THE EFFECT OF LOW DOSE ALCOHOL ON SIMULATED DRIVING AND COGNITIVE PERFORMANCE

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ABSTRACT

The current study investigated the effect of alcohol on simulated driving and cognitive performance across multiple blood alcohol levels (0.00, 0.02, 0.05 & 0.08%). The main objective was examine if the effect of alcohol was dose and task dependent and whether there was a mismatch in the development of acute tolerance across subjective and objective measures. Thirty participants (male & female) completed a simulated drive that comprised a rural highway which was divided into low and high traffic segments. In the driving scenario, a range of measures including speed maintenance, sign detection and hazard reaction were collected. Participants also completed a computer administered continuous performance test, a subjective measure of intoxication and had their breath alcohol level recorded. The experiment included a pre-alcohol, intoxicated and two post alcohol recovery conditions in which the measures were repeated at the same time intervals. Results showed no significant impairments in accelerator or brake reaction time but there was a significant increase in the number of crashes which increased in a dose dependent manner. There were no significant impairments in the sign detection task but traffic density was found to impair driving performance particularly in the heavy traffic segments. A significant Group*Density*Road interaction was also found, where the 0.05% group had a higher maximum speed on Road 4 than on Road 3 in the heavy traffic (70km/h) zone. There were no significant findings for the development of acute tolerance.

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INTRODUCTION

Alcohol has been shown to have a complex physiological effect which can directly and indirectly lead to the development of various adverse symptoms, for example changes in mood state, loss of coordination, slurred speech, unsteady gait, impaired judgement, dizziness and more (Vengeliene, Bilbao, Molander & Spanagel, 2009). At high levels, signs of intoxication can become progressively worse, often resulting in more global impairment, coma or even death. Vengeliene, et al (2009), reported that many of the pharmacodynamic effects of alcohol are due to its primary action on specific neurotransmitters in the brain. For example, recent studies have shown alcohol influences the function of NMDA, GABAa, glycine, serotonin, nicotinic ACh receptors, L-type Ca2+ channels and G-protein-activated inwardly rectifying K+ channels (Vengeliene, Bilbao, et al, 2009). Alcohol's direct action on the brains neurochemistry triggers a cascade of indirect effects on other neurotransmitter or neuropepetide systems which cause many of the behavioural impairments (Vengeliene, et al, 2009).

Overall, there has been consistent evidence heavy alcohol consumption can have many damaging effects on general health and brain function (Parsons and Nixon, 1998). However, there has been a common assumption most of the adverse effects associated with alcohol can be avoided if it is consumed in moderation (Eckardt, File, Gessa, Grant, Guerri, Hoffman, Kalant, Koob, Li & Tabakoff, 1998). Some studies have even suggested a small amount of alcohol can have a number of medicinal effects supporting the idea it is harmless to drink (Wallner, Hancher & Olsen, 2006). For example, low doses of alcohol have been thought to lower rates of myocardial infarction, reduce heart failure and more (Wallner, et al, 2006). However, over time the effect of moderate alcohol

consumption has been questioned, with other findings demonstrating even low levels can have a negative impact. For example, Vengeliene, et al (2008) reported a blood alcohol concentration within the legal driving limit can significantly impair NDMA function plus a number of other ion channels and receptors.

Alcohol related driving statistics

The fact alcohol is a widely used substance which can impair cognitive and psychomotor functions even at low to moderate levels has important implications. One major concern is the issue of alcohol consumption and driving which has been found to be associated with a number of significant safety risks. Statistical studies have consistently reported the link between alcohol consumption and road user fatalities, demonstrating the dramatic impact alcohol can have on driver performance (Langely & Marshall, 1994).

In New Zealand, statistics for motor vehicle crashes in NZ during 2010 showed there were 337 fatal crashes which resulted in 375 casualty deaths and 14, 031 injuries (Ministry of Transport, 2012). Alcohol was found to be a contributing factor in 121 of the fatal crashes which accounted for 142 deaths and 2, 111 casualties who sustained injury. Results from post mortem blood alcohol measurements taken from drivers killed in motor vehicle crashes, revealed 58 had a BAC above 80mg/100ml and 115 had a BAC within the range of 0 - 30mg/100ml (Ministry of Transport, 2012). The social cost of alcohol / drug related crash incidents in 2010 totalled approximately $898 million which equated a quarter of crash costs involving injury (Ministry of Transport, 2012).

The impact of driving under the influence of alcohol has also been shown to be a major factor which contributes to road trauma in other countries. For example, in Australia, driving under the influence contributes to a large number of

deaths and serious injuries on roads each year (Australian Transport Council, 2010). During 2010, it was reported there were 105 fatal crashes involving 118 fatalities and 886 serious crashes where 1,050 people sustained serious injuries (South Australia Police, 2010). It was reported 33% of those who were killed in road accidents were found to have a blood alcohol content of 0.05% or greater (South Australia Police, 2010). Overall, alcohol has been found to cause more than a third of all road fatalities with 1 in 5 people killed who have a BAC greater than the legal limit of 0.05% (Australian Transport Council, 2010).

The problem of drink driving is similar in other countries like Great Britain, Canada and America with crash statistics revealing a similar trend (Chowdury & Kilbey, 2011), (Transport Canada Road Safety and Motor Vehicle Regulation, 2011), (National Highway Traffic Administration, 2009) . Overall, when one considers the available evidence, it is clear drunk driving continues to be a major public health concern both locally and internationally. As a result, different countries established statutory blood alcohol limits as an attempt to reduce the incidence of road accidents (Albalate, 2008). However, this raises some interesting questions as not all countries have the same legal blood alcohol content (BAC) for driving. For example, in New Zealand the current legal BAC limit for driving is 0.08% for fully licensed drivers while in Australia it is 0.05%. Although efforts have been directed toward setting statutory BAC limits for driving, what constitutes a safe or rational BAC is an issue which continues to be debated (Rothengatter, 2002). For example, there is accumulating evidence which suggests any detected alcohol consumption or legal impairment significantly increases the severity of crash morbidities and the number of road fatalities (Traynor, 2005).

The effect of alcohol on driving and cognitive processes

Psychomotor functions

There are many studies which have shown alcohol can impair psychomotor functions which are essential for being able to make quick and coordinated responses while driving. Alcohol induced deficits in any area of psychomotor function are often evidenced by impairments in reaction time and a deterioration in overall manual handling or driving precision which poses a risk to driver safety. For example, Rzepecki-Smith, Meda, Calhoun, Stevens, Jafri, Astur and Pearlson (2010) investigated the effect of alcohol within the range of 0.071 - 0.10%, using an adapted driving simulator that could be used with a functional magnetic resonance imaging (fMRI) scanner. Rzepecki-Smith, et al (2010), found that the brain circuit connectivity between the frontal-temporal, basil ganglia (FTBG) and cerebellar networks was disrupted following the consumption of high alcohol doses. The disturbance noted in the specific brain regions was shown to be correlated with different impairments in driving performance. For example, a significant amount of unstable vehicle steering occurred with BACs of 0.071 - 0.10% due to the disruption of functional networks in cerebellum and frontal temporal lobes which play a role in the ability to plan and coordinate gross and fine motor skills.

Similar results were found in another fMRI study carried out by Meda, Calhoun, Astur, Turner, Ruopp and Pearlson (2009), who examined the effect of multiple BACs (0.00, 0.05 & 0.08%) on simulated driving. Meda, et al (2009), also found that alcohol exerted a negative effect on brain circuit activity in areas which are important for motor planning (Meda, et al, 2009). The changes observed in the brains activity was linked with a significant increase in centreline

crossings and steering variability which indicates participants psychomotor functions were impaired (Meda, et al, 2009). Together each of the fMRI studies were able to accurately identify central areas of brain that are activated during various driving tasks, providing concrete evidence of the specific effects alcohol can have. However, the external validity of the studies could also be limited because the experiments were conducted in a setting which was quite different to the normal driving context. For example, participants would have had to drive while lying down inside the fMRI scanner which could have been a confounding factor.

While fMRI studies have some inherent limitations, similar results have been found with research which has examined the effect of alcohol using a standard driving simulator which emulates the natural driving context reasonably well. For instance, Miller, Weafer and Fillmore (2009), reviewed the data from seven studies in which the effect of alcohol on simulated driving performance with a BAC of 0.08% was explored. To assess the effects of alcohol, participants had to complete a simulated driving scenario in which they had to maintain an appropriate lane position and drive at a constant speed of 88.50kms. Miller, et al, (2009) found participants displayed a greater amount of lane deviation and steering variability following an alcohol dose of 0.65g/kg, indicating there were impairments in motor coordination (Miller, et al, 2009).

Deficits in psychomotor performance were also identified by Marczinski, Harrison and Fillmore (2008), who also examined alcohol's effect on simulated driving following a dose of 0.65g/kg (BAC 0.08%). Findings demonstrated that a range of psychomotor skills were impaired by moderate alcohol consumption. Similar to Miller, et al (2009), Marczinski, et al (2008) found that there was a

significant increase in within-lane variability with a BAC of 0.08%. Participants were also unable to maintain an appropriate speed, indicating they made more errors compared to their performance in the placebo condition. Together, the studies conducted by Miller, et al, (2009) and Marczinski, et al (2008) confirm that higher doses of alcohol (BAC $\geq$ 0.08%) impair different psychomotor functions, but it would have been interesting to know whether the impairments would have occurred with lower doses of alcohol. Other studies which have investigated a broader range of BACs have shown that even at lower dosages of alcohol can produce impairments in various psychomotor functions. For example, Ligouri, D'Agostino Jr, Dworkin, Edwards and Robinson (1999) assessed the effect of different BACs ranging from 0.00%, 0.06% to 0.10% on participants individual's equilibrium and simulated driving performance. The results showed that brake and accelerator reaction time in response to a yellow barrier presented suddenly in the path of the participant's vehicle was significantly impaired following the consumption of moderate and high doses of alcohol. Participants also displayed a greater amount of body sway which indicates equilibrium was disrupted. Psychomotor speed was also impaired on a choice reaction time task, where participants had to respond by pressing a key on a keyboard which corresponded with a stimulus displayed on a computer screen, but only for the high alcohol dose 0.80g/kg (Ligouri, et al, 1999).

Other studies have used actual on road tests and found low to moderate doses of alcohol impair different psychomotor functions. For instance, West, Wilding, French, Kemp and Irving (1993) examined how low and moderate doses of alcohol (BACs of 0.025% & 0.05%) affected simple psychomotor functions. To assess performance, participants drove a car around a set circuit in which they had to maintain a constant speed. The results revealed that alcohol did not have a

significant effect on speed maintenance with a BAC of 0.025 or 0.05%, which suggests higher doses are needed to produce impairments in simple driving skills. Yet, when one considers the nature of the experiment it needs to be recognised that the results may have been limited by the way the driving test was conducted. As an experimenter had to sit in the back seat of the car while the participants completed each drive, it is possible testing effects confounded the results of the study. Although, participants were not informed that their speed was being recorded, merely having the experimenter in the back seat could have changed the way participants drove.

In another study, Domingues, Mendonca, Laranjeira and Nakamure-Palacios (2009) found evidence that psychomotor functioning was significantly impaired in drivers who had a BAC equal or above 0.06%, compared to those with a BAC of 0.00%. Deficits in psychomotor functions were identified on a sub-test from an executive frontal test battery which required individuals to execute a series of motor functions in a correct order. Impairments on the test indicated complex psychomotor functions were affected by moderate to high doses of alcohol (Domingues, et al, 2009). Similar to the study conducted by West, et al (1993), some limitations were apparent in the methodology of the experiment. The main limitation which reduced the validity of the research was the sampling method Domingues, et al (2009) utilised. For the study 490 drivers were randomly selected by agents of the State Traffic Department and Military police who were concurrently running a campaign to detect the presence of alcohol in nocturnal drivers. No screening criterion was used for the selection of participants which meant the effect of alcohol could have been influenced by a number of other factors. For example, participants who took part in the study may have been on medication or they could have had a pre-existing medical condition

which could have interacted with the effects of alcohol. Secondly, there would have been differences in the type and amount of alcohol each individual had consumed prior to being tested which influenced the outcome of the results. As there were uncontrolled variables, it is difficult to compare the results with other studies which were conducted in more controlled contexts.

Kuypers, Samyn and Ramackers (2006) conducted a more controlled actual on road experiment and found alcohol psychomotor functions were impaired by moderate doses of alcohol. In this study participants completed an on road driving test which included road tracking and car following tasks that took one hour to complete. For the road tracking task, participants had to maintain an appropriate lane position and drive at a constant speed of 95km/h (Kuypers, et al, 2006). In the car following task, participants had to maintain a headway distance of 15 – 30 meters behind a lead car which drove at 70km/h. The lead car slowed down six times at different points on the circuit which required participants to adjust their speed accordingly (Kuypers, et al, 2006). The findings revealed different aspects of driving performance on the road tracking and car following task were significantly impaired with a BAC of 0.05mg/ml alcohol (Kuypers, et al, 2006). Driving while intoxicated increased the standard deviation of lateral position on the road tracking task in comparison to the non-alcohol condition. However, speed and overall lateral position were not affected. In the car following test, significant changes in brake reaction time were also observed but there were no significant differences in the accuracy in which headway distance was adjusted (Kuypers, et al, 2006). It is important to note the drive took one hour to complete and a top up dose was given before the start to help maintain a stable BAC of 0.05mg/ml which could have been a limiting factor. As oral dosing is known to produce a wide variation in BACs, it is unclear whether the BACs

following the top up dose remained below the target level during the actual driving test. BACs were taken at the start and end of the drive which meant BACs could have peaked to a higher level during the test which may have impacted the results.

While every effort may be taken to ensure peak BACs are achieved within a certain range, individual differences in the metabolism and bioavailability of alcohol will often result (Zoethout, Schoemaker, Zuurman, van Pelt, Dahan, Cohen, van Gerven, 2009). For example, in a tightly controlled experiment Grant, Miller and Kenny (2000) compared the effect of alcohol administered orally and intravenously on psychomotor skills for BACs of 20, 50 and 80mg/100ml. Their results revealed significant psychomotor impairment occurred at BACs of 50 - 80mg/100mls in a choice reaction time task compared to baseline (Grant, et al, 2000). During a dual tracking task, impairment was also observed in psychomotor function evidenced by a significant reduction in reaction time at BACs of 50mg – 80mg/100mls (Grant, et al, 2000). The results were easily distinguished when alcohol was intravenously administered. However, when alcohol was given orally, psychomotor performance was difficult to assess as BACs varied too widely (Grant, et al, 2000). These findings indicate the rate in which BACs increase and decline can often vary significantly following oral dosing which is a limiting factor for most of the other studies.

Information processing (hazard perception)

Information processing refers to the encoding, storage and manipulation of sensory information. A deficit in the way one receives and processes information is therefore likely to have important implications for perception of hazards while driving. For example, Sewell, Poling and Sofuoglu (2009), reviewed a number of

experimental studies which measured the effect of alcohol on different aspects of cognition and driving. Their findings revealed alcohol at a high dose of 0.75g/kg, significantly impaired hazard perception. The ability to perceive a negative consequence associated with risk taking was impaired, and individuals who consumed alcohol drove faster, made more errors and attempted to overtake other vehicles more frequently (Sewell, et al, 2009).

Hazard perception has been shown to be impaired by even moderate doses of alcohol, Deery and Love (1996) investigated the effect of moderate alcohol (0.05% BAC) on young driver's ability to identify traffic hazards, using a series of videos taken from the driver's perspective. The videos were 10 minutes long and participants had to view and rate how dangerous they perceived the hazards to be. Deery and Love (1996) found that participants with a BAC of 0.05% took significantly longer to detect hazards and reacted to them more abruptly. Interestingly, traffic hazards which occurred as a result of the driver's own actions were rated as less dangerous than those caused by another driver. Factors like tailgating, passing a truck on an inside lane were regarded as less hazardous than a car pulling out of a side road or a pedestrian running across the road. The finding that drivers who are moderately intoxicated may not regard their own errors as being hazardous, could explain why some take more risks and don't necessarily make any allowances.

