Drunker than you think: Delayed performance impairment from moderate amounts of alcohol

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Abstract

The goal of the research was to evaluate the effects of alcohol on the performance of New Zealand drivers across .05 and .08 BAC levels. An experimental test was conducted with 61 participants assigned to one of two alcohol dose conditions or a placebo control group. Comparison of alcohol doses showed that a BAC of .08 produced a level of impairment significantly worse than the placebo control. Impairment included edge and centre line crossings in the driving simulator, disinhibition of reactions to vehicles at intersections, higher peak speeds, and errors learning and recalling a computer-based maze. Drivers with a BAC of .05 also displayed some performance decrements, but not to the same degree as a BAC of .08, and with the exception of their steering reactions to hazards, not significantly different to the placebo control. An analysis comparing the impairment associated with peak and post-peak intoxication revealed that response disinhibition and participants self-ratings of intoxication showed acute tolerance (higher levels during ascending and peak BAC levels) while maze learning and recall errors, edge and centre line crossings, and maximum speeds showed acute protracted errors (poorer performance during the post-peak phase). This asymmetry is found even when equivalent BACs levels are compared during ascending and post-peak phases and occurred for both alcohol groups. Finally, participants were not able to accurately judge how much alcohol they had consumed or their level of intoxication (particularly the high dose group), and subjective ratings of intoxication were not a reliable indicator of their performance impairment.

Introduction

It is widely recognised that alcohol has an adverse effect on driving performance. Increasing blood alcohol concentrations (BACs) are associated with increased crash risk (Blomberg et al 2009) and a higher likelihood of involvement in a serious injury or fatal crash (Maycock 1997; Phillips and Brewer 2011). The current legal BAC limits for drivers in different countries, however, vary widely from zero in some countries (e.g., Czech Republic, Russia and Romania) to .08 mg/ml in other countries, including New Zealand, US, and the UK. The majority of countries, however, currently have a limit of .05 (e.g., Australia). Part of the reason for this range of legal BAC limits is the variety of findings surrounding the amount of impairment produced by alcohol, which in turn can influence policy and public support for the limits.

Driving is a complex task which is dependent on a variety of specific functions some of which are affected by alcohol to a greater degree than others. For example, complex tasks such as divided attention show decrements in performance at lower BACs (<.01), whereas performance on reaction time tasks only become consistently impaired at BACs higher than .10 mg/ml (Moskowitz and Fiorentina 2000). In simulated driving tasks, BACs as low as .02 and .05 have been reported to result in a significant increase in speed (Lenné et al 2010; Veldstra et al 2012) however others suggest that only higher BACs (.08 and .11) produce significant alterations in speed (variability rather than speed per se: Mets et al 2011). Alcohol is also reported to lead to a dose-dependent increase in weaving (as measured by standard deviation of lane position, SDLP), from BACs of .02 to .011 (Lenné et al 2010; Mets et al 2011; Veldstra et al 2012). Similarly, drivers' reaction times to detect and respond to hazards has been reported to be impaired at BACs of .05 (West et al. 1993), although other studies failed to detect any impairment at BACs between .03 and .08 on complex

driving tasks such as reacting to a car pulling out, running red lights or frequency of crashes (Veldstra et al 2012). In general, higher-order attention and information processing abilities are impaired at lower BAC levels (.05 mg/ml) than simple motor coordination elements of driving which may not show outward signs of impairment until .08 mg/ml. It has been suggested that this difference may mislead drivers into thinking that because they are still capable of steering that they are safe to drive (Liu and Fu 2007).

Further, some cognitive and behavioural components of driving recover more quickly than others, and drivers are often unaware of their diminished capacity (Cromer et al 2010). As early as 1919, researchers noted that motor skills were impaired at a lower BAC level when BACs were rising compared to when they were descending (Mellanby 1919, cited in Schweizer and Vogel-Sprott 2008), a phenomena referred to as '*acute tolerance'*. A recent review (Schweizer and Vogel-Sprott 2008) found that speed of cognitive performance (on tasks of inhibition, information processing and selective attention) was better on the descending limb compared to at the same BAC on the ascending limb, i.e., acute tolerance. Errors, however, failed to diminish during descending BACs and in some case even rose, a phenomena termed '*acute protracted error'* (Schweizer and Vogel-Sprott 2008). These phenomena have safety implications in terms of driving: The rapid recovery of some behavioural functions when BAC remains high (acute tolerance) may lead drivers to think they are safe to drive even when they still are over the legal limit. In contrast, the acute protracted error effect suggests that some cognitive functions remain impaired even when BACs are below the legal limit.

