THE RELATIONSHIP BETWEEN THE ALCOHOL USE PATTERNS AND COGNITIVE AND SYMPTOMATIC FUNCTIONING OF RHODES UNIVERSITY STUDENTS

Tamara Ann Mayson
Supervised by Professors Charles Young and Ann Edwards

A thesis submitted in fulfilment of the requirements for the degree of Master of Arts

The Psychology Department,
Rhodes University,
Grahamstown,
South Africa,

Abstract

Introduction: Studies reveal differential cognitive effects of alcohol use, with moderate to heavy alcohol having some beneficial effects in older adults, whereas in young adulthood, heavier alcohol use produces deficits. The aim of the research was to investigate the relationship between patterns of alcohol use and the cognitive and symptomatic functioning of university students over one year, and to discern which demographic, clinical and alcohol factors might moderate cognitive outcomes.

Method: The sample included undergraduate Psychology students from a South African University, aged 18 to 23 years (n = 86). The sample was divided into two alcohol use groups based on four categories from the Alcohol Use Disorders Identification Test (AUDIT) (Babor et al., 2001), including a lighter drinking group (Safe/Hazardous, n = 74) and a heavier drinking group (Harmful/Dependent, n = 12). The two drinking groups were statistically equivalent for distribution of age, gender, English first language, race groups, history of neurological or psychiatric vulnerability, a measure of initial intelligence/cognitive reserve (Peabody Picture Vocabulary Test: PPVT-4) (Dunn & Dunn, 2007) and for a measure of test-taking validity (Immediate Postconcussion Assessment and Cognitive Test: ImPACT Impulse Control) (p = < 0.05 in all instances). Participants were tested on the computerized ImPACT battery (ImPACT, 2007) at three intervals over an academic year (May, August, October 2012). Repeated measures ANOVAs were used to investigate cognitive and symptomatic differences between the two alcohol use groups, in association with moderating factors.

Results: The heavier drinking group revealed: (i) poorer performance on ImPACT Visual Motor Speed, Reaction Time and Cognitive Efficiency Index scores, including less ability to benefit from practice over time; (ii) symptom reports of severe Numbness/Tingling, Balance problems and Dizziness. Deleterious cognitive outcome was exacerbated by longer lifetime duration of alcohol use, passing out monthly, beer or wine drinking, smoking or substance use.

Conclusion: Deleterious cognitive and symptomatic effects of heavy alcohol use are in evidence for undergraduate university students implicating current and/or cumulative alcohol induced brain impairment. The results of the study are of concern for optimal academic performance of students at an early stage of university level study, who engage in heavier (Harmful/Dependent) drinking patterns, with negative implications for future career attainment.

Keywords: neuropsychological impairment, cognitive functioning, alcohol use, university students, South Africa, AUDIT, ImPACT, practice effects

Declaration

I declare that "**The Relationship Between The Alcohol Use Patterns And Cognitive And Symptomatic Functioning Of Rhodes University Students**" is my own work, that it has not been submitted for any degree or examination in any other university, and that all the sources I have used or quoted have been indicated and acknowledge by complete references.

Tamara Ann Mayson

Signed: ……………………………

May, 2015

Table of Contents

List of Tables

List of Figures

Acknowledgements

I would like to thank the following people:

- Professor Ann Edwards for use of some of her NRF research grant.
- Doctor Victoria Alexander for helping with the ImPACT program and administration.
- Ronald Coombs and the ImPACT team for allowing me to use the ImPACT program pro bono.
- Ms Kathy Wakashe and Ms Vuyokazi Kofi for helping with administration and printing in the Psychology department.
- Wiebke Wilhelm-Welgemoed, Henriette van Zyl and Tinashe T.R. Ndoro for use of their lecture slots and helping to administer the surveys and tests in Phase 1.
- Professor Sarah Radloff for helping me with the statistical analyses.
- My family for making it possible for me to do my Masters, especially since it took longer than I originally thought it would.
- Nicole Laubscher, Daniel Greenslade, Ruth Mhlanga and Anzet du Plessis for keeping me sane and helping with proof reading and making sense of a sometimes tired mind.

Chapter 1: Literature Review

Section 1: Introduction

Alcohol use is a worldwide phenomenon whose prevalence in this day and age is common, especially amongst young adults (Ham & Hope, 2003). Those at university are perceived to be consuming alcohol excessively, which has adverse consequences for the drinker as well as other people and the university as an institution (Rickwood, George, Parker, & Mikhailovich, 2011). In 2012, alcohol use was linked to 3.3 million deaths in the world, which was approximately 5.9% of all deaths worldwide (World Health Organisation, 2014). Harmful use of alcohol is one of the top five risk factors for death, disease and disability in the world. Disability may be caused by diseases or by accidents linked with alcohol use. Harmful use can lead to diseases such as liver cirrhosis, cancers and brain disorders, among others. It has particularly acute, as well as chronic, effects in the brain as it can cross through the blood brain barrier (Rapoport, 1976). This makes it especially dangerous in the university student population as university students rely on their cognitive functioning to thrive and better themselves. This is an important area, specifically in the university context in which further research is required.

The following literature review will cover many aspects of alcohol use and cognitive functioning. It will start with some relevant information regarding young adult development and alcohol use, followed by a definition of alcohol and a discussion of the various definitions of alcohol misuse. Then the prevalence rates of alcohol use are reviewed, in general populations and amongst students, internationally and in South Africa. The reasons why university students use alcohol, sometimes to excess, will be examined. The effects and consequences of alcohol considered will be both social and physical and in the short term as well as the long term. The factors affecting alcohol-related cognitive damage will be discussed. Various types of cognitive functioning assessment will then be reviewed, followed by a discussion on the tests used in this research. A rationale will subsequently be given as to why this study was undertaken.

Section 2: Young Adult Development

In order to fully appreciate the consequences of alcohol use in students, one needs to start with a discussion of young adult development. This section will cover a basis of information on the biological development of young adults. Young adulthood is a time for many cognitive, biological and social changes. There is an abundance of research on the subject of development and its many aspects, such as research by Arnett (2001), Berger (2001), Birch (1997), Demetriou and van Lieshout (1998), Gogtay et al. (2004), Louw, van Ede and Louw (1998), and Sowell, Thompson and Toga (2004). While there are very distinct domains of development, it is important to note that these influence each other; for example, the biological development of a person's brain will influence their cognitive development and vice versa (Pally, 2000). It was previously thought that the brain stopped developing in infancy (Blakemore & Choudhury, 2006), however studies such as those by Giedd (2008) and Gogtay et al. (2004) show that during adolescence and early adulthood the brain continues its development and refinement. This section will focus on young adult development, between the ages of 18 and 25, and specifically the cognitive and neurological development that takes place during this time.

Pally (2000) states that brain development is in response to the genetic makeup of a person as well as the environment and the circumstances to which the person is exposed. New experiences bring about growth in various areas, and growth in various areas brings about a new way to experience and understand one's world. Each mental activity is the result of interconnecting neurons firing in a controlled and organised fashion to produce a result, such as storing a new memory or focusing one's attention. These mental activities are known as cognitive functions. When a specific brain region develops, so do the cognitive functions that are governed by that region; for example the executive functions, such as reasoning, develop slowly with the frontal lobe sections that produce them (Pally, 2000). Frontal lobe development peaks in puberty but still continues to develop into the young adult years and only reaches full maturity by the age of 25 (Giedd et al., 1999; Gogtay et al., 2004).

As the brain is still developing into young adulthood and alcohol use is common in young adulthood (Ham & Hope, 2003), it follows then that alcohol may be an influencing factor on how it develops (Brown et al., 2009; Pally, 2000; Sowell et al., 2004; Zeigler et al., 2005). Research has shown that the hippocampus and frontal lobe are the areas that are most likely to be affected by alcohol use (Brown et al., 2009). The hippocampus is the area involved with learning and storing memories. It myelinates until the second decade of life and new neurons continue to grow into adulthood (Pally, 2000; Sowell et al., 2004). The frontal lobe area is used for executive functioning such as attention, inhibition and cognitive control (Casey, Giedd, & Thomas, 2000). The prefrontal cortex, part of the frontal lobe, begins myelination at approximately three months and continues into adulthood (Giedd et al., 1999; Gogtay et al., 2004). Even though research has shown that the hippocampus and frontal lobe areas are most likely to be affected, alcohol has the potential to affect the whole brain, as will be discussed in the following section.

<u>Section 3: Alcohol as a substance</u>

As alcohol may have an effect on the development and functioning of the brain, alcohol as a substance will be briefly introduced. The addictive properties of alcohol will be reviewed along with the basic pathways that alcohol takes through the body.

There are many types of substances known as alcohols and most of these are deadly if ingested in even small doses, such as methanol for example (Fort, 1973; Fox & Cameron, 1970; Royal College of Psychiatrists, 1979). Ethyl alcohol or Ethanol is rarely deadly, except in large doses. It is a colourless, flammable liquid and has a weak but distinctive smell. It is soluble in both lipids and water (Zeigler et al., 2005). Ethanol is the only type of alcohol that is used in alcoholic beverages as it is the least toxic of all the alcohols. It is produced by fermenting certain grains (such as barley and corn), fruit (such as grapes and apples) and other carbohydrates (Fort, 1973; Fox & Cameron, 1970; Royal College of Psychiatrists, 1979). The type of alcoholic beverage depends on the production and ingredients; for example, wine is made from crushed grapes and grape juice, whilst beer is made from barley. Much of the research places alcoholic beverages into five main categories: Beers and Ciders, Table Wines, Dessert or Cocktail Wines, Distilled Spirits, and Liqueurs (Fort, 1973; Fox & Cameron, 1970; Royal College of Psychiatrists, 1979). The types of alcoholic beverages have differing alcohol concentrations which make them stronger or weaker. The higher the concentration of alcohol, the faster it is absorbed in the body and the more it affects the body. Although alcohol is absorbed into the blood stream fairly quickly, it is metabolised fairly slowly, thus the alcohol stays in the system for a long time.

The absorption and metabolism of alcohol is affected by a number of factors, including the person's body weight, alcohol dehydrogenase level and the amount of food in the stomach (Fort, 1973; Fox & Cameron, 1970; Royal College of Psychiatrists, 1979). Alcohol dehydrogenase is produced by the body to help metabolise the alcohol. In Parlesak, Billinger, Bode and Bode's (2002) investigation, lower levels of alcohol dehydrogenase were found when the Blood Alcohol Concentration (BAC) was higher. The reason for this is that alcohol dehydrogenase is used to metabolise the alcohol, thus there is a lower level of alcohol dehydrogenase in the body. Parlesak et al. (2002) also suggested that the level of alcohol dehydrogenase is influenced by gender and age. The findings indicated that young females between 20 and 40 years have lower alcohol dehydrogenase levels than young males.

However, in the older age groups, between 41 and 60 years, the reverse was found, that is, that older males had lower levels than the females. The researchers stated that the age differences could be due to certain factors decreasing the gastric alcohol dehydrogenase levels over time, such as diseases, genetics, drugs or medicine. A literature review by Ramchandani, Bosron and Li (2001) suggested that the gender difference in the metabolism of alcohol could be due to the variation in the proportion of water between the genders. Females have lower proportions of water in their bodies and thus achieve greater BAC levels when consuming the same amount of alcohol as males.

The BAC indicates the grams of alcohol per 100 millilitres of blood; for example, a BAC of 0.01 is equivalent to 0.01 grams of alcohol in 100 millilitres of blood (Fort, 1973). The higher the BAC, the more the person is affected, both physically and cognitively (Fort, 1973; Fox & Cameron, 1970; Royal College of Psychiatrists, 1979). People are affected cognitively because alcohol is one of a select few substances that crosses from the blood stream into the brain, through the blood brain barrier (Rapoport, 1976). The blood brain barrier is made up of endothelial cells, packed close together, that border most of the blood capillaries in the brain. It separates the circulating blood from the cerebrospinal fluid in the brain and restricts the flow of substances between the brain and blood so as to protect the brain from most substances that could harm it. The cells of the barrier let some substances through, such as those with small molecules and those which are soluble in water. Other substances can go through, but they either need the assistance of a protein molecule, or they get through with the expenditure of extra energy. Alcohol can cross the barrier on its own because it is a small, water-soluble molecule. Since it has this ability, it can affect the brain and its chemistry directly.

Alcohol is an addictive substance which is legal to consume over the drinking age of 18 in South Africa (World Health Organisation, 2014). This makes it very popular and easy to get. As alcohol is an addictive and harmful substance when used excessively, it can give rise to many problems and disorders, such as those described in the section below.

Section 4: Alcohol use and misuse around the world

4.1 Diagnostic Criteria of Alcohol use and Misuse

Both the World Health Organisation's International Classification of Diseases (ICD) version ten (ICD-10) (1992) and the Diagnostic and Statistical Manual for Mental Disorders (DSM) fourth edition text revision (DSM-IV-TR) (American Psychiatric Press, 2000) have sections devoted to disorders that come about due to the use of substances that affect the mind, including alcohol. Acute intoxication, or substance intoxication is a reversible and transient condition resulting from alcohol (or other substance) ingestion that produces disturbances in physical and cognitive functioning, such as disinhibition and lack of coordination (American Psychiatric Press, 2000; World Health Organisation, 1992). This can be complicated by injury, delirium, coma, perceptual disturbances and convulsions.

Harmful use is a "pattern of psychoactive substance use that is causing damage to health" (World Health Organisation 1992, p. 74). This damage can be neurological as well as physiological. According to the DSM-IV-TR, alcohol abuse, similar to the ICD-10's definition of harmful use, is a maladaptive pattern of alcohol use that occurs in a twelve month period (American Psychiatric Press, 2000). This pattern leads to clinically significant impairment or distress by resulting in a failure to fulfil certain obligations, such as at work, school or home, or by causing legal issues that relate to the alcohol use. It also leads to alcohol use in dangerous situations, for instance drinking and driving. This pattern includes continued alcohol use regardless of problems caused by the alcohol use.

Dependence syndrome is a collection of symptoms, both mental and physical, that indicate that the individual regards the substance as a much higher priority in their life than previously (American Psychiatric Press, 2000; World Health Organisation, 1992). The substance now takes precedence over other behaviours and objects that were once highly valued. The DSM-IV-TR diagnostic guidelines required for this diagnosis include at least three of the following criteria occurring in the previous 12 months: unintentional extended or excessive use, considerable time spent on activities related to use, withdrawal when the substance is not taken, tolerance when taking the substance, neglect of previously pleasurable activities and persistent use or unsuccessful efforts to decrease use, even though there are adverse repercussions as a direct consequence of taking the substance (American Psychiatric Press, 2000).

The DSM version 5 (DSM-5) was recently released with some changes in how substance use disorders are classified (American Psychiatric Association, 2013). The DSM-5 still divides the disorders in terms of type of substance and the criteria are largely the same as in the previous version, except that one criterion was removed and another added. The criterion for legal problems was removed while strong compulsion or craving for the substance was included. Where the DSM-IV-TR split the substance disorders into substance intoxication, abuse and dependence discussed above, the DSM-5 groups these disorders together as a single substance-use disorder and classifies this as mild, moderate and severe. The classifications now depend on the number of criteria met; the more criteria met, the more severe the disorder. A diagnosis of substance use disorder is given when a person meets two or more of the listed diagnostic criteria.

Binge drinking is a useful and popular clinical term in alcohol research, even though the DSM-IV-TR, DSM-5 and the ICD-10 do not classify or define it. There are various definitions of binge drinking, sometimes known as heavy episodic drinking. The most widely used definition is that presented by Wechsler and Nelson (2008) where a binge is defined as consuming five or more drinks for males, and four or more drinks for females, on a single occasion within a two week period. This definition is supported and validated by research done by Weingardt et al. (1998). This pattern of drinking is prevalent amongst college-age drinkers and is very problematic, for others as well as the binge drinkers (Wechsler & Nelson, 2008; Weingardt et al., 1998).

4.2 Prevalence

4.2.1 Worldwide and South Africa

According to the World Health Organisation's Global Status Report's 2010 figures (2014), 48% of the World's population aged 15 and older had never consumed alcohol, which has increased from the 2004 figure of 45% (World Health Organisation, 2011a). In 2004, South Africa had a lifetime abstention rate of 65.2%, which was much higher than the World's average of 45% (World Health Organisation, 2011b). However, in the World Health Organisation's 2010 figures, South Africa's lifetime abstention rate had decreased to 42% indicating a significant increase in the country's alcohol consumption prevalence rate (World Health Organisation, 2014). Even though South Africa has an abstention rate similar to the World average, according to the 2010 figures, it has a high prevalence rate of heavy episodic drinking. The report showed that in South Africa, 25.6% of current drinkers (13.9% of females and 31.7% of males) had taken part in heavy episodic drinking in the last year. The same study reported that the World's average heavy episodic drinking figures are 5.7% of current drinking females and 21.5% of current drinking males. This shows that although less than half of the South African population do not consume alcohol, those people that do drink, do so heavily when compared to the World averages.

The World Health Organisation's (2014) report also presented data for the alcohol per capita consumption of 15 year olds and above, in litres of pure alcohol, consumed. The amount for South Africa for both sexes was 11.0 litres for 2010. This amount places South

Africa at the highest in Africa, narrowly beating Gabon (10.9) and Namibia (10.8). It also ranks amongst the highest in the world, on par with New Zealand (10.9), Belgium (11.0) and UK (11.6). Generally the highest alcohol per capita consumption, above 12.5 litres, is found in the developed European regions.

Alcohol use has been studied extensively around the world, with an abundance of research coming from America and Europe, including those mentioned above by the World Health Organisation (2011a, 2011b, 2014). South Africa is not dissimilar in exploring alcohol use and its effects, and a few notable articles have been published in the last few years. For example, Peltzer and Ramlagan (2009), whose meta-analysis explored the alcohol use patterns and prevalence rates from a range of studies in South Africa spanning 12 years, and Parry et al. (2005), who studied adolescent drug use in South Africa between 1997 and 2001. Both publications found similar results as those reported by the World Health Organisation (2014) and reported that approximately 40-45% of men and 16-17% of women had consumed alcohol in the last month. Parry et al. (2005) found that approximately a third of current youth drinkers (between 15 years and 24 years) were binge drinkers. Patrick et al. (2009) also studied drug use in Cape Town, South Africa by conducting an investigation over one year on coloured adolescents and the stages of substance use initiation during that year. The results showed that the sampled adolescents tried alcohol or cigarettes first, then both alcohol and cigarettes, followed by marijuana and then inhalants. A further study was that by van Heerden et al. (2009), which was a part of the South African Stress and Health study. This study included data on the use of addictive drugs including tobacco and alcohol. The data showed that alcohol was the most prevalent of the drugs being used, with 38.7% of the sample mainly using alcohol over the other drugs. Their data also suggested that South Africans preferred alcohol over other drugs, and that alcohol is often used as a "starter" drug before moving on to other addictive drugs like cigarettes, marijuana and illegal drugs.

In a study of adults over the age of 18 years, Clausen, Rossow, Naidoo and Kowal (2009) investigated alcohol use, using questions set by the researchers, across 20 countries in Africa, including South Africa. It was found that 22.9% of South Africans were current alcohol drinkers, 29% of the current drinkers drank hazardously and 47.5% of the current drinkers drank heavily or binge drank. In comparison, Peltzer, Davids and Njuho (2011) studied South Africans over the age of 15 using the Alcohol Use Disorders Identification Test (AUDIT) and found that 27.7% of the sample participants were current drinkers. It was found that 31.5% of the current drinking sample consumed alcohol hazardously. These are similar figures to those found by Clausen et al. (2009), Peltzer and Ramlagan (2009) and Parry et al. (2005). Suliman, Seedat, Williams and Stein (2010) found slightly higher current drinking rates, using the World Health Organisation Composite International Diagnostic Interview, than both Clausen et al. (2009) and Peltzer et al. (2011) with 34% of the over 18 aged sample currently consuming alcohol. The measures used by the numerous studies assessed multiple aspects of alcohol use and the samples, even though covering the whole country, encompassed different age groups, with Clausen et al. (2009) and Suliman et al. (2010) sampling over 18 year olds while Peltzer et al. (2011) sampled over 15 year olds. These factors could account for the differences in results. Bear in mind, too, that the measures also have diverse descriptions of alcohol use, binge drinking and hazardous drinking. Nevertheless, these results, even though they differ, seem very high when compared to the World Health Organisation's (2014) prevalence rate figure for weekly heavy episodic drinking of 16% and confirm what was stated above, that South Africans consume alcohol more heavily when compared with the World averages.

4.2.2 Young Adults and University Students

Generally, studies have shown that alcohol use peaks at a certain age and then decreases (Jackson, Sher, Gotham, & Wood, 2001; Karlamangla, Zhou, Reuben, Greendale, & Moore, 2006; McCambridge, McAlaney, & Rowe, 2011; O'Malley, 2004; Patrick & Schulenberg, 2011). Patrick and Schulenberg (2011) and O'Malley (2004) found that alcohol consumption increased until age 22 and then started to decline. In contrast, Karlamangla et al. (2006) found that alcohol use only started declining after the age of 35 in a longitudinal study covering 21 years. Jackson et al. (2001) also researched changes in patterns of alcohol use over time. Their inquiry followed university students over seven years, starting at age 18 when they entered university. They found that alcohol consumption decreased over the study period. The researchers suggested that this decrease is due to the decrease in drinking to get drunk, rather than more people abstaining. The findings have not been conclusive as there are many things that can influence why alcohol use changes across the age groups, such as socio-economic status, gender and life events (McCambridge et al., 2011).

A review by Ham and Hope (2003) suggests that there is a higher rate of alcohol use by young adults aged 18 to 24 years when compared to other age groups. Blanco et al. (2008) produced similar results when interviewing 19 to 25 year old individuals. Blanco et al. (2008) found that 20.37% of college students had alcohol use disorders in the past year, compared to 16.98% of non-college attending individuals. While this difference between these disorder rates is statistically significant, this ceased to be the case after adjusting for socio-demographic variables, such as gender and race, and socioeconomic variables, such as income. The researchers concluded that alcohol use disorders were common in college-aged individuals as a whole - that is individuals between 18 and 25 years of age - rather than only those individuals who attended college. Similarly, Hingson, Zha and Weitzman (2009) found that there were no significant differences between the proportion of college-attending adults and non-college-attending adults that consume alcohol. Slutske et al. (2004) came to similar conclusions in their sample which consisted of sets of female twins. Slutske et al. (2004) determined that the differences observed between the drinking patterns of college-attending and non-college-attending adults were mostly due to demographic variables, such as family background. The only difference they found to be significant was that college students are more likely to occasionally binge drink when compared to their non-college-attending peers. Hingson et al. (2009) also reported this.

Though differences between the drinking patterns of college-attending young people and those not attending college might not be as large as often thought, problematic drinking on college campuses is considered a public health concern that has attracted a great deal of research. Some notable studies by Gill (2002), Wechsler and Nelson (2008) and Windle (2003) reviewed literature, specifically focusing on college students, spanning over 25 years on a wide range of alcohol related topics such as prevalence rates, binge drinking and reasons for use. Wechsler and Nelson's (2008) research reviewed literature obtained from studies concerning alcohol use across 120 colleges in America over nine years. Using Wechsler and Nelson's (2008) definition of binge drinking (four or more drinks for females, or five or more drinks for males, in one drinking session in the previous 14 days), it was found that 44% of the students in the sample were binge drinkers. Windle (2003) studied numerous national surveys conducted in America and found a similar figure for binge drinking (40.1%) using a slightly differing binge drinking definition to the one used by Wechsler and Nelson (2008). Windle's (2003) definition of binge drinking is drinking five or more drinks in a single drinking session at least once in the previous 30 days. Although Gill (2002) also reviewed college alcohol use research, a discernible pattern for binge drinking could not be found in the results. This was mainly because the studies included by Gill (2002) had contrasting definitions of binge drinking. These studies, along with many others, are difficult to compare because of the multiple definitions of binge drinking and the various countries being studied. However, the definitions used in Wechsler and Nelson’s (2008) research and Windle’s (2003) review are fairly similar and thus the finding, that approximately 40% of the student population binge drink, seems to be a good indication of the rate of university student binge drinking, at least in America.

Apart from using definitions of binge drinking, another way to measure and compare alcohol use is to employ a commonly used alcohol use questionnaire, such as the AUDIT. Rickwood et al. (2011) used the AUDIT to measure alcohol use and its associated harms in a sample of Australian university students. Approximately half of the sample reported abstaining from alcohol use or drinking at Safe levels, according to the AUDIT, while the rest drank at levels considered harmful or hazardous. Miles, Winstock and Strang (2001) used a shortened version of the AUDIT, the five-item AUDIT, as well as the recommended guidelines set by the United Kingdom (UK) government, to measure alcohol use patterns in a sample of UK university students. The researchers found that more than 90% of the sample consumed alcohol regularly, approximately a fifth of the sample consumed alcohol over and above the recommended guidelines set by the UK government and approximately a third scored in the Hazardous range of the AUDIT. The researchers state both the five-item AUDIT and the original AUDIT are acceptable measures of alcohol use in university student settings.

As a whole, South African universities have not been explored as extensively as universities in America or Europe, though there are a number of revealing published studies as well as numerous Master’s research projects, though much of them unpublished. For instance, Abels (2007) conducted a Master’s thesis on risky behaviour contributing to Human Immunodeficiency Virus (HIV) and Acquired Immune Deficiency Syndrome (AIDS). The study focused on students at the University of the Western Cape. The risky behaviours that were taken into account were alcohol and other drug use as well as young age at first sexual encounter, sex for money and cigarette smoking. In the sample of first year students, 44% were current drinkers. Of those current drinkers, 51% had consumed alcohol in the last 30 days of which 57% had binge drank. The analyses did not find any significant results between alcohol use and risky sexual behaviours. However, alcohol use was significantly related to cigarette smoking and substance use.

Kheswa (2006) wrote a thesis on the risk factors for alcohol and drug use of adolescents in Soweto. The mixed design research found that half of the sampled adolescents drank alcohol. They were more inclined to consume alcohol if they lived with step-parents, were male and were older than 17. Those adolescents in the sample that lived with step-parents had more negative experiences than the adolescents who lived in other living arrangements such as with both mother and father or with single parents. It was concluded that the adolescents who had negative self concepts had a greater tendency to consume alcohol and smoke marijuana.