Generally, there is converging evidence the way individuals perceive hazards is impaired to some extent when BACs exceed 0.05%, but it is unclear whether a lower BAC of 0.025% has an impact. Some studies have examined lower dosages of alcohol, but the evidence is not entirely conclusive. For example, West, et al (1993), examined how low and moderate doses of alcohol

(BACs of 0.025% & 0.05%) affected hazard perception. Similar to the method used by Deery and Love (1996), participants sat in a simulator and viewed several videos of different car driven routes which contained different types of hazards. The results of the study revealed that participants with a BAC of 0.05% were slower to perceive hazards, but there were no significant differences at a BAC of 0.025% (West, et al, 1993). While there was no evidence that hazard perception was impaired at a lower BAC level, it cannot be entirely excluded because the BACs achieved in the study varied wildly which made it difficult to compare the results.

Overall, the results from Sewell, et al (2009), West et al (1993) and Deery and Love (1996), confirm there were notable differences in the perception of hazards following moderate and high doses of alcohol. However, the studies conducted by West, et al (1993) and Deery and Love (1996) used a hazard perception simulation task that was non-interactive which could have limited the validity of the results obtained. It would have been interesting to know whether impairments in hazard perception would be the same if the participants had been able to drive the routes in an actual driving simulator. Actively being in control of the car would replicate the natural driving context more which could influence how dangerous participants rate both passive and active hazards. Leung and Starmer (2005) examined the effect of alcohol at BACs of 0.08% on mature and young driver's ability to detect and perceive hazards, using a driving simulator. Measures of gap acceptance were used to examine if alcohol increased the tendency to misjudge acceptable margins of safety. Results revealed that experienced drivers detected hazards more quickly on straight roads but on curved sections the opposite trend was observed. In an overtaking task, novice drivers remained in the opposite lane longer while more experienced drivers overtook

faster. Together, the findings suggest impairments in hazard perception could be due to a combination of cognitive and psychomotor impairments and that factors like driving experience can also play a role.

Attention

Driving involves a number of complex tasks which require focused attention. Deficits in the ability to attend are therefore often associated with marked deterioration in task performance. While various factors can affect one's ability to concentrate, alcohol-induced impairments have been found to be a key factor. There are various studies which have shown alcohol slows cognitive processes and restricts ones capacity to focus their attention however, the effects appear to be task dependent (Schulte, Muller-Oehring, Strasburger, Warzel and Sabel (2001). For example, studies have failed to detect impairments in sustained attention on tasks which require one to focus on a single task over prolonged period of time, but have found performance deteriorates when a dual task is performed (Schulte, et al, 2001). In a review Moskowitz and Fiorentino (2000) conducted for the U.S. Transportation National Highway Traffic Safety Administration, low of doses of alcohol were found to impair the ability to carry out primary and secondary tasks at BACs as low as 0.005%. The fact that alcohol-induced impairment increases on tasks which require divided attention is not surprising, as a greater amount of attention and cognitive processing is required to carry out multiple tasks at the same time (Chamberlian & Solomon, 2002). For example, completing secondary in-vehicle tasks while driving creates a high degree of resource conflict which can often impair task performance which often means drivers shed performance on secondary tasks to in attempt to maintain primary goals of driving (Sewell, et al, 2009). While drivers may

attempt to compensate by either increasing their effort or lowering their performance, coping strategies are usually insufficient to overcome impairments in attention produced by alcohol (Sewell, et al, 2009).

Rakauskas, Ward, Boer, Bernat, Cadwallader and Patrick (2008) demonstrated the effect of alcohol (BAC 0.08%) and distraction in an experiment where participants were required to follow a lead vehicle and maintain a safe headway distance. Task demand was increased by having the participants complete a variety of in-vehicle tasks while driving which included things like having to adjust the temperature or radio or answering questions on a hands free cell phone. Rakauskas, et al, (2008) found participants had to make greater attempts to maintain their within lane position and they also increased the distance in which they followed the lead car in order to lower the demand of the tasks. The result also showed that distraction alone caused a significant amount of impairment, but when combined with alcohol at a BAC of 0.08%, the level of impairment was greater (Rakauskas, et al, 2008). This is not surprising as alcohol has also been shown to increase distraction and interference from secondary stimuli which makes it more difficult for individuals to selectively focus their attention while performing secondary task. For example, Wester, Verster, Volkerts, Bocker and Kenemans (2010), examined the effects of alcohol (BACs of 0.00, 0.02, 0.05, 0.08 & 0.10%) on attention and dual task performance using a Divided Attention Steering Simulator (DASS) and an oddball task. The oddball task involved the presentation of irrelevant and standard auditory tones in both single and dual task conditions which was used to measure how well participants were able to focus their attention. Wester, et al, (2010) found that following the consumption of alcohol, participants made more errors, their reaction time increased and there was a greater amount of steering error showing alcohol

increased interference and distraction from secondary tasks which caused drivers to shift their attention away from the primary task of driving.

Other studies have also shown that sign detection and distance estimation tasks are particularly sensitive to alcohol's effects (Ogden & Moskowitz, 2010). Decreased accuracy rates on sign detection tasks has been known to occur at BACs as low as 0.02% indicating the ability to discriminate and monitor changes in the environment can be impaired by alcohol. Response times have also been found to increase, suggesting cognitive processes are slower following low amounts of alcohol consumption (Liu & Ho, 2010). Impairments in judgment have also been shown to occur where drivers often over estimate or under estimate the relative distance of a target object. Deficits that have been found in judgment suggest attention and the ability to process information can be impaired by low dose alcohol when performing a secondary task (National Traffic Highway Safety Association, 2000).

The effects of alcohol on tasks like sign detection and distance estimation were demonstrated in a study conducted by Yung-Ching and Shing-Mei (2007) who investigated the effect of alcohol on divided attention and simulated driving comparing BACs of 0.00, 0.02, 0.05, 0.08 and 0.10%. To measure the effect of alcohol, signs which randomly displayed a left or right arrow for five seconds were posted on the side of the road, across low and high load conditions. In the high load condition the density of traffic was greater, the lane width was narrower, the road contained more curves and there were more intersections. In comparison, the low load condition had fewer curves, a wider lane width and less traffic. Participants had to detect the signs and indicate what symbol was displayed using the cars corresponding indicator. Similar to other findings, the

results revealed participants took longer to react and made more errors across both the low and high load conditions. The effect of alcohol was found to increase in a dose dependent manner, with greater impairments observed at BACs of 0.08% (Yung-Ching & Shing-Mei, 2007). What was interesting about the findings of Yung-Ching and Shing-Mei (2007) was the fact impairment was found in the low and high load condition which suggests tasks that require divided attention can cause impairment, even though the driving context itself may not be particularly challenging. Yung-Ching and Shing-Mei (2007) concluded that small quantities of alcohol can impair the ability to carry out secondary or dual tasks well before the effect on the mechanics of driving are demonstrated. The reason for this is because alcohol increases the workload required to divide attention which causes impairment in executive function to occur first. While the findings reveal some interesting factors to consider, a small sample size (N = 8) was used in the study which limits the extent to which the results can be generalized. A larger sample would ideally need to be studied so the results could be better generalized to the true population as a whole. However, the results of Yung-Ching and Shing Mei (2007) are supported by the fact other studies which have used much larger samples (N = 168) with an equal ratio of male and female participants revealed similar results (National Traffic Highway Safety Association, 2000).

While studies have consistently shown that alcohol induced impairments are worse in conditions where there is distraction, the threshold in which impairment occurs does not always necessarily increase in stepwise, linear manner (Ogden & Moskowitz, 2010). Some studies have revealed that the effect of alcohol can plateau after a certain BAC is reached. For example, Verster, Wester, Goorden, van Wieringen, Olivier and Volkerts (2009), examined the effect of multiple BACs (0.00, 0.02%, 0.05%, 0.08% & 0.10%). Results indicated

driving performance was impaired by alcohol at BACs ranging from 0.05 – 0.10% in both the single and dual task DASS conditions (Verster, et al, 2009). While a dose dependent difference was observed between a BAC of 0.02% and 0.05%, interestingly the level of alcohol impairment did not vary after the dosage exceeded 0.05% (Verster, et al, 2009). The fact that impairment levelled from a BAC of 0.05% suggests there is no real way of predicting at what point alcohol may have a significant effect on performance.

Inhibitory control

Alcohol has been known to increase impulsive behaviours such as where drivers may make risky attempts to overtake other vehicles or to run a red light, speed and more. Research has shown that alcohol impairs the ability to inhibit impulsive behaviours, which tends to increase in conditions where there is response conflict, where two actions have equal motivational value (Fillmore, Blackburn & Harrison (2007). For example, Fillmore, et al (2007), examined the effects of alcohol (0.56g/kg) on inhibitory control and response conflict using a driving simulator. Participants had to drive on a busy city road which had twenty intersections that were controlled by traffic lights. At five of the intersections, red traffic lights were displayed where participants were required to stop, while all of the other intersections had green or orange lights (Fillmore, et al, 2007). To create response conflict, a monetary incentive was given to the participants for completing the drive quickly but also for stopping at the red lights and driving carefully (Fillmore, et al, 2007). Findings revealed the impairing effect of alcohol was more significant in driving conditions where there was response conflict (Fillmore, et al, 2007). For example, alcohol combined with response conflict resulted in greater speed, increased brake reaction time and greater failures to

stop. Given this, response conflict tends to interact with the impairing effect of alcohol, promoting the likelihood of impulsive, risky driving behaviour (Fillmore, et al, 2007).

The effect of alcohol on response inhibition has also been demonstrated on cued go/no tasks, with both moderate and high doses of alcohol (0.05% & 0.08%) being associated with an increase in response inhibition failures (Fillmore, Ostling, Martin & Kelly, 2009). In a cued go / no go task, cues (go or no-go) are displayed on a computer screen either horizontally or vertically. Cues that were presented horizontally indicated the following target response was a go, 80% of the time and when displayed vertically the ratio was reversed (Fillmore, et al, 2009). On cued go/no go tasks, participants become reliant on the cues presented which establishes a prepotent response (pre-established motor pattern). When the cue does not correspond with the following target, the prepotent action is difficult to inhibit due to a response conflict (Fillmore, et al, 2009).

It is possible alcohol increases the likelihood of impulsive behaviour because it reduces the conscious control of intentional behaviour while automatic processes or influences remain unaffected (Fillmore, Vogel-Sprott & Gavrilescu, 1999). Easton, Vogel and Sprott (2000) demonstrated alcohol impaired response inhibition flexibility on a change task. The change task is similar to the cued go/no go tasks, but it requires participants to provide an alternative response to no-go targets which measures flexibility. Interestingly, the results showed that the ability to inhibit an on-going response in order to initiate an alternative response was significantly impaired with a BAC of 0.08% (Easton, et al, 2000). The effect of alcohol on the relative influence of controlled and automatic cognitive processes has also been demonstrated by Fillmore, et al (1999), in an experiment

which used a word stem test. In the word stem test, participants were required to study a list of 40 words that are presented one at a time on a computer screen for 1.5 seconds. Following the study phase, word stems with the first three letters of a word were displayed which required participants to fill in the blanks. In one condition, words that were previously shown could be used to complete the word stems, but in the other test session participants could only use words they had not studied (Fillmore, et al, 1999). The results revealed that participants made more action slips when they were required to complete the word stems with words they had not previously studied. An increase in action slips meant that alcohol impaired the conscious control over behaviour and that automatic processes generated responses that opposed the intended action. Overall, when different words had to be used to complete the word stems it created a response conflict between intentional and automatic processes. When alcohol was consumed cognitive control was impaired so automatic response patterns had the greatest influence (Fillmore, et al, 1999).

Interestingly, some studies have been able to identify some areas of brain function which shed light on as to why automatic response patterns may have the greatest influence. Gundersen, et al (2008) examined the effects of alcohol (BACs of 0.00, 0.02 & 0.08%) on neuronal activation using functional magnetic imaging and found alcohol impairs cognitive functions in the dorsal anterior cingulate cortex (dACC) and cerebellum, particularly at a BAC of 0.08%. Studies have shown the dACC is important for cognitive control, decision making and error monitoring while the cerebellum plays a vital role in the control of voluntary and involuntary motor actions (Weissman, Gopalakrishnan, Hazlett & Woldorff, 2004).

Biphasic effects of alcohol

Alcohol has been shown to have a biphasic affect which means impairment does not solely depend on a specific BAC level. With advances in research some aspects of impairment have been shown to even remain after blood alcohol levels return to zero. For example, Liu and Ho (2010) examined the difference between drunk and post alcohol driving with BACs of 0.00, 0.05, 0.08 and 0.10%. Speed variance was found to increase significantly following the consumption of alcohol but there was no difference in the post alcohol sessions. Scores from a NASA-TLX mental workload questionnaire which asked participants to rate the mental workload of tasks also revealed alcohol continued to have an effect, with highest values obtained in the post alcohol driving condition (Liu & Ho, 2010). In view of these findings it can be concluded alcohol produces a lingering impairment which can jeopardise road user safety (Liu & Ho, 2010). While it is clear alcohol induced impairments can persist even after BACs return to zero, other studies have shown some cognitive functions can recover while others remain impaired. Given this, different levels of impairment can be observed across subjective and objective measures, depending on which phase of the blood alcohol curve performance is tested. Cromer, Cromer, Maruff and Snyder (2010) examined the effects of varying levels of alcohol on people's cognitive functioning and subjective perception of intoxication. Cromer, et al (2010) found that as BAC levels increased, participants perceptions of intoxication were significantly impaired, but as BACs declined from a maximum level, subjective evaluations improved (Cromer, et al, 2010). In contrast, no tolerance effect between ascending and descending levels of alcohol intoxication occurred for impairments observed in executive functioning. For example, higher order cognitive processes like error monitoring and spatial short term memory

remained significantly impaired (Cromer, et al, 2010). Overall, a discrepancy was identified in the subjective perception of intoxication and participants' level of cognitive function; showing acute tolerance can develop (Cromer, et al, 2010).

Acute tolerance is used to describe a mismatch in the recovery of cognitive impairments across the ascending and descending limb of the blood alcohol curve. There have been a number of studies which have confirmed the development of acute tolerance effects. For example, Scheweizer and Vogel-Sprott (2008) reviewed a number of studies which measured the effects of moderate alcohol ingestion across both phases of the blood alcohol curve. Scheweizer and Vogel-Sprott (2008) found there was a significant mismatch between speed and accuracy in cognitive performance. Reaction time was found to rise on the ascending limb of the alcohol curve, but decline as blood alcohol levels declined suggesting speed of cognitive performance was recovered (Scheweizer & Vogel-Sprott, 2008). In contrast, alcohol increased errors on all tasks involving inhibition, selective attention and information processing across each phase of the blood alcohol curve with no recovery of impairment (Scheweizer & Vogel-Sprott, 2008).

While some studies have indicated impairment either recovers as BACs decline or fails to diminish, other findings have also revealed an opposite can occur. For example, on verbal short term memory and visual memory tasks results revealed accuracy was not impaired on the ascending limb, but it deteriorated when BACs declined (Scheweizer & Vogel-Sprott, 2008). Acute tolerance effects may be seen as BACs rise, but not necessarily when they decline. Overall, the differences in the development of acute tolerance have important implications. According to this research, the incidence of 'protracted error' poses a threat to road safety because people may subjectively assume they are able to

drive when in fact cognitive processes remain significantly impaired (Scheweizer & Vogel-Sprott, 2008).