Studies examining differential rates of recovery from alcohol have found that performance on visuomotor tasks recovered from the effects of alcohol more rapidly than executive function abilities, and that drivers' perceptions of their level of intoxication appeared to be based on only their visuomotor abilities (Cromer et al., 2010). Similarly, Weafer and Fillmore (2012) found that motor coordination, subjective intoxication and willingness to drive showed acute tolerance whereas driving performance and inhibitory control showed slower recovery. In other words, subjective estimates of one's own level of intoxication appear susceptible to acute tolerance effects. Self-evaluation of alcohol intoxication and impairment is generally poor and typically underestimated, and is most inaccurate during declining BACs and thus drivers may decide to drive even though crucial aspects of their driving and cognitive performance are still impaired (Beirness 1987; Harrison and Fillmore 2005; Weafer and Fillmore 2012).

The goals of this research were to determine the driving and cognitive impairment associated with BAC levels of .05 and .08 mg/ml and to investigate any performance asymmetries (acute tolerance and acute protracted error) accompanying the onset and recovery from intoxication. The research also aimed to examine drivers' perceptions of their own intoxication and fitness to drive.

Method

Participants

Ethical approval was received from the School of Psychology Research Ethics Committee at the University of Waikato. Participants were recruited via word of mouth, advertisements on notice-boards, and online. Participants were eligible to take part if they were aged between 20-50 years, held a full New Zealand drivers licence, were in good health and drank occasionally. Volunteers were screened to ensure that they were in good health, they had no neurological /psychological conditions (e.g., head injury, stroke), were not taking any contra-indicated medication, and for female participants no possibility of being pregnant, and they consumed alcohol occasionally but not excessively, with a score <8 on the Alcohol Use Disorders Identification Test (AUDIT) (Babor et al 2001).

The 61 participants (33 male, 28 female) recruited for the experiment had an average age of 31.11 years (*SD* = 8.34, range 20-50 years). The majority (*n* = 45) of the participants were of New Zealand European descent, 7 self-identified as Māori and 9 of were of 'other' ethnicity (including British, Dutch, Chinese, and Indian). The participants held full New Zealand driving licences for an average of 14.03 years (*SD* = 8.19).

Apparatus and materials

Cognitive performance tasks

Three tasks from the Cogstate Research software (Chase task, Groton Maze task and Card Identification) were administered on an Acer Iconia (W510) touch screen tablet computer to assess the participants' cognitive performance. The Chase task was used to assess visual motor function, the Groton Maze learning task assessed executive functions and spatial problem solving while the delayed maze recall task provided a measure of visual learning and memory. The Card Identification (a choice reaction time task), was used to provide a measure of visual attention and vigilance.

Simulated driving task

The participants' driving performance was assessed using the University of Waikato driving simulator consisting of a complete automobile (BMW 314i) positioned in front of three angled projection surfaces. The details of the simulator have been described fully elsewhere (Charlton & Starkey, 2011). The participants' steering, acceleration, braking, and responses to cars at intersections were recorded continuously throughout each simulated drive. The simulation scenarios were based on an 11 km-long section of rural road containing 20 intersections with vehicles waiting. At six of the intersections, as the driver reached a point 80 m from the intersection, a vehicle waiting on the left moved 2.6 m forward [at 1 m per sec] to partially obstruct the driver's lane (see Figure 1). There were also four "false alarm" vehicles that moved 0.8 m in 1 sec when the driver was 70 m away, to a point past the marked limit line, but not obstructing the driver's lane (shown in the lower panel of Figure 1). The waiting vehicles were included in the simulated drive to provide a go no-go measure of inhibitory control. Five unique combinations of intersections, vehicles and backgrounds were presented in a counterbalanced order across participants.

Figure 1. Scenes from the simulation. A car waiting at an intersection (top left) and pulling out 2.6 m as the participant approaches (top right). The lower panel shows a false alarm car waiting (bottom left) and moving 0.8m ahead as the participant approaches (bottom right).

A 12.5 km practice road was also developed to allow the participants to practise controlling the simulated car. The participants were coached in how to respond to the vehicles at the intersections, sounding the horn and braking only for vehicles moving into their lane and making no response to the false alarm vehicles. Participants were asked to perform the simulated driving task just as they would drive a real car; following all posted speed limits until they arrived at a stop sign at the end. The participants were also informed that at some of the intersections during the drive they might encounter a car pulling out in front of them. The participants were instructed that if that occurred, they were to first signal that they detected the car by moving the headlight control stalk on the right side of the steering column towards them, as if they were flashing their headlights (which also produced a horn sound), and then brake and steer to avoid the car.

