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Effects of Alcohol on Psychomotor Performance and Perceived Impairment in Heavy Binge Social Drinkers

Ty Brumback¹, Dingcai Cao², and Andrea King¹

¹The University of Chicago, Department of Psychiatry Chicago, IL 60637 USA

²The University of Chicago, Departments of Health Studies and Ophthalmology and Visual Science Chicago, IL 60637 USA

Abstract

Alcohol intoxication often results in negative consequences; however, specific behavioral and subjective effects often vary as a function of individual differences. The present study utilized an alcohol challenge paradigm to examine whether heavy binge social drinkers (HD; $n=77$), compared to light social drinkers (LD; $n=55$), exhibit: 1) greater tolerance in psychomotor task performance under the influence of alcohol, and 2) differential perceptions of the impairing effects of alcohol. The study included three test sessions in which participants consumed either a low (0.4 g/kg) or a high (0.8 g/kg) dose of ethanol or a placebo beverage administered in random order and counterbalanced within group. Participants completed the Digit Symbol Substitution Task (DSST) and the Grooved Pegboard at pre-drink baseline and at multiple time points after beverage consumption. They also completed a scale of perceived impairment at several intervals after beverage consumption. Ethanol impaired performance at the high dose, but not at the low dose ($ps < .001$). The groups exhibited similar alcohol-induced impairment. However, HD reported lower self-perceived impairment compared to LD, particularly during the early portion of the blood alcohol curve ($p < .001$) when actual impairment was most pronounced. Thus, this study extends prior research in that habitual binge social drinking does not appear to be associated with tolerance to alcohol's impairing effects on select psychomotor skills. Further, results may have implications for alcohol-related harm as binge social drinkers regularly consume intoxicating doses of alcohol but may not be aware of the physical and cognitive impairments produced by alcohol.

Keywords

alcohol; binge social drinkers; psychomotor; DSST; Pegboard; perceived impairment

1. Introduction

Acute alcohol intoxication results in diminished cognitive and psychomotor abilities, which have important implications for public health and safety. For example, it is well established that the risk of automobile accidents increases dramatically when the driver consumes alcohol

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Send inquiries to: Ty Brumback, MA University of South Florida- Department of Psychology 4204 E. Fowler Ave. PCD 4118G Tampa, FL 33620-8200 TEL: 813-974-8198 FAX: 813-974-3409 Email: tbrumbac@mail.usf.edu.

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(Williams, 2006). In fact, alcohol related deaths comprised 39% of traffic fatalities in the US in 2005 (NHTSA, 2007). While most statutory limits for driving are set at blood alcohol concentrations (BACs) of 0.08 g/dl and higher, human laboratory research has shown that BACs above 0.05 g/dl significantly impair performance on some motor tasks such as tracking, tapping, reaction time, and body sway (Mitchell, 1985; for reviews see Eckardt et al., 1998; and Finnigan & Hammersley, 1992). Alcohol levels >0.05 have also been shown to impair more cognitively demanding psychomotor tasks such as inhibiting responses in a go/no-go task (e.g., Fillmore et al., 2005), and responding correctly in paradigms employing unrelated choice-response tasks for left and right hands simultaneously (e.g., Schweizer et al., 2004). In general, sensitivity to the impairing effects of alcohol is relative to the complexity of the psychomotor task, so more demanding tasks may be hindered by alcohol at lower levels than easier tasks (e.g., Hindmarch et al., 1991).

In addition to psychomotor impairment, alcohol affects cognitive processes such as perception and judgment. Moderate doses of alcohol (0.5 to 0.8 g/kg) have been shown to increase confidence in performance on a general knowledge test (Tiplady et al., 2004), and to decrease judgments of the probability of negative versus positive consequences (Fromme et al., 1997). Furthermore, BACs as low as 0.07 reduce social drinkers' ability to estimate their BACs and their fitness to drive a car (Bierness, 1987). Taken together, these studies demonstrate that moderate to heavy alcohol consumption alters judgment, reasoning, and decision making that could put drinkers at risk for serious harm and related consequences.

There is a long history of research over the past eighty years examining motor and performance-related behaviors during acute intoxication as well as in non-intoxicated states in persons of various drinking behaviors, from abstainers to moderate drinkers to alcoholics (e.g., Hollingsworth 1923, 1924; Goldberg, 1943; Ekman et al., 1964; Mitchell 1985; and Dengerink et al., 1978). These studies have shown that the heaviest and most experienced drinkers often demonstrate behavioral tolerance or lesser impairment to alcohol than lighter drinkers or very infrequent drinkers. These findings are consistent across many studies on measures of sensory perception, memory tasks, psychomotor tasks, and steadiness of gait or body sway. Subjective responses after drinking may also differ in very heavy drinkers compared to novice or light drinkers, with the former group anticipating less intoxication and underestimating post-drinking intoxication levels relative to the latter group (Gabrielli et al., 1991). However, on other responses, such as divided attention skills, very heavy drinkers are not more tolerant to alcohol's impairing effects compared to novice drinkers (see Mitchell, 1985).