Aim of the present study

Different aspects of driving and cognitive performance have been shown to be impaired by alcohol within the legal limit, increasing the odds of being involved in a road crash. Various studies have shown a broad range of driving and cognitive skills like information processing, dual task performance can be affected by a range of alcohol dosages. The fact alcohol's effect can vary across individuals' raises the question as to whether there is a safe BAC level in which the vast majority of drivers are unlikely to be affected. Although there has been a considerable amount of research which has examined several areas of driving related behaviour to date, little research in New Zealand has been done. Since alcohol produces a wide array of effects across the blood alcohol curve, the current study sought to investigate the impact of low dose alcohol on simulated driving and cognitive performance across multiple BAC levels (0.00, 0.02, 0.05 & 0.08%) extending on other findings. The fact alcohol has been reported to produce lingering impairments even after BACs return to zero has importance and as many previous studies have tended to focus on ascending effect of alcohol rather than when BACs decline, further research in this area seemed warranted. In the present study asked 3 questions (1) if the effect of low dose alcohol was task dependent, that is, whether any observed impairment became greater as task complexity increased; (2) whether the effect of alcohol increased in a dose dependent manner for example, if decrements in performance were more evident at the highest BAC level; (3) if a mismatch in acute tolerance developed across subjective and objective measures as BACs increased and declined back to zero.

METHODS

Participants

A sample of 30 participants was recruited from the local community by word of mouth and via notices placed on the university psych café virtual notice board (see Appendix A). Participants were aged between 20 – 64 years, with a mean age of 40.03 years (SD = 12.63), 14 were male and 16 were female. In terms of ethnicity, 86.7% of the sample identified themselves as NZ European, 6.7% Maori, 3.3% NZ/European Maori and 3.3% NZ European/Tongan.

To take part in the study, participants were required to possess a current full New Zealand driver license, with a minimum of three years driving experience. The average number of years participants held a full driver's license was 21.90 years (SD = 13.21) with a range of 3 – 47 years. All subjects were non-smokers and had a history of drinking at least one alcoholic beverage per week. The mean drinking frequency was 5.90 standard drinks per week (SD = 6.31). To ensure the consumption of alcohol would not exacerbate any medical condition or interact with any medication, all participants' completed a detailed eligibility questionnaire (see Appendix D). Each participant reported they had no medical, psychological, substance or alcohol abuse disorder. The study was approved by the School of Psychology Ethics Committee.

Experimental design

Participants were randomly allocated to one of three groups (0.00%, 0.02% and 0.05%) that were based on target BACs. The participants were blind to the group they were assigned to and were assessed at multiple points. There were four experimental trials which comprised a baseline, alcohol and two post alcohol

sessions (see Figure 1). Although baseline measures were obtained for the alcohol groups, a control, placebo group was incorporated as a way of measuring any practice effects which might confound results.

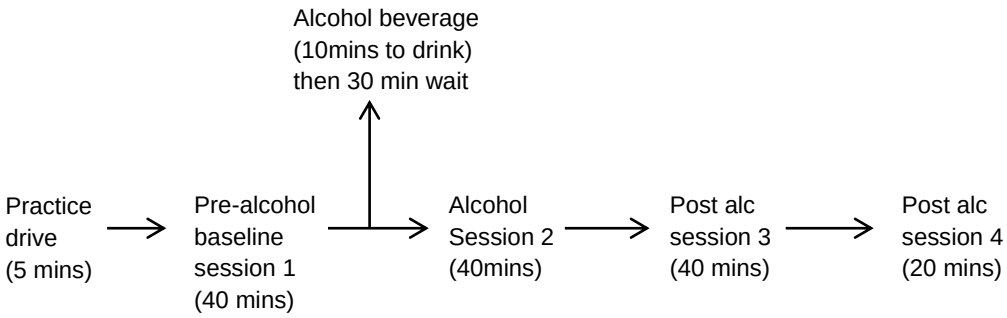


Figure 1. Format of the experimental design.

Alcohol administration

The amount of alcohol (grams) needed to reach a target BAC (0.02%, 0.05% and 0.08%) was calculated for each participant using the Widmark formula (as cited in Liu & Fu, 2007):

Men's TBW = 2.447 − 0.09516 × Age + 0.1074 × Height (cm) + 0.3362 × Weight (kg)

Women's TBW = −2.097 + 0.1069 × Height (cm) +0.2466 × Weight (kg)

Alcohol dose (g) = [(10 × BAL × TBW)/0.8] + 10 × MR × (DDP + TPB) × (TBW/0.8)

TBW is total body water which is adjusted for gender differences in total body water content. BAL is the target blood-alcohol level, MR is the metabolic rate generally set at 0.015 g/100 ml/h, DDP is duration of the drinking period (0.166 hrs) and TPB is the time to peak BAL set at 0.5 h.

Each dose of alcohol was mixed with pure orange juice to mask the flavour of the vodka, with a total liquid volume of 450mls per cup. The quantity of juice was adjusted depending on the amount of alcohol added, to keep the total volume equal. For example, one participant was given 158mls of vodka which was topped up with 292mls of orange juice. The placebo beverage contained the same fruit juice and volume of liquid per cup without alcohol. In order to help standardize the administration of alcohol, participants were asked to abstain from drinking any alcohol 24hrs prior to the experiment and to refrain from ingesting anything 2 hours before testing. Fasting 2 hours prior helped ensure the alcohol was absorbed rapidly. Participants were given 10 minutes to consume the beverage and then they waited another 30 minutes before the experimental trial was started.

Experimental groupings

To begin with participants were going to be assigned to groups on the basis of alcohol dosage. However, the Widmark formula was found to underestimate the amount of alcohol needed to achieve certain BAC levels. As the effect of alcohol varied, participants were allocated to groups according to their BAC rather than the dose given to reach a target BAC (see Figure 2). There were 6 participants' who received a pre-calculated dose of alcohol to achieve a target BAC of 0.05% that ended up with BACs of 0.022%, 0.028%, 0.03%, 0.025% and 0.03%. As there were problems in even achieving the low to moderate target BAC levels the original plan to have a 0.08% group, also had to be aborted due to the problems with the dosing method. Some people struggled to consume the 0.05% dose so the amount of vodka required to get BACs up to 0.08% would have been too excessive for participants to drink within 10 minutes

without being sick. The average BAC for the 0.02% group was 0.023% (SD 0.006) with a range of 0.015 – 0.030 and the 0.05% group's average BAC was 0.042% (SD 0.007) ranging from 0.034 – 0.058. There was no detectable alcohol in the control group. The average dose of alcohol given in the 0.02% group was 102.50mls and for the 0.05% group 119.94mls.

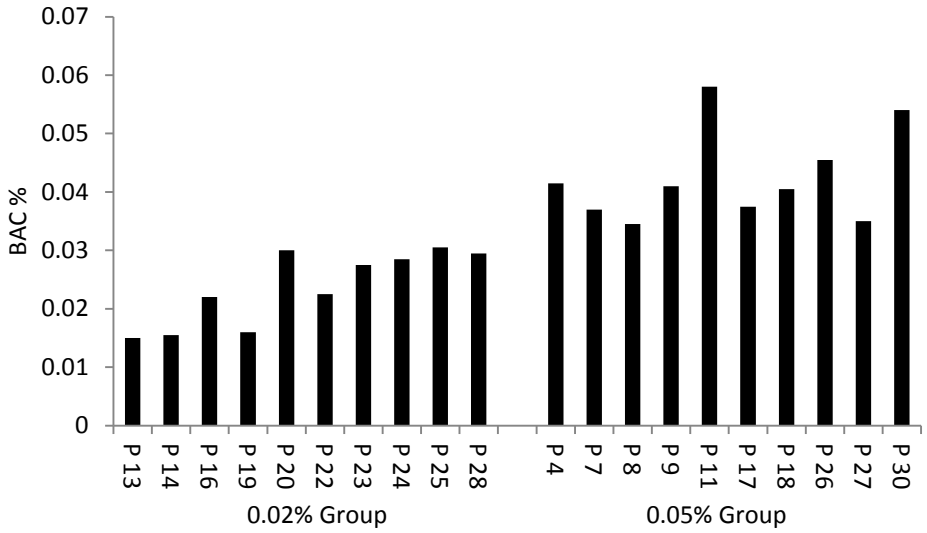


Figure 2: Participants allocated to each alcohol group based on actual BAC, 30 minutes after the end of drinking.

Materials and Measures

Simulator

This study used the University of Waikato driving simulator (see Figure 3). The simulator consisted of a full car body (BMWi) which had all of the characteristics and interior features (automotive display, steering wheel, direction indicators, horn, mirrors etc) of a normal car. The car was positioned 2.42 metres in front of a large central projector screen with two other peripheral screens connected at an angle of 62 degrees. Visual driving scenes were projected on to the three screens which were slanted away from the driver at an angle of 14

degrees producing 175 degree by 41 degree vertical forward view. The image projected central to the driver was 2.64 metres wide and 2.10 meters high at a resolution of 1280 by 1024 pixels. Visual images projected onto the other two peripheral projector screens were 2.65 by 2 metres at resolution of 1024 by 768 pixels. The car was also equipped with two side mirrors and a rear vision mirror which had colour LCD screens attached (12.06 by 7.49 cm in size) with a resolution of 640 by 480 pixels to provide drivers with rear view driving scene. To help emulate the feel of a real car the steering was set up to provide tactile feedback to the driver and a sub-woofer underneath the car and four other speakers inside the car were used to generate engine and road noise. Information related to driver such as speed, lane position, lateral displacement, braking, acceleration and steering variability were automatically recorded by the simulation software (Charlton & Starkey, 2011).

Figure 3. The University of Waikato driving simulator.

Simulated driving

For the simulated driving, participants' drove on four roads which were each 12 km long with a standard lane width. Each road was the same type, comprising a two lane rural highway with some straights, gentle hills, sweeping corners and one tunnel. The sky was a twilight colour and the road was a dark grey with white and or double yellow centre line markings on different segments along the route. There were some buildings and trees featured in the roadside landscape which was the same for every road.

To manipulate task demand, all the roads were dived into two light traffic (LT) and heavy traffic (HT) segments which were each 3km long (see Table 1). In the low traffic segment there were 3 vehicles which approached in the opposite lane, spaced one kilometre apart. In contrast, the high traffic segment had 69 oncoming vehicles which were spaced at different distances. Speed limit signs were posted on both sides of the road at every kilometre, with the left sign facing the direction of the driver and the other facing away. The speed sign changes were varied slightly across each road to prevent participants from becoming too familiar with each drive (see Table 1). The changes in the speed limit were also reversed across each road so that the speed transitions were not always the same.

Table 1. Signage, speed changes and traffic conditions for Roads 1 - 4.

	Light traffic (LT)						Heavy traffic (HT)						Light traffic (LT)						Heavy traffic (HT)						
Distance (km)	0	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5	5.5	6	6.5	7	7.5	8	8.5	9	9.5	10	10.5	11	11.5	12
Road 1 (speed km/h)	100	100	100	70	70	70	70	70	100	100	100	100	100	100	80	80	80	80	80	80	100	100	100	100	100
Road 2 (speed km/h)	100	100	100	100	80	80	80	80	80	100	100	100	100	100	100	70	70	70	70	70	100	100	100	100	100
Road 3 (speed km/h)	100	100	100	70	70	70	70	70	70	100	100	100	100	100	100	80	80	80	80	80	80	100	100	100	100
Road 4 (speed km/h)	100	100	100	100	80	80	80	80	80	80	100	100	100	100	100	100	100	70	70	70	70	70	100	100	100

Driving performance measures

Hazard car detection

On Roads 1 – 4, a car (Audi, Camaro or BMW325) was placed on a left-hand side road 11.89kms from the start. On Roads 2 and 4 the hazard car pulled out 1.65 metres in the traffic lane, but on Roads 1 and 3 it remained stationary. The car could be seen sitting on the side road 413m ahead and was triggered to move forward when the participants' vehicle was 313m away. Two other cars were placed on different left-hand side roads (8.57 km & 5.14 km from the start) on Roads 1- 4 to serve as decoys and help minimise any familiarisation or expectancy effects. Participants' reaction times to the hazard cars were recorded in terms of the time taken to react by removing their foot from the accelerator (accelerator reaction time or ART) and move it to the brake pedal (brake reaction time or BRT). Both ART and BRT were calculated from the time the hazard car started moving into the traffic lane. The number of times participants crashed in to the hazard cars was also recorded.

Centreline crossings

Participants' steering performance was assessed by measuring the number of times their vehicles right wheels crossed the centreline into the oncoming traffic lane. The total number of line crossings was recorded in each drive and averaged for each group.

Speed change task

The speed change task was used to assess driver's perceptual abilities in terms of being able to monitor and adapt their speed respectively. Speed signs were posted at every kilometre on the left hand side of the road, participants had

to identify changes in the speed limit while maintaining an appropriate lane position. The amount participants either increased or decreased their speed 100m before and after the posted speeds changed was recorded. There were four speed transition scores obtained on each road these included 100 - 70k, 100 -80k, 70 - 100k and 80 - 100k. The maximum speed participants drove within the low traffic and high traffic 100km/h, 70km/h and 80km/h speed zones was also recorded. There were two maximum speeds for each light and heavy (70, 80 and 100km/h) zone, giving a total of 6 measures.

Integrated visual and auditory continuous performance test (IVACPT)

A computer administered integrated visual and auditory continuous performance test (IVACPT) obtained from BrainTrain (2012), used as a supplementary test for the current study. The IVACPT is normally used to assist in the diagnosis of ADHD but has also been used to assess other attention /impulse disorders and medication effects with low to moderate test-retest reliability (Strauss, Sherman & Spreen, 2006). As alcohol has been shown to affect different aspects of cognitive performance in other studies the IVACPT seemed a relevant choice.

The IVACPT was administered using a laptop computer placed in the same room with the driving simulator. The test contained a warm up phase, practice test, a main test and a cool down period. For the two minute warm up period, participants had to respond to twenty visual and auditory targets presented separately**.** Participants were instructed to respond only when they saw or heard a "1" (rather than a "2"). The practice test involved the presentation of visual and auditory targets in random order. The main test consisted of 500 trials in which an equal number of auditory and visual targets (1's) and non- targets (2's) were

presented. The 500 trials were divided into five 100 trial blocks in which the target (1's) and non-target (2's) ratio was presented in pseudo random order and lasted 15 minutes. In the first 50 trials, the visual and auditory presentations were predominately targets (1's) while the remaining stimuli were non-targets (2's) which created a higher task demand. In the second set of 50 trials, an opposite target/ non-target ratio was presented meaning there were more non-targets (2's) versus targets (1's) forming a low task demand condition. The cool down phase involved the presentation of visual and auditory targets as for the warm up period (BrainTrain, 2012).

The IVACPT provides a number of quotient scores which can be divided into four categories. There is a full-scale response control quotient and full scale attention quotient score, which provides a summary how individuals performed on the visual and auditory modalities combined. Two separate full scale auditory and visual quotients for attention and response control which are each based on six primary scales are also provided. Individual quotients for each primary scale are also produced. For this study only the Full scale response control and attention quotients were utilised.

Breathalyser

An Alcomate AccuCell AL9000 professional grade breathalyser was used to measure participants' breath alcohol level (BrAc), which had a detection range of 0.000 – 0.400% BAC with a sensor accuracy of +/- 0.005%. The breathalyser automatically converted BrAc to equivalent BAC units (AK Solutions, 2012).

Subjective measure of intoxication

To obtain a measure of participant's subjective perception of intoxication a visual-analogue scale was used (Cromer, et al, 2010). Individuals in each group were asked to rate how intoxicated they felt by placing a vertical mark through a 15cm line with left side (0cm) indicating "not at all" and the right side (15cm) indicating "very much" The millimetre distance was used as measure of each person's subjective level of intoxication. The subjective measure of intoxication was administered at the end of each experimental session (see Appendix F).

