAC × alcohol response moderation

Previous research has suggested that within-person variation in the experience of subjective intoxication may be protective against driving after drinking via a unique mechanism (Marczinski & Fillmore, 2009; Quinn & Fromme, 2012). Specifically, among (objectively) intoxicated individuals, greater perceived intoxicated has been associated with reduced driving after drinking. We attempted to replicate this finding using the event-level BBAES measures of subjective stimulation and sedation. We began with a main effects model with of daily stimulation and sedation. Daily sedation was not associated with driving after drinking (b = -.05, OR = 0.95, p = .64), however, and in

contrast with expectations, greater stimulation was associated with a *greater* likelihood of driving after drinking, $b = .17$, $OR = 1.19$, $p = .01$. That is, holding daily eBAC constant, risk was greater during episodes in which participants experienced greater-than-their usual stimulation.

This main effect was, as predicted, moderated by daily eBAC such that the association decreased in magnitude as participants became more (objectively) intoxicated, $b = -.02$, $OR = 0.98$, $p = .052$. The daily sedation × eBAC interaction was not significant, $b = .01$, $OR = 1.01$, $p = .60$. As illustrated in Figure 16, the risk for driving after drinking associated with subjective stimulation was lower when participants had consumed more alcohol. This pattern suggests that, partially consistent with previous research, evaluations of subjective stimulation may not confer risk for driving after drinking when combined with greater levels of objective alcohol intoxication.

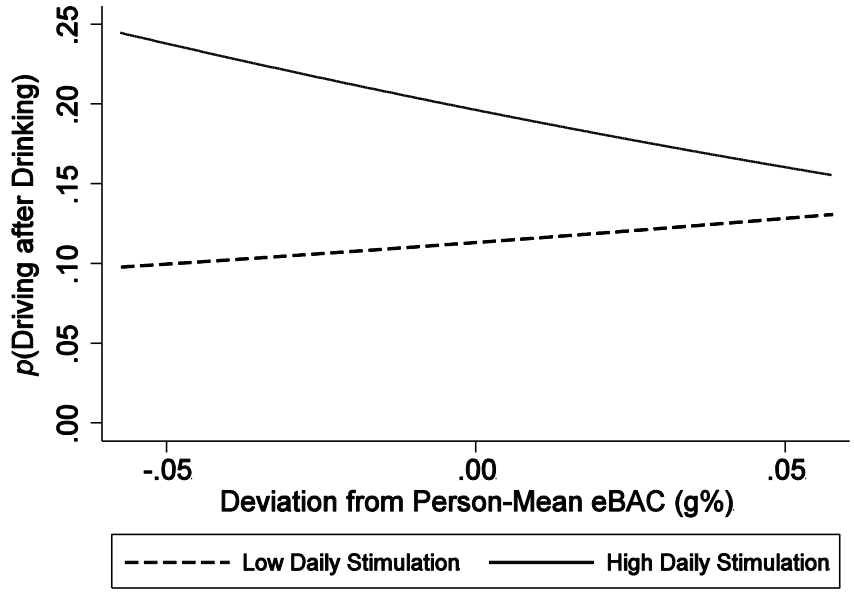

Figure 16: Association between daily eBAC and probability of driving after drinking at one standard deviation above and below the mean of daily stimulation.

Alcohol Responses and Disinhibited Personality

As shown in Table 12, the sedation factor was significantly but modestly associated with lower perseverance but was not associated with any other disinhibited personality facets. Stimulation was also not associated with any personality facets.

Personality Scale	Subjective Stimulation	Subjective Sedation
Brief Self-Control Scale (BSCS)	-.09	-.19
Monetary Choice Questionnaire[a] (MCQ)	.10	-.14
UPPS Premeditation (UPPS Pre)	.10	.14
UPPS Perseverance (UPPS Pers)	-.13	**-.16**
UPPS Urgency (UPPS Urg)	.06	.28[b]
UPPS Sensation Seeking (UPPS Sen)	-.02	-.11
Positive Urgency Measure	-.09	.02

Table 12: Correlations between disinhibited personality and alcohol responses.

Note. Correlations estimated in M*plus* using final measurement model described above (STDYX output). $N = 80$. Model fit was adequate, χ^2 (58) = 84.34, $p = .01$, CFI = .94, RMSEA = .07. Bolded values were significant, $p < .05$.
[a] Correlations are with natural log transformed MCQ scores.
[b] $p = .055$.

Moreover, perhaps as a consequence of range restriction (given selection criteria), there were few significant associations between disinhibited personality and event-level outcomes. In fact, the 2 associations with sensation seeking and 1 significant association with positive urgency are fewer than would be expected by chance alone (3/126 = 2.39%). Correlations between measures of disinhibited personality and outcomes (across all 1,668 quality-controlled observations) are presented in Table 13. This pattern of associations suggested that underlying associations with disinhibited personality dispositions were unlikely to explain any global associations with alcohol responses.

Personality Scale	BSCS	MCQ[a]	UPPS Pre	UPPS Pers	UPPS Urg	UPPS Sen	Positive Urgency
M (SD)	3.34 (0.56)	-4.84 (1.41)	2.91 (0.39)	2.95 (0.36)	2.09 (0.47)	2.92 (0.46)	1.74 (0.41)
Observed range	2.23 – 4.92	-8.75 – -2.14	1.73 – 3.55	2.00 – 3.80	1.08 – 3.50	2.00 – 3.92	1.00 – 2.64
Correlations with Outcome (Endorsed at Least Once)							
Illicit drug use	-.04	-.04	-.13	-.07	-.06	.11	-.05
Riding with drinking driver	.10	.18	-.14	.10	-.01	.04	-.12
Unsafe oral sex	.02	-.02	.07	.05	-.11	.06	-.19
Unsafe vaginal sex	-.09	-.06	.01	-.05	-.02	.12	.02
Verbal aggression	-.08	.06	.00	.00	.05	-.08	-.08
Driving after drinking	-.07	.05	-.09	-.10	.09	.01	-.03
Blackout	.08	-.10	.15	.15	-.02	**-.33**	-.08
Hangover	.00	-.03	.10	-.10	.09	-.16	.03
Other consequences	-.17	-.03	.10	-.15	.02	-.13	-.06
Correlations with Outcome (Percent Days Endorsed)							
Illicit drug use	-.01	.01	.08	-.03	.03	.20	.02
Riding with drinking driver	-.04	.10	-.08	.06	.02	.01	-.06
Unsafe oral sex	.00	-.06	.16	.03	-.15	-.12	**-.25**
Unsafe vaginal sex	-.13	-.07	-.04	-.10	-.06	.01	-.04
Verbal aggression	.01	.06	.05	.14	-.02	-.05	-.17
Driving after drinking	-.04	.09	-.04	-.06	-.08	-.01	-.02
Blackout	.11	-.02	.13	.21	-.08	-.20	-.09
Hangover	-.08	-.03	.04	-.12	.18	**-.24**	-.14
Other consequences	-.15	.13	.14	-.03	.12	-.07	.00

Table 13: Summary statistics and between-persons correlations for facets of disinhibited personality.

