Alcohol-Induced Performance Impairment in Heavy Episodic and Light Social Drinkers

ANDREA C. KING, PH.D., AND JOANNE A. BYARS, B.A.

Department of Psychiatry, Pritzker School of Medicine, University of Chicago, 5841 S. Maryland Avenue, Chicago, Illinois 60637

ABSTRACT. Objective: The purpose of this study was to examine performance effects of alcohol in young adult heavy drinkers (HDs) and light drinkers (LDs). Prior research has shown that HDs have alterations in subjective alcohol response in comparison with LDs, with greater reported stimulant-like effects and reduced sedative-like effects. It is unclear whether these quantitative differences extend to performance and objective effects. Method: Thirty-four subjects participated (20 HDs and 14 LDs) in three early evening individual alcohol challenge sessions. Subjects were examined on eye movement and psychomotor performance tasks before and several times after consuming either 0.8 g/kg or 0.4 g/kg ethanol or placebo beverage. Results: Alcohol produced similar impairment for the groups in psychomotor performance and saccadic velocity measures, with blood alcohol concentration dependent group differences on the smooth pursuit task and a marginally lower threshold for impairment for HDs on the saccadic latency task. Covarying for personality differences (i.e., disinhibition and boredom susceptibility traits) and family history of alcoholism did not significantly alter the findings. Conclusions: Despite prior findings of differential subjective response to alcohol in HDs and LDs, alcohol-induced performance impairment was comparable between the groups. Our findings suggest HDs may be particularly at risk for alcohol-related consequences because their greater sensitivity to positive alcohol effects and/or tolerance to sedative effects may not be accompanied by a lesser degree of alcohol-induced performance impairment. (J. Stud. Alcohol 65: 27-36, 2004)

It is well known that ethanol intoxication can impair performance in a variety of psychomotor and performance tasks. Alcohol has been shown to diminish performance on motor performance tests (Fogarty and Vogel-Sprott, 2002), eye movement responses (Blekher et al., 2002; Holdstock and de Wit, 1999; Moser et al., 1998) and short-term memory tasks (Chait and Perry, 1994; Heishman et al., 1997; Mattila et al., 1996). Studies examining objective alcohol response have focused mainly on nonalcoholics deemed at risk by virtue of biological (or paternal) family history of alcoholism (FH+). Some of these findings have indicated that FH+ subjects, as compared with family history negative subjects (FH-), experience behavioral tolerance to alcohol or less alcohol-related impairment in certain measures such as body sway (Schuckit, 1985, 1988) or electroencephalograph recordings (Begleiter and Porjesz, 1988). Other studies, however, have shown that FH+ subjects compared with FH- subjects exhibit greater or comparable performance impairments (Kaplan et al., 1988; Lex et al., 1994; McCaul et al., 1990; Nagoshi and Wilson, 1987; Vogel-Sprott and Chipperfield, 1987) or have variable performance decrements, depending on the task or method employed (i.e., eye movement latency versus velocity, body sway with eyes closed versus open, etc.) (Lipscomb et al., 1979; Ramchandani et al., 1999).

Recent studies examining persons at risk for alcohol dependence by our group have broadened the range of “at-risk” persons examined in alcohol challenge studies and examined how young adult habitual heavy drinkers compare with light drinkers (Holdstock et al., 2000; King et al., 2002). Heavy drinkers were chosen because a consistent pattern of heavy drinking during the early adult years presents a risk factor in its own right and is associated with enhanced vulnerability to alcohol use disorders and numerous future consequences of alcohol (Blane, 1979; Chou and Pickering, 1992; Hasin et al., 1990; Hilton, 1991; Zucker, 1987). In alcohol challenge paradigms, heavy drinkers have exhibited higher subjective stimulant-like responses (Holdstock et al., 2000; King et al., 2002) and lower sedative-like and cortisol responses than have light drinkers (King et al., 2002). Group differences in subjective responses were most apparent during a high alcohol dose session (i.e., 4 drink equivalent) and during the rising portion of the blood alcohol concentration (BAC) curve. A different pattern emerged for the stress hormone cortisol, with heavy drinkers showing lower cortisol response with the most apparent effects during the declining limb of the BAC. It is important to note that the groups were selected to be comparable in terms of prevalence of FH+ and statistical analyses covarying for this factor did not alter the results.