Subjective rating scales & gross motor function and co-ordination

Subjective intoxication was assessed using a visual analogue scale (Cromer et al 2010). Participants were asked to respond to the question "How intoxicated do you feel right now?" by placing a mark on a 200mm line. Response anchors ranged from "Least intoxicated I've ever felt in my life" (at 0mm) to "Most intoxicated I've ever felt in my life" (at 200mm). The participant's momentary willingness to drive was assessed by responding on a 100mm visual analogue scale ranging from "Not at all" (0mm) to "Very much" (100mm) (Beirness 1987). The Karolinska Sleepiness Scale (Akerstedt and Gillberg 1990) was used to ask participants to rate how sleepy they feel on a 9 point scale ranging from 1 (very alert), 5 (not sleepy or alert) to 9 (very sleepy). The Walk and Turn portion of the New Zealand Police Compulsory Impairment Test was used to assess gross motor function and co-ordination. The task assessed the participant's ability to walk nine heel-to-toe steps along a marked line on the floor while the following errors were marked by an observer: inability to maintain balance at start, stepping off line, not touching heel-to-toe, raised arms (for balance), incorrect turn, stopping, and wrong number of steps.

The measures comprising each test block are summarised in Table 1 with each block taking approximately 20 minutes to complete (Test Block 1 did not include measurements of BAC, walk and turn, or subjective ratings).

Table 1. A summary of the tests included in each block

Test	Purpose	Duration (minutes)
Chase Task	Visuo-motor function	1.0
Groton Maze Learning Task	Visuo-motor function, executive function, spatial problem solving, visual learning, memory	4.5
Identification Task	Visual attention, vigilance	1.0
Groton Maze Recall Task	Visuo-motor function, executive function, visual learning, memory	1.5
Breathalyser 1	Measurement of BAC	0.5
Driving Task	Driving performance including lane position, hazard reaction time, inhibitory control, and speed	8.0
Breathalyser 2	Measurement of BAC	0.5
Walk and Turn	Gross motor control and co-ordination	2.0
Subjective ratings	Perceived intoxication, fitness to drive and feelings of sleepiness	1.0

Procedure

Potential participants contacted the researchers to receive more information about the study and then completed an eligibility screening form. All volunteers provided informed consent before participating in the study. Participants completed a 40 min familiarisation session at the laboratory consisting of: completion of informed consent, sample of BAC via breathalyser, weighing,

completion of a short demographic questionnaire and several short questions about drinking and driving (e.g., current drink drive limit; the number of drinks a person could consume and still drive safely; the number of drinks to be under the legal limit). This was followed by a practice trial of the Cogstate tests, and a practice drive in the simulator. After this a full experimental session was scheduled and they were given a $10 gift voucher to thank them for their participation thus far. Participants were asked to refrain from drinking alcohol the evening before the full session and to consume no caffeine or food in the three hours before the session. Breath alcohol levels were measured using an Alcomate AccuCell AL9000 professional grade breathalyser that automatically converted breath alcohol readings into blood alcohol equivalents (AK Solutions 2012).

Participants were randomly assigned to one of three dosage groups: Placebo, Medium (a goal of .05 mg/ml BAC), or High (a goal of .08 mg/ml BAC). Three drinks were prepared for each participant based on the following dosages and the participant's body weight: women received doses of .6g/kg or .75g/kg (for the Medium / .05 and High /.08 groups respectively); men received .75g/kg or 1.0g/kg (for the Medium / .05 and High /.08 groups respectively). The alcohol (vodka, 37.5%) was mixed with orange juice at a ratio of 30% vodka: 70% orange juice, which was then divided into three equally sized drinks. Participants in the Placebo group received an equal drink volume as the other participants, but their drinks consisted of orange juice with 5 ml of vodka added to the top.

All participants began the full session with confirmation they had a BAC of zero, a reminder of the test protocol, and another practice drive. This was followed by Test Block 1 during which the participants completed the Cogstate test battery (Chase, Groton Maze, and Card Identification tasks) followed by the simulated drive. Immediately after completing the simulated drive the participants were served their first drink and given 5 minutes to consume it. At the end of the 5 minutes, a second drink was served, and participants were again given 5 minutes to finish the drink. Test Block 2 began 5 minutes after the second drink was consumed and from Test Block 2 onwards, each block consisted of: Cogstate test battery, pre-drive BAC measurement, simulated drive, post-drive BAC measurement, walk and turn task, subjective ratings of intoxication, sleepiness, and willingness to drive. Following completion of Test Block 2 the participants were served their third drink, unless their BAC measured during Block 2 was within 0.01 of the desired BAC (>.04 or >.07 depending on their dosage group), in which case the third drink was omitted. The timing of the test blocks were as follows: Block 2 – 15 mins after first drink was served; Optional 3rd drink – 35 mins after first drink was served; Block 3 – 45 mins after first drink was served; Block 4 –75 mins after first drink was served. Block 5 (150 mins after the first drink was served) occurred after a 55 min rest break, during the second half of which the participants were allowed a variety of snack foods. At the end of Test Block 5, participants were asked how many standard drinks they thought they had consumed during the session, they were then informed how much alcohol they had consumed (but not their BAC), thanked and given a $50 gift voucher for their participation, and provided a taxi ride home. Each full session took approximately 3.5 hours to complete.