More recent research over the past decade area has focused on comparing alcohol impairment within a more tightly defined range of drinking, namely, heavier or lighter nonalcoholic *social drinkers*. Some of these studies have supported the results of the past studies, with experienced social drinkers exhibiting less alcohol impairment than less-experienced drinkers, as measured by the pursuit rotor task (Fillmore & Vogel-Sprott, 1995, 1996) and the Pegboard and Digit-Symbol Substitution Task (DSST) (Evans & Levin, 2004). However, other studies have not supported behavioral tolerance in heavier social drinkers, as measured by eye movement tasks and event-related potentials (Ramchandani et al., 2002) and in cognitive tasks such as pattern matching and spatial recognition (Weissenborn & Duka, 2003). The variability in results across studies may be due to several factors, including small sample sizes, lack of parallel self-report measures of impairment, and widely ranging definitions for heavy social drinking, which has been determined by duration of drinking (2 or more years), drinking quantity (35 or more weekly drinks) or recent drinking history (e.g., post-hoc determination of the heaviest alcohol consumers over the 28-day period before testing).

In a recent preliminary study, our group sought to clarify the issue by comparing alcohol response factors within social drinkers and by selecting two distinct social drinker groups a

priori. Heavy drinking was defined using a combination of quantity and frequency, as well as habitual alcohol misuse via weekly “binge” drinking (5+ drinks consumed on an occasion; 4+ for females; SAMHSA, 2005). The comparison group was also well-defined in terms of habitual light social drinkers, defined by a lifetime predominance of consuming one to six drinks weekly with no or very rare binge drinking occasions. Findings from this study showed mixed results for behavioral tolerance in the heavy drinkers, as heavy social drinkers exhibited less alcohol-induced impairment on a smooth pursuit eye tracking but comparable impairment on the DSST as light drinkers (King & Byars, 2004). Moreover, heavy drinkers showed more sensitivity to impairment on the latency to initiate saccadic eye movements than light drinkers, but this was observed only at a low dose of alcohol (0.4 g/kg) and not a high dose (0.8 g/kg) (King & Byars, 2004). While this preliminary investigation had strengths in examining various responses to multiple doses of alcohol (and placebo) in two well-defined groups of social drinkers, limitations were similar to prior studies, with a modest sample size ($n = 34$) and lack of concurrent assessment of subjects' perception of impairment.

The goal of the present study was to examine both psychomotor performance responses and self-rated perceptions of impairment in two well-defined groups of young adult social drinkers: habitual heavy binge and light social drinkers. The main study question was whether binge social drinkers would show evidence of behavioral tolerance to alcohol's effects on psychomotor performance compared to their light-drinker counterparts, and if so, would this response be dose- or time-dependent? Based on prior studies, we hypothesized that a high alcohol dose (0.8g/kg) would produce significantly more impairment on two psychomotor tasks than a low dose (0.4g/kg) or placebo beverage. We also predicted that heavy drinkers would underestimate alcohol's impairing effects by reporting lower *perceived* impairment than light drinkers.

2. Method

2.1 Participants

Healthy male and female study candidates, aged 21-35 years, were recruited through flyers, advertisements in local Chicago newspapers, websites, and by word-of-mouth referrals. Subjects for this study were taken from the larger, ongoing Chicago Social Drinking Project (CSDP), an experimental and longitudinal study of social drinkers and risk for alcohol problems. Two groups were recruited for CSDP and were based on predominant lifetime adult drinking patterns (i.e., at least the last two years or longer). Heavy social drinkers (HD) were nonalcoholic persons who regularly consumed 10 or more drinks weekly and had regular (one to five times weekly) occurrences of “binge” drinking (5+ drinks on one occasion, 4+ drinks for females). These patterns were selected to be consistent with prior studies on heavy social drinkers and are reflective of patterns that likely produce intoxication and some loss of control on a regular basis (King & Epstein, 2005; King & Byars, 2004; King et al., 2002). Light social drinkers (LD) drank fewer than 6 drinks weekly with rare or no “binge” drinking history. Drinking patterns were determined by the Quantity-Frequency Index (QFI; Cahalan, et al., 1969) and a Timeline Follow-Back interview (TLFB; Sobell & Sobell, 1995; Sobell et al., 1979).

Interested candidates were screened over the telephone for basic study inclusion criteria, including a body mass index between 19-30, no major current medical or psychiatric condition, and specific alcohol use patterns. Those persons who met the basic criteria were subsequently invited for an in-lab screening session, where they underwent a physical examination by a resident physician, a pregnancy test (females only), a urine toxicology test, a blood test to examine blood chemistry and liver functioning, a diagnostic interview, and several psychosocial and health history questionnaires. The questionnaires included the Beck Depression Inventory (BDI; Beck et al., 1961), State-Trait Anxiety Inventory (STAI;

Spielberger et al., 1970), and the Short Michigan Alcoholism Screening Test (SMAST; Selzer et al., 1975). The diagnostic interview included the screening portion of the Structured Diagnostic Interview for the DSM IV (SCID; First et al., 1995), as well as specific modules for mood and substance use disorders. Participants were excluded if they were taking any psychotropic medications, had any major medical or psychiatric conditions including past or current alcohol or substance dependence (history of alcohol abuse permitted), scored outside of standard cutoff thresholds on the questionnaires, or had a positive urine toxicology screening (except for marijuana).