---


*This research was supported by a grant from the Alcoholic Beverage Medical Research Foundation and National Institute on Alcohol Abuse and Alcoholism grant K08-AA00276 to the first author, and by General Clinical Research Center grant M01-RR00055 to the University of Chicago.

Correspondence may be sent to Andrea C. King at the above address, or via email at: aking@yoda.bsd.uchicago.edu.
There are currently two main hypotheses proposed to explain the mechanism underlying persons vulnerable to alcohol dependence: (1) the low level of response model (i.e., tolerance) that states persons at risk are less sensitive to the effects of alcohol and therefore show behavioral tolerance to it (Schuckit, 1994; Schuckit et al., 1994), and (2) the differentiator model (Newlin and Thompson, 1990, 1999) that states at-risk individuals are more sensitive to alcohol during rising BACs and less sensitive during declining BACs. Prior studies examining objective alcohol response in FH+ persons on psychomotor tasks and body sway, neuroendocrine measures and other psychophysiological indices have supported both models: the diminished response (Begleiter and Porjesz, 1988; Schuckit, 1985, 1988; Schuckit et al., 1996) and differentiator model (Conrod et al., 2001; Finn and Pihl, 1987; Gianoulakis et al., 1996; Peterson et al., 1996; Vogel-Sprott and Chipperfield, 1987; Wilson and Nagoshi, 1988). Reasons for the variable pattern of results are unclear but may relate to differences in the type of performance measure examined, the timing when measures are taken on the BAC course and the degree of FH risk in subjects (i.e., uni- versus multi-generational). Although there is evidence for both models in terms of FH, other risk groups have not been systematically examined.

To investigate the different hypotheses in drinkers at risk on the basis of early adult drinking patterns, the present study examined a variety of performance measures sensitive to alcohol's effects in the heavy and light drinker sample from our prior article (King et al., 2002). Whereas the main objective of the first article was to investigate limb-dependent subjective indices in the drinker groups, the goal of this article is to characterize detailed objective response (psychomotor performance and eye movements) over the course of the BAC curve and as a function of alcohol dose or placebo. If heavy drinkers show a lower level of impairment in comparison with their light-drinking counterparts, then the low level response model would be supported. If, however, the heavy drinkers show greater or similar impairment to light drinkers during rising BACs, with less impairment during declining BACs, then the results would be more consistent with the tenets of the differentiator model. Psychosocial factors were also examined as possible contributing factors to alcohol response, particularly since heavier drinkers have shown a propensity for impulsive personality features (Conrod et al., 1997a; Nagoshi et al., 1991) and mood dysregulation (Cloninger, 1987; King et al., 2003).

Method

Subjects

The volunteer subjects were 34 healthy men and women between the ages of 24 and 38 who were recruited through newspaper advertisements, flyers and word of mouth referrals. Eligible candidates from a phone interview were invited to attend further medical and psychological screening in the laboratory. On the basis of interview and cut-off thresholds on standard screening questionnaires (Brief Michigan Alcohol Screening Test [Pokorny et al., 1972], quantity-frequency index [Cahalan et al., 1969] and the Symptom Checklist-90 [Derogatis, 1983]), candidates were excluded from participation if they had a history of alcohol or substance dependence, regular heavy use of drugs other than alcohol, current or past major psychiatric or medical disorders, a positive urine toxicology, abnormalities on hepatic function tests or other medical disorders as determined during screening by the resident physician’s physical examination. Female candidates were given pregnancy tests at screening and prior to each laboratory session, and all tests were negative. The University of Chicago Institutional Review Board approved the protocol, and eligible subjects read and signed the study consent form prior to participating. A more detailed description of the subject recruitment and screening methods can be found in King et al. (2002).