Results

BAC and alcohol consumption

The mean BAC levels associated with each alcohol dose group across the five test blocks are shown in Figure 2. The dosage groups displayed considerably different BAC levels, peaking at Test Block 3. A series of univariate ANOVAs revealed that the BACs of the three dose groups were significantly different at Test Block 2 [$F(2,58) = 108.52$, $p < .001$, $\eta_p^2 = .789$], Block 3 [$F(2,58) = 242.55$, $p < .001$, $\eta_p^2 = .893$], Block 4[$F(2,58) = 166.86$, $p < .001$, $\eta_p^2 = .852$], and Block 5 [$F(2,58) = 93.43$, $p < .001$, $\eta_p^2 = .766$]. For each test block, post hoc pairwise comparisons (using the Bonferroni adjustment) indicated that the three alcohol dose groups were significantly different from one another (all $p's < .01$).

Participants' estimates of the number of standard drinks they thought they had consumed differed significantly across the three dose groups [$F(2, 55) = 19.86$, $p < .001$, $\eta_p^2 = .419$]. The Placebo group's estimates ($M = 1.42$, $SD = .892$) were higher than their actual level of alcohol consumption (.5 of a standard drink). Their estimates, however, were significantly lower ($p < .001$) than the Medium group's estimates ($M = 3.83$, $SD = 1.46$) who underestimated the amount of alcohol they consumed (actual drinks consumed $M = 4.78$, $SD = 1.43$). The estimates from the High dose group ($M = 4.08$, $SD = 1.88$) were significantly higher than the Placebo group's estimates ($p < .001$), and much lower than the actual number of standard drinks they consumed ($M = 7.01$, $SD = 2.86$). There was no significant difference between the Medium and High group's estimates.

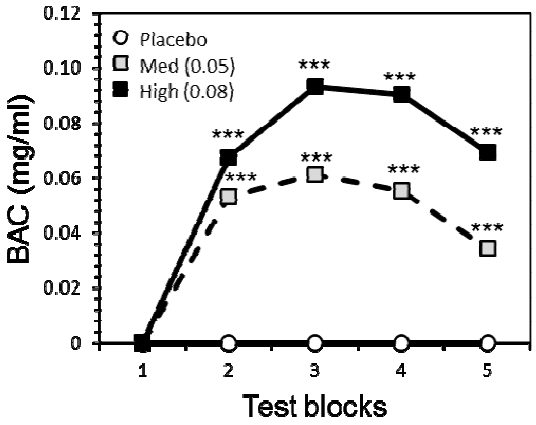

Figure 2. BAC (mg/ml) levels across the five test blocks for each alcohol dose group. Data are presented as the mean for each group (Placebo, Medium and High alcohol dose) for each test block. *** p < .001 compared to Placebo group.

Analysis by BAC level

An initial analysis of the differences between the three alcohol dose groups (Placebo, Medium and High) revealed reliable differences in task performance, however not every participant was at their target BAC level, some were over and some were under. To more clearly assess the differences between 0.05 and 0.08 BAC levels, the participants were grouped according to their observed BAC levels (rather than alcohol dose). Participants were assigned to one of three BAC groups: Placebo, Medium (BAC of .04 to .06), or High (BAC of .07 to .09) based on their BAC level during Test Blocks 2 or 3. A total of 51 participants were assigned to one of the three BAC groups (10 participants whose BAC levels fell outside the desired ranges were excluded). Sixteen participants were classified as being in the Medium BAC range (Mean BAC = .053, $SD = .006$), and 15 were classified as being in the High BAC range (Mean BAC =.078, $SD = .006$).

The participants' performance on the cognitive tasks varied as a function of their BAC. Generally the Placebo group performed the tasks faster and with fewer errors than either the Medium or High BAC group. Multivariate analysis of the cognitive performance tasks' primary outcome variables indicated a statistically significant difference between the three BAC groups, [Wilks' lambda = .640, $F(8, 90) = 2.81$, $p = .008$, $\eta_p^2 = .200$]. The univariate analyses showed significant differences between the groups for maze learning errors [$F(2,48) = 6.76$, $p = .003$, $\eta_p^2 = .220$], maze recall errors [$F(2,48) = 9.44$, $p = <001$, $\eta_p^2 = .282$], and speed of response in the card identification task [$F(2,48) = 3.17$, $p = .051$, $\eta_p^2 = .117$], but not for the chase task. The Placebo group made significantly fewer errors than the High BAC group on the learning and recall tasks ($p < .01$), and the Medium BAC group made significantly fewer recall errors compared to the High group ($p < .05$).