Note. *N*s = 80 except *n* = 79 for MCQ. Bolded values were significant, $p < .05$.
[a] Correlations are with natural-log transformed MCQ scores.

Chapter 6: Discussion

This study attempted to integrate individual differences in responses to the intoxicating effects of alcohol into research on drinking-episode-level negative consequences. We collected data from a relatively diverse sample of moderate-to-heavy drinkers aged 21 – 25 in two consecutive research protocols: an alcohol challenge in a simulated bar laboratory (N = 82) and an event-level daily self-monitoring protocol (n = 78). The combination of these two methods enabled this study to employ a rigorous, placebo-controlled assessment of alcohol responses while also measuring behavioral risks and other alcohol-related consequences as they actually occurred during specific drinking episodes in natural drinking environments. As such, it represented a refinement of our previous efforts at detecting the contributions of individual differences in alcohol responses to intoxicated behavioral risk-taking.

Alcohol Responses and Inhibitory Impairment

Replicating previous research, alcohol intoxication impaired response inhibition as assessed by the Cued Go/No-Go Task (Fillmore & Weafer, 2004; Marczinski & Fillmore, 2003; Weafer & Fillmore, 2008). Moreover, consistent with previous research (Marczinski & Fillmore, 2003), this impairment appeared stronger when cues falsely signaled response targets. This pattern has been interpreted as providing evidence that alcohol most strongly impairs the inhibition of prepotent (i.e., cued) responses. However, further examination revealed that this increase in impairment was primarily driven by a small number of participants who reported a large number of inhibitory failures; transforming inhibitory failure scores rendered the alcohol effect non-significantly larger in the Invalid Go cue condition relative to the Valid No-Go cue condition. Taken with our finding that alcohol also slowed response execution equivalently across cue conditions—

which is consistent with some (Fillmore & Weafer, 2004) but not all (Marczinski & Fillmore, 2003) previous findings—this pattern indicates a broader cognitive impairment beyond response inhibition. Nevertheless, alcohol's impairing effect on response inhibition, a component of executive cognitive control, is consistent with the Alcohol Myopia model, in which alcohol intoxication is predicted to impair higher-order, controlled processing (Moss & Albery, 2009).

A principal goal of this study was to determine whether individual differences in responses to alcohol's subjective effects corresponded with individual differences in alcohol-induced inhibitory impairment. We therefore required a strong assessment of alcohol responses, which we obtained by following Ray and colleagues (2009) in evaluating alcohol responses with multiple measures of stimulant-like and sedative-like effects. As expected, alcohol (relative to placebo) produced stimulant-like subjective effects on the ascending limb of the breath alcohol concentration (BrAC) curve and at its peak and produced sedative-like subjective effects through the descending limb. In addition, alcohol increased two objective indices of response to alcohol: Heart Rate Reactivity (HRR) and psychomotor impairment, as evaluated with the Digit Symbol Substitution Task (DSST).

Moreover, placebo-adjusted subjective alcohol responses at the peak of the BrAC curve could be described well using a two-factor model, which is consistent with previous literature. The two factors corresponded to subjective stimulation (with highest loadings for the *POMS Energetic* and *SEAS High Arousal Positive* scales) and sedation (with highest loadings for the *SHAS* and the *SEAS Low Arousal Negative* scale) and were moderately but not significantly correlated. Interestingly, not all measures loaded cleanly onto the two factors. The *POMS Energetic* scale loaded inversely onto the sedation factor, and the *POMS Intoxicated* and *BAES Sedation* scales loaded inversely onto the

stimulation factor. If this finding is replicated in other samples, future research may benefit from employing either a similar measurement model approach or the use of specific scales that are highly correlated with one but not the other factor (e.g., the *SEAS* scales or the *SHAS*). Neither HRR nor DSST performance was significantly associated with subjective responses, suggesting that, at least in this population, individual differences in these physiological/psychomotor responses may be empirically distinct from subjective responses to alcohol.

Most importantly, participants who reported greater placebo-adjusted subjective stimulation also experienced significantly greater alcohol-induced inhibitory impairment on the Cued Go/No-Go Task Invalid Go cue condition. In contrast, subjective sedation was not associated with inhibitory impairment. That is, we found support for our hypothesis that greater alcohol responders would experience more inhibitory impairment, although this support was limited to stimulation. It is important to note that this significant association was only apparent when we controlled for gender, which was also significantly associated with inhibitory impairment: Controlling for placebo performance, men made more alcohol-condition inhibitory failures, and they additionally made more placebo-condition inhibitory failures. Fillmore and Weafer (2004), in a much smaller sample, similarly found stronger alcohol-induced impairment of response inhibition among men, although they found no difference in placebo performance.

Beyond the subjective alcohol responses, the association between greater HRR and inhibitory impairment did not reach significance. This finding differed from a previous study, in which greater heart rate responders made more alcohol-induced commission errors (i.e., inhibitory failures; $\beta = .19$) controlling for baseline performance (Assaad et al., 2006b). We see two possible explanations for this difference. First, Assaad and colleagues (2006b) measured resting heart rate continuously (within a five-minute

period) rather than at discrete points, which may have reduced measurement error. Second, Assaad and colleagues (2006) employed a no-alcohol control rather than placebo, meaning that expectancy effects may have inflated differences between alcohol and control-condition performance in that study relative to ours.

Individuals who experienced less alcohol-induced psychomotor impairment, as measured by the DSST change score, also made fewer placebo-condition response inhibition failures but *more* alcohol-condition failures. Although the current analyses were unable to determine why alcohol-induced psychomotor and response inhibition impairment would be inversely correlated, we speculate that response strategies may have produced differential success in the DSST relative to the Cued Go/No-Go Task. For example, responding as rapidly as possible on the DSST would help increase the number of correct responses, whereas the same strategy might also increase the likelihood of inhibition failures on the Cued Go/No-Go Task. Indeed, Valid Go reaction times and Invalid Go proportions of inhibitory failures on the Cued Go/No-Go Task were significantly but inversely correlated in both the placebo and alcohol conditions, which is consistent with this possibility.

