# An investigation into the prolonged effects of excessive acute alcohol consumption on motor vehicle driving skills

By

**Dustin Silvey** 

A thesis submitted to The School of Graduate Studies In partial fulfillment of the Requirements for the degree of Master of Science (Kinesiology)

School of Human Kinetics and Recreation Memorial University

May 2014

St. John's

Newfoundland

# ABSTRACT

The purpose of the current thesis was twofold: 1) to review the literature while linking the prolonged effects of excessive acute alcohol consumption (hangover) to decrements in complex vigilance tasks and 2) to determine if the prolonged effects of excessive acute alcohol consumption have an affect on driving a motor vehicle. Driving involves great requirements for attention, vigilance, and motor control. It has been previously demonstrated that alcohol, while in the body, or recently removed does cause decrements in attention span, judgment, psychomotor vigilance, and memory. However, there has been little consistency between the testing; for example, some researchers did not wait until participants' blood alcohol concentration (BAC) was 0.00 g% before conducting testing and therefore, the remaining alcohol in the participants' bodies may have affected the results. In the present study it was indicated that hangovers do not have an effect on driving a motor vehicle, but with a BAC above the legal limit (0.08 g%) there is a significant decrement in driving ability. In conclusion, hangovers may not affect driving a motor vehicle, but future research should examine if more severe hangovers do have a detrimental effect.

ii

# ACKNOWLEDGEMENTS

I would like to thank my supervisor Dr. David Behm for all his patience and understanding over the past 3 years. Without his constant vigilance I would still be nowhere near completing this thesis. Dr. Jeannette Byrne for always laughing at me and keeping me in a good frame of mind when things just would not work out how they were suppose to in a timely manner. Dr Saied Jalal Aboodarada for his constant support throughout the final months of writing. Dr. Michael Johnson for allowing me to conduct my data collection at the Université de Moncton. Marie-Belle Adrao for all her patience with aiding me in conducting much of the research required to complete this thesis and putting up with my constant phone calls and emails. Lastly, all the staff and faculty of the School of Human Kinetics and Recreation at Memorial University of Newfoundland for their considerable support and for making learning fun and interesting.

# Table of Contents

ABSTRACT	ii			
ACKNOWLEDGEMENTS	iii			
LIST OF TABLES	v			
LIST OF FIGURES	vi			
LIST OF ABBREVIATIONS	vii			
1. LITERATURE REVIEW Introduction				
Hypothesis	5			
General Physiological and Cognitive Effects of Alcohol Physiological Effects of Alcohol				
Endocrine/Psychological Effects of Alcohol	6			
Cognitive Effects of Alcohol	7			
Chronic Effects of Alcohol Cognitive Complex Vigilance Tasks				
Physiology of Complex Vigilance Tasks				
Hangover Effects				
Physiological Effects Cognitive Effects				
Conclusions				
References				
2. CO-AUTHORSHIP STATEMENT				
3. RESEARCH MANUSCRIPT				
ABSTRACT				
INTRODUCTION	-			
METHODOLGY				
Research design				
Participants				
Procedure				
Measures				
Statistical Analysis				
RESULTS				
DISCUSSION CONCLUSION				
REFERENCES				
KEFEKENLEJ				

# LIST OF TABLES

Table 1.	Absolute values for each simulation and <u>Useful Field of View (</u> UFOV) test at 4, 7, and 24 hours
Table 2.	Overall score normalized statistical values for each simulator trial at 4, 7, and 24 hours with percent differe <u>n</u> ces
Table 3.	Fuel Economy normalized statistical values for each simulator tr <u>ia</u> l at 4, 7, and 24 hours with percent differences
Table 4.	Control of Vehicle normalized statistical values for each simulator tr <u>ia</u> l at 4, 7, and 24 hours with percent differences
Table 5.	Road Safety normalized statistical values for each simulator tr <u>ia</u> l at 4, 7, and 24 hours with percent differences45
Table 6.	Legal Aspect normalized statistical values for each simulator tr <u>ia</u> l at 4, 7, and 24 hours with percent differences
Table 7.	Mobility normalized statistical values for each simulator tr <u>ia</u> l at 4, 7, and 24 hours with percent differences
Table 8.	Road Sharing normalized statistical values for each simulator tr <u>ia</u> l at 4, 7, and 24 hours with percent differences
Table 9.	<u>Useful Field of View (</u> UFOV) normalized statistical values for each trial at 4, 7, and 24 hours

# **LIST OF FIGURES**

Figure 1. Useful Field of View Test (UFOV)	37
Figure 2. Participant in the Virage Simulator	38
Figure 3. The view of participants while driving the Virage Simulator	39
Figure 4. Overall Score normalized percentile for each driving trial and group	43
Figure 5. Fuel Economy score normalized percentile for each driving trial and group	. 43

# LIST OF ABBREVIATIONS

- ALDH Aldehyde Dehydrogenase
- ANOVA Analysis of Variance
- **BAC Blood Alcohol Concentration**
- CBF Cerebral Blood Flow
- CI Confidence Intervals
- CNS Central Nervous System
- COCOM Contextual Control Model
- **CRP C-Reactive Protein**
- ES Effect Size
- FAA Federal Aviation Administration
- NHTSA U.S National Highway Traffic Safety Administration
- OFI Opuntia ficus indica
- SD Standard Deviation
- SPSS Statistical Package for the Social Sciences
- UFOV Useful Field of View

# **1. LITERATURE REVIEW**

# Effects of Alcohol on the Human Mind and Body During and After Consumption

Author: Dustin Silvey School of Human Kinetics and Recreation Memorial University St. John's, NL, A1C 5S7

<u>Running Title:</u> Effects of Hangovers on the Human Mind and Body

#### Introduction

Alcohol is one of the most commonly used drugs in the world (Model, 1990). Alcohol is used regularly in social settings, social traditions and many religious rituals (Mayer, 1998). As alcohol is legal in most countries in the world, including every country in the western hemisphere, it is extremely accessible and therefore is often used in excess (Lewis, 1996). The United States of America Department of Health indicates that alcohol abuse has a serious impact on society; in the USA over \$160 billion a year is spent on alcohol related crimes, accidents and treatments for alcohol abuse. Alcohol intoxication is connected in over 40% of fatal automobile accidents in the USA. These accidents have been related to the consumption of alcohol prior to or during the operation of a motor vehicle.

Alcohol has a depressant effect on the central nervous system (CNS) (Model, 1990). Studies have indicated that the depressant effects of alcohol on the CNS can occur at a blood/alcohol level of 0.025 g%. As alcohol levels increase to a level of 0.05 g% a high level of CNS function is lost (Model, 1990). If the CNS is impaired, operating a motor vehicle will be difficult. While driving a vehicle an individual is forced to be in a rapidly changing, three-dimensional environment. If an individual has consumed alcohol and thus impaired the function of the CNS their ability to drive will decrease; as they will not be able to analyze the constantly changing environment in which they are driving (Model, 1990).

Ingesting alcohol has been shown to cause many physiological and cognitive changes in an individual (Goldberg, 1966). A "standard drink" is defined as 44 ml (1.5oz) of distilled liquor (40% alcohol) 360 ml (12oz) of beer (5% alcohol) or 150

ml (5oz) of wine (12% alcohol). Each of theses drinks contains about 15 grams of alcohol (Miller, 1991). A 70 kg person who drinks one "standard drink" will have a blood alcohol of 0.02 g% to 0.04 g%. This number is dependent on the rate of ingestion and how rapidly the alcohol is absorbed (Model, 1990). This same average person eliminates alcohol from the body at a rate of 8 g per hour, therefore in order to have a blood/alcohol level of zero it could take up to 2 hours or even more depending on absorption rates. Thus, after one "standard drink" a person could be under the physiological and cognitive influences of alcohol for a minimum of 2 hours.

Current Federal Aviation Administration (FAA) regulations indicated that a pilot may operate a commercial airplane 8 hours after their last "standard drink" (Federal Aviation regulation 91.17). FAA suggests that 8 hours is enough time for impairments of alcohol on the CNS to cease - although the FAA does state that if possible wait 24 hours before piloting an aircraft after consuming alcohol (Federal Aviation Administration, 2012). The FAA suggests the 24 hours of recovery because of the prolonged effects of excessive acute alcohol consumption or hangover effects that occur once alcohol has been eliminated from the body.

A hangover is the prolonged ill effects caused by drinking an excessive amount of alcohol (Lemon, 1993). Hangovers can last for up to 72 hours after the last alcoholic beverage is consumed and many of the cognitive effects remain from the alcohol ingestion (Yesavage and Leirer, 1986). Because of the cognitive effects of hangovers, driving after alcohol has been eliminated from the body may still be dangerous. Yesavage and Leirer (1986) showed that hung-over military pilots

scored significantly lower in a piloting simulator indicating that hangovers still affect the CNS and reaction time. Piloting a plane is similar to driving, in that the three-dimensional environments are both changing at a rapid pace and cognitive function must be high in order to fully analyze and react to the situations. This suggests that driving a motor vehicle while impaired by a hangover could also be dangerous.

There have been numerous studies conducted on how alcohol affects complex vigilance tasks such as driving during the consumption of alcohol or while an individual has alcohol in their system (Koelega, 1995). There are also several studies on how hangovers affect psychomotor performance, piloting, and cognition (Goldberg, 1966; Lemon, 1993; Seppala, 1976; Yesavage and Leirer, 1986). There, however, is very little research that has addressed the prolonged effects of a hangover on physiological measures, such as simple and complex vigilance tests, that are directly related to driving.

The majority of government funding given towards alcohol related motor vehicle accidents examine how an intoxicated person's (blood alcohol > 0.05 g%) driving ability decreases. The U.S National Highway Traffic Safety Administration (NHTSA) has funded numerous studies in order to prevent individuals from driving under the influence of alcohol (Rothschild, 2006). The Canadian Institutes of Health Research also funded several studies related to driving while under the influence of numerous drugs; including alcohol, cannabis and cocaine (Adlaf et al., 2003; Macdonald et al., 2005; Macdonald et al., 2004; Walsh and Mann, 1998). While governments seem to place an exceptional emphasis on the direct effects of alcohol

on driving there is very little emphasis on the prolonged ill effects (hangover) caused after alcohol has been eliminated from the body.

#### Purpose

The purpose of this review is to examine the literature regarding acute and chronic effects of alcohol consumption on physiological and cognitive responses as they relate to simple and complex vigilance tasks. This review will specifically attempt to highlight areas of future research especially concerning the prolonged effects of excessive acute alcohol consumption and the effects it has on simple and complex vigilance tasks.

## **Objectives**

The objective of the research is to determine if the prolonged effects of excessive acute alcohol consumption effect complex vigilance tasks such as driving a motor vehicle.

## **Hypothesis**

It is hypothesized that a hangover will cause impairments in completing complex vigilant tasks such as driving a motor vehicle after 7 and 24 hours after the consumption of alcohol has ceased.