Subjects were included if they met the criteria for the Light Drinker (LD) or Heavy Drinker (HD) group on the basis of their reported drinking patterns. The LD group (n = 14; 10 men and 4 women) was defined by consumption of fewer than five drinks weekly (one to three drinks per occasion, one or two times weekly) with no history of regular heavy episodic alcohol drinking. The HD group (n = 20; 16 men and 4 women) included those drinkers consuming a minimum of 10 drinks per week, with consumption of five or more drinks (four drinks for women) on one occasion (heavy episodic drinking) one to four times each week. Consumption of five or more drinks (four for women) on one occasion is considered heavy episodic drinking because it departs from normative social drinking, may indicate loss of control (Dawson, 2000; Dufour, 1999) and often is linked to negative drinking-related consequences (Dawson, 1999; Single, 1996). The groups did not differ in family history of alcoholism (i.e., 31% of LDs and 50% of HDs had at least paternal or three secondary biological relatives with alcohol dependence [χ²=1.45, 1 df, p = .22]).

Procedure

The study employed a two-group (HD vs LD), within-subject design. Subjects participated individually in three experimental sessions in which they received either a low (0.4 g/kg) or high (0.8 g/kg) dose of ethanol or a placebo administered in random order and counterbalanced within group. The study was double-blind: Neither the subject nor the experimenter knew the contents of the beverage. To reduce expectancy effects, the placebo beverage contained 1% ethanol as a taste mask. Sessions were conducted in the late afternoon/evening in a comfortable living-room-like laboratory environment, with at least 48 hours separating
each session. Subjects were transported home at the end of each session.

Upon arrival at the laboratory, at approximately 4:30 PM (i.e., the -60 minute time point), the subject ate a noncaffeine, low-fat snack at 15% calories according to body weight to reduce potential nausea resulting from alcohol consumption. The subject then completed several demographic and personality questionnaires. The subject then completed physiological and performance measures at -15 minutes. At the 0 minute time point, the subject drank either a placebo beverage or 8% volume 190 proof alcohol (0.4 g/kg) or 16% volume alcohol (0.8 g/kg) mixed with water, NutraSweet (NutraSweet Co., Chicago, IL) and sugar-free grape Kool-Aid (Kraft, Northfield, IL) in the presence of the experimenter. To control for pacing effects, the alcohol drinking interval lasted 15 minutes, with half the beverage consumed in the first 5 minutes and half consumed in the last 5 minutes with a rest of 5 minutes in between. The objective measures were repeated at 15, 45, 105 and 165 minutes after the completion of the drinking interval. When subjects were not completing tasks or forms, they were allowed to read or watch videotapes.

Measures

BAC. Baseline BAC was obtained from breath samples using the Alco-Sensor III (Intoximeter Inc., St. Louis, MO), a unit that displays the BAC reading, to ensure that subjects had not consumed alcohol prior to the session. Post-alcohol consumption BACs were measured using the Alco-Sensor IV (Intoximeter Inc., St. Louis, MO), which provides improved experimenter blindness to alcohol dose. This breath analyzer reads .000 for all measures during testing, with actual BAC levels downloaded to a computer after the subject has completed the study.

Digit Symbol Substitution Test. The Digit Symbol Substitution Test (DSST) (WAIS-R; Wechsler, 1958) is a pencil and paper test that requires subjects to substitute a series of symbols for numbers within 90 seconds. Scores are based on the number of correct responses. Five equivalent forms of the DSST were used to minimize order and learning effects. The DSST is a psychomotor task incorporating motor speed, memory and learning of the digit-symbol pairs, attention and concentration and visual scanning (Kaplan, 1990). Alcohol has been shown to impair performance on the DSST (Chait and Perry, 1994; Heishman et al., 1997, 1988; Matilla et al., 1996).

Eye movement. Smooth pursuit tracking and saccadic eye movements were measured using a noninvasive oculographic device (EyeTrac Model 210, ASL Laboratories, Waltham, MA). The procedures for eye movement recording and data extraction have been described in detail in Holdstock and de Wit (1999) and are based on previously published techniques (Radant et al., 1997; Roy-Byrne et al., 1993). Eye movements were recorded using infrared sensors mounted on a glasses-like frame and positioned at the level of the subject’s lower eyelid and pointed at the pupil. The targets were white dots presented on a computer screen at a distance of 43 cm from the subject. Subjects rested their heads against forehead and chin barriers to prevent head movement.

In the smooth pursuit task, the subject tracked a target (0.5 cm white dot) that moved back and forth continuously on a black background at 20°/second, starting in the middle and moving horizontally from side to side. Artifacts (i.e., eye velocity > 40°/second) were removed by a custom-designed software package. The main dependent variable from the smooth pursuit task was the time on target, which is defined as the time the subjects focused their eyes on the target. As a result of instrumentation error and some equipment difficulties, smooth pursuit data were evaluated on 22 of 34 total subjects (10 HD, 12 LD).