Taken together, our results suggest an association between individual differences in alcohol responses and alcohol-induced inhibitory impairment that may be specific to responses to stimulant-like effects. Given evidence that physiological arousal can mimic alcohol's myopic effects (Ward et al., 2008), we hypothesized that a subjective-stimulation-specific effect would stem from individual differences in sensitivity to the physiologically arousing effects of alcohol. However, our results did not generalize to HRR, which argues against this possibility. If future studies replicate this differentiation, it may be rather that differential inhibitory impairment reflects differential hedonic or euphoric response. Many measures of subjective stimulation confound stimulant-like

effects with positively valenced effects (Morean & Corbin, 2010), and a rewarding response may impel some intoxicated individuals to "Go" with reduced attention to inhibitory cues. This pharmacological effect would be akin to outcome expectancies of "liquid courage" (Fromme, Stroot, & Kaplan, 1993). Although we are aware of limited evidence for this possibility, we note that Cyders and colleagues (2010) found that a positive mood induction increased alcohol consumption, particularly among those higher in trait-level positive urgency. A test of whether positive mood induction or receipt of other rewarding stimuli can impair response inhibition would be a possible avenue for future research.

ALCOHOL RESPONSES AND ALCOHOL-RELATED NEGATIVE CONSEQUENCES

The second goal of this study was to determine whether laboratory-assessed alcohol responses were associated with elevated risk for negative outcomes in real-world drinking events. In daily self-monitoring, participants provided evidence that they engaged in heavy drinking and a variety of other behavioral risk-taking. Average drinking-day estimated blood alcohol concentrations (eBACs) were at the NIAAA criterion for binge drinking (.08 g%), and most participants experienced at least one intoxicated behavioral risk or other negative consequence, most commonly including driving after drinking, riding with a driver who had been drinking, blackout, and hangover. Other behavioral risks, such as physical aggression, gambling, and property crime, were rarer.

At the event-level, eBACs were associated with increases in the probability of verbal aggression and riding with a drinking driver but no other behavioral risks. Previous event-level research has found similar results for aggression (e.g., Neal & Fromme, 2007; Quinn et al., 2013; Wells et al., 2008). Other research has consistently

demonstrated that heavier drinkers are more likely, on average, to ride with a drinking driver (Calafat et al., 2009; Cartwright & Asbridge, 2011; Kim & Kim, 2012; Pedersen & McCarthy, 2008; Poulin, Boudreau, & Asbridge, 2007), but this study is the first, to our knowledge, to find evidence of an event-level association.

In all, we found little evidence that alcohol responses were associated with behavioral risks. No global associations between alcohol responses and behavioral risks reached conventional significance, although greater stimulation was marginally associated with unsafe oral sex, whereas greater sedation was marginally associated with illicit drug use and (less) unsafe vaginal sex. Similarly, neither alcohol response was significantly associated with strengthened associations between eBAC and the behavioral risks. In fact, the only significant moderation effect, in which the association between eBAC and riding with a drinking driver was greater among those *lower* in sedation, was opposite to the expected direction. Given the association between measures of subjective sedation and perceived impairment (see Table 4), we speculate that individuals who typically experience less alcohol-induced sedation might view internal cues associated with increasing BAC as indicators that they are unable to drive, whereas greater sedation responders might not. As a consequence, lower sedation responders may actually replace one behavioral risk with another by choosing to ride with a drinking driver rather than drive themselves when they reach higher BACs. Because this pattern of results was not hypothesized, however, we are reluctant to draw firm inferences unless it can be replicated in other event-level research.

These results failed to support the hypothesis that alcohol responses amplify general risk for intoxicated behavioral outcomes. Relative to previous studies, the current study incorporated several levels of increased experimental control, including adjustment for placebo response, stronger measurement, and standardization of the laboratory

drinking environment. It is possible that our preliminary findings may have been inflated by confounds that were reduced here. In sum, whereas this study found that subjective stimulant-like responses were associated with inhibitory impairment, the small-to-moderate size of this association may have limited its ability to relate to actual behavioral outcomes, which were relatively rare in the follow-up period and were subject to other contextual influences in real-world drinking environments.

More promisingly, however, we found that individuals who experienced greater subjective sedation were more likely to experience blackout and other (broadly defined) negative consequences over and beyond typical levels of drinking. This pattern of aversive outcomes may help explain why sedative-like responses predicted lower levels of binge drinking in a longitudinal study (King et al., 2011). A previous event-level study by Wetherill and Fromme (2009) found that self-reported sedation and stimulation were associated with blackout and hangover during 21st birthday celebrations. In the present investigation, we were able to extend this finding by distinguishing individual differences from other variation in responses, and results suggested that greater sedation responders may be at elevated risk for blackout and other consequences, if not for hangover. Alcohol blackout is a complex, problematic phenomenon, and identifying why some individuals are more predisposed to experience blackout relative to others is a major focus of research. One study estimated that around half of college drinkers experience blackout and that many engage in dangerous behavior during periods of alcohol-induced amnesia (White, Jamieson-Drake, & Swartzwelder, 2002). The experience of blackout is driven in part by genetic influences, even after controlling variation shared with frequency of alcohol intoxication (Nelson et al., 2004). The current results suggest that individual differences in the subjective sedative effects of alcohol may be valuable targets for future behavioral genetic research on alcohol-induced blackout.

Event-Level Alcohol Responses and Negative Consequences

Previous research has found that within-person, episode-to-episode variation in subjective intoxication is associated with at least some behavioral risks over and beyond eBACs (e.g., Quinn et al., 2013). Although it was not a primary goal of the present study, we replicated some of these associations using a brief measure of alcohol's stimulant-like and sedative-like effects (Rueger & King, 2013; Rueger et al., 2009). Specifically, greater stimulation at the event level was associated with riding with a drinking driver, unsafe oral sex, blackout, hangover, and other consequences, and greater event-level sedation was associated with hangover and was marginally associated with blackout and other consequences. Moreover, participants were also more likely to drive after drinking during episodes in which they experienced greater stimulation, although this association was weaker as participants became more objectively intoxicated (i.e., at higher eBACs).

A challenge for the interpretation of these associations is that episode-level variation in drinking contexts may alter alcohol responses (Ray, Miranda et al., 2010). Thus, we cannot determine whether within-person variability confers risk or whether risk *and* response increase in certain contexts. For example, if social environments increase stimulant-like effects (Ray, Miranda et al., 2010) and if driving after drinking requires participants to be out of the home while drinking, then the association between stimulation and driving after drinking may be explained by social context. That this association was moderated by eBAC, however, suggests that subjective stimulation may be interpreted differently at higher eBACs, with eBAC and stimulation interacting to buffer against decisions to drive while impaired (Quinn & Fromme, 2012). Future research in the laboratory (in which drinking contexts could be randomly assigned) or using experience sampling (to establish temporal precedence) would help test these possibilities.

LIMITATIONS AND FUTURE DIRECTIONS

Our conclusions were constrained by several limitations of our methodology. Most importantly, our use of non-experimental methods prevented us from being able to draw causal inferences regarding statistical associations between alcohol responses, inhibitory impairment, and outcomes. Additionally, our sample, though reasonably diverse and adequate for a laboratory alcohol challenge, was relatively small for a study of individual differences (fewer than 598 observations among 78 participants in follow-up), and we included only moderate-to-heavy drinkers in order to ensure sufficient variability in behavioral risks. This selection criterion may have resulted in range restriction on alcohol responses, meaning that some associations of interest could have been detected in a larger, more representative sample.