Figure 3 shows the effects of BAC for participants' performance on the simulated driving task and the number of false alarms to vehicles at intersections. The multivariate ANOVA was statistically

significant [Wilks' lambda = .640, $F(8, 90)= 2.81$, $p = .008$, $\eta_p^2 = .200$]. There were significant BAC differences for the number of centre line crossings [$F(2, 48) = 10.62$, $p < .001$, $\eta_p^2 = .307$], seconds spent over the centre line [$F(2, 48) = 9.16$, $p < .001$, $\eta_p^2 = .276$], the number of edge line crossings [$F(2, 48) = 4.15$, $p < .022$, $\eta_p^2 = .148$], seconds spent over the edge line [$F(2, 48) = 3.62$, $p < .034$, $\eta_p^2 = .131$], and maximum speed [$F(2, 48) = 4.21$, $p < .021$, $\eta_p^2 = .149$]. The average number of false alarms also showed a significant difference across the groups [$F(2, 53) = 4.26$, $p = .019$, $\eta_p^2 = .139$]. The placebo group made significantly fewer centre line crossings (and spent less time over the centre line) compared to the Medium ($p < .01$) and High BAC groups ($p < .001$). They also made fewer edge line crossings (and spent less time over the edge lines) and had fewer false alarms compared to the High BAC group ($p < .05$). With regard to peak speed, the Medium BAC group displayed a significantly ($p = .02$) lower peak speed than High BAC group (but there was no significant difference between the Medium or High BAC group compared to placebo).

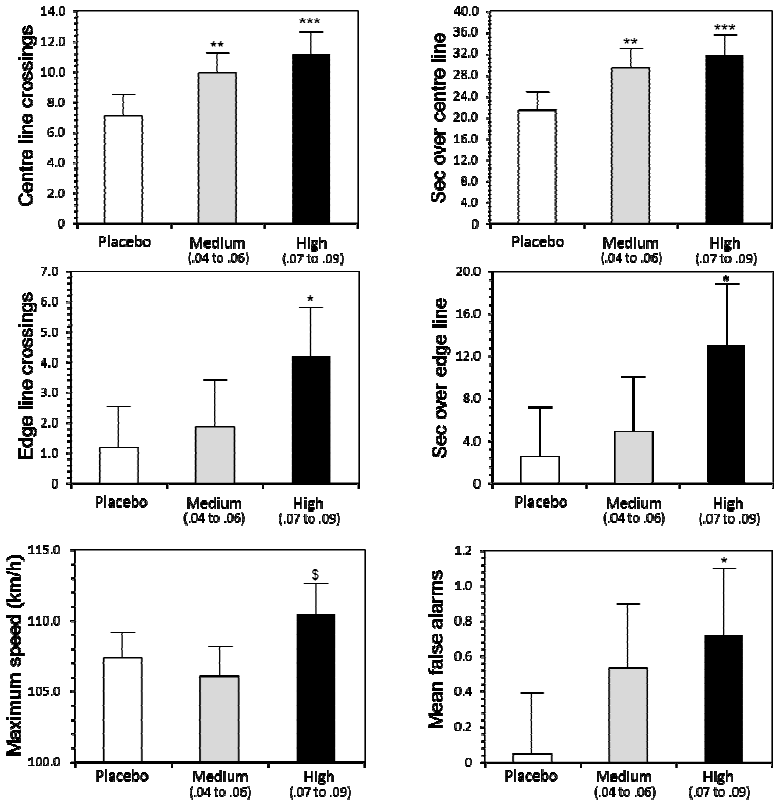

Figure 3. Participants' driving performance as a function of their BAC level. Error bars represent 95% confidence intervals *** = p < .001, ** = p < .01, * = p < .05 compared to Placebo group; $ = p < .05 compared to Medium BAC group.

Analysis comparing the ascending and descending limbs of the intoxication curve

To compare the effects of equivalent BAC during the ascending and descending limbs of the intoxication curve, two new groupings of the participants' data were formed based on their ascending and descending BAC levels. Participants' ascending BACs (during Block 2 or 3) were matched with their equivalent descending BACs (Block 3, 4 or 5) and they were then allocated into one of two groups, Medium (BAC of .04-.069) or High (BAC >.07). Fourteen participants were allocated to the Medium group with an average ascending BAC of .056 (range .044 -.069) and an average descending BAC of .053 (range .045 - .064). Fifteen participants were allocated to the High group with an average ascending BAC of .094 (range .070 - .126) and an average descending BAC of .092 (range .064 and .125). Twelve participants could not be classified into one of these two groups because they did not display ascending and descending BAC levels that could be compared.