Olwagen (2003) qualitatively explored patterns of substance abuse across generations in families. The researcher considered the factors that can manipulate the way that patterns of alcohol use are transferred across the generations. The sampled adults each had an alcohol abuse problem and a history of parental alcohol use and abuse. It was found that when one or both of the parents abused alcohol, the parents were emotionally and physically absent. This caused a reduced sense of unity and communication in the family which in turn caused a reduced sense of belonging experienced by the child. These patterns in the family influenced how the children grew up. This negative influence showed itself in adulthood where the children of alcoholics had and have a challenging time trying to form and maintain interpersonal relationships. This in turn hampers their abilities to cope with life's stresses and thus leads to coping mechanisms such as alcohol use and abuse.

Mhlongo (2008) studied personality, parental bonding and alcohol use in African students at the University of Limpopo. The Five Factor Model of personality was employed and the personality domains of the model are Neuroticism, Extraversion, Agreeableness, Conscientiousness and Openness-to-Experience. Drinking behaviour was used to split the sample into three groups; 58% were Abstainers, 31% moderate drinkers and 11% heavy drinkers. The researcher found that a high score on the Openness factor of the Five Factor Model of personality was associated with higher levels of alcohol use. The Agreeableness factor was associated with lower levels of alcohol use, with abstainers scoring higher than the heavy drinkers. Conversely to Kheswa (2006) and Olwagen (2003), there was no significant relationship between alcohol use and parenting.

Research published about South African university students includes Smit, Pretorius and Joubert's (2010) inquiry at the University of the Free State. There the students were asked about their views on at-risk drinking behaviour. The researchers found that the students thought that the number of drinks required to be considered at-risk drinking levels were much lower than those set out by the American Department of Health and Human Services. The levels considered at-risk are more than 14 standard drinks for men and seven standard drinks for women over one week, or four standard drinks per day for males and three standard drinks per day for females, at any time in the past year. Despite the students thinking that the at-risk levels were lower than the actual criteria, these students still consumed alcohol at levels considered to be at-risk when compared to American standards. Approximately half of the students (55.3%) met the criteria for drinking at at-risk levels.

Similar research at the University in the Western Cape investigated actual and perceived levels of alcohol use (Steyl & Phillips, 2012). The majority of the students thought that their peers drank more than they did in reality. The students thought that 93.5% of their peers had consumed alcohol in the previous 30 days, where in fact only 54.8% had. Young and de Klerk (2012) also researched these perceptions of drinking norms. The researchers found that Rhodes University students overestimated how much their peers consume alcohol and that these misperceptions were implicated in the participants' own alcohol consumption rates. One of the theories used to explain this overestimation of normative drinking and subsequent drinking behaviour is the Social Norms Theory (Steyl & Phillips, 2012; Young & de Klerk, 2012).

Researchers studied drinking patterns and consequences of student athletes at three university campuses in Gauteng (Surujlal, Nolan, & Ubane, 2012). The results suggested that even though the student athletes drank alcohol only occasionally (between once a month and less than once a week), the volume of alcohol that they drank during those single occasions was quite high (over six units of alcohol). The researchers suggest that sport in university is related to high alcohol consumption and binge drinking, which is supported by research in America (Wechsler and Nelson, 2008). Surujlal et al. (2012) also state that a large percentage of the athletes experience negative consequences as a result of their drinking, such as hangovers and nausea as well as risky behaviours including driving while intoxicated. Rickwood et al. (2011), who investigated physical and social harms experienced by university students in relation to their drinking habits, reported similar findings. This suggests that the severity and range of harms experienced by students increases as their drinking level increases.

Peltzer (2000) administered health behaviour surveys to black university students at the University of the North, which is now called the University of Limpopo. Health behaviours such as alcohol use, exercise, smoking and diet were researched. It was found that 61.8% of the students were non-drinkers, 22.2% consumed alcohol only very occasionally, 10.9% consumed less than one alcoholic drink in the past two weeks and 5.1% consumed more than one alcoholic drink in the past 14 days. Young and de Klerk (2008) at Rhodes

University reported Safe drinking rates of 58.3% for black students in 2008, 48.8% for black students in 2007 and, for the total student sample, in 2008 a Safe drinking level of 49.8% was reported by the AUDIT screening and 48.8% was reported in 2007. This suggests that more students consumed alcohol at safe levels at the University of the North than at Rhodes University. The figures from Rhodes University and University of the North are very different and it could be that the universities themselves have different drinking cultures that promote varied behaviours in the students. Young and de Klerk (2008) found that more black students (58.3%) drank at Safe levels when compared with the white students (43.2%). The difference in drinking behaviours could be because Rhodes University is historically a white university and the black students and other races, are influenced by the drinking culture that was set by the white students (Young & de Klerk, 2008). Another reason why the universities could have different drinking cultures is that, for a variety of geographical, historical and economic reasons, they attract different proportions of socioeconomically privileged and disadvantaged students.

Young and Mayson (2010) looked at prevalence rates of alcohol use at Rhodes University also using the AUDIT. In both Rhodes University studies (Young & de Klerk, 2008; Young & Mayson, 2010), it was found that approximately half of the students reported that they drank at Safe levels and about 30-40% were categorised as hazardous drinkers. Young and de Klerk (2008) found approximately 20% of the students had scores which fell into the Harmful and Dependent drinkers categories, while Young and Mayson (2010) found approximately 13% were Harmful and Dependent drinkers. Young and Mayson (2010) only sampled those students staying in residences, whereas Young and de Klerk (2008) sampled the whole university. The difference in the Harmful and Dependent drinking figures in the two studies could be due to students having more opportunity to consume alcohol when they live outside the residential system than when they stay in a residence with supervision.

Research conducted at universities indicates that alcohol use is high, particularly at Rhodes University. Thus, the need for further research on alcohol use in the university context is emphasised.

4.3 Reasons for and against student drinking

The reasons people consume alcohol have been researched broadly, along with why they do not consume alcohol. Below, follows an exploration of student drinking patterns and the reasons behind them.

4.3.1 Reasons for abstaining

Although there are multiple studies exploring the reasons for alcohol use, there are far fewer studies focusing on the reasons for abstaining from alcohol use. Luhtanen and Crocker (2005) studied the effects of some personality factors and contingencies of self-worth on alcohol use. The researchers found that if the students' self-worth were based on religion, morals/virtues or academic achievement, they were less likely to binge drink and more likely to abstain from consuming alcohol altogether. Slicker (1997) and Mikhailovich, George, Rickwood and Parker (2011) both studied the reasons for abstaining or discontinuing alcohol use. Some similar reasons were found for not drinking including religion, the taste of alcoholic drinks, health reasons (alcohol damaging their health or interfering with medication), financial costs and disapproval of family or friends.

These may be useful in prevention techniques; however social norms theory has provided the most successful prevention techniques recently (Perkins, 2003).

4.3.2 Social Norms Theory

Social norms theory, as previously mentioned, is largely based on research done by Perkins (2003). The theory suggests that students believe that their peers consume alcohol more frequently and more in volume than they actually do (descriptive norms) and that their attitudes towards consuming alcohol are more permissive than they really are (injunctive norms) and that this overestimation of peer norms is implicated in their own alcohol consumption rates (Borsari & Carey, 2003; Perkins, 2003).

Perkins (2003) argues that these normative misperceptions come about on the basis of four reasons. The first is that people tend to remember and talk about only the out of the ordinary occurrences that happen, rather than things that happen frequently, for example, extremely drunken behaviour. These occurrences are then further exaggerated when the story is repeated and as the story is repeated, the occurrence appears more commonplace than it actually is. The second reason is that when people observe these out of the ordinary occurrences, they have nothing to compare them to, thus they assume that this occurrence happens often and is normal for that environment. The third and fourth reasons are that the media and community encourage the exaggerations by advertising alcohol use as more common and glamorous than it actually is. Attention is then given to the one off occurrences, for example the occurrences of extreme drunkenness or the consequences of drunkenness, like drunken driving and arrests. These are seen as newsworthy, whereas the safe and healthy behaviours, that are actually practiced, are not. By concentrating on the problem behaviour, rather than the desired behaviour, the problem behaviour comes to be seen as the norm, which perpetuates the cycle. The reasons that these misperceptions are causing higher alcohol use are that students will consume more alcohol because they feel that they are expected to, they think other students are drinking more and they want to conform to what they think their peers are doing and thinking. Therefore, in perpetuating this behaviour, the students thereby encourage other students, creating a vicious cycle.

This theory has also been researched by Neighbors, Lee, Lewis, Fossos and Larimer, (2007), while interventions to correct inflated social norms have been validated in terms of its use in prevention techniques (for example, Borsari & Carey, 2003). The prevention techniques often include the use of mass media and feedback sessions, for example to change the language used about alcohol use and abuse (de Klerk & Young, 2012) with the aim of rectifying the gap between the perceived norm and actual norm of alcohol abuse and alcohol use levels (Borsari, Murphy, & Barnett, 2007). A Cochrane review by Moreira, Smith and Foxcroft (2009) has also confirmed that efforts to correct inflated alcohol use levels using social norms theory results in reduced consumption. The research showed that web-based feedback provided the best results with individual face to face feedback also helping in reducing the misperceptions. The feedback produced improvements in alcohol related problems, drinking frequency, drinking quantity and binge drinking, as well as a significant reduction in peak BAC. Other sorts of feedback included mailed feedback after alcohol use tests, as well as group face to face feedback, which were generally less effective.

4.3.3 Drinking Motives and Personality

The Drinking Motives model of alcohol use is based on Cox and Klinger's (1988) research. The model that the researchers proposed assumes that the decision to drink or not drink is based on "nonconscious and automatized" emotional and logical processes that involve what the person expects will happen once they have had a drink compared to if they do not drink (Cox & Klinger 1988, p. 171). The model categorised four expectancies with regards to alcohol's effect on affect: a) positive affect will be enhanced, b) positive affect will be reduced, c) negative affect will be enhanced and d) negative affect will be reduced.

Cox and Klinger's (1988) motivational model was used as a basis for Cooper's (1994) drinking motives measure called the Drinking Motives Questionnaire (DMQ). The DMQ categorises reasons for drinking into four classes: Social, Coping, Enhancement and Conformity. The Social class is comprised of items pertaining to drinking to enhance social rewards, the Coping class includes items concerning reducing negative emotions, items associated with increasing well-being are classed in the Enhancement group and those items relating to avoiding rejection are in the Conformity class. Much research has been done using the DMQ, some concerning how the numerous drinking motives influence patterns of alcohol use and the volume of alcohol consumed. For example, Theakston, Stewart, Dawson, Knowlden-Loewen and Lehman (2004) used the DMQ with a test for the Five-Factor Model personality domains along with an alcohol use questionnaire. It was found that some personality traits predicted certain drinking motives and drinking levels. For example, low Emotional Stability and low Extraversion were predictors of Coping motives, which was related to heavy drinking. Williams and Clark (1998) found that drinking to escape (similar to Coping motives) was associated with binge and social drinking and high alcohol consumption. Social motives are associated with moderate drinking, Enhancement with heavy drinking and Coping with drinking disorders (Kuntsche, Knibbe, Gmel & Engels, 2005).This model gives insight into how harmful drinking can be curbed. For example, if students are drinking to cope with stress, universities can introduce stress management techniques in classes.

Some studies have been done linking social motives theory and social norms theory in terms of drinking. For example, Neighbors et al. (2007) found that students driven by social motives are more likely to respond to social norms interventions than those students driven by coping motives. This is expected as the social norms theory primarily focuses on an external cause for drinking, for example drinking to fit in, rather than an internal cause, such as coping with negative emotions and stress.

4.3.4 Coping Models

Schulenberg and Maggs (2002) proposed several coping skill models. The Overload model considers rapidly changing roles within a student's life when they enter university and how they can overload the students' coping strategies and abilities. Some of the role changes that occur during this period include the change from high school student to university student and from adolescent to young adult. The Developmental Mismatch model emphasises an individual's needs and how well those needs are met in their current environment, as well as how those needs change and how the environment changes. When the needs of the individual cannot be matched by the environment or if the student's needs clash, the student's well-being may suffer. The Increased Heterogeneity model suggests that the transitions that an individual goes through when entering university can aggravate a person's adaptive functioning. Well-adjusted individuals with good coping skills will adapt to changing environments while those with poor coping skills, as well as poor social, academic and/or organisational skills, will not adapt well to the changing environments and roles. The Transition Catalyst model considers risk-taking in university and its advantages and disadvantages. With regards to alcohol, risk taking can be a way to make new friends and create a new social circle, however excessive alcohol use may result in academic problems as well as a risk to safety and health. According to the Heightened Vulnerability to Chance Events model, individuals at a stage of transition in their lives are more likely to want to experience new things. This may make them vulnerable to adverse consequences of chance events, such as the negative consequences of alcohol use and abuse.

These models cover the changes and transitions that students go through, as well as how the student copes with these. If the student does not cope well, then they are more likely to indulge in alcohol and may use it to excess. The student however may also use drinking to their advantage, for example drinking to make new friends.

4.3.5 Biological theories

Biological theories on why students consume alcohol at university include theories of human development, specifically brain development. Spear's (2002) research showed that the adolescent brain is still developing and refining itself, which influences the sensitivity of the brain to the effects of alcohol. For example, adults will experience the sedating effects of alcohol at a lower threshold than young people, the result being that the young individual would continue drinking long after the older individual would have stopped. Bava and Tapert's (2010) study showed that the young individual may be more inclined to use alcohol if their brain develops abnormally, specifically when the prefrontal lobe and limbic systems do not develop at the same time and pace. This asynchronous development affects alcohol use; however the individual's environment and behaviour may be factors behind the asynchronous development.

Yet more research has shown that serotonin levels have an effect on alcohol use as well (Fils-Aime et al., 1996; Lovinger, 1999). Lower levels of serotonin in the brain have been found in chronic alcoholic patients. Low serotonin could be both a cause and effect of alcohol use. Research has shown that low serotonin levels are linked to higher impulsivity, which is implicated in excessive drinking behaviours. However, chronic alcohol use also has been shown to lower serotonin levels.

Lyvers (2000) noted in most biological theory papers that it is unclear which comes first, the brain damage, or the alcohol use. It could be that the brain develops in such a way that causes increased use of alcohol, or it could be that excessive alcohol use causes some of the differences seen in the brain. It could also be the case that both the alcohol and the brain's development work together to cause the alcohol misuse and damage to the brain.

4.3.6 University environments

Presley, Meilman and Leichliter (2002) focused more on the university environments rather than the students themselves to explain student drinking. It was suggested that drinking is more prominent in universities that are historically white, have a strong involvement in sport and have a Greek system of residence. The Greek system of residence is particular to American universities. It mainly houses undergraduate students and includes small, generally single-sex residences. The students need to apply to be accepted and members of the house vote on which students can become residents. The researchers also found that university size is associated with the amount of alcohol consumed and students at a smaller university would consume less alcohol than students at a larger institution. This seems not to be the case at Rhodes University, however, where opinion is that the high alcohol use at Rhodes University is caused, at least in part, by its small size and isolation (Young & de Klerk, 2008). Presley et al. (2002) also found that the area surrounding the campus impacts how much alcohol the students consume, for example the alcohol outlet density and the settings where the actual drinking takes place. More alcohol outlets mean greater access to alcohol, so the students consume more alcohol. This could be the reason that alcohol use is high at Rhodes University, as it is surrounded by many alcohol outlets and places to drink (Young & de Klerk, 2008).

Another reason that Young and de Klerk (2008) suggested for the high alcohol use at Rhodes University is that more than half the students live in unsupervised living arrangements, for example in rented housing not owned and supervised by the university. The researchers also suggest that the diversity of the student demographics is linked to drinking rates and patterns on campus. Wechsler and Kuo (2003) researched demographic factors that may have an influential effect on campus drinking. The results suggested that students who did not binge drink in high school were more likely to start binge drinking if their university had fewer senior students and a less ethnically diverse environment; that is less minority (or black) students. It was also found that drinking rates in high-risk students tended to be lower when the student body is diverse in terms of race with higher proportions of females and older students. High risk students in Wechsler and Kuo's (2003) study were white, male and under 21 years old. Young and de Klerk (2008) had findings that mostly supported this. Compared to the other races, fewer white students reported drinking at Safe levels, according to the AUDIT, and more white students drank at Hazardous, Harmful and Dependent levels than the other races. More males than females reported drinking at higher levels. Young and de Klerk (2008) found that the undergraduate and postgraduate students had roughly the same average AUDIT scores in 2007 (8.97 and 8.79 respectively), however, there were far fewer postgraduate students in the study compared to undergraduate students. In 2008, the respective mean scores changed slightly – with the undergraduate mean score of 9.05 and postgraduate mean score of 7.56. Even though these differences were not significant at the 0.01 significance level, the younger students did consume alcohol at more hazardous levels than the more senior students.

Arnett's theory (Colby, Colby, & Raymond, 2009) claims that students perceive the time spent at university as their 'last chance' to live life freely, that is, without restrictions, responsibility and negative consequences. This 'care-free' attitude is often even expected by other students. New students feel that, in order to be accepted by and to 'fit-in' with other students, they need to consume alcohol to the extent that other students do. This would affect those starting at university, rather than those who have been there for a while already.

Another interesting factor related to university alcohol use could be participation in university level sport. Surujlal and colleagues' (2012) Gauteng university student research, mentioned above (see p. 17), found that playing sports in university is related to higher alcohol use when compared to those students who did not play sports. Presley et al. (2002) support this finding in a review of literature. Martens, Dams-O'Connor and Beck (2006) conducted a systematic review and meta-analysis on research regarding sport and alcohol use. It was found that there were similar figures between the athletes and non-athletes in terms of alcohol use prevalence rates, which were between 80% and 87%. The athletes in the sample did have slightly higher prevalence rates but the results were not significant. As was found in Surujlal et al. (2012), Martens et al. (2006) found that although the students had similar prevalence rates, the athletes had heavier drinking patterns than the non-athletes because the athletes consumed more alcohol per drinking day, than the non-athletes consumed. The researchers also reviewed literature covering why there is a relationship between college athletes and drinking. The researchers state that one of the more common explanations is that the participation in college level sport brings a certain amount of pressure to perform which may produce anxiety. The students may then consume alcohol to relieve the pressure and anxiety, similar to the reasons stated above. Another reason is that of peer pressure or influence. The student may feel that in order to fit in with the team, they need to behave as the team behaves. This is related to social norms theory, as the student athlete may perceive their teammates to be drinking much more than they actually do, thus the student athlete drinks more.

4.3.7 Other findings

Other social research on why students consume alcohol includes that by White, Fleming, Kim, Catalano and McMorris (2008) which suggested that university students consume more alcohol than their non-university-attending peers because they have left home and not because they are attending university. It was also suggested that drinking in the final year of high school before going to university is associated with more drinking in university than if the student did not drink in high school.

Humensky (2010) proposed that students consume more alcohol if their parents have a higher level of education and have a higher income, because the students have more money to spend on alcohol. Also pertaining to education, Hayatbakhsh, Najman, Bor, Clavarino and Alati (2011) alternately found that those children who performed worse in school drank more in young adulthood and in adulthood. It was suggested that this could be because both the poor school performance and alcohol use are expressions of socially deviant behaviour. Another reason is that the students consume alcohol to reduce their frustration and anxiety caused by their low marks at school and then this becomes a cycle with alcohol use causing lower marks.

In Peltzer and Ramlagan's (2009) literature review, it was found that two of the included studies reported that lower levels of education were associated with heavier drinking patterns. Similarly, Karlamangla et al. (2006) reported that heavy alcohol use was associated with having less education and a lower income. It was also reported that greater average consumption was related to higher socio-economic status. The researchers suggested that the higher socio-economic individuals consume alcohol more on average but at a more constant rate, while the lower socio-economic individuals drank heavily episodically. Peltzer and colleagues' (2011) research reported that more than 50% of their participants who had higher than a grade 12 education reported being current drinkers. Contrary to Karlamangla et al. (2006), Peltzer et al. (2011) reported that the proportions of current and binge drinkers increased with increased income and additionally found that hazardous and harmful drinkers were equally dispersed across education and income levels. Slutske et al. (2004) followed female twins starting from age 13 to 21 years. It was found that a college education was related to higher frequency of drinking, binge drinking and getting drunk in the past year. It was concluded that the increase in alcohol use was due to a range of demographic variables working together, for example socio-economic status, race, single, living alone and not employed full-time.

All these theories suggest that there are many possible reasons that students consume alcohol during their college attending years and there are many possible factors leading to higher drinking rates. At Rhodes University, for example, it could be that the students are away from home for the first time, trying to make friends, cope with the university work as well as the high density of places to consume alcohol. A contributing factor could also be that students see it as the norm to consume alcohol frequently and to excess because the University is believed, by popular opinion, to be the university with possibly one of the highest drinking rates in South Africa. This excess drinking can lead to many consequences, both positive and negative, which are discussed in the next section.

Section 5: Consequences of Alcohol Use

Alcohol use can lead to many dangers, both to other people as well as the drinker. These effects include social consequences and biological effects at the time of drinking and long after the drinking has stopped.

5.1 Social consequences

The World Health Organisation (2014) stated that harmful alcohol use can be attributed to 3.3 million deaths a year worldwide, which is approximately 5.9% of global deaths. The Global Status Report for 2004 (World Health Organisation, 2011a) stated alcohol use is one of the top five risk factors for disease and death in the world, along with underweight childhood, unsafe sexual practices, unhygienic water and sanitation and high blood pressure. In South Africa, harmful alcohol use is estimated to have cost the country R300 billion in 2009 (World Health Organisation, 2014). These costs include direct costs like health care, police and justice system response costs as well indirect costs due to loss of productivity and reduced quality of life. The costs are experienced by the drinkers themselves, their friends, families and people associated with them, as well as the country as a whole.

Alcohol use dangers were studied by Rickwood et al. (2011) and Park (2004) who specifically looked at the physical and social harms that resulted from alcohol use in university students. McGee and Kypri (2004) similarly researched the reported problems of alcohol use experienced by New Zealand university students approximately 20.5 years old. The students’ alcohol use was measured using the AUDIT while a survey was used for the alcohol-related harms. Similarly, Rickwood et al. (2011) used the AUDIT to measure alcohol use while Park (2004) used questions set by the author. Hingson et al. (2009) examined the trends in mortality and morbidity related to alcohol use in 18 to 24 year old adults, from 1998 to 2005, using data from various National Health sources in America. Hingson and colleagues’ (2009) sample included both those attending and those not attending university. In Rickwood and colleagues’ (2011) sample of Australian university students, 17.7% of non-drinkers and 45% of drinkers had been a passenger in a car being driven by an intoxicated person and 17.2% of the drinkers had driven a car after consuming too much alcohol. Hingson et al. (2009) reported that, in a sample of young adults, 26.5% in 1999 had driven a vehicle when they were under the influence of alcohol. This figure increased to 28.9% in 2005. Rickwood and colleagues’ (2011) figures also show that drunken behaviour is accompanied by unwanted sexual advances, sexual harassment and assault. Hingson et al.

(2009) reported that, in 2001, 12% of the students in the sample had been assaulted by a drunken college student and 2% had reported being sexually assaulted.

In terms of university property, Rickwood et al. (2011) found that 24.4% of the sample had experienced an intoxicated person damaging university property. Park (2004) found that aberrant behaviour or being out of control was more common in females than in males, but males were more likely to be in trouble with the authorities or police. Rickwood et al. (2011) presented that 7.6% of the sample reported losing consciousness due to their own alcohol use, while 37.1% had witnessed someone else lose consciousness due to alcohol use. McGee and Kypri (2004) reported that 42.8% of females and 52.3% of males had reported blackouts due to their alcohol use. A blackout is different to losing consciousness and is where an intoxicated person is unable to recall events that occurred during the intoxicated period (White, 2003). An alcohol-induced memory blackout is mainly due to alcohol's deleterious effects on the hippocampal region (White, 2003) while losing consciousness is largely due to alcohol's excitatory effects on the Gamma-amino Butyric Acid (GABA) inhibitory neurotransmitter system (Barclay et al., 2008).

Park (2004) also investigated positive effects of alcohol use. Positive effects include meeting new friends, meeting romantic partners, relieving stress and having fun. Even though there are some positive effects of alcohol use, especially from the students' point of view, the negative effects outweighed the positive ones, particularly when taken from university administrations' perspectives.

The World Health Organisation (2014) stated that heavy alcohol use can lead to a decrease in productivity, for example in the work place and at university. In Rickwood and colleagues' study (2011), this is shown in the percentages of those who had missed a class (25.6%) or had poor academic performance (11.5%) because of drinking. Park (2004) found that approximately 12-13% of the sample had experienced school problems as a result of drinking. McGee and Kypri (2004) found 23.5% of the female students reported being late for class, 42.9% reported missing a class, 40.4% reported not concentrating and 7% reported not completing assignments. The researchers found that 29% of the males reported being late for class, 47.7% reported missing a class, 40.9% reported not concentrating and 11.1% reported not completing assignments. Aertgeerts and Buntinx (2002) did not include self-reports but rather reports from the university on grades and pass rates. The researchers specifically focussed on alcohol abuse and academic performance in first year university students in Belgium. The alcohol abuse and dependence diagnoses were made using the criteria from the DSM-IV. The results showed that there was no relationship between academic performance and alcohol abuse. Alcohol dependence, however, was related to a 25% increased risk of failing the year.

Alcohol use and misuse at university has been shown to be a national issue, causing many problems for not only the drinkers themselves, but those exposed to drunkenness. It also causes problems for the university as a whole, as well as putting a burden on the country's resources.

5.2 Biological Effects

The main concern of this research is the effect of drinking on university students' brains and subsequent cognitive functioning. It was alluded to earlier (see Section 4.3.5, p. 24) that alcohol use has an effect on the development of the young adult brain (Fils-Aime et al., 1996; Lovinger, 1999; Spear, 2002; Bava & Tapert, 2010). Research has shown that, because the young adult brain is developing, it is susceptible and sensitive to the damaging effects of alcohol (Spear, 2002). This could cause changes in normal brain development (Bava & Tapert, 2010) or serotonin levels (Fils-Aime et al., 1996; Lovinger, 1999). These changes could either be a cause or an effect, or possibly both, of the alcohol use, as pointed out by Lyvers (2000).

