Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml
December 2013

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NZ Transport Agency research report 541
Contracted research organisation – Traffic and Road Safety Research Group,
University of Waikato
Charlton, SG and NJ Starkey (2013) Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml. NZ Transport Agency research report 541. 63pp.

The Traffic and Road Safety Research Group, University of Waikato, was contracted by the NZ Transport Agency in 2012 to carry out this research.

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Keywords: acute tolerance, alcohol, cognitive performance, driving, driving simulator
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Acknowledgements

The authors would like to thank all those who took part in this study and our research assistants who provided invaluable help with data collection and data entry as well as members of the steering committee; Paul Graham of the NZTA, Rachael McLaren of the Ministry of Transport, and Inspector Dave Parsons of NZ Police. We would also like to express our thanks to the peer reviewers of this report, Barry Hughes at the University of Auckland and Craig Gordon of the Health Promotion Agency.

Abbreviations and acronyms

BAC  blood alcohol concentration
SDLP  standard deviation of lane position
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Executive summary

The research described in this study had two main objectives: 1) evaluate the effects of alcohol on the psychomotor, cognitive and driving abilities of New Zealand drivers across the 0.05% and 0.08% blood alcohol concentration (BAC) levels1, and 2) identify the relationship between drivers' perception of intoxication and the actual level of impairment produced. In order to address these objectives, several preliminary questions needed to be addressed.

The first of these questions was how to produce BACs at the levels desired for comparison. The results of a series of dosage trials showed that the amount of alcohol required to produce BACs of 0.05% and 0.08% varied considerably from person to person, even taking into account differences in their body weight. In particular, alcohol produced substantially higher BAC levels for women than for men consuming equivalent amounts of alcohol. Based on the results, a dosage protocol that used a titration procedure was established whereby two initial drinks were followed by an optional third drink only if needed. This protocol was developed in order to ensure an ethical treatment of the participants while still ensuring the desired BAC levels were achieved.

A second preliminary question concerned what performance measures would best detect meaningful and practically significant differences between the alcohol levels of interest. A pilot test was conducted with six participants with whom we tested a combination of cognitive function, driving performance and subjective measures that had been suggested by the published literature. The results of the pilot test indicated that the alcohol dosing protocol was successful in producing the desired range of BAC levels and the length and timing of the testing procedures were manageable by the researchers and acceptable to participants. Based on the findings from the pilot test a final set of test measures was developed to address the research objectives.

The dosage trials and pilot test were followed by the full experiment in which 61 participants (33 men and 28 women) were recruited and randomly assigned to one of three alcohol dose groups: a high alcohol group (0.08% BAC); a medium alcohol group (0.05% BAC); and a placebo control group. An analysis of performance associated with specific BAC levels achieved by the participants allowed a direct comparison of the two BAC levels of interest (0.05% and 0.08%). That analysis showed that 0.08% BAC produced significant impairment across a broad range of cognitive and driving measures, relative to the participants in the placebo condition. The participants with a BAC level of 0.08% had significant increases in edge and centre line crossings in the driving simulator, spent significantly longer amounts of time over the edge line and centre line, displayed a disinhibition of reactions to hazard 'false alarms' (vehicles at intersections) and had much higher peak speeds. In a test battery of cognitive performance measures participants made significantly more errors learning and recalling a computer-based maze and longer response times on a card identification task, measures of executive function, problem solving, memory and visual attention.

The participants with a BAC of 0.05% also showed some performance impairment on these measures, but the level of impairment was not large enough to be statistically worse than the placebo control condition for the number of edge line crossings, seconds spent over the edge line, peak speed, or any of the cognitive performance measures. At 0.05% BAC only the number of centre line crossings and amount of time spent over the centre line were significantly worse than the performance seen for the placebo condition. The centre line crossing measures were distinct from the edge line crossings in that centre line

1 Throughout this report blood alcohol concentrations (BAC) are expressed in units of milligrams per 100 millilitres (for example 50mg/100ml) or the equivalent percentage (for example 0.05%, or 0.05).
crossings were designed to include participants' steering reactions to hazard vehicles at intersections. The
centre line crossing measures indicated that participants at both BAC levels (0.08% and 0.05%) 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.

A second research objective was to identify the relationship between drivers’ ratings of their intoxication
and the actual level of impairment produced. The results showed that participants in the two alcohol
groups rated themselves as significantly more intoxicated than did the participants in the placebo
condition. The ratings of intoxication of the two alcohol groups did not differ from one another, however,
indicating that although the participants could tell they were intoxicated, they could not accurately
determine how intoxicated they were. Similarly, the participants were not able to correctly judge how
much alcohol they had consumed. Both of the alcohol groups underestimated the amount they had
consumed, and the amounts they estimated were very similar, which meant that participants receiving the
high dose were extremely inaccurate, approximately half of their actual dose. Participants’ ratings of their
willingness to drive throughout the experimental sessions 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 results of the full experiment demonstrated findings that have been reported elsewhere in the
published literature; substantial impairment produced by a BAC of 0.08%, with more subtle effects of
0.05% being evident only for more complex tasks such as hazard avoidance (Leung and Starmer 2005; Liu
and Fu 2007; Moskowitz and Fiorentina 2000; West et al 1993). Participants’ subjective estimates of their
own levels of intoxication and impairment were shown to be relatively insensitive to actual BAC levels and
a poor indicator of their performance impairment (Cromer et al 2010; Schweizer and Vogel-Sprott 2008;
Weafer and Fillmore 2012).

The results of the present study also replicated some interesting asymmetries in performance impairment
that have been reported in the recent literature. Some aspects of performance show greater impairment
when BAC levels are rising, as compared with somewhat better performance when BAC levels are falling
(post-peak), even when the absolute BAC levels are the same, a phenomenon known as ‘acute tolerance’. Other measures of performance display poorer performance during the post-peak stage than during rising
or peak BACs (for equivalent BAC levels), a phenomenon known as ‘acute protracted error’. In the present
study, the number of edge line crossings, time spent over the centre line and edge line, maximum speeds,
and maze learning and maze recall errors all showed poorer performance during the post-peak phase than
that seen for ascending or peak BAC levels (acute protracted errors). Response disinhibition (reactions to
hazard false alarms) and ratings of subjective intoxication during the ascending/peak BAC were worse
than that seen during post-peak, meaning the effects were greater during the initial stages of intoxication
(acute tolerance). These findings replicate recent published reports (Cromer et al 2010; Schweizer and
Vogel-Sprott 2008; Weafer and Fillmore 2012), albeit the present study showed them to occur even in the
medium to low range of BACs (0.01% to 0.055%).

As others have noted, the combination of these two effects, specifically acute tolerance for self-ratings of
intoxication and acute protracted errors for many 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 much faster than their BACs 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 a level of delayed impairment that was nearly equivalent to that seen for
substantially higher BAC levels, at the same time when those participants were indicating an increasing
willingness to drive.
Finally, the present study produced some rather interesting findings with regard to New Zealand drivers’ knowledge of the law associated with drink-driving and attitudes towards that law. Fewer than half of the participants from the general population of drivers were able to correctly state the current adult drink-drive limit (in either blood or breath alcohol concentration). However, of the sample of drivers in the present study, the majority (57.3%) thought the current drink-drive limit should be reduced. This result coincides with other recent surveys of the larger driving population; the Ministry of Transport’s 2012 driving attitudes survey found that 60% of New Zealanders favoured a lower legal blood-alcohol limit for driving (41% thought the limit should be lowered from 80mg/100ml to 50mg/100ml and a further 19% wanted it lowered to zero) (Ministry of Transport 2012b). Similarly, a New Zealand Herald DigiPoll released at the time this report was being prepared indicated that 65% of New Zealanders were in favour of reducing the drink-drive limit from 80mg/100ml to 50mg/100ml.

The results of the present study suggest an important focus for public education regarding alcohol and driving, beyond simple information on the enforced alcohol limits for drivers. One message might make the public aware of the fact that when intoxicated at the currently enforced adult limit most drivers cannot accurately judge the amount of alcohol they have consumed or their level of performance impairment. After drinking even moderate amounts of alcohol, drivers’ judgement of their intoxication is impaired. A second message might address the issue of the delayed or protracted effects of alcohol intoxication on motor performance; some aspects of safe driving recover very slowly, and may persist even after BACs have fallen to below legal limits. There is recent evidence of considerable impairment on some aspects of cognitive performance (eg attention) that last until the morning after alcohol has been consumed (McKinney et al 2012; Verster et al 2003).
Abstract

The goal of the research was to evaluate the effects of alcohol on the performance of New Zealand drivers across 0.05% and 0.08% blood alcohol concentration (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 0.08% 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, and errors learning and recalling a computer-based maze. Moderate alcohol (BAC of 0.05%) produced some performance decrements, but not to the same degree as a BAC of 0.08%. An analysis comparing the impairment associated with peak and post-peak intoxication revealed that while some aspects of performance (eg motor coordination and response inhibition) showed acute tolerance, other measures (eg maze learning and recall errors, edge and centre line crossings, and maximum speeds) showed acute protracted errors. 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.
1 Introduction

The deleterious effects of alcohol on people’s ability to drive safely have been recognised for well over a century. As driving became widespread in the early 1900s drunk-driving became a crime, but as the level of alcohol could not be accurately measured, arrest rates remained low (the defence in court often being that the driver was tired). By 1910, however, Eric Widmark, a Swedish physiologist, developed a procedure for measuring the amount of alcohol in the blood. In 1931 Rolla Harger (an American biochemist), assisted by Robert Borkenstein (head of a forensic police laboratory in Indiana), used Widmark’s technique to invent the drunkometer, a device that measured blood alcohol from air blown into a balloon. A portable version (the precursor to the modern breathalyser) was developed and patented by Robert Borkenstein in 1954. The ability to measure blood alcohol concentrations (BAC) led to a rapid rise in the number of arrests for drunk-driving, and different countries adopted a range of BAC limits for driving, a legacy that is still in evidence today. Currently, some countries have a zero tolerance (eg Czech Republic, Russia and Romania), others have limits of 0.02% (Sweden and Poland), and in some countries, including New Zealand, the USA and the UK, the current legal drink-drive limit is 0.08% (for adult drivers with full licences). The majority of countries, however, currently have a limit of 0.05%, including Australia and many European countries such as France, Spain and Switzerland (Lerner 2012). The setting of BAC limits for drinking and driving are influenced by a variety of factors including crash risk and personal safety, and the studies that have been influential in setting current BAC limits are outlined below.

Between 1962 and 1963, Borkenstein et al (1964) conducted one of the largest case-controlled studies examining the effects on alcohol on driving: ‘The Grand Rapids Study’. The data was used to generate relative risk calculations for crash involvement as a function of BAC and informed the setting of BAC limits in many countries. One of the most cited aspects of this case-control study is the high elevation in crash risk (4.79) associated with a BAC of 0.10% (Compton et al 2002). In addition, the data also showed what has become known as the ‘Grand Rapids Dip’, an apparent decrease in crash risk for BACs between 0.01% and 0.04%. This data, in addition to that from a study by Borkenstein in 1981 (funded by the liquor industry) led to the suggestion that small amounts of alcohol might even improve driving performance, although Borkenstein himself promoted abstinence from alcohol when driving (Economist 2002). Subsequent reports (eg Hurst et al 1994; Zador 1989) have failed to find such a dip and it has been suggested that it was in fact an artefact of the data resulting from differences in the driver populations at each BAC (Allsop 1966) and the use of univariate data analysis techniques (Blomberg et al 2009). A more recent case-control study was conducted in Long Beach and Fort Lauderdale to address the limitations of the Grand Rapids study and provide data that represented the current driving population and their driving habits (Blomberg et al 2005; 2009). In this study, data was collected from 2871 crashes at two study sites over a 12-month period and a control group, matched on time, location and direction of travel was also recruited. The data (adjusted for covariates and data biases) showed a dose-related increase in crash risk from BACs over 0.04% which increased exponentially at BACs over 0.10%. At a BAC of 0.04% the adjusted relative crash risk was 1.38, which increased significantly to 2.69 at a BAC of 0.08%, much higher than the original relative risk estimate of 1.88 at the same BAC in the original Grand Rapids study (Allsop 1966).

Not only does the relative risk of crash involvement rise with increasing BAC, so does the risk and severity of personal injury. For fatal crashes in the USA between 1994 and 2008, the severity of in-car injuries increased significantly at BACs as low as 0.01% compared with fatal crashes involving drivers who had not been drinking. In addition the ratio of serious to non-serious injuries increases with increasing BACs (Phillips and Brewer 2011). In Great Britain, Maycock (1997) estimated that the risk of being involved in an injury accident is 2.9 times greater at a BAC of 0.05% and 5.6 times greater at a BAC of 0.08% (compared with a BAC of 0). At a BAC of 0.05%, the risk of being killed is increased by a factor of 5 and at a 0.08%
BAC, the risk of being involved in a fatal crash is increased 12.4 times. New Zealand based estimates are similar and indicate that for every 20mg/dl increase in BAC above zero the risk of driver fatal injury doubles (Keall et al 2004), with the risk being greatest for young males driving on rural roads on summer weekend nights (Keall et al 2005).

It is widely recognised that alcohol impairment represents one of the largest causes of serious road crashes in New Zealand (and internationally). The number of road deaths attributed to alcohol-impaired driving in New Zealand has risen in the past decade, contributing to over 30% of road deaths in the 2005-2011 reporting period. Taking just one year for which extensive analysis of alcohol-related crashes has been conducted, 1996, alcohol-related crashes were calculated to cost $1.2 billion, 38.5% of the total road crash costs in New Zealand. An additional tragedy is that of those who were killed or injured by alcohol related crashes in 1996, nearly half (48%) were innocent victims (Miller and Blewden 2001). In 2011 alcohol and other drugs were identified as a causal factor in at least 77 fatal crashes resulting in 85 causality deaths and 466 serious injuries. Of the drivers and motorcycle riders who died during 2011, it is estimated that 26% had a BAC exceeding the current legal limit of 0.08% ( Ministry of Transport 2012a).