Second, given our interest in assessing intra-individual change, the within-person, counterbalanced design was more optimal than would have been a between-persons design. Repeated laboratory sessions did, however, introduce other limitations, including attrition, differing numbers of participants and, in select cases, differing members in laboratory session groups. We note, though, that all alcohol response assessments were conducted individually and that we found little evidence of within-group dependency.

Third, our approach to the measurement of subjective alcohol responses had the advantage of reducing measurement error by including multiple measures of subjective sedation and stimulation, in addition to adjusting for baseline individual differences and placebo responses. These methodological choices had costs as well, however. We selected measures that captured subjective stimulant-like and sedative-like effects, but, as Morean and colleagues have argued, measures of stimulation and sedation confound high and low arousal with positive and negative valence, respectively (Morean et al. 2013, May 6; Morean and Corbin, 2010). We therefore cannot distinguish whether our results

reflect differences in subjective arousal or hedonic effects. The recently validated Subjective Effects of Alcohol Scale may help future research make this important distinction (Morean et al., 2013, May 6). Moreover, our use of difference scores to adjust for baseline and placebo scores may have resulted in reduced measure reliability. Although this loss of reliability would be attenuated by our latent measurement approach, examination of how alcohol response measurement models vary when using difference scores and raw alcohol-condition scores would improve understandings of the latent structure of alcohol's subjective effects.

Fourth, our alcohol challenge approach focused on the peak assessment (King et al., 2011), and we were only able to assess response inhibition with a single task. Future research should test whether our results generalize across limbs of the BrAC curve and across other measures. Finally, although we failed to detect alcohol response associations with many behavioral outcomes, such failure does not necessarily demonstrate a lack of true association. Other alternative approaches to the assessment of behavioral risk-taking, such as reported intentions or behavioral analogues (e.g. Taylor Aggression Paradigm, Balloon Analogue Risk Task; Lejuez et al., 2002; Taylor, 1967) could help ensure response conflict, reduce contextual influences, maximize variability, and therefore increase the probability of detecting associations—albeit at the expense of external validity and the assessment of diverse behavioral risks.

CONCLUSIONS

In spite of these limitations, our findings have several implications for future research. First, high-quality measurement of subjective responses to alcohol is crucial, and our findings add to previous studies in indicating that subjective stimulation and sedation can and should be differentiated. Future research could continue to use a

measurement model approach or select measures that cleanly capture subjective stimulation and sedation. Second, this study is the first, to our knowledge, to demonstrate an association between subjective stimulation and alcohol-induced inhibitory impairment. Although this association was not strong, was only apparent when controlling for gender, and did not extend to the prediction of consequences in real-world drinking, it suggested that individual differences in sensitivity to the subjective effects of alcohol may correspond with inhibitory sensitivity as well. Future research is needed to characterize this relation more fully. Third, sedative-like subjective responses appear to be associated with risk for blackout if not for behavioral outcomes. Further research on sedative-like responses may provide insight into why some individuals are at greater risk for blackout and its consequences.

Finally, our results cumulatively highlight the complexity of alcohol intoxication. There are individual differences in sensitivity to alcohol's effects, and its diverse physiological, cognitive, and behavioral consequences vary both across and within individuals. Understanding this complexity will be a key to developing effective interventions to reduce the public health cost of emerging adult alcohol use.

Appendix: Self-Report Measures

Baseline Self-Report Measures

Demographics

1. What is your biological sex?
   ○ Female ○ Male

2. What is your birth date?
   Month __ Day __ Year ____

3. What is your race/ethnicity (mark all that apply):
   ○ American Indian/Alaskan Native
   ○ Asian
   ○ Black or African American
   ○ Hispanic or Latino(a)
   ○ Native Hawaiian or Pacific Islander
   ○ White or Caucasian

4. Prior to high school graduation, what was your family's estimated annual income?
   ○ under $19,999
   ○ $20,000 - 29,999
   ○ $30,000 - $39,999
   ○ $40,000 - $49,999
   ○ $50,000 - $59,999
   ○ $60,000 - $69,999
   ○ $70,000 - $99,999
   ○ $100,000 or over

5. What is the highest grade in school or year in college that your mother completed?
   ○ Did not complete high school or obtain GED
   ○ High school diploma/GED
   ○ Some college
   ○ Junior college/trade school degree
   ○ College degree
   ○ Post-graduate degree
   ○ Not sure/don't know

6. What is your mother's occupation?
   ○ Currently unemployed
   ○ Deceased
   ○ Retired
   ○ Not sure/don't know
   ○ Currently employed/describe occupation: _____

7. What is the highest grade in school or year in college that your father completed?
   ○ Did not complete high school or obtain GED
   ○ High school diploma/GED
   ○ Some college
   ○ Junior college/trade school degree
   ○ College degree
   ○ Post-graduate degree
   ○ Not sure/don't know

8. What is your father's occupation?
   ○ Currently unemployed
   ○ Deceased
   ○ Retired
   ○ Not sure/don't know
   ○ Currently employed/describe occupation: _____

9. How often do you drive an automobile (car, truck, motorcycle)?

- ○ Never/almost never
- ○ Monthly
- ○ Weekly
- ○ Daily or almost every day

Family History of Alcohol Problems

Family Tree Questionnaire

INSTRUCTIONS: For each relative listed below, we want to know your impressions of their drinking behavior. Please categorize each relative into the category you think best describes their drinking behavior. Only include blood relatives; that is, relatives by birth. Do not include relatives who are adopted, half-siblings, in-laws, or step-relatives. If you have fewer than 4 brothers, sisters, aunts, or uncles, mark "N/A" on any remaining lines.

CODE EACH RELATIVE USING ONE OF THE FOLLOWING 5 CATEGORIES:

1. NEVER DRANK: A person who has never consumed alcoholic beverages (i.e., a lifelong abstainer or teetotaler).
2. SOCIAL DRINKER: A person who you think drinks moderately and is not known to have a drinking problem.
3. POSSIBLE PROBLEM DRINKER: A person who you or others believe may have a past or current drinking problem, but you are not actually certain whether they ever had a drinking problem.
4. DEFINITE PROBLEM DRINKER: Only include persons who you think either have received treatment for a drinking problem (i.e., Alcoholics Anonymous), or who are known to have experienced several negative consequences of their drinking.
5. DON'T KNOW/DON'T REMEMBER: Please indicate only if you do not know the relative, or have no memory of their drinking behavior.