In the saccade task, the subject looked at a black background on which a series of 25 targets appeared in pseudorandom order at various angles (7.5, 15, 22.5 and 30 degrees) from the center. Subjects were instructed to follow the target with their eyes. There were two main dependent variables of interest from the saccade task: average saccadic latency (time lag to make the saccade) and peak saccadic eye velocity (speed of eye movement to target). The equation for peak saccadic velocity is peak velocity = a - b e^c, where a, b and c are constants, x is saccade amplitude, and the peak velocity for a 20-degree saccade was calculated; e = exponential (base of the natural log).

Psychosocial. During presession baseline, subjects completed the Sensation Seeking Scale Form V (SSS: Zuckerman et al., 1978) and the Eysenck Personality Questionnaire (EPQ; Eysenck, 1975). The SSS is a 40-item questionnaire yielding a total sensation seeking score (total SSS) as well as four factor-derived subscales: Thrill and Adventure Seeking, Experience Seeking, Disinhibition and Boredom Susceptibility. Total SSS score was the primary dependent measure, with the subscales examined as secondary dependent measures. The SSS is a reliable, well-validated instrument of impulsive and behavioral undercontrol traits (Zuckerman 1974, 1978, 1979a,b). The EPQ is a reliable and valid 90-item true-false questionnaire measuring three personality traits: Neuroticism, Psychoticism and Extraversion (Eysenck and Eysenck, 1975).

In addition, affective and stressful events were examined by the Beck Depression Inventory (BDI; Beck et al., 1961), the Spielberger State-Trait Anxiety Inventory (STAI; Spielberger et al., 1970) and the Scaling of Life Events questionnaire (SLE; Paykel et al., 1971). The BDI is a 21-item self-report questionnaire assessing current (past 7 days) depressive symptomatology and has been validated in psychiatric (Beck et al., 1961) and normal healthy (Blumberg et al., 1987) samples. The STAI is a reliable and valid 40-
item self-report questionnaire; the main dependent measure used here was the STAI-Trait T score. The SLE is a 61-item questionnaire measuring stressful life events that have occurred in the past year. The dependent variable used was the weighted total score, which was the total number of events endorsed by the subjects multiplied by their rated (0-20) perception of stress. All three measures have been shown to be higher in alcohol dependent and problem drinker subjects than in lighter drinkers (King et al., 2003).

Statistical analyses

To evaluate the effects of ethanol on performance measures, change scores were calculated by subtracting baseline scores (i.e., preconsumption values) from scores obtained at the various postdrinking timepoints (Kenny, 1975). Repeated measures analyses of variance (ANOVA) were conducted with two within-subjects factors (Dose and Time) and one between-subjects factor (Group). For all ANOVAs, Greenhouse-Geisser adjustments were used to protect against violations of sphericity. Significant main effects or interactions were followed by simple effects tests, where appropriate. Student’s t tests compared groups on psychosocial and demographic factors. Exploratory analyses of covariance (ANCOVAs) were conducted to examine the contribution of subject background variables to predrink baseline and alcohol response variables that differed between groups. For all analyses, F and t values were considered significant at p < .05.

Results

Demographic and psychosocial measures

Table 1 displays general demographics and psychosocial measures for the groups. As reported in our earlier article, the groups were similar in the majority of demographic factors (age, race and gender composition), except for HDs’ lower education levels (p < .05). The groups were also similar on EPQ personality factors and affective and stress-related measures (see Table 1). As expected, the HDs were significantly higher than LDs in typical alcohol quantity and frequency, as well as estimated number of episodes of heavy drinking over the past 6 months.

The only psychosocial factor that differed between the groups was the SSS. The total SSS score was significantly higher in HDs compared with LDs (t = 3.89, df = 32, p < .001), and between-group analyses of the subscales revealed that the HDs were higher than LDs on Disinhibition (t = 4.99, df = 32, p < .001) and Boredom Susceptibility (t = 2.35, df = 32, p = .05) but not on the other subscales. Since the Disinhibition subscale contains items directly related to heavy alcohol drinking (i.e., 3 out of the 10 items), a secondary analysis examined the groups on a modified Disinhibition subscale without those items, and the HDs remained significantly elevated compared with LDs (t = 3.08, df = 32, p < .01).