This section will deal with the effects that alcohol can have on the body and brain, immediately after consumption and over time.

5.2.1 Immediate Effects

When alcohol is consumed, most of it makes its way to the stomach and small intestine where it is absorbed into the blood stream (Fort, 1973; Fox & Cameron, 1970; McCrady & Epstein, 1999). The metabolism of alcohol takes place very slowly at a rate of about seven grams of alcohol per hour, or approximately one alcoholic beverage per hour, depending on the concentration of the alcoholic beverage. A small percentage of the consumed alcohol (about 2-10%) bypasses the blood stream and is excreted by the lungs and in the urine (Fort, 1973; Fox & Cameron, 1970; McCrady & Epstein, 1999). The alcohol that is excreted by the lungs can be measured using a breathalyser and is used to estimate the BAC (Marshall, Humphreys and Ball, 2010). As such, breathalysers are used in roadside testing to see whether a person is over the legal drinking and driving limit. A BAC of 0.05 grams of alcohol in 100 millilitres of blood is the current legal limit for driving under the influence in South Africa (World Health Organisation, 2014). According to Fort (1973) a BAC of 0.05 grams of alcohol in 100 millilitres of blood will produce mild cognitive deficits and a slightly impaired driving ability.

Once in the bloodstream, the alcohol affects the circulatory system (Fort, 1973). The person experiences the characteristic warm feeling and red face associated with alcohol consumption. This is because most of the blood vessels in the body expand at low doses. The muscular system is then also affected. The muscles of the body are normally in a slightly contracted, tensed state and alcohol has the effect of relaxing them. The muscles then are weaker than they normally are. This is why a person feels weak after drinking too much alcohol. The heart muscle is also weakened at the time of consumption and is less able to contract and circulate blood, thus blood pressure falls slightly with low to moderate alcohol doses (G. Hanson, Venturelli, & Fleckenstein, 2008). There is also a slight increase in heart rate at low to moderate alcohol doses because the heart works harder to circulate blood around the body, thus the pulse increases while the blood pressure drops.

Due to the ease with which alcohol crosses the blood brain barrier, alcohol almost immediately affects cognitive processing (Rapoport, 1976). When the alcohol reaches the brain, the higher brain functions moderated by the pre-frontal cortex, are affected first (Royal College of Psychiatrists, 1979). Many studies have found that even at low doses alcohol affects mood, inhibition and attention span (Friedman, Robinson, & Yelland, 2011; Grattan-Miscio & Vogel-Sprott, 2005; Guillot, Fanning, Bullock, McCloskey, & Berman, 2010; Levenson, 1987; Saults, Cowan, Sher, & Moreno, 2007; Schweizer & Vogel-Sprott, 2008). As the BAC rises, the alcohol then affects more of the brain, such as the areas that control the muscles and voluntary movement, impairing these (Barclay et al., 2008; Fort, 1973). The result is reduced coordination and clumsiness, as well as slurring as the speech muscles are affected. If the BAC rises still, then the hind-brain is affected. This controls all the involuntary actions of the body, such as breathing and circulation. When the BAC gets too high, that is over approximately 0.3 grams of alcohol in 100 millilitres of blood, people often lose consciousness. Death often occurs at BAC levels of between 0.5 and 0.8 grams of alcohol in 100 millilitres of blood. This can occur if the person consumes a large amount of alcohol very quickly.

Goble (2013) conducted a Master’s thesis on the immediate effects of alcohol use on information processing in Rhodes University students. The findings indicated that the time of day of alcohol consumption affected performance significantly. It was concluded that when alcohol was consumed in the morning, the students scored worse on performance tests (for example, memory and motor performance) and experienced bigger changes on physiological tests (for example, increased heart rate and decreased skin temperature) than if the alcohol was consumed in the evening.

Alcohol also affects the receptors and proteins involved in neuronal signalling in the neurotransmitter pathways (Marshall et al., 2010). It mainly interacts with GABA and Glutamate, but also affects the Dopamine, Opioid and Serotonin neurotransmitter systems (Chastain, 2006; Marshall et al., 2010; McCrady & Epstein, 1999; Yudofsky & Hales, 2002). GABA is an inhibitory neurotransmitter that interacts with many other neurotransmitter systems. Glutamate also interacts with many neurotransmitter systems, but it is an excitatory neurotransmitter. Alcohol enhances GABA's inhibitory actions and decreases Glutamate's excitatory ones. This is thought to give alcohol its depressant nature. Alcohol's effects on Glutamate are thought to be the reason for its deleterious effects on memory and learning. As the brain tries to compensate for alcohol's actions on neurotransmitters and receptor sites, so it modifies its structure. These modifications remain for a period of time after the alcohol is gone from the body and the brain functions differently to the way it did before, for at least a short amount of time. For example, as alcohol dulls the effect of Glutamate receptors, the brain tries to compensate by increasing the number of receptors over time. When there is no alcohol in the system, the brain still has this excess of Glutamate receptors. This modification in the brain is one of the reasons that people sometimes experience hallucinations and seizures during alcohol withdrawal (De Witte, Pinto, Ansseau, & Verbanck, 2003).

5.2.2 Hangovers and Withdrawal

As discussed, alcohol changes the brain, both chemically and physically, so that when the alcohol has been metabolised fully and there is no alcohol left in the system, the brain functions differently from when it was under the influence (Chastain, 2006; Marshall et al., 2010; McCrady & Epstein, 1999; Yudofsky & Hales, 2002). This is usually temporary while the brain adjusts. This difference in functioning causes cognitive and physical symptoms, including any combination of the following: headaches, nausea, dizziness, lack of concentration, decreased attention and fatigue (Swift & Davidson, 1998; Wiese, Shlipak, & Browner, 2000). These are the symptoms of withdrawal and hangover. The difference between withdrawal and a hangover is that a hangover can occur after a single bout of drinking and withdrawal occurs after multiple drinking occasions followed by abstinence. Hangovers may last for several hours, whereas withdrawal may last for several days. Both withdrawal and hangovers can include the above symptoms, but withdrawal symptoms are much more severe than those experienced during a hangover and can be complicated by hallucinations, seizures and, in severe cases, death.

Research has been conducted to investigate how alcohol hangovers affect cognitive functioning (Anderson & Dawson, 1999; Finnigan, Hammersley, & Cooper, 1998; Ilan & Gevins, 2001; McKinney & Coyle, 2006). Finnigan et al. (1998) studied 40 social drinkers (aged between 18 and 41 years), comparing cognitive abilities such as motor ability, attention and memory, when hungover and when not. It was found that alcohol affected the drinkers' feelings about their abilities but not the abilities themselves. This is a singular finding and is in contrast to other research findings, such as the studies below.

Anderson and Dawson (1999) and McKinney and Coyle (2006) studied student drinkers with a mean age of approximately 21 years (Anderson & Dawson, 1999) and 24 years (McKinney & Coyle, 2006). Anderson and Dawson (1999) used the Symbol modalities test, Paced Auditory Serial Addition Test (PASAT) and Cancellation tests to measure attention, information processing and visual scanning to research South African university students. McKinney and Coyle (2006) used a task battery measuring recall, reaction time and attention among other domains to research American university students. The students from both Anderson and Dawson's (1999) and McKinney and Coyle's (2006) samples were assessed when hungover and when not. Contrary to Finnigan et al. (1998), both Anderson and Dawson (1999) and McKinney and Coyle (2006) found that, even when the students' BAC levels had returned to zero, their attention was impaired 11 to 16 hours after alcohol consumption. Anderson and Dawson (1999) also included a control group to measure any practice effects that may occur. Both the hangover and control groups displayed significant practice effects. However, these effects were significantly greater for the control group. The researchers suggested that the practice effects, along with the main test results, showed that the hangover group had attentional problems.

Hartshorne (2000) conducted Master's research based on Anderson and Dawson's (1999) methodology. Hartshorne's (2000) research was conducted on South African students, like Anderson and Dawson (1999), but using the California Computerised Assessment Package (CALCAP) and driving skill tasks. Hartshorne (2000) found that the students who were hungover performed worse than those who were not hungover in terms of reaction time and driving precision.

Ilan and Gevins (2001) looked at the effects of social drinking, specifically the social consumption of wine, on neurophysiological functioning in eight social drinkers between 23 and 33 years old. The findings indicated that moderate alcohol use had effects on brain function that persisted for hours after the alcohol had left the system. It was suggested that the brain function was slowed because the resources necessary for normal cognitive functioning were being used elsewhere, for example the resources normally used for stimulus processing might be used in making sure the bodily functions were working correctly, such as keeping the heart pumping.

Still other research has been conducted to explore the effects of multiple hangovers and withdrawals on cognitive functioning (Brown, Tapert, Granholm, & Delis, 2000; Loeber et al., 2009; Tapert & Brown, 1999). Loeber et al. (2009) studied alcoholic patients in Germany. The researchers compared cognitive functioning across three groups of participants: a healthy control group and two alcoholic patient groups. The alcoholic patients were split into two groups; one with fewer than two medically supervised detoxifications and the other group with more than two detoxifications. The participants were between the ages of 21 and 65 years and were not addicted to any other substances. The researchers used Intelligence Quotient (IQ) performance (as measured by the Wortschatztest, a German vocabulary test) and memory functions to control for interfering variables. The results showed that both the alcoholic patient groups performed worse than the healthy controls in terms of attention shifting and other executive functions. However, the research found no significant results that supported that the two groups of alcoholic patients differed in terms of memory and frontal lobe functions. Tapert and Brown (1999) studied a younger sample, aged 13 to 19 years, from in-patient substance abuse treatment centres, over four years. It was found that withdrawal symptoms and severity were associated with lower scores on tests for visuospatial functioning and non-verbal processing tasks. Similarly, Brown et al. (2000) showed that the adolescents who experienced more withdrawal symptoms and more severe symptoms scored lower on tests of visual motor integration and visuo-perception. This seems to suggest that the younger the sample, the more seriously they are affected by multiple and severe withdrawals.

5.2.3 Alcohol use consequences over time

Barclay, Barbour, Stewart, Day and Gilvarry (2008), McIntosh and Chick (2004), and Schuckit (2009) performed reviews of alcohol use and the physical effects that excessive alcohol use can cause over time. Excessive alcohol use can cause physical diseases and ailments that involve almost every system in the body, such as the liver, cardiovascular system, respiratory system and the skin amongst others, as well as the brain, which is of interest in this research (Barclay et al., 2008). Excessive alcohol use is also associated with nervous system degeneration which can appear as problems with balance and gait (Barclay et al., 2008; McIntosh & Chick, 2004) as well as dizziness (Barclay et al., 2008) and neuropathy or numbness in the extremities, particularly the legs (Barclay et al., 2008; McIntosh & Chick, 2004; Schuckit, 2009).

The physical consequences of alcohol use are fairly easy to measure and produce easily replicable results. However, the effects that alcohol has on cognitive functioning are not as easy to measure. For this reason and others, the relationship between alcohol use and cognitive functioning over time is highly debated with often contradictory findings. Some studies have found moderate alcohol use had beneficial effects (Britton, Singh-Manoux, & Marmot, 2004; Corley et al., 2011; Elias, Elias, D'Agostino, Silbershatz, & Wolf, 1999; Rodgers et al., 2005; Smith & Fein, 2010; Wright, Elkind, Luo, Paik & Sacco, 2006), while others either find no relationship at all (Leroi, Sheppard, & Lyketsos, 2002; Lobo et al., 2010) or that moderate alcohol use is linked to cognitive deficits and brain damage (K. L. Hanson, Medina, Padula, Tapert, & Brown, 2011; Hermens et al., 2013; Sher, Martin, Wood, & Rutledge, 1997; Sullivan & Pfefferbaum, 2005; Zeigler et al., 2005).

Britton et al. (2004) researched the effects of alcohol consumption in adults, who were 46 to 68 years old at the start of the study. The researchers measured the participants’ alcohol use over approximately 11 years with five testing sessions and tested the participants cognitive functioning at the end of the study. The researchers used a word list test, mathematical reasoning tasks, vocabulary tests and verbal fluency tests to assess the participants’ short term memory, reasoning abilities and verbal abilities. The participants’ alcohol use was estimated using questions devised by the researchers. When comparing average alcohol use over the duration of the study and cognitive functioning, the results showed that those participants who drank alcohol in the previous week scored better on all the cognitive tests compared to those adults who abstained. In trying to explain this beneficial relationship, some confounding factors were mentioned that could influence the alcohol use and cognitive functioning relationship, such as mental and physical health and social position. It was also mentioned that alcohol use is related to lower risks of cardiovascular diseases, which may themselves be related to poor cognitive functioning. However, the researchers stated that those participants with a history of cardiovascular disease were excluded, thereby reducing the possible effects that the disease could have as an influential factor.

Elias et al. (1999) and Corley et al. (2011) also discovered that alcohol use is associated with better scores on cognitive tests. Elias et al. (1999) studied a sample of 55 to 88 year olds while Corley et al. (2011) included a sample of adults of approximately 70 years old. Elias et al. (1999) measured participants' alcohol use using questions that were devised by the researchers and Corley et al. (2011) used the Scottish Collaborative Group 165-item Food Frequency Questionnaire to measure alcohol use over the previous two to three months. Corley et al. (2011) used subtests from the Wechsler Adult Intelligence Scale (WAIS) Third Edition (WAIS-III) and a computerised reaction time task to test memory, reaction time, verbal ability and information processing. Corley et al. (2011) controlled for age, IQ scores and socio-economic status in the analyses. Elias et al. (1999) also used subtests from the WAIS, as well as the Wechsler Memory Scale (WMS) and the Multilingual Aphasia Examination. These subtests constitute the Kaplan-Albert Neuropsychological Test battery which measures verbal memory, learning, visual organisation, attention, abstract reasoning and concept formation. Elias et al. (1999) controlled for age, education and occupation in the study and analysed males and females separately. Elias et al. (1999) found that those who consume alcohol scored better on tests for attention and concentration. Corley et al. (2011) found that the women who reported drinking at moderate to substantial levels scored worse on tests for processing speed. Verbal ability in men was affected by alcohol type whereby wine and sherry were related to better scores while beer was related to worse scores. The drinking of spirits was related to better memory scores. Both Elias et al. (1999) and Corley et al. (2011) found that those participants who consumed alcohol scored higher on memory tests and had higher total cognitive composite scores than those who abstained from alcohol.

Rodgers and colleagues (2005) studied three particular age groups; 20-24 years, 40-44 years and 60-64 years. The researchers measured alcohol use by administering the AUDIT and used WMS subtests, Spot-the-word test, California Verbal Learning Test and a Reaction time test (using a special device) to measure verbal ability, working memory, psychomotor speed and episodic memory, as well as reaction time. The Mini-Mental State Examination (MMSE) was used to detect the presence of dementia in the older group (60-64 year olds). The researchers showed that those who abstained and those who drank at hazardous or harmful levels performed worse on cognitive tests than those who drank lightly or at safe levels, regardless of age group. Wright et al. (2006) measured participants' alcohol use and cognitive functioning using a modified version of the Telephone Interview for Cognitive Status over a duration ranging from 6 months to 4.4 years. The participants were all over the age of 40 years. It was observed that drinking more than one drink weekly, and up to two drinks daily, was related to less cognitive decline when compared with drinking more than two drinks daily or less than one drink weekly.

Smith and Fein (2010) compared the cognitive functioning of treatment-naïve alcoholic (TNAD) and non-alcoholic participants between the ages of 19 and 51 years. The TNAD participants were alcoholic adults who had not received any treatment for alcohol use or abuse. The groups were matched for age and gender and had comparable years of education. The subjects were tested using the MicgroCog Assessment of Cognitive Functioning which is a computerised battery that assesses a wide range of cognitive domains. A variety of paper and pencil tests were also administered, for example the Rey-Osterrieth Complex Figure Test, the Trail Making Test (TMT) parts A and B, PASAT and Stroop Colour-Word test. The tests utilised gave scores for attention, verbal processing, abstraction, cognitive flexibility, psychomotor speed, reaction time, spatial processing and various kinds of memory. Initial intelligence was estimated using the National Adult Reading Test (NART). The results showed that the TNAD and non-alcoholic participants scored similarly on all of the tests, except for that of attention, where the TNAD participants scored better.

The above studies seem to suggest that light to moderate alcohol use is associated with some sort of cognitive benefit over time (Britton et al., 2004; Corley et al., 2011; Elias et al., 1999; Rodgers et al., 2005; Smith & Fein, 2010). However the researchers do state that using alcohol to try and achieve these results is not advocated or recommended. Indeed, there could be other reasons that account for the cognitive benefits. For instance, alcohol increases blood flow to the brain, which counteracts the effects that might come about due to low blood flow to the brain and other parts of the body in cardiovascular and cerebrovascular diseases which can occur later in life.

Leroi et al. (2002) and Lobo et al. (2010), in contrast to the above mentioned studies, discerned almost no differences in the cognitive functioning of those who do and do not consume alcohol. Leroi et al. (2002) found that women experienced less cognitive decline over time compared to men, but found no association between alcohol use and cognitive decline. Leroi et al. (2002) included participants between the ages of 18 and over 70 years at the start of the almost 12 year study, with the highest proportions in the 18-30 years (37%) and 31-40 years (21%) age brackets. The participants in the almost five year Lobo et al. (2010) study were over the age of 55 years at baseline. Both studies used the MMSE as a measure of memory change over time (Leroi et al., 2002; Lobo et al., 2010). Psychometrically the MMSE is a relatively gross measure used in the detection of dementia and assesses orientation, attention, recall and language (Lezak, Howieson, & Loring, 2004). As such it is unlikely to detect the subtle differences that are associated with the diffuse brain damage that can be caused by alcohol use.

Most of the research results discussed fall into a ‘J’ or ‘U’ shaped curve in terms of alcohol-related harms, where those who abstain from drinking and those who consume alcohol heavily show more cognitive harm than those who consume alcohol moderately (Elias et al., 1999; Rodgers et al., 2005). This can especially be seen in the studies with older samples, such as Britton et al. (2004), Corley et al. (2011) and Wright et al. (2006) who conducted longitudinal studies and Elias et al. (1999) and Smith and Fein’s (2010) single testing session research.

The ‘J ‘or ‘U’ shaped curve hypothesis is not as prevalent in younger samples: literature reviews covering research from studies of young adults and adolescents observe that alcohol use and cognitive functioning have an inverse relationship (Hermens et al., 2013; Sullivan & Pfefferbaum, 2005; Zeigler et al., 2005). These reviews observed that most of the literature involving imaging of the brain and cognitive tests showed that greater alcohol consumption was related to greater damage to the brain, especially in the frontal lobe region and especially during the adolescent and young adult years. Additionally, Hermens et al. (2013) and Sullivan and Pfefferbaum (2005) suggested that the alcohol-related damage during young adulthood is conceivably reversible if the alcohol abuse is discontinued. This damage was stated to be particularly pertaining to working memory, visuospatial abilities and balance/gait. However, if the alcohol abuse continues or recommences, the damage may become permanent and impede future brain development and treatment, with regards to both alcohol abuse treatment and brain damage treatment.

Montgomery, Fisk, Murphy, Ryland and Hilton (2012) performed a two-part study on how social drinking by a non-clinical sample can affect the brain, particularly executive functioning. The first part of the study took the form of an extensive literature review and meta-analysis of research involving social drinking and executive functioning. This part of the study found no significant results relating social drinking and executive functioning in all of the research included. Part two of the study was experimental research, involving a between-groups design, looking at heavy social drinkers and light social drinkers. The participants were between the ages of 18 and 35 years and were tested on a range of computerised attention switching and executive function tasks and paper and pencil working and semantic memory tasks, mood tests and the AUDIT. The results for part two of the study were discrepant from those of part one. It was found that the heavy social drinkers scored significantly lower than the light social drinkers on the tests of semantic memory, inhibition and attention switching. The researchers stated the differences between the two parts of the research could be attributed to different aspects of executive functioning being tested, the tests being used and cut offs for social drinking.

Other experimental research has also found that alcohol use during the younger years is associated with cognitive functioning deficits. Sher et al. (1997) used subtests from the WAIS revised battery (WAIS-R), the Halstead-Reitan Neuropsychological Test Battery and the WMS to measure visuospatial abilities, attention, motor speed and different kinds of memory and recall. Alcohol use was measured using the Short Michigan Alcoholism Screening Test and the Family History-Research Diagnostic Criteria. The variables of age and education were controlled by using first year undergraduate students who were similarly aged and educated. The researchers found that heavy alcohol use and alcohol use disorders in first year university students (approximately 18 years old) were related to lower scores on visuospatial and attention tests, and slower motor speeds.

K. L. Hanson et al. (2011) also found lower scores on visuospatial tests for those who use alcohol. The researchers conducted a ten year study of drug use on adolescents and young adults who were between 13 and 18 years old at the start of the study. They separated the participants into three groups: no alcohol or substance use disorder, persisting alcohol or substance use disorder and remitted alcohol or substance use disorder. The researchers also used subtests from the WMS, as well as from the Wechsler Intelligence Scale for Children (WISC) revised edition, amongst others. Alcohol and drug use was measured using the Customary Drinking and Drug Use Record. Age at the start of the research and years of education at year 10 were used as control variables in the analyses. It was observed that the visuospatial construction performance of the participants with a history of alcohol use disorders declined over the 10 year research period but the performance of those without such a history did not. The research found no significant differences between the alcohol use disorder groups in terms of their performance on visual learning and memory, verbal learning and memory, executive functioning and motor speed. Further analysis showed that alcohol use during the research period predicted worse scores on verbal learning and memory and executive functioning tests. The researchers also studied the effects of withdrawal on cognitive functioning, but did not find any significant results.

In summary, adolescent and young adult research seems to suggest that alcohol specifically affects numerous types of memory and executive functions, as well as inhibition, attention processes, visuospatial abilities and response time. However, not all the research findings are the same. The reasons for this are unclear. It could be that the findings are contradictory because the damage to the brain is diffuse and subtle, and therefore not easy to detect. It could also be that there are other forces at play in the relationship between alcohol use and cognitive functioning. In the next section multiple factors are discussed that may affect the relationship between alcohol use and cognitive functioning.

<u>Section 6: Factors affecting the cognitive functioning and alcohol use relationship</u>

Cognitive Reserve Theory

Cognitive reserve is one theory used to explain why some people recover faster or cope better than others with damage to the brain (Stern, 2002). Stern (2002) suggests that cognitive reserve is the dynamic ability of the brain to use brain networks, other than the ones damaged, to perform the actions that the damaged sections would normally perform. This can be measured by intelligence test scores, levels of education and occupation achievements. The research suggested that physical, intellectual and social leisure activities also contribute to higher cognitive reserve (Scarmeas and Stern, 2003). Stern (2009) stated that there are physical measures of cognitive reserve, such as brain volume and size and number of synapses. These are not easily measured, so levels of intelligence are often used as a predictor or proxy for cognitive reserve, like in Corral, Rodríguez, Amenedo, Sánchez and Díaz's (2006) research. Corral et al. (2006) studied the relationship between cognitive reserve, age and cognitive functioning. This study predicted cognitive reserve by measuring pre-morbid IQ or initial intelligence using the WAIS Vocabulary subtest. Verbal ability, like that measured by the WAIS Vocabulary test, has been shown to be correlated with initial intelligence and is relatively unimpaired by mild cognitive damage, such as that caused by alcohol use (Lezak et al., 2004). Corral et al. (2006) found that performance on the cognitive functioning tests declined with age, but low cognitive reserve at all ages was associated with deficient scores on the cognitive functioning tests. It has been suggested that those study participants who have lower cognitive reserve, as measured by initial intelligence scores, to begin with, show more cognitive decline later, as a result of alcohol use. The term 'initial intelligence' will be used as a proxy for the concept of an individual's level of cognitive reserve from this point.

Wood, Sher and Bartholow (2002) found that those participants who had an alcohol use disorder and who scored lower on visuospatial performance tests at the beginning of the study, showed greater visuospatial deficits at the end of the study, seven years later, when compared to those who did not have an alcohol use disorder. Corley and colleagues (2011) used IQ scores measured at age 11 (using the Moray House Test) and compared them to the ones obtained during testing of the subjects at age 70 years (using the NART). The researchers also tested the subjects using measures for alcohol use and cognitive functioning (see p. 40). It was observed that the subjects who drank moderately and heavily had better cognitive scores than those who abstained or drank at lower levels. However, when the researchers controlled for IQ scores and socio-economic status at age 11, the relationship between alcohol use and cognitive functioning weakened and the only result that remained significant was that of memory performance, which increased as alcohol use increased.

Similarly, Glass et al. (2009) studied the effects of both alcohol and cigarette use on cognitive functioning in adult men over a period of 12 years. The researchers measured IQ levels using a short form of the WAIS-R and alcohol use by administering the Drinking and Drug History Questionnaire. The researchers administered a multitude of tests including the Stroop Colour-Word test, TMT, PASAT, Controlled Oral Word Association Test (COWAT) and a computerised version of the Wisconsin Card Sorting Test. The tests assessed verbal word fluency, set shifting, flexibility, verbal memory, attention, concentration, processing speed, visual motor skills and reaction time. The researchers ran three separate analyses; one where IQ levels were controlled, another where education was controlled and another where neither IQ nor education were controlled. The results showed that smoking was correlated with response speed. Alcohol use and smoking together were related to lower scores for the executive function composite scores. When education was controlled, alcohol use and smoking remained significantly correlated with the executive function composite scores, especially with regards to response speed. The results also showed that alcohol was significantly negatively correlated with many of the cognitive test scores, but not when the IQ was controlled. When IQ was controlled none of the alcohol use relationships remained significant, which suggests that initial intelligence is an influential factor in the alcohol use and cognitive functioning relationship. This can be estimated by previous education length and type, as well as any cognitive or abilities test, or even the presence of learning disorders.