	Family Member	N/A	1	2	3	4	5
01.	Maternal Grandmother (Mother's Mother)	■	①	②	③	④	⑤
02.	Maternal Grandfather (Mother's Father)	■	①	②	③	④	⑤
03.	Paternal Grandmother (Father's Mother)	■	①	②	③	④	⑤
04.	Paternal Grandfather (Father's Father)	■	①	②	③	④	⑤
05.	Mother	■	①	②	③	④	⑤
06.	Father	■	①	②	③	④	⑤
07.	Maternal Aunt (Mother's Sister)	○	①	②	③	④	⑤
08.	Maternal Aunt (Mother's Sister)	○	①	②	③	④	⑤
09.	Maternal Aunt (Mother's Sister)	○	①	②	③	④	⑤

10.	Maternal Aunt (Mother's Sister)	○	①	②	③	④	⑤
11.	Maternal Uncle (Mother's Brother)	○	①	②	③	④	⑤
12.	Maternal Uncle (Mother's Brother)	○	①	②	③	④	⑤
13.	Maternal Uncle (Mother's Brother)	○	①	②	③	④	⑤
14.	Maternal Uncle (Mother's Brother)	○	①	②	③	④	⑤
15.	Paternal Aunt (Father's Sister)	○	①	②	③	④	⑤
16.	Paternal Aunt (Father's Sister)	○	①	②	③	④	⑤
17.	Paternal Aunt (Father's Sister)	○	①	②	③	④	⑤
18.	Paternal Aunt (Father's Sister)	○	①	②	③	④	⑤
19.	Paternal Uncle (Father's Brother)	○	①	②	③	④	⑤
20.	Paternal Uncle (Father's Brother)	○	①	②	③	④	⑤
21.	Paternal Uncle (Father's Brother)	○	①	②	③	④	⑤
22.	Paternal Uncle (Father's Brother)	○	①	②	③	④	⑤
23.	Brother	○	①	②	③	④	⑤
24.	Brother	○	①	②	③	④	⑤
25.	Brother	○	①	②	③	④	⑤
26.	Brother	○	①	②	③	④	⑤
27.	Sister	○	①	②	③	④	⑤
28.	Sister	○	①	②	③	④	⑤
29.	Sister	○	①	②	③	④	⑤
30.	Sister	○	①	②	③	④	⑤

Alcohol Response during Early Drinking Experiences

Self-Rating of the Effects of Alcohol

For each of the time periods listed, please estimate the number of standard drinks required for you to experience each of the following four conditions. One Standard Drink is equal to 12 oz of beer, 5 oz of wine, or 1.5 oz of liquor in a shot or mixed drink.

During the…	***…first five times you had ever taken a drink***	***…most recent 3 consecutive months on which you drank at least once***	***…period that you drank the heaviest***
1. Begin to feel any different			
2. Feel a bit dizzy or begin to slur your speech			
3. Begin stumbling or walking in an uncoordinated manner			
4. Pass out, or fall asleep when you did not want to			

Personality Dispositions toward Behavioral Risks

UPPS Impulsivity Scale

Using the scale provided, please indicate how much each of the following statement reflects how you **TYPICALLY** are.		Disagree Strongly			Agree Strongly
01.	I have a reserved and cautious attitude toward life.	1	2	3	4
02.	My thinking is usually careful and purposeful.	1	2	3	4
03.	I am not one of those people who blurt out things without thinking.	1	2	3	4
04.	I like to stop and think things over before I do them.	1	2	3	4
05.	I don't like to start a project until I know exactly how to proceed.	1	2	3	4
06.	I tend to value and follow a rational, ``sensible'' approach to things.	1	2	3	4
07.	I usually make up my mind through careful reasoning.	1	2	3	4
08.	I am a cautious person.	1	2	3	4
09.	Before I get into a new situation I like to find out what to expect from it.	1	2	3	4
10.	I usually think carefully before doing anything.	1	2	3	4
11.	Before making up my mind, I consider all the advantages and disadvantages.	1	2	3	4
12.	I have trouble controlling my impulses.	1	2	3	4
13.	I have trouble resisting my cravings (for food, cigarettes, etc.).	1	2	3	4
14.	I often get involved in things I later wish I could get out of.	1	2	3	4
15.	When I feel bad, I will often do things I later regret in order to make myself feel better now.	1	2	3	4
16.	Sometimes when I feel bad, I can't seem to stop what I am doing even though it is making me feel worse.	1	2	3	4
17.	When I am upset I often act without thinking.	1	2	3	4
18.	When I feel rejected, I will often say things that I later regret.	1	2	3	4
19.	It is hard for me to resist acting on my feelings.	1	2	3	4
20.	I often make matters worse because I act without thinking when I am upset.	1	2	3	4
21.	In the heat of an argument, I will often say things that I later regret.	1	2	3	4
22.	I am always able to keep my feelings under control.	1	2	3	4
23.	Sometimes I do things on impulse that I later regret.	1	2	3	4
24.	I generally seek new and exciting experiences and sensations.	1	2	3	4
25.	I'll try anything once.	1	2	3	4
26.	I like sports and games in which you have to choose your next move very quickly.	1	2	3	4

27.	I would enjoy water skiing.	1	2	3	4
28.	I quite enjoy taking risks.	1	2	3	4
29.	I would enjoy parachute jumping.	1	2	3	4
30.	I welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional.	1	2	3	4
31.	I would like to learn to fly an airplane.	1	2	3	4
32.	I sometimes like doing things that are a bit frightening.	1	2	3	4
33.	I would enjoy the sensation of skiing very fast down a high mountain slope.	1	2	3	4
34.	I would like to go scuba diving.	1	2	3	4
35.	I would enjoy fast driving.	1	2	3	4
36.	I generally like to see things through to the end.	1	2	3	4
37.	I tend to give up easily.	1	2	3	4
38.	Unfinished tasks really bother me.	1	2	3	4
39.	Once I get going on something I hate to stop.	1	2	3	4
40.	I concentrate easily.	1	2	3	4
41.	I finish what I start.	1	2	3	4
42.	I'm pretty good about pacing myself so as to get things done on time.	1	2	3	4
43.	I am a productive person who always gets the job done.	1	2	3	4
44.	Once I start a project, I almost always finish it.	1	2	3	4
45.	There are so many little jobs that need to be done that I sometimes just ignore them all.	1	2	3	4