Baseline performance

Baseline measures on the performance tasks revealed no group differences on average predrinking baseline for the DSST and the saccadic velocity tasks. However, predrinking group differences were noted for smooth pursuit and saccadic latency (see Table 1), with HDs showing less time on target for smooth pursuit tracking and shorter latency to initiate saccades than LDs showed.

BACs

Analysis of BAC levels for the two groups has been reported previously (King et al., 2002). In brief, BAC significantly increased over time with a curvilinear pattern and also increased as a function of increasing alcohol dose (Dose × Time: F = 158.45, 8/232 df, p < .0001). The groups did not differ in BAC levels. The BACs peaked in the range of .03-.04 mg% (low dose) and .07-.08 mg% (high dose), respectively.

Table 1. Demographic and background variables by group

<table>
<thead>
<tr>
<th>Demographics and drinking behaviora</th>
<th>Light drinkers</th>
<th>Heavy drinkers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>28.7 (0.7)</td>
<td>28.5 (0.8)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>17.2 (0.6)</td>
<td>15.2 (0.3)*</td>
</tr>
<tr>
<td>Race: Black/white</td>
<td>5/8</td>
<td>5/15</td>
</tr>
<tr>
<td>Gender: Male/female</td>
<td>10/4</td>
<td>16/4</td>
</tr>
<tr>
<td>Alcohol drinks/occasion</td>
<td>1.9 (0.2)</td>
<td>4.8 (0.4)*</td>
</tr>
<tr>
<td>Drinking occasions/week</td>
<td>1.2 (0.11)</td>
<td>3.4 (0.2)*</td>
</tr>
<tr>
<td>No. episodes of heavy drinking in last 6 months†</td>
<td>0.5 (0.2)</td>
<td>45.0 (6.1)*</td>
</tr>
<tr>
<td>Sensation Seeking Scale (SSS)</td>
<td>21.1 (1.3)</td>
<td>26.9 (0.8)*</td>
</tr>
<tr>
<td>Total SSS</td>
<td>4.5 (0.5)</td>
<td>7.6 (0.3)*</td>
</tr>
<tr>
<td>Disinhibition</td>
<td>2.7 (0.5)</td>
<td>4.4 (0.5)*</td>
</tr>
<tr>
<td>Boredom Susceptibility</td>
<td>7.6 (0.6)</td>
<td>7.8 (0.4)</td>
</tr>
<tr>
<td>Thrill and Adventure Seeking</td>
<td>6.3 (0.6)</td>
<td>7.2 (0.4)</td>
</tr>
<tr>
<td>Experience Seeking</td>
<td>9.8 (1.4)</td>
<td>10.0 (1.3)</td>
</tr>
<tr>
<td>Eysenck Personality Questionnaire (EPQ)</td>
<td>3.7 (0.5)</td>
<td>4.0 (0.7)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>14.4 (1.1)</td>
<td>16.4 (0.8)</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>4.9 (1.0)</td>
<td>3.6 (0.9)</td>
</tr>
<tr>
<td>Affective and stress-related measures</td>
<td>47.7 (2.2)</td>
<td>47.9 (1.8)</td>
</tr>
<tr>
<td>Beck Depression Inventory</td>
<td>59.4 (8.5)</td>
<td>70.9 (15.6)</td>
</tr>
<tr>
<td>Spielberger Trait Anxiety T score</td>
<td>75.0 (3.1)</td>
<td>70.0 (2.7)</td>
</tr>
<tr>
<td>Smooth pursuit (time on target)</td>
<td>17.7 (0.7)</td>
<td>15.2 (1.0)*</td>
</tr>
<tr>
<td>Saccadic latency (ms)</td>
<td>197.0 (8.5)</td>
<td>179.6 (3.4)*</td>
</tr>
<tr>
<td>Saccadic velocity (°/sec)</td>
<td>315.7 (12.0)</td>
<td>330.7 (10.0)</td>
</tr>
</tbody>
</table>

Notes: Data presented are mean (SEM). aDrinking behavior derived from 6-month QFI. bEpisodes of heavy drinking defined as 5+ drinks/occasion (men), 4+ drinks (women).