Family history (FH) for alcohol use disorders was also obtained at baseline by a two generational family tree to include all primary and secondary biological relatives. When participants identified a family member with an alcohol use disorder, they were asked follow-up questions consistent with the FH-RDC for drinking consequences (Andreasen et al., 1977). Because we enrolled participants without regard to FH, there was considerable variability in the number of relatives with alcohol use disorders. In order to include as many participants as possible in secondary analyses using FH classifications, a broad definition of FH+ was employed which included those persons with two or more secondary biological relatives or one or more primary relative identified with an alcohol use disorder. FH- was defined as having no biological relatives with alcohol use disorders in the last two generations. Participants who could not be classified on family history, due to insufficient information provided or only one secondary relative with an alcohol problem, were excluded from these analyses (i.e., 35 subjects, or 26% of the sample).

2.2 Procedure

During the screening session, participants signed informed consent, which was approved by the University of Chicago Institutional Review Board. To reduce potential alcohol expectancies, participants were informed that they might receive a stimulant, sedative, alcohol, or placebo or any combination of these during their three experimental sessions. All participants actually received (on separate sessions) a placebo beverage (1% volume of ethanol as a taste mask), a low alcohol dose beverage (0.4 g/kg), and a high alcohol dose beverage (0.8 g/kg), in random order and counterbalanced within group. The drinks were prepared with Kool-Aid®, water, Splenda®, and the appropriate dose of 190-proof ethanol based on body weight. Adjustments were made for women to receive an approximate 90 percent dose for that of men due to differences in total body water affecting blood alcohol concentrations (Frezza et al., 1990; Sutker et al., 1983). A separate coder prepared the beverages so that the research assistant was kept blind to the beverage content.

Each participant completed three early evening 4 to 5 hour laboratory sessions separated by at least 48 hours, which were conducted in a comfortably furnished room with a television and reading materials. They were instructed to abstain from alcohol and recreational drugs for 48 hours before each session, and to abstain from caffeine, food, and cigarettes for three hours prior to each session. Upon arrival, they were interviewed to assess compliance with these alcohol, drug, caffeine, food, and smoking abstinence requirements. The participant arrived at the laboratory between 15:00 and 17:00h and underwent a pregnancy test (if female), a breathalyzer test, and a random urine toxicology test at one of the three sessions. After the participant completed pre-session measures, he or she consumed a low-fat snack (20% daily calories) to avoid hunger effects on mood state and to help reduce the potential for alcohol-induced nausea.

Thirty minutes after arriving, the participant completed baseline questionnaires and performance measures. Approximately 45 minutes after arriving, the participant consumed the assigned beverage through a straw from a plastic, lidded cup, in order to help conceal the scent and identification of the alcohol content. Prior to consuming the beverage, he or she was again

reminded that the drink could contain alcohol, a stimulant, a sedative, a placebo, or some combination of the substances. The participant had 5 minutes to consume one half of the beverage, followed by a 5-minute rest period, and then another 5 minutes to consume the second half of the beverage. Similar beverage administration procedures have been used in the past and have shown reliable rising and declining blood alcohol curves across participants (King & Epstein, 2005; King & Byars, 2004; King et al., 2002). All three sessions were identical with the exception of beverage alcohol content. The participant then completed the performance measures, questionnaires, and breathalyzer readings at 15, 45, 105, and 165 minutes after consuming the beverages. The questionnaire of perceived impairment was administered at 15 and 165 minutes after completion of the beverage consumption. These two intervals were chosen to avoid complacency with multiple measurements while capturing the early rising BAC as well as the latter excretion phase of alcohol metabolism. Participants were allowed to read or watch selected emotionally neutral videos during the session when study measures were not being recorded. At the end of each session, the participants were transported home. The participants were debriefed at the end of the study and were mailed a check for \$200 (\$50 per session plus a \$50 bonus for compliance) within 2 weeks after study completion.

2.3 Dependent Measures

Objective measures of impairment were obtained through two performance tasks. The DSST (from WAIS-R; Wechsler, 1958), which is a pencil and paper psychomotor task that measures perceptual-motor processing speed. Participants were given 90 seconds to complete as many items as possible and scores were based on the number of correct items coded in the time allowed. This task has been shown to be sensitive to drug and alcohol-related impairment (e.g., Holdstock & de Wit, 2001; Doty et al., 1997). Five different but equivalent forms were used so that within each session participants completed each form only once to reduce order and learning effects.

The Grooved Pegboard (Lafayette Instruments, Lafayette, IN), a test of motor speed and fine motor coordination, followed the DSST at each time point. This test has been used for evaluation of lateralized brain damage in neuropsychological test batteries. It has also been used as a general test of motor speed and fine motor coordination and has been shown to be sensitive to alcohol-induced impairment (e.g. Ramchandani et al., 1999; Ramchandani et al., 2002). The test requires participants to quickly retrieve, rotate, and insert 25 small metal pegs into 25 slotted holes with random orientations on a board. Each participant completed the task using only the non-dominant hand. Two dependent measures are derived from this test, including the time in seconds for correct positioning of all pegs and the number of pegs dropped. To reduce a potential confound of early learning effects on performance, candidates practiced the task 1 to 2 times during the screening session (Ramchandani, personal communication).