Age and General Health

Along with initial intelligence, age appears to be an important influential factor in the cognitive function and alcohol use relationship (see Section, 5.2.3 p. 38). It seems that the younger the sample, the more noticeable the effects of heavy alcohol use on the brain and cognitive functioning. This is in the acute stages of alcohol use (Spear, 2002) and possibly in the long term (Brown et al., 2000; K. L. Hanson et al., 2011; Sher et al. 1997; Sullivan & Pfefferbaum, 2005; Tapert and Brown, 1999; Zeigler et al., 2005). El-Guebaly (2007) suggested that the reason results in research studies on the relationship between alcohol use and cognitive functioning are often inconsistent is that there are many factors, not just age, that can affect alcohol-related brain damage, such as age of first alcohol use (age of onset), gender and pattern of alcohol use as well as initial intelligence stated above. These are not always adequately controlled. Tapert, Caldwell and Burke (2005) and Zeigler et al. (2005) support this suggestion. Since there are many influential factors, most studies only research and control certain factors; for example Skogen and colleagues (2011) focussed on general health and explored why alcohol use can be related to cognitive gains, rather than deficits. The researchers suggested that those who abstain from drinking may have done so for health concerns, such as interactions with medications taken or disease. These health concerns may be the reason that the abstainers in the research scored poorly on cognitive tests.

Elias et al. (1999) made another suggestion for the higher cognitive scores by drinkers, stating that the positive association could be the result of the beneficial effects of alcohol on psychological health. The researchers state that the psychological well-being of a person influences their cognitive functioning, so that when a person is, for example, less stressed and anxious, they will perform better on the cognitive tests. For example, drinkers may socialise more, have a wider network of friends and thus have a wider support network. This may make them feel better about themselves, thereby increasing their self-esteem, which helps them perform better on tests. Levenson (1987) supports this in research covering the beneficial aspects of alcohol on psychological health and why people may consume alcohol. It was suggested that people may use alcohol because it reduces anxiety and makes people feel good physically, according to research findings. Peele and Brodsky (2000) also support this in a review of the benefits of drinking and found that moderate alcohol use is linked to better moods and mental health. These studies show that general health both before and at the time of testing is an influential factor in the relationship between cognitive functioning and alcohol use. Included in general health are current and past physical health and mental health.

Patterns of alcohol use

Alcohol use related factors, like those stated above by El-Guebaly (2007), include patterns of alcohol use. Thoma et al. (2011) researched alcohol use patterns and their effect on cognitive functioning. The researchers studied an adolescent sample of alcoholic patients in a treatment centre and non-alcoholic controls, between the ages of 12 and 18 years. The researchers used the Wechsler Abbreviated Scale of Intelligence (WASI), TMT (parts A and B), subtests from the WAIS-III, Wisconsin Card Sorting Test (computerised version) and the COWAT, amongst others, to test for verbal reasoning, visuospatial ability, memory, processing speed and attention. Alcohol and substance use was measured using the Structured

Interview for the DSM version Four (DSM-IV). The results showed that more drinks per drinking day and more drinking days were associated with poorer scores on tests of attention and executive functioning.

Parada et al. (2012) found similar results in a sample of university students aged between 18 and 20 years. The Galician version of the AUDIT was used to measure alcohol use. The cognitive domains tested were different types of memory, planning, cognitive flexibility and concept formation. The researchers measured these domains by using subtests from the WMS version Three (WMS-III) and the Behavioural Assessment of Dysexecutive Syndrome, among others. It was found that those students who binge drank scored lower on tests of memory and executive functioning, which is a product of the prefrontal cortex. As has been discussed before, the prefrontal cortex is still developing in young adulthood (Giedd, 2008; Gogtay et al., 2004) (see p. 3) and, as such, is susceptible to alcohol-related damage (Brown et al., 2009; Pally, 2000; Sowell et al., 2004) (see p. 4). This is supported by research by Bednarski et al. (2012) and Squeglia, Schweinsburg, Pulido and Tapert (2011), who studied the brain images of young adults and adolescents during cognitive tests. Bednarski et al. (2012) studied 18 to 34 year olds using a computerised Stop Signal task to measure reaction time during functional Magnetic Resonance Imaging (fMRI) and used the AUDIT for alcohol use measurements. Squeglia et al. (2011) likewise used an fMRI and studied the brains of adolescents between the ages of 16 and 19 years during a multitude of cognitive tests assessing spatial abilities, attention, memory and executive functions. Squeglia et al. (2011) measured average alcohol use with the Customary Drinking and Drug Use Record and Timeline Follow-back questionnaires. Both studies found that the participants who drank more heavily had differential activation in certain sectors of the brain, such as in the frontal and temporal lobes. This differential activation can show up in poorer scores on tests for cognitive functioning.

Type of drink

Related to alcohol drinking patterns, is the type of alcohol most often consumed. Mortensen, Jensen, Sanders and Reinisch (2001) performed research on different types of alcohol and the relationships they may have with many factors, such as socio-economic factors, personality and cognitive functioning. The researchers used the WAIS battery to assess a wide array of cognitive functions and the Alcohol Usage Questionnaire to gauge alcohol patterns and preferences. The sample consisted of Danish adults between 29 and 34 years old. It was found that the drinking of wine was related to higher scores on cognitive functioning measures as well as higher parental educational level, high socio-economic status, sensation seeking (among men only) and lower neuroticism. On the other hand, it was found that a preference for beer was related to almost the opposite of all those factors, with lower scores on cognitive tests, lower parental education level and so on. This could be an interesting finding considering that wine has been researched and found to be a healthier form of alcohol with many health benefits (though, overall, still more harms than benefits when consumed to excess), such as having protective effects against heart problems, inhibition of cancerous cell growth and greater antioxidant activity (de Lorimier, 2000; Peregrin, 2005). These protective effects are said to be more noticeable with red wine when compared to white wine. According to de Lorimier (2000) and Peregrin (2005), these protective effects are due to the phenols found in the wine. However, Mortensen et al. (2001) suggest that the benefits of wine may be due to the individual's better psychological functioning, rather than the individual's psychological functioning being better because of the wine. Additionally, the researchers suggested that high social status may be a contributing supportive factor, as social status has been shown to be an influential factor in the alcohol use and cognitive functioning relationship (Karlamangla et al., 2006; Peltzer et al., 2011; Slutske et al., 2004) (see p. 28).

Age of onset

Fils-Aime et al. (1996) (see p. 24) focussed on the effects that the age of onset of alcohol use (before or after 25 years old) and parental alcohol use may have on the relationship between alcohol use and cognitive functioning. The researchers determined that those participants who had started drinking earlier in life, before the age of 25, had a more severe course of alcoholism and lower concentrations of serotonin in their brains than those who started drinking later in life. The participants with alcoholic parents were also found to have weaker concentrations of serotonin in their brains, as well as weaker concentrations of dopamine and tryptophan. This could give a reason as to why some people present with more cognitive deficits than others.

The research that Hermens et al. (2013) reviewed suggested that cognitive deficits and neurological damage may be greater in those with a younger age of onset of alcohol use disorders. Tapert et al. (2005) also reviewed research that found that an earlier onset of alcohol use disorder was related to a smaller hippocampal region volume. The researchers also reviewed literature that said that those participants who had longer durations of alcohol use disorders also had smaller hippocampal regions. Tapert et al. (2005) suggested that smaller hippocampal regions are associated with impairments in the memory domains. This could be a contributing factor in the memory deficits associated with alcohol abuse. This suggests a strong link between the durations of alcohol use, and alcohol use disorders, and cognitive deficits.

Duration of drinking

The duration of drinking is an important factor which is why longitudinal and prospective methodologies have been used to describe the relationship between alcohol use and cognitive functioning over time. In serious cases, the drinker can develop Wernicke-

Korsakoff's syndrome which is a severe consequence of excessive and prolonged alcohol use in conjunction with a deficiency of thiamine. The deficiency in thiamine can be brought on by malnutrition, genetic defect or liver disease (Kopelman, Thomson, Guerrini, & Marshall, 2009; Lezak et al., 2004; Royal College of Psychiatrists, 1979; Zubaran, Fernandes, & Rodnight, 1997). The syndrome consists of abrupt and severe memory impairments and is accompanied by paralysis of eye movements, unsteady and unbalanced gait, mental confusion, psychosis and peripheral neuropathy, which is damage to the nerves in the hands and feet. If the syndrome is caught early enough and the patient is treated with high doses of thiamine, certain symptoms may improve, such as those of the visual and gait problems. However, according to research, not all patients suffering from Wernicke-Korsakoff's syndrome will recover completely, and will require custodial care. The recovery outcome is separated into quarters, with 25% not recovering at all, 25% recovering slightly, 25% recovering significantly and 25% recovering completely (McIntosh & Chick, 2004).

In less severe cases, and especially with drinking patterns that are not as severe or long lasting, cognitive impairments due to alcohol use can still be seen over time. Tapert and Brown (1999) and Tapert, Granholm, Leedy and Brown (2002) studied the cognitive effects of alcohol use and withdrawal over a period of time, between 4 and 8 years, so that any changes could be tracked. The participants were between 13 and 19 years old at the start of the study. Tapert and Brown (1999) reported the results after four years and four testing sessions and Tapert et al. (2002) reported the results after eight years and seven testing sessions. Tapert et al. (2002) included a healthy control group to provide comparisons. The groups were matched for age, gender, socioeconomic status, years of education and family history of substance use. Alcohol use was measured using the Customary Drinking and Drug Use Record. The researchers used subtests from the WISC Revised Edition (WISC-R) and the WMS for the younger participants and the WAIS-R and WMS for the older participants and as the younger participants got older. Various tests were changed during the eight year period but the tests all gave an overall impression of the participants' cognitive functioning. The researchers discovered that high alcohol use during the eight year period predicted poorer attention and mental manipulation of information at the last follow-up test. Additionally, both studies found that more withdrawal symptoms resulted in poorer visuospatial and attention scores. Withdrawals and hangovers have been discussed (see Section 5.2.2, p. 35) and are also suggested as influential factors in the relationship between alcohol use and cognitive functioning.

Even though alcohol has been shown to cause some damage in the brain, this damage can often be reversed if the brain is given time to recover. Kopera and colleagues' (2012) tested abstinent alcoholic male patients (mean age 44 years) and healthy controls (mean age 40 years) using tests from the Cambridge Neuropsychological Test Automated Battery (CANTAB). Firstly, the researchers found that the abstinent alcoholic participants scored lower than the healthy controls on cognitive tests, such as working memory, attention shifting and reaction time tests. Secondly, those patients who had been abstinent for more than a year showed less impairment in attentional control and spatial working memory, when compared to those patients who had been abstinent for less than a year. Rosenbloom, Pfefferbaum and Sullivan (2004) tested 43 alcoholic adult abstinent women (28-63 years) and 47 healthy control women (20-85 years) using the NART, distracter tests (short-term memory), Dementia Rating Scale memory subscale (memory status), WAIS Digit Symbol subtest (visuospatial ability) and walk-a-line ataxia battery (gait and balance). The participants were retested after one year and again after four years. Fourteen alcoholic participants and fourteen healthy controls completed the testing at the four year interval. The two groups were not significantly different in terms of education or handedness, however the control participants were slightly older, had higher initial intelligence (NART) scores and lower rates of depression than the alcoholic participants. Those alcoholic participants who had maintained their sobriety by year-four had returned to normal levels of memory and psychomotor speed. However, the participants standing balance remained impaired. In another study, even after as little as three weeks of abstinence, improvement in cognitive functioning was found: Manning et al. (2008) studied 30 alcohol dependent adult males with a mean age of 44 years. The NART, CANTAB tests and paper and pencil tests (WMS-III subtests, Motor Screening Test, Pattern Recognition Memory Test and others) were completed. There were significant improvements in performance on tests of memory and verbal skills. However, skills relating to executive functioning, such as mental flexibility and planning remained impaired.

Substance use

Alcohol is not the only substance that has been shown to affect cognitive functioning, as Glass and colleagues' (2009) results suggested above (see p. 47). The results suggested that cigarette smoking, alone and together with alcohol, is related to cognitive deficits. Marijuana use was linked to poorer memory scores in Thoma and colleagues' (2011) adolescent sample. Tapert and Brown (1999) also found that substance use was related to poor attention scores (see p. 38). These substances should also be taken into account as their consumption along with, or separate from, alcohol could cause some of the cognitive deficits seen in research, thus influencing the results.

Gender and Race Factors

Besides factors related directly to alcohol and substance use, demographic variables are of interest, specifically gender and race. Squeglia et al. (2011) suggested that adolescent females may be more sensitive to the neurotoxic action of alcohol and male brains may be more durable in terms of alcohol's damaging effects. Leroi et al. (2002) found, in a sample of older adults, that there was less cognitive decline over time in the females in the research, but not so for males. Smith and Fein (2010) (see p. 41) on the other hand found no gender effect differences between the two groups of participants. Mann and colleagues (2005) suggested that the combination of gender and duration of the alcohol use could contribute to the damaging effects of alcohol. The researchers studied brain atrophy and alcohol use in 158 participants. There were 42 alcoholic females, 34 healthy females, 34 alcoholic males and 48 healthy males. The researchers included initial intelligence (measured by the Mehrfachwahl-Wortschatz-Intelligenztest, a German vocabulary test) and weight as control variables in the analyses. The researchers found that the females drank slightly less than the males, but this effect was negated when the researchers adjusted the analyses for the difference in weight, which affects how much alcohol is absorbed. Even though females drank less than males, both genders absorbed similar amounts of alcohol over time. Both the female and male participants' brains atrophied to the same extent, but the significant finding that was discovered was that the females had been alcoholics for a much shorter duration than the males. This suggested to the researchers that females have a faster onset of brain damage in response to alcohol dependence when compared to males.

Unlike gender, race is not often included in the analyses of studies. More often than not, it was found that race was controlled for in the analyses but not studied as a contributing factor. Very little research was found to include race as a factor in the relationship between cognitive functioning and alcohol use (Castaneda & Galanter, 1988; Mulia, Ye, Greenfield, & Zemore, 2009; Pedersen, & McCarthy, 2013). Pedersen and McCarthy (2013) studied the differences in immediate responses to alcohol between African Americans and European Americans and found that African Americans showed stronger responses to the alcohol administered. Mulia et al. (2009) mainly studied the differences between the races in the social effects of drinking and included dependence symptoms as well. The analyses found that the African American participants were more likely to report social consequences as a result of their drinking and reported more alcohol dependence symptoms, when compared to white participants. Castaneda and Galanter's (1988) research was the only study found to focus on racial differences with regard to alcohol-related cognitive impairment. Hispanic, black and white participants were included in Castaneda and Galanter's (1988) study of detoxifying inpatients and measured cognitive functioning using the Blessed Orientation-Memory-Concentration test and a Computerised Tomography (CT) scan. While it was found that the Hispanic participants recorded the most cognitive impairment, it was also found that the Hispanics were the participants with the highest daily drinking rates and most severe drinking patterns. The white participants recorded the least cognitive impairment and alcohol use out of the three groups. The researchers suggested that the differences in cognitive impairment between the races may not be a product of race itself, but rather, as a result of the drinking practices observed by the individuals, such as the drinking patterns mentioned above (see p. 49), that is, the drinking patterns are racialised, as suggested by Young and Mayson (2010).

Family History

The relationship between cognitive functioning and alcohol use has also been shown to be moderated by family history of alcohol use (Fils-Aime et al., 1996) (see p. 52). The researchers found that those with a positive family history of alcohol abuse had much lower concentrations of tryptophan, serotonin and dopamine neurotransmitters in their cerebrospinal fluid than those with no family history of alcohol abuse. Moriyama, Muramatsu, Kato, Mimura and Kashima (2006) researched the effects that family alcohol use can have on an individual regarding cognitive recovery during subacute alcohol withdrawal. The sample consisted of 39 alcoholic inpatient male adults with a mean age of approximately 50 years.

The participants completed a computerised TMT (parts A and B), Figure Position, Symbol Digit Modalities, Block Design and Reaction time tests. These tests measured memory, visual abilities and attention. The researchers found that the recovery was better among those patients who had no family history of alcohol abuse, especially regarding memory and attention.

Foetal Alcohol Syndrome (FAS) is related to this area of research as it is a collection of the physical and neurological impairments in the children of mothers who consume alcohol during pregnancy (Poskitt, 1984; Royal College of Psychiatrists, 1979). These cognitive impairments can present as deficits in attention span, intellectual performance and reaction time. It is uncertain as to the level of alcohol use during pregnancy that will cause FAS, but research has shown that moderate to heavy drinking by the mother can cause FAS symptoms (Royal College of Psychiatrists, 1979). Even if a child of alcoholic parents does not have FAS, other abnormalities can be present. For example, research by Herting, Fair and Nagel (2011), and Wetherill et al. (2012) found that adolescent children, who have not started using alcohol, of alcoholic parents had abnormalities in the frontal lobe regions of their brains. The authors state that these abnormalities may be a predictive marker for future alcohol abuse disorders. Family history of alcohol abuse has been found to be a strong predictor of alcohol abuse (Dick & Foroud, 2006) and as such it can be an influential factor in the cognitive functioning and alcohol use relationship (Thoma et al., 2011). This can be in terms of developing alcohol use patterns that could cause damage as well as having damage from birth and making the brain more susceptible to alcohol-related brain damage.

Summary of factors

In summary, there is a diverse range of factors that can form part of the relationship between alcohol use and cognitive functioning. These factors include initial intelligence, current age, general health, pattern of alcohol use, type of alcohol consumed, age of onset, duration of alcohol use, hangovers, substance use, gender, race and family history of alcohol use.

Section 7: Cognitive Functioning Assessment

There are many kinds of tests for cognitive functioning, as can be seen from the previous sections that focus on alcohol use and its effects on cognition. Cognitive functioning tests are the most accessible means to assess changes related to physical alterations in the brain or to assess differences between individuals in terms of cognitive functioning. As Pally (2000) stated, each cognitive function is the result of interconnecting neurons firing at the same time to produce some result and when a specific brain region develops, so do the cognitive functions that are produced by that region. It follows then that alterations in the brain can produce changes in cognitive functioning. These changes can take the form of development, as Pally (2000) stated, or take the form of damage to the brain, such as damage caused by disease, injury or toxin (Lezak et al., 2004). The following section will give an overview of assessments of cognitive functioning changes in relation to some of the various ways in which the brain can be damaged for reasons other than alcohol use. The overview has relevance when considering the possible tests that can be used as well as possible confounding variables in an investigation on the cognitive effects of alcohol use.

7.1 General Cognitive Testing

Some neurodevelopmental disorders can cause differences in cognitive functioning, for example Wolf, Sambataro, Lohr, Steinbrink, Martin and Vasic (2010) studied the brains of dyslexic and control adolescents to see if there were differences in connectivity in the brain during certain tasks. The researchers also wanted to look at the relationship between the connectivity in certain areas of the brain and scores on cognitive tests. The dyslexic and control adolescents were matched for age, gender and IQ. The researchers found that those participants who had dyslexia showed dysfunction in the frontoparietal area of the brain and scored lower on the verbal working memory task when compared to the control adolescents. The researchers suggest that the structural abnormalities seen in dyslexic brains could be associated with altered verbal abilities like phonological processing.

Meyer and Sagvolden (2006) studied another neurodevelopmental disorder, Attention Deficit Hyperactivity Disorder (ADHD), and its relationship with cognitive functioning, specifically motor skills, and ethnicity. It was found that in simple motor tasks the ADHD students did not score significantly differently compared to the control group. However, on more complex tests, the ADHD group performed worse on motor control, accuracy and speed tests. The researchers also found that the older children produced less noticeable differences between those with ADHD and those without. No significant differences were found in the different ethnic groups with regards to motor skills. The findings suggest that ADHD symptoms may reduce with age and are not related to ethnicity. Likewise, Halperin, Trampush, Miller, Marks and Newcorn (2008) found that those participants who had ADHD as a child had lower scores on the neuropsychological battery when compared to the control participants. The researchers state that poor readiness to receive information and a bias to respond too readily may be the core problems of ADHD individuals that affect their performance on cognitive tests. The results suggest that ADHD children may respond too quickly which is why they seem to have lower motor control and accuracy scores.

Stevens, Sidener, Reeve and Sidener (2011) looked at another neurodevelopmental disorder: Autism. The researchers were interested in praise for specific behaviours in the learning and maintenance of tact in autistic children. The Peabody Picture Vocabulary Test (PPVT) version Four (PPVT-4) was used to measure minimum mental age and general mental ability. Glenwright and Agbayewa (2012) also used the PPVT-4 to assess minimum mental age in children with and without high functioning autism spectrum disorder. The PPVT-4 was used to assure that the two groups were matched in terms of verbal ability and initial intelligence. The research was concerned with comparing the understanding of verbal irony between those with an autism spectrum disorder and those without. The original version of the PPVT was used by Guthrie and Seifert (1977) when measuring the verbal ability of good readers and poor readers in first, second and third grade. The researchers looked at sentence completion in children in remedial and regular classes. The PPVT was used in a group setting rather than in an individual setting. The researchers found reliable group-administration results for the verbal intelligence scores.

As distinct from neurodevelopmental disorder, there are mental disorders that may not affect cognitive functioning; for example Twamley, Hami and Stein (2004) performed a study on Post Traumatic Stress Disorder (PTSD) and its effects on cognitive functioning in college students. The researchers found no significant differences in cognitive functioning regarding PTSD symptoms, trauma exposure or any clinical variables, such as anxiety and depression. This is a surprising finding, as high anxiety levels have been found to have a negative effect on working memory, particularly storage and processing capacity (Darke, 1988).

As stated above, initial intelligence and age are two of the many subjects that have been assessed using cognitive testing. Corral et al. (2006), mentioned above (see p. 46), researched the relationship between initial intelligence, aging and cognitive functioning. The sample was split into four age groups: 20 to 35 years, 36 to 50 years, 51 to 65 years and 66 to 80 years. The researchers found that performance on cognitive testing decreased with age. Interestingly, the decreases in performance were not consistent through the age groups or in the different domains tested. It was discovered that the participants' performance on abstract reasoning, attention (using the WAIS Digit span backward test) and visuomotor abilities changed significantly between the 20 to 35 year age group and the 36 to 50 year age group. However, differences were seen more in conceptualisation, attention (using the WAIS Digit span forward test), verbal memory and visual memory between the 36 to 50 year and 51 to 65 year age groups. This suggested that aging affects different domains of functioning at different stages. Additionally, the participants with lower initial intelligence were more likely to have lower scores for memory, global functioning and attention. The researchers stated that those participants in the lower initial intelligence group were six times more likely to have lower scores in the cognitive test battery than those who were in the higher initial intelligence group.

Tlustos et al. (2010) combined the topics of brain injury and initial intelligence. The PPVT-4 was administered along with other tests to ensure that the control and Traumatic Brain Injury (TBI) groups were similar in terms of initial intelligence. The researchers found that the control group with no incidence of TBI did not score significantly differently to the group with incidence of TBI. Dawson, Batchelor, Meares, Chapman and Marosszeky (2007) also combined initial intelligence and brain injury and looked at TBI in adolescents and brain activation in interference tasks. Dawson et al. (2007) aimed to study the relationship between the duration of Post-Traumatic Amnesia (PTA) and initial intelligence. The researchers also studied the factors that could affect initial intelligence, like alcohol and drug use patterns prior to TBI as measured by the AUDIT and a drug use questionnaire, as well as previous neurological disease or damage and emotional state after TBI. The researchers found that those participants with higher initial intelligence and education had shorter PTA durations. Dawson et al. (2007), like others (Corley et al., 2011; Corral et al., 2006; Stern, 2002; Wood et al., 2002), concluded that higher levels of initial intelligence mediated the damage caused by mild brain injury.

The brain can also be influenced due to internal causes such as nutritional deficiencies, for example Blanton, Green and Kretsch (2013) studied the effects of body iron levels on cognitive functioning in non-anaemic undergraduate women. The researchers stated that iron levels in college women were lower than women in the rest of the pre-menopausal female population, thus it was important to investigate the effects of low iron levels on cognitive functioning. The researchers reported that planning speed significantly decreased as body iron decreased. This study demonstrates that any factor, even those not obviously associated with the brain, like iron levels, can affect cognitive functioning.

Gender is another factor that may have an effect on cognitive functioning, for example Weiss, Kemmler, Deisenhammer, Fleischhacker and Delazer (2003) suggested that males and females may perform differently on different kinds of tests. The researchers found that men scored higher on visual-spatial tasks while women scored higher on verbal tasks. The researchers, however, also reported that effect sizes were small and concluded that the similarities between male and female scores were much more substantial than the differences.

Wang, Chan and Deng (2006) hypothesised that the health status, or perceived health status of individuals may have an effect on cognitive functioning, similar to Elias et al. (1999), Levenson (1987) and Skogen et al. (2011) mentioned above (p. 48). Wang et al. (2006) reported on the findings of an investigation on the relationship between postconcussion-like symptoms, in the absence of head injury, to cognitive functioning. The researchers concluded that university students experienced a high rate of postconcussion-like symptoms. These symptoms were not related to scores on the cognitive functioning tests but were related to more depressive symptoms as reported by the Beck Depression Inventory (BDI) responses.

The above research suggests that cognitive functioning deficits may appear in different domains and ages depending on the area damaged and the nature of the damage, including aging as well as pathological conditions such as iron deficiency, learning disability, autism, ADHD and TBI. It also suggests that injury to the brain and cognitive functioning in general, can be influenced by certain factors, such as initial intelligence, severity of damage, time, gender, mental health and age.

7.2 Prospective Cognitive Testing

Multiple cognitive testing sessions can give insights into the changes in cognitive functioning over time; that is the recovery, decline or lack of change. For example, Kersel, Marsh, Havill and Sleigh (2001) evaluated the cognitive recovery over time after severe TBI in adults. The results showed that a greater number of participants showed impairment in verbal learning, complex attention and verbal fluency but not in initial intelligence, simple attention and perceptual abilities. At the follow-up sessions, verbal memory was most likely to be impaired and visual-perception was least likely to be impaired.

Moser and Schatz (2002) explored the enduring effects that TBI, specifically concussion, can have on healthy young adults. The researchers concluded that those participants who had been concussed two or more times in the past had similar cognitive profiles to those who were recently concussed. Similarly, Hessen, Nestvold and Anderson (2007) investigated the enduring effects of head injuries. The results indicated that mild TBI in children could predict poor cognitive performance 23 years later, while similar injuries in adults could not. The researchers concluded that the younger brain is more vulnerable to damage than the adult brain and thus less likely to recover to pre-injury levels.