*p < .05; †p < .001; ‡p < .0001.
Smooth pursuit eye movement

For smooth pursuit, there was a significant three-way interaction (Group × Dose × Time: $F = 2.25, 6/120 \text{ df, } p < .05$; Figure 1), with both HDs and LDs exhibiting significant alcohol-induced impairment (i.e., less time on target) during the early declining BAC phase (105 minutes; simple effects, $p < .05$) but differential effects during the rising phase. Specifically, the HDs were impaired during the early, initial rising phase of the BAC (15 minutes, simple effects, $p = .02$), but the reverse was noted during the latter portion of the rising phase (i.e., LDs impaired at 45 minutes; simple effects, $p = .001$). All effects were notable for the high alcohol dose but not the low dose.

Saccadic latency

For saccadic latency, in contrast to smooth pursuit, there was a trend for a significant two-way interaction (Group × Dose: $F = 2.71, 2/62 \text{ df, } p = .07$ [Figure 2]), with HDs exhibiting more sensitivity to impairment. As seen in Figure 2, for HDs saccadic latency increases were apparent at both the low and high alcohol doses, but the LDs were affected only at the high dose. The HDs therefore showed a tendency, although nonsignificant, for a lower threshold for the slowing effects of alcohol on the latency of saccades, but this was not BAC-limb dependent.

Saccadic velocity and DSST

Alcohol produced comparable impairment between the groups for the DSST and saccadic velocity measures (see Figures 3 and 4). As indexed by the DSST task, psychomotor performance was impaired by alcohol (Dose × Time: $F = 2.25, 6/186 \text{ df, } p < .05$), with the high dose producing impairments during the rising limb (simple effects at 15 minutes, high > low > placebo, $p < .003$; 45 minutes, high > low = placebo, $p < .0001$) and the low dose impairing

---

**Figure 1.** Mean smooth pursuit tracking change scores in the LD compared with HD group for the placebo (open circles), low alcohol dose (0.4 g/kg; filled triangles) and high alcohol dose (0.8 g/kg; filled squares) sessions. X-axis indicates time intervals after the completion of beverage consumption. Main effect of dose (simple effects: high > low = placebo, $p < .01$) and Group × Dose × Time interaction (simple effects, LD > HD [more impairment] at 45 min; HD > LD at 15 min, $p's < .05$).

**Figure 2.** Mean saccadic latency change scores (ms) for the HD and LD groups for the placebo (open circles), low alcohol dose (0.4 g/kg; filled triangles) and high alcohol dose (0.8 g/kg; filled squares) sessions. X-axis indicates time intervals after the completion of beverage consumption. Main effect of dose (high > low > placebo, $p < .01$) and Group × Dose trend ($p = .07$).
performance only at the initial rising limb time point (15 minutes, simple effects low > placebo, p < .005). The velocity of saccades was also decreased by the high dose of alcohol (dose: F = 10.97, 2/62 df, p < .0001; simple effects, high > low = placebo, p < .0005) but was not limb-dependent.

**Relationship of subject-related factors to performance**

Given the elevations in HDs' disinhibition and boredom susceptibility, ANCOVAs were conducted to examine the influence of these personality factors on performance. The results revealed the group differences in baseline performance were no longer apparent after covarying for disinhibition (latency: $F = 0.44, 1/30$ df, $p = .51$; smooth pursuit: $F = 2.46, 1/18$ df, $p = .13$) but not boredom susceptibility (latency: $F = 4.31, 1/30$ df, $p < .05$; smooth pursuit: $F = 4.91, 1/18$ df, $p < .05$). Further exploratory analyses examining the influence of these traits on alcohol response factors showed that covarying for disinhibition or boredom susceptibility did not significantly alter previous group findings for smooth pursuit or saccadic latency. Finally, given the extensive literature on FH effects on alcohol response, post hoc analyses were also conducted using FH as a covariate and as a grouping variable (FH+ vs FH-). Although ANCOVA showed that FH did not alter the significant effects of drinking group on performance, FH as a grouping variable showed a three-way marginal interaction effect for saccadic latency ($F = 1.94, 6/156$ df, $p < .08$), with FH+ subjects showing a tendency for more sensitivity to alcohol-induced impairment at the low dose (i.e., similar to the results found with HDs).