#### **General Physiological and Cognitive Effects of Alcohol**

A drug is considered to be any substance that causes a physiological effect on an individual when ingested or introduced to the body. Alcohol causes many acute and long-term physiological and cognitive effects (Heffernan, 2008). The acute

effects differ based on the amount of alcohol consumed (moderate, less than 5 standard drinks or high amounts, more than 5 standard drinks) while the long-term effects are consistent with chronic alcohol use in excess (Abrasom, 2010; Goldberg, 1966; Heffernan, 2008; Model, 1990; Ryback, 1971; Tolentino, 2011; Zirkle, 1959).

# **Physiological Effects of Alcohol**

The common acute effects observed when a moderate amount of alcohol is ingested are: lower control of balance, reduced vision, dehydration and increased cerebral blood flow (Tolentino, 2011). Moderate levels of alcohol can also impair fine motor control (Ryback, 1971). When an excessive amount of alcohol is ingested the acute physiological effects are more severe: vomiting, decreased heart rate, severe nausea, and even death can occur (Kupari, 2007).

#### **Endocrine/Psychological Effects of Alcohol**

An increase in cerebral blood flow to the brain during the consumption of alcohol causes the release of adrenocroticotropin and prolactin (Tolentino, 2011). Adrenocroticotropin, which is also released when cocaine is ingested, acts on the adrenal glands and can cause an increase in mood and social ability in drinkers (Mendelson, 1992). Prolactin counteracts the effects of dopamine causing an individual to feel "sexually gratified" and more positive (Turner, 2002). High levels of prolactin can lead to impotence when high amounts of alcohol are consumed (Besser, 1972). When alcohol stimulates the release of these two hormones in an individual, that person will feel more positive, more confident and become more social (Besser, 1972; Mendelson, 1992). Some individuals have a lower reaction to

alcohol and require more alcohol to stimulate an increase in cerebral blood flow. Tolentino et al. (2011) has indicated that individuals with a lower reaction to alcohol are at a higher risk of alcoholism and alcohol abuse.

#### **Cognitive Effects of Alcohol**

After the ingestion of moderate levels of alcohol, an individual could experience a shortened attention span, disruption of sleep patterns, impaired judgment, and memory loss (Model, 1990). The cause of memory loss or blackouts when alcohol is consumed is relatively unknown. It has been suggested that blackouts occur due to the toxic effect of alcohol on the brain; however, this has not been shown in research (Goodwin, 1970; Wetherill, 2011). When a high amount of alcohol is ingested the effects are much worse: unconsciousness, impaired speech and impaired reflexes (i.e. pupil dilation) (Zirkle, 1959).

# **Chronic Effects of Alcohol**

There are long-term cognitive effects of regular excessive alcohol consumption, one of which is a decline in prospective memory (i.e. forgetting to lock the car door when walking away) (Heffernan, 2008). Heffernan et al. (2002) and Ling et al. (2003) found that heavy alcohol users showed global impairments in prospective memory when compared to individuals that did not consume alcohol. Chronic excessive alcohol users have shown severe impairments when learning simple cognitive tests such as word lists, short- and long-term memory tests and general working memory (Ambrose, 2001). There are also findings that show

teenagers with a history of excessive alcohol abuse have difficulty with verbal and non - verbal memory tasks, language tasks, and attention tasks (Brown, 2000).

The long-term effects of alcohol consumption are observed much later in an individual's life and are often caused by chronic excessive alcohol use. One longterm physiological effect of alcohol is increased blood pressure (Abrasom, 2010). Another long-term physiological effect indicates that chronic alcohol consumption can break down bone tissue and lower bone density (Maurel et al., 2010). Vingren et al. (2005) also found that excessive alcohol consumption over time could decrease the amount of androgen receptors found in muscle tissue preventing muscle growth due to the inability to bind testosterone.

## **Cognitive Complex Vigilance Tasks**

Alcohol also impairs an individual's ability to complete complex vigilance tasks such as operating a motor vehicle (Koelega, 1995). This is due to more than just the cognitive effects of alcohol. Reduced vision, loss of balance and nausea would also make the completion of complex tasks difficult which as stated earlier are physiological effects.

People complete simple and complex vigilance tasks every day. A simple vigilance task involves one stimulus or event that causes an individual to determine an appropriate response, a complex vigilance task requires an individual to observe more than one event and then chose an appropriate response. A basic representation of what occurs during a simple vigilance task by Hollnagel and Woods (2005) is called the contextual control model (COCOM):

1) External event  $\rightarrow$ 2) Evaluating/assessing the situation  $\rightarrow$ 3) Intention  $\rightarrow$ 4) Choosing what to do  $\rightarrow$  5) Action  $\rightarrow$ 6) Carrying out the action  $\rightarrow$  7) Feedback  $\rightarrow$  8) Another external event and cycle starts again.

The COCOM describes the basic function of a complex task however it does not indicate the time between each step. The human body is a group of systems and processes that work together in order to function thus time is required to make any type of change or adaption (Naatanen, 1992). Between step 1 and step 2 time is needed to assess the situation, between 3 and 4 time is required to choose an appropriate reaction, step 5 is the available time to complete the action, and lastly the estimated performance time in what would be called the window of opportunity (Cook et al., 2007). This time has two major implications, first there is a limited amount of time for an individual to evaluate the situation, choose what to do and to act. The second implication is that the world is in constant flux and thus a reaction to one situation may not work for the next situation (Cohen, 1993). For example, if a person reacted one way to a situation in the past and continued to react the same way to a similar but not the same situation they may make the wrong choice. If a person constantly relies on an event to occur before reacting they may react too slowly to the situation. Individuals try to avoid this happening by "looking ahead", predicting what will happen (Cook et al., 2007). Being aware of what will happen before it does would allow individuals more time to complete their task, however, if the prediction is inaccurate then the chosen response will not be appropriate to the occurring event.

As has been discussed above, timing is important in completing complex vigilance tasks. There are many modes in which an individual can make an error and thus fail at the task. There are eight major failure modes that can occur during a complex task: 1) The timing of the action performed if performed too early or too late, 2) If the action performed is too long or too short, 3) Object which is being moved or controlled is moved too far or too short, 4) Action is performed too fast or too slow, 5) Action is performed in the wrong direction, 6) Action is performed with too much, or too little force, 7) The action is performed on the wrong object (object not involved in event), 8) Two or more of the actions are performed in the wrong order (Cook et al., 2007). There are many areas in which a small error can cause the complete failure in a simple or complex task. Thus an individual needs to be completely aware of the event occurring and have complete control of their motor responses in order to succeed at a simple let alone a complex vigilance task.

In order to be completely aware, an individual must maintain a high level of vigilance. The definition of vigilance has changed over time. In 1923 a British neurologist named Henry Head suggested that vigilance was "the extent to which the activities of a particular portion of the CNS exhibit at any moment signs of integration and purposive adaptation indicate its vigilance. When vigilance is high, the body is more prepared to respond to an effective stimulus with a more or less appropriate reaction" (Head, 1923). Davies and Parasuraman (1982) suggest that vigilance is the ability to sustain attention to a task for a period of time. Vigilance is now defined as "a state of readiness to detect and respond to certain specified small changes occurring at random time intervals in the environment" (Mackworth,

1957). Taking this information into account along with the above cognitive information, vigilance can be defined as a physiological and psychological readiness to an external event or stimuli.

In short, vigilance is the ability to stay aware for long periods of time (Cohen, 1993). One of the earliest studies of vigilance conducted by Mackworth (1950) tested the ability to detect radar signals. The findings indicated that after a 2 hour period the participants suffered from fatigue and their ability to react suffered greatly. In the past 60 years several more studies have been published on vigilance and fatigue. The findings of these studies suggest that the greatest level of fatigue is seen under conditions of high stimulus rate and low target rate. In other words, subjects become fatigued and their reaction time slows due to an information overload (Broadbent, 1950, 1971; Colquhoun and Baddeley, 1964, 1967; Corcoran and Houiston, 1977; Jerison, 1957; McGrath, 1963).

## **Physiology of Complex Vigilance Tasks**

In order for an individual to react to an event during a complex vigilance task a myriad of physiological responses must occur. First an individual must become aware of the event that is occurring. This awareness can be detected by vision, hearing or physical contact. Briefly, vision begins with the lens of the eye, which changes its curvature in order to focus the event onto the retina. The rods and cones of the eye are located in the retina and allow individuals to convert images of light into neurological signals (Nakatani and Yau, 1988). These signals are then sent via the ocular nerve to the primary visual cortex in the occipital lobes of the brain (Martini, 2001).

If the information is heard, a sound wave enters the external acoustic meatus of the ear and causes the tympanic membrane to shift its position. This movement causes information to be transmitted to the inner ear, specifically the cochlea. As a hydromechanical frequency analyzer, the cochlea's principle role is to "perform a real-time spectral decomposition of [an] acoustic signal in producing a spatial frequency map" (Dallas, 1992). Receptor cells located in the spiral organ of the cochlea send impulses back to the auditory cortex of the temporal lobe located in the brain via the vestibulocohlear nerve (VIII) (Spoendin, 1984; Martini, 2001).

Touch or physical contact is defined as "direct contact between two physical bodies" (Gardner, 2010). Four types of mechanoreceptors - Meissner corpuscles, Merkel cells, Pacinian Corpuscles and Ruffini endings - located in the skin transmit information to the cerebral cortex about the vibration, motion, weight, and form of the object or event. Each mechanoreceptor has its own field of reception that corresponds to the anatomical location of the receptor in the body. There are a higher number of receptors in areas that require a more precise response to touch. For example, the fingers have a far higher number of receptors than the forearm. The information is transmitted to the brain via the dorsal columns, medial lemniscuses and ventral posterior thalamus to the parietal lobe. Here, the information is integrated and reconstructed into an image of the object or event (Gardner, 2010). Normally an event involves a combination of vision, hearing and physical contact. Being able to hear, see and feel the event allows an individual to make a better choice about the course of action to follow: more appropriate information typically equals a better response.