Figure 4 presents the measures that showed evidence of poorer performance on the ascending limb of the intoxication curve compared to the descending limb for equivalent BAC levels (acute tolerance). A 2 x 3 mixed ANOVA indicated that the ratings of subjective intoxication were lower during the descending limb [Intoxication Curve x BAC interaction $F(2,46) = 2.799$, $p = .071$, $\eta_p^2 = .109$], predominantly due to the participants in the medium BAC group. Ratings of willingness to drive showed a similar pattern with medium BAC participants showing greater willingness to drive during the descending limb, reflected in a marginally significant interaction between intoxication curve and BAC group [$F(2,46) = 3.005$, $p = .059$, $\eta_p^2 = .116$]. The total errors for Groton maze recall showed a marginally significant effect of the intoxication curve comparison [$F(2,46) = 3.591$, $p = .064$, $\eta_p^2 = .072$], with more errors on the ascending limb than the descending limb, and a significant BAC effect. Even though there was no statistically significant interaction, the data indicate that the High group made fewer maze recall errors during descending BAC compared to ascending BAC.

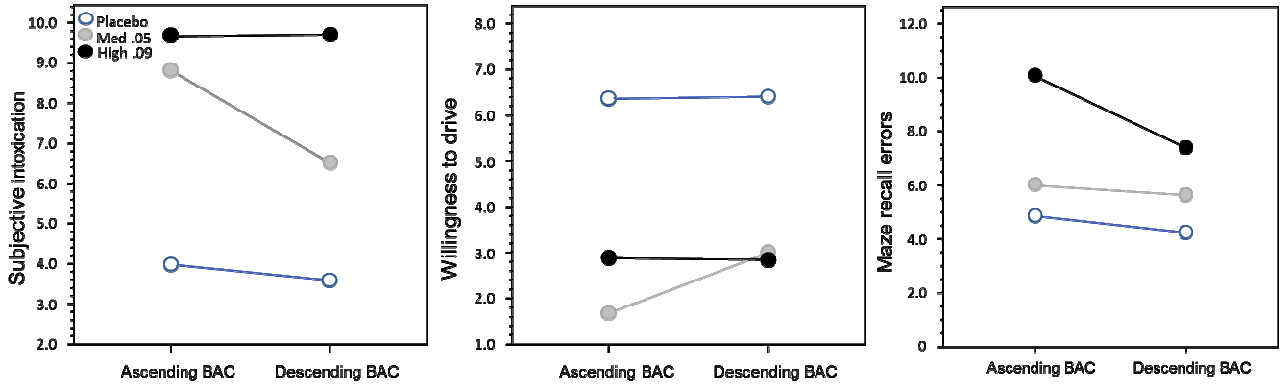

Figure 4. Participants' performance measures displaying evidence of acute tolerance (poorer performance on the ascending limb for equivalent BAC levels).

Figure 5 shows the driving and cognitive test measures that displayed evidence of acute protracted errors. For the cognitive tasks, descending BACs resulted in more maze learning errors, particularly for the High group, reflected in a significant interaction between intoxication curve and BAC [$F(2,46) = 3.710$, $p = .032$, $\eta_p^2 = .139$] In the chase task, there was significantly slower performance by the BAC groups on the descending limb but not by the Placebo group, [$F(2,46 = 2.772$, $p = .073$, $\eta_p^2 = .108$]. The time spent driving over 100 km/h was significantly greater during the descending limb for both BAC groups [Intoxication x BAC interaction $F(2,46) = 4.401$, $p = .018$, $\eta_p^2 = .164$]. The time spent over the centre line and the number of centre line crossings also showed evidence of acute protracted error with both measures showing an increase during the descending limb of the intoxication curve.

Measures of participants' maximum speed while driving [$F(2,46) = 10.994$, $p < .001$, $\eta_p^2 = .328$], their number of edge line crossings [$F(2,46) = 5.968$, $p = .005$, $\eta_p^2 = .210$] and time over the edge line [$F(2,46) = 5.332$, $p = .008$, $\eta_p^2 = .192$], the SD of their lane positions [$F(2,46 = .477$, $p = .039$, $\eta_p^2 = .134$) and the number of responses to false alarm vehicles [$F(2,46) = 4.168$, $p < .022$, $\eta_p^2 = .156$] all showed significant BAC effects. The High alcohol group was significantly worse than the Placebo group for all of these measures (all p's <.05), while the Medium group's maximum speed was significantly lower than the High BAC group ($p < .01$).