**Discussion**

The present study demonstrated generally comparable levels of alcohol-induced performance impairment in the
HD and LD groups, although a few exceptions were noted. Both groups showed similar alcohol-induced slowing of speed in psychomotor processing and encoding (DSST task) and saccadic eye movement (i.e., velocity). Impairment of smooth pursuit eye movement in HDs was observed during the early, initial phase of drinking with evidence for rapid development of acute tolerance in HDs compared with LDs. In contrast to the saccade velocity and DSST measures, smooth pursuit is a relatively repetitive and predictable task, with some evidence for an accumulation of steady-state sequenced learning (Barnes and Schmid, 2002; Chakraborti et al., 2002).

It may be speculated that HDs have greater alcohol-related behavioral tolerance to tasks of a stereotyped, repetitive nature than to those requiring the processing and execution of new strategies, higher cortical input or shifting of set. The latter task processes are ostensibly more reflective of the DSST and saccadic eye tasks than the smooth pursuit task because of their sudden onset stimuli and multiple forms (Reingold and Stame, 2002). It is interesting to note that the HD group did not show tolerance to alcohol’s impairing effects on these tasks.

Alternatively, the behavioral tolerance in HDs on the smooth pursuit eye task, in the absence of impairment differences on the saccade task, may lie in differential sensitivity to the proposed separate fixation systems for these two types of eye movement (Kimmig et al., 2002). Ethanol is theorized to impair saccadic eye movements by increasing subjects’ “disengage” time (i.e., disengage attention from the fixation point and focus on the target) (Wegner and Fahle, 1999), but this disengagement process is not a component of the smooth pursuit task. It is therefore possible that the HDs show sensitivity comparable to LDs in alcohol-induced impairment of saccadic fixation system but not to disruption of the smooth pursuit system.

For latency of saccadic eye movements, the HDs showed more sensitivity to alcohol’s impairing effects during the low alcohol dose, but this was not limb-dependent. In terms of the theoretical models tested in this study, mixed support was found for both positions. Diminished impairment to alcohol was evident in the at-risk group (i.e., HDs) for smooth pursuit, but only after the initial acute rising BAC phase. Contrary evidence for the lower level of response model was found for two tasks, with comparable impairment for the groups on saccadic velocity and DSST. Finally, more sensitivity to alcohol-induced disruption was found in the high-risk drinkers on saccadic latency, providing some support for the differentiator model. However, the effects were observed at the lower threshold dose of alcohol during both limbs of the BAC and were not specific to the rising phase, which is suggested to be the key sensitization phase according to the differentiator model.

Our earlier article on these subjects showed that the HDs had increased subjective stimulation and reduced sedation from alcohol compared with the LDs (King et al., 2002). Similar findings (i.e., differential subjective report but similar performance impairments) have been found in other studies examining social drinkers with higher quantity-frequency scores (Schuckit and Klein, 1991) or with greater subjective alcohol stimulation (Holdstock and de Wit, 1999) compared with their respective control groups. It has been posited that the dissociation between subjective and objective responses may reflect differential neural mechanisms for these effects (Holdstock and de Wit, 1999). Objective indicators, such as eye movement responses, are reliable measures of drug effects that are unlikely to be affected by expectation or measurement “noise” inherent in self-report measures (Leigh and Zee, 1991). Furthermore, they are particularly sensitive to alcohol as a result of alcohol’s specific action on the oculomotor control system and its subcomponents (Wegner and Fahle, 1999) rather than as a function of overall sedation (Blekher et al., 2002; Baloh et al., 1979; Jantti et al., 1983; Moser et al., 1998). Continued research on high-risk drinkers, who may show tolerance to subjective alcohol sedation but exhibit comparable performance decrements to those of light drinkers, may help to further elucidate these complex mechanisms.