Eligibility questionnaire

An eligibility questionnaire was formulated which outlined the inclusion criteria for the study which was used to screen participants. Questions covered the type of driving license participants held, whether they had prior experience in a simulator and how much alcohol they consumed per week. Participants were asked whether they had a medical condition, if they were taking any medication or had a history of substance abuse. Those who were female were also asked whether they were pregnant or breast feeding (see Appendix D).

Demographic and driving questionnaire

To obtain demographic information and details about each participant's driving history an additional questionnaire was devised. Questions covered gender, whether participants had any driving infringements, prior driving convictions or motor vehicle crashes. Participants were also asked to provide details about how much alcohol they consumed per week and what their weight and height was (see Appendix E).

Procedure

Participants who expressed interest in taking part in the experiment were explained the purpose of the study and sent an information sheet and a copy of the eligibility questionnaire to complete. An instruction sheet that provided additional details about the experiment (see Appendix B) was emailed to the participants and a time was arranged to meet them at the driving simulator. On the day of the experiment, participants were given the opportunity to ask any questions and informed consent was obtained (see Appendix C). Each participant's BrAC level was then measured using the breathalyser to ensure no alcohol had been consumed prior to testing.

After individuals were confirmed to have a BAC level of 0.00% they completed a demographic and driving questionnaire (see Appendix E) and then had a practice session (5 minutes) to become familiar with the driving simulator to ensure it didn't make them feel sick. After the practice session each person was given the opportunity to ask any questions or discuss any concerns they might have. The first test session which included the baseline drive, computer administered test (IVACPT) and subjective measure of intoxication was then completed. Once the pre-alcohol session had been carried out, those in the alcohol groups were given pre-calculated doses of vodka mixed in orange juice to be consumed within 10 minutes. Participants in the control group were given a placebo beverage without alcohol. After consuming the beverage, participants waited in an assigned area for 30 minutes at the end of the 10 minute drinking session where they could relax or read. The three remaining sessions (intoxicated and two post alcohol sessions) were completed at the specified interval, BAC levels were measured at the start and end of every test session at the same time

points. When the experiment was completed participants were asked to remain until their BAC level returned to zero. Participants were debriefed about how much alcohol they had drunk and the group that they were in and questions they had were answered. Participants were then given a $10 MTA voucher and were taken home by their designated driver.

Statistical analysis

IBM SPSS version 20.0 was used to analyse the data. A between group mixed ANOVA was conducted to evaluate whether alcohol had an effect on simulated driving and cognitive performance. Some of the factors analysed included Group (between) and Traffic Density (within). Greenhouse glassier corrections were used when sphericity could not be assumed. Post hoc tests were conducted when the ANOVA revealed a significant difference to identify the source of difference, the significance level was set at $p = 0.05$. Bonferroni adjustment to the alpha level was used for each comparison to correct for multiple comparisons. In cases where there was a significant interaction additional analyses of simple effects were carried out by using a univariate ANOVA. Pearson product moment correlation coefficients were also conducted as a supplement to examine the relations between BACs and subjective intoxication ratings. Variable in the study that were analysed included accelerator reaction time, brake reaction time, crashes, maximum speed within speed zones, speed changes positive and negative, centreline crossings, IVACPT global full scale response control and attention quotients , BAC and subjective intoxication ratings.

RESULTS

Reaction times

The time (seconds) it took for participants to take their foot off the accelerator in response to a hazard car (ART) which pulled out on Roads 2 & 4 was calculated. Shown in Figure 4 are the group averages which were analysed to investigate whether alcohol impaired accelerator reaction time. Note that negative reaction times refer to accelerator responses that occurred before the hazard car began moving i.e. anticipating reactions. It can be seen that each group's accelerator reaction time (ART) decreased from Road 2 to Road 4, with the 0.05% group having the quickest ART compared with the other two groups on Road 4. Analysis with a 3 (Group) x 2 (Road) mixed ANOVA of the variation observed between each group's ART was not statistically significant $F(2, 25) = 0.273$, $p = 0.763$, $\eta p^2 = 0.021$. There was also no main effect of Road $F(1, 25) = 3.011$, $p = 0.095$, $\eta p^2 = 0.108$ or Road*Group interaction evident $F(2, 25) = 0.405$, $p = 0.671$, $\eta p^2 = 0.031$.

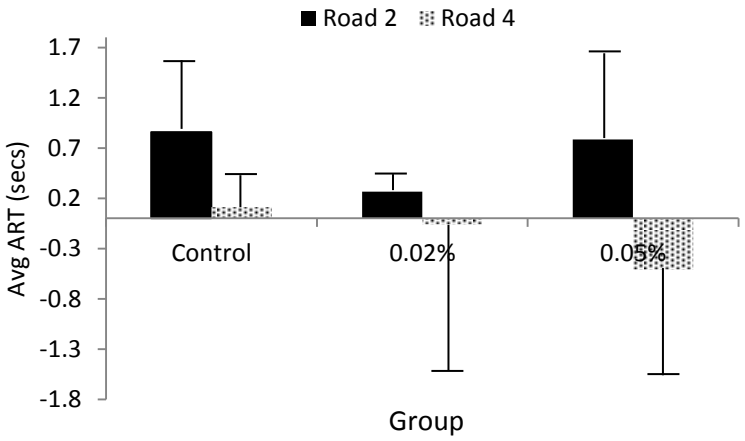


Figure 4. The average accelerator reaction time (ART) for each group in response to a hazard car that pulled out on Road 2 & 4. Error bars indicate 95% confidence intervals.

Shown in Figure 5 are the average brake reaction times (BRTs) for each group. Looking at the figure, the control group's average BRT was similar on Roads 2 & 4. In contrast, the 0.02% and 0.05% groups' average BRT was faster on Road 4 than on Road 2. Brake reaction time (BRT) was analysed by 3 (Group) x 2 (Road) mixed ANOVA. A significant effect of Road was obtained $F(1, 21) = 7.946$, $p = 0.010$, $\eta p^2 = 0.275$ but no significant difference between the groups $F(2, 21) = 0.152$, $p = 0.860$, $\eta p^2 = 0.014$ or a Group*Road interaction was observed $F(2, 21) = 0.908$, $p = 0.419$, $\eta p^2 = 0.080$.

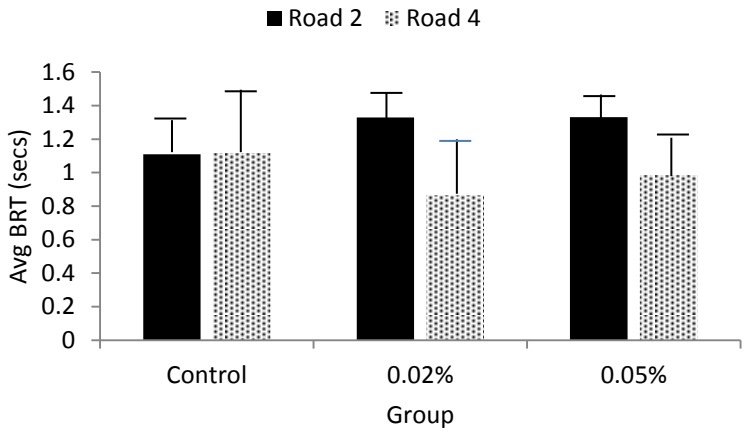
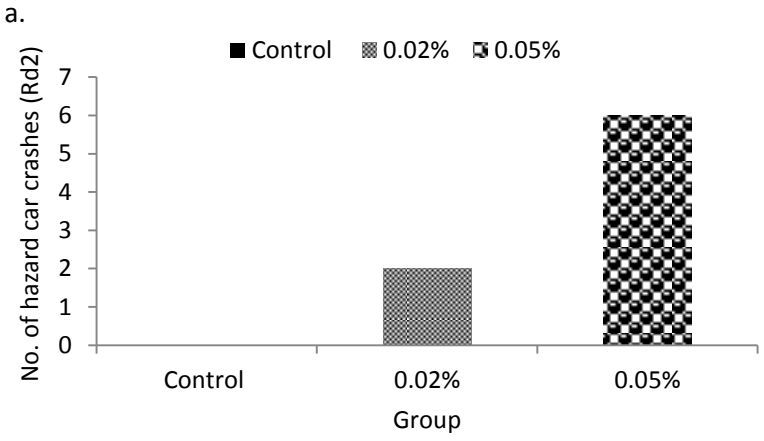


Figure 5. The average brake reaction time (BRT) for each group in response to a hazard car that pulled out on Road 2 & 4. Error bars indicate 95% confidence intervals.

In summary, a practice effect was observed for both ART and BRT where participants' reaction time decreased from Road 2 to Road 4, but there was no effect of alcohol dose.

Hazard car crashes

The total number of times participants in each group crashed into the hazard car on Roads 2 and 4 was calculated to assess the effects of alcohol on vehicle control. Figure 6 (a) and (b), presents the number of times participants in each group crashed into the hazard car on Road 2 and 4. Looking at the figure, all of the participants' in the control group avoided crashing while a number of participants' in the other groups crashed into the hazard car. On Road 2, the 0.05% group had the highest number of crashes but on Road 4 the 0.05% and 0.02 % group had a similar number of crash incidents. Apart from one participant in the 0.05% group who crashed once on both Road 2 and Road 4 all of the other crash incidents were caused by different participants. Analysis with a 3 (Group) x 2 (Road) mixed ANOVA showed a significant main effect of Road $F(1, 27) = 9.529$, $p = 0.005$, $\eta p^2 = 0.261$ and Group $F(2, 27) = 4.061$, $p = 0.029$, $\eta p^2 = 0.231$. A significant interaction between Road*Group was also obtained $F(2, 27) = 5.559$, $p = 0.010$, $\eta p^2 = 0.292$ revealing participants in the 0.05% group crashed a significantly higher number of times on Road 2 in comparison to the control and 0.02% group.



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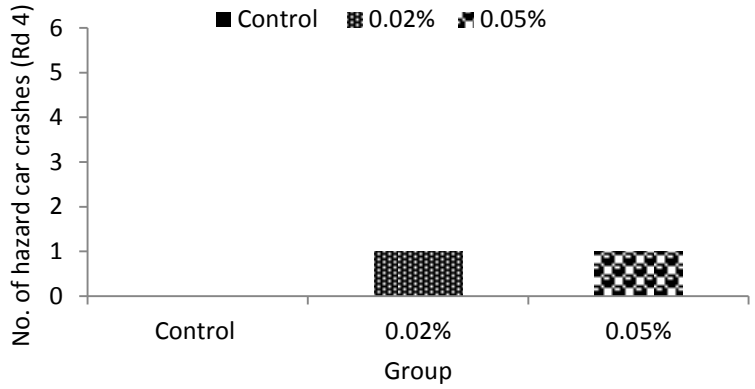


Figure 6. The total number of hazard car crashes for each group with (a) showing the total number of crashes on Road 2 and (b) the number of crashes on Road 4.

Average number of centreline crossings.

Figure 7 shows the average number of times the centreline was crossed in each group, which was calculated to assess whether alcohol affected driving precision. In panel (a), one participant's score in the control group was identified as an outlier so this was removed from the data set when the averages were calculated and analysed.

Looking at the figure, the 0.02% group had a marginally higher average number of centreline crossings than the control and 0.05% group, on Road 1. There did not seem to be any major difference in the group averages across Roads 2-3. On Road 4, the 0.02% and 0.05% group crossed the centreline more frequently. The average number of centreline crossings was analysed by a 3 (Group) x 4 (Road) mixed ANOVA. Results showed there was no main effect of Group $F(2, 26) = 0.103$, $p = 0.902$, $\eta p^2 = 0.008$ or significant Road*Group interaction $F(4.351, 56.560) = 0.344$, $p = 0.862$, $\eta p^2 = 0.026$. Greenhouse glassier adjustment was used because sphericity could not be assumed. However, there was a main effect of Road $F(2.175, 56.560) = 6.450$, $p = 0.002$, $\eta p^2 = 0.199$.

Pairwise comparisons showed a significant increase in the number of centreline crossings between Road 3 – 4 $p < 0.001$, all other Roads were $> p = 0.055$.

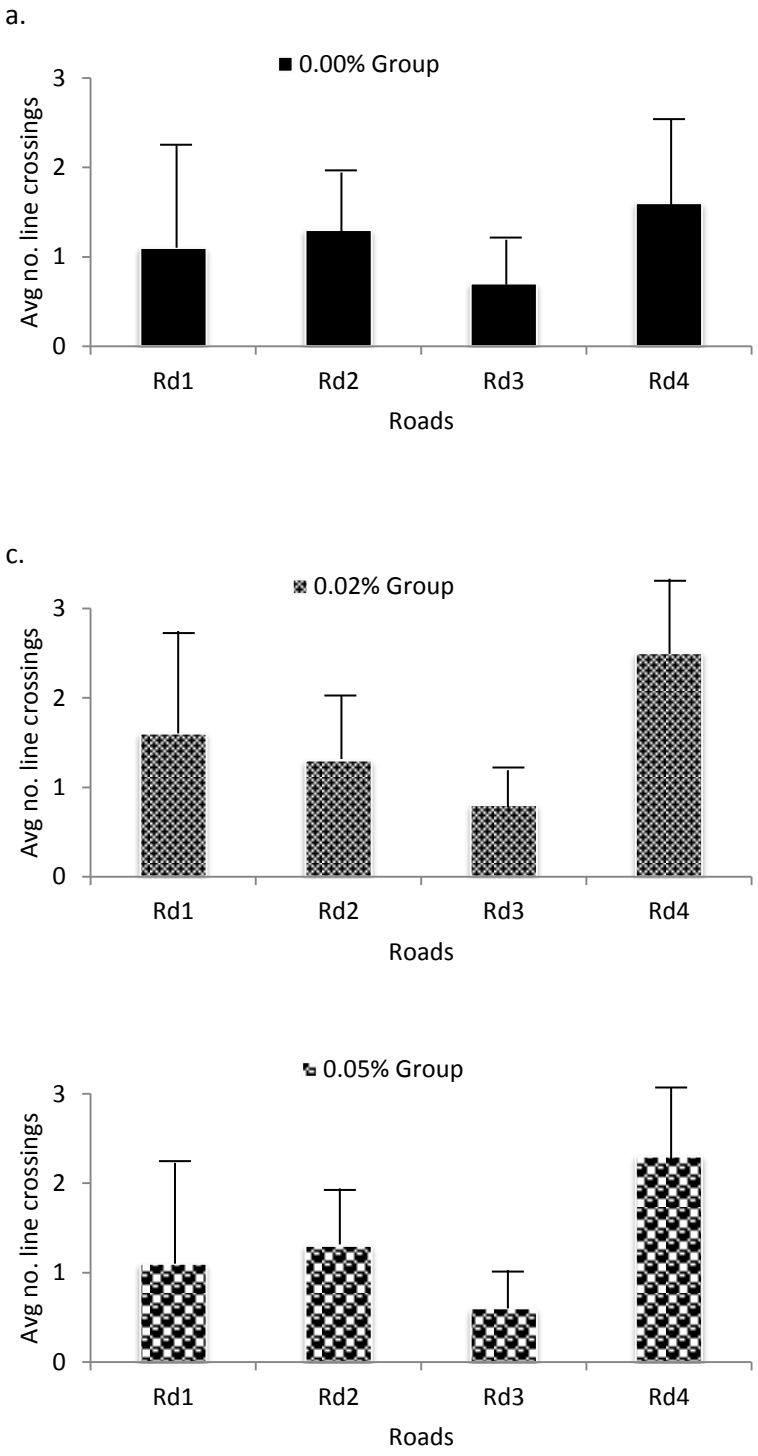


Figure 7. The average number of centreline crossings for each group with (a) showing the 0.02% group, (b) the 0.02% group and (c) the 0.05% group. Error bars indicate 95% confidence intervals.