There are other causes for cognitive change over time, for example Becker et al. (2011) used the computerised cognitive test battery Computer Assessment of Mild Cognitive Impairment (CAMCI) to detect cognitive changes in HIV positive adults and healthy adults over a period of 24 weeks. The researchers stated that the CAMCI was sensitive to mild cognitive impairment, such as that caused by HIV. It was suggested that the CAMCI is useful in detecting defects related to many brain problems, not just those associated with HIV. Also using a computerised cognitive test battery, S. Thomas et al. (2010) used the CogState and the Stroop Colour-Word Test (paper and pencil version) to measure cognitive changes over one year in teenagers. The inquiry focused on practice effects as well as changes due to cell phone use. The researchers found that generally reaction time improved and the number of errors remained the same between the time periods. No significant relationship was found between frequency of cell phone use and cognitive functioning. The researchers concluded that the changes over time may be due to practice effects. Collie, Maruff, Darby and McStephen (2003) specifically looked at practice effects in older adults on a group of cognitive tests over a shorter period than S. Thomas et al. (2010). Collie et al. (2003) found that there was improvement in all cognitive tests and that improvement was most noticeable between the first and second assessments.

Prospective studies can show more detailed change over time than single assessments. These types of studies can also give a wider view of how certain factors affect cognitive functioning over time, rather than a single snapshot of how a person functions at a certain point in time. Practice effects have been shown to present certain analytic problems, but alternate forms of tests and practice examples in cognitive tests can serve to mediate those effects. However, the absence of practice effects when they are expected can also be used diagnostically in prospective studies, such as has been done in studies looking at cognitive decline in Mild Cognitive Impairment, HIV and Huntington's disease (Duff et al. 2007) as well as due to concussive brain injury (Shuttleworth-Edwards, Radloff, Whitefield-Alexander, Smith, & Horsman, 2013).

Duff et al. (2007) researched three different samples to assess the relationship between practice effects and cognitive functioning. The three different samples were of three progressive, neurodegenerative disorders: Mild Cognitive Impairment, HIV and Huntington's disease. The samples were tested on different cognitive tests and over different timelines. All three of the samples showed a relationship between practice effects and cognitive functioning, even after controlling for the baseline testing. Practice effects accounted for 79% of the variance in the memory scores of the Mild Cognitive Impairment group, 83% of the variance in the memory scores in the HIV positive group and 31% of the variance in the Digit symbol task in the Huntington's disease sample. Duff et al. (2007) concluded that practice effects need to be thought of as diagnostic results, rather than as erroneous results as they may hold valuable diagnostic information. This is supported in Duff's (2012) literature review.

Duff, Callister, Dennett and Tometich (2012) explored practice effects and the relationship they may have with demographic variables. The results indicated that the practice effects were not significantly related to demographic variables such as age, education, initial intelligence, gender or psychological wellbeing. The researchers concluded that practice effects may be a unique diagnostic tool because they seem to be independent of many variables that normally affect cognitive testing. The researchers, however, do state that ceiling effects may influence the practice effects relationship. It was stated that if a person performs well on a cognitive test, there is not much room for improvement and therefore practice effects may not be as significant. Therefore, the researchers concluded that practice effects need to be explored more before they are used as definitive diagnostic tools.

In South Africa, Shuttleworth-Edwards, Radloff et al. (2013) researched whether there were long-term visual motor effects of repetitive brain injuries, like those encountered in Rugby. The article combined the data of three studies that used the Immediate Postconcussion Assessment and Cognitive Test (ImPACT) (Horsman, 2010; Shuttleworth-Edwards, Smith, & Radloff, 2008; Whitefield, 2007). The results from the combined analyses revealed that there were significant improvements in the Visual Motor Speed scores of the control participants that were not present in the Rugby players. These were seen as practice effects. The researchers concluded that practice effects can be seen as diagnostic in this area, as those who do not experience them may be suffering from cognitive damage.

7.3 Computerised Cognitive Testing

Computerised cognitive tests, like the ImPACT mentioned above, are gaining popularity lately due to their ease of administration and scoring. Snyder et al. (2011) surveyed 16 cognitive tests, most of which were computerised, to determine whether the tests were valid and useful in researching cognitive functioning and subtle decline in individuals. Most of the tests surveyed were focused on the elderly population and dementia; however the tests are applicable to younger samples and other forms of brain dysfunction. The researchers state that there is an abundance of good computerised cognitive tests out there and that there is no one test that the researchers would recommend over another. The researchers state that the choice of test or tests should be based on the needs of the study.

The many computerised cognitive tests are used to assess a wide variety of conditions that are subject to cognitive status and/or change. For example, the Automated Neuropsychological Assessment Matrices (ANAM) was developed by the Department of Defence over 20 years to measure human cognitive performance (Wilken et al. 2003). Wilken et al. (2003) used the ANAM test battery to assess cognitive functioning in Multiple Sclerosis (MS) patients. The researchers state that computerised tests are more efficient than paper and pencil test for assessing subtle cognitive decline because they have multiple alternate forms, accurately measure reaction time and require less administration time and training. The results indicated that the computerised test, ANAM, performance was significantly related to the performance on the paper and pencil tests. The researchers concluded that computerised tests, such as the ANAM, are highly useful in detecting cognitive impairment and decline in MS patients.

Younes et al. (2007) also studied cognitive impairment in MS patients, but using the Cognitive Stability Index (CSI). The CSI battery was developed by HeadMinder for use as a screening tool in individuals with suspected neurological illness. As above, the computerised test provided a novel and improved procedure to paper and pencil tests. Both studies from Wilken et al. (2003) and Younes et al. (2007) suggest that computerised cognitive tests may be used not just for the purposes the tests were developed for, but may also extend to other kinds of brain damage or dysfunction. This is supported by Keith, Stanislav and Wesnes (1998) who used the Cognitive Drug Research (CDR) computerised system to measure cognitive functioning in individuals with brain injuries or organic illnesses. The CDR system was initially developed to assess the cognitive effects of drug use but was found to be useful in assessing short-term memory, discrimination skills and identification skills in those with TBI.

CogState is another popular cognitive test that was developed for different types of cognitive evaluation. For example, Cysique, Maruff, Darby and Brew (2006) used CogState for evaluating the cognitive impact of HIV. The researchers found that the CogState was sensitive to cognitive impairments experienced by HIV positive individuals. Falleti, Maruff, Collie and Darby (2006) also used the CogState but were assessing practice effects and test-retest reliability at different intervals. The researchers suggested that practice effects seem to be related to the type of testing and not the actual test. For example, participants who are unaware of how a test works will obtain lower scores than those who have encountered a similar test before. These types of practice effects can be ameliorated by using example tests before the actual tests as well as by using alternate forms of the tests for multiple testing sessions.

The CANTAB is yet another popular computerised test. It was developed for the cognitive assessment of neurodegenerative diseases and brain damage (Fray & Robbins, 1996). It is a sensitive test that can detect subtle changes in Alzheimer's, Huntington's and Parkington's diseases, and AIDS, as well as damage to the brain like in TBI and tumours. Moore, Ashman, Cantor, Krinick and Spielman (2010) used the CANTAB in gender research on TBI recovery. The researchers concluded that males and females do not differ significantly in cognitive recovery after TBI, with the possible exception of visual memory functioning, where females scored higher than males. The CANTAB test battery has also been used in autism research, such as Ozonoff and colleagues' (2004) research. The researchers found that the CANTAB executive subtests did not predict autism severity or symptoms but was related to adaptive behaviour.

As shown in the research above, many general cognitive tests are administered via paper and pencil, but with technological advancement, tests have been developed for computer administration such as the CANTAB, the MicroCog test battery, the CogState and others which have been mentioned above. These batteries contain many subtests that are used to measure different cognitive functions, such as memory, inhibition and response time. Computerised neuropsychological testing has grown in popularity for many reasons, (Crawford, Parker, & McKinlay, 1992; Wilken et al., 2003) for example computers are more accurate than human testers when examining timed tests and response times. These cognitive functions are known to be sensitive to the non-specific effects of diffuse brain damage including alcohol-related effects on cognition (Lezak et al., 2004). A further benefit of using computerised testing is that examiner bias is decreased because computers are not able to give cues for the correct answers by way of tone or facial expression (Crawford et al., 1992). Moreover, according to Crawford et al. (1992), testing in larger groups is also more feasible with computers and the test administrators do not need to be trained extensively in order to oversee the testing or scoring processes. Another advantage of computerized testing over traditional paper and pencil tests, especially those developed within the sports concussion context - for example, the ImPACT test - is that they have multiple randomised versions suitable for repeat test occasions (ImPACT, 2007; Wilken et al., 2003) that serve to reduce practice effects which may obscure the extent of the deficits over several test occasions (Schnirring, 2001). A major disadvantage of computerised testing is that if a person does not know how to use a computer, then they are not able to do the test, but in a highly technological age, this is increasingly rare and can be overcome using practice examples and brief training (Fisher, 2009).

The above studies also show that there is an abundance of computerised tests that are used for a wide variety of cognitive related research. The tests' use depends on the needs of the investigation and the test's attributes (Keith et al., 1998; Wilken et al., 2003; Younes et al., 2007). For example, in concussion management, the tests used need to measure a wide variety of domains because concussions can cause diffuse damage to the brain (Halstead, Walter, & The Council on Sports Medicine and Fitness, 2010). The tests also need to be sensitive because the damage can be subtle. Concussions are common in contact sports, like rugby and soccer. As with all causes of brain damage, concussions can cause cognitive and emotional changes that will interfere with academic work and family and friend relationships. This is why concussion management tools are important in sports, as well as in other areas that are related to diffuse brain damage. Halstead et al. (2010) conducted an extensive literature review which covered the causes, signs and symptoms and assessment of concussions. The researchers stated that there are a variety of computerised concussion management assessments available, including, for example, the ImPACT battery mentioned above along with the CANTAB, ANAM, CogSport (previously Concussion Sentinel) and HeadMinder Concussion Resolution Index (CRI).

The reasons that computerised testing in concussion management is preferred over paper and pencil tests are the same as for any other area of cognitive assessment (see p. 70). However, according to Collie, Darby and Maruff (2001) there are limitations to using computerised tests in concussion management. These include the necessity of special equipment, for example, some tests require not only the computer and usual peripheries, but also touch-sensitive screens for example. Another limitation is the cost involved when setting up the system. The costs include the test itself, which can be expensive, as well as all the equipment required. Some tests also require neuropsychologists to be trained in the administration and scoring of the tests, as is also required in most paper and pencil tests. The last limitation suggested is that of the lack of normative and test-retest reliability data available. However, Collie et al. (2001) do mitigate some of the limitations by stating that they are being overcome – in fact, some computerised tests require very little training of personnel for the administration of the test as the instructions are included onscreen with the tests. Also, the scoring of the tests is done automatically by the computer. As for the lack of normative, reliability and validity data, this is rapidly being overcome as more and more people are using computerised tests and the results are being stored electronically in a central database for each test. Also, quite a few studies have been done since the publication of Collie and colleagues' (2001) article which deal with the reliability and validity of the tests.

For example, Schatz and Putz (2006) who cross-validated computerised tests and validated them against paper and pencil tests. The computerised tests studied by the researchers included the ImPACT, HeadMinder CRI and CogSport. The researchers found that there were correlations between all the computerised tests in terms of processing speed but little shared variance was observed in the memory domain. The reaction time domain was correlated across only a few of the tests, for example complex reaction time, as measured by the ImPACT, was correlated with the TMT (parts A and B) and the Digit symbol test. It was stated that the ImPACT shared the most consistent correlations with the other tests, except on the memory domain. The researchers suggested that because of discrepancies between some of the tests, a single test battery should be chosen for the baseline and follow-up tests. One test should not be used as a baseline measure for another test.

The CogSport battery is a popular choice as a computerised concussion management tool. Makdissi et al. (2001) used the CogSport battery to measure the cognitive impairment experienced by concussed athletes, specifically football players. The researchers found that only the simple reaction time score changed significantly pre- and post-concussion. It was suggested that computerised testing may prove to be a particularly effective concussion management tool in that is more sensitive to subtle cognitive change compared to paper and pencil tests especially when it comes to timed measures like response time. Broglio, Macciocchi and Ferrara (2007) and van Kampen, Lovell, Pardini, Collins and Fu (2006) used the ImPACT (version 2) to assess concussed athletes once the athletes had ceased to exhibit physical concussion symptoms such as headaches and nausea. Both of these studies found that even after the athletes ceased to report physical symptoms of concussion, the athletes still experienced cognitive symptoms of concussion, such as memory problems, as measured by the ImPACT, and as such, were still considered to be concussed. This methodology was replicated by Fazio, Lovell, Pardini and Collins (2007) with similar findings. Gardner, Shores, Batchelor and Honan (2012) compared the CogSport and the ImPACT in assessing concussed male athletes. The control and concussed athletes differed on reaction time, decision making, matching and working memory from the CogSport and Visual Motor Speed and Total symptom score from the ImPACT. It was found that the CogSport and ImPACT had identical classification accuracy figures (87.4%) which suggested the tests perform equally well in identifying concussed athletes.

Notebaert and Guskiewicz (2005) investigated the current trends of concussion management tools in athletic trainers. The researchers found that those athletic trainers with more years of certification and those athletic trainers in college or professional fields were more likely to use computerised concussion management tools. From the surveys returned, 14.5% reported using computerised concussion management tools. Of those, 75% reported using the ImPACT battery as the primary computerised concussion management tool. The rest of the computerised concussion management responses consisted of 10% for ANAM, 4.5% for CogState and 4.5% for the HeadMinder CRI. The last 6% used other unspecified tests. The researchers do not give reasons for the predominance of the use of the ImPACT amongst users of computerised concussion management tools. However a literature search implicates a higher level of technological efficiency in the tool now having had several revisions and a much wider research base than is evident for the other products.

While developed in the US, this 'gold standard' tool has been adopted for use quite extensively within the South African context also. Shuttleworth-Edwards et al. (2008) conducted research on South African university sport players using the ImPACT and other cognitive tests. The researchers concluded that rugby players are more vulnerable to cognitive damage and that attention and visual motor speed are the functions that are most likely to be affected. The Rugby players significantly lowered their attention and visuomotor domain scores over the course of the year, where the non-contact control group did not.

Clark (2010) performed doctoral research into the neurocognitive and symptomatic profiles of South African athletes, both concussed and non-concussed, over one Rugby season. The participants were tested with the ImPACT at the beginning and end of the Rugby season. The participants who sustained concussions during the research were assessed additionally. The participants were also tested using the WMS-III Visual Reproduction and Verbal Paired Associates subtests. The results indicated that the Rugby players scored lower on the ImPACT composite scores and higher on the symptom scales than the non-contact sport players, regardless of concussion status. Clark (2010) also found that the non-contact sport players benefited more from practice effects on the Verbal Memory, Reaction Time and Impulse Control composite scores than the Rugby players. This research demonstrates that the internationally developed ImPACT battery can efficiently distinguish between contact sports players and non-contact sports players internationally and in South Africa and that university students were able to successfully complete an international computerised test (Broglio et al., 2007; Clark, 2010; Fazio et al., 2007; Shuttleworth-Edwards et al., 2008; van Kampen et al., 2006).

In short, the research stated above shows that computerised testing is useful and often preferred over paper and pencil tests. This is because they give a more accurate measurement of timed tasks, they are easy to administer (individually and in groups) and they have multiple forms. Concussion management tools are of interest in this research because they are sensitive to diffuse brain damage and mild cognitive changes over time. Alcohol use has been shown, by the diverse range of findings, to also cause diffuse and subtle damage (see Sections 5 and 6) and this damage may be measurable using a concussion management tool, such as the ImPACT.

The ImPACT has also been used in settings that are not strictly sports related, such as Iverson's (2006) research on the relationship between cognitive functioning and depression in university athletes. Specifically, Iverson (2006) wanted to determine if the ImPACT battery was sensitive to the cognitive functioning effects of depression or distress. The researcher concluded that those students with more severe depression-related symptoms had significantly lower reaction times and processing speeds and scored non-significantly lower on verbal memory tests than those with less severe depression-related symptoms. Elbin and colleagues (2013) also used ImPACT in a setting that was not strictly sports related and found that athletes with a learning disorder or ADHD scored lower on the ImPACT baseline tests than those control athletes without a learning disorder or ADHD. The researchers state that the findings are similar to other studies on learning disorders that used paper and pencil tests.

Recently, the ImPACT has been used in non-sport settings in a few studies. For example, in a medical setting, Tsushima et al. (2013) compared the ImPACT battery to traditional paper and pencil tests in diagnosing minimal hepatic encephalopathy, which is a neurodegenerative disorder which affects cirrhotic patients. The researchers discerned that the ImPACT gave comparable results to the paper and pencil tests. It was concluded that the ImPACT is a good test when diagnosing minimal hepatic encephalopathy. Conklin et al. (2013) used the ImPACT as a computerised assessment of cognitive effects experienced by adolescent brain tumour survivors. The researchers tested the validity of using the ImPACT in this context by comparing it to the WASI and other cognitive tests measuring recognition memory and word memory. It was found that the ImPACT was useful and sensitive in monitoring the late cognitive effects of brain tumour survivors and gave comparable results to the other tests. D. G. Thomas et al. (2011) researched adolescents brought into emergency departments with head injuries and suspected concussions. The researchers used the ImPACT to measure concussion severity. It was concluded that cognitive testing, like that provided by the ImPACT, at the time of injury predicted the recovery rate better than clinical grading.

In summary, the above review reveals that computerised cognitive testing is an improvement over paper and pencil cognitive tests for various reasons (Crawford et al. 1992; Collie et al., 2001) and that there is a wide variety of tests to choose from (Snyder et al., 2011). The ImPACT has been shown to be a popular and well-researched test (Notebaert & Guskiewicz, 2005) useful in assessing damage to the brain over time all over the world (Broglio et al., 2007; Fazio et al., 2007; van Kampen et al., 2006) as well as in South Africa

(Clark, 2010; Shuttleworth-Edwards et al., 2008). It can also be used in settings other than those associated with sport and concussions (Conklin et al., 2013; Elbin et al., 2013; Iverson, 2006; D. G. Thomas et al., 2011; Tsushima et al., 2013).

7.4 South African Cognitive Testing

Even though most cognitive tests have been developed in America and Europe, these tests have been administered successfully in South Africa, for example in research mentioned above by Anderson & Dawson (1999), Clark (2010), Hartshorne (2000), Shuttleworth-Edwards et al. (2008) and Shuttleworth-Edwards, Radloff et al. (2013). Rushton, Skuy and Fridjhon (2003) also conducted cognitive testing on a South African sample, specifically using students from the University of the Witwatersrand. The research concerned the effects that race can have on cognitive functions, particularly those measured by the Raven's Progressive Matrices. The researchers noted significant differences in score according to race, which can be attributed to poverty and educational adversity in South Africa. The students all increased their scores through the testing sessions, which is possibly due to practice effects. Like Rushton et al. (2003), Skuy, Schutte, Fridjhon and O'Carrol (2001) also examined the effects race can have on commonly used cognitive functioning tests. Skuy et al. (2001) found that, likewise to Rushton et al. (2003), the American norms are inappropriate for African students.

Alant and Beukes (1986) and Pakendorf and Alant (1997) translated and culturally adapted the PPVT Revised version (PPVT-R) into Afrikaans and Sotho respectively and administered the tests individually to school children. The original PPVT tests are based on American English and the norms are based on American age and education levels. The studies attempted to translate and administer the PPVT-R test to South African children to determine whether American tests can be used and adapted successfully for a South African non-English context. Like Rushton et al. (2003) and Skuy et al. (2001) above, the researchers found that the South African scores were slightly lower than the American norms.

Olivier (2009) conducted a doctoral thesis also using a version of the PPVT, specifically version Three (PPVT-3), along with other tests from the Emergent Literacy Assessment, to assess literacy levels in children. The researcher found that participants in the higher socio-economic status groups scored significantly higher on the PPVT-3 than those in the lower socio-economic groups. It was also found that all the participants scored approximately 15 points lower than the mean standard score of 100 for the test. The researcher states that these scores are considered low average and are as a result of the test being normed on an America population.

The Raven’s Progressive Matrices and the PPVT are traditionally paper and pencil tests, however, computerised cognitive testing has recently become popular in South Africa; for example Fisher (2009) researched the use of computerised cognitive tests in the South African context. Most people in South Africa have very little or no access to computers and as such have very little, sometimes no, experience with using computers. The researcher found that computer mouse ability affected performance significantly on the computerised tests. It was also found that computer experience, computer confidence, gender and home language were associated with computer ability. However, when computer mouse ability was partialed out of the analysis there were no main effects for computer experience, computer confidence, gender or home language. Fisher (2009) concluded that the differences in computer ability between gender and home language may be due to other effects, such as differences in computer experience and confidence, rather than differences between the genders and languages. These results suggest that South Africans can be tested using computerised cognitive tests if the participants are given brief training and practice in using a computer and its peripheral attachments, like mice and keyboards.

The above results show that internationally developed cognitive tests may be administered to South African individuals but that South Africans may score lower than the international standard scores. This could be due to various reasons, for example, education and, specifically in terms of computerised testing, previous access and exposure, or lack thereof, to computers.

In terms of education, Shuttleworth-Edwards et al. (2004) looked at the effects that level and quality of education can have on cognitive test performance. The results showed that English first language white individuals with advantaged education (private schooling in South Africa and Zimbabwe and Model C schooling in South Africa) scored at least as well as those individuals from America and at least as well as American standards. Black participants, who received advantaged education, also achieved scores that were highly equivalent to the American standardization. However, those who had disadvantaged education, which is the former Department of Education and Training (DET) schools and current township schools, scored approximately 20 points lower than American standard scores on the Wechsler Adult Intelligence Scale (WAIS) Third Edition (WAIS-III). This research was subsequently refined, with a replication of the basic outcome implicating the deleterious effect of a lower quality of education amongst black Xhosa participants on the WAIS-III test (Shuttleworth-Edwards, Gaylard, & Radloff, 2013). This suggests that economic and educational deprivation impacts on cognitive test performance.

Shuttleworth-Edwards, Whitefield-Alexander, Radloff, Taylor, & Lovell (2009) conducted an investigation using the ImPACT on South African Rugby players. The ImPACT results showed that the white English first language individuals performed at least as well as the American athletes, but the researchers cautioned that this equivalence of scores might not apply to those with disadvantaged education. The above results from Shuttleworth-Edwards et al. (2004), Shuttleworth-Edwards, Gaylard et al. (2013) and Shuttleworth-

Edwards et al. (2009) indicate that if South Africans have had advantaged education, regardless of race, then they are likely perform close to American standards and score higher on cognitive tests, when compared to those with disadvantaged education.

7.5 Cognitive Testing in Alcohol use

In alcohol-related cognitive testing research, many cognitive domains have been investigated. From the studies on the consequences of alcohol use, stated in Sections 5 and 6, the most often tested domains are those of memory, attention, visuospatial abilities, reaction time, motor skills and reasoning. Many studies also estimated initial intelligence using vocabulary and verbal tests. For example, the NART (Corley et al. 2011; Manning et al. 2008; Rosenbloom et al., 2004; Skogen et al., 2011), the WAIS Vocabulary subtest (Corral et al., 2006; Sher et al., 1997; Wood et al., 2002) and the WISC Vocabulary subtest (Brown et al., 2000; K. L. Hanson et al. 2011; Tapert et al., 2002) are used among others.

As stated, memory is often assessed in alcohol related research. The research has looked at different kinds of memory such as non-verbal memory (Manning et al. 2008; Moriyama et al. 2006), short term memory (Britton et al. 2004; Brown et al., 2000; K. L. Hanson et al. 2011; Ilan & Gevins, 2001; Rosenbloom et al., 2004; Tapert et al., 2002; Thoma et al., 2011), verbal memory (Corley et al. 2011; Parada et al. 2012; Piechatzek et al., 2009; Rodgers et al., 2005; Sher et al., 1997) and working memory (Anderson & Dawson, 1999; Corley et al. 2011; Glass et al., 2009; Ilan & Gevins, 2001; Montgomery et al. 2012; Parada et al. 2012; Sher et al., 1997; Smith & Fein, 2010; Squeglia et al., 2011; Tapert et al., 2002; Thoma et al., 2011; Wood et al., 2002). The different kinds of memory are tested using many tests, like the Information, Coding and Arithmetic subtests from the WISC, Spatial and Digit span subtests from the WMS and word list type tests.

Attention is also a common construct tested in alcohol related cognitive functioning research (Anderson & Dawson, 1999; Moriyama et al. 2006; Rodgers et al., 2005; Smith & Fein, 2010; Tapert et al., 2002; Thoma et al., 2011). Alcohol related research discussed above tested attention by using the TMT (Glass et al., 2009; K. L. Hanson et al. 2011; Loeber et al. 2009; Moriyama et al. 2006; Sher et al., 1997; Smith & Fein, 2010; Tapert & Brown, 1999; Tapert et al., 2002; Thoma et al., 2011; Wood et al., 2002). The TMT assesses cognitive flexibility and visuomotor tracking as well as attention. The Stroop Colour-Word Test also measures attention as well as working memory (Glass et al., 2009; Piechatzek et al., 2009; Smith & Fein, 2010; Tapert et al., 2002). The PASAT measures attention and working memory and was used by Anderson and Dawson (1999), Glass et al., (2009), Smith and Fein (2010) and Tapert et al. (2002).

Alcohol related investigations, discussed above, that assess visuospatial abilities did so by using a few different tests, for example Anderson and Dawson (1999), Moriyama et al. (2006), Rodgers et al. (2005), Smith and Fein (2010), and Tapert et al. (2002) used the Symbol Digits Modalities test, which also tests for motor speed, processing and memory. The WISC Block Design subtest also measures visuospatial abilities, specifically visuoconstructional ability (Brown et al., 2000; K. L. Hanson et al. 2011; Tapert et al., 2002). The WMS Paired Associates subtest measures visuospatial abilities along with memory and learning (Manning et al. 2008; Wood et al., 2002). The Rey-Osterrieth Complex Figure Test was used by K. L. Hanson et al. (2011), Smith and Fein (2010) and Tapert et al. (2002) to measure visual perceptual organisation and visual recall.