Reducing the impact of alcohol-impaired driving is one of the areas of high concern identified in the New Zealand government’s Safer journeys: road safety strategy 2010-2020 (Ministry of Transport 2010). The role that alcohol plays in motor vehicle fatalities and crashes is also acknowledged by the general public. A survey examining New Zealanders’ opinions towards alcohol found that half to two thirds of the respondents thought that alcohol played a major or leading role in dangerous driving and traffic crashes, particularly in rural areas (Maclennan et al 2012). Furthermore, data from the Public attitudes to road safety: results of the 2012 survey (Ministry of Transport 2012b) indicated that 60% of New Zealanders were in favour of lowering the current legal BAC limit for driving.

The breath/blood alcohol limit for drivers should be based on clear evidence regarding the nature of the effects of alcohol on drivers and the likely consequence in terms of road casualties. Quantifying this latter aspect of the issue is problematic in that there has not been any legal requirement to record the BAC of drivers involved in crashes if their BAC reading is lower than the current legal limit (80mg/100ml for adults). Collection of more complete information regarding the BAC of crash-involved drivers is now being undertaken by New Zealand Police (since May 2011).

As regards the research evidence about the type of effect alcohol has on drivers, such information is essential to inform and support credible decision making in a way that can be expressed clearly to the New Zealand public, regardless of whether there is any reduction in the BAC limit (currently 0.08% BAC). A considerable amount of international research has identified adverse effects of alcohol on a number of behavioural and cognitive capabilities, but the majority of these studies have focused on the impairment produced by relatively high BAC levels such as 0.10%, which is above the current New Zealand limit. Further, these studies have typically examined performance decrements only at the peak of intoxication and only on one or two performance measures. Recently, however, there is evidence to suggest that recovery from the effects of alcohol may take longer for some cognitive and behavioural components of driving than others, and that drivers are often unaware of their diminished capacity on these aspects of their performance, even at relatively low BAC levels (Cromer et al 2010; Friedman et al 2011; Shinar 2007).

In a major review of the research literature published between 1981 and 1997, Moskowitz and Fiorentina (2000) found that alcohol produced a wide range of effects on divided attention, vigilance, tracking, perception, reaction time, critical flicker fusion, psychomotor skills and other cognitive functions (see table 1.1). Importantly, the amount of alcohol found to impair these functions differed markedly, as well as there being differences associated with the experimental methods used to assess impairment. Generally the more complex tasks, such as divided attention showed decrements in performance at lower
BACs (<0.01%), and at BACs of 0.05% information processing, choice reaction time and some higher order aspects of cognitive function became impaired (Tzambazis and Stough 2000), whereas simple reaction time tasks only became consistently impaired at higher BACs (>0.10%).

Table 1.1 Impairments associated with different BAC levels (adapted from Moskowitz and Fiorentina 2000)

<table>
<thead>
<tr>
<th>%BAC (g/100ml)</th>
<th>Lowest BAC at which impairment was found</th>
<th>First BAC at which 50% or more of behavioural tests indicated consistent impairment</th>
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<tr>
<td>0.100%</td>
<td>Critical flicker fusion</td>
<td>Simple reaction time, critical flicker fusion</td>
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<tr>
<td>0.090%–0.099%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.080%–0.089%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.070%–0.079%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.060%–0.069%</td>
<td>Cognitive tasks, psychomotor skills, choice reaction time</td>
<td></td>
</tr>
<tr>
<td>0.050%–0.059%</td>
<td>Tracking</td>
<td></td>
</tr>
<tr>
<td>0.040%–0.049%</td>
<td>Simple reaction time</td>
<td>Perception, visual functions</td>
</tr>
<tr>
<td>0.030%–0.039%</td>
<td>Vigilance, perception</td>
<td>Vigilance</td>
</tr>
<tr>
<td>0.020%–0.029%</td>
<td>Choice reaction time, visual functions</td>
<td></td>
</tr>
<tr>
<td>0.010%–0.019%</td>
<td>Drowsiness, psychomotor skills, cognitive tasks, tracking</td>
<td>Drowsiness</td>
</tr>
<tr>
<td>0.001–0.009%</td>
<td>Driving, flying, divided attention</td>
<td>Driving, flying, divided attention</td>
</tr>
</tbody>
</table>

Driving is a complex task comprising several complementary cognitive functions and there is accumulating evidence that some cognitive functions are affected by alcohol to a greater degree than others. With regard to alcohol’s effects on the ability to drive safely, a number of recent studies have examined driving performance using simulated driving tasks. Alcohol has been reported to result in a significant increase in speed at BACs as low as 0.02% and 0.05%; however the increase in speed was only slight ($M = 0.68\text{km}/\text{h}$) (Lenné et al 2010). Veldstra et al (2012) found that only BACs above 0.05% led to significant increases in speed compared with a placebo, but once again the increase was small and participants did not exceed the posted speed limit. Other findings suggest that driving speed is not affected at BACs of 0.05% (West et al 1993), and that higher BACs (0.08% and 0.11%) produce significant increases in the standard deviation of driving speed, rather than any increase in average speed per se (Mets et al 2011). Thus, an effect of alcohol may be to increase drivers’ speed variability, which does not always translate to increases in average driving speed.

Another key component of safe driving is hazard detection. West et al (1993) conducted two double-blind studies (with placebo controls) using a simulated driving task and found that a BAC of 0.05% produced significant impairments in drivers’ reactions to detect and respond to road hazards. Using a somewhat different task, however, Veldstra et al (2012) found no effect of BACs of 0.03%, 0.05% and 0.08% on complex driving tasks such as reacting to a car pulling out, running red lights or frequency of crashes. Therefore, in terms of drivers’ reactions to hazards, the effects of various levels of alcohol remain unclear.
As many alcohol-related crashes are a result of single vehicles leaving the road or crossing the centre line (Keall et al 2004), experimental studies of the effect of alcohol on driving often incorporate a measure of drivers’ lane position. This is most commonly reported as the standard deviation of lane position (SDLP) and reflects the amount the car is moving across the lane (ie weaving). The majority of driving simulator studies have found that alcohol leads to a dose dependent increase in SDLP, from BACs of 0.02% to 0.011% (Lenné et al 2010; Mets et al 2011; Veldstra et al 2012).

Information processing and divided attention also play an important role in safe driving skills and, as suggested by Moskowitz and Fiorentina’s (2000) review of the literature, it appears that these aspects of driving become impaired at BAC levels well below 0.08% (Chamberlian and Solomon 2002). Liu and Fu (2007) found that drivers’ attention and information processing abilities were impaired at lower BAC levels (0.05%) than simple psychomotor components of the driving task (0.08%). Using a driving simulator and a range of secondary tasks they tested participants’ performance under a range of BAC levels and found that higher-order cognitive components of driving involving divided attention, information processing, and memory showed significant deterioration in performance at much lower levels than outward signs of driving behaviour involving motor coordination. Importantly, Liu and Fu concluded that this asymmetry in the effects of alcohol on different components of driving may mislead drivers into thinking that because they are still capable of steering that they are safe from the potential dangers of drink-driving (Liu and Fu 2007). In keeping with these findings, Leung and Starmer (2005) found that although a BAC of 0.06% did not produce impairments in some driving-related decisions (such as estimates of time to collision and overtaking), it did produce a significant impairment in a divided attention task (vehicle detection) compared with the placebo control condition. It has been suggested that impairments in divided attention may result from alcohol decreasing the information processing capacity (Fillmore 2003), as well as reducing task switching efficiency (Moskowitz and Fiorentina 2000). Therefore when task demands exceed a driver’s capacity, which has been diminished by alcohol, performance suffers and the risk of a crash is increased.

The fact divided attention is affected at relatively moderate BAC levels is of some concern with regard to driving which takes place in a complex, constantly changing environment with multiple stimuli competing for the drivers’ attention. Studies examining the joint effects of alcohol and distraction have found that the impairments produced by alcohol (BAC of 0.07%) are exacerbated when the driver is required to complete a task (manipulating controls in the vehicle, completing verbal tests or responding to visual stimuli) in addition to the driving (Rakauskas et al 2008). Similarly, Harrison and Fillmore (2011) found that the combination of alcohol (BAC 0.08% – 0.09%) and completion of a secondary task (two-choice reaction time) produced a greater increase in SDLP and increased failure to stop at a red light compared with alcohol alone or placebo. Thus the effects of alcohol appear to be exacerbated in situations where the workload is high.

Young drivers are over-represented in crash statistics world-wide and this is in part due to high levels of risk taking and impulsivity (Dahl 2004). Interestingly, even low doses of alcohol can promote impulsive behaviour by impairing inhibitory behaviour control mechanisms. A task commonly used to assess the disinhibiting effects of alcohol is the go/no-go reaction time task, where participants have to respond as quickly as possible to a go signal and inhibit the response to a no-go signal (eg de Wit et al 2000). Similar alcohol-related impairments can be detected across a range of other impulsivity measures (Dougherty et al 2008). In addition, laboratory studies indicate that alcohol-induced disinhibition is most likely to occur when activating a behaviour is as desirable as inhibiting a behaviour (Steele and Southwick 1985), for example choosing between being late for an appointment and driving safely, or speeding and arriving on time. To examine the interaction between alcohol, disinhibition and response conflict in relation to driving, Fillmore et al (2008) conducted a study where participants completed a stimulated drive and a
cued go/no-go task. Response conflict was induced by rewarding participants for completing the drive as quickly as possible while penalising them for exceeding speed limits and failing to obey traffic signals. In the alcohol condition (peak BAC 0.09%), participants showed increased risk taking and poorer driving performance, suggesting that the disinhibiting effects of alcohol may increase risky driving and the likelihood of a crash.

The studies reviewed so far demonstrate that alcohol consumption impairs different aspects of driving at different BAC levels, and that task complexity and driving demands may exacerbate alcohol’s effects. However, one other important issue which influences the effects of alcohol on performance is whether the concentration of alcohol in the blood is rising or declining. Ingestion of an acute dose of alcohol leads to a relatively swift rise in BAC, which peaks and then gradually declines. This is commonly referred to as the BAC curve, with ascending (as BAC levels increase) and descending (decreasing BAC) limbs of the curve. 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; Goldberg 1943), a phenomenon referred to as ‘acute tolerance’. Initially it was thought that this could be explained by practice effects (performance was always measured on the ascending limb first). More recent studies using computerised tasks less susceptible to practice effects indicated that acute tolerance effects did occur; however, it was not observed across all tasks.

A recent review (Schweizer and Vogel-Sprott 2008) found that speed of cognitive performance (on tasks of inhibition, information processing and selective attention) tended to recover more quickly than the decline in BAC, so that performance at a specific BAC on the descending limb was better than at the same BAC on the ascending limb, i.e., acute tolerance. Behavioural activation also showed acute tolerance, with faster reaction times to go signals on a go/no-go task during descending BACs but not to no-go signals (Fillmore et al 2005; Ostling and Fillmore 2010). Errors, however, failed to diminish during descending BACs and in some cases even rose, a phenomenon termed ‘acute protracted error’ (Schweizer and Vogel-Sprott 2008). It has been suggested that the acute protracted error effect may be related to whether the task is under automatic or conscious control (Fillmore et al 1999; Schweizer and Vogel-Sprott 2008), but the reasons behind this and the acute tolerance effect are still not well understood. Nonetheless the occurrence of acute tolerance and acute protracted errors has safety implications in terms of driving. In terms of acute tolerance, the rapid recovery of some behavioural functions when BAC remains high may lead drivers to think they are safe to drive even when they are over the legal limit. In contrast, the acute protracted error effect suggests that some important cognitive functions may remain impaired even when BACs are below the legal limit.

Of related concern, acute tolerance effects appear to also affect subjective estimates of one’s own level of intoxication. Several studies have shown that the self-evaluation of the effects of alcohol (usually obtained by a rating of subjective intoxication) is generally quite poor, and levels of intoxication and impairment are often underestimated (e.g., Beirness 1987; Harrison and Fillmore 2005; Weafer and Fillmore 2012). Further, the amount of underestimation appears to be greatest during the descending limb of the intoxication curve, when BACs are declining (Bois and Vogel-Sprott 1974; Hiltunen 1997). In other words, drivers have a tendency to believe that they are recovering from the effects of alcohol much faster than is the case.

Perceptions of subjective intoxication have also been compared simultaneously to participants’ performance on a range of cognitive and driving tasks. Cromer et al (2010) used a computer-based maze task, the Groton maze learning test, to compare alcohol’s effects on visuomotor performance and higher-order executive functions. They found that performance on visuomotor tasks recovered from alcohol effects more rapidly than executive function abilities, and that drivers’ perceptions of their level of intoxication appeared to be based on only their visuomotor abilities. In other words, drivers’ cognitive
functions may remain impaired even though they subjectively feel as though they have recovered from the effects of alcohol. The authors concluded that 'Subjective perception of intoxication is a poor indicator of sobriety and of the ability to operate a motor vehicle' (Cromer et al 2010, p337).

With a focus on assessing acute tolerance and driving performance, Weafer and Fillmore (2012) used a 30-minute test battery consisting of a simulated driving task, reaction time task, subjective intoxication ratings, willingness to drive ratings, and a grooved pegboard task to assess the performance of participants in a placebo or alcohol condition (dose of 0.65g/kg, resulting in a BAC of 0.09%) on the ascending and descending limbs of the BAC curve. Interestingly, motor coordination, subjective intoxication and willingness to drive showed acute tolerance (ie better performance on the descending limb of the BAC curve compared with performance at the same BAC on the ascending limb) whereas driving performance and inhibitory control showed slower recovery. These findings reinforce those of Cromer et al (2010), and suggest that drivers have the potential to underestimate their level of intoxication particularly when their BAC is declining and they may decide to drive even though crucial aspects of their driving and cognitive performance are still impaired. Furthermore, the fact the motor coordination shows acute tolerance suggests that drivers’ performance on tasks included in the traditional field sobriety test may be misleading (Rubenzer 2011).

More recent studies suggest that the acute protracted error effect, which occurs on the descending limb of the BAC curve, may persist long after drinking has ceased, (until the morning after drinking) when BACs have returned to zero. Performance on cognitive tasks assessing reaction time, divided attention and selective attention the morning after alcohol consumption has been found to be as impaired or worse than participants’ performance at the legal driving limit of 0.08% (McKinney et al 2012; Verster et al 2003).