Once the signal reaches the brain it is run through several processing systems in order to determine the best choice of action in response to the event. One such system, the limbic system, consists of the hippocampus and the amygdala and is responsible for establishing emotional states, linking consciousness, intellectual functions of the cerebral cortex with the unconscious and autonomic functions of the brain stem (blood pressure, muscle tension, breathing patterns etc.) and lastly, facilitating memory storage and retrieval (Martini, 2001). In short, the limbic system gives meaning to events that have been recognized. The hippocampus is important for memory formation and retrieval. A human case study of an individual who had their hippocampus removed showed severe impairments in a wide range of memory functions such as recognition of previously presented words or figures, free recall of noun pairs, and memory of the position of objects (Milner et al., 1968). It has also been shown that severe damage to the hippocampus leads to severe and permanent memory deficits, as well as anterograde amnesia and variable retrograde amnesia (Rempel-Clower et al., 1996). There are several different views of the hippocampus' role in memory. One view suggests that the hippocampus binds together different components of a learning event by linking neuronal activation in different brain regions (Squire, 1992). A second view suggests that the hippocampus is involved in the detection of novelty of stimuli (Knight, 1996). In other words, the hippocampus determines which information is relevant in order to produce a response to an event based on previous learned events. The amygdala is an essential link in the neural system underlying emotional responses. Lesions of the amygdala have been shown to interfere with the ability to express emotional responses (Gentile et al.,

1986). The amygdala is also linked to the brain stem and spinal areas that control the motor expressions of emotional responses (Phillips and LeDoux, 1992). In short, the amygdala tells an individual if an event is good or bad. An example of a response caused by the amygdala would be fight or flight (Martini, 2001). With reference to complex vigilance tasks the hippocampus determines which events are relevant and require a response while the amygdala determines what type or response is required.

Goldberg et al. (1998) used the Wisconsin Card Sorting Test in order to determine brain response to complex cognitive tasks. The researchers' findings indicate that there is an increase in cerebral blood flow (CBF) around the prefrontal cortex when a complex cognitive task is being completed. These findings correlate to previous research, as the prefrontal cortex is responsible for planning complex cognitive behavior. Along with the limbic system the prefrontal cortex- the area of the brain that orchestrates actions and responses to events and goals- determines the best choice of action (Stanfield, 2005). Damasio et al. (1991) found that individuals with damage to the prefrontal cortex had severe impairments when it came to real-life decision-making even though they preserved their overall intellect. Other findings indicate that the actions of individuals with prefrontal cortex damage are not related to future prospects but only to the immediate rewards. Using a loan gain/loss card experiment, Bechara et al. (1994) found that the individuals with prefrontal damage could not calculate long-term gains or losses in the experiment. They were not able to decide advantageously over an extended a period of time. These findings indicate that the dorsal prefrontal cortex is also not functioning

correctly. The dorsal prefrontal cortex evaluates if the course of action chosen is appropriate and approves, overrides, or finely tunes the response chosen for a given event (Martini, 2001).

Once the course of action has been approved the information is transmitted to the premotor cortex and the cerebellum. The cerebellum is the most neurologically dense part of the brain and has more neurons than the remainder of the brain combined (Williams and Herrup, 1988). It receives and transmits information to all major subdivisions of the central nervous system (Allen et al., 1997). The cerebellum has two primary motor functions: adjusting the postural muscles of the body and programming and fine-tuning movements controlled at the conscious and subconscious levels. The cerebellum compares the course of action with current proprioceptive information (position of the body) and makes any adjustments to make the movement smooth and coordinated. As the course of action takes place proprioceptive feedback is transmitted to the cerebellum via the spinocerebellar pathway. This feedback allows for any quick corrections that need to be made to the response. If information was not transmitted to the cerebellum prior to the premotor cortex, the responses would be uncoordinated and rendered almost useless (Martini, 2001).

The premotor cortex is the control hub for individuals' voluntary actions. The premotor cortex along with the supplementary motor area and the primary motor cortex play a large role in motor learning (Halsband et al., 1993). From the premotor cortex the determined choice of action is sent via 3 pathways to motor neurons: the medial pathway, the lateral pathway and the corticospinal pathway

(Enoka, 2008). The medial pathway is involved in the conscious control of muscle tone and gross movements of the neck, trunk and proximal limb muscles. The lateral pathway is involved in the conscious control of muscle tone and the precise movements of the distal parts of the limbs. The corticospinal pathway provides voluntary control over skeletal muscles that move the eye, jaw, face and some muscles of the neck and pharynx (Martini, 2001). Damage to any of the above systems or the system as a whole would slow reaction time in a complex vigilance task. For example, alcohol has been shown to directly affect the cerebellum during consumption. While an individual is severely intoxicated the cerebellum cannot process propreopceptive information using the feedback loop discussed. This lack of information processing can lead to a loss of balance while standing or sitting. This would make accomplishing complex vigilance tasks that involve movement difficult to complete.

## **Hangover Effects**

The prolonged effects of excessive acute alcohol consumption, also known as a hangover occur 7 to 16 hours after alcohol consumption has ceased and can last from 12 to 72 hours (Kim, 2003; Verster, 2010). The symptoms of a hangover are both physiological and cognitive and are often dependent on the amount of alcohol consumed (Yesavage and Leirer, 1986). There has been a significant amount of research conducted on why a hangover occurs but the mechanisms remain elusive (Wiese, 2000).

Although a hangover may seem like it only affects an individual for the indulgence of alcohol, it also has substantial economic effects. The United Kingdom

recently released a study stating that over \$2 billion pounds (\$3.2 billion Canadian) is lost per year in wages due to work missed because of a hangover (Wiese, 2000). In Canada \$1.4 billion a year is lost due to productivity decreases in the work place caused by hangovers (Wiese, 2000). Individuals who regularly abuse alcohol (over 5 drinks in one day) are 22 times more likely to miss work (Verster, 2010). Hangovers obviously affect more than just the person suffering through them.

## **Physiological Effects**

A hangover is currently defined as the occurrence of at least two of the following physiological symptoms after a bout of alcohol consumption: anorexia, headache, nausea, fatigue, tremulousness, dry mouth, diarrhea, vomiting and/or an all around poor sense of well being (Harburg, 1981). If any two of these symptoms are severe enough to disrupt an individual's daily routine then the individual is classified as having a hangover (Kim, 2003). Dehydration is also a common symptom occurring after the consumption of alcohol due to the diuretic effects of alcohol but has not been linked to having a hangover (Newlin, 1990; Penning, 2010).

There are several findings that indicate why these physiological effects occur. Several researchers have suggested hormonal changes, dehydration, alteration in blood-glucose levels, ketone bodies, lactate, blood pH, and free fatty acids as the cause of hangovers (Penning, 2010). However, no studies on the above factors have concluded any significant results. One study conducted by Kim et al. (2003) indicated that one of the prolonged effects of excessive acute alcohol consumption is an increase of some cytokines: interleukin, interferon-gamma and tumor necrosis factor-alpha. An increase in these cytokines causes the cytokine pathway to become

over stimulated. This overstimulation indicates that alcohol has an effect on an individual's immune system because these three cytokines aid the body in fighting infection. Thus the reaction to a hangover is the similar to contracting a virus or bacterial infection. Kim et al. (2003) suggests that a hangover is actually an imbalance in the immune system. Wiese et al.'s (2004) findings also indicate that an imbalance in the immune system is the cause of a hangover. Wiese et al. (2004) examined the effectiveness of Opuntia ficus indica (OFI) on hangover symptoms. OFI is a cactus found abundantly in Texas, USA and has been suggested as a hangover cure (Verster, 2010). OFI is suggested to decrease the inflammatory response which alcohol causes. Wiese et al. (2004) determined that c-reactive protein (CRP) has high levels in the blood in response to inflammation. During a hangover, individuals had an increase in CRP, however, when individuals were given OFI there was no significant increase in CRP and the severity of the hangovers was much less. OFI prevented inflammation caused by excessive acute alcohol use and thus lowered the severity of the hangover. This supports that hangovers are linked to immune imbalances and may be linked to tissue damage caused by acetaldehyde (Penning, 2010).

Acetaldehyde is made during the break down of alcohol by alcohol dehydrogenase (Penning, 2010). Acetaldehyde is metabolized rapidly in the body and is thus highly reactive. Acetaldehyde reacts with tissue causing the breakdown of this tissue to a toxic level. This tissue damage is suggested to cause nausea, sweating, rapid pulse and headache during a hangover (Penning, 2010). Chauhan et al. (1991) conducted a study using a herbal formulation suggested to prevent

hangovers called Liv.52. When Liv.52 was ingested while consuming alcohol, participants the following day had higher levels of acetaldehyde in their blood, suggesting that acetaldehyde could not react with tissues in the body. Hangover symptoms of these participants were also much less severe. Other studies have shown people who do not have the gene for aldehyde dehydrogenase (ALDH), which is required to break down acetaldehyde to acetate (which is not reactive), could drink less alcohol and still experience severe hangovers as compared to individuals with the gene for ALDH (Karamanakos, 2007; Yokoyama, 2005). All of the above studies indicate that 2 major causes of hangovers are tissue damage caused by acetaldehyde and an imbalance of the immune system.

# **Cognitive Effects**

Hangovers have many cognitive effects on an individual (Ling, 2010). Rohsenow et al. (2010) conducted a study on university graduates on the effects of a hangover on continuous performance task latency, psychomotor vigilance task latency and finger tapping latency. After having the participants ingest either a placebo or 1.2g of alcohol/kg of weight they were asked to sleep at the lab. After 10 hours the participants were woken up and their breath alcohol was tested. Any participant with a breath alcohol greater than 0.01 g% did not complete the cognitive tasks, as this would suggest they are not in the full depth of the hangover. Rohsenow et al. (2010) then concluded from the testing group that hangovers increase cognitive task latency from as little as 2% up to as much as 4%.

Another study conducted by Howland et al. (2010) also used a placebo group but gave alcohol to participants at a rate of 1.1 g/kg of body weight, and stopped

once the breath alcohol was 0.12 g%. Again after 10 hours the participants' breath alcohol was tested but this time however, any participant with breath alcohol over 0.00 g% was not tested. Howland et al. (2010) concluded that a hangover increased psychomotor vigilance task latency, decreased visual span backwards accuracy and decreased pattern memory accuracy. These two studies show decrements in both sustained attention and memory (Ling, 2010). McKinney and Coyle (2007) conducted a study in which subjects informed the researchers when they would be consuming a high amount of alcohol. The next day tests would be conducted while the individuals were suffering from a hangover. Breath alcohol had returned to 0.00 g% for all subjects except for 4% of the sample who were less than 0.05 g%. Each subject was tested a minimum of 7 hours after the consumption of alcohol had ceased. The subjects also were given a stressor of white noise during the experiment to see how a subject responded under stress while having a hangover. The subjects showed lower ability in immediate word recall, poorer delayed word recognition, longer simple reaction times and longer movement times on a reaction time test. McKinney and Coyle (2007) also found that the hangover impaired decision time.

As has been discussed, a lower level of vigilance will impair the ability to accomplish complex vigilance tasks. Ling (2010), Howland et al. (2010), Mckinney and Coyle (2007) and Roshsenow et al. (2010) all found impairments in task completion with individuals who were suffering from the symptoms of hangovers. The individuals' reaction times, decision times and recall times were all impaired due to the hangover. This impairment may have been caused by a decrease in vigilance. As fatigue is a symptom of a hangover and fatigue is a major cause of

impairments in vigilance it could be rationalized that the decreased attention would be the reason that the times were slower. If the symptoms of a hangover were to affect an individual's vigilance it could in turn disrupt their ability to complete complex vigilance tasks (Mackworth, 1950). Due to the amount attention required for such complex vigilance tasks such as driving it would be imperative that an individual maintain a high level of vigilance.