Positive Urgency Measure

Using the scale provided, please indicate how much each of the following statement reflects how you **TYPICALLY** are.		Disagree Strongly			Agree Strongly
01.	When I am very happy, I can't seem to stop myself from doing things that can have bad consequences.	1	2	3	4
02.	When I am in great mood, I tend to get into situations that could cause me problems.	1	2	3	4
03.	When I am very happy, I tend to do things that may cause problems in my life.	1	2	3	4
04.	I tend to lose control when I am in a great mood.	1	2	3	4
05.	When I am really ecstatic, I tend to get out of control.	1	2	3	4
06.	Others would say I make bad choices when I am extremely happy about something.	1	2	3	4
07.	Others are shocked or worried about the things I do when I am feeling very excited.	1	2	3	4
08.	When I get really happy about something, I tend to do things that can have bad consequences.	1	2	3	4
09.	When overjoyed, I feel like I can't stop myself from going overboard.	1	2	3	4
10.	When I am really excited, I tend not to think of the consequences of my actions.	1	2	3	4
11.	I tend to act without thinking when I am really excited.	1	2	3	4
12.	When I am really happy, I often find myself in situations that I normally wouldn't be comfortable with.	1	2	3	4
13.	When I am very happy, I feel like it is OK to give in to cravings or overindulge.	1	2	3	4
14.	I am surprised at the things I do while in a great mood.	1	2	3	4

Brief Self-Control Scale

Using the scale provided, please indicate how much each of the following statement reflects how you **TYPICALLY** are.		Not at All				Very Much
01.	I am good at resisting temptation	1	2	3	4	5
02.	I have a hard time breaking bad habits	1	2	3	4	5
03.	I am lazy	1	2	3	4	5
04.	I say inappropriate things	1	2	3	4	5
05.	I do certain things that are bad for me, if they are fun	1	2	3	4	5
06.	I refuse things that are bad for me	1	2	3	4	5
07.	I wish I had more self-discipline	1	2	3	4	5
08.	People would say that I have iron self-discipline	1	2	3	4	5
09.	Pleasure and fun sometimes keep me from getting work done	1	2	3	4	5
10.	I have trouble concentrating	1	2	3	4	5
11.	I am able to work effectively towards long-term goals	1	2	3	4	5
12.	Sometimes I can’t stop myself from doing something, even if I know it is wrong	1	2	3	4	5
13.	I often act without thinking through all the alternatives	1	2	3	4	5

Monetary Choice Questionnaire

For each of the next 27 choices, please indicate which reward you would prefer: the smaller reward today or the larger reward in the specified number of days.

		Today	Delayed
01.	Would you prefer $54 today, or $55 in 117 days?	0	1
02.	Would you prefer $55 today, or $75 in 61 days?	0	1
03.	Would you prefer $19 today, or $25 in 53 days?	0	1
04.	Would you prefer $31 today, or $85 in 7 days?	0	1
05.	Would you prefer $14 today, or $25 in 19 days?	0	1
06.	Would you prefer $47 today, or $50 in 160 days?	0	1
07.	Would you prefer $15 today, or $35 in 13 days?	0	1
08.	Would you prefer $25 today, or $60 in 14 days?	0	1
09.	Would you prefer $78 today, or $80 in 162 days?	0	1
10.	Would you prefer $40 today, or $55 in 62 days?	0	1
11.	Would you prefer $11 today, or $30 in 7 days?	0	1
12.	Would you prefer $67 today, or $75 in 119 days?	0	1
13.	Would you prefer $34 today, or $35 in 186 days?	0	1
14.	Would you prefer $27 today, or $50 in 21 days?	0	1
15.	Would you prefer $69 today, or $85 in 91 days?	0	1
16.	Would you prefer $49 today, or $60 in 89 days?	0	1
17.	Would you prefer $80 today, or $85 in 157 days?	0	1
18.	Would you prefer $24 today, or $35 in 29 days?	0	1
19.	Would you prefer $33 today, or $80 in 14 days?	0	1
20.	Would you prefer $28 today, or $30 in 179 days?	0	1
21.	Would you prefer $34 today, or $50 in 30 days?	0	1
22.	Would you prefer $25 today, or $30 in 80 days?	0	1
23.	Would you prefer $41 today, or $75 in 20 days?	0	1
24.	Would you prefer $54 today, or $60 in 111 days?	0	1
25.	Would you prefer $54 today, or $80 in 30 days?	0	1
26.	Would you prefer $22 today, or $25 in 136 days?	0	1
27.	Would you prefer $20 today, or $55 in 7 days?	0	1

Risk Perceptions

"What is the likelihood that you would experience some negative consequences (e.g., become sick, be injured, be embarrassed, suffer legal consequences, or feel bad about yourself) if you…"		Not at all likely		Some -what likely		Very likely
1.	engaged in any type of gambling (e.g. casino, sports, track, bingo, online lottery, etc.) ?	1	2	3	4	5
2.	used illicit drugs (e.g. marijuana, cocaine, prescription drugs not prescribed to you by a physician)?	1	2	3	4	5
3.	drove after drinking?	1	2	3	4	5
4.	rode with a driver who had been drinking?	1	2	3	4	5
5.	had potentially unsafe oral sex with a romantic partner (e.g. did not use protection against STDs)?	1	2	3	4	5
6.	had potentially unsafe vaginal sex with a romantic partner (e.g. did not use protection against pregnancy or STDs)?	1	2	3	4	5
7.	had potentially unsafe anal sex with a romantic partner (e.g. did not use protection against STDs)?	1	2	3	4	5
8.	had potentially unsafe oral sex outside a romantic relationship (e.g. did not use protection against STDs)?	1	2	3	4	5
9.	had potentially unsafe vaginal sex outside a romantic relationship (e.g. did not use protection against pregnancy or STDs)?	1	2	3	4	5
10.	had potentially unsafe anal sex outside a romantic relationship (e.g. did not use protection against STDs)?	1	2	3	4	5
11.	got into a physical fight?	1	2	3	4	5
12.	got into a verbal argument?	1	2	3	4	5
13.	destroyed property or stole something?	1	2	3	4	5

Alcohol Use and Consequences

Timeline Follow-Back Interview

Study ID: _____

October 2012 (example month)						
Monday	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**	**Sunday**
1	2	3	4	5	6 UT vs. West Virginia	7
8	9	10	11	12	13 UT vs. Oklahoma	14
15	16	17	18	19	20 UT vs. Baylor	21
22	23	24	25	26	27 UT at Kansas	28
29	30	31 Halloween				

____ Max Drinks

____Total Drinks Consumed

____Binge Episodes

(4+/5+ Standard Drinks in a Sitting)

____ Times Drunk

____ Date of Period Onset

Frequency of:

____ Memory

____ Hangover

____ Gambling

____ Unsafe sex

____ Illicit drug use

____ Driving after drinking

____ Riding with a driver who had been drinking

____ Property crime (theft or vandalism)

____ Arguing verbally

____ Fighting physically

Scored by: _____

Checked and Entered by: _____

Alcohol Use Disorders Identification Test

Mark the box that best describes your answer to each question.