As discussed above, motor skills and reaction time are also often found to be influenced by alcohol use. Motor skills are related to aspects of speed, accuracy and visuospatial skills (Lezak et al. 2004). Manning et al. (2008) measured motor speed by using a Motor Screening Test, while Squeglia et al. (2011) used the WASI Block design subtest to assess motor skills and spatial abilities. Reaction time is related to both attention and motor skills (Lezak et al., 2004). Reaction time is easier to assess using computerised tasks (Rodgers et al., 2005) but can be measured using paper and pencil tasks as well (Corley et al. 2011; Moriyama et al. 2006). Stop signal tasks were used by Bednarski et al. (2012) and Glass et al. (2009) to measure participants' reaction times.

"Reasoning is thinking with a conscious intent to reach a conclusion" according to Lezak et al. (2004, p. 593). In alcohol related research, Britton et al. (2004) used a mathematical reasoning task to measure reasoning and pattern recognition while Smith and Fein (2010) used a Short Category Test to measure conceptual and spatial reasoning. The Halstead-Reitan Neuropsychological Test Battery Booklet Categories subtest also measures conceptual and spatial reasoning and it was used in research by Sher et al. (1997), Tapert and Brown (1999), Tapert et al. (2002) and Wood et al. (2002).

The most common tests used in alcohol-related cognitive functioning research are batteries evaluating a range of cognitive domains. The batteries by Wechsler are often used in the research stated above, including the WISC (Tapert & Brown, 1999), the WAIS (Elias et al. 1999; Mortensen et al. 2001; Tapert et al., 2002), the WMS (Elias et al., 1999), the Reduced Wechsler Intelligence Scale (Loeber et al., 2009) and the WASI (Manning et al. 2008; Thoma et al., 2011). The Wechsler scales do not have to be used in their entirety; the subtests can be used independently as in some of the studies mentioned above. The MMSE battery is generally used to test for dementia and can be used to detect gross changes in memory and general functioning (Leroi et al. 2002; Lobo et al., 2010; Rodgers et al., 2005). Computerised cognitive test batteries have also successfully been used in alcohol-related cognitive testing such as the CANTAB that was used by Kopera et al. (2012), Manning et al. (2008) and Piechatzek et al. (2009), the MicroCog used by Smith and Fein (2010) and the CALCAP used by Hartshorne (2000).

For the purposes of the present research it was considered important to take into account the functional modalities commonly affected by alcohol use as indicated above, and then to identify an efficient mode of evaluating changes in those sensitive modalities by means of an appropriate choice of cognitive tests.

Section 8: Measures chosen for application in this research

Three broad aspects informed the choice of tests for this research: (i) the use of an alcohol use questionnaire that will separate the drinkers into categories that can be easily analysed, (ii) the use of a test to measure initial intelligence as a control variable in alcohol-related cognitive functioning studies and (iii) the use of a computerised cognitive test battery. For the purposes of this research this included (i) the Alcohol Use Disorders Identification Test (AUDIT), (ii) the paper and pencil Peabody Picture Vocabulary Test version 4 (PPVT-4) and (iii) the computerised Immediate Postconcussion Assessment and Cognitive Test (ImPACT) battery.

8.1 The Alcohol Use Disorders Identification Test (AUDIT)

The AUDIT is a popular tool for measuring alcohol use and its patterns over the previous 12 months (Marshall et al., 2010). It is an internationally used questionnaire that was developed by the World Health Organisation in 1982 for the screening of hazardous use of alcohol and alcohol-related disorders (Babor, Higgins-Biddle, Saunders & Monteiro, 2001).

Structurally, the test consists of 10 multiple choice questions, each with a maximum of 5 answers (Babor et al., 2001). The questions deal with alcohol consumption patterns and are able to distinguish between drinkers whose patterns may lead to future health problems and those whose drinking had already caused health problems (Allen, Litten, Fertig & Babor, 1997). Each question is scored between 0 and 4, giving a total score out of 40, with higher scores indicating more hazardous drinking patterns. This score is categorised into one of four drinking patterns: Safe, Hazardous, Harmful and Dependent. The Safe group includes abstainers as well as drinkers who are least likely to experience negative consequences or harms related to their drinking. The Hazardous group includes those drinkers whose patterns of alcohol use may lead to future physical and mental health problems. Harmful drinkers currently experience harms related to alcohol use, such as mental and physical health problems. The Dependent group experience the symptoms of Dependence syndrome stated in Section 4.1 (see p. 7).

The AUDIT has been compared to other alcohol use tests, for example in studies evaluated in Schuckit's (2009) literature review on alcohol-use disorders. The paper was a general overview of the causes, diagnosis, consequences, treatment and rehabilitation of alcohol use disorders. Windle and Zucker (2010) also reviewed the AUDIT's usage in alcohol use studies in a literature review. Most studies used the AUDIT as a measure of alcohol use patterns in certain populations or as a screening measure for inclusion or exclusion into a study on the influence of alcohol use or what may cause risky alcohol use (Schuckit, 2009; Windle & Zucker, 2010).

The AUDIT has been effectively used extensively in many studies involving university settings around the world (for example, Andersson, Johnsson, Berglund, & Ojehagen, 2007; Fleming, Barry, & MacDonald, 1991; Granville-Chapman, Yu, & White, 2001; Kypri, Langley, McGee, Saunders, & Williams, 2002; McShane & Cunningham, 2003; Miles et al., 2001; O'Hare & Sherrer, 1999; Parada et al., 2012; Rickwood et al., 2011; Shields, Guttmannova, & Caruso, 2004). It has been used in South African contexts; for example Peltzer et al. (2011) used the AUDIT in a South African national study of alcohol use and misuse prevalence rates. It has also been used at Rhodes University in recent years by

Young and de Klerk (2008), and Young and Mayson (2010) (see p. 18). The Rhodes University studies reported on the prevalence rates in the university and investigated the relationship between normative estimates and alcohol consumptions. The AUDIT has also been used in cognitive functioning assessment research to separate drinkers into categories for either inclusion into the study or for analysis into the relationship between alcohol use and cognitive functioning (Bednarski et al., 2012; Dawson et al., 2007; Montgomery et al., 2012; Parada et al., 2012; Rodgers et al., 2005). The AUDIT will be used in this research to categorise the university students into drinking categories.

8.2 Peabody Picture Vocabulary Test (PPVT)

With a view to controlling for initial intelligence, and as with other alcohol-related cognitive research that have controlled for this influential variable, a measure of initial intelligence is used in this study, that is the Peabody Picture Vocabulary Test (PPVT) version Four (PPVT-4). As stated above, many of the cognitive assessment studies used a vocabulary test to measure verbal ability (p. 79) because verbal ability and vocabulary are known to be proxies for initial intelligence (Lezak et al., 2004). According to Betz, Eickhoff, Sullivan, Nippold and Schneider's (2013) survey research, the PPVT-4 was the third most used test by surveyed practitioners and 60% of the practitioners said that they used the PPVT-4.

Structurally, the PPVT-4 is an untimed neuropsychological test that measures the verbal ability of subjects through the use of a list of vocabulary words and coloured picture plates (Dunn & Dunn, 2007). The purpose of the PPVT scores in this research is for comparative group analyses of all South African groups with to the aim of controlling for initial intelligence, rather than an attempt to estimate IQ level per se. Therefore the fact that scores may be lower for South African samples than American standard scores does not present a problem.

As mentioned earlier, research has shown that the current version of the PPVT, that is the PPVT-4, has been used to determine initial intelligence successfully in previous research (Glenwright & Agbayewa, 2012; Stevens et al., 2011; Tlustos et al., 2010). The above investigations show that the first version of the PPVT has been used successfully in South African contexts (Alant & Beukes, 1986; Olivier, 2009; Pakendorf & Alant, 1997). Borovsky, Elman and Fernald (2012) showed that it can also be used successfully in a young adult sample by researching children and college-aged adults separating the sample into high and low vocabulary groups. Guthrie and Seifert (1977) showed that the PPVT has been used successfully in a group setting such as that which will be used in this research. Norris, Hottel and Brooks (1960) and Tillinghast and Renzulli (1968) also found that the test can be used in group form with reliable results.

8.3 Immediate Postconcussion Assessment and Cognitive Test (ImPACT)

The ImPACT test, being the most widely employed and well-researched of the computerized tests developed in the sports concussion arena, was the test selected for use in the present study (ImPACT, 2007; Notebaert & Guskiewicz, 2005). Structurally, the ImPACT programme is a computerised neuropsychological test battery that comprises six cognitive tests: Word Memory, Design Memory, X's and O's, Symbol Match, Colour Match and Three Letters (ImPACT, 2007). From these six cognitive tests, the programme automatically generates five cognitive composite scores: Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Cognitive Efficiency Index. It has a sixth composite score, Impulse Control, which is used as a validity indicator, where if a participant scores above 20 then the test result is considered invalid. In addition, the battery includes a Post-concussion Symptom Scale which consists of a list of 22 symptoms related to concussions, such as fatigue, nausea, headaches and trouble sleeping. These are measured on a Likert-scale from 0 to 6. The individual symptom scores and a composite symptom score are given in the test report along with the composite cognitive scores and the individual test scores.

The research reviewed above indicates that the ImPACT has been used successfully on South African athletes (Clark, 2010; Shuttleworth-Edwards et al., 2008; Shuttleworth-Edwards, Radloff et al., 2013) and has been shown to be sensitive to the effects of mild neurocognitive damage such as that caused by concussion (Broglio et al. 2007; Fazio et al., 2007; van Kampen et al., 2006). It was also able to determine the differences between those with mild neurocognitive damage and controls by showing that the participants with damage deteriorated over a year compared to the control participants who did not (Shuttleworth-Edwards et al., 2009). The ImPACT has been shown to be particularly efficient at measuring and detecting changes in visual motor and processing speed (Shuttleworth-Edwards, Radloff & Whitefield-Alexander, 2014). It is documented that processing speed is one of the most sensitive indicators of diffuse brain injury in general. This has also been shown to be the case in concussion research using the ImPACT battery (Lezak, 2004; Shuttleworth-Edwards et al. 2014). Additionally, it was also used successfully in contexts not related to concussion management including depression, learning disorders, diagnosis of minimal hepatic encephalopathy and brain tumours (Elbin et al., 2013; Conklin et al., 2013; D. G. Thomas et al., 2011; Iverson, 2006; Tsushima et al., 2013).

Since the ImPACT is very sensitive to brain injury symptoms, both physical and neuropsychological, and the changes of these over time, it is a useful test to use in this current study, as the damage caused by alcohol use may be subtle and not always overt as with the sequelae of the concussive brain injury and thus not easy to measure.

Section 9: Rationale for the research

As discussed above, a little over half of the South African population consumes alcohol (World Health Organisation, 2014). Clausen et al. (2009), Parry et al., (2005), Peltzer and Ramlagan (2009), and Peltzer et al. (2011) found that approximately 30% of the current drinkers in South Africa consume alcohol hazardously or binge drink. These figures are fairly high when compared to the World Health Organisation's (2014) weekly heavy episodic drinking rate of only 16% of all current drinkers in the world. This suggests that South Africans consume alcohol heavily, especially when compared to the world averages.

Young adulthood is a time for many changes, especially for those moving away from home and going to university (Colby et al., 2009; Schulenberg & Maggs, 2002; Steyl & Phillips, 2012; Young & de Klerk, 2012). During this time, university students often start using alcohol, some to excess (Ham & Hope, 2003). At Rhodes University, approximately half of the students are categorised on the AUDIT as Safe drinkers, which means that they consume alcohol at Safe levels or do not drink at all (Young & de Klerk, 2008; Young & Mayson, 2010). The remaining 50% of the students, however, either drink at Hazardous levels (30-40%) or at Harmful or Dependent levels (13-20%). This indicates that Rhodes University students drink at high levels which may cause problems with academic work and social lives, not to mention causing problems with the university as an institution (Park, 2004; Rickwood et al., 2011; World Health Organisation, 2011a). Rhodes University was chosen for this research because of the high drinking rates and possible consequences that those high drinking rates may have on the population.

Young adults at university were chosen because of the possibility of alcohol related damage to the brain during young adulthood. This may occur because the brain is still developing in this age group (Brown et al., 2009; Gazzaniga, Ivry & Mangun, 2009; Giedd, 2008; Gogtay et al., 2004). The research stated above showed that alcohol use has been linked to changes in the brain (Fils-Aime et al., 1996; Hermens et al., 2013; Squeglia et al., 2011; Sullivan & Pfefferbaum, 2005) and many cognitive domains, such as different types of memory (Brown et al., 2000; K. L. Hanson et al., 2011; Montgomery et al., 2012; Parada et al., 2012), motor speed (Sher et al., 1997), attention (Montgomery et al., 2012; Smith & Fein, 2010; Thoma et al., 2011), visuospatial ability (Brown et al., 2000; K. L. Hanson et al., 2011; Sher et al., 1997), reaction time (Glass et al., 2009; Hartshorne, 2000) and inhibition (Montgomery et al., 2012).

Previous studies discussed have indicated that the relationship between alcohol use and cognitive functioning is not clear, with some studies finding negative effects (K. L. Hanson et al., 2011; Hermens et al., 2013; Sher et al., 1997; Sullivan & Pfefferbaum, 2005; Zeigler et al., 2005), others showing no effects (Leroi et al, 2002; Lobo et al., 2010) and still others finding beneficial effects of alcohol use (Corley et al., 2011; Elias et al., 1999; Britton et al., 2004; Rodgers et al., 2005; Smith & Fein, 2010; Wright et al., 2006). These inconsistent findings could be explained by the many factors that can affect the relationship between alcohol use and cognitive functioning (El-Guebaly, 2007; Tapert et al. 2005) (see Section 6). These factors encompass demographic factors, alcohol and substance use related factors and current health factors. The demographic factors are gender, race, current age, initial intelligence and education. The alcohol and substance use related factors include age of onset, duration of alcohol use, parental alcohol use, hangovers, number of pass out incidents, type of alcohol consumed, severity of alcohol use pattern, substance use and cigarette smoking. The current health factors are current physical and psychological health. The analyses will attempt to control these factors and an exploratory analysis will be done on the substance related factors to determine any effects that they may have.

Another important factor for this research is time. Cognitive functioning changes over time due to many factors, such as aging (Corral et al., 2006) as well as damage to the brain

(Becker et al., 2011; Dawson et al., 2007; Hessen et al., 2007; Kersel et al., 2001; Moser & Schatz, 2002; Tlustos et al., 2010; Zeigler et al., 2005). The advantage of a short-duration prospective study is that the duration is short enough so that age-related damage is not a major contributing factor. The period of one academic year was chosen for this research to measure the effects that the pattern of alcohol can have on the brain. The students' alcohol patterns would be fairly stable at this point because they are still starting their academic careers (Jackson et al., 2001; Karlamangla et al., 2006; O'Malley, 2004; Patrick & Schulenberg, 2011) (see p. 12). Only major changes would be likely to change their drinking patterns from the previous 12 months, such as health, financial and family issues (Mikhailovich et al., 2011; Slicker, 1997).

The AUDIT was chosen because it is a well-validated, much used test and has been used successfully previously on university campuses (Miles et al., 2001; Rickwood et al., 2011), including Rhodes University (Young & de Klerk, 2008, 2012; Young & Mayson, 2010). It has also been used in alcohol-related cognitive functioning research (Bednarski et al. 2012; Manning et al. 2008; Montgomery et al. 2012; Parada et al. 2012; Rodgers et al., 2005; Skogen et al., 2011). The AUDIT measures alcohol use patterns in the previous 12 months and separates the drinkers into four categories: Safe, Hazardous, Harmful and Dependent (Babor et al., 2001). The Safe and Hazardous groups are not likely to have experienced alcohol-related harm yet, according to Babor et al. (2001) while the Harmful and Dependent categories most likely have. Therefore, for the purposes of this research, the four categories will be clustered into two groups. The Safe and Hazardous categories will be clustered together and the Harmful and Dependent categories will be grouped together.

Verbal ability will be tested as it has been shown to be related to initial intelligence and is relatively unimpaired by mild cognitive damage such as that caused by alcohol use (Lezak et al., 2004). It is thus a good indicator of initial intelligence levels. Verbal ability is often measured by vocabulary tests, for example the WISC and WAIS vocabulary subtest (Brown et al., 2000; Tapert et al., 2002). The PPVT-4 is the vocabulary test that was selected for this research. The PPVT-4 has not been used in alcohol related research before but has been used in university samples (Borovsky et al. 2012). The current version has not been used in South African research, however previous versions have been used (Alant & Beukes, 1986; Olivier, 2009; Pakendorf & Alant, 1997). Previous versions of the test have also been administered successfully in group settings (Guthrie & Seifert, 1977; Norris et al., 1960; Tillinghast et al., 1968). The PPVT-4 will be used in this research in group settings and on a university student population.

The ImPACT was chosen for this research because it is a popular test that has been shown to be sensitive to subtle diffuse brain damage, especially in the concussion area (ImPACT, 2007). Subtle diffuse brain damage is also caused by different levels of alcohol use, which is indicated by the diverse findings in alcohol-related cognitive functioning research. A computerised test was selected because computerised tests are more accurate than paper and pencil tests in assessing timed tasks, like those for reaction time and processing speed. These functions that are known to be negatively affected by diffuse brain injury in general, including alcohol use (see p. 45). The ImPACT also measures verbal and visual memory which, as stated above, is generally deleteriously affected by alcohol use. The ImPACT symptom list will also be used to identify any current health symptoms that may be related to alcohol use in university students.

As discussed above, the ImPACT has shown that practice effects can be diagnostic of cognitive deficits (Shuttleworth-Edwards, Radloff et al., 2013). According to Duff et al. (2012), Duff et al. (2007) and Shuttleworth-Edwards, Radloff et al. (2013), practice effects can be diagnostically relevant. For instance, if practice effects are expected, as they would be over the course of multiple testing sessions, and are not present, it can be deduced that there may be some cognitive functioning deficiency present. Only one alcohol related cognitive functioning study was located that included practice effects as a diagnostic tool (Anderson & Dawson, 1999). The researchers tested students when they were hungover and again when they were not hungover and included a control group of participants to assess any practice effects that may have occurred with the multiple testing sessions. Practice effects, however, have not previously been included in a study involving alcohol use patterns and cognitive functioning over time. Practice effects will be used diagnostically in this research as they have been used diagnostically in previous research (Shuttleworth-Edwards, Radloff et al., 2013) and have also been used to predict future cognitive functioning in progressive neurodegenerative disorders causing mild brain damage (Duff et al. 2007).

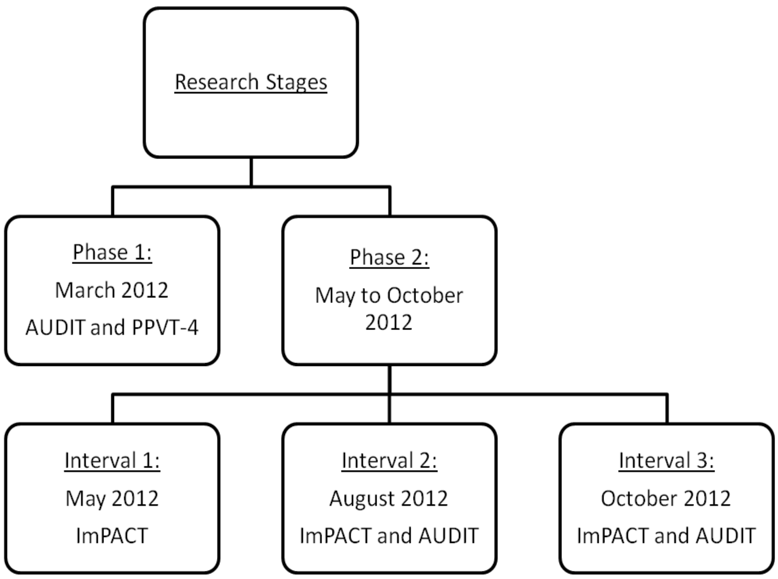
In the past, an alcohol-related prospective study has not been conducted with all the influential variables together in a university sample with a purely computerised cognitive test battery and controlling for cognitive ability. Therefore, this study hypothesises that those students in the heavier drinking categories (Harmful and Dependent) will score worse on cognitive tests than those in the lighter drinking categories (Safe and Hazardous). It is also hypothesised that practice effects will be seen in the lighter drinking categories but not to the same extent in the heavier categories due to cognitive vulnerability in association with the heavier drinking categories. In terms of the exploratory analyses, it is hypothesised that the substance related influential variables will have a significant effect on the alcohol use cognitive functioning relationship.

Chapter 2: Methodology

This section focuses on the methodological aspects of the current research. The methodological choices for the current research were informed by and based on the literature reviewed in the previous chapter. Participant selection is first discussed, along with the exclusion criteria. The final sample characteristics are then displayed. The data collection methods are presented, including the measures used, ethical considerations and the procedure followed. The data analysis is briefly described at the end of this chapter along with the research questions.

The study took place between March and November in the 2012 academic year and was conducted in two test-taking phases: Phase 1 (March) and Phase 2 (May to October). Phase 1 included a single testing protocol in March, repeated at three different sessions with different students. Phase 2 included three different testing intervals in May, August and October. The stages of the data collection procedure are shown in Figure 1 with the corresponding dates and the tests used. A total of 216 students completed Phase 1 and 92 students completed all three testing intervals of Phase 2.

Figure 1: Research Stages



Section 1: Participants

1.1 Population characteristics

In 2012, Rhodes University had 7395 registered students, of which approximately 58% were female (Rhodes University, 2013). Approximately 71% of the students were registered for undergraduate degrees and approximately 38% of the students were registered in the Humanities faculty.

Of the student population, 40% were white, while 60% were Black. The Black students were specifically comprised of 52% black African, 4% Indian and 4% coloured students (Rhodes University, 2013). Importantly, the terms black African, coloured, Indian and white are used in this research, as they were in the most recent 2011 census, where the demographics of South Africa encompassed approximately 52 million people of diverse origins, cultures, languages, and religions (Lehohla, 2012). Currently the term 'Black' is preferred when making a general reference to those race groups in the country that historically were referred to as 'non-white' (Bowman, Seedat, Duncan, & Burrows, 2006). Therefore, the term 'Black' has been applied in this thesis with reference to a group made up of the sub-groups of black African, coloured and Indian individuals included in the study.

Second and Third year Psychology students were approached for this research so as to attempt to capture a large sampling pool of students. Additionally, by this stage of their university studies these students would be likely to have an established drinking pattern at university level (Jackson et al., 2001; Karlamangla et al., 2006; O'Malley, 2004; Patrick & Schulenberg, 2011) (see Section 4.2.2 of the Literature Review, p. 12). There were 367 and 288 students registered in 2012 for Psychology 2 and Psychology 3 respectively. Participants in this study were drawn from those classes.

1.2 Exclusion criteria

1.2.1 Phase 1 exclusions

Of the 216 Psychology 2 and 3 students who completed Phase 1, 30 were excluded from the sample because they did not consent to participating in Phase 2. One student who did not consent to participating in Phase 2 did however attend Intervals 1 and 2 in Phase 2 and completed the Phase 2 consent form.

Additionally, initial intelligence was controlled by the PPVT-4 scores. Vocabulary is a relatively stable indicator of intelligence, as discussed above (see Section 7 of the Literature Review, p. 59). Given that these participants are all either second or third year university students, it is likely that the explanation for any poor performance on the PPVT-4 is that they either did not take the test seriously or that they have some difficulty with the English language. Thus, students having a PPVT-4 standard score lower than 75 were excluded. Three students were excluded on these grounds.

Two students were excluded because they did not include their year of birth in the initial questionnaire so their ages could not be calculated. Repeated attempts to contact them to get their year of birth were ignored by the participants. Finally, two students older than 26 years were excluded to control for age. This was done because the frontal lobe is thought to have reached full maturity by age 25 (Giedd et al., 1999; Gogtay et al., 2004). Thus, older age groups may respond differently to alcohol use with regards to cognitive functioning, as stated above in Section 5.2.3 of the Literature Review (p. 38).

None of the students who were excluded were informed of their exclusion. They were not invited to take part in Phase 2 of the study. This yielded a total of 179 students who were invited to take part in Phase 2.

1.2.2 Phase 2 exclusions

A total of 92 students completed all three of the testing intervals in Phase 2 of the research. The following section describes exclusions that took place after data collection and before data analysis.

a) Alcohol use

The students completed the AUDIT at three times during the year; in Phase 1 in March and in Phase 2 (Intervals 2 and 3) in August and October. From this it could be determined whether any of the students changed their alcohol use patterns markedly over the course of the year. Due to Intervals 1 and 3 taking place in the month before examination periods, the drinking patterns were expected to change slightly from one interval to the next. This slight change would be seen by scoring in the same AUDIT category (Safe, Hazardous, Harmful and Dependent) or by either increasing or decreasing by only one AUDIT category. If any of the students changed their alcohol use enough to shift between two of the AUDIT categories, it was concluded that their alcohol use patterns were highly variable. One student was excluded from the sample on these grounds by moving down two categories: from the Dependent category in Phase 1 to the Hazardous category in Intervals 2 and 3 in Phase 2. This yielded a sample of 91 students.

b) Education

Not only is the PPVT-4 a proxy for initial intelligence, as discussed above (see Section 8.2 in the Literature Review, p. 84), but it can also be an indicator of educational disadvantage, especially in a context such as South Africa where there are large differences between the educational institutions (Shuttleworth-Edwards et al., 2004; Shuttleworth-Edwards, Gaylard et al., 2013; Shuttleworth-Edwards et al., 2009). This educational disadvantage is likely to impact on their cognitive test performance because tests like these are generally normed on privileged English first language subjects.

Students educated at former DET/Township schools had significantly lower PPVT-4 raw and standard scores than students educated at other types of institutions. An Analysis of Variance (ANOVA) on the PPVT-4 raw scores showed significant differences between the Education types ($F(3, 87) = 7.09$, $p < .001$). A post hoc Tukey Honestly Significant Difference (HSD) test showed that the significant differences lay between the former DET/Township type mean score (177.5±10.08) and the other three education type mean scores: Private (204.00±10.30, $p < .001$), Government (201.87±11.30, $p = .001$) and Other (202.88±12.1, $p = .001$). Other high schools are those in other countries. An ANOVA on the PPVT-4 standard scores also showed that there was a significant difference across the Education types ($F(3, 87) = 4.47$, $p = .006$). A Tukey HSD test showed that the former DET/Township type mean score (83.00±6.63) was significantly lower than the mean scores of the other three Education types: Private (104.74±10.16, $p = .003$), Government (103.29±12.77, $p = .007$) and Other (104.24±12.17, $p = .007$).