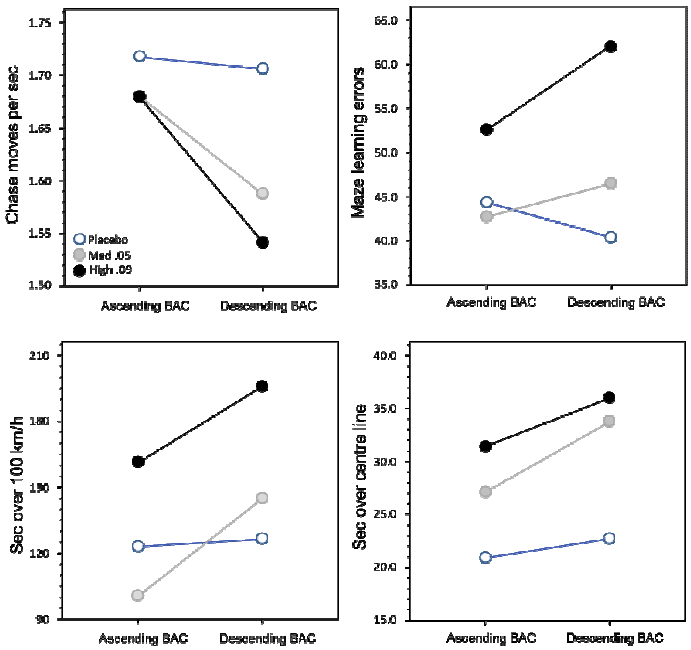

Figure 5. Participants' performance measures displaying evidence of acute protracted error (poorer performance on the descending limb for equivalent BAC levels).

Discussion

The main goals of this research were to determine the driving and cognitive impairment associated with BAC levels of .05 and .08 mg/ml and to investigate performance asymmetries (acute tolerance and acute protracted error) accompanying the onset and recovery from intoxication. Regarding the effective difference between .05 and .08 BAC levels, the present study demonstrated findings reported elsewhere in the literature; substantial impairment produced by a BAC of .08, with more subtle effects at .05 evident only for more complex tasks such as the correction of hazard avoidance (Liu and Fu 2007; Moskowitz and Fiorentina 2000; West et al 1993). Specifically, participants with a BAC level of .08 had significant increases in edge and centre line crossings in the simulator, spent significantly longer amounts of time over the edge line and centre line, displayed a disinhibition of reactions to false alarm vehicles at intersections, and had much higher peak speeds. In the cognitive test battery participants made significantly more errors learning and recalling a computer-based maze, a task of executive function, problem solving, memory and visual attention.

At .05 BAC only the number of centre line crossings and amount of time spent over the centre line were significantly worse than the performance of participants in the Placebo condition. It is interesting to note that the centre line and edge line crossing measures represent somewhat different aspects of driving performance. The edge line crossing measures were completely independent of the responses to vehicles at intersections, whereas the centre line crossings included participants' steering reactions to the hazard vehicles. While it was possible to avoid the hazard cars with little or no movement across the centre line, participants in the two alcohol groups tended to exaggerate their steering responses to avoid the cars, crossing into the opposing lane and remaining there significantly longer than drivers in the Placebo condition.

Regarding the asymmetries between intoxication onset and recovery, the participants' self-ratings of intoxication and willingness to drive showed acute tolerance (higher levels during the ascending limb) along with their error rates for the maze recall task. Other measures showed evidence of acute protracted errors (poorer performance during the descending limb) including: response speed on the chase task, errors on the maze learning task, the amount of time they exceeded the speed limit in a simulated driving task, and exaggerated steering responses to hazards. These findings replicate

recent published reports (Cromer et al 2010; Schweizer and Vogel-Sprott 2008; Weafer and Fillmore 2012), albeit the present study has shown them to occur with more moderate BAC levels.

As others have noted, the combination of these two effects, acute tolerance for self-ratings of intoxication and acute protracted errors for some components of the driving task, is a particularly dangerous mixture (Cromer et al 2010; Schweizer and Vogel-Sprott 2008). In essence, drivers mistakenly judge their sobriety as recovering faster than their BACs actually decline, at a time when their impairment on several important driving skills is actually getting worse. In the present study, the acute protracted error effect was associated with some delayed impairment that was nearly equivalent to that seen for substantially higher BAC levels, even while participants were reporting decreased feelings of intoxication and increased willingness to drive.