Participants completed three self-rated items of perceived impairment at 15 and 165 minutes after drink completion: Item 1: “how impaired do you think you are at present?”, Item 2: “how unsafe do you think it would be to drive an automobile at present?”, and Item 3: “if I were at work now, others might think I was intoxicated or behaving unusually” with 10-point rating scales for each, anchored at 1 for “not at all” and 10 for “extremely”.

2.4 Statistical Analyses

Participant characteristics were compared between HD and LD groups using Student t-tests for continuous variables or χ^2 tests for categorical variables. Analyses for the primary dependent variables used raw scores at each time point before and after beverage consumption. Mixed linear models were used to evaluate the main effects of dose, time, and group effects as well as their interactions for performance (Pegboard time and DSST number correct), self-rated perceived impairment, and BAC. Confirmatory analyses were conducted using change

scores, which were derived by subtracting each post-beverage measure from its respective within session baseline measure. The presence of drops in the Pegboard task, which was a binary variable, was analyzed using a generalized mixed linear model with a logit link function. Factors that differed between the groups or that have been shown to relate to psychomotor response in prior research, such as FH, were included in subsequent analyses to examine their influence on alcohol-induced performance impairment. Multiple linear regression was used to examine the relationship between self-rated and actual impairment. The criterion of $p < .05$ was used to determine significance of statistical tests.

3. Results

3.1 Background characteristics

There were 138 participants who enrolled in the study. However, 6 participants did not complete all experimental sessions due to various factors, including schedule conflicts ($n = 4$), an adverse response ($n = 1$), and a positive drug test ($n = 1$). Therefore, the final sample consisted of 132 participants, with $n = 77$ in the HD group and $n = 55$ in the LD group. Background and drinking characteristics are listed in Table 1. The HD had significantly more drinking occasions per week, drinks per occasion, and binges than the LD. The average duration of bingeing on a monthly or more frequent basis for HDs was 7.77 ± 0.38 years.

3.2 Main Dependent Variables

3.2.1 BAC—As expected, alcohol increased BAC levels in both HD and LD groups. Similar to prior research, the HD achieved higher BAC levels than LD after the low and high dose [Group $\times$ Time: $F(4,516) \geq 4.66$, $ps < .001$; Table 2]. Post-hoc tests revealed that the HD had significantly higher BAC levels during the rising limb of the BAC (15 to 30 min for low dose and 15 to 45 min for high dose, $p < .01$) compared to the LD.

3.2.2 Performance Measures—The top row of Figure 1 depicts Pegboard performance (i.e., time to complete) for each group at each dose across time. Analyses indicated that alcohol impaired performance on the Pegboard at the high dose of alcohol [Dose $\times$ Time: $F(8, 1040) = 11.7$, $p < .001$ (Tukey's HSD: high $>$ low = placebo)]. In both groups, Pegboard performance was relatively stable after consumption of the placebo and the low dose beverage. However, after the high dose of alcohol, impairment was evident at all time points except at the final time point (165 minutes). Pegboard performance did not differ between the HD and LD groups, as neither main effect nor interaction terms were significant. High dose alcohol, but not the low dose, also significantly increased the incidence of Pegboard drops [Dose $\times$ Time: $F(8, 1040) = 2.67$, $p < .01$], with significant increases in drops occurring at 15, 45, and 105 minutes [$\chi^2(1) \geq 10.58$, $ps \leq .001$]. As with Pegboard time, there were no differences between the groups on incidence of Pegboard drops [$\chi^2(1) \leq 1.22$, ns].

Alcohol also impaired performance on the DSST at the high dose [Dose $\times$ Time: $F(8, 1040) = 2.67$, $p < .001$ (Tukey's HSD: high $>$ low = placebo); bottom row of Figure 1]. Similar to the Pegboard, DSST performance (number of items correct) was relatively stable throughout the placebo and low dose sessions. After the high dose, impairment was evident throughout testing except for the last time point. As with Pegboard, the groups showed similar alcohol-induced impairment. Finally, analyses using change scores for both tasks confirmed the main analyses stated previously with raw scores [Dose $\times$ time: Pegboard, $F(6,780) = 8.16$, $p < .001$; DSST, $F(6,780) = 28.25$, $p < .001$].

After controlling for variables that differed between groups including years of education, sex, and BAC, results did not change for either the Pegboard or the DSST as both groups continued to show similar impairment. In terms of order effects, regardless of dose, participants performed

better at their second and third sessions compared to their first session [$F_s(2,393) \geq 11.64$, $p_s < .001$]. Analyses controlling specifically for the order in which participants received their high dose did not alter the results of the main analyses. That is, regardless of the order in which the high dose was received, alcohol produced impairment on Pegboard and the DSST tasks [time: $F_s(4,504) > 31.08$, $p_s < 0.001$]. Finally, secondary analyses of the influence of FH showed that alcohol affected performance similarly in the broadly defined FH+ and FH- groups. After controlling for FH, the main study findings were not altered.