As can be seen in the above brief overview of the published literature, there is a growing consensus that alcohol affects some aspects of driver performance to a greater degree than others, at BAC levels well below the current legal limit of 0.08%, and that drivers’ perception of their fitness to drive may not provide an accurate indication of their actual ability to drive safely. As such, there is a clear need to unambiguously assess drivers’ cognitive and performance impairments produced by alcohol in concert with any move to re-visit the New Zealand limits on drivers’ BAC. This assessment was conducted in the high-fidelity driving simulator at the University of Waikato using a sample of New Zealand drivers in order to provide up-to-date information that is directly relevant to the New Zealand driving population for decision makers, road safety educators and law enforcement personnel.

The focus of this research was therefore to provide research-based evidence on the behavioural and cognitive impairment associated with BAC levels under the current adult limit of 0.08%. The research was designed to replicate and extend some recent international findings about alcohol-impaired driving with a sample of New Zealand drivers. The research was also intended to investigate possible asymmetries across various cognitive and behavioural components of the driving task during the recovery from alcohol effects and drivers’ misperceptions of their own fitness to drive. The results of this research may thus be of relevance to not only policy makers, but also for education and enforcement programmes specific to the New Zealand situation.

The research objectives were to:

1. Evaluate the effects of alcohol on the psychomotor, cognitive and driving abilities of New Zealand drivers across the range of BAC levels.

2. Identify the relationship between drivers’ perception of intoxication and the actual level of impairment produced.
2 Participant recruitment and ethics

All of the methods used in this study were submitted for review by, and received ethical approval from, the School of Psychology Research Ethics Committee at the University of Waikato.

Volunteers were eligible to take part if they were aged between 20 and 50 years, held a full New Zealand driver licence, were in good health and drank occasionally. People expressing an interest in the experiment were first screened to ensure they were in good health with no neurological/psychological conditions (e.g., head injury, stroke), no contra-indicated medication, and for female participants no possibility of being pregnant. They had to consume alcohol occasionally but not excessively, with a score of <8 on the Alcohol Use Disorders Identification Test (AUDIT). The AUDIT is used as a screening test for hazardous drinking (recommended by the World Health Organisation) and consists of three domains (hazardous alcohol use, dependence symptoms, harmful alcohol use) assessed via 10 items (Babor et al. 2001).

Participants were recruited via word of mouth, advertisements on notice-boards (in the community and at the university) and on the university’s e-learning platform. Individuals who were interested in participating emailed or phoned the researchers to receive more information about the study. This was followed by completion of the eligibility screening form (either in person or over the telephone) and arrangements were made for them to attend the laboratory. All volunteers provided informed consent before participating in the study. Further details about the participants for each stage of the study are presented in the relevant sections below.
3 Dosage trial

Previous studies have used two different approaches to calculate alcohol doses for experimental research. The first of these approaches was based on participants consuming a dose of alcohol based on their body weight while the second approach involved participants consuming an alcoholic beverage at set time intervals until they reach a desired BAC.

Using the first approach, Fillmore and his colleagues at the University of Kentucky reported that an alcohol dose of 0.65g/kg of body weight would reliably produce a BAC of 0.08% (Fillmore et al 1999, 2005, 2008; Harrison and Fillmore 2005, 2011; Ostling and Fillmore 2010; Weafer and Fillmore 2012). These studies, however, typically focused on the performance effects resulting from the alcohol dose administered and did not often report individual BAC levels that resulted from this dose. In contrast, other researchers have reported that doses of 0.4g/kg and 0.6g/kg produce peak BACs of less than 0.025% and below 0.05% respectively, although the range of BACs produced by these doses varied considerably, from 0.01% to 0.05% for the lower dose and 0.02% to 0.08% for the higher dose (Lenné et al 2010). Other studies have used lower alcohol doses for women, for example Leung and Starmer (2005) used an alcohol dose of 0.70g/kg for men and 0.60g/kg for women and reported that a mean BAC of 0.06% resulted (although their goal had been 0.08%). Women frequently achieve a higher BAC from a given dose of alcohol compared with men and it is thought that this may be due to differences in body composition, metabolism and hormonal fluctuations (Frezza et al 1990). Methodological differences between these studies (eg carbonated vs uncarbonated mixers and time course of alcohol administration) may have accounted for some of the differences in BAC levels produced, but typically ‘dose’ based alcohol administration results in quite varied BACs.

Studies using the second approach (consuming alcohol until the desired BAC is achieved) have typically asked participants to consume a series of 250ml beverages (approximately 40% vodka) at intervals of up to half an hour until participants reached the desired BAC (eg Cromer et al 2010; Tzambazis and Stough 2000). Although this approach is effective in assessing participants at specific BACs, the varied dosing regimen can lead to extended test sessions in which the amount of practice on assessment tasks can differ between participants. A third approach has been to use a combination of these two approaches; using a specific dose of alcohol as a guide, but adjusting or titrating the final number of drinks consumed depending on the participants’ BAC. For example, Veldstra et al (2012) calculated the volume of alcohol based on the participants’ weight, but adjusted their drink consumption to achieve and maintain the desired BAC.

As can be seen from this brief overview, there is no single reliable method for achieving specific BACs. Therefore, in order to select alcohol dosages and an administration protocol for the 0.05% and 0.08% comparison that was to be the focus of the present study, a dosage trial (that turned into a series of trials) was conducted with volunteer New Zealand drivers.

3.1 Trial 1

The first trial was conducted with seven volunteers, two males and five females ranging in age from 18 to 34 ($M = 25.71$). The volunteers were asked to consume no food or caffeine for three hours prior to the trial and to refrain from consuming any alcohol the evening before the test. A BAC of zero was confirmed for each participant on arrival at the laboratory. During the trial each participant was given an alcohol dose of either 0.45g/kg or 0.65g/kg by means of vodka (37.5% alcohol) mixed with orange juice in a 3:7 ratio. The alcohol-orange juice mixture was served in two drinks and participants were given five minutes to
consume each drink. Throughout the two-hour trial, the participants’ BACs were measured at 10-minute intervals with an Alcomate AccuCell AL9000 professional grade breathalyser that automatically converted breath alcohol readings into blood alcohol readings. The AL9000 breathalyser had a detection range of 0.000% – 0.400% BAC with a sensor accuracy of +/- 0.005%. At the conclusion of the trial, the participants were provided with a $20 gift voucher and given a ride home.

The participants’ BACs produced during the trial are shown in figure 3.1. As can be seen in the figure, BAC levels for both groups peaked 20 to 30 minutes into the trial (10 to 20 mins after consuming the alcohol), but none of the participants in the 0.45g/kg group reached their target BAC of 0.05%. The two female participants who received 0.65g/kg did reach 0.05%, and peaked at a BAC of approximately 0.06% (0.057% and 0.062%). For the males, the dose of 0.45g/kg produced a rapid peak BAC of 0.048% but the 0.65g/kg dose produced a BAC of 0.045%. Neither alcohol dose was sufficient to produce the higher BAC target of 0.08% in any of the participants tested.

Figure 3.1  BAC levels for seven participants measured every 10 minutes during the first trial of two alcohol doses. The alcohol was administered in two drinks consumed in the first 10 minutes of the trial.

3.2 Trial 2

Because the dosage methodology in Trial 1 produced such a rapid peak in BAC, and the doses used failed to result in the desired BAC levels, a second trial was conducted using a somewhat different alcohol administration protocol. For this trial, three drinks were prepared, two consumed at five minute intervals at the start, as in the previous trial, and a third consumed 30 minutes after finishing the second drink. Once again, BACs were collected at 10-minute intervals throughout the trial, and in an attempt to provide more reliable BAC readings, no BACs were collected for a five minute interval after any drink was consumed. Three alcohol dosages were used, ranging from 0.50g/kg to 0.75g/kg, spread across the three drinks. As before, all participants were given a $20 gift voucher to thank them for their participation.

Six volunteers were recruited for this trial, three men and three women ranging in age from 28 to 36 (M = 31.83). As can be seen in figure 3.2, neither participant receiving the 0.50g/kg dose achieved the target BAC of 0.50%. The male recorded a peak BAC of 0.032% five minutes after consuming his second drink and the female participant’s peak was 0.037% recorded 25 minutes after finishing her third drink. In comparison, the female receiving the 0.65g/kg dose recorded a peak BAC of 0.07% 25 minutes after consuming her third drink (although the polynomial line fit through her data points shows a peak of 0.06% BAC). The male receiving the 0.65g/kg dose peaked at 0.03% BAC 15 minutes after his third drink. The
female participant receiving the highest dose of alcohol, 0.75g/kg, recorded a peak BAC of 0.079% 25 minutes after consuming her third drink; the male’s BAC peaked at 0.046%, 35 minutes after his third drink.

Figure 3.2  BAC levels for six participants measured every 10 minutes during the second trial of three alcohol doses. The alcohol was administered in two drinks consumed in the first 10 minutes of the trial, followed by a third drink 30 minutes later.

This revised protocol provided a closer approximation to the desired BACs, particularly for the female participants at the two higher doses, and provided a slower onset of intoxication, a useful aspect given the duration of testing planned for the full experimental trials. The BAC levels for the female participants receiving these doses were very close to the two target levels for the full experiment. The BAC levels for male participants, however, still fell somewhat short of the target BACs, so a decision was made to examine a higher dose using only male participants while also replicating the effects of the 0.75g/kg dose.
3.3 Trial 3

Five male volunteers were recruited for this trial, ranging in age from 22 to 31 ($M = 25.6$). The participants were recruited as before, and given a $20 gift voucher to thank them for their participation. Two alcohol doses of 0.75g/kg and 0.90g/kg were evenly spread across three drinks. As can be seen in figure 3.3, the two males in the 0.75g/kg group peaked at 0.055% and 0.059% after consuming their third drink, very close to the lower of the two target BACs (0.05%). The three participants receiving the higher dose reached 0.074%, 0.055% and 0.045% after consuming their third drink, short of the target of 0.08% BAC. By 30 minutes after that drink their BAC levels were still well under the target of 0.08% (0.052%, 0.053% and 0.056%). At that point a decision was made, with the participants’ informed consent, to serve a fourth drink of equal volume to the first three bringing their total alcohol dose to 1.20g/kg. Shortly after consuming this fourth drink two of the participants recorded peak BACs of 0.10% and 0.089%, exceeding the target, while the third participant recorded a peak of 0.071%, 20 minutes after receiving his fourth drink.

**Figure 3.3** BAC levels for five male participants measured every 10 minutes during a trial of two higher alcohol doses. The alcohol was administered in two drinks consumed in the first 10 minutes of the trial, a third 30 minutes later, and for the participants receiving the higher dose, a fourth drink was consumed after another 30 minutes.

Based on a review of all of the results of the three dosage trials, alcohol doses for female participants were set at 0.60g/kg (to achieve a BAC of 0.05%) and 0.75g/kg (to achieve a BAC of 0.08%). For the men, a dose of 0.75g/kg was chosen to produce the target BAC of 0.05%, and a dose of 1.0g/kg was chosen to produce the target BAC of 0.08%. The next step in the experiment was a pilot test of these dosages, and the three drink administration protocol, in the context of collecting performance data from the participants using both a set of cognitive and driving performance measures.
4 Pilot test of experimental protocol

The next part of the study focused on testing the cognitive and driving performance measures in conjunction with the alcohol administration protocol to ensure 1) that data could be collected during the ascending, peak and descending portions of the BAC curve (as previously mentioned, the behavioural effects of alcohol have been shown to differ at equivalent BACs on the ascending and descending limbs) and 2) that the measures were sensitive to changes in intoxication. The cognitive and driving performance measures were selected on the basis of their use in previous studies.

4.1 Method

4.1.1 Participants

Six participants were recruited for the pilot test via posters placed on university notice boards and word of mouth. The six participants, two males and four females, whose ages ranged from 21 to 31 years \( (M = 25.80 \text{ years}, SD = 3.63) \), reported that they had an average of 9.60 years of driving experience (range 5.5-15 years, \( SD = 3.66 \)).

4.1.2 Apparatus

As with the dosage trials, an Alcomate AccuCell AL9000 professional grade breathalyser that automatically converted breath alcohol readings into blood alcohol readings was used to measure the participants' BAC level.

The cognitive performance tasks were presented on an Acer Iconia (W510) touch screen tablet computer. The Cogstate Research software was installed directly from the Cogstate website and configured on the touch screen computer to present the Groton chase task, the Groton maze learning task and the card identification task.

The experimental apparatus was the University of Waikato driving simulator consisting of a complete automobile (BMW 314i) positioned in front of three angled projection surfaces (as shown in figure 4.1). The centre projection surface was located 2.42m in front of the driver's seat with two peripheral surfaces connected to the central surface at 62º angles. The entire projection surface was angled back away from the driver at 14º (from the bottom to the top of the projection surface) and produced a 175º (horizontal) by 41º (vertical) forward view of the simulated roadway from the driver's position. The image projected on the central surface measured 2.64m wide by 2.10m high (at a resolution of 1920 by 1200 pixels) and each of the two peripheral images measured approximately 2.65m by 2.00m (at resolutions of 1024 by 768 pixels). In addition, two colour LCDs with an active area of 12.065cm by 7.493cm each at a resolution of 640 by 480 pixels were mounted at the centre rear-view mirror and driver's wing mirror positions to provide views looking behind the driver's vehicle. The simulated vehicle's dashboard displayed accurate speed and engine RPM data and vehicle performance was determined by a multi-body vehicle dynamics model configured as an automobile with automatic transmission, three litre engine (making 170kW power), and power steering. The projected images and vehicle model were updated at a minimum rate of 100 frames per second. The steering wheel provided tactile feedback to simulate the forces produced when steering the vehicle. Four speakers located inside the car and a sub-woofer underneath the car presented realistic engine and road noises as appropriate. The simulation software recorded the participant's speed, lane position and control actions automatically throughout designated sections of the simulation scenario. A digital video camera was located in the rear seat of the vehicle to record the participants' behaviour during the experimental sessions.

Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml
4.1.3 Simulation scenarios

The simulation scenarios used for this study were based on an 11km-long section of rural road containing a combination of straights and gentle horizontal and vertical curves. The road geometry was an accurate representation of a rural two-lane state highway in New Zealand and was based on the surveyed three-dimensional road geometry of the highway. Lane widths were a constant 3.6m with a 1.7m sealed verge on either side of the edge lines. The road had a posted speed limit of 100km/h with the exception of a 400m section posted with a 60km/h speed limit midway through the road (see figure 4.2). The road contained 12 intersections and at 10 of the intersections stationary cars were positioned to enter the roadway, nine from the driver’s left and three from the driver’s right. At two of the intersections, as the driver reached a point 80m from a vehicle stopped on the left, it moved 2.6m forward (at 1m per second) to partially obstruct the driver’s lane (see figure 4.3).

Twenty oncoming vehicles were placed into the simulation, of a variety of types and colours, all travelling towards the driver at the posted speed limit. Five unique combinations of allocating vehicles to intersections were formed and placed into one of five distinct background scenes to make the roads appear dissimilar. The five experimental roads were then presented in counterbalanced order across participants.
Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml

Figure 4.2 A map of the 11km simulated road used for the pilot test. Waiting cars were positioned at 12 intersections, 10 remaining stationary (shown in green), two moving to partially block the drivers lane (shown in red) as the participants approached.

Figure 4.3 Scene from the simulation scenario with a car waiting at an intersection (top panel) and pulling out 2.6 m as the participant approaches (lower panel).

In addition to the experimental roads, one 12.5km practice road was developed with the same lane width and general configuration as the experimental roads (ie 100km/h speed limit with a 60km/h speed zone in the middle). The first one-third of the road contained no other vehicles in order to allow the participants to concentrate on acquiring the feel of controlling the simulated car. During the rest of the practice road, the number of oncoming vehicles and cars waiting at intersections were gradually increased.
4.1.4 Performance measures

Three tasks from the Cogstate Research software (chase task, Groton maze task and card identification) were administered on the tablet computer (see figure 4.4) to assess the participants’ cognitive performance. The tasks were administered according to the supervisor’s script (provided by Cogstate Ltd) and the on-screen instructions. This computerised test battery was selected due to the standardised administration and the availability of parallel forms which allowed repeated administration without appreciable learning across testing blocks. In addition, the test has good construct validity (Pietrzak et al 2008) and has been used to evaluate the cognitive effects of various psychopharmacological drugs including alcohol (Cromer et al 2010).

Figure 4.4  A participant completing the tablet-based cognitive performance tasks

The first task, the chase task, is typically used as an introduction to the Groton maze and assesses visual motor function. The participant was presented with a 10 x 10 grid of tiles on the computer screen and was instructed to ‘chase the target’. Starting with the tile in the top corner, the participant chased the target as quickly and as accurately as possible by tapping on the tiles. There were two rules associated with the task: the same tile could not be tapped twice and diagonal moves were not allowed. Once the participant understood the rules of the task they repeated the same task, timed over a period of 30 seconds. The primary outcome variable was the number of correct moves per second, where a higher score indicated better performance.

The practice for the Groton maze learning task was the second task in the series. Participants were presented with a 5 x 5 grid of tiles and were instructed to find the hidden pathway from the top left-hand corner to the bottom right-hand corner of the grid by trial and error, with a green tick indicating a correct choice and a red cross, an incorrect choice (the same rules applied as for the chase task). Once this was completed, participants were then presented with an identical 5 x 5 grid twice more and were asked to find the same hidden pathway, after which the practice trial was complete. The full Groton maze learning task requirements were the same as the practice trials, only this time they had to find a 28-step hidden pathway across a 10 x 10 maze, as quickly and as accurately as possible. Once completed, participants were then asked to recall the same route through the maze on four more consecutive trials. The Groton maze learning task assesses executive functions and spatial problem solving with the primary outcome variable being the total number of errors across the five consecutive learning trials (a lower score indicating better performance).
The next task in the series was card identification (a choice reaction time task), where participants were presented with a virtual stack of playing cards on the tablet screen, and when the top card flipped over participants had to indicate by a ‘Yes’ or ‘No’ mouse key press if the card was red. Practice trials were presented until the participant was comfortable with the task requirements. Prior to the real task participants were reminded to respond as quickly and as accurately as they could. This task provided a measure of visual attention and vigilance, the primary outcome variable being the speed of performance calculated as the mean of the log_{10} transformed reaction times for correct responses (a lower score indicated a better performance).

After the card identification task, participants were once again presented with the 10 x 10 Groton maze grid and were asked to recall the previously learnt pathway through the maze. This delayed maze recall task provided an indication of visual learning and memory, as measured by the total number of recall errors. Each administration of the full cognitive testing battery took approximately eight minutes to complete.

The participants' were also asked to complete a series of subjective rating scales during each test block. Subjective intoxication was assessed using a visual analogue scale (Cromer et al 2010). In this scale, participants were asked to respond to the question ‘How intoxicated do you feel right now?’ by placing a mark on a 200mm line centred on a white page. 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 100mm (‘very much’) (Beirness 1987). The Karolinska Sleepiness Scale (Akerstedt and Gillberg 1990) was used to ask participants to rate how sleepy they feel on a nine-point scale ranging from 1 (very alert), 5 (not sleepy or alert) to 9 (very sleepy).

The walk and turn portion of the NZ Police compulsory impairment test (see appendix A for scoring sheet and instructions) was used to assess gross motor function and coordination. 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 (see figure 4.5).

Participants were asked to perform the simulated driving task just as they would drive a real car: following all posted speed limits and following the main road 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 this 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. This embedded detection procedure was adapted from previous studies (Charlton 2006; Charlton and Starkey 2011) in which moving the headlight control (producing a single horn beep) was found to be an effective method for participants to indicate detection of objects and other vehicles.

During the practice drive participants were shown examples of the stationary and moving cars and coached on how to respond: toggling the headlight/horn first, and then braking and steering to avoid any car in their path. The participants’ steering, acceleration, braking and responses to the cars at the intersections were recorded continuously throughout each simulated drive. The following driving measures were calculated for each experimental drive: the standard deviation of the driver’s lane position, the standard deviation of speed, the detection reaction time (measured in seconds) for cars pulling out from the intersection and the brake reaction time in response to cars pulling out.
The measures comprising each test block are summarised in table 4.1 with each test block taking approximately 20 minutes to complete (test block 1 did not include measurements of BAC, walk and turn, or subjective ratings).

Table 4.1 A summary of the tests included in each block

<table>
<thead>
<tr>
<th>Test</th>
<th>Purpose</th>
<th>Duration (minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chase task</td>
<td>Visuo-motor function</td>
<td>1.0</td>
</tr>
<tr>
<td>Groton maze learning task</td>
<td>Visuo-motor function, executive function, spatial problem solving, visual learning, memory</td>
<td>4.5</td>
</tr>
<tr>
<td>Identification task</td>
<td>Visual attention, vigilance</td>
<td>1.0</td>
</tr>
<tr>
<td>Groton maze recall task</td>
<td>Visuo-motor function, executive function, visual learning, memory</td>
<td>1.5</td>
</tr>
<tr>
<td>Breathalyser 1</td>
<td>Measurement of BAC</td>
<td>0.5</td>
</tr>
<tr>
<td>Driving task</td>
<td>Driving performance including lane position, hazard reaction time, false alarms, and driving speed</td>
<td>8.0</td>
</tr>
<tr>
<td>Breathalyser 2</td>
<td>Measurement of BAC</td>
<td>0.5</td>
</tr>
<tr>
<td>Walk and turn (field sobriety test)</td>
<td>Gross motor control and co-ordination</td>
<td>2.0</td>
</tr>
<tr>
<td>Subjective ratings</td>
<td>Perceived intoxication, fitness to drive and feelings of sleepiness</td>
<td>1.0</td>
</tr>
</tbody>
</table>
4.1.5 Procedure

Following the phone screening procedure, participants completed a 40 minute familiarisation session 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 (ie current drink-drive limit, perceived safety when driving after drinking (lunch time and evening), 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. Participants were allowed to repeat the practice drive in the simulator if they so desired. Following successful completion of the familiarisation session, participants were scheduled for a full experimental session (if they agreed to continue) and were given a $10 gift voucher to thank them for their participation thus far. In preparation for their full session, 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.

Prior to arriving in the laboratory for the full session, participants were randomly assigned to one of three dosage groups: placebo, medium (a goal of 0.05% BAC), or high (a goal of 0.08% BAC). Just prior to each full session, three drinks were prepared for each participant based on the following dosages and the participant’s body weight: women received doses of 0.6g/kg or 0.75g/kg (for the medium/0.05% and high/0.08% groups respectively), men received 0.75g/kg or 1.0g/kg (for the medium/0.05% and high/0.08% 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 consisting of orange juice with 5ml of vodka added to the top of the drink.

All participants began the full session with confirmation they had a BAC of zero, a reminder of the test protocol, and another completion of the 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 following completion of the simulated drive the participants were served their first drink and given five minutes to consume it. At the end of the five minutes, a second drink was served, and participants were again given five minutes to finish the drink. Test block 2 began five minutes after the second drink was consumed and from test block 2 onwards, each block consisted of: Cogstate test battery, followed by BAC measurement, followed by simulated drive, followed by another BAC measurement (see figure 4.6), completion of the walk and turn task, completion of subjective ratings of intoxication, momentary 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 (>0.04% or >0.07% depending on their dosage group), in which case the third drink was omitted. The timing of the test blocks was as follows: test block 2 – 15 minutes after first drink was served; optional 3rd drink – 35 minutes after first drink was served; block 3 – 45 mins after first drink was served; test block 4 –1 hour 15 minutes after first drink was served. Test block 5 followed a 55 minute rest break, during the second half of which the participants were allowed to choose from a variety of snack foods. The timeline for the full session is presented in figure 4.7. At the conclusion of the session (completion 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, even if they asked), 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.
4 Pilot test of experimental protocol

4.2 Results of pilot test protocol

The effects of the three alcohol conditions (placebo, medium/0.05% BAC and high/0.08% BAC) were assessed separately for the two participants in each group. The primary driving performance data for the two participants in the placebo group (one male and one female) across the five test blocks are shown in figure 4.8. As can be seen in the figure, horn and brake reaction times measured from the time vehicles moved into their lane, remained very consistent across the five test blocks. Similarly, the participants’ steering as measured by the SDLP also remained very consistent.

Figure 4.9 shows the same measures collected for the two participants (one male and one female) in the medium alcohol dose group along with their BAC levels associated with each of the five test blocks. As can be seen in the figure, although horn reaction times and steering showed only small changes as the participants’ BAC levels increased, in the later test blocks the participants began to fail to press the brake pedal after sounding their horn for vehicles moving into their lane from the intersection (shown as missed brake in the figure). The BAC levels for these two participants came close to the target of 0.05% for this dose of alcohol, peaking at 0.043% BAC in test block 2 for one participant and at 0.05% in test block 5 for the other.
Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml

Figure 4.8  Driving performance for the two participants in the placebo condition. The male participant’s performance is shown in the left panel and the female participant’s in the right panel.

HRT = horn reaction time; BRT = brake reaction time

Figure 4.9  Driving performance and BAC levels for the two participants in the medium alcohol (target 0.05%) condition. The missed brake measure refers to occasions when a participant failed to brake for a car moving into the roadway.

Figure 4.10 shows the driving performance and BAC levels for the two participants (both female) receiving the high alcohol dose. One of the participants reached a peak BAC of 0.066% at test block 2, and the other recording a peak of 0.069% during test block 4. For one participant, brake reaction times appeared to decrease gradually over the course of the experiment, whereas the other participant showed an increase in horn reaction time immediately after consuming the alcohol, followed by progressively faster horn reaction times in subsequent test blocks. SDLPs showed some increase in steering variability associated with consuming the alcohol, but then declined to baseline levels in the later test blocks.

For the cognitive tasks, the two participants in the placebo condition (see figure 4.11) showed little variability in their performance across the five test blocks for the moves per second on the chase task, speed of card identification and the number of maze learning and recall errors.
Figure 4.10  Driving performance and BAC levels for the two female participants in the high alcohol (target 0.08%) condition

Figure 4.11  Cognitive test performance of the two participants in the placebo group. The male participant’s performance is shown in the left panel and the female participant’s in the right panel. The upper panel shows the participants’ performance on the chase and card identification task and the lower panel shows the number of errors on the Groton maze tasks.

The data from the participants in the medium alcohol group can be seen in figure 4.12. Speed of card identification remained stable for both participants across the varying of BACs. In contrast, for the male participant the number of moves per second on the chase task increased with rising BAC, whereas the female participant’s performance on this task was slowest when her BAC reached its peak. For the Groton
maze, the number of learning and recall errors increased for both participants in line with their increasing BACs.

Figure 4.12 Cognitive task performance and BAC for the two participants in the medium alcohol group. The male participant’s performance is shown in the left panel and the female participant’s in the right panel. The upper panel contains data for the chase and card identification task, the lower panel shows the number of errors on the Groton maze task.

For the participants in the high alcohol group, BAC did not appear to affect performance speed on the card identification task (figure 4.13), nor was there a clear relationship between BAC and moves per second on the chase task. The total errors on the Groton maze learning and recall task (presented in the lower left panel) show a high degree of variability across the test blocks, which does not appear to be related to BAC; however, data from the other participant (lower right panel) suggests that the number of maze learning and recall errors increased and decreased in line with the participant’s BAC.

From test block 2 onwards, participants also provided ratings of their subjective intoxication, their sleepiness and their willingness to drive. Ratings from the two participants in the placebo group at each test block are presented in figure 4.14. The ratings of subjective intoxication for both participants were above zero (0 indicates least intoxicated) for test blocks 2, 3 and 4, suggesting that neither participant had realised they were in the placebo group. The willingness to drive ratings mirrored to some extent the ratings of subjective intoxication (a score of 0 on this scale indicates they would not be willing to drive); for both participants their willingness to drive increased as the level of subjective intoxication decreased. These two participants’ ratings of sleepiness did not exceed the midpoint (not sleepy or alert) for any of the test blocks and did not appear to be related to levels of subjective intoxication.
Figure 4.13  Cognitive task performance and BAC of the two female participants receiving the high alcohol dose. The upper panel contains data for the chase and card identification task, the lower panel shows the number of learning and recall errors on the Groton maze task.