Thus, the former DET/Township educated participants' scores were excluded from the final analysis. To avoid stigmatisation of participants, all those who consented and who were not initially excluded were allowed to complete the tests and nobody was informed that their data would be excluded from the final analysis. Four students were excluded on these grounds, yielding a sample of 87 students.

c) Impulse Control

According to the ImPACT (2007) manual, the Impulse Control composite score can be used as a validity indicator. It suggests that a score of 20 or more can indicate an unreliable test taker. A high score in Impulse Control may indicate purposefully under-performing on the tests. Additionally, Shuttleworth-Edwards, Radloff et al. (2013) suggested that the test taker may naturally have an erratic test taking style which may present in the Impulse Control composite score. As in the Shuttleworth-Edwards, Radloff et al. (2013) study, the participants in this study did not stand to gain anything by purposefully performing badly. The mean scores for each alcohol group over the time intervals in Table 1 show that, in general, the 87 students were motivated and co-operative which is indicated by (i) low average scores on the Impulse Control composite that were at worst approximately 5, with standard deviations being at most approximately 4, and therefore were well below the cut off point of 20 and (ii) an equivalence between the alcohol groups being investigated on this variable ($p > 0.05$). Therefore, it is argued that the scores do not serve as an explanation for any of the between groups differences found on the dependent variables.

Table 1: Impulse Control Means across Alcohol Groups before exclusion

		M (SD)				
Alcohol groups	***n***	**Interval 1**	**Interval 2**	**Interval 3**	***F*(2, 170)**	***p***
Safe/Hazardous	74	3.52 (2.70)	4.23 (3.79)	4.44 (3.53)	0.29	.746
Harmful/Dependent	12	3.83 (2.48)	4.67 (2.46)	5.42 (3.87)		

Note: *p < .05, **p < .01

There was only one student who scored above 20 on the Impulse Control composite; specifically at Intervals 2 (25) and 3 (22). It was decided that this student be excluded because he also scored low on many of the other tests and had many of the neurological vulnerabilities discussed in Section 1.3 (ADD/Hyperactivity, Learning disability, Psychiatric disorder, Grade repeated, Neurological disorder, Dyslexia and Concussion). The mean Impulse Control scores after exclusion are shown in Table 2. There were still no significant differences after exclusion.

Table 2: Impulse Control Means across Alcohol Groups after exclusion

		M (SD)				
Alcohol Groups	***n***	**Interval 1**	**Interval 2**	**Interval 3**	***F*(2,168)**	***p***
Safe/Hazardous	74	3.39 (2.48)	3.95 (2.93)	4.20 (2.89)	0.39	.676
Harmful/Dependent	12	3.83 (2.48)	4.67 (2.46)	5.42 (3.87)		

Note: $^*p < .05$, $^{**}p < .01$

This exclusion yielded a total sample of 86 students. The final sample was divided into two groups based on the AUDIT categories; Safe/Hazardous and Harmful/Dependent. There were 74 students in the Safe/Hazardous group and 12 in the Harmful/Dependent group.

1.3 Neurological vulnerabilities

Using the ImPACT's demographic questions, information was obtained on Neurological vulnerabilities. These vulnerabilities are history of ADD/Hyperactivity, Learning disability, Psychiatric disorder, Grade repeated, Neurological disorder, Dyslexia and Concussion. Previous research has excluded participants on the grounds of some of these variables, and others, for example Brown et al. (2000) excluded participants if they did not speak English, had a psychiatric or neurological disorder or a history of concussion. Loeber et al. (2009) and Moriyama et al. (2006) also excluded participants who had neurological or psychiatric disorders. These variables all may have a negative effect on how the participants take the tests and thus may adversely affect the results of the test (Duff et al., 2007; Elbin et al., 2013; Iverson, 2006; Meyer & Sagvolden, 2006; Tlustos et al., 2010; Wolf et al., 2010).

In Table 3 below, Chi-squared test results are given for the criteria mentioned above, for each of the alcohol use groups.

Table 3: Neurological Exclusion Criteria Distribution across Alcohol Groups

Variables	Total (*n* = 86)	Safe/ Hazardous (*n* = 74)	Harmful/ Dependent (*n* = 12)	$\chi^2(1)$	*p*
	***n* (%)**				
ADD/Hyperactivity					
Yes	12 (13.95%)	11 (14.86%)	1 (8.33%)	0.02	.876
No	74 (86.05%)	63 (85.14%)	11 (91.67%)		
Learning disability					
Yes	7 (8.14%)	7 (9.46%)	0 (0.00%)	0.29	.587
No	79 (91.86%)	67 (90.54%)	12 (100%)		
Psychiatric disorder					
Yes	21 (24.42%)	20 (27.03%)	1 (8.33%)	1.07	.300
No	65 (75.58%)	54 (72.97%)	11 (91.67%)		
Grade repeated					
Yes	5 (5.81%)	4 (5.41%)	1 (8.33%)	0.07	.793
No	81 (94.19%)	70 (94.59%)	11 (91.67%)		
Neurological disorder					
Yes	4 (4.65%)	3 (4.05%)	1 (8.33%)	0.01	.931
No	82 (95.35%)	71 (95.95%)	11 (91.67%)		
Dyslexic					
Yes	1 (1.16%)	1 (1.35%)	0 (0.00%)	1.09	.295
No	85 (98.84%)	73 (98.65%)	12 (100%)		
Concussions					
Yes	17 (19.77%)	13 (17.57%)	4 (33.33%)	0.78	.378
No	69 (80.23%)	61 (82.43%)	8 (66.67%)		

Note: $^*p < .05$, $^{**}p < .01$

As can be seen from Table 3 the Neurological vulnerabilities are evenly distributed across the alcohol groups as none of the Chi-squared significance values were significant at the .05 alpha level. Thus the alcohol groups are matched on these variables. For this reason, the participants who meet these criteria were not excluded.

Another student was considered for exclusion, but it was ultimately decided not to exclude her. She sustained a concussion on the 26th of May 2012, between Interval 1 and Interval 2 of Phase 2. It resulted in confusion but not amnesia and no loss of consciousness. She had no history of concussions prior to this date. It was thus considered to have been a mild concussion. Her next testing date during data collection was on the 8th of August 2012; 74 days after her concussion diagnosis. According to Zuckerman et al. (2012), 90% of older adolescents (aged 18 to 22) will return to baseline cognitive and symptomatic functioning levels within 1 month of sustaining a concussion. Eisenberg, Andrea, Meehan and Mannix's (2013) research showed that those who had sustained previous concussions had a longer recovery period than those who had sustained no previous concussions. Those who had no previous concussions recovered within approximately 12 days. Thus, the student in question had ample time, over two months, to recuperate from her concussion and return to baseline levels before the ImPACT testing and was included in the data analysis. The Final sample therefore consists of 86 students.

1.4 Control Variables

Previous research has made use of control variables, such as age, gender, initial intelligence and certain neurological vulnerabilities, which were matched across different alcohol groups (Bednarski et al., 2012; Britton et al., 2004; Loeber et al., 2009; Mann et al., 2005; Smith & Fein, 2010; Tapert et al., 2002). The control variables considered for this research include: Gender, Race groups, English first language, Neurological vulnerability presence, PPVT-4 raw score, PPVT-4 standard score, Age in years and Years at Rhodes. The Neurological vulnerability presence variable indicates the presence of any one of the Neurological vulnerability criteria in the previous section. The Years at Rhodes variable gives an indication of full years at Rhodes University and education length.

Chi-squared tests were performed on Gender (female and male), Race groups (Black and white), English first language (English and other-than-English), Neurological vulnerability presence (yes and no). The Black race group consists of black African, Indian and coloured students. The other-than-English group consists of the all those students who do not speak English as a first language. The Chi-squared test results are shown in Table 4.

Table 4: Chi-squared Test Results for Control Variables

Variables	**Totals (*n* = 86)**	**Safe/ Hazardous (*n* = 74)**	**Harmful/ Dependent (*n* = 12)**	**$\chi^2(1)$**	***p***
Gender					
Female	68 (79.07%)	60 (81.08%)	8 (66.67%)	0.57	.450
Male	18 (20.93%)	14 (18.92%)	4 (33.33%)		
Race groups					
†Black	28 (32.56%)	26 (35.14%)	2 (16.67%)	0.87	.350
White	58 (67.44%)	48 (64.86%)	10 (83.33%)		
English first language					
English	67 (77.91%)	57 (77.03%)	10 (83.33%)	0.01	.910
Other-than-English	19 (22.09%)	17 (22.97%)	2 (16.67%)		
Neurological vulnerability presence					
Yes	40 (46.51%)	34 (45.95%)	6 (50.00%)	0.00	.960
No	46 (53.49%)	40 (54.05%)	6 (50.00%)		

Note: **p* < .05, ***p* < .01
† Importantly, the terms black African, coloured, Indian and white are used in this research, as they were in the most recent 2011 census, where the demographics of South Africa encompassed about 52 million people of diverse origins, cultures, languages, and religions (Lehohla, 2012). Currently the term 'Black' is preferred when making a general reference to those race groups in the country that historically were referred to as 'non-white' (Bowman et al. 2006). Therefore, the term 'Black' has been applied in this thesis with reference to the sub-groups of black African, coloured and Indian individuals included in the study.

Independent *t*-tests were performed on the PPVT-4 raw scores, PPVT standard scores, Age in years and Years at Rhodes control variables. The results are shown in Table 5 below.

Table 5: Independent *t*-test Results for Control Variables

	M (SD)			
	Safe/Hazardous	**Harmful/Dependent**		
Variables	**(*n* = 74)**	**(*n* = 12)**	***t*(84)**	***p***
PPVT-4 raw	203.77 (10.95)	199.58 (10.25)	-1.24	.219
PPVT-4 standard	105.05 (11.20)	99.75 (11.72)	-1.51	.134
Years at Rhodes	2.65 (0.83)	2.92 (1.16)	0.97	.333
Age in years	19.96 (0.99)	20.33 (1.37)	1.15	.253

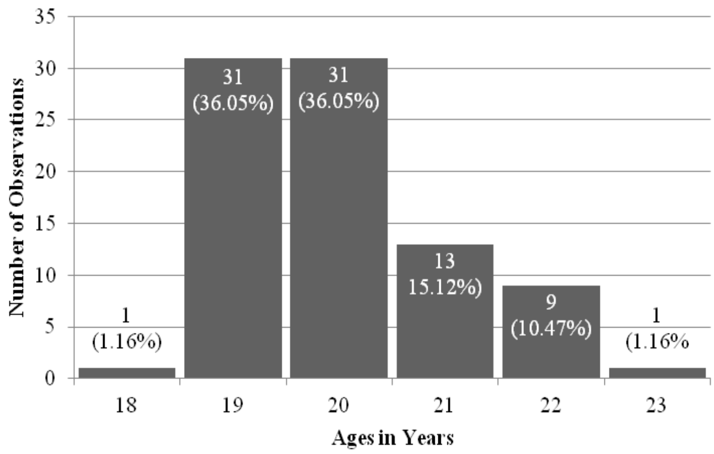
Note: $*p < .05$, $**p < .01$

The Chi-squared test and independent *t*-test results show that the alcohol groups are matched for all of the control variables: Gender, Race groups, English first language, Neurological vulnerability presence, PPVT-4 raw score, PPVT-4 standard score, Years at Rhodes and Age in years.

1.5 Sample characteristics

The Final sample consisted of 86 students, of which 68 were female (79.07%) and 18 were male (20.93%). The average age of the students was 20.01 years with a standard deviation of 1.05. The minimum age was 18 years and the maximum was 23 years. Figure 2 shows the distribution of the ages across the sample.

Figure 2: Age distribution for Final Sample



The racial distribution of the sample was 58 white (67.44%) and 28 Black (32.56%). The Black students consisted of 21 black African students (24.42%) and smaller numbers of coloured (4, 4.65%) and Indian (3, 3.49%) students. The Nationality breakdown is shown in Table 6. The main groups (South Africa, Other African Countries and Other World Countries) are shown in bold, while the countries that make up the main groups are shown underneath the group name, in descending *n* order.

Table 6: Nationality Breakdown for Final Sample

Countries	***n***	**%**
South Africa	**69**	**80.23%**
Other African Countries:	**13**	**15.12%**
Zimbabwe	10	11.63%
Namibia	2	2.33%
Botswana	1	1.16%
Other World Countries:	**4**	**4.65%**
Britain	3	3.49%
Germany	1	1.16%

The First Language breakdown is shown in Table 7. The main groups (English and Other-than-English) are in bold. The groups that make up the Other-than-English group are shown in descending *n* order underneath the main group name.

Table 7: First Language Breakdown for Final Sample

Countries	*n*	%
English	**67**	**77.91%**
Other-than-English:	**19**	**22.09%**
Zulu	7	8.14%
Xhosa	4	4.65%
German	2	2.33%
Shona	2	2.33%
Ndebele	1	1.16%
Sepedi	1	1.16%
Setswana	1	1.16%
Sotho	1	1.16%

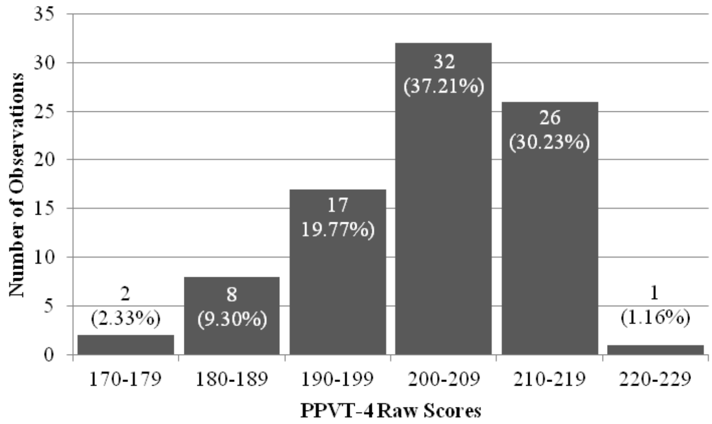
The students' high school education distribution is as follows: 38 attended Private high schools (44.19%), 31 attended Government high schools (36.05%) and 17 attended other high schools (19.76%). As stated above, other high schools are those in other countries.

At the time of the research, the greatest percentage of students had attended Rhodes University for at least one full year of academic study (98.84%) while only one student had started at Rhodes University in 2012 (1.16%). The mean time spent at Rhodes University was 2.69 years with a standard deviation of 0.88. The maximum time registered as a student at Rhodes University was four full academic years.

During the research, participants resided in the university residences (54, 62.79%), digs (29, 33.72%), at home (2, 2.33%) or at one of the schools in Grahamstown (1, 1.16%). Digs are flats, apartments and houses, not owned by the university, rented by students. Some students work part time at the schools in Grahamstown and stay in the dormitories with the school students.

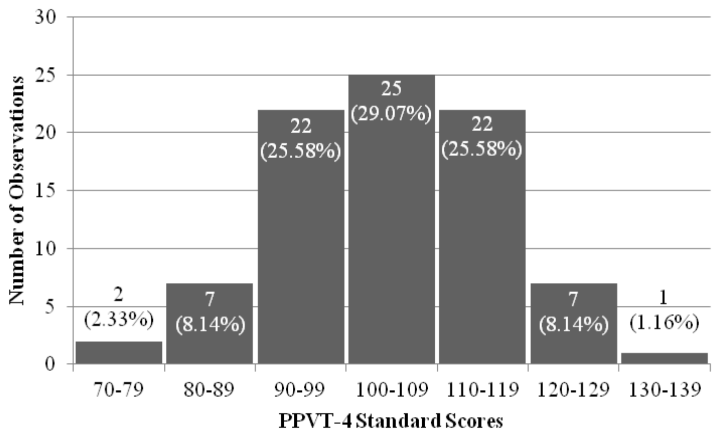
The average PPVT-4 raw score was 203.19 with a standard deviation of 10.89. The minimum raw score was 171 and the maximum was 221. The distributions of the raw are shown in Figure 3.

Figure 3: PPVT-4 Raw Scores for Final Sample



The average PPVT-4 standard score was 104.31 with a standard deviation of 11.35. The minimum standard score was 78 and the maximum was 132. The distributions of the standard scores are shown in Figure 4.

Figure 4: PPVT-4 Standard Scores for Final Sample



As stated, the sample was divided into two alcohol groups for analyses (Safe/Hazardous and Harmful/Dependent). Table 8 shows the distribution of the sample between the traditional AUDIT categories (Safe, Hazardous, Harmful and Dependent) as well as the alcohol use groups used for the analyses (Safe/Hazardous and Harmful/Dependent).

This grouping was done because, according to Babor et al. (2001), the drinkers classified as Safe or Hazardous have not experienced harms related to their alcohol consumption (see Section 8.1 in the Literature Review, p. 82). It was on this assumption, that the researcher did not expect to find any harmful effects, cognitive or symptomatic, related to alcohol consumption categorised as Safe or Hazardous by the AUDIT. Thus it was decided to combine the two groups that were unlikely to have experienced alcohol-related cognitive and symptomatic harms. This formed the Safe/Hazardous group which consisted of 74 students (86.05%). The Harmful and Dependent groups were expected to have experienced alcohol-related cognitive and symptomatic harms, and it was decided to combine these into one group. This formed the Harmful/Dependent group which included of 12 students (13.95%). These figures are similar to those found in two previous Rhodes University studies by Young and de Klerk (2008) and Young and Mayson (2010) (see Section 4.2.2, p. 12 of the Literature Review Chapter).

Table 8: Alcohol Group Distribution

Alcohol groups	***n***	**%**
Safe/Hazardous	**74**	**86.05%**
Safe	34	39.53%
Hazardous	40	46.51%
Harmful/Dependent	**12**	**13.95%**
Harmful	8	9.30%
Dependent	4	4.65%

Along with the alcohol groups, the sample was also analysed using various moderating substance related variables (First drunk, First age, Drink, Hangover, Pass out, Parental alcohol use, Smoking and Substance use). The distributions of these variables for the total sample and for the alcohol groups are shown in Table 9. Of note, there were empty categories that contained either no Safe/Hazardous drinkers or no Harmful/Dependent drinkers. These are displayed using a zero count and zero percentage. If a category had no participants, that category was not included in this table. For example, in the First age variable, the Primary school category was empty and thus not included in the table.

Table 9: Substance related Variable Distributions

Substance related variables	Safe/Hazardous		Harmful/Dependent		Total	
	n	%	*n*	%	*n*	%
First drunk						
Never	12	16.22%	0	0.00%	12	13.95%
Primary School	3	4.05%	2	16.67%	5	5.81%
High School	38	51.35%	8	66.67%	46	53.49%
Over 18	21	28.38%	2	16.67%	23	26.74%
First age						
Never	15	20.27%	0	0.00%	15	17.44%
High School	14	18.92%	5	41.67%	19	22.09%
Over 18	45	60.81%	7	58.33%	52	60.47%
Drink						
Never	7	9.46%	0	0.00%	7	8.14%
Wine	23	31.08%	1	8.33%	24	27.91%
Spirits	5	6.76%	0	0.00%	5	5.81%
Beer	4	5.41%	5	41.67%	9	10.47%
Mixture	26	35.14%	6	50.00%	32	37.21%
Other	9	12.16%	0	0.00%	9	10.47%
Hangover						
Abstain	7	9.46%	0	0.00%	7	8.14%
Not in year	2	2.70%	0	0.00%	2	2.33%
Never	16	21.62%	0	0.00%	16	18.60%
Less than monthly	27	36.49%	2	16.67%	29	33.72%
Monthly	15	20.27%	3	25.00%	18	20.93%
Weekly	7	9.46%	7	58.33%	14	16.28%

Substance related variables	**Safe/Hazardous**		**Harmful/Dependent**		**Total**	
	n	**%**	***n***	**%**	***n***	**%**
Pass out						
Abstain	7	9.46%	0	0.00%	7	8.14%
Not in year	2	2.70%	0	0.00%	2	2.33%
Never	52	70.27%	4	33.33%	56	65.12%
Less than monthly	12	16.22%	5	41.67%	17	19.77%
Monthly	1	1.35%	2	16.67%	3	3.49%
Weekly	0	0.00%	1	8.33%	1	1.16%
Parental alcohol use						
Abstain	18	24.32%	3	25.00%	21	24.42%
Light	52	70.27%	9	75.00%	61	70.93%
Heavy	4	5.41%	0	0.00%	4	4.65%
Smoking:						
Yes	15	20.27%	7	58.33%	22	25.58%
No	59	79.73%	5	41.67%	64	74.42%
Substance use						
Yes	5	6.76%	1	8.33%	6	6.98%
No	69	93.24%	11	91.67%	80	93.02%

Section 2: Data collection

This section will cover the measures used to collect the data at the various testing sessions as well the procedure followed to collect the data. This research made use of a quasi-experimental design with a purposive sampling technique (Terre-Blanche, Durrheim, & Painter, 2006). It is considered a quasi-experimental design as there was no randomised assignment to different alcohol use groups, but rather assignment to the groups on the basis of the AUDIT scores and categories.

2.1 Measures

2.1.1 Alcohol use test

a) Alcohol Use Disorders Identification Test (AUDIT)

The AUDIT was administered once in Phase 1 in order to get a baseline measure of the sample's alcohol use patterns. It was administered twice in Phase 2 (Intervals 2 and 3) in order to determine if the students changed their alcohol use drastically over the course of the year.

History and Previous research

The AUDIT is an internationally used questionnaire that was developed by the World Health Organisation in 1982 for the screening of hazardous use of alcohol and alcohol-related disorders (Babor et al., 2001). In test development, the questions were selected based on their correlations with daily alcohol intake, frequency of binge drinking and their ability to distinguish between drinkers whose patterns of alcohol use may lead to future physical and mental health problems and those whose drinking has already caused physical and mental health problems (Allen, Litten, Fertig & Babor, 1997).

The AUDIT has been effectively used in university settings around the world (Miles et al., 2001; Reinert & Allen, 2007; Rickwood et al., 2011; Rodgers et al., 2005; Shuckit, 2009; Windle & Zucker, 2010). It has been used successfully in South African contexts (Peltzer et al., 2011) and specifically at Rhodes University by Young and de Klerk (2008), Young and Mayson (2010) and Young and de Klerk (2012). The AUDIT has also been used in cognitive functioning assessment research to separate participants into drinking categories (Bednarski et al., 2012; Dawson et al., 2007; Montgomery et al., 2012; Parada et al., 2012; Rodgers et al., 2005).

Psychometric Properties

According to Babor et al., the AUDIT "has been found to provide an accurate measure of risk across gender, age, and cultures" for hazardous drinking (2001, p. 10). The measure has a high internal consistency with a reliability coefficient of between .75 and .97 according to Reinert and Allen's (2007) review of 18 studies. Furthermore, the AUDIT is a reliable and valid tool in the detection of hazardous alcohol use and the measurement of alcohol use patterns. Young and Mayson (2010) calculated the reliability coefficient of the AUDIT to be .83 for their multi-racial university student sample at the same institution as the present study, which is equal to the mean reliability coefficient from Reinert and Allen's (2007) literature review. Similarly, Kokotailo et al. (2004) researched the psychometric properties of the AUDIT and its use in college students. The Cronbach's alpha reliability coefficient was found to be .81, also similar to Young and Mayson's (2010) figure. Kokotailo et al. (2004) found that the AUDIT has reasonable psychometric properties in college students and displayed a sensitivity of 91.0% with regards to a Safe cut-off score of 6 and a specificity of 60.0% in detecting high-risk drinkers. Thus the AUDIT has shown adequate psychometric properties across a variety of university settings and with a variety of test subjects. As such, it is an appropriate measure of alcohol consumption patterns and problems for the present study.

Administration and Scoring

The questionnaire is easy to administer and complete, requiring very little time to complete and score. It comprises ten questions that quantify different aspects of alcohol use (Babor et al., 2001). Questions one, two and three measure hazardous alcohol consumption, questions four, five, and six measure the symptoms of alcohol dependence and questions seven to ten measure harmful effects of heavy alcohol use. The AUDIT questions each give a score of between 0 and 4, which are added together to produce a maximum total AUDIT score out of 40. These scores are classified into the following categories: Safe drinking, Hazardous drinking, Harmful drinking and Dependent drinking.

According to the AUDIT manual, participants who score between 0 and 7 are considered to be Safe drinkers and experience no negative consequences (Babor et al., 2001). This Safe drinking cut-off score of 7 has been contested in recent years, specifically for females. Reinert and Allen's (2007) research showed that females are more sensitive to alcohol's effects and thus more susceptible to its harms. Those researchers suggested that a lower Safe drinking cut-off score should be used for females. The Safe cut-off scores suggested by Reinert and Allen (2007), and used in the current research, are 7 for males and 6 for females. Participants are considered Hazardous drinkers when their scores are between the Safe cut-off score (7 for females and 8 for males) and 15, inclusive (Babor et al., 2001). According to Babor et al. (2001), hazardous drinkers have not experienced alcohol-related harms yet but hazardous drinking increases the risk of harmful effects to the drinker as well as to others; for example, physical and emotional harm to the drinker and criminal damage to the property of others.

The higher categories' cut-off scores were not altered as currently no recommendation exists to do so. Participants are considered Harmful drinkers when their scores fall between 16 and 19, inclusive. This range indicates that their drinking has resulted in harm to the mental, physical and social health of themselves and others. Those who obtain a score of 20 or more are considered to be dependent on alcohol. The dependent drinkers experience various cognitive, physical and behavioural dependence symptoms related to their alcohol use, such as a strong urge to consume alcohol and withdrawal symptoms when alcohol is not consumed over a period of time.

As stated above in Section 9 of the Literature Review chapter (see p. 87), the AUDIT categories were combined into two alcohol groups for statistical analyses: Safe and Hazardous as one group (Safe/Hazardous) and Harmful and Dependent as another (Harmful/Dependent). The Safe/Hazardous group had scores that fell between 0 and 15 on the AUDIT, while the Harmful/Dependent group scored between 16 and 40. This grouping was used because Safe and Hazardous drinkers have not experienced alcohol-related harms yet (Babor et al., 2001), thus the researcher did not expect to find evidence of harmful cognitive and symptomatic effects in the Hazardous group. It is on that assumption that the Hazardous group was then combined with the Safe group for the purposes of this study. The Harmful and Dependent groups were expected to have experienced negative consequences due to their alcohol use, thus, those two groups were clustered together.