3.2.3 Self-Rated Responses—Figure 2 illustrates the mean scores of the self-rated response items by dose. LD reported higher ratings than HD on item 2 (ability to drive an automobile) and item 3 (if other's would detect impairment) at the first time interval following low dose alcohol, and on items 1 (overall impairment), 2, and 3 at both post-drinking intervals following high dose alcohol [Group: $F_s(1,130) \geq 4.75$, $p_s < .05$; post hoc pairwise comparisons, $p_s < .05$; Figure 2, middle graph & right graph]. After consuming either dose, subjects self-rated impairment for item 2 higher than for items 1 or 3 [$F_s(2,260) \geq 33.41$, $p_s < .001$; post hoc pairwise comparisons: item 2 > item 1 = item 3]. Since there was a high intercorrelation among the three items [$r_s(130) \geq 0.71$, $p_s < .001$], ratings for the 3 items were averaged to form a single composite perceived impairment index for subsequent regression-based analyses.

3.2.4 Association between perceived and actual performance—After controlling for group, sex, and family history of alcoholism, the perceived impairment index score was significantly associated with actual performance impairment on the DSST at both post-beverage intervals [15 min: $\beta(\text{se}) = -0.20 (0.09)$, $p < .05$; 165 min: $\beta(\text{se}) = -0.19 (0.09)$, $p < .05$], and with the Pegboard at the latter interval [165 min: $\beta(\text{se}) = 0.22 (0.09)$, $p < 0.01$]. Both the LD and HD groups showed a similar positive relationship between perceived and actual impairment.

3.2.5 Dose-masking manipulation check—Finally, since oral alcohol administration may contain cues such as taste and smell than can be only partially masked, a supplemental manipulation check analysis was performed. Actual performance impairment on DSST and Pegboard and self-rated impairment were compared in participants who identified alcohol as the only substance received (42% of LDs and 47% of HDs; $\chi^2(1) = .27$, ns) versus those who did not (i.e., identified another substance either with or without alcohol). Results showed that there was no difference in actual or self-rated performance impairment in those who solely identified alcohol versus those who did not [actual performance: $F_s(1,130) \leq 2.77$, ns ; self-rated impairment: $F(1,130) \leq .66$, ns].

4. Discussion

This study demonstrated that heavy social drinkers, categorized by their persistent and predominant adult pattern of drinking and regular bingeing, exhibited comparable alcohol-induced impairment to that of light social drinkers in such areas as fine motor and dexterity skills (Pegboard) and processing and encoding functions (DSST) following consumption of a moderate-to-heavy dose of alcohol (4-5 standard drink equivalent). These results replicate findings pertaining to the DSST from our prior study of a smaller overall sample (34 participants; King & Byars, 2004), and extend these results to the Pegboard task. In addition, the HD reported significantly lower self-rated perception of alcohol's impairing effects than LD, even though they were actually equally impaired by alcohol. These effects were noted even after controlling for BAC level, and other potential confounds, such as sex, education level, or FH.

Repeated exposure to alcohol may result in behavioral tolerance, that is, a neuroadaptive process resulting in diminished response to alcohol after short-term (acute) or long-term

(chronic) consumption (Kalant et al., 1971). Individuals who persistently drink alcohol to the point of intoxication may gain experience under the influence of alcohol, and previous research supports that this experience can translate into tolerance through physiological compensation and cognitive control (e.g., Zack & Vogel-Sprott, 1995; Kalant, 1998). Contrary to past research demonstrating that heavier drinkers exhibit behavioral tolerance in cognitive and behavioral domains, the current study indicates that heavy binge social drinkers show similar cognitive-motor performance impairment after alcohol compared to their light drinking counterparts. These results are similar to a recent study showing that after drinking alcohol, binge social drinkers were either equally or even more impaired on memory tasks compared to non-binge social drinkers (Weissenborn & Duka, 2003). The current findings may also be a reflection of the group definitions used in this study which utilized strict criteria based on drinking experience and bingeing behavior that resulted in distinct drinking groups without co-existing Axis I disorders.

While both groups showed similar degrees of psychomotor impairment, the perception of impairment was significantly lower in the chronic binge social drinkers than in the light drinkers. Heavy drinkers self-rated lower perception than light drinkers of their impairment level, ability to drive an automobile, and whether they thought others would perceive their behavior as affected. These items require one to utilize interoceptive cues and to apply their perceptions to external contexts. Heavier drinkers have been shown to exhibit more alcohol-induced positive-like euphoric effects and reduced sedative-like effects after compared with light social drinkers, and these mood effects may play a role in their judgments of intoxication level (Holdstock et al., 2000; King et al., 2002). Others have suggested that heavier drinkers may deny the effects of alcohol when reporting perceived impairment (e.g., Gabrielli et al., 1991). It is also possible that heavy binge social drinkers judge interoceptive cues from alcohol intoxication to be less severe because they are more familiar with them. Thus, while it has been demonstrated that social drinkers under the influence of alcohol poorly estimate risk, performance, and intoxication levels (Tiplady et al., 2004; Fromme et al., 1997; Bierness, 1987), the current study indicates that regular binge drinking in particular is associated with lower awareness of alcohol-induced motor and cognitive impairments.