Figure 4.14  The subjective ratings of the participants in the placebo group across test blocks.

The subjective ratings and BACs of the two participants in the medium alcohol group can be seen in figure 4.15. For the male participant (left panel), his increase in BAC between test blocks 2 and 3 was accompanied by an increase in subjective intoxication, and a decrease in willingness to drive; however, as his BAC continued to climb to just above 0.05%, his ratings of subjective intoxication decreased and willingness to drive increased. In contrast, his sleepiness increased along with his BAC. The pattern for the female participant was somewhat different. The drinks she consumed rapidly produced a peak BAC of around 0.045%. Her ratings of subjective intoxication were highest as her BAC began to fall in test block 4, and as her BAC decreased, her willingness to drive increased. Her levels of sleepiness increased across the test blocks.
Figure 4.15  The subjective ratings of the participants in the medium alcohol group across test blocks. The male participant's performance is shown in the left panel and the female participant's in the right panel. The top panel presents data for BAC, subjective intoxication and willingness to drive, the lower panel shows ratings of sleepiness in relation to BAC and test block.

Ratings provided by the two female participants in the high alcohol group are presented in figure 4.16. The ratings from the participant in the left panel show that as her BAC increased, her level of subjective intoxication increased and her willingness to drive decreased; however, by test block 5 her subjective intoxication was very low even though her BAC was still above 0.05%. This participant’s sleepiness ratings showed an increase across test blocks. The data from the other participants shows that her subjective ratings of intoxication and her sleepiness followed a similar pattern to her BAC; however, her willingness to drive was consistently very low.

Although the six pilot participants completed the walk and turn test after each simulated drive, the scoring criteria used in New Zealand were not available to the researchers at the time the pilot testing was conducted, therefore the data is not presented here (but is included for the full experiment).
4.3 Conclusions

Overall, the results from the pilot test of the experimental protocol were promising. In terms of the alcohol dosing protocol, the ratings of subjective intoxication provided by the placebo group participants suggested the drink preparation meant they were unaware of being assigned to the placebo group, and the BACs achieved by the participants in the medium and high alcohol dose groups were within the target range. The length and timing of the test blocks were acceptable to participants and did not place them under undue pressure while the timing was also feasible for the researchers, allowing data collection on the ascending and descending limbs of the BAC curve. In terms of sensitivity of the measures, for the driving performance data, horn and brake reaction time appeared the most promising in terms of sensitivity to the effects of alcohol. For the cognitive performance measures, errors on the Groton maze learning and recall tasks seemed to be most sensitive. The subjective ratings seemed to be suitable and for some participants there was evidence of a mismatch between their actual BAC and their perceived level of intoxication. On the basis of these findings, a decision was made to retain the cognitive measures and subjective ratings unchanged for the full study, but to make the simulated drive more challenging with the addition of more cars at intersections. At the intersections, cars would either: remain stationary, pull out into the path of the oncoming vehicle and require evasive action (as in the current scenarios), or pull out slightly, but not enough to require the driver to take evasive action (termed false alarms). The participants were instructed to only respond to a real hazard (i.e., a car that pulls into their path, a ‘go’ stimulus) which required evasive action, and inhibit a response to the cars that moved forward slightly as they approach (a ‘no-go’ or false alarm stimulus). This task mimics the computerised go/no-go tasks which have been
Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml

found to be sensitive to the effects of alcohol consumption (eg. Weafer and Fillmore 2012). In addition, in order to minimise learning effects, all participants were also asked to complete the practice drive again at the start of the full experimental session (in addition to the familiarisation session), before baseline testing.
5 Full experiment

The pilot test demonstrated that the alcohol administration protocol produced intoxication curves that allowed examination of the ascending and descending limbs using the range of test measures chosen for the experiment. The test measures themselves appeared to be fairly robust and could be conducted in a timely fashion, with minor changes to the driving simulation scenarios. The goal of the full experiment was to explicitly compare the performance and subjective effects associated with peak BACs of 0.05% and 0.08% during the ascending and descending limbs of the BAC curve.

5.1 Method

5.1.1 Participants

One hundred and thirty-four potential participants contacted the researchers for further information about the study. Of these, 23 participants did not meet the eligibility criteria (as described earlier in chapter 2) and were excluded after the telephone screening process, 34 withdrew prior to their first familiarisation session, 13 withdrew after the familiarisation session (mainly due to feelings of discomfort in the driving simulator), and three male volunteers were declined by the researchers as sufficient male participants had been recruited. Sixty-one participants took part in the full experiment.

The 61 participants (33 male, 28 female) recruited for the full 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, seven self-identified as Māori and nine were of ‘other’ ethnicity (including British, Dutch, Chinese and Indian). A third (n = 20, 32.8%) of the participants were married, 11 (18%) were in de-facto relationships, 26 (42.6%) were single and four (6.6%) were separated or divorced.

In terms of their driving history, all participants held full New Zealand driving licences for 14.03 years on average (SD = 8.19). In the previous 12 months, four participants had been involved in a crash and 14 had received at least one traffic infringement notice (including parking tickets). Most of the participants reported that they most often drove a midsize car (n = 40, 65.6%), 13 (21.3%) drove a compact car, five (8.2%) drove a van or ute, two (3.3%) rode a motorbike, and one (1.6%) participant reported that they usually drove a truck.

5.1.2 Apparatus

The experimental apparatus, the Alcomate AccuCell AL9000 breathalyser, Cogstate test battery and the University of Waikato driving simulator described for the pilot test remained the same for the full experiment.

5.1.3 Simulation scenarios

As mentioned in the conclusions from the pilot test (see section 4.3) the simulation scenarios were changed somewhat for the full test protocol. The 11km section of simulated road was modified to include a total of 20 intersections with vehicles waiting (a 21st intersection located in the 60km/h zone never contained a vehicle). At 10 intersections there were stationary vehicles; five located on the left side, two located on the right, and three intersections where there were vehicles located on both the left and right side of the intersection (see figure 5.1). The first intersection of the simulated drive always contained one or more of the stationary vehicles (a car on the left, right or a double). At six of the intersections cars moved into the driver’s lane from the left (as described for the pilot test), but at two of these intersections there were cars waiting on both sides of the roadway, although only the car on the left moved. There were
also four false alarm vehicles which moved 0.8m in 1 second when the driver was 70m away, to a point past the marked limit line, but not obstructing the driver’s lane (shown in figure 5.2). Three of these false alarm vehicles were located on the left side of the intersection, one of them paired with another stationary vehicle waiting on the right side of the intersection, and the fourth was positioned on the right. As with the simulated roads developed for the pilot test, five unique combinations of intersections and vehicles were formed and placed into one of five distinct background scenes to make the roads appear dissimilar. The five experimental roads were then presented in counterbalanced order across participants.

The practice road remained the same as in the pilot test except it included examples of each type of intersection, stationary cars, false alarms and cars moving into the driver’s lane from the left. As before, the participants were coached in how to respond to the vehicles, sounding the horn and braking only for vehicles moving into their lane and making no response to the false alarm vehicles.

Figure 5.1 An example scene from the simulation scenario modified to include waiting cars on both sides of the roadway. Note that only cars from the left side moved into the roadway

Figure 5.2 An example scene showing a false alarm car waiting at the intersection (top panel) and moving 0.8m ahead as the participant approaches (lower panel)
5.1.4 Performance measures

The same cognitive performance test battery used for the pilot test was again used for the full experiment (please refer to table 4.1). The measures of driving performance, however, were changed slightly to reflect the changes to the simulation scenarios and capture some additional aspects of the effects of alcohol on driving. Additional driving performance measures collected included: the participants’ maximum speed, the number of seconds spent driving over 100km/h, the number of times the participants steered across the centre line of the simulated road, the number of seconds the participants spent with their wheels across the centre line of the road, the number of edge line crossings, the number of seconds spent over the edge line, average speed in the 60km/h zone, the number of times participants failed to indicate detection of a car pulling out, the number of times participants failed to brake in response to a car pulling out and the number of times participants responded to false alarm vehicles.

The measures of subjective intoxication, willingness to drive and sleepiness all remained the same as in the pilot test. Participants also completed the walk and turn part of the Police compulsory impairment test after each test block drive. One additional question was added to the participant debriefing at the end of the experiment: at the end of the session each participant was asked an open-ended question requesting them to explain factors that influenced their responses to the willingness to drive scale.

5.1.5 Procedure

Participant recruitment and the familiarisation session remained the same as for the pilot test; however, when participants arrived at the laboratory for the full session, they were asked to complete another practice drive in the simulator before baseline testing (test block 1) began. After this, the alcohol administration protocol, the timing and order of the test blocks, and participant remuneration all remained the same as that described for the pilot test.

5.2 Results

In order to address all of the research goals, the data was examined using three complementary analysis methods. The first of these was to compare the performance changes produced by the two doses of alcohol to the placebo group (the effect of alcohol dose) over the time course of each experimental session. The second analysis was directed at a more select comparison of the two BAC levels of interest (0.08% and 0.05%) using the simulated driving and cognitive performance measures, once again comparing them to the performance of the placebo group on these measures (the effect of the BAC level). This analysis is important given the range of individual differences in the BACs resulting from a given dose of alcohol. Finally, to compare the full range of BACs that would result from any change to the adult legal BAC limit, and to assess the differences associated with ascending and descending portions of the intoxication curve (becoming intoxicated versus recovering from intoxication), an analysis based on BAC thresholds was conducted. This analysis is important because in naturalistic settings drivers will typically manage their consumption in order to remain below the legal BAC threshold (rather than drink to reach a target limit), and prior research has shown that there are important differences in impairment produced during the ascending and post-peak stages of intoxication. These three distinct analysis approaches are described separately in the sections below.

5.2.1 Effect of alcohol dose

The differences between the three alcohol dose groups were assessed separately for the driving performance measures, the embedded detection task and the cognitive performance tasks. The mean BAC levels associated with each alcohol dose group across the five test blocks are shown in figure 5.3, along
with the primary driving performance data. As can be seen in the figure, the dosage groups displayed considerably different BAC levels, peaking at test block 3. A series of univariate analyses of variance (ANOVAs) revealed that the BACs of the three dose groups were significantly different at test block 2 \( F(2, 58) = 108.52, p < .001, \eta^2_p = .789 \), test block 3 \( F(2, 58) = 242.55, p < .001, \eta^2_p = .893 \), test block 4 \( F(2, 58) = 166.86, p < .001, \eta^2_p = .852 \) and test block 5 \( F(2, 58) = 93.43, p < .001, \eta^2_p = .766 \). For each test block, post hoc pairwise comparisons (using the Bonferroni adjustment for multiple comparisons) indicated that the alcohol group differences were significantly different from one another \((p < .01)\). Because of the participants’ weight differences, and due to the dose titration methodology in which participants only received a third drink if needed, not all of the participants in each group consumed the same amount of alcohol. On average, the participants in the medium dose group received the equivalent of 4.78 \((SD = 1.43)\) standard drinks and those in the high dose group received an average of 7.01 \((SD = 2.86)\) standard drinks. All of the participants in the placebo group received the same amount of alcohol: 0.5 of a standard drink.

Also shown in the figure are the driving performance measures collected at each alcohol dose. As can be seen, driving performance throughout the test period was clearly affected by the alcohol dose received, with the placebo group having lower maximum speeds and the lowest number of centre line and edge line crossings. A multivariate analysis of variance indicated that during test block 1 there was no statistically reliable difference between the dosage groups across the full range of driving performance measures collected \([Wilks’ lambda = .553, F(22, 96) = 1.51, p = .090, \eta^2_p = .257]\). Immediately after consuming the first two drinks (test block 2), however, a significant difference in driving performance was observed \([Wilks’ lambda = .449, F(22, 96) = 2.15, p = .006, \eta^2_p = .330]\). Univariate analyses of the test block 2 driving performance measures showed significant group differences for the number of centre line crossings \(F(2, 58) = 7.19, p = .002, \eta^2_p = .199\), the number of seconds spent over the centre line \(F(2, 58) = 3.96, p = .024, \eta^2_p = .120\) and the number of seconds spent over the edge line \(F(2, 58) = 3.38, p = .041, \eta^2_p = .104\). The group means for maximum speed, the number of edge line crossings, the standard deviation of lane position, average speed and the other driving performance measures were not statistically different. The participants’ gender was also entered as a variable for the analyses (and the ones to follow), but after verifying there were no statistically reliable gender differences, gender was removed from consideration in the statistical models.

Post hoc pairwise comparisons (Bonferroni adjusted) indicated that the placebo group was reliably lower \((p < .05)\) than the two alcohol groups for three measures (centre line crossings, seconds over centre line and seconds over edge line) but for the seconds over edge line measure the placebo group and high dose group did not differ sufficiently to meet the Bonferroni adjustment threshold. The medium and high dose groups were not reliably different on any of the three performance measures.

A similar analysis was performed on the driving performance measures at test block 3 (after the second alcohol dose, for those requiring one), and it too indicated a statistically reliable difference between the dosage groups across the driving performance measures \([Wilks’ lambda = .468, F(22, 96) = 2.01, p = .011, \eta^2_p = .316]\). The univariate analyses at test block 3 showed significant group differences for the number of centre line crossings \(F(2, 58) = 6.73, p = .002, \eta^2_p = .188\), the number of seconds spent over the centre line \(F(2, 58) = 5.16, p = .009, \eta^2_p = .151\), the number of edge line crossings \(F(2, 58) = 4.85, p = .011, \eta^2_p = .143\) and the number of seconds spent over the edge line \(F(2, 58) = 3.32, p = .043, \eta^2_p = .103\). In addition, the participants’ maximum speed during the drive was significantly different for the three groups \(F(2, 58) = 4.53, p = .015, \eta^2_p = .135\), as was their lane position variability (SDLP) during the 400m reduced speed section \(F(2, 58) = 4.53, p = .015, \eta^2_p = .135\). The group means for SDLP throughout the entire drive, average speed during the drive, and the other driving performance measures were not statistically different.
Post hoc pairwise comparisons (Bonferroni adjusted) indicated that the placebo group was reliably lower ($p < .05$) than the two alcohol groups for the centre line and edge line crossings and significantly lower than the medium dose group for the seconds over the centre line measure. The high dose group had significantly higher maximum speeds than the placebo and medium dose groups, which did not differ from one another.