1.	How often do you have a drink containing alcohol?	Never	Monthly or less	2-4 times a month	2-3 times a week	4 or more times a week
2.	How many drinks containing alcohol do you have on a typical day when you are drinking?	1 or 2	3 or 4	5 or 6	7 to 9	10 or more
3.	How often do you have six or more drinks on one occasion?	Never	Less Than Monthly	Monthly	Weekly	Daily or Almost Daily
4.	How often during the last year have you found that you were not able to stop drinking once you had started?	Never	Less Than Monthly	Monthly	Weekly	Daily or Almost Daily
5.	How often during the last year have you failed to do what was normally expected of you because of drinking?	Never	Less Than Monthly	Monthly	Weekly	Daily or Almost Daily
6.	How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?	Never	Less Than Monthly	Monthly	Weekly	Daily or Almost Daily
7.	How often during the last year have you had a feeling of guilt or remorse after drinking?	Never	Less Than Monthly	Monthly	Weekly	Daily or Almost Daily
8.	How often during the last year have you been unable to remember what happened the night before because of your drinking?	Never	Less Than Monthly	Monthly	Weekly	Daily or Almost Daily
9.	Have you or someone else been injured because of your drinking?	No		Yes, but not in the last year		Yes, during the last year
10.	Has a relative, friend, doctor, or other health care worker been concerned about your drinking or suggested you cut down?	No		Yes, but not in the last year		Yes, during the last year

Age at First Drink and First Intoxication

How old were you when you…

1. Took your first drink on your own rather than just a sip from an adult's glass, not including drinking as part of religious ceremonies? _____

2. First got drunk after drinking alcohol? _____

Self-Report Alcohol Challenge Measures

Subjective Alcohol Response Measures

Biphasic Alcohol Effects Scale

Please rate the extent to which these words describe your feelings at the present time**.**

		Not at All					Moderately				Extremely
1.	Difficulty Concentrating	0	1	2	3	4	5	6	7	8	9	10
2.	Down	0	1	2	3	4	5	6	7	8	9	10
3.	Elated	0	1	2	3	4	5	6	7	8	9	10
4.	Energized	0	1	2	3	4	5	6	7	8	9	10
5.	Excited	0	1	2	3	4	5	6	7	8	9	10
6.	Heavy Head	0	1	2	3	4	5	6	7	8	9	10
7.	Inactive	0	1	2	3	4	5	6	7	8	9	10
8.	Sedated	0	1	2	3	4	5	6	7	8	9	10
9.	Slow Thoughts	0	1	2	3	4	5	6	7	8	9	10
10.	Sluggish	0	1	2	3	4	5	6	7	8	9	10
11.	Stimulated	0	1	2	3	4	5	6	7	8	9	10
12.	Talkative	0	1	2	3	4	5	6	7	8	9	10
13.	Up	0	1	2	3	4	5	6	7	8	9	10
14.	Vigorous	0	1	2	3	4	5	6	7	8	9	10

Subjective High Assessment Scale

Please place an “X” on each line which you feel best estimates how you feel now.

<table>
<tr><th></th><th></th><th colspan="4">NO CHANGE ... MOST EXTREME</th></tr>
<tr><td>1.</td><td>Normal</td><td>0</td><td>|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|</td><td>36</td><td>High</td></tr>
<tr><td>2.</td><td>Normal</td><td>0</td><td>|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|</td><td>36</td><td>Clumsy</td></tr>
<tr><td>3.</td><td>Normal</td><td>0</td><td>|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|</td><td>36</td><td>Confused</td></tr>
<tr><td>4.</td><td>Normal</td><td>0</td><td>|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|</td><td>36</td><td>Dizzy</td></tr>
<tr><td>5.</td><td>Normal</td><td>0</td><td>|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|</td><td>36</td><td>Drunk</td></tr>
<tr><td>6.</td><td>Normal</td><td>0</td><td>|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|</td><td>36</td><td>Difficulty concentrating</td></tr>
<tr><td>7.</td><td>Normal</td><td>0</td><td>|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|</td><td>36</td><td>Feeling alcohol effects</td></tr>
</table>

Drug Effects Questionnaire

Do you feel any drug effects?

0 | | | | | | | | | | | 25 | | | | | | | | | | | 50 | | | | | | | | | | | 75 | | | | | | | | | | | 100

Not at all Extremely

Do you like the effects you are feeling now?

0 | | | | | | | | | | | 25 | | | | | | | | | | | 50 | | | | | | | | | | | 75 | | | | | | | | | | | 100

Not at all Extremely

Would you like more of what you consumed, right now?

0 | | | | | | | | | | | 25 | | | | | | | | | | | 50 | | | | | | | | | | | 75 | | | | | | | | | | | 100

Not at all Extremely

Profile of Mood States (Energetic and Intoxication Subscales Only)

Below is a list of words that describe feelings people have. Please read each one carefully. Then circle the number of the answer to the right which best describes HOW YOU FEEL RIGHT NOW.

	Not at all	A little	Moderately	Quite a bit	A lot
1) lively	1	2	3	4	5
2) sleepy	1	2	3	4	5
3) tired	1	2	3	4	5
4) off-balance	1	2	3	4	5
5) energetic	1	2	3	4	5
6) flushing	1	2	3	4	5
7) nauseous	1	2	3	4	5
8) probable impairment of driving ability	1	2	3	4	5
9) exhausted	1	2	3	4	5
10) dizziness	1	2	3	4	5
11) uncoordinated	1	2	3	4	5

Subjective Effects of Alcohol Scale

Instructions: The following adjectives describe feelings that are sometimes produced by drinking alcohol.
On a scale of 1-10, please rate the extent to which drinking alcohol has produced these feelings in you **at the present time**.

		Not At All					**Moderately**					**Extremely**
1.	Sociable	0	1	2	3	4	5	6	7	8	9	10
2.	Moody	0	1	2	3	4	5	6	7	8	9	10
3.	Demanding	0	1	2	3	4	5	6	7	8	9	10
4.	Mellow	0	1	2	3	4	5	6	7	8	9	10
5.	Carefree	0	1	2	3	4	5	6	7	8	9	10
6.	Rude	0	1	2	3	4	5	6	7	8	9	10
7.	Relaxed	0	1	2	3	4	5	6	7	8	9	10
8.	Woozy	0	1	2	3	4	5	6	7	8	9	10
9.	Fun	0	1	2	3	4	5	6	7	8	9	10
10.	Lively	0	1	2	3	4	5	6	7	8	9	10
11.	Calm	0	1	2	3	4	5	6	7	8	9	10
12.	Aggressive	0	1	2	3	4	5	6	7	8	9	10
13.	Dizzy	0	1	2	3	4	5	6	7	8	9	10
14.	Anxious	0	1	2	3	4	5	6	7	8	9	10
15.	Attractive	0	1	2	3	4	5	6	7	8	9	10
16.	Ill	0	1	2	3	4	5	6	7	8	9	10
17.	Funny	0	1	2	3	4	5	6	7	8	9	10
18.	Talkative	0	1	2	3	4	5	6	7	8	9	10
19.	Confident	0	1	2	3	4	5	6	7	8	9	10
20.	Happy	0	1	2	3	4	5	6	7	8	9	10
21.	Drunk	0	1	2	3	4	5	6	7	8	9	10
22.	Wobbly	0	1	2	3	4	5	6	7	8	9	10

Perceived Impairment and Willingness to Drive

On a scale of 1-10, circle the number of the answer which best describes you **at the present time**.