These findings may have particular clinical relevance and public safety implications. The peak impairment occurred within one hour of consuming a dose of alcohol that put participants at or above the legal BAC limit for driving an automobile in the United States. Experienced habitual binge drinkers may have false beliefs in their behavioral tolerance to psychomotor impairing effects of alcohol as indicated by their self-rated impairment. We may speculate that this could render them at greater risk for engaging in dangerous activities such as driving after drinking possibly resulting in harm to themselves and others. Lighter non-bingeing social drinkers appear to be at lower levels of risk after drinking as they rated their impairment levels significantly higher than heavy binge social drinkers. The difference in awareness of impairment under the influence of alcohol may be a direct result of one's experience under the influence of alcohol and one's drinking habits given that actual performance on two psychomotor tasks were comparable.

Some potential limitations in the study should be noted. First, peak BACs were higher in HD than in LD, which may be due to differences in first pass metabolism to increase alcohol bioavailability or to metabolizing enzymes in the liver (such as CYP2E1), which can be increased by chronic heavy drinking (Lieber, 1994). Although the BAC levels (between groups) were statistically significant, they are unlikely to be clinically significant since they were of a small magnitude and analyses controlling for BAC did not alter the main study findings. Second, while the high dose clearly impaired performance, which supports prior research, the low alcohol dose did not affect performance for either group. Peak BACs for the low dose session were ≤ 0.04 , but past research in general social drinkers indicates that

behavioral impairment is typically recorded at BACs ≥ 0.05 (Mitchell, 1985). Thus, future dose-ranging studies of alcohol to produce BACs at or above this level would provide an extension of the present results at more distinct subthreshold intoxication levels. Third, since the study used oral dosing, expectations regarding alcohol consumption and subsequent behavioral consequences could not be completely eliminated (Fillmore & Vogel-Sprott, 1996). However, precautions were taken to limit such cue-related expectancies and analyses indicated that performance and self-rated impairment was similar in those participants who believed they received alcohol alone versus those who believed they received another substance either with or without alcohol. Finally, because there are no validated instruments of self-rated perceived alcohol impairment, we derived several self-rated impairment items based on face validity. While similar measures have been used in the past, they are idiosyncratic and make it difficult to compare findings across studies.

In conclusion, the present study demonstrated that habitual binge social drinkers show comparable alcohol-induced behavioral impairment but less self-rated perception of impairment than their light social drinking counterparts. This result was evident even after controlling for the effects of BAC levels, dose order, sex, education, and family history of alcoholism. On novel cognitive-motor tasks, more experienced heavy drinkers appear to be similarly affected by alcohol as less experienced light drinkers. Given that they report less perceived alcohol-induced impairment than lighter drinkers despite similar levels of impairment, they could be at even greater risk for accidents stemming from poor judgments while intoxicated. The findings warrant continued research on risky drinking behavior, with particular interest in persistent heavy drinking at non-dependent levels.

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References

- Andreasen NC, Endicott J, Spitzer RL, Winokur G. The family history method using diagnostic criteria: Reliability and validity. *Arch. Gen. Psychiatry* 1977;10:1229–1235. [PubMed: 911222]
- Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch. Gen. Psychiatry* 1961;4:561–571. [PubMed: 13688369]
- Beirness DJ. Self-estimates of blood alcohol concentration in drinking-driving context. *Drug Alcohol Depend* 1987;19:79–90. [PubMed: 3816540]
- Cahalan, V.; Cisin, I.; Crossley, HM. American drinking practices: A national study of drinking behavior and attitudes. Rutgers Center for Alcohol Studies; New Brunswick, NJ: 1969.
- Dengerink HA, Mead JD, Bertilson HS. Individual differences in response to alcohol. *J. Stud. Alcohol* 1978;39:12–18. [PubMed: 633895]
- Doty P, Kirk JM, Cramblett MJ, de Wit H. Behavioral responses to ethanol in light and moderate social drinkers following naltrexone pretreatment. *Drug Alcohol Depend* 1997;47:109–116. [PubMed: 9298332]
- Eckardt MJ, File SE, Gessa GL, Grant KA, Guerri C, Hoffman PL, Kalant H, Koob GF, Li T-K, Tabakoff B. Effects of moderate alcohol consumption on the central nervous system. *Alcohol. Clin. Exp. Res* 1998;22:998–1040. [PubMed: 9726269]
- Ekman G, Frankenhaeuser, Goldberg, Hagdahi R, Myrsten A. Subjective and objective effects of alcohol as a function of dosage and time. *Psychopharmacology (Berlin)* 1964;6:399–409.
- Erblich J, Earlywine M. Children of alcoholics exhibit attenuated cognitive impairment during an ethanol challenge. *Alcohol. Clin. Exp. Res* 1999;23:476–482. [PubMed: 10195821]