The significant differences between the groups persisted during the later test blocks; during block 4 the number of centre line crossings [$F(2, 57) = 4.25, p = .019, \eta^2_p = .130$], the number of edge line crossings [$F(2, 57) = 3.51, p = .036, \eta^2_p = .110$], the number of seconds spent over the edge line [$F(2, 57) = 3.59, p = .034, \eta^2_p = .112$], the maximum speed during the drive [$F(2, 57) = 4.17, p = .020, \eta^2_p = .128$] all showed
significant differences, and the number of seconds spent over the centre line was marginally significant \(F(2, 57) = 3.01, p = .057, \eta^2_p = .095\). During block 5 the number of centre line crossings \(F(2, 56) = 4.02, p = .023, \eta^2_p = .125\), the number of seconds spent over the centre line \(F(2, 56) = 3.75, p = .030, \eta^2_p = .118\), the number of edge line crossings \(F(2, 56) = 3.74, p = .038, \eta^2_p = .110\), the maximum speed during the drive \(F(2, 56) = 4.85, p = .011, \eta^2_p = .148\), the number of seconds spent over 100km/h \(F(2, 56) = 4.65, p = .014, \eta^2_p = .142\) and the maximum speed in the 400m reduced speed area \(F(2, 56) = 3.32, p = .044, \eta^2_p = .106\), all showed significant differences between the groups. Pairwise comparisons of the means showed that the two alcohol groups were typically significantly different from the placebo group \((p < .05)\) but were never significantly different from each other.

The participants' performance on the embedded detection task (their responses to cars at intersections) was analysed separately to the above driving performance measures. A multivariate analysis of variance across the full range of measures associated with the detection task did not reveal any differences between the groups during block 1 \([\text{Wilks' lambda} = .760, F(8, 98) = 1.81, p = .085, \eta^2_p = .128]\). After consuming the first two drinks there was a significant multivariate difference between the groups at test block 2 \([\text{Wilks' lambda} = .653, F(8, 98) = 2.91, p = .006, \eta^2_p = .192]\) and block 3 \([\text{Wilks' lambda} = .717, F(8, 100) = 2.27, p = .029, \eta^2_p = .153]\). The univariate analysis, however, revealed that the only measure of detection task performance that showed a reliable difference between the dose groups was the average number of false alarms (where the participants sounded their horn for the distractor vehicles that moved slightly but did not enter their driving lane). None of the other measures of the detection task performance such as horn reaction time, brake reaction time, failure to brake, or reaction time variability showed any statistically reliable differences. Figure 5.4 shows the average number of false alarms for each dosage group across the five test blocks and as can be seen, the effect of alcohol apparently made it difficult for drivers to resist reacting to the false alarm vehicles. For test block 1 the univariate result for participants' false alarms was \(F(2, 55) = 0.27, p = .764, \eta^2_p = .010\) indicating no difference between the groups. After consuming alcohol (test block 2), however, there was a significant difference between the groups \(F(2, 55) = 6.62, p = .003, \eta^2_p = .194\). Similarly, for test block 3 the result was \(F(2, 55) = 4.78, p = .012, \eta^2_p = .148\), for block 4 the result was \(F(2, 55) = 1.62, p = .207, \eta^2_p = .056\), and for block 5 it was \(F(2, 55) = 6.12, p = .004, \eta^2_p = .182\). The post hoc pairwise comparisons (Bonferroni adjusted) indicated that the high dose group had significantly more false alarms at blocks 2, 3 and 5 compared with the placebo group (but the differences between the medium and high groups and the placebo and medium groups did not meet the Bonferroni adjustment criterion).

Figure 5.4 The average number of false alarms for each dosage group across the five test blocks

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<table>
<thead>
<tr>
<th>Test blocks</th>
<th>Placebo</th>
<th>Med (0.05)</th>
<th>High (0.08)</th>
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<tbody>
<tr>
<td>1</td>
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<tr>
<td>5</td>
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Mean false alarms

0 0.2 0.4 0.6 0.8 1.0 1.2

1 2 3 4 5

Test blocks
The three alcohol doses also resulted in differences in the participants’ performance on the cognitive tests (shown in figure 5.5), with the placebo group performing the tasks most quickly and with fewest errors. The cognitive performance data was analysed using the same method as the driving performance measures, with multivariate analyses of the cognitive tests’ primary outcome variables carried out separately for each test block. As with the driving performance data, the analyses were initially conducted with the participants’ gender included as a variable; however, as there were no statistically significant gender effects, gender was removed from the statistical models.

Figure 5.5  Cognitive test performance across the five test blocks for each alcohol dose group

There were no significant differences between the three alcohol dose groups at test block 1, (prior to alcohol consumption) for any of the cognitive performance measures. For test block 2 (post-drink) a multivariate analysis of variance revealed a significant difference between the dose groups [Wilks’ lambda = .706, $F(8, 110) = .2.61, p = .012, \eta^2_p = .160$]. The univariate analyses showed significant group differences for the number of maze recall errors [$F(2,58) = 5.49, p = .007, \eta^2_p = .159$] and speed of performance in the card identification task [$F(2,58) = 4.59, p = .014, \eta^2_p = .137$], but the group means for performance on the chase task and total errors on the learning task were not statistically different. In test block 3 the multivariate analysis failed to show a reliable difference across the groups based on participants’ performance in all four cognitive tests [Wilks’ lambda = .801, $F(8, 110)= 1.61, p = .130, \eta^2_p = .105$]; however, as in test block 2, the univariate analyses indicated a statistically significant difference between the dose groups for maze recall errors [$F(2,58) = 3.62, p = .033, \eta^2_p = .111$] and speed in the card identification task [$F(2,58) = 4.10, p = .022, \eta^2_p = .124$]. There was a statistically significant difference between the groups for cognitive task performance for test block 4 [Wilks’ lambda = .689, $F(8, 110)= 2.81$,
Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml

\[ p = .007, \eta_p^2 = .170 \] with the total number of maze learning errors \[ F(2, 58) = 3.57, p = .034, \eta_p^2 = .110 \] and recall errors \[ F(2, 58) = 8.29, p = .001, \eta_p^2 = .222 \] showing a reliable difference across the three alcohol dosage groups. For the final test block (5), there were no statistically reliable differences between the groups.

The post-hoc comparisons (Bonferroni corrected) showed that in test blocks 2, 3 and 4 the group receiving the high alcohol dose made significantly more errors recalling the route through the maze compared with the placebo group \( (p < .05) \). During block 4, the high dose participants’ maze recall errors were significantly higher than the medium dose group as well \( (p < .05) \), and their maze learning errors were significantly higher than the placebo group \( (p < .05) \). There were no statistically significant differences between the placebo and medium dose groups for maze learning or recall errors for any test block. For the card identification task in blocks 2 and 3, the response speed of the placebo group was significantly faster than the medium dose group \( (p < .05) \); however, the performance of the high alcohol dose group did not differ significantly from the placebo group.

5.2.2 Effect of the BAC level

Although the alcohol doses described above produced reliable differences in average BAC levels, the participants displayed a range of individual differences in their BAC levels (not every participant hit their ‘target’ BAC). In order to remove some of the variability from the analyses and more clearly assess the differences between 0.05% and 0.08% BAC levels, the participants’ data was classified according to their observed BAC levels (as opposed to their alcohol dose). Participants’ BAC levels during test blocks 2 and 3 were examined and the participants were assigned to one of three BAC groups: placebo, medium (a BAC level of 0.04% to 0.06%), or high (a BAC level of 0.07% to 0.09%).

BAC levels during block 2 were examined first and, where possible, participants were assigned to groups on that basis. BAC levels during block 3 were then considered and group assignments were revised to most closely match the desired BAC ranges. Based on this process, 51 participants were assigned to one of the three BAC groups using data from either their second or third test block (the 10 participants whose BAC levels fell outside the desired ranges were excluded for the purposes of this analysis). Sixteen participants were classified as being in the medium BAC range (10 during block 2 and six during block 3), and 15 were classified as being in the high BAC range (eight from block 2 and seven from block 3). All 20 participants receiving the placebo dose remained in the placebo BAC group and their block 2 data was used in the analyses. (Note that this analysis was fully between-subjects; participants were assigned so that their data contributed to only one of the BAC groups, ie block 2 and 3 data from any one participant could not be used for different BAC groups).

A multivariate analysis of variance indicated a statistically reliable difference between the BAC groups across all of the driving performance measures \[ \text{Wilks' lambda} = .347, F(22, 76) = 2.41, p = .003, \eta_p^2 = .411 \]. The larger effect size resulting from this analysis (compared with the analyses based on dose groups) provides evidence of the reduction in variability achieved by forming groups based on BAC levels. Univariate analyses of the driving performance measures showed significant BAC differences across a wide range of driving performance measures: the number of centre line crossings \[ F(2, 48) = 10.62, p < .001, \eta_p^2 = .307 \], the number of seconds spent over the centre line \[ F(2, 48) = 9.16, p < .001, \eta_p^2 = .276 \], the number of edge line crossings \[ F(2, 48) = 4.15, p = .022, \eta_p^2 = .148 \], the number of seconds spent over the edge line \[ F(2, 48) = 3.62, p = .034, \eta_p^2 = .131 \] and for the participants’ maximum speed \[ F(2, 48) = 4.21, p = .021, \eta_p^2 = .149 \].

Figure 5.6 shows the effects of BAC for these variables as well as the false alarm measure from the embedded detection task. For this latter measure (average number of false alarms), the univariate analysis
showed a significant difference between the BAC groups \( F(2, 44) = 3.54, p = .038, \eta^2_p = .139 \). None of the other detection task measures were associated with significant differences between the BAC groups. In general, a BAC of 0.04% to 0.06% had an intermediate effect between the placebo and high BAC conditions for the same measures indicated by the analysis based on dose groups. The only exception to this pattern was for the measure of maximum speed, where the medium BAC group displayed a lower peak speed than either the placebo or high BAC groups.

**Figure 5.6** Participants’ driving performance as a function of their BAC level. Lines indicate 95% confidence intervals.

Pairwise comparisons (Bonferroni adjusted) showed that the two alcohol groups were significantly different from the placebo group \( (p < .01) \) but not from one another for the number of centre line crossings and seconds over the centre line. For the number of edge line crossings and seconds spent over the edge line, however, the medium BAC group was not significantly different from the placebo group; only the high BAC group was significantly worse than the placebo group \( (p < .05) \). This was the pattern for the average number of false alarms as well – the placebo group had significantly fewer than the high BAC
group ($p = .045$), but the medium BAC group did not differ from either the placebo or high BAC groups. For maximum speed, the medium BAC group was significantly lower than the high BAC group, but was not significantly lower than the placebo group. The placebo and high BAC groups did not differ reliably from one another.

The participants’ performance on the cognitive tasks as a function of their BAC level is presented in figure 5.7. Generally the placebo group performed the tasks faster and with fewer errors than either the medium or high BAC group. For the maze learning and maze recall errors there is also some evidence of BAC-dependent decrements in performance, with errors increasing linearly with BAC levels. Multivariate analysis of the cognitive performance tasks’ primary outcome variables indicated a statistically significant difference between the three BAC groups [Wilks’ lambda = .640, $F(8, 90)= 2.81$, $p = .008$, $\eta^2_p = .200$]. The univariate analyses showed significant differences between the groups for maze learning errors [$F(2,48) = 6.76$, $p = .003$, $\eta^2_p = .220$], maze recall errors [$F(2,48) = 9.44$, $p < .001$, $\eta^2_p = .282$] and speed of response in the card identification task [$F(2,48) = 3.17$, $p = .051$, $\eta^2_p = .117$], but not for the chase task. Bonferroni corrected post hoc pairwise comparisons showed that the placebo and medium BAC groups made significantly fewer errors than the high BAC group on the maze learning and maze recall tasks ($p <.05$). There were no statistically significant differences between the placebo group and the medium BAC group. The pairwise comparisons for card identification speed showed no reliable differences between the three BAC groups, presumably as a result of the higher Bonferroni criterion for multiple comparisons.

Figure 5.7 Participants’ cognitive test performance as a function of BAC level. Lines indicate 95% confidence intervals

5.2.3 BAC threshold analysis

In addition to comparing participants’ performance based on the dosage group they were assigned to and the BAC levels resulting from the alcohol dose received, another important way to view the results is to
consider how any legislative reduction in BAC limits might be reflected in the driving performance exhibited on the roads. In contrast to the experimental protocols used in the present study, drivers do not consume alcohol to achieve a specific BAC level, instead most drivers will typically manage their consumption in order to remain below the legal BAC threshold. Thus it is an interesting question to ask how reducing the threshold to 0.05% would differ from the performance resulting from the current threshold of 0.08% BAC.

In order to address that question, two new groupings of the participants’ data were formed based on whether their ascending or peak BAC level was 0.055% or below (as it would be if drinking to a new enforced limit of 0.05%), or whether it was between 0.056% and 0.085% (a range intended to correspond to those exceeding the new limit but at or under the currently enforced limit of 0.08%). Because drivers often cease drinking for a time prior to driving, it was also of interest to examine performance associated with post-peak BAC levels (ie on the descending limb of the intoxication curve) across these two ranges. Although there would be more variability inherent as a result of the wider range of BACs contained in these ranges, the approach was intended to reflect how many people currently respond to the enforced limit, managing drinking to remain under a target or threshold BAC level, and sometimes delaying their driving until their BAC falls below that threshold.

Using a similar procedure as the one described for the BAC analysis, participants’ peak or ascending BAC during block 2 (or block 3 if needed for a closer approximation) was used to classify them into the BAC threshold groups (five participants whose BAC exceeded 0.085% by block 2 were excluded from this analysis). Similarly, participants’ performance during block 4 (or 5) was grouped according to whether their post-peak BAC during these latter stages was 0.055% or below or whether it was between 0.056% and 0.085% (seven participants whose BACs peaked during blocks 4 or 5 or who still exceeded 0.085% by block 4 were excluded from this analysis). Using this grouping procedure, 17 participants were allocated to the group with an ascending or peak BAC below the 0.055% threshold (using block 2 data for 12 and block 3 data for five of them), and 19 participants were allocated to the group with a BAC of below the 0.085% threshold (using block 2 data for 13 and block 3 data for six of them). For the post-peak threshold groupings, 17 participants were allocated to the below 0.055% group (using block 4 data for 13 of them) and 17 participants were allocated to the below 0.085% group (using block 4 data for nine of them). It should be noted that the participants were not necessarily in the same group (medium or high) for the ascending and post-peak BACs.