Speed Change

Figure 8 displays the average amount speed was decreased for each group 100m before and after the speed signs changed from 100 – 70km/h on Roads 1 - 4 in the light traffic zone. Looking at the figure, the control and 0.02% groups' average speed change was fairly consistent across the four roads. The 0.05% group appeared to have a sharper speed reduction on Road 2 but then had a smaller speed reduction on Road 3, suggesting participants in the group experienced more difficulty in regulating their speed. A 3 (Group) x 4 (Road) mixed ANOVA of the average speed differences revealed there was no main effect of Group, $F(2, 27) = 0.003$, $p = 0.997$, $\eta p^2 = 0.001$ or Road, $F(1.825, 49.287) = 0.532$, $p = 0.574$, $\eta p^2 = 0.019$. There was also no significant Road*Group interaction $F(3.651, 49.287) = 0.202$, $p = 0.924$, $\eta p^2 = 0.015$ and no significant effect of Road, $F(1.825, 49.287) = 0.532$, $p = 0.574$, $\eta p^2 = 0.019$.

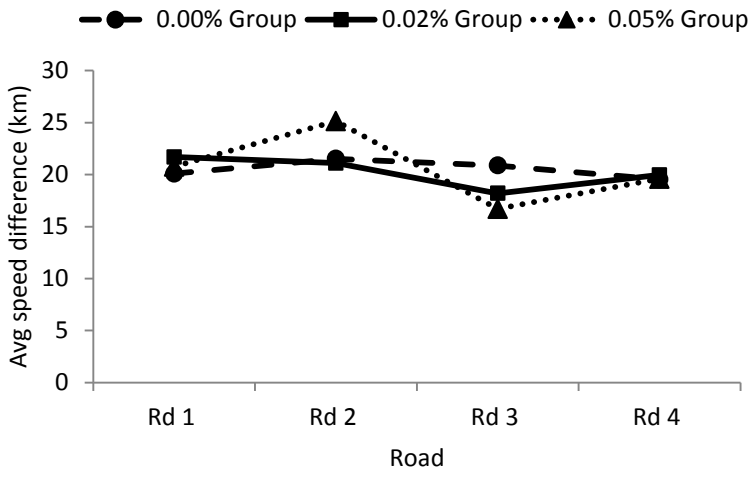


Figure 8. The average amount each group changed speed 100m before/after the transition from 100 - 70km/h for each group, Roads 1-4.

Figure 9 displays the average amount speed was decreased for each group 100m before and after the speed signs changed from 100 – 80km/h on Roads 1 - 4

(in the light traffic zone). There was a marked difference in the amount participants in each group decreased their speed on Road 3 compared to Roads 1, 2 and 4. On Roads, 1, 2 and 4 each group appeared to drive slower displaying a greater reduction in speed, but on Road 3 speed was not reduced to the same extent, indicating participants' maintained a higher speed. A 3 (Group) x 4 (Road) mixed ANOVA showed a significant effect of Road, $F(3, 81) = 13.052$, $p = 0.001$, $\eta p^2 = 0.326$ and pairwise comparisons showed the speed change on Road 3 was significantly lower than on the other three roads $< p = 0.05$. There was no main effect of Group, $F(2, 27) = 0.524$, $p = 0.598$, $\eta p^2 = 0.037$ or significant Road*Group interaction, $F(6, 81) = 2.032$, $p = 0.071$, $\eta p^2 = 0.131$. Variation in where the 100-80km/h speed transition occurred, could explain why participants' had a smaller speed reduction on Road 3 due to changes in the road geometry.

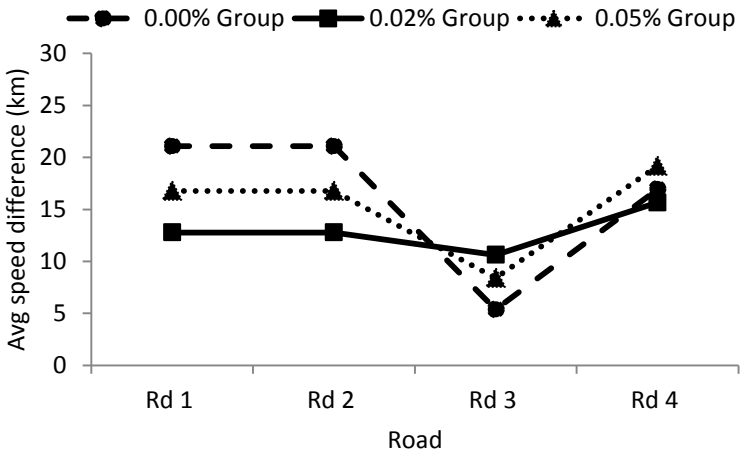


Figure 9. The average amount each group changed speed 100m before/after the transition from 100 -80km/h for each group, Roads 1-4.

The average amount speed was increased 100m before and after the signs changed from 70 – 100km/h in the heavy traffic zone was calculated. Figure 10 displays each groups average speed increase for the speed transition from 70 – 100km/h, on Roads 1 - 4. Looking at the figure, the control and 0.05% groups'

average speed increase varied slightly across Roads 1 - 4, while the 0.02% group's speed change remained relatively even, but the differences across each group was not large. Analysis with a 3 (Group) x 4 (Road) mixed ANOVA of the speed changes showed there was no main effect of Group, $F(3, 8) = 2.224$, $p = 0.092$, $\eta p^2 = 0.076$ or Road, $F(3, 8) = 2.224$, $p = 0.092$, $\eta p^2 = 0.076$. There was also no significant Road*Group interaction, $F(6, 81) = 0.652$, $p = 0.688$, $\eta p^2 = 0.046$.

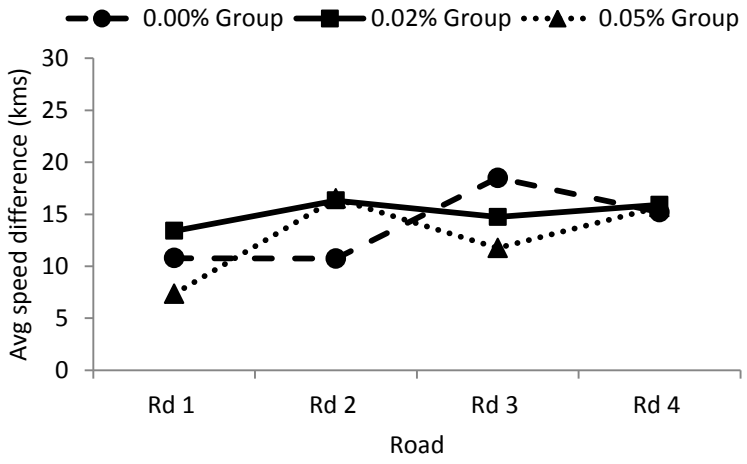


Figure 10. Average speed difference 100m before/after signs changed in the 70 - 100km/h speed transition for each group, Roads 1 - 4.

Figure 11 displays the average amount speed was increased for each group 100m before and after the speed signs changes from 80 – 100km/h on Roads 1 - 4 in the heavy traffic zone. Looking at the figure, there were no obvious differences in the average amount each group increased their speed. An analysis with a 3 (Group) x 4 (Road) mixed ANOVA showed there were no significant differences across each Group $F(2, 27) = 0.620$, $p = 0.545$, $\eta p^2 = 0.044$. There were no significant differences in the average amount speed was increased across each Road $F(3, 81) = 2.201$, $p = 0.094$, $\eta p^2 = 0.075$ and no significant interaction between Road*Group was revealed $F(6, 81) = 1.197$, $p = 0.316$, $\eta p^2 = 0.081$.

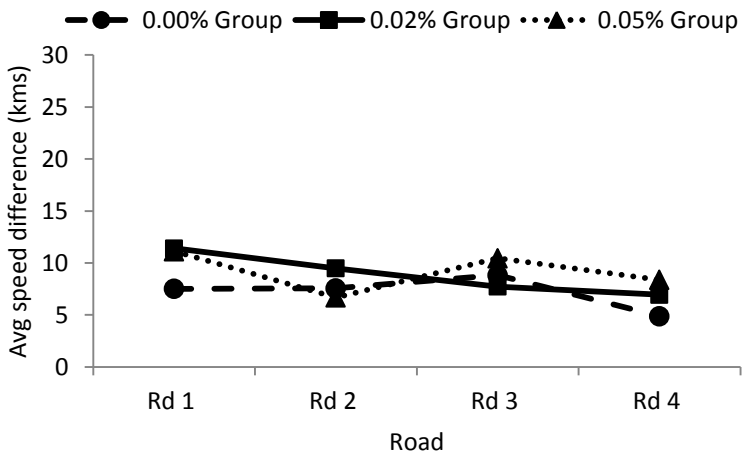


Figure 11. Average speed difference 100m before/after signs changed in the 80 - 100km/h speed transition for each group, Roads 1 - 4.

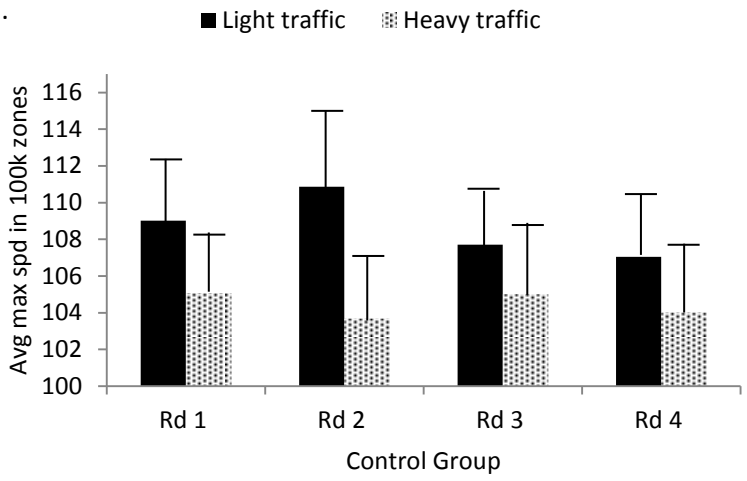
Overall there was no effect of alcohol on speed regulation (70 – 100km/h, 80 – 100km/h, 100 – 70km/h & 100 - 80km/h) but a main effect of road was observed in the 100 – 80km/h speed transition. Participants in each group had a smaller speed reduction on Road 3 for the 100 – 80km/h speed transition which may have been due to differences in the road geometry.

Speed maintenance

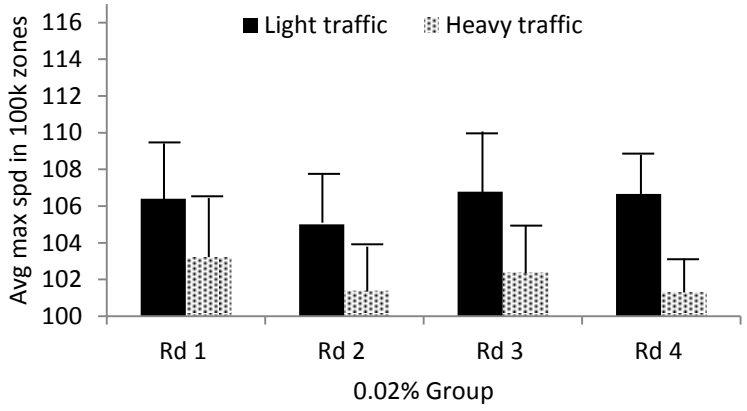
The average maximum speeds within the low and high traffic 100k zones for each group are plotted in Figure 12. Group averages were calculated to examine the effects of alcohol on speed regulation. Looking at the figure, it can be seen that all the groups' drove faster in the light traffic (100km/h) zone and slower in the heavy traffic (100km/h) zone for all four roads. A 3 (Group) x 2 (Density) x 4 (Road) mixed ANOVA confirmed there was a main effect of traffic density, $F(1, 27) = 58.149$, $p = 0.001$, $\eta p^2 = 0.683$ revealing driving speed was significantly slower in the heavy traffic zones. While there was a small variation in each group's average speed across Roads 1 - 4, the effect of Road was not

significant F (2.032, 54.851) = 1.594, p = 0.212, ηp^2 = 0.056. There was no significant Road*Group interaction F (4.063, 54.851) = 0.548, p = 0.704, ηp^2 = 0.039, Density*Group interaction F (2, 27) = 0.992, p = 0.384, ηp^2 = 0.068 or Density*Road*Group interaction F (4.814, 64.991) = 1.109, p = 0.364, ηp^2 = 0.076. The results for Group were also not significant F (2, 27) = 0.713, p = 0.499, ηp^2 = 0.050 showing alcohol did not have a significant effect on speed maintenance.

a.



b.



c.

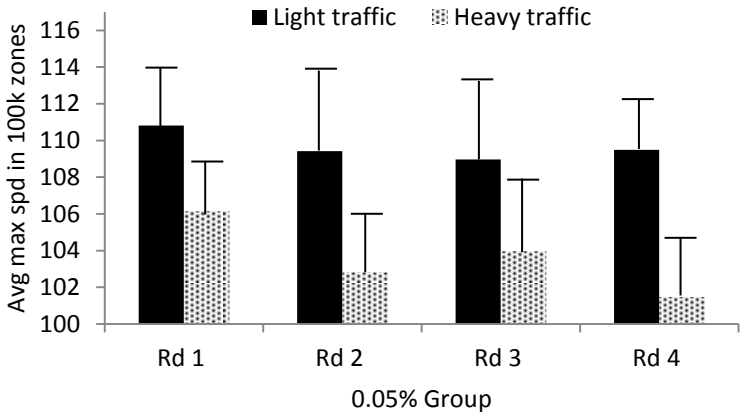
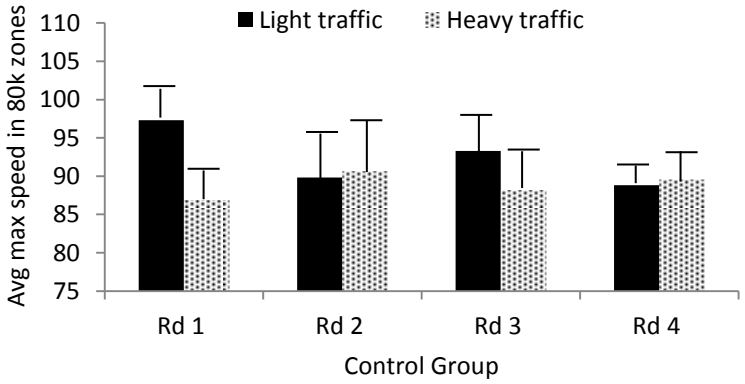


Figure 12. The average maximum speed each group drove within light traffic (LT) and heavy traffic (HT) 100km/h zones with (a) showing the control group, (b) the 0.02% group and (c) the 0.05% group. Error bars indicate 95% confidence intervals.

Figure 13 shows each group's average maximum speed in the light traffic (LT) and heavy traffic (HT) 80km/h zones. The maximum speed of each group varied across the low traffic and high traffic zones on Roads 1 and 3 while there was little difference observed on Roads 2 and 4. A 3 (Group) x 2 (Density) x 4 (Road) mixed ANOVA revealed there was a main effect of Density, $F (1, 27) = 30.076$, $p = 0.001$, $\eta p^2 = 0.527$ showing traffic density had a significant effect on the speed in which participants in each group drove. There was also a significant Density*Road interaction, $F (2.271, 61.327) = 14.802$, $p = 0.001$, $\eta p^2 = 0.354$ which showed there was a larger speed difference between light and heavy traffic zones on Road 1. No significant Density*Group interaction, $F (2, 27) = 1.475$, $p = 0.247$, $\eta p^2 = 0.099$ or significant effect of Road noted, $F (3, 81) = 1.491$, $p = 0.223$, $\eta p^2 = 0.052$. There was also no significant Road*Group interaction, $F (6, 81) = 1.247$, $p = 0.298$, $\eta p^2 = 0.085$ or main effect of Group, $F (2, 27) = 0.125$, $p = 0.883$, $\eta p^2 = 0.009$.

a.