- Evans S, Levin F. Differential response to alcohol in light and moderate female social drinkers. *Behav. Pharmacol* 2004;15:167–181. [PubMed: 15187575]
- Fillmore M, Marczinski C, Bowman A. Acute tolerance to alcohol effects on inhibitory and activational mechanisms of behavioral control. *J. Stud. Alcohol* 2005;66:663–672. [PubMed: 16331852]
- Fillmore M, Vogel-Sprott M. Behavioral effects of alcohol in novice and experienced drinkers: Alcohol expectancies and impairment. *Psychopharmacology (Berl)* 1995;127:359–364. [PubMed: 8923572]
- Fillmore M, Vogel-Sprott M. Social drinking history, behavioral tolerance and the expectation of alcohol. *Psychopharmacology (Berl)* 1996;127:359–364. [PubMed: 8923572]
- Finnigan, F.; Hammersley, R. Effects of alcohol on performance. In: Jones, DM.; Smith, AP., editors. *Handbook of Human Performance*, Vol. 2: Health and Performance. Academic Press; London: 1992. p. 73-126.
- First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. *Structured Clinical Interview for DSM-IV Axis I Disorders – Patient Edition (SCID-I/P, Version 2.0)*. Biometrics Research Department; New York, NY: 1995.
- Frezza M, di Padova C, Pozzato G, Terpin M, Baraona E, Lieber CS. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. *N. Engl. J. Med* 1990;322:95–99. [PubMed: 2248624]
- Fromme K, Katz E, D'Amico E. Effects of alcohol intoxication on the perceived consequences of risk taking. *Exp. Clin. Psychopharmacology (Berl)* 1997;5:14–23.
- Gabrielli WF, Nagoshi CT, Rhea SA, Wilson JR. Anticipated and subjective sensitivities to alcohol. *J. Stud. Alcohol* 1991;52:205–214. [PubMed: 2046370]
- Goldberg L. Quantitative studies on alcohol tolerance in man. *Acta Physiol. Scand* 1943;5(Suppl 16):1–128.
- Hindmarch I, Kerr JS, Sherwood N. The effects of alcohol and other drugs on psychomotor performance and cognitive function. *Alcohol Alcohol* 1991;26:71–79. [PubMed: 1854375]
- Holdstock L, de Wit H. Individual differences in responses to ethanol and D-amphetamine: A within-subject study. *Alcohol. Clin. Exp. Res* 2001;25:540–548. [PubMed: 11329494]
- Holdstock L, King AC, de Wit H. Subjective and objective responses to ethanol in moderate/heavy and light social drinkers. *Alcohol. Clin. Exp. Res* 2000;24:789–794. [PubMed: 10888066]
- Hollingsworth HL. The influence of alcohol. *J. Abnormal Psychol* 1923;18:204–237.
- Hollingsworth HL. The influence of alcohol (Part II). *J. Abnormal Psychol* 1924;18:311–333.
- Kalant H. Research on tolerance: What can we learn from history? *Alcohol. Clin. Exp. Res* 1998;22:67–76. [PubMed: 9514287]
- King AC, Byars J. Alcohol-induced performance impairment in heavy episodic and light social drinkers. *J. Stud. Alcohol* 2004;65:27–36. [PubMed: 15000501]
- King AC, Epstein AM. Alcohol dose-dependent increases in smoking urge in light smokers. *Alcohol. Clin. Exp. Res* 2005;29:547–552. [PubMed: 15834219]
- King AC, Houle T, de Wit H, Holdstock L, Schuster A. Biphasic alcohol effects differ in heavy versus light drinkers. *Alcohol. Clin. Exp. Res* 2002;26:827–835. [PubMed: 12068251]
- Lieber CS. Metabolic consequences of ethanol. *Endocrinologist* 1994;4:127–139.
- Mitchell M. Alcohol-induced impairment of central nervous system function: Behavioral skills involved in driving. *J. Stud. Alcohol* 1985;10(Suppl):109–116.
- National Highway Traffic Safety Administration (NHTSA), Dept of Transportation (US). *Traffic safety facts 2005: Alcohol*. NHTSA; Washington DC: 2007. http://199.79.179.188/portal/nhtsa_static_file_downloader.jsp?file=/staticfiles/DOT/NHTSA/NCSA/Content/TSF/2006/TSF2005.pdf <accessed on 15 February 2007>
- Ramchandani VA, Flury L, Morzorati S, Kareken D, Blekher T, Foroud T, Li T-K, O'Connor S. Recent drinking history: Association with family history of alcoholism and acute response to alcohol during a 60 mg% clamp. *J. Stud. Alcohol* 2002;63:734–744. [PubMed: 12529074]
- Ramchandani VA, O'Connor S, Blekher T, Kareken D, Morzorati S, Nummerger J, Li TK. A preliminary study of acute responses to clamped alcohol concentration and family history of alcoholism. *Alcohol. Clin. Exp. Res* 1999;23:1320–1330. [PubMed: 10470974]