Alcohol-induced subjective stimulation (King et al., 2002) during the early rising phase of the BAC does not seem to relate to system-wide stimulation or performance enhancement as indexed by the psychomotor and eye movement tasks measured in the present study. Prior research, however, indicates that other stimulatory drugs, such as amphetamine, produce increases in subjective stimulation but do not impair psychomotor (Holdstock and de Wit, 2001) or saccadic eye movement performance (Tedeschi et al., 1983). Unlike amphetamine, which affects dopamine receptors throughout the brain, alcohol’s dopaminergic effects appear to be localized in the reward-mediating mesolimbic region (Gianoulakis, 2001; Robinson and Berridge, 2001). Dopamine and opioid receptor blockers have been shown to dampen alcohol-induced subjective stimulation (Enggasser and de Wit, 2001; King et al., 1997; Swift et al., 1994); however, neither class of medication has been shown to dampen alcohol-induced performance impairment (Mattila et al., 1996; Swift et al., 1994). This finding suggests further evidence for independent mechanisms for alcohol reinforcement and performance indices. However, objective measures other than performance measures, such as heart rate or cardiovascular response, may be sensitive objective indicators of the stimulant properties of alcohol (Conrod et al., 1997b).

Persons with significant major psychiatric comorbidity were not included in the present study, which may limit generalizations to the greater population of heavy episodic drinkers. On the other hand, this omission may strengthen the findings by ruling out group differences attributed to an overall spectrum of psychiatric problems. The majority of
background measures were similar between the groups, except for significantly greater sensation-seeking traits in the HD group. Increased disinhibited personality in the HDs was not specific to the need to disinhibit behavior through alcohol drinking because a post hoc analysis removing the items referring to alcohol showed that the modified scale remained elevated in HDs in comparison with LDs. Prior research (Conrod et al., 1997a) has indicated a possible role of disinhibited personality and alcohol-induced cardiovascular acceleration as independent correlates of drinking behavior. In the present study, disinhibited personality was also associated with baseline performance differences: HDs' elevated disinhibition accounted for their predrink baseline shorter latency for eye movement responses and less accurate smooth pursuit tracking. However, the baseline differences observed in the present study are in contrast to data from alcoholic samples (Bauer et al., 1993). Therefore, future research is needed to discern the precise interrelationships between risk for alcoholism, disinhibited personality traits and performance (Conrod et al., 1997a; de Wit et al., 1987; Nagoshi et al., 1991; Sher, 1993).

This study had several limitations worth noting. First, the sample size was relatively small, which precluded further investigation of mediators of the different performance measures. Second, the subjects were mostly male, and given the small number of women in the study, specific conclusions regarding possible gender effects could not be discerned. Third, performance tasks were conducted using standard methods, but modifications of these procedures in future studies may be useful to differentiate more specific alcohol effects and corresponding neurological pathways (Kaplan, 1990). For example, adding a measure to record error rates on the saccadic eye movement task and/or employing refinements or process measures on the DSST might aid in our understanding of the locus of alcohol's performance decrements.

The present study showed that the performance effects of alcohol in at-risk groups for alcohol use disorders, beyond family history, is complex and specific to the task and mechanisms employed in each task. While there were data consistent with both the diminished response and differentiator models, there were no overwhelming data to support either theory. Given the public health concerns about alcohol-induced psychomotor decrements, the findings presented suggest the assumed lack of performance impairment or tolerance effects in regular heavy alcohol consumers is not valid (see Streufert et al., 1992). Moreover, heavy episodic drinkers, who may show a propensity for alcohol-induced subjective stimulation, could be at greater risk for accidents resulting from drinking, not only as a result of habitual alcohol misuse but also as a consequence of sensitivity to mood-enhancing effects of ethanol with similar relative motor and eye movement performance decrements as in lighter drinkers.

Acknowledgments

The authors wish to thank Dr. Ted Karrson and Dr. Dingcai Cao, Department of Health Studies, for their guidance on statistical analyses. They would also like to acknowledge Alyson Schuster for her work in conducting the experimental sessions, and Louis Holdstock, Ph.D., and Harriet de Wit, Ph.D., for their assistance and expertise with the eye scanner and other laboratory measures. Appreciation is also extended to the staff at the Clinical Research Center for obtaining baseline tests during screening and for providing calorie-controlled snacks.

References

Conrod P.J., Peterson, J.B., Phih, R.O. and Markowski, S. Biphasic effects of alcohol on heart rate are influenced by alcoholic family history and rate of alcohol ingestion. Amsm Clin. Exp. Res. 21: 140-149, 1997b.