## Conclusions

As seen in the literature, cognitive and motor function are affected by a hangover. Alcohol-induced hangovers are the most commonly reported consequence of excessive alcohol consumption (Verster et al., 2010). As has been discussed, hangovers contribute to absence from the workplace, impaired job performance, lower productivity, and may even compromise daily activities such as operating a motor vehicle (Ling, 2010). Excessive use of alcohol can lead to tissue damage and cause imbalances in the immune system (Karamanakos, 2007; Penning, 2010; Yokoyama, 2005). These imbalances can lead to severe symptoms such as headache, nausea and fatigue. Fatigue causes a decrease in vigilance thus leading to impairments in attention. If an individual's attention is impaired than the completion of a complex vigilance task such as driving a motor vehicle or piloting a plane will be affected (Ling, 2010). Ironically, Rohsenow et al. (2009) found that while participants were suffering the symptoms of a hangover they gave an improved self-rating of their ability to drive the morning after alcohol consumption. Having more confidence in the ability to drive while suffering from the symptoms of a hangover could increase the likelihood of an accident.

Studies on pilots while having the symptoms of a hangover have shown a decrease in performance (Yesavage and Leirer, 1986). Yet, hangovers have received very little scientific attention and studies that have been conducted yielded inconclusive results (Verster et al., 2010). Therefore, future research is necessary to develop a full understanding of hangovers' effects on other complex vigilance tasks such as driving a motor vehicle.

# References

Adlaf, E.M., Mann, R.E., Paglia, A. (2003). Kids, drugs and cars: Alcohol, cannabis and driving among Ontario students. *Canadian Meidcal Association Journal*, *168*, 565-566.

Allen, G., Buxton, R.B., Wong, E.C., Courchesne, E. (1997). Attentional activation of the cerebellum independent of motor involvement. *Science*, *275*(*5308*), 1940-1943.

Ambrose, M.L., Bowden, S.C., Whelan, G. (2001). Working memory impairments in alcohol-dependent participants without clinical amnesia. *Alcohol Clinical and Experimental Research, 25*, 185-191.

Bechara, A., Damasio, A.R., Damasio, H., Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, *50*, 7-15.

Besser, G.M., Parke, L., Edwards, C.R.W., Forsyth, I.A. McNeilly, A.S. (1972). Galactorrhoes: successful treatment with reduction of plasma prolactin levels by brom-ergocryptine. *British Medical Journal, 3*, 669-672.

Broadbent, D.E. (1950). The twenty dials test under quiet conditions. *Associated Press Report, 130*.

Broadbent, D.E. (1971). Decision and stress. London Academic Press.

Brown, S.A., Tapert, S.F., Granholm, E., Delis D.C. (2000). Neurocognitive functioning of adolescents: effects of protracted alcohol use. *Alcohol Clinical Experimental Research 24*, 164-71.

Chauhan, B.L., Kulkarni, R.D. (1991). Effect of Liv.52, a herbal preparation, on absorption and metabolism of ethanol in humans. *European Journal of Clinical Pharmacology*, *40*, 189-191.

Cohen, R.A. (1993). The Neuropsychology of Attention. *New York: Plenum Press*.

Coluqhoun, W.P. Baddeley, A.D. (1964). Role of retest expectancy in vigilance decrement. *Journal of Experimental Psychology*, *68*,156-160.

Coluqhoun, W.P., Baddeley, A.D. (1967). Influence of signal probablkiltiy during pretraining on vigilance decrement. *Journal of Experimental Psychology, 73*, 153-155.

Cook, I.A., Bookheimer, S.Y., Mickes, L., Leuchter, A.F., Kumar, A. (2007). Aging and brain activation with working memory tasks: an fMRI study of connectivity. *International Journal of Geriatric Psychiatry*, *22*(*4*), 332-342.

Corcoran, D.W., Houston, T.G. (1977). Is the lemon test an index of arousal level? *British Journal of Psychiatry, 68*, 361-364.

Dallas, P. (1992). The active chochlea. *Journal of Neuroscience*, 2(12), 4575-4585.

Damasio, A.R., Tranel, D., Damasio, H. (1991). Somatic markers and the guidance of behavior. Frontal lobe function and dysfunction. *New York: Oxford University Press*, 217-228.

Davies, D.R., Parasuraman, R. (1982). The psychology of vigilance. *Academic Press, London and New York.* 

Enoka, R.M., Duchateau, J. (2008). Muscle fatigue: what, why and how it influences muscle function. *The Journal of Physiology*, *586(1)*, 11-23.

Gardner, E.P. (2010). Touch. Encyclopedia of Life Sciences.

Gentile, C.G., Jarrel, T.W., Teich, A., McCabe, PM., Scheider-man, N. (1986). The role of amygdaloid central nucleus in the retention of differential Pavlovian conditioning of bradycardia in rabbits. *Behavioral Brain Research, 20*, 263-273.

Goldberg, L. (1966). Behavioral and physiological effects of alcohol on man. *Behavioral-Physiological Effects, 28(4),* 570-595.

Goodwin, D.W., Othmer, E., Halikas, J.A., Freemon, F. (1970). Loss of short term memory as a predictor of the alcoholic "Blackout". *Nature, 227*, 201-202.

Halsband, U., Ito, N., Tanji, J., Freund, H.J. (1933). The role of premotor cortex and the supplementary motor area in the temporal control of movement in man. *Brain*, *116*(*1*), 243-266.

Harburg, E., Davis, D., Cummings, M.K., Gunn, R. (1981). Negative affects, alcohol consumption and hangover symptoms among normal drinkers in a small community. *Journal of Studies on Alcohol, 42(11),* 998-1012.

Head, H. (1923). The conception of nervous and mental energy. *British Journal of Psychology*, *14*, 126-147.

Heffernan, T. (2008). The impact of excessive alcohol use on prospective memory: a brief review. *Current Drug Abuse Reviews, 1,* 36-41.

Hollnagel, E., Woods, D.D. (2005). Joint cognitive systems: foundation of cognitive systems engineering. *Taylow and Francis/CRC Press, Boca Raton, FL.* 

Howland, J., Rohsnenow, D. J., Greece, J. A., Littlefield, C. A, Almeida, A., Heeren, T., Winter, M., Bliss, C.A., Hunt, S., Hermos, J. (2010). The effects of binge drinking on

college students' next-day academic test-taking performance and mood state. *Addiction*, *105(4)*, 655-665.

Jerison, H.J. (1957). Performance on a simple vigilance task in noise and quiet. *Journal of the Acoustical Society of America, 29*, 1163-1165.

Karamanakos, P.N. (2007). Pharmaceutical agents known to produce disulfiram-like reaction: effects on hepatic ethanol metabolism and brain monoamines. *International Journal of Toxicology*, *26*(*5*), 423-432.

Kim, D., Won, K., Yoon, S., Choi, B., Kim, J., Go, H., Kim, Y., Jeong, J. (2003). Effects of alcohol hangover on cytokine production in healthy subjects. *Alcohol*, *31*, 167-170.

Knight, R.T. (1996). Contriution of human hippocampal region to novelty detection. *Nature, 383*, 256–259.

Koelega, H.S. (1995). Alcohol and vigilance performance: a review. *Psychopharmacology, 118,* 233-249.

Kupari, M., Koskinen, P. (2007). Alcohol, cardiac arrhythmias and sudden death. *Novartis Foundation Symposium, 216*.

Lemon, J., Chesher, G., Fox, A., Greeley, J., Nabke, C. (1993). Investigation of the "hangover" effects of an acute does of alcohol on psychomotor performance. *Alcoholism: Clinical and Experimental Research, 17*, 665-668.

Lewis, R.K., Fawcett, S.B., Richter, K.P. (1996). Evaluating the effects of a community coalition's efforts to reduce illegal sales of alcohol and tobacco products to minors. *Journal of Community Health, 21(6),* 429-436.

Ling, J., Heffernan, T.M., Buchanan, T., Rodgers, J., Scholey, A.B., Parrott, A.C. (2003). Effects of alcohol on subjective ratings of prospective and everyday memory deficits. *Alcohol Clinical Experimental Research*, *27*, 1-5.

Ling, J., Stephens, R., Heffernan, T.M. (2010). Cognitive and psychomotor performance during alcohol hangover. *Current Drug Abuse Reviews*, 3(2), 80-87.

McGrath, J.J. (1963). Irrelevant stimulation and vigilance performance. *Vigilance: A Symposium. New York: McGraw-Hill.* 

McKinney, A., and Coyle, K. (2006). Next day effects of alcohol and an additional stressor on memory and psychomotor performance. *Journal of Studis on Alcohol and Drugs*, *68(3)*, 446-454.

Macdonald, S., Anglin-Bodrug, K., Mann, R., Chipman, M. (2005). Driving while impaired (DWI) by alcohol convictions among alcohol, cocaine, and cannabis clients in treatment. *Traffic Injury Prevention*, *6*(*3*), 207-211.

Macdonald, S., Rylett, M., Chipman, M., Mann, R., Anglin Bodrug, K., Ercikson, P., Hathaway, A., MacIntyre, P. (2004). The effects of cocaine and cannabis on driving. *Proc:* 7<sup>th</sup> World Conference on Injury Prevention and Safety Promotion. Vienna, June 6-9.

Mackworth, N.H. (1950). Researches on the measurement of human performance. *London: Medical Research Council Special Report*.

Mackworth, N.H. (1957). Some factors affecting vigilance. *Advancement of Science*, *53*, 389-393.

Martini, F.H. (2001). *Fundamentals of Anatomy and Physiology*. 5<sup>th</sup> edition. New Jersey: Prentice Hall.

Maurel, D.B., Boisseau, N., Ingrand, I., Dolleans, E., Benhamou, C., Jaffre, C. (2011). Combined effects of chronic alcohol consumption and physical activity on bone health: study in a rat model. *European Journal of Applied Physiology*, *111*, 2931-2940.

Mendelson, J.h., Teoh, S.K., Mello, N.K., Ellingboe, J. (1992). Buprenorphine attenuates the effects of cocaine on adrenocorticotropin (ACTH) secretion and mood states in man. *Neuropsychopharmacology*, *7*(*2*), 157-162.

Miller, W.R., Heather, N., Hall, W. (1991). Calculating standard drink units: international comparisons. *British Journal of Addiction, 86*, 43-47.

Milner, B., Corkin, S., Teuber, H.L. (1968). Further analysis of the hippocampal amnesic syndrome: 14-year follow-up study of H.M. *Neuropsychologia*, *6*, 317-388.

Modell, J.G., Mountz, J.M. (1990). Drinking and flying – the problem of alcohol use by pilots. *The New England Journal of Medicine, 323*, 455-461.