- Substance Abuse and Mental Health Services Administration (SAMHSA). National Survey on Drug Use and Health. Office of Applied Studies; Bethesda, MD: 2005.
- Schweizer TA, Jolicœur P, Vogel-Sprott M, Dixon MJ. Fast, but error-prone, responses during acute alcohol intoxication: Effects of stimulus response mapping complexity. *Alcohol. Clin. Exp. Res* 2004;28:643–649. [PubMed: 15100617]
- Selzer ML, Vinokur A, van Rooijen LA. A self-administered Short Michigan Alcoholism Screening Test (SMAST). *J. Stud. Alcohol* 1975;36:117–126. [PubMed: 238068]
- Sobell LC, Maisto SA, Sobell MB, Cooper AM. Reliability of alcohol abusers' self-reports of drinking behavior. *Behav. Res. Ther* 1979;17:157–160. [PubMed: 426744]
- Sobell, LC.; Sobell, MB. Alcohol Timeline Follow-Back Users' Manual. Addiction Research Foundation; Toronto, Canada: 1995.
- Spielberger, CD.; Gorsuch, RL.; Luchene, RE. Test Manual for the State-Trait Anxiety Inventory. Consulting Psychologists Press; Palo Alto, CA: 1970.
- Sutker PB, Tabakoff B, Goist KC, Randall CL. Acute alcohol intoxication, mood states and alcohol metabolism in women and men. *Pharmacol. Biochem. Behav* 1983;18(Suppl):349–354. [PubMed: 6634848]
- Tiplady B, Franklin N, Scholey A. Effect of ethanol on judgments of performance. *Br. J. Psychol* 2004;95:105–118. [PubMed: 15005871]
- Wechsler, D. WAIS-R Manual: Wechsler Adult Intelligence Scale-Revised. Harcourt, Brace, & Jovanovich; New York: 1981.
- Weissenborn R, Duka T. Acute alcohol effects on cognitive function in social drinkers: Their relationship to drinking habits. *Psychopharmacology (Berl)* 2003;165:306–312. [PubMed: 12439627]
- Williams A. Alcohol-impaired driving and its consequences in the United States: The past 25 years. *J. Safety Res* 2006;37:123–138. [PubMed: 16647085]
- Zack M, Vogel-Sprott M. Behavioral tolerance and sensitization in alcohol in humans: The contributions of learning. *Exp. Clin. Psychopharmacol* 1995;3:396–401.

Figure 1.

Mean scores for Pegboard (time to complete task, top row) & DSST (number correct, bottom row) ($\pm$ SEM) for LD and HD at baseline and all post-beverage consumption time points during each of the three doses separately. Alcohol impaired performance at the high alcohol dose but not the low dose. There were no differences between the groups.

Figure 2.

Mean scores of self-rated impairment items ($\pm$ SEM) for LD and HD groups at each dose. X-axes indicate time since the completion beverage. ** $p < .001$; * $p < .01$; ^a $p = .06$; Indicate HD reported significantly less impairment than LD.

Table 1

Participant Characteristics

General	Light Drinkers (n = 55)	Heavy Drinkers (n = 77)
Age (year)	25.76 (0.463)	25.18 (0.361)*
Education (year)	16.32 (0.282)	15.72 (0.152)*
Race (% Caucasian)	43(79 %)	46(60 %)*
Sex (% female)	36(65%)	34(44%)*
Family History		
FH+	24 (44%)	32 (42%)
FH-	17 (31%)	24 (31%)
Alcohol Drinking		
Days drinking/ week	1.46 (0.111)	3.58 (0.140)**
Drinks ^a / occasion	1.98 (0.001)	6.14 (0.452)**
Drinks/ week	2.81 (0.228)	20.87 (1.36)**
Binges ^b / month	0.11 (0.019)	9.51 (0.539)**
Duration of Bingeing ^c (yrs.)	-- (--)	7.77 (0.380)
S-MAST Score	0.22 (0.077)	0.95 (0.14)**

Note. Data are Mean (SEM) or N (%).

^a Drink based on standard definition of one drink = 12 oz. beer, 5 oz. wine, or 1.5 oz. liquor.

^b Binge criteria were ≥ 5 drinks per occasion for males and ≥ 4 drinks for females.

^c Number of years of monthly or more frequent intoxication. An insufficient number of LD reported any bingeing history so means were not calculated.
S-MAST = Short Michigan Alcoholism Screening test (range 0-13; >3 indicates alcohol problem)

*
 $p = .05$

**
 $p < .01$

Table 2
BAC data for Low (0.4g/kg) and High (0.8g/kg) Dose of Alcohol

Time after drink (min)	Mean BAC (g/dl)	
Low Dose	Light Drinkers	Heavy Drinkers
15	0.035 (0.002)	0.042 (0.001)*
30	0.036 (0.002)	0.040 (0.001)*
45	0.034 (0.001)	0.037 (0.001)
105	0.019 (0.001)	0.019 (0.001)
165	0.006 (0.001)	0.004 (0.001)
180	0.003 (0.001)	0.001 (<0.001)
High Dose		
15	0.074 (0.002)	0.084 (0.002)*
30	0.082 (0.003)	0.090 (0.002)*
45	0.082 (0.002)	0.092 (0.002)*
105	0.070 (0.002)	0.074 (0.002)
165	0.053 (0.002)	0.057 (0.002)
180	0.048 (0.002)	0.049 (0.002)

Note. Data are Mean (SEM). A time point for BAC was included at 30 minutes to capture the acute changes during the rising limb. Note that performance was not assessed at that interval. Post hoc analysis of linear mixed model

*
 $p < .01$