Figure 5.8 shows the driving performance of these two new ascending/peak and post-peak threshold groupings compared with the placebo group’s performance (during block 2 for comparison to peak BACs and block 4 for post-peak). As can be seen in the figure, for some measures post-peak performance for the alcohol threshold groups was actually worse than their performance at ascending BACs, even though the BAC levels at these two times were categorised as equivalent (medium: ascending BAC $M = .047 SD = .008$, descending BAC $M = .050 SD = .005$; high: ascending BAC $M = .073 SD = .008$, descending BAC $M = .066 SD = .013$).

Multivariate analysis of variance of the driving performance measures indicated a significant difference between the threshold groups during ascending/peak BACs [Wilks’ lambda = .469, $F(22, 86) = 1.80$, $p = .029$, $\eta^2_p = .262$] and for performance during post-peak BACs [Wilks’ lambda = .395, $F(22, 80) = 2.37$, $p = .004$, $\eta^2_p = .372$]. Univariate results showed significant group differences at ascending/peak BACs for the number of centre line crossings [$F(2, 53) = 9.40$, $p < .001$, $\eta^2_p = .262$], the number of seconds spent over the centre line [$F(2, 53) = 7.87$, $p = .001$, $\eta^2_p = .229$], the number of edge line crossings [$F(2, 53) = 4.16$, $p = .021$, $\eta^2_p = .136$] and for the number of seconds spent over the edge line [$F(2, 53) = 3.70$, $p = .031$, $\eta^2_p = .123$], but not for maximum speed or the other driving performance measures. Post-peak performance showed significant group differences for centre line crossings [$F(2, 49) = 3.35$, $p = .043$, $\eta^2_p = .120$], the
number of edge line crossings \([F(2, 49) = 5.56, p = .007, \eta^2_p = .185]\), for the number of seconds spent over the edge line \([F(2, 49) = 6.22, p = .004, \eta^2_p = .202]\), as well as for maximum speed \([F(2, 49) = 4.51, p = .016, \eta^2_p = .156]\) and seconds spent driving over 100km/h \([F(2, 49) = 4.44, p = .017, \eta^2_p = .154]\, not included in figure 5.8), but not for seconds spent over the centre line or any other driving performance measure.

Figure 5.8  Participants' driving performance as a function of BAC threshold category: ascending/peak/BAC (test block 2 or 3) and descending/post-peak (test block 4 or 5). Lines indicate 95% confidence intervals

The above analysis by BAC threshold shows a similar set of significant effects on the driving performance measures to the earlier analyses by dose group and BAC level. Closer inspection of the pairwise differences, however, reveals some interesting differences between the two BAC thresholds. In contrast to the earlier analyses where only the high alcohol group was typically significantly different from the placebo group while the medium group was intermediate between them (but not statistically different from either), this comparison based on BAC thresholds indicates that the performance of the medium alcohol group was similar to the placebo group when measured during the ascending/peak BAC phase, but closer to the high BAC threshold group when measured post-peak.
During the ascending/peak BAC phase, the placebo group was significantly better ($p < .05$) than the high threshold group in terms of the centre and edge line crossings measures (both number and time over the line), but was better than the medium threshold group only for the number of centre line crossings (the medium threshold group did not differ from the high threshold group for any of these measures). During the descending phase the placebo group was still significantly better than the high threshold group on all of the above measures except seconds spent over the centre line and did not differ significantly from the medium threshold group on any of the above measures. The medium threshold group displayed an average maximum speed that was significantly higher than the placebo group during the post-peak phase ($p = .013$), and the high threshold group on average spent more time over the 100km/h speed limit than the placebo group during the post-peak phase ($p = .014$).

An exception to this pattern can be seen in the results from the embedded detection task. Multivariate analysis of the detection task measures indicated a significant difference between the threshold groups during the peak BAC phase [Wilks’ lambda = .569, $F(14, 84) = 1.95$, $p = .032$, $\eta^2_p = .245$] and during the post-peak phase [Wilks’ lambda = .503, $F(12, 78) = 2.67$, $p = .005$, $\eta^2_p = .291$]. Once again, for the ascending BAC phase the only measure showing significant effects was the average number of false alarms [$F(2, 48) = 4.02$, $p = .024$, $\eta^2_p = .143$]. During the post-peak phase, however, in addition to the significant difference in the average number of false alarms [$F(2, 44) = 6.26$, $p = .004$, $\eta^2_p = .222$], the standard deviation of reaction times (to beep the horn) was also significantly different across the three groups [$F(2, 44) = 3.41$, $p = .042$, $\eta^2_p = .134$] (not pictured in figure 5.8). Pairwise comparisons (Bonferroni adjusted) indicated that the placebo group was significantly lower than the high BAC threshold group during the ascending BAC phase ($p = .021$) for the average number of false alarms, but the medium threshold group was not significantly different from the other two groups. During the post-peak phase the medium and placebo groups were both significantly lower than the high BAC threshold group ($p < .05$) for the false alarm measure. For the standard deviation of horn reaction times, the placebo group was significantly lower than the high BAC threshold group during the post-peak BAC phase ($p = .051$) but the medium threshold group was not significantly different from the other two.

The participants’ cognitive test performance based on BAC threshold categories is presented in figure 5.9. Multivariate analysis of variance revealed a reliable group difference for the ascending/peak phase for cognitive test performance [Wilks’ lambda = .648, $F(8, 100)= 3.02$, $p = .004$, $\eta^2_p = .195$] but not for the descending or post-peak phase [Wilks’ lambda = .840, $F(8, 94)= 1.07$, $p = .389$, $\eta^2_p = .084$]. As with the earlier results, during the ascending/peak phase, there were significant differences between the BAC threshold groups for maze learning errors [$F(2,53) = 7.84$, $p = .001$, $\eta^2_p = .228$] and recall errors [$F(2,53) = 9.36$, $p < .001$, $\eta^2_p = .261$], but not for performance on the chase or card identification task. Post hoc pairwise comparisons revealed that the high threshold group made significantly more maze learning and recall errors compared with the medium and placebo threshold groups during the ascending/peak phase. There were no reliable differences between the placebo and medium threshold groups in this phase, and in the post-peak phase there were no significant differences between the three groups on any of the cognitive performance measures.

The findings from these analyses of the cognitive test data are similar to those based on dose and BAC level. That is, those in the high dose/high BAC groups made the greatest number of errors on the maze learning and recall tasks, whereas the performance of those in the medium dose/medium BAC groups were not reliably different from the placebo group. There was, however, an interesting and important asymmetry in the participants’ performance during the descending or post-peak phase of the BAC curve. As can be seen in figure 5.9, the numbers of maze learning and maze recall errors were greatest during the post-peak phase. Similarly, the driving measures of edge line crossings, time spent over the centre line and edge line, and maximum speeds all showed poorer performance during the post-peak phase than that
seen for ascending or peak BAC levels (acute protracted errors). In contrast, participants’ reactions to the false alarm vehicles during the ascending/peak BAC was worse than that seen during post-peak, meaning the effects were greater during the initial stages of intoxication (acute tolerance). This asymmetry is particularly noteworthy given that the absolute BAC levels used to compare post-peak and peak performance were equivalent as a result of the classification procedures used to define the threshold groups. The average ascending/peak BAC for the participants in the medium threshold group was 0.048% compared with 0.049% during the post-peak phase (the average ascending/peak BAC for participants in the high threshold group was 0.074%, and post-peak it was 0.066%). It should be noted that these asymmetries appeared predominately for the medium range of BACs (0.01% to 0.055%), although acute protracted errors were seen for the edge line measures in the high threshold group as well.

Figure 5.9  Participants’ cognitive test performance as a function of BAC threshold category; ascending/peak BAC (test block 2 or 3) and descending/post-peak BAC (block 4 or 5). Lines indicate 95% confidence intervals.

The degree of performance impairment seen for these post-peak measures of edge and centre line crossings, maximum speeds, and maze learning and recall was to a level where the impairment was equivalent to substantially higher BAC levels. In other words, for some tasks the degree of impairment from even moderate amounts of alcohol (0.055% and below) appeared to be particularly great after some time had elapsed (post-peak) than the level of impairment produced while participants’ BACs were reaching their peak. For other tasks (eg inhibition of responses to false alarm), post-peak performance was much better than that seen when BACs were ascending or at their peak.

5.2.4 Subjective perceptions of intoxication

As well as the objective performance measures obtained from the driving and cognitive tasks, participants were also asked to complete the walk and turn portion of the NZ Police compulsory impairment test and provide self-report ratings of their subjective intoxication, willingness to drive and sleepiness at the end test block. Comparing participants’ responses on the self-report measures with the objective performance...
data provides insights into how accurately the participants were able to judge their level of intoxication and how it may influence their behaviour.

There were statistically significant differences across the three alcohol dose groups for participants' ratings of subjective intoxication at each test block: block 2 \([F(2, 57) = 14.82, p < .001, \eta^2_p = .342]\), block 3 \([F(2, 58) = 11.64, p < .001, \eta^2_p = .286]\), block 4 \([F(2, 57) = 9.59, p < .001, \eta^2_p = .252]\) and block 5 \([F(2, 57) = 8.98, p < .001, \eta^2_p = .240]\). As can be seen in figure 5.10, the placebo group rated their intoxication significantly lower than the medium or high alcohol dose groups at test blocks 2, 3 and 4 (all \(p\)'s <.01 with Bonferroni correction). By block 5, only the high dose group’s ratings remained significantly higher \((p<.001) than the placebo group. There were no statistically significant differences between the medium and high dose group’s rating of subjective intoxication at any test block. It should be noted that the placebo group rated their subjective intoxication as greater than zero at every test block, suggesting that participants were unaware they had been allocated to the placebo group.

**Figure 5.10** Participants’ subjective ratings and walk and turn performance by alcohol dose group and test block

The participants’ willingness to drive ratings followed a similar pattern to the ratings of subjective intoxication, in that there were significant differences across the groups at test block 2 \([F(2, 58) = 16.75, p < .001, \eta^2_p = .366]\), block 3 \([F(2, 58) = 7.09, p = .002, \eta^2_p = .196]\), block 4 \([F(2, 57) = 6.33, p = .003, \eta^2_p = .182]\) and block 5 \([F(2, 57) = 6.29, p = .003, \eta^2_p = .181]\). At each test block, the placebo group rated themselves as significantly more willing to drive compared with the medium and high alcohol dose groups (all \(p\)'s <.05, with Bonferroni correction), but there were no reliable differences between the medium and high alcohol dose groups (see figure 5.10).
At the end of the test session, each participant was asked an open-ended question regarding the factors that influenced their willingness to drive ratings. These responses were grouped into four broad categories: feeling physically impaired, feeling fine, safety (self or others) and fear of being caught. The number of participants in the placebo, medium and high dose alcohol groups giving responses in each category is shown in table 5.1. The main factor influencing participants’ willingness to drive was feelings of physical impairment from the alcohol consumption. These feelings were described by participants as ‘light-headed’, ‘dizzy’, ‘intoxicated’, ‘tipsy’, ‘too drunk’, ‘foggy’ and interestingly almost half of the participants in the placebo group reported these factors as influencing their ratings of willingness to drive (additional evidence that these participants were unaware of their group allocation). The second most common explanation for the ratings was that participants ‘felt fine, didn’t feel any physical effects of alcohol’ while the remaining participants’ ratings were influenced by concerns for their or others’ safety (‘worried about crashing, safety: don’t want to hurt anybody’) or being caught (‘embarrassing to be caught and over the limit’).

Table 5.1 Participants’ explanations of their willingness to drive ratings

<table>
<thead>
<tr>
<th>Willingness to drive reason</th>
<th>Placebo</th>
<th>Medium dose</th>
<th>High dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physically impaired (n)</td>
<td>9</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>Physically fine (n)</td>
<td>7</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Safety (n)</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Fear of being caught (n)</td>
<td>2</td>
<td>0</td>
<td>4</td>
</tr>
</tbody>
</table>

Another way of viewing the participants’ subjective intoxication and willingness to drive ratings can be seen in figure 5.11. Shown here are the ratings from 48 participants where ascending and descending BAC levels have been matched for each participant in each of the three groups (the resulting mean BAC levels for the three groups were 0%, 0.056% and 0.093% on both ascending and descending limbs). As can be seen in the left side of the figure there is a significant difference in the ratings of subjective intoxication for the three groups \( F(2, 46) = 13.89, p < .001, \eta^2 = .377 \). There is also, however, a significant asymmetry between the ascending and descending stages of intoxication \( F(1, 46) = 4.47, p = .040, \eta^2 = .088 \), even though the BAC levels are the same. This effect is particularly pronounced for the medium alcohol (0.05%) group.

Figure 5.11 Participants’ subjective ratings of intoxication and willingness to drive compared for matched BAC levels at ascending and descending stages of intoxication
As can be seen in the right panel of the figure, these patterns of subjective intoxication were mirrored in the participants’ willingness to drive ratings. There was a significant difference between the three groups in their willingness to drive ratings \( F(2,46) = 10.75, p < .001, \eta^2 = .319 \), and a significant difference between the ascending and descending stages of intoxication \( F(1,46) = 3.183, p = .081, \eta^2 = .065 \).

Participants’ performance on the walk and turn test followed a pattern very similar to the ratings of subjective intoxication and willingness to drive. That is, the total number of errors differed significantly across the groups at each test block: block 2 \( F(2, 58) = 10.36, p < .001, \eta^2 = .263 \), block 3 \( F(2, 58) = 12.57, p = .001, \eta^2 = .302 \), block 4 \( F(2, 57) = 7.34, p = .001, \eta^2 = .205 \) and block 5 \( F(2, 57) = 8.55, p = .001, \eta^2 = .234 \). Pairwise comparisons indicated that participants in the placebo group made significantly fewer errors compared with those in the medium and high alcohol dose groups for test blocks 2, 3 and 5 \((p’s < .05)\). In test block 4, the only reliable difference was between the placebo and high alcohol dose groups \((p = .001)\). There were no statistically significant differences between the medium and high alcohol dose groups. No statistically reliable group differences in sleepiness ratings were seen for any of the test blocks.