Naatanen, R. (1992). Attention and brain function. *Lawrence Erlbaum Associates*.

Nakatani, K., Yau, K.W. (1988). Calsium and light adaptation in retinal rods and cones. *Nature, 334(6177),* 69-71.

Newlin, D.B., Pretorius, M.B. (1990). Sons of alcoholics report greater hangover symptoms than sons of nonalcoholics: a pilot study. *Alcoholism: Clinical and Experimental Research*, 14(5), 713-716.

Penning, R., Nuland, M., Fliervoet, L.A.L., Olivier, B., Verster, J.C. (2010). The pathology of alcohol hangover. *Current Drug Abuse Reviews*, *3(2)*, 68-75.

Phillips, R.G., LeDoux, J.E. (1992). Differential contribution of amygdala and hippocampus to cued and contextual fear conditioning. *Behavioral Neuroscience*, *106(2)*, 274-285.

Rempel-Clower, N.L., Zola, S.M., Squire, L.R., Amaral, D.G. (1996). Three cases of enduring memory impairment after bilateral damage limited to the hippocampal formation. *Journal of Neuroscience*, *16*, 5233–5255.

Rohsenow, D.J., Howland, J., Arnedt, J.T., Almeida, A.B., Greece, J., Minsky, S., Kempler, C.S., Sales, S. (2009). Intoxication with bourbon versus vodka: effects on hangover, sleep and next day neurocognitive performance in young adults. *Alcoholism: Clinical and Experimental Research*, *34*(*3*), 509-518.

Rothschild, M.L., Mastin, B., Miller, T.W. (2006). Reducing alcohol impaired driving crashes through the use of social marketing. *Accident Analysis and Prevention, 38(6)*, 1218-1230.

Ryback, R.S. (1971). The continuum and specificity of the effects of alcohol on memory: A review. *Quarterly Journal of Studies on Alcohol, 32(4),* 995-1016.

Seppala, T., Leino, T., Linnoila, M., Huttunen, M., Ylikahri, R. (1976). Effects of hangover on psychomotor skills related to driving: modification by fructose and glucose. *Acta Pharmacol. Et Toxicol, 38(3),* 209-218.

Spoendin. H. (1984). Factors inducing retrograde degeneration of the cochlear nerve. *The annals of Otology, Rhinology and Laryngology, 112,* 76-82.

Squire, L.R. (1992). Memory and the hippocampus: a synthesis from findings with rats, monkeys, and humans. *Psychological Review*, *99(2)*, 195–231.

Stanfield, K.H., Kirstein, C.L. (2005). Neurochemical effects of cocaine in adolescence compared to adulthood. *Developmental Brain Research*, *159(2)*, 119-125.

Tolentino, N.J., Wierenga, C.E., Hall, S., Tapert, S.F., Paulus, M.P., Liu, T.T., Smith, T.L. Schuckit, M.A. (2011). Alcohol effects on cerebral blood flow in subjects with low and high responses to alcohol. *Alcoholism: Clinical and Experimental Research*, *35(6)*, 1-7.

Turner, R.A., Altemus, M., Yip, D.N., Kupferman, E., Fletcher, D., Bostrom, A., Lyons, D.M., Amico, J.A. (2002). Effects of emotion on oxytocin, prolactin and ACTH in Women. *Stress, 5(4),* 269-276.

Verster, J.C., Stephens, R. (2010). Editorial: the importance of raising the profile of alcohol hangover research. *Current Drug Abuse Review*, *3*(*2*), 64-67.

Verster, J.C., Stephens, R., Penning, R., Rohsenow, D., McGeary, J., Levy, D., McKinney, A., Finnigan, F., Piasecki, T.M., Adan, A., Batty, G.D., Fliervoet, L.A., Heffernan, T., Howland, J., Kim, D.J., Kruisselbrink, L.D., Ling, J., McGregor, N., Murphy, R., van Nuland, M., Oudelaar, M., Parkes, A., Prat, G., Reed, N., Slutske, W.S., Smith, G., Young, M. (2010). The alcohol hangover research group consensus statement on best practice in alcohol hangover research. *Current Drug Abuse Reviews*, *3(2)*, 116-126.

Walsh, G., Mann, R.E. (1999). On the high-road: Driving under the influence of Cannabis in Ontario. *Canadian Journal of Public Health, 90,* 260-263.

Wetherill, R.R., Fromme, K. (2011). Acute alcohol effects on narrative recall and contextual memory: an examination of fragmentary blackouts. *Addictive Behaviors*, *36(8)*, 886-889.

Wiese, J.G., McPherson, S., Odden, M.C., Shlipak, M.G. (2004). Effects of Opuntia ficus indica on symptoms of alcohol hangover. *Archives of Internal Medicine*, *164(12)*, 1334-40.

Wiese, J.G., Shlipak, M.G., Browner, W.S. (2000). The Alcohol Hangover. *Annals of Internal Medicine*, 132(11), 897-902.

Williams, R.W., Herrup, K. (1988). The control of neuron number. *Annual Review of Neuroscience*, *11*, 423-453.

Yesavage, J.A., Leirer, V. (1986). Hangover effects on aircraft pilots 14 hours after alcohol ingestion: a preliminary report. *American Journal of Psychiatry*, *143(12)*, 1546-1550.

Yokoyama, M., Yokoyama, A., Yokoyama T. (2005). Hangover susceptibility in relation to aldehyde dehydrogenase-2 genotype, alcohol flushing, and mean corpuscular volume in Japanese workers. *Alcoholism: Clinical and Experimental Research, 29,* 1165-1171.

Zirkle, G.A., King, P.D., McAtee, O.B., Van Dyke, R. (1959). Effects of chlorpromazine and alcohol on coordination and judgment. *Journal of the American Medical Association*. *171(11)*, 168-171.

# **2. CO-AUTHORSHIP STATEMENT**

The following statements clearly identify my role in the development, execution, and preparation of this thesis:

- Design and identification of the research proposal: The research idea was created by myself and expanded upon by Dr. David Behm and Dr. Michael Johnson.
- Practical aspects of research: Raw data was collected by Marie-Belle Adrao and myself.
- Data analysis: Was performed by myself under the supervision of Dr. Saied Jalal Aboodarada and Dr. David Behm.
- Manuscript preparation: Under the supervision of Dr. David Behm I prepared this manuscript.

## **3. RESEARCH MANUSCRIPT**

# An investigation into the prolonged effects of excessive acute alcohol consumption on simulated motor vehicle <u>driving skills</u>

Author: Dustin Silvey School of Human Kinetics and Recreation Memorial University St. John's, NL, A1C 5S7

Running Title: Hangover Effects on Driving

#### ABSTRACT

*Background*: The prolonged effects of excessive acute alcohol consumption (hangover) have been shown to directly affect motor and cognitive functions, however, there has been little research on how this directly affects complex vigilance tasks.

*Objective/Purpose*: To examine the effects of excessive acute alcohol consumption on complex vigilance tasks, specifically driving a motor vehicle simulator. *Methods*: Fourteen healthy participants, <u>8</u> males and 6\_females who socially consume alcohol were divided into two groups: control and intervention. The participants had to <u>complete</u> a Useful Field of View\_test (UFOV) and a <u>15-minute</u> driving scenario on a driving simulator\_at four different times (0, 4, 7, and 24 hours). Directly after the first testing sequence, participants in the intervention group were given 1 ounce of 40% vodka per 11\_kg of body weight. A series of one-way ANOVAs was performed on the two groups for all dependent variables.

<u>*Results*</u>: There were no significant differences between conditions for any of the dependent variables between groups at 7 and 24 hours. However, at 4 hours there were significant differences between groups for the overall and fuel economy scores.

<u>*Conclusions*</u>: The effects of excessive acute alcohol consumption d<u>id</u> not have an effect on complex vigilance tasks 7 or 24 hours after consumption, but alcohol consumption 4 hours prior to driving a motor vehicle simulator does significantly inhibit performance.

Key words: Complex Vigilance Task, Cognitive Functions, Field of View, Hangover

#### INTRODUCTION

Alcohol is extremely accessible, used in regular social settings, social traditions and many religious rituals and is therefore one of the most commonly used drugs in the world (Lewis, 1996; Mayer, 1998; Mode, 1990;). Alcohol is also often used in excessive amounts. The prolonged effects of excessive acute alcohol consumption, also known as a hangover, can last from 12 to 72 hours and occur 7 to 16 hours after alcohol consumption has ceased (Kim, 2003; Verster, 2010). There are both physiological and cognitive symptoms of a hangover <u>that</u> are often dependent on the amount of alcohol consumed (Yesavage and Leirer, 1986). There has been a significant amount of research conducted on why a hangover occurs but the data remains inconclusive (Wiese, 2000).

A hangover is currently defined as the occurrence of at least two of the following physiological symptoms after a bout of alcohol consumption: anorexia, headache, nausea, fatigue, tremulousness, dry mouth, diarrhea, vomiting and/or a general poor sense of well being (Harburg, 1981). If any two of these symptoms are severe enough to disrupt an individual's daily routine then the individual is classified as hungover (Kim, 2003). Hangovers have many cognitive effects on an individual (Ling, 2010) such as impaired attention, memory, pattern memory accuracy, as well as increased cognitive task latency, and psychomotor vigilance task latency, (Howland et al., 2010; Ling, 2003, 2010; McKinney and Coyle, 2007; Rohsenow et al., 2010; Yesavage and Leirer, 1986). The question still remains regarding the duration of significant physiological impairments associated with excessive alcohol consumption.

Current Federal Aviation Administration (FAA) regulations indicate that a pilot may operate a commercial airplane 8 hours after they <u>consume</u> their last alcoholic beverage (Federal Aviation regulation 91.17). However, because of the cognitive effects of hangovers, piloting after alcohol is no longer directly affecting the central nervous system may still be dangerous. Reaction time is important <u>for</u> complex vigilance tasks such as driving or piloting a plane, in that the threedimensional environments are changing at a rapid pace and cognitive function must be high in order to fully analyze and react to the changing situations. This suggests that driving a motor vehicle or piloting a plane while impaired by a hangover could also be dangerous.

There have been numerous studies conducted on how alcohol <u>consumption</u> or the presence of alcohol in the system affects complex vigilance tasks such as driving (Koelega, 1995). Although there have been several studies on the effects of hangovers on cognitive function there is little consistency with the procedures (Howland et al., 2010; Ling, 2010; McKinney and Coyle, 2007). Many of the studies conducted did not establish a blood alcohol level of zero during the testing (Howland et al., 2010; Ling, 2010; McKinney and Coyle, 2007). Other studies have not used a placebo control, have not controlled for the amount of alcohol consumed by the participants, have not used a standard amount of alcohol for each participant and have not used a standard time from consumption until testing (Howland et al., 2010; Ling, 2010; McKinney and Coyle, 2007). Thus, the objective of this research was to determine if the prolonged effects of excessive acute alcohol consumption have any effect on complex vigilance tasks such as driving a motor vehicle. It is

hypothesized that a hangover will cause impairments in completing complex vigilant tasks <u>such as performance in a</u> driving simulator.