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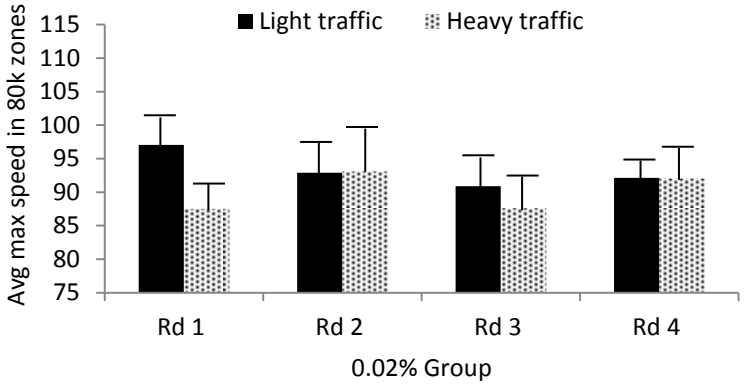


c.

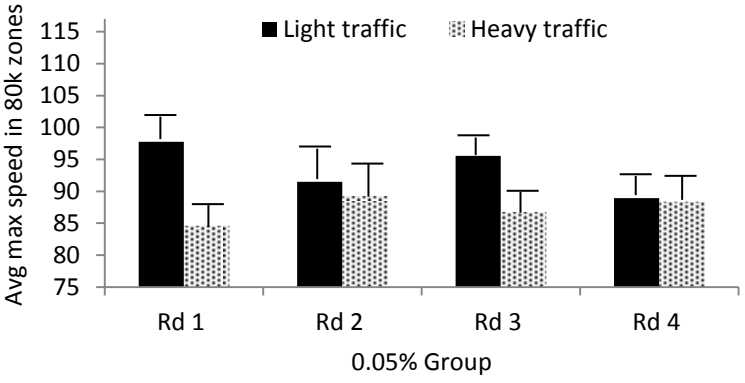
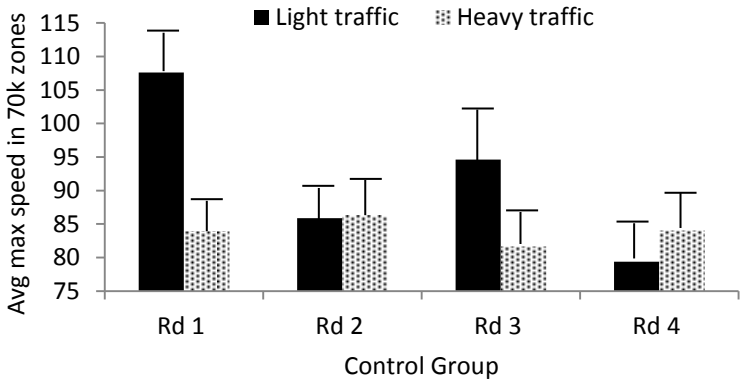


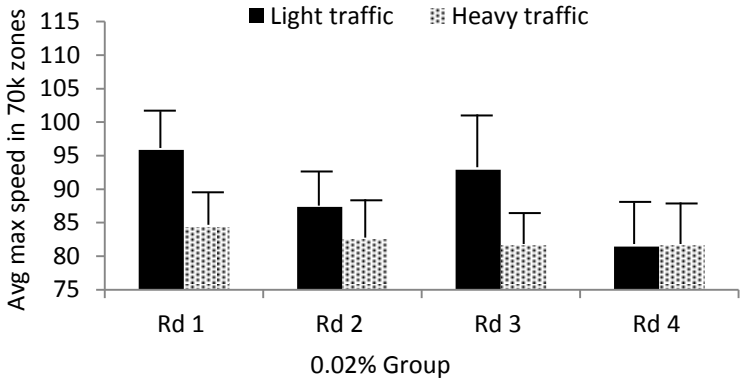
Figure 13. The average maximum speed each group drove in the light traffic (LT) and heavy traffic (HT) 80km/h zones with (a) showing the control group, (b) the 0.02% group and (c) the 0.05% group. Error bars indicate 95% confidence intervals.

Figure 14 displays the differences in each group's average speed within the light traffic (LT) and heavy traffic (HT) 70km/h zones. It can be seen, participants' generally drove faster in the LT70km/h zone than within the HT70km/h zone on Roads 1 - 3, suggesting both road and traffic volume altered driving speed. Panel 8c, indicates there was a marked difference in the average speed of the 0.05% group in the HT70km/h zone compared to the other two groups on Road 3 - 4. Those in the 0.05% group appeared to drive slower on Road 3 and faster on Road 4 in the HT70km/h zone. A 3 (Group) x 2 (Density) x 4 (Road) mixed ANOVA showed a main effect of Density, $F(1, 27) = 50.650$, $p = 0.001$, $\eta p^2 = 0.652$ confirming participants' drove faster in the light traffic (70km/h) zone and slower in the heavy traffic zone. There was main effect of Road, $F(3, 81) = 16.907$, $p = 0.001$ and pairwise comparisons showed significant differences between all Roads $p = < 0.048$ except Roads 2 - 3 $p = 0.589$ and Roads 2 – 4 $p = 0.214$. The results also revealed a significant Density*Road interaction, $F(3, 81) = 36.624$, $p = 0.001$, $\eta p^2 = 0.576$ and Density*Road*Group $F(6, 81) = 2.295$, $p = 0.043$, $\eta p^2 = 0.145$. There was a significant Density*Road*Group interaction $F(6, 81) = 2.295$, $p = 0.043$, $\eta p^2 = 0.145$, such that the participants' in the 0.05% group had a higher maximum speed on Road 4 than their speed on Road 3. This was reflected in a significant main effect of Road for the 0.05% group when tested with a one-way ANOVA, $F(3, 27) = 3.257$, $p = 0.037$, $\eta p^2 = 0.266$. There was no significant Road*Group interaction $F(6, 81) = 0.970$, $p = 0.451$, $\eta p^2 = 0.067$, $\eta p^2 = 0.385$ or main effect of Group was noted $F(2, 27) = 0.154$, $p = 0.858$, $\eta p^2 = 0.011$.

a.



b.



c.

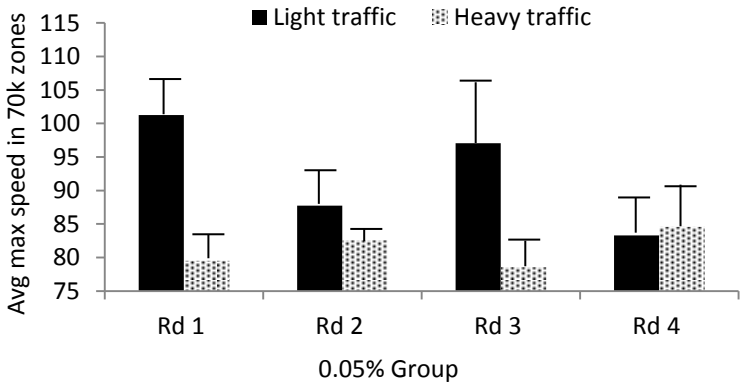


Figure 14. The average maximum speed participants' drove in the light traffic (LT) and heavy traffic (HT) 70km/h zones with (a) showing the control group, (b) the 0.02% group and (c) the 0.05% group. Error bars indicate 95% confidence intervals.

In summary, there was a main effect of traffic density on speed maintenance in each speed zone (70km/h, 80km/h & 100km/h). There was no main effect of Group but a Road*Density interaction in the 80km/h zone and a significant Road*Group*Density interaction in the heavy traffic (70km/h) zone was observed.

Sustained attention

To examine the effects of alcohol on cognitive control, full scale global quotients from the IVACPT were calculated, which provide an overall summary of participants' performance on the auditory and visual response control composite scales combined. Figure 15 shows the average Full Scale Response Control quotient for each group across the three time points. In the figure, all the groups scores declined slightly on over time but the control and 0.05% groups scores remained $\geq$ 90 which was within the IVACPT average performance standard score range. The 0.02% group scores dropped a little lower than 90 on the second and third measure, falling within the slightly impaired standard score range. A 3 (Group) x 3 (Time) mixed ANOVA revealed there was no significant effect of Time, $F (2, 54) = 2.585$, $p = 0.085$, $\eta p^2 = 0.087$ or Group, $F (2, 27) = 0.273$, $\eta p^2 = 0.020$. There was also no significant Time*Group interaction, $F (4, 54) = 0.233$, $p = 0.918$, $\eta p^2 = 0.017$.

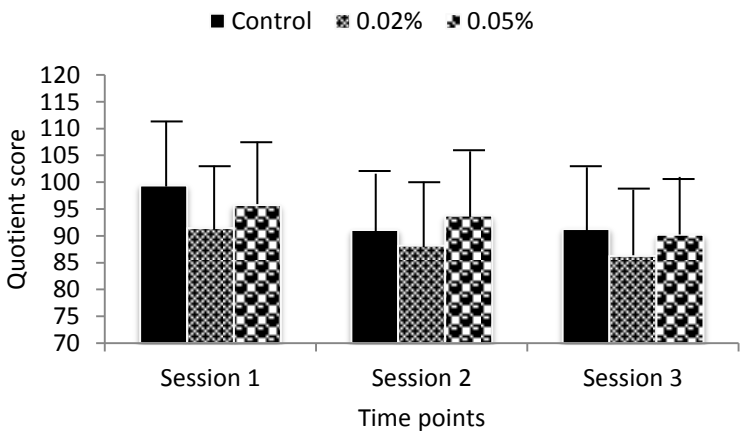


Figure 15. The average global full-scale response control quotients for each group (visual & auditory combined) across time points 1 - 3. Error bars indicate 95% confidence intervals.

To examine the effects of alcohol on attention, group average full scale global quotients were calculated, which provide an overall summary of how well participants' performed on the auditory and visual attention composite scales. Shown in Figure 16 are the average full scale attention quotients for each group. Participants in the 0.05% group obtained slightly higher scores on the third test administration compared to the other groups which one would not have generally expected. However, while some small variations were evident, all the groups scores remained within the average standard score range, suggesting there was no significant impairment. The analysis with a 3 (Group) x 3 (Time) mixed ANOVA revealed there was no significant main effect of Time, $F (1.284, 34.663) = 0.577$, $p = 0.0.494$, $\eta p^2 = 0.021$ or significant Time*Group interaction, $F (2.568, 34.663) = 1.239$, $p = 0.308$, $\eta p^2 = 0.084$. There was also no significant main effect of Group, $F (2, 27) = 0.013$, $p = 0.835$, $\eta p^2 = 0.013$.

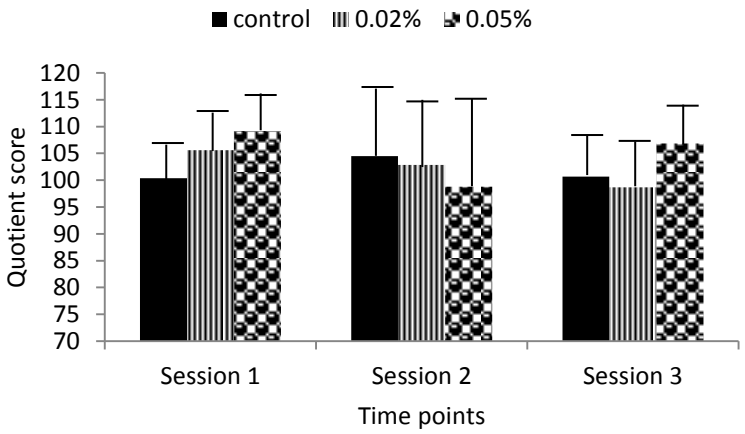


Figure 16. The global full-scale attention quotients for each group (visual and auditory combined) across time points 1 - 3. Error bars indicate 95% confidence intervals.

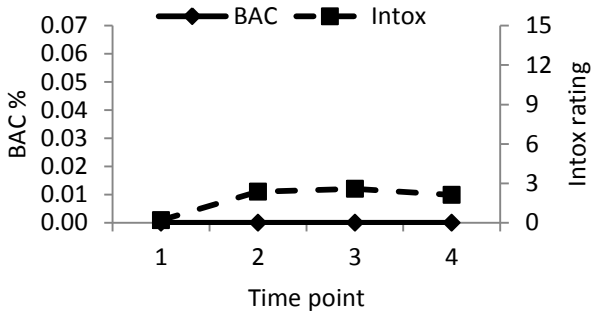
In summary, for the IVACPT there was no effect of alcohol, Group or Time on attention or response control measures. However, there was a large amount of variation in the participants' scores. For example the 0.05% group had the highest baseline quotient, then the lowest score on the second session and the highest on the third measure which was not expected.

BAC and subjective intoxication ratings

The average BAC and subjective rating of intoxication was calculated to examine whether there was a development of acute tolerance (a mismatch as BACs increased and declined back to zero)**.** Figure 17 shows the results for each group's average BAC and subjective intoxication ratings. It can be seen that participants in the control group felt slightly intoxicated, indicating that they were blind to the experimental condition they were in and there was a possible placebo effect. In comparison, participants in the 0.05% group felt less intoxicated in relation to their BAC than those in the 0.02% group, but an acute tolerance effect was not evident.

To examine whether there was a significant difference across each group's intoxication rating, a 3 (Group) x 4 (Time) mixed ANOVA was used. There was no main effect of Group, $F (2, 27) = 0.059$, $p = 0.943$, $\eta p^2 = 0.004$ but there was a significant effect of Time, $F (2.274, 61.401) = 32.996$, $p = 0.001$, $\eta p^2 = 0.074$ showing the ratings of subjective intoxication varied significantly across each test administration. Pairwise comparisons revealed there were significant differences between all time-points ($p = < 0.003$ apart from 2 & 3 $p = 1.00$). There was no significant Time*Group interaction, $F (4.548, 61.401) = 1.079$, $p = 0.378$, $\eta p^2 = 0.074$ evident. As a mixed ANOVA only compared each group's average, Pearson correlations were also conducted to see if there was a significant relationship between actual BAC and intoxication level at each point in time. There was no significant correlation between BAC and rating of subjective intoxication across time points 2-4, (BAC/Intox2) $r = - 0.047$, $p = 0.805$, (BAC/Intox3) $r = - 0.023$, $p = 0.906$ and (BAC/Intox4) $r = 0.167$, $p = 0.379$.

a.



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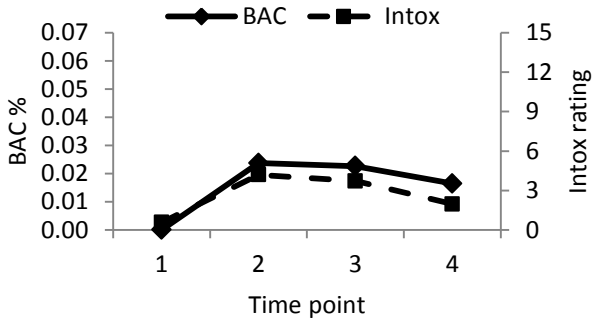

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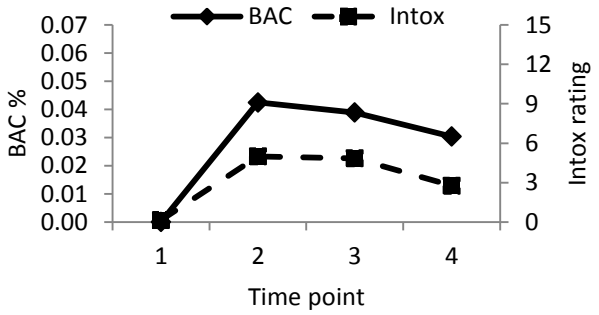


Figure 17. Average BACs and subjective intoxication ratings with (a) showing the control group, (b) the 0.02% group and (c) the 0.05% group.