At the conclusion of the experiment, all participants were also asked to provide an estimate of the number of standard drinks they thought they had consumed. These estimates differed significantly across the three dose groups \( F(2, 55) = 19.86, p < .001, \eta^2 = .419 \). The placebo group’s estimates of the number of drinks they consumed \((M = 1.42, SD = .892)\) provides more evidence that these participants were unaware they had been allocated to a low alcohol condition (.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, as a whole tended to underestimate the amount of alcohol they consumed (actual drinks consumed \(M = 4.78, SD = 1.43\)).

The drink estimates of participants in the high dose group \((M = 4.08, SD = 1.88)\) were also significantly higher than the placebo group’s estimates \((p < .001)\), and much lower than the actual number of standard drinks they consumed \((M = 7.01, SD = 2.86)\). The degree of their underestimation meant there was no statistically significant difference in the amount they estimated when compared with the medium group’s estimate. Interestingly, however, the female participants in the high alcohol group estimated they had drunk less than the females in the medium alcohol group, leading to a significant group x gender interaction \( F(2, 58) = 4.39, p = .017, \eta^2 = .145 \). Females in the high alcohol group estimated they had consumed 2.72 standard drinks, whereas in reality they had consumed an average of 4.81. In comparison, the females in the medium group estimated they had consumed 3.37 drinks after having received 3.78 standard drinks. Men in the medium group estimated they had consumed 4.25 drinks after consuming 5.6 drinks, and men in the high group estimated 5.3 drinks when in actuality they had consumed nearly twice that amount, an average of 9.01 standard drinks.

### 5.2.5 Knowledge and attitudes to drink-driving

At the start of the experimental testing participants were asked what the current drink-drive limit (blood or breath) is for drivers over 20 years and their responses were scored as being correct or incorrect (scoring was based on the numbers provide by participants rather than the units of measurements). Of the 61 participants in the full experiment, eight were employed by the NZ Police so their data was analysed separately. All of the NZ Police employees correctly knew the current legal limit for drinking and driving, and of the remaining participants fewer than half (47%, \(n = 25\)) could accurately report the current drink-drive limit (based on blood or breath alcohol concentration).

Participants were also asked if they thought the current limit should be changed and their responses were grouped into six categories: zero, lower than current, stay the same, higher, no limit, and don’t know (regardless of their knowledge of the current drink-drive limit). The majority of police employees \((n = 6)\)
thought the drink-drive limit should be reduced with only two suggesting it should remain the same. Of the remaining participants, the majority thought it should be zero ($n = 12$) or lower than the current limit ($n = 17$). Seventeen participants thought it should remain the same, one thought the limit should be higher, one thought there should be no limit and five said that they didn’t know. Overall the majority of participants (57.3%) thought the current drink-drive limit should be reduced.

Participants were also asked to rate how safe they would feel driving home or being driven home by a friend, after consuming three standard drinks over a long lunch or dinner. Overall participants rated themselves driving as being marginally safer than a friend driving at lunch (self $M = 5.36$ $SD = 2.50$; friend $M = 5.84$ $SD = 2.40$) or dinner (self $M = 5.52$ $SD = 1.28$; friend $M = 5.93$ $SD = 2.37$). When asked how many drinks they thought they could consume and still drive safely participants estimated an average of 2.02 standard drinks ($SD = 1.28$). When asked how many standard drinks they could consume and be under the legal limit participants’ estimates averaged 3.26 standard drinks ($SD = 1.94$).

Together these findings indicate that the majority of participants did not know the current drink-drive limit (for those over 20 years of age), but they felt the current drink-drive limit should be decreased. In addition, participants thought that the number of drinks they could consume and still drive safely was, on average, one drink less than the amount of alcohol they thought would bring them to a point just under the legal drink-drive limit.
The main objective of the present research was to evaluate the effects of alcohol on the psychomotor, cognitive and driving abilities of New Zealand drivers across the 0.05% and 0.08% BAC levels. The results of the dosage trial showed that the amount of alcohol required to produce BACs of 0.05% and 0.08% varied considerably from person to person, even taking into account differences in their body weight. In particular, alcohol produced substantially higher BAC levels for women compared with men consuming equivalent amounts of alcohol.

The study showed that once a BAC level of 0.08% had been reached, significant performance impairment could be seen (relative to placebo controls) across a broad range of cognitive and driving measures. Participants with a BAC level of 0.08% had significant increases in edge and centre line crossings in the driving 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 and longer response times on a card identification task, measures of executive function, problem solving, memory and visual attention.

Participants with a BAC of 0.05% also showed some performance impairment on these measures, but the level of impairment was not large enough to be statistically worse than the placebo control condition on the number of edge line crossings, seconds spent over the edge line, peak speed, or any of the cognitive performance measures. At 0.05% BAC only the number of centre line crossings and amount of time spent over the centre line were significantly worse than the performance seen for 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 embedded hazard detection task, 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.

A second research objective was to identify the relationship between drivers' perception of intoxication and the actual level of impairment produced. In the present study participants in the two alcohol groups rated themselves as significantly more intoxicated than did the participants in the placebo condition. The two alcohol groups' ratings of intoxication did not differ from one another, however, indicating that although the participants could tell they were intoxicated, they could not accurately determine the level of their intoxication. Similarly, the participants were not able to correctly judge how much alcohol they had consumed. Both of the alcohol groups underestimated the amount they had consumed, and the amounts they estimated were very similar, which meant that participants receiving the high dose were extremely inaccurate, 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.

One more subjective estimate of intoxication should be mentioned at this point. The walk and turn test represents an observer's subjective estimate of someone else's level of intoxication, albeit there are quantitative scoring criteria associated with the test. The results of the walk and turn test mirrored the other subjective estimates with the participants in the two alcohol groups scoring as performing significantly worse than the placebo participants. Although there was a tendency for participants in the high dose group to score slightly worse than those in the medium group, there was no statistically reliable
difference in their scores. (It should be remembered that the observers in the laboratory were not blind to the amount of alcohol actually consumed by the participants, which was a very different situation from that of a police officer administering the test in the field.)

The effects reported above demonstrate findings reported elsewhere in the published literature: substantial impairment produced by a BAC of 0.08%, with more subtle effects of 0.05% being evident only for more complex tasks such as hazard avoidance (Leung and Starmer 2005; Liu and Fu 2007; Moskowitz and Fiorentina 2000; West et al 1993). Participants’ subjective estimates of their own levels of intoxication and impairment were again shown to be relatively insensitive to actual BAC levels and degree of impairment (Cromer et al 2010; Schweizer and Vogel-Sprott 2008; Weafer and Fillmore 2012).

The findings of the present study also showed the asymmetries associated with the alcohol intoxication curve, ie acute tolerance and acute protracted error effects, that have been reported in the recent literature. Maze learning and maze recall errors, edge line crossings, time spent over the centre line and edge line, and maximum speeds all showed poorer performance during the post-peak phase than that seen for ascending or peak BAC levels (acute protracted errors). Response disinhibition (reactions to the false alarm vehicles) and ratings of subjective intoxication during the ascending/peak BAC were worse than that seen during post-peak, meaning the effects were greater during the initial stages of intoxication (acute tolerance). These findings replicated recent published reports (Cromer et al 2010; Schweizer and Vogel-Sprott 2008; Weafer and Fillmore 2012), although the present study showed them to predominate in the medium range of BACs (0.01% to 0.055%). As others have noted, the combination of these two effects, specifically acute tolerance for self-ratings of intoxication and acute protracted errors for many 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 much 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 a level of delayed impairment that was nearly equivalent to that seen for substantially higher BAC levels, at the same time when these participants were indicating an increasing willingness to drive.

The acute tolerance of participants’ ratings of subjective intoxication suggests that drivers may be judging their sobriety based on some explicit or conscious elements of performance whereas other aspects of their performance (particularly driving) remain less accessible to these explicit judgements. Whether this is because performance on tasks like driving do not lend themselves to unambiguous pass/fail judgements, or because drivers simply do not consciously monitor their performance on these types of tasks to the same degree is not clear, but there is evidence that many aspects of driver performance may be controlled by automatic processes that drivers do not consciously monitor (Charlton and Starkey 2011). Previous researchers (Cromer et al 2010; Schweizer and Vogel-Sprott 2008) have speculated that prepotent responses (well-practised or reflexive) might be more likely to demonstrate acute tolerance to alcohol whereas the present findings suggest that prepotent responses, such as those used when steering a car, actually demonstrate acute protracted errors and a longer recovery time.

In either case, the findings of poor self-assessments of intoxication and the performance asymmetry during recovery suggests an important focus for public education regarding alcohol and driving. Drivers intoxicated at the currently enforced adult limit cannot accurately judge the amount of alcohol they have consumed or their level of performance impairment. After drinking even moderate amounts of alcohol, drivers’ judgement of their intoxication is impaired; in fact some drivers in the present study reported that they thought their driving had improved after drinking although it clearly had not. A second public education message might address the issue of the delayed or protracted effects of alcohol intoxication on motor performance: some aspects of safe driving recover very slowly, and may persist even after BACs have fallen to below legal limits.
Finally the present study produced some rather interesting findings with regard to New Zealand drivers’ knowledge of the law associated with drink-driving and attitudes towards that law. Fewer than half of the participants from the general population of drivers were able to correctly state the current adult drink-drive limit (in either blood or breath alcohol concentration). While this situation might not seem particularly surprising given that drivers could not accurately judge their intoxication or the amount of alcohol they had consumed, it does cast a somewhat disquieting light on the driving public’s attitudes to the current drink-driving law. Of the sample of drivers in the present study, the majority (57.3%) thought the current drink-drive limit should be reduced. This result coincides with other recent surveys of the larger driving population; the Ministry of Transport’s 2012 driving attitudes survey found that 60% of New Zealanders favoured a lower legal blood-alcohol limit for driving (41% thought the limit should be lowered from 80mg/100ml to 50mg/100ml and a further 19% wanted it lowered to zero) (Ministry of Transport 2012b). Similarly, a New Zealand Herald DigiPoll released at the time this report was being prepared indicated that 65% of New Zealanders were in favour of reducing the drink-drive limit from 80mg/100ml to 50mg/100ml.

The artificiality of laboratory procedures generally, and simulations specifically, must be taken into account when generalising the results from such procedures to real-life situations. In the present case, the hazards included in the simulation scenario were all plausible, but their concentration in a short section of road was unlikely. This does, however, speak to one advantage of simulation, as the possibility of even a low rate of crashes would make replication of this sort of experiment on the highway unethical and impractical. Further there is an emerging consensus that relative validity (i.e., the equal generalisability of the conditions being compared in a simulated environment) may be more important than absolute validity (e.g., the veridical correspondence between driver speeds and reaction times obtained in simulation and on real roads) (Godley et al. 2002; Törnros 1998). The number of laboratories using simulation has increased dramatically in recent years, perhaps reflecting this understanding (Bella 2008). In the specific case of research into alcohol’s effects using a driving simulator, a recent paper comparing driving simulator performance with on-road performance showed that driving simulation is a sensitive measure of alcohol impaired driving and good external validity of the results (Helland et al. 2013).
7 Recommendations

1. Convey the findings from the present research to the public, particularly as regards the inaccuracy of self-assessments of intoxication and the delayed or protracted driving impairment resulting from alcohol.

2. Consider extending the research approach and methodology to examine the degree of driving impairment associated with common prescription medications such as amitriptyline, fluoxetine, and benzodiazepines such as oxazepam.


Borkenstein, RF, RF Crowther, RP Shumate, WB Ziel and R Zylman (1964) *The role of the drinking driver in traffic accidents*. Bloomington, IN: Department of Police Administration, Indiana University.


Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml


Phillips, DP and KM Brewer (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.


Driver risk from blood alcohol levels between 50mg/100ml and 80mg/100ml


Veldstra, JL, KA Brrohuis, D de Waard, BHW Molmans, AG Verstrate, G Skopp and R Jantos (2012) Effects of alcohol (BAC 0.5%) and ecstasy (MDMA 100mg) on simulated driving performance and traffic safety. Psychopharmacology 222: 377-390.


# Appendix A: Walk and turn scoring sheet

## Walk and Turn Assessment

**Explain assessment:**
- Stand and place your left foot on the line. Place your right foot on the line in front of your left foot, touching heel to toe.
- Place your arms by your sides. Remain in this position until I tell you to start.
- When I say start take nine heel to toe steps along the line counting each step out loud.
- After nine heel to toe steps turn around by pivoting your front foot on the line and taking a series of small steps with the other foot.
- After turning take nine heel to toe stops back along the line.
- Leave your arms by your sides throughout the assessment, watch your feet at all times and count each step out loud.
- Once you start, do not stop until you have completed the assessment (demonstrate the assessment).

**"Do you understand?"**

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
</table>

| "Start" |

## OBSERVATIONS

<table>
<thead>
<tr>
<th>Poor balance during instructions?</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Starts walking before directed to do so?</td>
<td>YES</td>
<td>NO</td>
</tr>
</tbody>
</table>

## RESULTS

<table>
<thead>
<tr>
<th>Stops while walking?</th>
<th>YES</th>
<th>NO</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walked off line?</td>
<td>YES</td>
<td>NO</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>Heel to toe missed / doesn’t touch?</td>
<td>YES</td>
<td>NO</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>Incorrect number of steps?</td>
<td>YES</td>
<td>NO</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>Turns incorrectly / loses balance on turn?</td>
<td>YES</td>
<td>NO</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>Arms to balance?</td>
<td>YES</td>
<td>NO</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>Ability to follow directions?</td>
<td>YES</td>
<td>NO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>If no, describe behaviour</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Was the Walk and Turn assessment completed?</td>
<td>YES</td>
<td>NO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Was the Walk and Turn assessment completed satisfactorily?</td>
<td>YES</td>
<td>NO</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Record any observations made