#### **METHODOLGY**

#### **Research design**

An experimental study was conducted on the acute effects of alcohol binge drinking on complex vigilance tasks. In this study alcohol was used as the intervention (independent variable) and participants were tested using a <u>Useful</u> <u>Field of View (UFOV)</u> test and a driving simulator which included the following variables; control of the vehicle, the level of road safety, ability to follow legal aspects, mobility within the vehicle, road sharing, and lastly fuel economy (dependent variables) at 4 time periods including 0, 4, 7, and 24 hours after alcohol consumption.

#### **Participants**

Based on a power analysis <u>of related articles (Kim, 2003; Lemon, 1993;</u> Seppala, 1976; Yesavage and Leirer, 1986) fourteen subjects, <u>8</u> males (77.58 ± 14.04 kg, 181.42 ± 6.43 cm, 24 ± 7.82 years) and <u>6</u> females (83.1 ± 44.03 kg, 179.07 ± 8.98 cm, 22.17 ± 2.64 years) participated in this study. Accidental <u>and snowball</u> sampling were employed. All participants had a valid driver's license and had consumed alcohol at some point in their lives. Participants were screened by a registered nurse prior to the study to ensure they had no medical issues restricting them from the use of alcohol. <u>Participants were excluded from the data collection if they did not</u> have a valid driver's license, if they experienced motion sickness during the practice <u>scenario on the driving simulator, or if they had any conditions that would make the</u> <u>acute consumption of alcohol harmful.</u> Participants were paid \$50<u>to offset expenses</u> to participate in this study.

<u>Ethical</u> approval <u>was obtained from the Human Research Ethics Authority of</u> Memorial University <u>(primary investigator) (Reference # 12.216)</u>, <u>as well as from</u> l'Université de Moncton <u>(data collection site) (Reference # 1213-032)</u>.

#### Procedure

Prior to the evaluation session, each participant attended the driving lab<u>oratory</u> at l'Université de Moncton to assess motion sickness<u>in the driving</u> <u>simulator (Virage</u> Simulation Inc, Montreal, Quebec, Canada). The <u>driving</u> scenario during this <u>orientation</u> session was not the same as the <u>scenario</u> utilised during the data collection session. None of the participants who tried the simulator experienced motion sickness prior to or during the data collection session.

During the data collection session, the participants attended the driving lab<u>oratory</u>, in pairs, early in the morning. The consent form was explained and signed. A registered nurse to ensure that the subjects did not have any medical restrictions associated with the consumption of alcohol then interviewed the participants. The <u>pass/fail</u> breathalyzer was also administered to ensure the participants' blood-alcohol level was zero. <u>Subsequently, the participants</u> had to <u>complete</u> the Useful Field of View\_test (UFOV) and a <u>15-minute</u> driving scenario on the driving simulator <u>for the baseline measures</u>.

The participants were weighed and alcohol was <u>provided</u> to each participant based on body weight (1 ounce of vodka per 11\_kg). Forty percent alcohol vodka

was provided and participants could drink the alcohol pure, or mix it with noncarbonated water at the ratio of 2:1 (water: vodka). The participants had one hour to drink the <u>prescribed</u> amount of alcohol and the alcohol was divided in 6 equal amounts, which was <u>consumed</u> every 10 minutes. During this hour, participants were not allowed to drink or eat anything else.

Four hour<u>s</u> post<u>-</u>alcohol consumption, participants were asked to complete both evaluations again (UFOV and driving simulator). The participants were again administered the breathalyzer, which they all failed 4 hour<u>s after</u> alcohol consumption. <u>During the testing day, the participants were allowed to eat and play</u> <u>video games</u> both of which were provided in the laboratory.

Participants <u>took</u> the breathalyzer <u>test</u> again at 7 hours post\_alcohol consumption at which point all participants passed (blood alcohol of zero percent). Once the participants passed the breathalyser, they were asked to complete the evaluations again (UFOV and driving simulator). Once they had completed these evaluations, participants were sent home by taxi. They were asked not to consume any alcohol again until after the completion of the test the following morning. Twenty-four hour post\_alcohol consumption, participants returned to the driving lab and the breathalyzer was administered prior to the evaluation to ensure all participants were sober. The participants then completed both evaluations (UFOV and driving simulator) for a final time. The control group underwent the same procedures without the consumption of alcohol. All data was then normalized, post test time courses (4, 7, and 24 hour) with the pre-test for each of the variables in each group (Intervention and Control).

## **Measures**

<u>Measuring devices included the UFOV, Virage driving simulator, cardiac</u> <u>monitor and portable breathalyzer.</u>

The UFOV (Visual Awareness Research Group, Punta Gorda, Florida, USA) is a computer-based test used to evaluate the visual field in which a person can extract information in a brief glance through the evaluation of processing speed, divided attention and selected attention by measuring the speed of visual processing under increasingly complex task demands (Figure 1). Using both eyes, the examinee must detect, identify and localize briefly presented targets. <u>Goode et al. (1998) have</u> investigated the use of the UFOV and found that "in terms of cognitive assessment of driving risk, the results of the current investigation support the use of a stand-alone measure of visual attention for assessing older adults' risk for automobile crashing" (Goode et al., 1998).

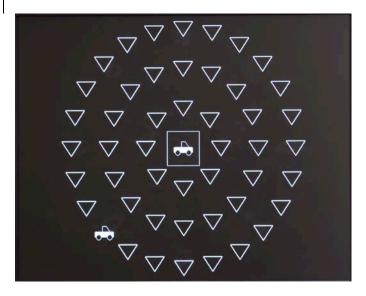


Figure 1. Useful Field of View Test (UFOV). Participants are shown the above image for a fraction of a second after which they have to determine where the outside car was located on the screen.

The Virage Simulator (VS500M, Virage Simulation Inc, Montreal, Quebec, Canada) is a level 3 simulator (uses screens, a cabin, and vibrates similar to a normal vehicle) used to simulate driving conditions in numerous environments and situations. (Figure 2 and Figure 3). Prior to the simulation, participants were given the following instructions: "You will be driving a 15 minute driving scenario that will include city driving and highway driving. You will receive verbal instructions via the simulator in the scenario on where to go. Please drive as you would normally drive and follow all the speed limits and driving rules." The simulator graded the participants out of 100 for the following: control of the vehicle, the level of road safety, ability to follow legal aspects, mobility within the vehicle, road sharing, and lastly fuel economy. A score from each of these was taken and an overall score was given for each participant each time they drove the simulator.



Figure 2. Participant in the Virage Simulator. The three large screens give a full frontal view similar to that of a windshield and the two smalls screens mimic the blind spots found while driving a motor vehicle.



Figure 3. The view of participants while driving the Virage Simulator. The small screen in the top right functions as a rear view mirror while the small one on the lower left functions as a side mirror.

<u>A portable pass/fail breathalyzer (Alcotest</u>® 7410 GLC Manufacturer: Draeger Canada Ltd. 7555 Danbro Crescent, Mississauga, ON, L5N 6P9) <u>was used</u> <u>during the procedure to evaluate participants' blood alcohol levels at different times</u> <u>during the data collection session. The breathalyzer was on loan from the Royal</u> <u>Canadian Mounted Police (RCMP) and the researchers received a 3 hour course on</u> <u>the use of the portable breathalyzer by a certified instructor from the RCMP.</u>

## **Statistical Analysis**

Statistical Analysis were computed on normalized data using SPSS (Version 16.0, SPSS, Inc, Chicago, IL). A series of <u>separate</u> one way measures analysis of variance (ANOVA) w<u>ere</u> used to identify the <u>differences between the</u> 2 conditions (Intervention and Control) <u>at the</u> 3 times (4, 7, and 24 hours). <u>Times were</u> <u>normalized to pre-test scores and analyzed separately as differences between drunk</u> (<u>4 hours</u>) and hungover conditions was not the objective of the present study. Independent variables including an overall score, control of the vehicle, the level of road safety, ability to follow legal aspects, mobility within the vehicle, road sharing, fuel economy, and UFOV. Post hoc (Tukey) was used to identify significant difference between the two groups. Significance was defined as p < 0.05. Additionally, Cohen effect size statistics (ES) were conducted to evaluate the magnitude of the changes in various DJs according to the criterion of >0.80 large, 0.4-0.79 moderate, and 0.2-0.39 small (Cohen, 1988). Lastly, percent differences were calculated between the mean scores for each dependent variable at each time.

#### RESULTS

There was a lack of significant results for the UFOV and all variables of the simulator other than the overall score (p < 0.05) and fuel economy (p < 0.015). The mean absolute values and standard deviation for each variable of the simulator and the UFOV are shown in Table 1.

		Overall	Control of Vehicle	Road Safety	Legal Aspect	Mobility	Road Sharing	Fuel Economy	UFOV
		Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Pretest									
	interv	46.14 (23.23)	52.83 (21.32)	36.83 (20.28)	49.29 (22.05)	75.43 (15)	63.17 (27.01)	45 (16.85)	97.88 (36.67)
	control	53.14 (24.7)	52.29 (28.48)	58.43 (20.21)	66.71 (13.78)	59.57 (31.41)	58.71 (34.69)	53.71 (16.99)	68.63 (20.47)
4 hours									
	interv	32 (22.46)	57.33 (22.96)	16 (20.58)	33.14 (30.67)	71.57 (35.11)	57.67 (25.45)	18.86 (21.15)	94.25 (31.56)
	control	54.86 (17.78)	52.14 (17.65)	41.29 (26.88)	64.86 (15.73)	71.71 (23.25)	67.71 (18.81)	48.71 (10.95)	57.57 (14.15)
7 hours									
	interv	52.71 (19.65)	74.5 (6.89)	43 (48.45)	53.43 (15.8)	89.29 (24.95)	82.5 (11.33)	46.43 (16.01)	74.25 (34.49)
	control	60.29 (18.51)	62 (19.31)	48.29 (29.71)	66.86 (10.32)	77.57 (19.6)	72.14 (24.43)	53.14 (20.09)	54 (16.43)
24 hours									

Table 1. Absolute values for each simulation and <u>Useful Field of View (</u>UFOV<u>)</u> test at 4, 7, and 24 hours.

interv	56.86 (19.63)	74.5 (6.03)	49.5 (21.48)	54.57 (25)	93.29 (10.26)	87 (7.54)	45 (16.53)	72 (28.44)
control	74.14 (13.61)	78 (9.68)	67.71 (22.57)	74 (13)	91.86 (11.05)	85.85 (12.35)	66 (19.52)	36.43 (10.29)