Overall, there was no statistical relationship between alcohol dose and subjective ratings of intoxication, or evidence of acute tolerance, but a main effect of Time was observed. There was however a large amount of variation BAC and subjective rating of intoxication which could have obscured any differences.

DISCUSSION

This study set out to measure the effects of low dose alcohol on simulated driving and cognitive performance across multiple BACs (0.00, 0.02, 0.05 and 0.08%). It was expected that the effect of alcohol would be task and dose dependent and that there would be a mismatch in the development of acute tolerance. Interestingly, the outcomes of this study did not entirely go in the direction anticipated, so the findings from previous research were only partially supported, exemplifying that the effect of low dose alcohol can vary widely.

Psychomotor function

On the hazard car task there were no significant impairments in accelerator and brake reaction time noted on Roads 2 and 4 when the hazard car pulled out. Studies have tended to show that impairments in simple psychomotor functions like reaction time, often only become evident with higher doses of alcohol $\geq$ 0.05%, which could explain why no effects were found. In the current study, only a few participants' achieved a BAC of 0.05%, most were lower than this level. It was also interesting that accelerator and brake reaction time actually improved, rather than declined on Road 4 which does not correspond with other studies findings. The reason reaction time became faster is likely because participants started to anticipate or drive more cautiously after encountering the hazard car the first time round. For example, a participant in the 0.05% group said he was prepared for the hazard car when it pulled out the second time and because he felt intoxicated it made him try to focus harder to compensate. Another participant mentioned she felt as though she was driving to the limit and didn't feel safe, she said her reaction time felt slower and that she was more prone to make mistakes, so she drove slower. The changes in driving behaviour were evident by the

negative ART values obtained which showed participants in the 0.02% and 0.05% group took their foot off the accelerator in advance. The fact that participants' may have compensated their driving, reflects there may have been some kind of learning effect which prevented any small effects of alcohol impairment being detected.

Although no impairments in brake or accelerator reaction time were found, there was a significant difference in the number of hazard car crashes revealed on Roads 2 and 4 when participants BACs were highest. On Road 2, the 0.05% group had a higher number of crashes in comparison to the other two groups. However, on Road 4, there were fewer crash incidents with the 0.05% and 0.02% groups' each having the same number. There are several possible reasons as to why participants in the 0.05% group crashed more frequently on Road 2. Firstly, Sewell et al (2009) found low doses of alcohol significantly impaired hand eye coordination and hazard perception, therefore it is possible participants in the 0.05% group were less able to perceive or react in a coordinated manner. For example, during the experiment some participants in the 0.05% group appeared to brake more abruptly and they skidded into the hazard car without changing course. In comparison, participants in the control group seemed to brake less intensely and were able to drive around the hazard car. The differences observed in braking behaviour and the way participants' handled the car also suggests more complex, rather than simple psychomotor skills may have been impaired. As discussed in the literature, Ligouri (1999) found that complex motor skills and equilibrium were impaired but simple psychomotor skills were not affected. Other studies also revealed complex executive functions were often impaired and participants' strategies to cope or compensate may not be sufficient to offset the increased crash risk (Sewell, et al, 2009). One could argue that if this was the

case, why did participants in the 0.05% group crash less on Road 4, it is likely because they had encountered the hazard car before or their BACs were lower. In a normal context, drivers would not normally encounter the same hazard twice which would prevent them from being able to anticipate the event in advance.

Speed change / maintenance

There are numerous driving and cognitive studies which have shown low dose alcohol can significantly impair executive frontal functions on tasks. However, in this study the results from the speed change manipulation revealed a significant effect of Road but it did not detect any alcohol impairment. The reason why all of the groups increased or decreased their speed more on some Roads and not others is likely because the speed transitions started and ended in different places across Roads 1 - 4. Differences in the placement of the signs meant that some of the speed changes occurred at the bottom of a hill or around a corner which could have influenced the results.

In terms of alcohol, impairment may not have been detected because of several factors. To start with, some participants may have possibly employed some kind of behavioural strategy to resist the effects of alcohol. For example, one participant commented "because it was a test, I felt I drove to the signage more than I would normally." Another participant said that in the simulator she was more conscious of monitoring her speed than in normal everyday driving, so was more focused on that aspect. As participants were able to make a more conscious effort, this indicates there may have been some testing and carry over effects which confounded the results. Participants' likely interpreted the meaning or intention of the study and changed their driving behaviour accordingly which spilled over to affect subsequent repeated measures. It is also possible the speed

change task itself was not sensitive enough to detect any effect of alcohol impairment. For example, the speed signs could often be seen some way ahead before passing them which could mean the task was not difficult or sufficiently challenging. Yung-Ching and Shing-Mei (2007) used a sign detection task in their study to examine the effects of low dose alcohol. However, the signs were less conspicuous and the driving scenario in which the task was performed was more complex. This draws attention to the research findings which have shown that the signs of alcohol induced impairment are often task dependent and that detection may require additional behaviours to be noted.

The effect of task demand was reflected in the results of speed maintenance task in the current study. Generally, participants in each group drove faster in the 70km/h, 80km/h and 100km/h light traffic (LT) zones and slower in the heavy traffic (HT) zones which correspond with the findings reported in literature. Distraction alone has consistently been shown to impair driving performance because it increases the demand of cognitive processing (Rakauskas, et al, 2008). The discovery that alcohol when combined with distraction exacerbates impairment was also partially confirmed in this study. For example, it was interesting to find there was a marked difference in the average maximum speed of the 0.05% group in the low traffic (LT) 70km/h zone compared to the other two groups on Roads 3 - 4. Those in the 0.05% group drove slower on Road 3 and faster on Road 4, revealing there was a significant Road*Density*Group interaction. There are several possible reasons why participants' drove slower on Road 3 and faster on Road 4, in comparison to the other groups. One possible explanation is that participants in the 0.05% group may have found it difficult to regulate their speed because some aspect of response control might have been impaired by alcohol. Some participants' mentioned that 70kms/h felt slow in the

simulator, so those with a higher BAC may have found it more difficult to maintain the correct speed because it was boring. The conflict between perhaps wanting to go faster, yet knowing they needed to comply with the speed limit could have generated some level of response conflict.

Another reason could be that alcohol made participants feel more confident to take risks. For example, one participant said "I drove more carefully in the first drive, was conscious of my speed but after drinking orange and vodka I felt less conscious, more fluid though I doubt more prepared should the circumstance have changed from a normal road..." Generally, the participant felt less concerned about the environment but at the same time less prepared if any changes were to occur. Studies have shown performance often decreases in conditions of high resource conflict and alcohol interacts to promote risky behaviour (Fillmore, et al 2007). Sewell, et al (2009) found the ability to perceive negative consequences associated with risk taking was impaired. Individuals who consumed alcohol drove faster and made more errors and attempted to overtake other vehicles more often. Participants with a higher BAC may have also not regulated their speed due to a mismatch in the perception of alcohol intoxication and the recovery of cognitive performance.

Biphasic effects

The fact the difference in the 0.05% group's speed in the heavy traffic (HT) 70km/h zone occurred when BACs should have been declining suggests cognitive performance may have been affected differently across the descending BAC curve. While it is possible that there could have been a mismatch in the development of acute tolerance, it was difficult to compare results across the repeated measures because of problems encountered with the oral dosing method.

The Widmark formula underestimated BAC levels more than expected and the oral dosages of alcohol produced considerable variability in peak BACs and the rate in which BACs declined. In most cases, the target BAC of 0.05% was not achieved using the dosing method, which effectively narrowed the range of BACs in which the effect of alcohol could be compared. As there was a wide variation in BACs it was unclear whether there were any differences in cognitive functions like, accuracy and speed of information processing. Results from the IVACPT showed there was no difference in response control or measures of attention to suggest alcohol produced a mismatch in cognitive recovery. Factors like speed and accuracy which were factors the IVACPT measured would not have likely shown any impairment, as research has failed to detect alcohol impairment with similar measures (Schulte, et al, 2001). The IVACPT only required the participants to complete a single task at one time and the test itself was probably too simple. The difference in each group's average intoxication rating also did not reach statistical significance in this study. Although the results did not replicate the acute tolerance effects in subjective measures of intoxication, it was evident that subjective perceptions of intoxication were an unreliable means of estimating ones actual BAC level. For example, some participants' in the control group felt intoxicated even though they received no alcohol, while some of those given alcohol felt less intoxicated. Overall, the findings confirm oral dosing produces wide variability due to differences in metabolic metabolisms and bioavailability of alcohol which corroborates with results in literature.

Summary

The link between alcohol consumption and road user fatalities has been consistently reported, demonstrating the impact alcohol can have on driving.

While efforts have been directed toward lowering BAC limits, much debate continues about what constitutes a safe or rational BAC level. Research has shown, alcohol produces a wide array of effects across the blood alcohol curve and that different aspects of driving and cognitive performance are task dependent. Given this, alcohol may not affect performance on simple tasks but when task complexity is increased, impairments even with low dose alcohol often become evident. Despite some discrepancies, this study also generally showed the effect of low dose alcohol is task dependent and that impairment can vary considerably across different measures. Differences in the way individuals respond to alcohol and the variation in BACs achieved, means the effect of alcohol varied widely. Results showed there were no significant impairments in reaction time but the consumption of low dose alcohol lead to an increased number of crashes. While strategies to cope or compensate may mitigate the effects of alcohol to some extent, this is not sufficient to offset the crash risk associated with alcohol. Overall, it was apparent that people cannot reliably judge how intoxicated they are in relation to their actual BAC level, which is a relevant safety issue.

Limitations

The dosing method used in the current study consistently provided lower than expected BACs therefore another procedure would be needed in any future experiment. As alcohol had such a varied effect across individuals, a within subjects design may have been better in order to reduce subject variability. The study had a small sample size which meant if the effect of alcohol was small there may not have been enough statistical power to detect it. As the study was based on a repeated measures design, testing and carry over effects likely reduced the sensitivity of the measures used. The results of the study were generated in a

laboratory setting using a driving simulator, which itself limits the external validity of the findings. However, while there were some limitations inherent in the design of the experiment, the use of a driving simulator enabled the effect of alcohol to be examined in a more controlled environment in which tasks could be manipulated.

Directions for future research

Future research could examine the effects of low dose alcohol using a larger sample size and a more challenging driving scenario. To explore in more depth the effects of alcohol on attention and driving tasks would need to be more unexpected and demanding than those used in the current study. As participants did not reach the desired target BAC levels, research using a different dosing method could be utilised to explore the effect of alcohol as BACs rise and decline. Future studies could also examine if differences in the rate in which peak BACS are achieved (rapid versus gradual dosing methods), could itself influence the effects of alcohol on driving and cognitive performance. Greater consideration into how driver skill or other personal factors can mediate alcohol's effect is another area that could be investigated further.

REFERENCES

Albalate, D. (2008). Lowering blood alcohol content levels to save lives: The European experience. *Journal of Policy Analysis and Management 27*(1), 20 - 39.

AK Solutions. (2012). *AlcoMate AccuCell*. Retrieved from http://alcomate.net/alcomate-accucell.html

Australian Transport Council. (2010). *National Road Safety Action Plan 2009 and 2010.* Retrieved 3 June 2011 from http://www.atcouncil.gov.au/documents/files/ATC_actionplan0910.pdf

BrainTrain. (2012). IVA+Plus: *Integrated Visual and Auditory Continuous Performance Test (CPT).* Retrieved from http://www.braintrain.com/ivaplus/

Chamberlian, E., & Solomon, R. (2002). The case for a 0.05% criminal law blood alcohol concentration limit for driving. *Injury Prevention, 8* (3), iii1- iii17.

Charlton, S.G., & Starkey, N. J. (2011). Driving without awareness: The effects of practice and automaticity on attention and driving. *Transportation Research Part F: Traffic Psychology and Behaviour, 14* (6), 456 - 471.

Chowdhury, K., & Kilbey, P. (2011). *Reported road casualties Great Britain 2009*. Retrieved from http://www.dft.gov.uk/adobepdf/162469/221412/221549/227755/rrcgb2009.pdf

Cromer, J. R., Cromer. J. A., Maruff, P., & Snyder, P. J. (2010). Perception of alcohol intoxication shows acute tolerance while executive functions remain impaired. *Experimental and Clinical Psychopharmacology, 18* (4), 329 – 359.

Deery, H. A., & Love, A. W. (1996). The effect of a moderate dose of alcohol on the traffic hazard perception profile of young drink-drivers. *Addiction, 91*(6), 815 – 827.

Domingues, S. C. A., Mendonca, J. B., Laranjeira, R., & Nakamura-Palacios, E. M. (2009). Drinking and driving: a decrease in executive frontal functions in young drivers with high blood alcohol concentration. *Alcohol, 43,* 657 – 664.

Easdon, C. M., & Vogel-Sprott, M. (2000). Alcohol and behavioural control: Impaired response inhibition and flexibility in social drinkers. *Experimental and Clinical Psychopharmacology, 8* (3), 387 – 394.

Eckardt, M. J., File, S. E., Gessa, G. L., Grant, K. A., Guerri, C., Hoffman, P. L., Kalant, H., Koob, G. F., Li, T., & Tabakoff, B. (1998). Effects of moderate alcohol consumption on the central nervous system. *Alcoholism Clinical and Experimental Research, 22*(5), 998 - 1040.

Fillmore, M. T., Vogel-Sprott. M., & Gavrilescu, D. (1999). Alcohol effects on intentional behaviour: Dissociating controlled and automatic influences. *Experimental and Clinical Psychopharmacology, 7* (4), 372 – 378.

Fillmore, M. T., Blackburn, J. S., & Harrison, E. L. R. (2007). Acute disinhibiting effects of alcohol as a factor in risky driving behaviour. *Drug and Alcohol Dependence, 95,* 97 – 106.

Fillmore, M. T., E. W., Ostling & Martin, C. A., & Kelly, T. H. (2009). Acute effects of alcohol on inhibitory control and information processing in high and low sensation-seekers. *Drug and Alcohol Dependence, 100,* 91 -99.

Grant, S., Millar, K., & Kenny, G. N. C. (2000). Blood alcohol concentration and psychomotor effects. *British Journal of Anaesthesia, 85*(3), 401-406.

Gundersen, H., Gruner, R., Specht, K, & Hugdahl, K. (2008). The effects of alcohol intoxication on neuronal activation at different levels of cognitive load. *The Open Neuroimaging Journal, 2*, 65 – 72.

Harrison, L. R., Marczinski, C.A., & Fillmore, M. T. (2007). Driver training conditions affect sensitivity to the impairing effects of alcohol on a simulated driving test to the impairing effects of alcohol on a simulated driving test. *Experimental and Clinical Psychology, 15* (6), 588 – 598.

Kuypers, K. P.C, Samyn, N., & RMckers, J. G. (2006). MDMA and alcohol effects, combined and alone on objective and subjective measures of actual driving performance and psychomotor function. *Psychopharmacology, 187*, 467 - 475.

Langley, J., & Marshall, S. W. (1994). The severity of road traffic crashes resulting in hospitalisation in New Zealand. *Accident Analysis and Prevention 26* (4), 549 - 554.

Leung, S., & Starmer, G. (2005). Gap acceptance and risk taking by young and mature drivers, both sober and alcohol intoxicated, in a simulated driving task. *Accident Analysis & Prevention, 37,* 1056 – 1065.