A secondary research objective was to identify the relationship between drivers' perception of intoxication and the actual level of impairment produced. Although the two alcohol groups rated themselves as more intoxicated than participants in the Placebo condition, the two alcohol groups' ratings did not differ from one another. In other words, although the participants could tell that they were intoxicated, they could not accurately determine the level of their intoxication. Nor were the participants able to judge how much alcohol they had consumed: Both alcohol groups underestimated the amount they had consumed, and high dose participants' estimates were approximately half of their actual dose. The willingness to drive ratings displayed a similar pattern, participants in the two alcohol groups rated themselves less willing to drive than the Placebo participants, but there was no difference between the two alcohol groups. The poor self-assessments of intoxication and the performance asymmetry during recovery suggest an important focus for public education regarding alcohol and driving. After drinking even moderate amounts of alcohol, drivers' judgement of their intoxication is impaired.

References

Akerstedt, T., Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *Int J Neuro*, *52*(1-2), 29-37.

Babor, T.F., Higgins-Biddle, J.C., Saunders, J.B., Monteiro, M.G. (2001). *AUDIT: The Alcohol Use Disorders Identification Test—Guidelines for Use in Primary Care*. 2nd ed. Geneva, Switzerland: World Health Organization. 40pp.

Beirness, D.J. (1987). Self-estimates of blood alcohol concentration in drink-driving context. *Drug Alc Dep*, *19*, 79-90.

Blomberg, R.D., Peck, R.C., Moskowitz, H., Burns, M., Fiorentino, D. (2009). The Long Beach / Fort Lauderdale relative risk study. *J Saf Res*, *40*, 285-292.

Charlton, S.G., Starkey, N.J. (2011). Driving without awareness: The effects of practice and automaticity on attention and driving. *Transp Res F: Traffic Psychol Behav*, *14*, 456-471.

Cromer, J.R., Cromer, J.A., Maruff, P., Snyder, P.J. (2010). Perception of alcohol intoxication shows acute tolerance while executive functions remain impaired. Exp Clin *Psychopharmacol*, *18*, 329 – 359.

Harrison, E., Fillmore, M.T. (2005). Social drinkers underestimate the additive impairing effect of alcohol and visual degradation on behavioural functioning. *Psychopharmacol*, *177*, 459-467.

Lenné, M.G., Dietz, P.M., Trigg, T.J., Walmsley, S., Redman, J.R. (2010). The effects of cannabis and alcohol on simulated arterial driving: Influences of driving experience and task demand. *Accid Anal and Prev*, *42*, 859-866.

Liu, Y-C., Fu, S-M. (2007). Changes in driving behaviour and cognitive performance with different breath alcohol concentration levels. *Traffic Inj Prev*, *8*, 153 – 161.

Maycock, G. (1997). *Drinking and Driving in Great Britain – A Review*. TRL Report 232. Crowthorne: Transport Research Laboratory, Great Britain.

Mets, M.A.J., Kuipers, E., de Senerpont Domis, L.M., Leendeers, M., Olivier, B., Verster, J.C. (2011). Effects of alcohol on highway driving in the STISIM driving simulator. Human *Psychopharmacol*, *26*, 434-439.

Moskowitz, H., Fiorentino, D. (2000). *A Review of the Literature on the Effects of Low Doses of Alcohol on Driving-Related Skills*. NHTSA Report DoT HS 809 028 Washington DC: U.S. Department of Transportation.

Phillips, D.P., Brewer, K.M. (2011). The relationship between serious injury and blood alcohol concentration (BAC) in fatal motor vehicle and accidents: BAC = 0.01% is associated with significantly more dangerous accidents then BAC = 0.00%. *Addiction*, *106*, 1614-1622.

Schweizer, T.A., Vogel-Sprott, M. (2008). Alcohol-impaired speed and accuracy of cognitive functions: A review of acute tolerance and recovery of cognitive functions. Exp Clin *Psychopharmacol*, *16*, 240-250.

Veldstra, J.L., Brookhuis, K.A., de Waard, D., Molmans, B.H.W., Verstraete, A.G., Skopp, G., Jantos, R. (2012). Effects of alcohol (BAC 0.5%) and ecstasy (MDMA 100 mg) on simulated driving performance and traffic safety. *Psychopharmacol*, *222*, 377-390.

Weafer, J., Fillmore, M.T. (2012). Acute tolerance to alcohol impairment of behavioral and cognitive mechanisms related to driving: drinking and driving on the descending limb. *Psychopharmacol*, *220*, 697-706.

West, R., Wilding, J., French, D., Kemp, R., Irving, A. (1993). Effect of low and moderate doses of alcohol on driving hazard perception latency and driving speed. *Addiction*, *88*, 527-532.