There were significant reductions at the 4-hour testing period for the overall

(p < 0.05: Table 2, Figure 4) and fuel economy (p < 0.015: Table 3, Figure 5) scores

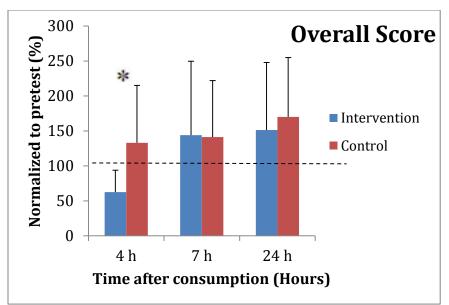
between the intervention and control group<u>s.</u>

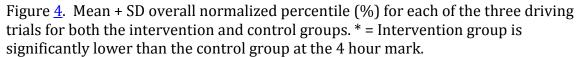
Table 2. Overall score normalized statistical values for each simulator trial at 4, 7, and 24 hours with percent differences. <u>SD: Standard deviation. CI: Confidence</u> <u>Intervals. ES: effect size.</u>

				95	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	62.48	31.30	33.54	91.43			
4 hrs		<u>∆</u> 72.08%				4.47	0.05	1.13
	control	132.90	82.33	56.76	209.04			
	interv	143.91	105.88	45.99	241.84			
7 hrs		<u>∆</u> 1.83%				0.003	0.96	0.03
	control	141.30	80.74	66.63	215.97			
	interv	151.20	96.60	61.85	240.54			
24 hrs		<u>∆</u> 11.71%				0.15	0.71	0.21
	control	170.00	85.09	91.28	248.67			

Table 3. Fuel Economy normalized statistical values for each simulator tr <u>ia</u> l at 4, 7,
and 24 hours with percent differences. <u>SD: Standard deviation. CI: Confidence</u>
Intervals. ES: effect size.

				95%	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	43.00	16.91	1.62	84.37			
4 hrs		<u>∆</u> 74.25%				8.10	0.02	3.18
	control	93.78	15.01	79.90	107.65			
	interv	104.68	17.85	88.18	121.19			
7 hrs		<u>∆</u> 34.40%				0.06	0.81	0.13
	control	101.14	33.43	70.22	132.05			
	interv	112.45	53.32	63.13	161.76			
24 hrs		<u>∆</u> 14.41%				0.46	0.51	0.36
	control	129.91	42.46	90.64	169.17			





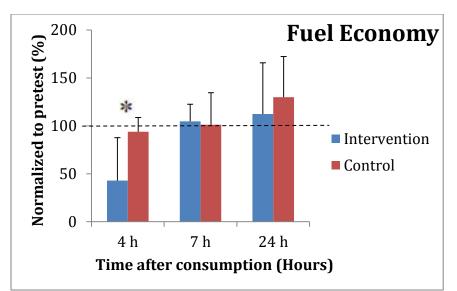


Figure <u>5</u>. Mean + SD fuel economy normalized percentile (%) for each of the three driving trails for both the intervention and control groups. \* = Intervention group is significantly lower than the control group at the 4 hour mark.

Tables 4 - 9 <u>illustrate</u> no significant results at 4, 7 or 24 hours between the control and interventions groups. However, <u>based on effect size calculations</u>, there were several moderate magnitude based changes. Tables 5 - 7 indicate that there <u>were</u> moderate changes at 4 hours for road safety (ES = 0.76), legal aspect (ES = 0.47), mobility (ES = 0.64), and road sharing (ES = 0.77) respectively. Table 5\_7 indicate that there <u>were</u> moderate changes at 7 hours for road safety (ES = 0.59), legal aspect (ES = 0.5) and mobility (ES = 0.55). Table 7 also indicates a moderate change at 24 hours for mobility (ES = 0.73).

Table 4. Control of Vehicle normalized statistical values for each simulator trial at 4, 7, and 24 hours with percent differences. <u>SD: Standard deviation. CI: Confidence</u> <u>Intervals. ES: effect size.</u>

				95	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	121.53	54.36	64.48	178.57			
4 hrs		<u>∆</u> 27.98%				0.39	0.55	0.35
	control	161.06	146.13	25.91	296.21			

	interv	190.57	159.72	22.96	358.18			
7 hrs		<u>∆</u> 11.52%				0.70	0.80	0.15
	control	169.81	122.26	56.74	282.88			
	interv	190.58	159.65	23.03	358.12			
24 hrs		<u>∆</u> 27.50%				0.29	0.60	0.31
	control	251.34	231.89	36.88	465.81			

Table 5. Road Safety normalized statistical values for each simulator tr<u>ia</u>l at 4, 7, and 24 hours with percent differences. <u>SD: Standard deviation. CI: Confidence Intervals.</u> <u>ES: effect size.</u>

				95	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	39.01	48.25	-11.63	89.65			
4 hrs		<u>∆</u> 64.81%				1.84	0.20	0.76
	control	76.41	50.66	29.56	123.27			
	interv	127.14	80.00	43.19	211.09			
7 hrs		<u>∆</u> 37.38%				1.15	0.31	0.59
	control	87.10	54.38	36.80	137.39			
	interv	162.69	104.15	53.89	271.99			
24 hrs		<u>∆</u> 27.30%				0.73	0.41	0.46
	control	123.61	58.42	69.58	177.64			

				95	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	74.17	78.11	1.93	146.41			
4 hrs		<u>∆</u> 32.66%				0.78	0.39	0.47
	control	103.12	37.67	68.28	137.96			
	interv	140.62	96.45	51.41	229.83			
7 hrs		<u>∆</u> 29.22%				0.88	0.37	0.50
	control	104.91	29.87	77.28	132.54			
	interv	131.05	87.93	49.73	212.37			
24 hrs		<u>∆</u> 7.66%				0.30	0.59	0.29
	control	112.40	18.03	95.72	129.07			

Table 6. Legal Aspect normalized statistical values for each simulator tr<u>ia</u>l at 4, 7, and 24 hours with percent differences. <u>SD: Standard deviation. CI: Confidence</u> <u>Intervals. ES: effect size.</u>

Table 7. Mobility normalized statistical values for each simulator tr<u>ia</u>l at 4, 7, and 24 hours with percent differences. <u>SD: Standard deviation. CI: Confidence Intervals. ES:</u> <u>effect size.</u>

				95%	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	92.97	45.77	50.63	135.30			
4 hrs		<u>∆</u> 65.15%				1.44	0.25	0.64
	control	182.80	192.89	4.40	361.19			
	interv	119.68	36.78	85.67	153.69			
7 hrs		<u>∆</u> 49.37%				1.04	0.33	0.55
	control	198.14	199.87	13.29	382.98			
	interv	127.05	23.60	105.22	148.88			
24 hrs		<u>∆</u> 55.79%				1.85	0.20	0.73
	control	225.34	189.56	50.03	400.66			

				95%	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	97.92	33.80	62.45	133.38			
4 hrs		<u>∆</u> 59.31%				1.76	0.21	0.77
	control	180.47	148.43	43.19	317.74			
	interv	173.11	136.72	29.63	316.60			
7 hrs		<u>∆</u> 6.91%				0.02	0.88	0.09
	control	185.50	153.03	43.98	327.03			
	interv	184.10	147.06	29.77	338.42			
24 hrs		<u>∆</u> 19.88%				0.20	0.66	0.25
	control	224.73	173.13	64.61	384.85			

Table 8. Road Sharing normalized statistical values for each simulator tr<u>ial</u> at 4, 7, and 24 hours with percent differences. <u>SD: Standard deviation. CI: Confidence</u> <u>Intervals. ES: effect size.</u>

Table 9. <u>Useful Field of View (</u>UFOV) normalized statistical values for each trial at 4, 7, and 24 hours. <u>SD: Standard deviation. CI: Confidence Intervals. ES: effect size.</u>

				95%	% CI			
Time	Group	Mean	SD	Lower	Upper	F value	Sig	ES
	interv	97.86	19.36	81.68	114.04			
4 hrs		<u>∆</u> 0.77%				0.002	0.97	0.02
	control	97.05	53.49	47.58	146.52			
	interv	78.34	38.15	46.44	110.23			
7 hrs		<u>∆</u> 9.96%				0.14	0.71	0.19
	control	86.55	46.27	43.75	129.35			
	interv	75.37	25.33	54.19	96.55			
24 hrs		<u>∆</u> 27.38%				2.05	0.18	0.74
	control	57.22	23.43	35.55	78.88			

#### DISCUSSION

The most important findings of the present study were contrary to the hypothesis; the prolonged effects of excessive acute alcohol consumption (a hangover<u>at 7 and 24 hours</u>) did not have a debilitating effect on complex vigilance tasks. Secondly, when an individual is under the influence of alcohol (blood alcohol concentration (BAC) greater than 0.08 g%) their ability to complete complex vigilance tasks such as driving a motor vehicle decreases.

Contrary to the literature, this study indicates that the prolonged effects of excessive acute alcohol consumption (hangover) did not have an effect on complex vigilance tasks such as driving a motor vehicle simulator. Several studies have indicated that the effects of a hangover hinder functions required to complete complex vigilance tasks such as cognitive function, psychomotor vigilance, pattern memory accuracy, attention, reaction speed, movement speed, and decision making (Howland et al., 2010; Ling, 2010; McKinney and Coyle, 2007; Mackworth, 1950; Rohsenow et al., 2010; Seppala et al., 1976; and Yesavage and Leirer, 1986). Rohsennow et al. (2010) and Howland et al. (2010) both found that 10 hours after excessive alcohol consumption, participants had a decrease in task latency (increase in task completion) and memory when their BAC was 0.00 g%. Another study by McKinney and Coyle (2007) found that participants suffering from a hangover had a lower ability for word recall and had decrements in word recognition, reaction times, decision times and movement times after a night of binge drinking. However, McKinney and Coyle (2007) did not wait for all participants to have a blood alcohol level of 0.00 g%, therefore, their findings were more likely caused by the cognitive

effects of alcohol ingestion rather than the prolonged effects of excessive acute alcohol consumption. Lastly, Yesavage and Leirer (1986) conducted a study on military pilots, who used a flight simulator 16 hours after a BAC of 0.10 g%. All the pilots scored significantly lower on the simulator while enduring a hangover, indicating that hangovers did have an effect on complex vigilance tasks. The present study did not have any significant differences in the completion of complex vigilance tasks 7 or 24 hours after consuming enough alcohol to have a BAC above 0.08 g% or any decrements in the Useful Field of View test, indicating that during a hangover participants did not have decrements in their visual processing speed, divided attention or selective attention.