Ligouri, A., D'Agostino Jr, R. B., Dworkin, S. I., Edwards, D., & Robinson, J. H. (1999). Alcohol effects on mood, equilibrium and simulated driving. *Alcoholism: Clinical and Experimental Research, 33* (5), 815 – 821.

Liu, Y-C., & Ho, C. H. (2010). Effects of different blood alcohol concentrations and post alcohol impairment on driving behaviour and task performance. *Traffic Injury Prevention, 11*, 334- 341.

National Highway Traffic Safety Administration. (2000). *Driver characteristics and impairment at various BACs*. Retrieved 5 June 2011 from http://www.nhtsa.gov/people/injury/research/pub/impaired_driving/BAC/index.html

National Highway Traffic Administration. (2009). *Traffic safety facts for 2009.* Retrieved 4 June 2011 from http://www-nrd.nhtsa.dot.gov /Pubs/811 385.PDF

Marczinski, C. A., Harrison, E. L. R., & Fillmore, M. T. (2008). Effects of alcohol on simulated driving and perceived driving impairment in binge drinkers. *Alcoholism: Clinical and Experimental Research, 32* (7), 1329 - 1337.

Meda, S. A., Calhoun, V. D., Astur, R. S., Turner, B. M., Ruopp, K., & Pearlson, G. D. (2009). Alcohol dose effects on brain circuits during simulated driving: An - fMRI Study. *Human Brain Mapping, 30,* 1257 – 1270.

Miller, M. A., Weafer, J., Fillmore, M. T. (2009). Gender influences in alcohol impairment of simulated driving performance and driving related skills. *Alcohol & Alcoholism, 44* (6), 586 – 593.

Ministry of Transport. (2012). *Alcohol and drugs crash facts*. Retrieved 4 June 2011 from http://www.transport.govt.nz/research/Pages/MotorVehicle CrashesinNew Zealand2010.aspx

Moskowitz H, Fiorentino D. (2000). *A review of the literature on the effects of low doses of alcohol on driving related skills*. Retrieved from http://www.nhtsa.gov/people/injury/research/pub/hs809028/title.htm

Ogden, E.J.D., & Moskowitz H. (2010). Effects of alcohol and other drugs on driver performance. *Traffic injury prevention, 5,* 185 – 198.

Parsons, O. A., & Nixon, S. J. (1998). Cognitive functioning in sober social drinkers: a review of the research since 1986. *Journal of Studies on Alcohol, 59*(2), 180 – 190.

Rakauskas, M. E., Ward, N. J., Boer, E. R., Bernat, E. M., Cadwallader, M., & Patrick, C. J. (2008). Combined effects of alcohol and distraction on driving performance. *Accident Analysis and Prevention, 40,* 1742 – 1749.

Rothengatter, T. (2002). Drivers' illusions – no more risk. *Accident Analysis and Prevention, 41,* 1053-1063.

Rzepecki-Smith, C. I., Meda, S. A., Calhoun, V.D., Stevens, M. C., Jafri, M. J., Astur, R. S., & Pearlson, G. D. (2010). Disruptions in functional network connectivity during alcohol intoxicated driving. *Alcoholism: Clinical and Experimental Research, 34*(3), 479 – 487.

Sayette, M. A., Reichie., & Schooler, J. W. (2009). Lost in the sauce the effects of alcohol on mind wandering. *Psychological Science, 20* (6), 747 – 752.

Schulte. T., Muller-Ohring. E. M., Warzel, H., & Sabel, B. A. (2001). Acute effects of alcohol on divided attention and covert attention in men. *Pharmacology, 154*, 61 -69.

Schweizer, T. A., & Vogel-Sprott, M. (2008). Alcohol-impaired speed and accuracy of cognitive functions: A review of acute tolerance and recovery of cognitive performance. *Experimental and Clinical Psychopharmacology, 16* (3), 240 – 250.

Sewell, R. A., Poling, J., & Sofuoglu. M. (2009). The effect of cannabis compared with alcohol on driving. *American Journal on Addictions, 18* (3), 185 – 193.

South Australia Police (2011). *Road statistics*. Retrieved 3 June, 2011 from http://www.sapolice.sa.gov.au/sapol/road_safety/road_statistics.jsp

Strauss. E., Sherman. E. M. S., & Spreen. O. (2006). *A compendium of neuropsychological tests: Administration, norms and commentary* (3rd ed.). Oxford University Press: New York.

Transport Canada Road Safety and Motor Vehicle Regulation. (2011). *Canadian motor vehicle traffic collision statistics: 2009*. Retrieved 2 June 2011 from http://www.tc.gc.ca/eng/roadsafety/tp-tp3322-2009-1173.htm

Traynor, T. L (2005). The impact of driver alcohol use on crash severity: a crash specific analysis. *Transportation Research Part E, 41*, 421 - 437.

Van Skike, C. E., Botta, P., Chin, V., S., Tokunaga, S., McDaniel, J. M., Venard, J., Diaz-Granados, J. L., Valenzuela, C. F., & Matthews, D. B. (2010). Behavioural effects of ethanol in cerebellum are age dependent: Potential system and molecular mechanisms. *Alcoholism: Clinical and Experimental Research, 34*(12), 2070 – 2080.

Vengeliene, V., Bilbao, A., Molander, A., & Spanagel, R. (2009). Neuropharmacology of alcohol addiction. *British Journal of Pharmacology, 154*(2), 299 – 315.

Verster, J. C., Wester, A. E., Goorden, M., van Wieringen, J.P., Olivier, B., & Volkerts, E. R. (2009). Novice drivers' performance after different alcohol dosages and placebo in the divided-attention steering simulator. *Psychopharmacology, 204*, 127-133.

Wallner, M., Hancher, H. J., & Olsen, R. W. (2006). Low dose acute effects on GABAa receptor subtypes. *Pharmacology and Therapeutics, 112* (2), 513 – 528.

Wallner, M., & Olsen, R. W. (2008). Physiology and pharmacology of alcohol: the imidazobenodiazepine alcohol antagonist site on subtypes of GABAa receptors as an opportunity for drug development*? British Journal of Pharmacology, 154* (2), 288 – 298.

Weissman, D. H., Gopalakrishnan, A., Hazlett, C. J., & Woldorff, M.G. (2005). Dorsal anterior cingulate cortex resolves conflict from distracting stimuli by boosting attention toward relevant events. *Cerebral Cortex, 15*(2), 229 – 237.

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

Wester, A. E., Verster, J. C., Volkerts, E. R., Böcker, K. B. E., Kenemans, J. L. (2010). Effects of alcohol on attention orienting and dual-task performance during simulated driving: an event-related potential study. *Journal of Psychopharmacology, 24*(9), 1333 - 1348

Yung-Ching, L., & Shing-Mei, F. (2007). Changes in driving behaviour and cognitive performance with different breath alcohol concentration levels. *Traffic Injury Prevention, 8,* 153 – 161.

Zoethout, R. W, Schoemaker, R. C., Zuurman, L., van Pelt, H., Dahan, A., Cohen, A. F., & van Gerven, J. M. (2009). Central nervous system effects of alcohol at a pseudo-steady-state concentration using alcohol clamping in healthy volunteers. *British Journal of Clinical Pharmacology*, *68* (4), 524 - 34.

APPENDICES

Appendix A: Research information sheet

THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

Low Dose Alcohol, Simulated Driving & Cognitive Performance Research

What is this study about?

- The aim of this study is to examine the effects of low dose alcohol on simulated driving and cognitive performance. We will be looking to see if the effect of alcohol is task dependent and whether any changes occur in a dose dependent manner. Furthermore, efforts will be focused on assessing if the impact of alcohol varies when blood alcohol levels peak and then decline.

Am I eligible to participate?

- You are aged between 20 – 65 years.
- Hold a full drivers licence with at least 3 years driving experience.
- Have no history of a medical, psychological, substance or alcohol abuse disorder that might affect ones performance.
- Be a non-smoker.
- Have a history of consuming at least one alcoholic beverage per week.
- You must be able to provide a designated driver to bring and take you home.

What am I being asked to do?

- To abstain from drinking alcohol 24hrs prior to the experiment.
- To abstain from ingesting food or other beverages 2 hrs before the experiment.
- Answer a health screening questionnaire and provide details about your weight.
- Have your Breath alcohol (BrAC) level tested before & during the study
- Drink a beverage containing a pre-calculated dose of alcohol within a BAC range of 0.00 – 0.08%.
- Complete a practice drive followed by 4 test sessions involving a simulated drive, computer administered test and measure of intoxication which will take approximately 3 hrs.

Participants

- All participants receive a $10 MTA voucher and students in PSYC102 or PSYC103 will obtain 3% course credit.

Who can I contact to participate in this study or ask any questions?

- Please email Paula Beard (pjbeard@orcon.net.nz) or Dr Nicola Starkey nstarkey@waikato.ac.nz) (K1.10) or Assoc Prof Sam Charlton (samiam@waikato.ac.nz) (K1.09) (supervisors).

Appendix B: Participant instructions

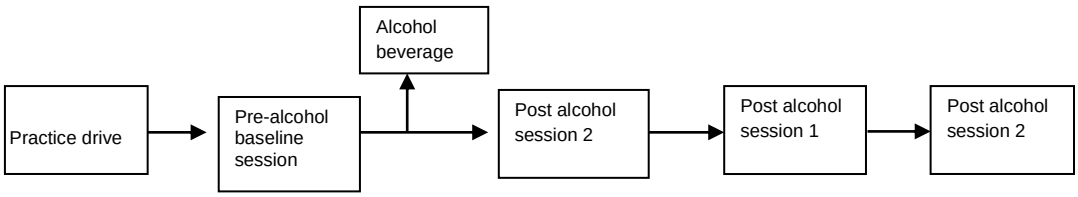
Participant Instructions

The purpose of this study is to examine the effect of low dose alcohol on simulated driving and cognitive performance.

For this study we are asking participants to:

- Abstain from drinking alcohol 24hrs prior to the experiment.
- Abstain from ingesting food or other beverages 2 hrs before the experiment.
- Answer a health screening questionnaire and provide details about your weight.
- Have your Breath alcohol (BrAC) level tested before & during the study.
- Drink a beverage containing a pre-calculated dose of alcohol within a BAC range of 0.00 – 0.08%.
- Come to the lab once and complete a practice drive followed by 4 test sessions involving a simulate drive, computer administered test and measure of intoxication which will take approximately 3 hours.
-

The study will be divided in to five phases as outlined in the diagram below:



- Before testing begins, you will need to complete an informed consent form, questionnaire and have your BrAC level tested. After this, we would like you to take a short practice drive to become familiar with the driving simulator and to see how you feel. The first test session will then be done and after this you will be given a beverage to drink within 10 minutes. Following this, you will be asked to do the remaining test sessions. Once the experiment has been completed we would like you to remain until your BrAC level approaches zero and then have your designated driver take you home.

- If you begin to feel unwell at any stage while in the driving simulator, please tell us and if you have any questions feel free to ask. You will be able to withdraw at

any stage during the experiment. All information you provide will remain strictly confidential.

Thank you for your participation

Paula Beard (researcher), Dr Nicola Starkey & Assoc Prof Sam Charlton (supervisors).

Appendix C: Informed consent form

University of Waikato
School of Psychology
CONSENT FORM

PARTICIPANT'S COPY

Research Project: The effect of low dose alcohol on simulated driving and cognitive performance

Name of Researcher: Paula Beard Supervisors: Dr. S. G. Charlton & Dr. N. J. Starkey

I have received an information sheet about this research project or the researcher has explained the study to me. I have had the chance to ask any questions and discuss my participation with other people. Any questions have been answered to my satisfaction.

I agree to participate in this research project and I understand that I may withdraw at any time. If I have any concerns about this project, I may contact the convenor of the Research and Ethics Committee (Dr Robert Isler, phone: 838 4466 ext. 8401, e-mail r.isler@waikato.ac.nz)

Participant's
Name:_____Signature:_____Date:_____

==========

University of Waikato
School of Psychology
CONSENT FORM

RESEARCHER'S COPY

Research Project: The effect of low dose alcohol on simulated driving and cognitive performance

Name of Researcher: Paula Beard Supervisors: Dr. S. G. Charlton & Dr. N. J. Starkey

I have received an information sheet about this research project or the researcher has explained the study to me. I have had the chance to ask any questions and discuss my participation with other people. Any questions have been answered to my satisfaction.

I agree to participate in this research project and I understand that I may withdraw at any time. If I have any concerns about this project, I may contact the convenor of the Research and Ethics Committee.

Participant's

Name: _____Signature:_____ Date:_____

Appendix D: Eligibility Questionnaire

THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

Eligibility Questionnaire

(all information provided will be kept in strict confidence)

1. What is your date of birth? ___

2. What type of driving licence do you currently hold? (tick one)

☐ Learner's licence
☐ Restricted licence
☐ Full (unrestricted licence)

3. How long have you held a Full licence? (since you attained your unrestricted)_____ (years)

4. Have you had prior experience in a driving simulator? Yes No (circle one)

5. Do you drink more than one alcoholic beverage a week? Yes No (circle one)

6. Do you take or have you recently taken (within the last 48 hrs) any of the following medications /drugs?

☐Yes ☐No Antidepressants
☐Yes ☐No Antihistamines
☐Yes ☐No Beta blockers
☐Yes ☐No Benzodiazepines
☐Yes ☐No Diuretics
☐Yes ☐No Thyroid medication
☐Yes ☐No Amphetamines
☐Yes ☐No Opiates
☐Yes ☐No Barbiturates
☐Yes ☐No Cocaine
☐Yes ☐No Marijuana
☐Yes ☐No Herbal, synthetic psychoactive substances eg party pills

7. Do you smoke tobacco? YES NO (circle one)

8. Do you have any of the following medical problems? (tick yes or no)

☐ Yes ☐ No Current physical disease / illness
(if yes, please specify)_____

☐Yes ☐No Psychiatric disorder
☐Yes ☐No Substance use disorder
☐Yes ☐No Head trauma or other CNS injury
☐Yes ☐No Neurological disorder
☐Yes ☐No Stroke
☐Yes ☐No Thyroid disease
☐Yes ☐No Diabetes
☐Yes ☐No Cardiovascular disorder
☐Yes ☐No Chronic obstructive pulmonary disease
☐ Other which may affect your performance (please specify)
_____

9. If female, are you pregnant or breast-feeding? YES NO (circle one)

If you have any questions please feel free to ask, thank you for your answers.

Appendix E: Demographic & Driving Questionnaire

THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

Demographic & Driving Questionnaire

(all information provided will be kept in strict confidence)

1. What is your gender? M F
(circle one)

2. Which ethnic group do you belong to? (tick the box or boxes that apply to you)

- ❑ New Zealand European
- ❑ Maori
- ❑ Samoan
- ❑ Cook Island Maori
- ❑ Tongan
- ❑ Niuean
- ❑ Chinese
- ❑ Indian
- ❑ Other (eg Dutch, Japanese, Tokelauan)

Please specify_____

3. Have you received any driving infringements (including speed camera fines)? Yes No
(circle one)

If yes, how many? _____

4. In the past year, have you been involved in any motor vehicle crashes? YES NO
(circle one)

If yes, how many? _____

5. Have you had any drink driving convictions? YES NO
(circle one)

If yes, how many? _____

6. How many standard drinks do you consume per week? (standard drink = 1 bottle of beer, 1 small glass of wine, 1 shot of spirits)_____

7. What is your height (cm) _____

and weight (kgs)_____

(Information regarding height & weight is requested for the purpose of calculating alcohol dosage).

If you have any questions please feel free to ask, thank you for your answers.

Appendix F: Subjective intoxication rating

Subjective Intoxication Rating (1)

Please place a mark on the horizontal line below to indicate how intoxicated you feel right now.

Least intoxicated
ever felt in life

Most intoxicated
ever felt in life