	Not At All				**Moderately**						**Extremely**
1)	How impaired do you think you are at present?										
	0	1	2	3	4	5	6	7	8	9	10
2)	How unsafe do you think it would be to drive an automobile at present?										
	0	1	2	3	4	5	6	7	8	9	10
3)	If you were at work now, would others think you were intoxicated or behaving unusually?										
	0	1	2	3	4	5	6	7	8	9	10
4)	How willing would you be to drive an automobile at present?										
	0	1	2	3	4	5	6	7	8	9	10

Placebo Manipulation Check

Research experiments do not always use the same standard servings as those typical used at bars or parties. Please estimate the number of standard alcoholic drinks you were served during this experiment. (1 Standard Drink = 12 ounces of beer, 1.5 ounces of liquor (1 shot straight or in a mixed drink), or 5 ounces of wine) _____

Follow-Up Daily Self-Monitoring Measures

Please enter your Study ID _____

Entry Date (The date your diary responses refer to, NOT today's date) _____

Relationship Status

What is your current relationship status?

- ○ Not dating
- ○ Dating, but not exclusively
- ○ Dating exclusively
- ○ Engaged
- ○ Married
- ○ Other (please specify):_____

Estimated Blood Alcohol Concentration; Co-ingestion with Caffeine and Tobacco

NOTE: One standard drink is equivalent to 12 oz. of beer, 1.5 oz. of liquor in a shot or mixed drink, or 5 oz. of wine.

1. How many standard drinks did you consume yesterday? _____

Questions 2-11 only if participant drank alcohol yesterday.

2. How many of those standard drinks also contained energy drinks (e.g., vodka and red bull, Jagerbomb)? _____

3. How many standard drinks also contained other caffeinated beverages (e.g., rum and coke, Irish coffee)? _____

4. When you drank yesterday, how many energy drinks that were NOT mixed with alcohol did you consume? _____

5. When you drank yesterday, did you also use any tobacco products (e.g., cigarettes, cigars, pipes, smokeless tobacco)? ○ Yes ○ No

6. How many times did you drink yesterday? (e.g., if you drank at a sporting event in the morning and drank at a party in the evening, you drank two times) _____

7. Of the times that you drank yesterday, how long (in hours) was your heaviest drinking episode (from the time you started your first drink to the time you finished your last drink)?_____

Brief Biphasic Alcohol Effects Scale

8. Please rate the extent to which these words describe your feelings during your heaviest drinking episode yesterday.

		Not at All					**Moderately**					**Extremely**
a.	Energized	0	1	2	3	4	5	6	7	8	9	10
b.	Excited	0	1	2	3	4	5	6	7	8	9	10
c.	Sedated	0	1	2	3	4	5	6	7	8	9	10
d.	Slow Thoughts	0	1	2	3	4	5	6	7	8	9	10
e.	Sluggish	0	1	2	3	4	5	6	7	8	9	10
f.	Up	0	1	2	3	4	5	6	7	8	9	10

Subjective Intoxication

9. During your heaviest drinking episode yesterday…

	Not at all drunk		Slightly drunk		Moderately drunk				Very drunk		Extremely drunk
How drunk did you feel?	0	1	2	3	4	5	6	7	8	9	10

Drinking Context

10. Where or in what location did the drinking take place? Mark all that all that apply.

- O My own residence
- O My parents home
- O A restaurant or cafe
- O My friends residence
- O A bar or club
- O A recreational event (e.g., sports, music, festival)
- O Other (please specify) _____

11. Who were you with when the drinking took place? Mark all that all that apply.

- O I was alone
- O Close friends
- O People I didn't know
- O Acquaintances
- O My boyfriend/girlfriend/partner
- O Parents
- O Brothers or sisters
- O Other family members
- O Other (please specify) _____

Behavioral Risks

In which of the following activities did you participate yesterday? (*mark all that apply*)	Did not engage in activity	Engaged in activity when I had NOT been drinking	Engaged in activity when I HAD been drinking
Gambled (e.g., casino, sports, track, bingo, online lottery).	○	○	○
Had potentially unsafe sex (e.g., did not use condom or other protection against STDs and pregnancy).	○	○	○
Used illicit drugs (e.g., marijuana, cocaine, prescription medication not prescribed to you by a physician).	○	○	○
Drove after drinking.	○	○	○
Rode with a driver who had been drinking.	○	○	○
Destroyed property or stole something.	○	○	○
Got into a verbal argument.	○	○	○
Got into a physical fight.	○	○	○
Engaged in at least one instance of oral sex.	○	○	○
If you engaged in at least one instance of oral sex,			
Was this with a romantic partner?	○ Yes		○ No
Did you regret having oral sex?	○ Yes		○ No
Was the oral sex potentially unsafe (e.g., you did not use a condom or other protection against STDs)?	○ Yes		○ No
Engaged in at least one instance of vaginal sex.	○	○	○
If you engaged in at least one instance of vaginal sex,			
was this with a romantic partner?	○ Yes		○ No
did you regret having vaginal sex?	○ Yes		○ No
was the vaginal sex potentially unsafe (e.g., you did not use a condom or other protection against STDs and pregnancy)?	○ Yes		○ No
Engaged in at least one instance of anal sex.	○	○	○
If you engaged in at least one instance of anal sex,			
was this with a romantic partner?	○ Yes		○ No
did you regret having anal sex?	○ Yes		○ No
was the anal sex potentially unsafe (e.g., you did not use a condom or other protection against STDs)?	○ Yes		○ No

Alcohol-Related Consequences

Which of the following consequences did you experience as a result of your drinking last night (*mark all that apply*)?	Did not experience consequence	Experienced consequence
1. Emotional (e.g., had regrets, felt angry, or felt worried)	○	○
2. Social (e.g., felt rejected or hurt your reputation)	○	○
3. Disciplinary (e.g., got caught, arrested, or punished)	○	○
4. Financial (e.g., spent or lost money)	○	○
5. Academic or work (e.g., missed class or work, failed an exam)	○	○
6. Sickness (e.g., felt sick, vomited)	○	○
7. Hangover (e.g., the next day, had a headache, felt sick, vomited)	○	○
8. Memory (had difficulty remembering things you said or did or events that happened that night)	○	○
9. Injured yourself or were injured	○	○
10. Injured someone else	○	○

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