Several explanations can be offered for the results of the lack of hangover findings in this investigation. The first being that the participants were mentally preparing, self-motivating or "psyching themselves up" prior to completing the tests at 7 hours. Several studies have shown that individuals have higher performance scores if they "psych up" prior to completing motor and cognitive tasks (Caudill et al., 1983; Sackett, 1934, 1935; Shelton and Mahoney, 1978; Weinberg et al., 1980). Shelton and Mahoney (1978) measured grip strength in two groups of individuals; the first group was told to count backwards by sevens from a four-digit number prior to completing the test while the other group was told to "psych up" prior to the test. The group that was instructed to "psych up" had a higher grip score than those that were told to count backwards. Another study by Weinberg et al. (1980) showed that not only does "psyching up" increase strength test results, but increases attention levels as well. It was clear to the researchers that the

intervention group were mentally preparing <u>and motivating</u> themselves prior to driving the simulator at 7 hours, but during the pre-test this <u>arousal</u> was not noticeable. In addition, "psyching up" was not seen prior to the control group driving the simulator at any time. This "psyching up" may have influenced the intervention group's scores by allowing them to score higher. As individuals often do not "psych up" before driving home after a night of ingesting alcohol this may be the reason for not seeing a significant difference between the control and intervention group at 7 hours for any of the dependent variables.

The present experiment was one of the first to look at the prolonged effects of excessive acute alcohol consumption after a period of 24 hours. Yesavage and Leirer (1986) suggested that a hangover can last up to 72 hours if enough alcohol is consumed at one time and conducted a similar study 16 hours after ingestion. In the present study, we may have not given the intervention group sufficient alcohol to induce a strong enough hangover to last for the duration of 24 hours. Perhaps if a higher amount of alcohol were given to participants this may have induced a stronger hangover (more severe symptoms) <u>resulting in possible significant effects</u> <u>at the extended duration testing period (24 hours)</u>. To determine the validity of these explanations, however, will require further research.

The finding of decreases in the ability to complete complex vigilance tasks as a result of a blood alcohol level greater than 0.08 g% is similar to other studies that measured the effects of alcohol consumption on driving (Arndet et al., 2001; Fillmore et al., 1994; Legel, 1994; <u>Modell, 1990;</u> Morrow et al., 1990; Moskowitz, 1973; and Moskowtiz and Robinson, 1988). Moskowtiz (1973) found that when

isolated, specific functions required for driving a motor vehicle such as vision, tracking and division of attention were not hindered by a 0.03 g% BAC. However, when these same specific functions were examined together during a complex task there was a large performance decrement at the same BAC. Fillmore et al. (1994) also found that when participants had a BAC of 0.054 g% their adaptive tracking abilities used for driving were significantly hindered. In another study, Morrow et al. (1990) found that a BAC of 0.001 g% impaired the ability to operate a flight simulator. The BAC difference between the studies may be related to the complexity of each test. Morrow et al.'s (1990) flight simulator test was considered extreme, with participants performing 5 tasks required for flying at one time, while Fillmore et al.'s (1994) study simply required participants to visually track an object for a short period of time. In the present study, higher BACs were used (minimum 0.08 g%) and a complex vigilance task was used, but the task was not as complex as Morrow et al.'s (1990); it required fewer tasks to be completed at one time. In a review by Moskowtiz and Robinson (1988) it was indicated that divided attention and tracking are the first two complex vigilance behaviors affected by alcohol consumption. They showed that at BAC 0.02 g%, divided attention starts to become impaired and at BAC 0.05 g% tracking ability starts to decrease. Since 1980 there have been few studies that suggest alcohol does not hinder driving performance (Moskowtiz and Fiorentiono, 2000). The present study provides support to the theory that alcohol inhibits complex vigilance tasks.

A limitation of this study is the small sample size, increasing the risk of Type II statistical errors. Another limitation of this study was the use of the same

simulator course for each trial (pre-test, 4 hours, 7 hours, and 24 hours). Having the participants complete the same course 4 times in 24 hours allowed them to learn where each incident would occur and gave them the ability to prepare themselves. However the use of a comparative control group ensured that the learning effect was taken into consideration with the analysis. One last limitation of this study was the assumption that all hangovers were equivalent in severity. In order to improve this study we suggest the use of a larger sample size, the use of 4 different driving courses with the same incidents but randomized, and lastly, examining the severity of the hangovers prior to testing.

#### CONCLUSION

The results of the present study indicate that the prolonged effects of excessive acute alcohol consumption do not have an effect on driving a motor vehicle simulator 7 or 24 hours after consumption. However, the present study does reinforce the well-known idea that consuming alcohol prior to completing complex vigilance tasks will hinder performance (Mean  $\Delta$ 72.08%). Thus, alcohol should not be consumed 4 hours before operating any type of motor vehicle and future studies should address the effects of higher levels of alcohol consumption on operating a motor vehicle after longer durations once consumption has stopped.

#### REFERENCES

Arnedt, J. T., Wilde, G. J. S., Munt, W. P., MacLean, W. A. (2001). How do prolonged wakefulness and alcohol compare in the decrements they produce on a simulated driving task? *Accident Analysis & Prevention*, *33*(*3*), 337-344.

Caudill, D., Weinberg, R., Jackson, A. (1983). Psyching-up and track athletes: A preliminary investigation. *Journal of Sport Psychology*, *5*(*2*), 231-235.

Cohen J. (1988). Statistical power analysis for the behavioral sciences (2nd ed.) Hillsdale, New Jersey: Lawrence Erlbaum Associates.

Fillmore, M.T., Voget-Sprott, M. (1995). Behavioural effects of alcohol in novice and experienced drinkers: Alcohol expectancies and impairment. *Psychopharmacology*, *122*, 175-181.

Goode, K.T., Ball, K.K., Sloane, M., Roenker, D.L., Roth, D.L., Myers, R.S., & Owsley, C. (1998). Useful field of view and other neurocognitive indicators of crash risk in older adults. Journal of Clinical Psychology in Medical Settings, 5,

425-440.

Harburg, E., Davis, D., Cummings, M.K., Gunn, R. (1981). Negative affects, alcohol consumption and hangover symptoms among normal drinkers in a small community. *Journal of Studies on Alcohol, 42(11),* 998-1012.

Howland, J., Rohsnenow, D.J., Greece, J.A., Littlefield, C.A, Almeida, A., Heeren, T., Winter, M., Bliss, C. A., Hunt, S., Hermos, J. (2010). The effects of binge drinking on college students' next-day academic test-taking performance and mood state. *Addiction*, *105(4)*, 655-665.

Kim, D., Won, K., Yoon, S., Choi, B., Kim, J., Go, H., Kim, Y., Jeong, J. (2003). Effects of alcohol hangover on cytokine production in healthy subjects. *Alcohol*, *31*, 167-170.

Koelega, H.S. (1995). Alcohol and vigilance performance: a review. *Psychopharmacology, 118,* 233-249.

Lewis, R.K., Fawcett, S.B., Richter, K.P. (1996). Evaluating the effects of a community coalition's efforts to reduce illegal sales of alcohol and tobacco products to minors. *Journal of Community Health*, *21(6)*, 429-436.

Ling, J., Heffernan, T.M., Buchanan, T., Rodgers, J., Scholey, A.B., Parrott, A.C. (2003). Effects of alcohol on subjective ratings of prospective and everyday memory deficits. *Alcohol Clinical Experimental Research*, *27*, 1-5.

Ling, J., Stephens, R., Heffernan, T.M. (2010). Cognitive and psychomotor performance during alcohol hangover. *Current Drug Abuse Reviews, 3(2),* 80-87.

McKinney, A., and Coyle, K. (2006). Next day effects of alcohol and an additional stressor on memory and psychomotor performance. *Journal of Studis on Alcohol and Drugs, 68(3),* 446-454.

Mackworth, N.H. (1950). Researches on the measurement of human performance. *London: Medical Research Council Special Report*.

Model, J.G., Mountz, J.M. (1990). Drinking and flying – the problem of alcohol use by pilots. *The New England Journal of Medicine, 323*, 455-461.

Morrow, D., Leirer, V., Yesavage, J. (1990). The influence of alcohol and again on radio communication during flight. *Aviation, Space and Environmental Medicine, 61,* 12-20.

Moskowitz, H. (1973). Laboratory studies of the effects of alcohol on some variables related to driving. *Journal of Safety and Research*, *5(3)*, 185-199.

Moskowitz, H., Burns, M. (1973). Alcohol effects on information processing time with an overlearned task. *Perceptual and Motor Skills, 37,* 835-839.

Moskowtiz, H, Robinson, C.D. (1988). Effects of low doses of alcohol on drivingrelated skills, a review of the evidence. *U.S. Department of Transportation*.

Rohsenow, D.J., Howland, J., Arnedt, J.T., Almeida, A.B., Greece, J., Minsky, S., Kempler, C.S., Sales, S. (2009). Intoxication with bourbon versus vodka: effects on hangover, sleep and next day neurocognitive performance in young adults. *Alcoholism: Clinical and Experimental Research*, *34*(*3*), 509-518.

Sackett, R.S. (1934). The influences of symbolic rehearsal upon the retention of maze habit. *Journal of General Psychology, 10,* 376-395.

Sackett, R.S. (1935). The relationship between amount of symbolic rehearsal and rention of maze habit. *Journal of General Psychology*, *13*, 113-128.

Seppala, T., Leino, T., Linnoila, M., Huttunen, M., Ylikahri, R. (1976). Effects of hangover on psychomotor skills related to driving: modification by fructose and glucose. *Acta Pharmacol. Et Toxicol, 38(3),* 209-218.

Shelton, T.O., Mahoney, M.J. (1978). The content and effect of "psyching-up" strategies in weight lifters. *Cognitive Therapy and Research, 2,* 275-284. Verster, J.C., Stephens, R. (2010). Editorial: the importance of raising the profile of alcohol hangover research. *Current Drug Abuse Review, 3(2),* 64-67.

Verster, J.C., Stephens, R., Penning, R., Rohsenow, D., McGeary, J., Levy, D., McKinney, A., Finnigan, F., Piasecki, T.M., Adan, A., Batty, G.D., Fliervoet, L.A., Heffernan, T., Howland, J., Kim, D.J., Kruisselbrink, L.D., Ling, J., McGregor, N., Murphy, R., van Nuland, M., Oudelaar, M., Parkes, A., Prat, G., Reed, N., Slutske, W.S., Smith, G., Young, M. (2010). The alcohol hangover research group consensus statement on best practice in alcohol hangover research. *Current Drug Abuse Reviews*, *3(2)*, 116-126.

Weinberg, R.S., Yukelson, D., Jackson, A. (1980). Effect of public and private efficacy expectations on competitive performance. *Journal of Sport Psychology*, *2*, 340-349.

Wiese, J.G., Shlipak, M.G., Browner, W.S. (2000). The Alcohol Hangover. *Annals of Internal Medicine*, *132(11)*, 897-902.

Yesavage, J.A., Leirer, V. (1986). Hangover effects on aircraft pilots 14 hours after alcohol ingestion: a preliminary report. *American Journal of Psychiatry*, *143(12)*, 1546-1550.