2.1.2 Cognitive and Symptomatic Tests

The cognitive and symptomatic tests used in this research, with a view to investigate the neurocognitive effects in association with alcohol use, are the paper and pencil Peabody Picture Vocabulary Test version 4 (PPVT-4) and the computerised Immediate Postconcussion Assessment and Cognitive Test version 6 (ImPACT).

a) The Peabody Picture Vocabulary Test Version 4 (PPVT-4)

The PPVT-4 was administered only once, in Phase 1 of the study, in order to establish a comparative level of initial intelligence across the comparative groups. Vocabulary tests, such as the PPVT-4, are not expected to be lowered in association with the diffuse brain injury effects of a neuropathological condition such as excessive alcohol use (Lezak et al., 2004). Vocabulary tests are considered to be ‘hold’ tests and therefore suited to being used as tests of initial (i.e., pre-morbid) intelligence. Typically the test would not need to be repeated, on the assumption that it would not change significantly over the data collection period of one year, and therefore is only administered once, at the baseline test phase.

History and Previous research

The first edition of the PPVT came out in 1959 and was specifically focused on children, but since then it has undergone various changes through the versions, from PPVT to PPVT-R, PPVT-3 and PPVT-4 (Dunn & Dunn, 2007). The fourth edition can be administered to subjects from the age of 2 years and 6 months up to and over the age of 90 years. The fourth and latest edition (PPVT-4) was published in 2007. As mentioned in the Literature Review chapter (see Section 8.2, p. 84), research has shown that the PPVT-4 has been used to determine initial intelligence successfully in previous research (Borovsky et al., 2012; Glenwright & Agbayewa, 2012; Stevens et al., 2011; Tlustos et al., 2010). Previous versions of the PPVT have been used successfully in South African contexts (Alant & Beukes, 1986; Olivier, 2009; Pakendorf & Alant, 1997).

Psychometric Properties

According to Dunn and Dunn (2007), the PPVT-4 has a mean internal consistency split half reliability of approximately .94 and alpha reliability of approximately .97 for Form A. The PPVT-4 has a mean alternate-form reliability of approximately .89 and a mean test-retest correlation of .93. Gray, Plante, Vance and Henrichsen (1999) researched the diagnostic accuracy of four vocabulary tests, including the PPVT-3. The results showed that the PPVT-3 had a sensitivity of 74% and a specificity of 71%. The PPVT-4 manual does not state the specificity or sensitivity values for the current test and no study was found to state these values for the PPVT-4.

Administration and Scoring

Each of the forms, A and B, has 228 test items, sorted into 19 sets with 12 items in each set (Dunn & Dunn, 2007). Each test item is numbered and consists of a vocabulary word and a picture plate displaying 4 pictures. The pictures are in full colour. Traditionally, the examiner says the word out loud and asks the examinee to either point to the relevant picture or say the letter corresponding to the picture. The examiner then records the answer on an answer sheet and moves onto the next item until a ceiling level has been established. According to Dunn and Dunn (2007), the ceiling level is reached when the examinee has answered eight words out of the set incorrectly. The sets are sorted with an increasing level of difficulty corresponding with different age groups. Depending on the age of the examinee, the examiner starts at a different set. This research sample focused on students between the ages of 18 and 26 years. Consequently, this research started with Set 13, for ages 17-18 and continued until set 19, which is the end of the test. This was to make sure that a strong baseline and ceiling level were identifiable for each participant. Form A was used for this research.

Since there has been research that has used the PPVT in group settings effectively (Guthrie & Seifert, 1977; Norris et al., 1960; Tillinghast et al., 1968) and the initial sampling pool of students was large, the PPVT-4 was used in group form using Microsoft PowerPoint. While the group administration deviates from the standard individual administration of the test, the purpose of using the test was for group comparative purposes, and not for the identification of an IQ level in itself. The comparative groups were given the identical group administration version of the test, thereby validating this deviation in administration procedure for the group research purposes. Each picture plate was scanned and then placed on a PowerPoint slide, with the number of the picture slide and the corresponding word on top of the slide. The word was placed on the slide, as well as spoken aloud, so as to counteract the effects of mispronunciation of the researcher and mishearing of the students. The students were given an answer form with tables corresponding to the picture slides (attached in the booklet in Appendix 1).

The scoring of the PPVT-4 involves the examiner computing the total number of wrong answers in each set (Dunn & Dunn, 2007). If the number of wrong answers in a given set is more than eight, that set is considered the Ceiling set. The number of wrong answers up to and including those in the ceiling set is then subtracted from the number of the last item of the Ceiling set to produce the raw score. For example, a participant in the research scores 3 out of 12 for Set 15. The Ceiling set would be Set 15. The last item of the set is item number 180. The participant scores 8 out of 12 for set 14 and 12 out of 12 for set 13. The total number of errors would be 13. The participant would have a raw score of 180 minus 13 which is 167.

This raw score can be converted into a standard score by means of norm tables. "A standard score indicates the distance of the examinee's raw score from the average for people of the same age" or grade (Dunn & Dunn, 2007, p. 17). The PPVT-4 has two sets of norm tables, which are normed by either student grade or by age. This research made use of the age normed tables because the grade normed tables refer to American grades which are not consistent with South African school grades and the grade normed tables do not extend to university. To use the age normed tables, the age needs to be calculated to the year and month. The participants' ages were calculated from the month of the test, which was taken in March 2012. The PPVT-4 uses a normal distribution for the standard scores, thus the mean standard score is 100 with a standard deviation of 15. The PPVT-4 was normed using representative samples in America closely matched for Sex, Race, Socio-Economic Status, Geographic region and Education. Since the PPVT-4 was normed in America, and South Africans have been seen to score lower on American tests, and previous versions are thought to underestimate verbal abilities, a low standard score of 75 was used as a cut-off for this research.

b) Immediate Postconcussion Assessment and Cognitive Test (ImPACT)

The ImPACT test was used over the three repeat testing intervals in Phase 2 of the study to measure the participants' cognitive and symptomatic functioning and any changes in these that may have occurred over the year.

History and Previous research

The first version of the ImPACT was produced as a downloadable program in America (ImPACT, 2011). It was introduced as a research edition in 1998 and after its clinical utility was tested, it was made commercially available to some universities and high schools in 2000. Version 2.0 included a Design Memory module that provided data on visuospatial and recognition memory abilities. Version 2.0 was introduced in 2001 and until 2007 no major changes to the test stimuli and presentation were made. In 2007, version 6 was presented as both a desktop version, as before, and as an online version. Version 6 is the current version and the online version was utilised in this research. The test was administered in English, but is available in 20 languages including Afrikaans. Small updates have been done on this version since its introduction. For example, a Cognitive Efficiency Index score was added to the composite scores (Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Impulse Control) to provide information on the effort that the participant put into the test. The test producers also included a validity index which helped to identify invalid baseline tests. The validity index is based on an algorithm concerning individual scores from the tests. The report states whether the test result should be considered valid or not.

The current version gives composite scores for Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time, Impulse Control and Cognitive Efficiency Index as well as a Total Symptom score (ImPACT, 2007). It has been used primarily in concussion diagnosis and management. In that the ImPACT battery is made up of test modalities that show a high level of sensitivity to the diffuse brain injury effects of concussion, these modalities can also be expected to show sensitivity to other neuropathological conditions with diffuse brain injury consequences. Therefore the use of the test has expanded beyond being used purely in the concussion arena, including being used in research on depression, learning disorders, diagnosis of minimal hepatic encephalopathy and brain tumours (Conklin et al., 2013; Elbin et al., 2013; Iverson, 2006; D. G. Thomas et al., 2011; Tsushima et al., 2013). As it has been used in areas other than for concussion management, and is sensitive to diffuse brain injury, it will be used as a measure of cognitive and symptomatic functioning, and changes in these, in this research.

Psychometric Properties

The ImPACT battery has been approved for clinical use in South Africa (Health Professions Council of South Africa, 2010). It has been shown to be valid and reliable in terms of its use in concussion management in America (Broglio et al. 2007; Fazio et al., 2007; van Kampen et al., 2006) as well as in South Africa (Clark, 2010; Shuttleworth-Edwards et al., 2008; Shuttleworth-Edwards, Radloff et al., 2013).

The ImPACT test-retest intervals have been shown to be reliable when compared to paper and pencil tests measuring similar cognitive functioning constructs (Iverson, Lovell, & Collins, 2002). Iverson et al. (2002) calculated Cronbach's Alpha as a measure of reliability and internal consistency. The Alpha value ranged from .88 to .94 for the sample, with a mean value of .93. Schatz and Sandel (2013) researched the sensitivity and specificity of the online ImPACT test, and reported statistics of 91.4% and 69.1% respectively.

Administration and Scoring

The ImPACT uses six test modules to measure neurocognitive functioning (ImPACT, 2007). The modules are Word memory, Design memory, X's and O's, Symbol match, Colour match and Three letters. There are also distracter tasks in between some of the tests, such as a timed task requiring the participant to click on numbers backwards from 25 to 0. The test takes approximately 30 minutes to complete. Rather than giving an overall cognitive score, which might obscure deficits in particular cognitive domains, the program computes a differentiated set of indicators of possible dysfunction. It reports the scores for each test and five composite scores for Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Impulse Control. The Verbal Memory composite score is a measure of verbal attention, learning and memory. It is computed using the Word memory, Symbol match and Three letters modules. The Visual Memory composite score is a measure of visual learning, memory, attention and scanning. It is calculated using Design memory and X's and O's. The Visual Motor Speed composite score is a measure of visual motor response speed, processing, learning and memory. It is based on scores from the X's and O's and Three letters modules. The Reaction Time composite score is a measure of average response speed in milliseconds and is established using the X's and O's, Symbol match and Colour match modules. The Colour match module is a choice reaction time task. The Impulse Control composite score is a measure of errors on the X's and O's and Colour match tests.

Along with those composite and individual test scores, the report gives the composite score for Cognitive Efficiency Index. This score gives an indication of the effort that the participant put into testing. It is a measure of the interaction between speed and memory accuracy, specifically whether the participant sacrificed their accuracy by working too quickly on the Symbol Match test or sacrificed their speed by working slowly on the test to maximise their accuracy. The ImPACT report states that the Cognitive Efficiency Index scores normally range between 0 and .70. The participant performed well in both speed and memory on the Symbol Match test if the score is higher than the mean score of .34. If the score is lower than .20, the participant scored poorly on both speed and memory accuracy. It is also possible for the score to be negative, in which case, the participant did badly in the reaction time component.

The ImPACT program also inquires about the symptoms a person is experiencing, in the Postconcussion Symptom Scale. The symptoms are generally concussion-related but can be related to general symptomatic functioning as well. As stated in the Literature Review (see Section 5.2.3, p. 38 on Alcohol consequences and Section 5.2.2, p. 35 on Hangover symptoms), some symptoms that are related to excessive alcohol use are balance, headaches, nausea, vomiting, dizziness and numbness or tingling. These, among others, are in the Postconcussion Symptom Scale. The full symptom list is shown in Table 10. The symptom scale takes the form of a Likert scale. The participant states whether they have experienced the symptom in the previous 24 hours, in giving an answer from 0 to 6, where 0 indicates that they have not experienced the symptom and 6 indicates they have been severely affected by the symptom. The test report gives the individual symptom scores and a total symptom score along with the cognitive scores.

Table 10: ImPACT Postconcussion Symptom Scale

Headache	Noise sensitivity
Nausea	Irritability
Vomiting	Sadness
Balance Problems	Nervousness
Dizziness	Feeling more emotional
Fatigue	Numbness or tingling
Trouble falling asleep	Feeling slowed down
Sleeping more than usual	Feeling mentally foggy
Sleeping less than usual	Difficulty concentrating
Drowsiness	Difficulty remembering
Light sensitivity	Visual problems

The cognitive scores used for analyses in this research are Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Cognitive Efficiency Index. Impulse Control was not included as it was used as an exclusion criterion. All of the Postconcussion Symptom Scale scores were used in the analyses.

2.2 Procedure

This section will deal with the ethical considerations taken in this research as well as the data collection procedures used in both Phase 1 and Phase 2 of this study.

2.2.1 Ethical Considerations

a) Approval from relevant ethics committees

At the end of 2011, ethical clearance was obtained from the Rhodes University Psychology Department's Research Projects and Ethics Review Committee. At the beginning of the 2012 academic year, permission was obtained from the Rhodes University Registrar and the Head of the Rhodes University Psychology Department to recruit Psychology 2 and 3 students as research participants.

b) Informed Consent

The participants in this research were required to sign and date two consent forms for the two phases of the research. These are included in Appendices 2 and 3. The forms detailed the reasons for the research and the contact details for the researcher and supervisors. The forms included information regarding their participation roles and stated that they could withdraw from the study at any time with no negative consequences. The forms also stated that the de-indentified data from the tests may be used in future research and publications.

c) Privacy and Confidentiality

As most of the tests were administered face-to-face with the researcher, anonymity could not be guaranteed for the participants, however confidentiality was ensured by only the researcher and supervisors having access to any identifiable information. If the students took part in the second phase of the research, a coding system was implemented where they were assigned random names to input into the ImPACT program. This ensured that the students' participation and data remained confidential and that their progress could be tracked through the year. Only the researcher and supervisors had access to the data linking the student numbers and assigned names. The ImPACT data was stored online and accessible only through a password protected website. As there is no way for the general population to link the results of this research to individual students, overall anonymity and confidentiality for the students was ensured.

d) Risks and Benefits

The risks that the participants may have experienced as a result of this research were minimal. The participants may have been alerted to existing problems as a result of completing the tests, for example, they may realise that their alcohol use is higher than they previously thought, as a result of answering the AUDIT questions. The students may also have had difficulty in answering and completing the cognitive tests which may have resulted in distress. The participants were informed that if they experienced any distress, they were free to withdraw from the test and/or talk with the researcher and supervisors concerning a pre-arranged list of counselling services.

The benefits to the students include an incentive for taking part in the research, discussed in Section 2.2.1 e) below, and also exposure to a comprehensive cognitive test battery. The exposure is a benefit because it may enable them to learn more about themselves as well as gain experience in cognitive testing. As Psychology students, they may be interested in cognitive tests such as those used in this research.

e) Incentive

Incentive for participating in the first phase of the research was given in the form of entry into random prize draws of R1000 for the Psychology 2 students and Psychology 3 students. Entry into another random prize draw was used for the second phase of the research, where two students would win R1000 each. As a minor incentive for each of the testing intervals in May, August and October 2012, the students were offered a small chocolate on completion of the test.

f) Test availability and Funding

The PPVT-4 is freely available to Rhodes University students for academic use through the Rhodes University Psychology Clinic. The AUDIT is freely available on the Internet. The ImPACT battery developers granted free access to the ImPACT battery specifically for this research for the period of one year (the 2012 academic year), during which time the data was collected. The research was largely funded by the private funds of the researcher and additional funding was supplied by Professor Ann Edwards' NRF research grant.

2.2.2 Phase 1

Psychology 2 and Psychology 3 lecturers were approached at the beginning of the 2012 academic year and permission was obtained to access the students during an arranged lecture time. All the second and third year Psychology students were invited to attend one of three special lectures where they were asked to participate in a Masters Research project. The special lectures were advertised on the second and third year Psychology notice boards and were announced in emails. The students were told that if they completed the surveys and test they would be entered into a random prize draw for R1000 as an incentive to participate in the research. There was one prize for the second years and another for the third years.

In the special lecture, the students received a booklet containing a consent form explaining the purpose of the study, a form for answering the PPVT-4, a biographical questionnaire and the AUDIT. This booklet is attached in Appendix 1. The students were also invited to take a separate page explaining the research in full and contained the contact details of the researcher and supervisors (Appendix 4). At the end of the consent form, the students were asked if they wanted to participate in the second phase of the research. The students were then told how to fill in the PPVT-4 form and the PowerPoint presentation was shown to the students. The PowerPoint presentation started with four practice examples which were not included the students' final scores. After the test, they were asked to fill in the biographical questionnaire and the AUDIT. A total of 216 students completed the first testing phase.

2.2.3 Phase 2

Consenting students, who completed Phase 1 and who were not excluded using the criteria in Section 1.2.1 (p. 94), were emailed and asked to book a testing slot for Phase 2 of the research. These students were told that if they completed all three testing intervals of Phase 2 of the research they would be entered into another random prize draw where another two students would win R1000 each. They were notified that the testing would take place in the evenings, so as not to conflict with lectures, or they could book an alternate time slot if they could not attend an evening slot. These participants were informed that the test would take approximately 45 minutes.

Each testing session required the students to obtain a testing booklet from the researcher which detailed the test procedure and included additional questions. In the first testing session of this phase the students were required to sign a second consent form. The testing booklets for each interval are attached in Appendices 5, 6 and 7. The students were asked to follow the procedure notes in the booklets and the instructions onscreen in the ImPACT program to complete the online ImPACT battery. If they required additional information or assistance, they were to raise their hand and wait silently until the researcher came to them. They were asked to complete the test in silence so that they would not disturb any of the other participants. Once they had completed the ImPACT, they were asked to complete the additional questions in the booklet and return them to the researcher.

The ImPACT requires that the test taker input a name, so that the tests from one person can be saved together. In order for the participants to remain confidential, random names were assigned to each test taker. Each participant had the same name for all three testing intervals so that changes could be tracked through the year. The data for the test takers from this research were stored on the ImPACT online system, for ease of access for the researcher.

2.3 Variables

The independent variable is alcohol use behaviour and the dependent variables are the students' cognitive and symptomatic functioning over the three time intervals. These are listed in Table 11. The dependent variables were obtained from the ImPACT reports for each participant for each interval and are continuous variables.

Table 11: Variables

Dependent ImPACT Cognitive Scores
Verbal Memory, Visual Memory, Visual Motor, Reaction Time, Cognitive Efficiency Index
Dependent ImPACT Symptom Scores
Total Symptom Score, Headache, Nausea, Vomiting, Balance, Dizziness, Fatigue, Difficulty falling asleep, Sleeping more than usual, Sleeping less than usual, Drowsiness, Light sensitivity, Noise sensitivity, Irritability, Sadness, Nervousness, Emotional, Numbness, Mental Slowness, Fogginess, Difficulty concentrating, Difficulty remembering, Visual problems
Main Independent Variable
Alcohol groups
Moderating Substance Related Variables
First drunk, First age, Drink, Hangover, Pass out, Parental alcohol use, Smoking, Substance use

Considering the expected change in alcohol use over the year (see Section 1.2.2 a), p. 95), the alcohol groups obtained from Phase 1 were chosen for the analyses. The Phase 1 alcohol groups were expected to be unrestrained drinking behaviours as they were measured at the beginning of the year when there is expected to be less stress as the students had just returned from December holidays. The alcohol groups analysed were Safe/Hazardous and

Harmful/Dependent (see Section 1.5, p. 106 for discussion on alcohol groups and AUDIT categories).

The substance related variables were obtained from additional questions that the students answered during the research. The answer options for the questions are shown in Table 12. The First drunk variable states when the participant first experienced a drunken episode. The First age variable describes when the participants first started consuming alcohol regularly. The Drink variable describes what type of alcoholic drink the participant consumes most often. The Hangover variable describes how often the participants experience a hangover after drinking. The Pass out variable describes how often the participants lost consciousness, or passed out, due to excessive alcohol consumption. The Parental alcohol use variable describes the alcohol use patterns of the parents of the participants, as described by the participants. The answers in Table 12 for Parental alcohol use were combined into Abstain, Light (Light and Moderate) and Heavy (Heavy and Alcoholic) for analysis. The Smoking variable indicates whether the participant smokes cigarettes or not. The Substance use variable stated whether the participants uses any illegal substances or not, as reported by the participant.

Table 12: Substance Related Variables Answer Options

Variables and Possible answers
First drunk
Never, Primary, High school and Over 18
First age
Never, Primary, High school and Over 18
Drink
Never, Beer, Wine, Spirits, Mixture and Other
Hangover
Abstain, Never, Not in year, Less than monthly, Monthly, Weekly and Daily
Pass out
Abstain, Never, Not in year, Less than monthly, Monthly, Weekly and Daily
Parental alcohol use
Abstain, Light, Moderate, Heavy and Alcoholic
Smoking
Yes and No
Substance use
Yes and No

Section 3: Data Analysis

STATISTICA version 10 was used to analyse the data from this research. The data set was analysed using descriptive statistics, Chi-squared tests (with Yates' correction when necessary), independent *t*-tests, repeated measures ANOVA tests and Tukey HSD multiple comparison tests, when necessary. Kolmogorov-Smirnov Normality distribution tests were run on the data, as were Levene's Tests for Homogeneity of Variance. The results from these confirmed that the main variables were normally distributed but in some cases, not homogenous in terms of variance.

3.1 Null Hypothesis

The significance level is the probability that the null hypothesis is rejected when it is actually true (Tredoux & Durrheim, 2008). The null hypothesis states that there is no difference between the groups being tested. Type I errors occur if the null hypothesis is rejected when it is true while Type II errors occur if the null hypothesis is accepted when it is false. The significance level is a balance between the two error types. If the significance level is too high, there is more chance of committing Type I errors, however if it is too low there is more chance of committing Type II errors. Statistical convention is to set the significance level at either .05 or at .01. This research will make use of the .05 significance level.

3.2 Statistical Power

Statistical power for each analysis is shown by Effect sizes and Observed power values. Observed power is the probability of correctly rejecting the null hypothesis when it is false (Tredoux and Durrheim, 2008). Observed power should be higher than .80 as a smaller value will increase the risk of accepting a null hypothesis that is false, that is, committing a Type II error (Cohen, 1992). The .80 value will be further referred to as Cohen's cut-off.

While statistical significance indicates that there is a difference between groups due to an effect, effect size indicates the magnitude of that effect. Effect size generally conveys "the difference between means in standard deviation units" (Tredoux and Durrheim, 2008, p. 236). Effect sizes in repeated measures ANOVA tests are shown by Partial eta-squared (η_p^2) values. The η_p^2 values can be interpreted using the following values: .01 as a small effect, .06 as a medium effect and .14 as a large effect (Richardson, 2011).

3.3 Research aims, questions and hypotheses

The aim of this research was to investigate the relationship between alcohol use patterns and the cognitive and symptomatic functioning of Rhodes University Psychology students over time. The research aimed to also explore certain factors that may influence the cognitive functioning and alcohol use relationship. Thus, the research questions that guided this research are as follows:

1. Whether or not there is a relationship between alcohol use patterns and the cognitive functioning of university students over the course of one academic year.
2. Whether or not there is a relationship between the alcohol use patterns and the symptoms experienced by university students over the course of one academic year.
3. Whether or not any of the substance related factors exert a significant effect on the cognitive functioning and alcohol use relationship stated in question 1?

Following on from these research questions, it was hypothesised that the Harmful/Dependent group would score worse on the ImPACT cognitive tests than the Safe/Hazardous group. It was also hypothesised that practice effects would be seen in the Safe/Hazardous group but not to the same extent in the Harmful/Dependent group. It was hypothesized that the Harmful/Dependent group would score higher on the symptom scores when compared to the Safe/Hazardous group. In terms of the exploratory statistical analyses, it was hypothesised that the various subcomponents of the designated substance related moderating factors (including First drunk, First age, Drink, Hangover, Pass out, Parental alcohol use, Smoking and Substance use) would have a significant effect on the relationship between alcohol use and cognitive functioning.

3.4 Statistical Analyses

The Kolmogorov-Smirnov test for normality was run for the main dependent variables, including the ImPACT Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Impulse Control Composite scores, as well as the ImPACT Cognitive Efficiency Index, and the total ImPACT Symptom score. In all instances the test of normality for these variables was passed at the 1% level of significance ($p > 0.01$). For the further subset analyses on the moderating factors and individual symptom scores, it is understood that the subgroup sample numbers were very small, such that in many instances the assumption of normality would not be met. Accordingly, that aspect of the investigation has been done for exploratory purposes only.

Chi-squared tests and independent *t*-tests were run on Control and Neurological vulnerability variables, to ensure that the alcohol groups were matched according to those variables (see Sections 1.3 and 1.4, p. 98 and 100 respectively). Independent *t*-tests were performed on the continuous PPVT-4 raw score, PPVT-4 standard score, Age in years and Years at Rhodes variables while Chi-squared analyses were performed on the rest of the Control and Neurological categorical variables. Sample numbers were small, thus some of the expected frequencies in the Chi-squared tables were less than 5. The Yates' correction was used in place of Pearson's Chi-squared to correct for this in tables that were 2x2 in size. The Control and Neurological vulnerability tables were all 2x2 in size with the alcohol groups.

The Cognitive Composite scores and the Symptom Scores over the three intervals were analysed using repeated measures ANOVAs with the Alcohol group variable. This was done to measure the effects of the alcohol use patterns as well as the interaction effect of alcohol use patterns on the cognitive and symptomatic functioning of the students over time. Tukey HSD tests were used to explore any significant relationships in depth when necessary. Those analyses were used to answer research questions 1 and 2. To answer research question

3, an additional analysis was conducted using repeated measure ANOVA tests and Tukey HSD tests with the Cognitive Composite scores over the three intervals with the alcohol group variable and each of the substance related variables (see Table 11).

Chapter 3: Results

This chapter will report on the results from three sets of statistical analyses relating to each of the three research questions: (i) Statistical analysis of the ImPACT cognitive scores over three repeat time intervals with the alcohol use group variable; (ii) Statistical analysis of the ImPACT symptom scores over three repeat time intervals with the alcohol use group variable and (iii) Exploratory Statistical analyses of the ImPACT cognitive scores over three repeat time intervals with the alcohol use group variable and each of the substance related factors (First drunk, First age, Drink, Hangover, Pass out, Parental alcohol use, Smoking and Substance use). The first two sets of analyses, i.e., the Cognitive and Symptom analyses, were conducted using repeated measures ANOVA tests with the ImPACT cognitive and symptom scores at three time intervals as the dependent variables and alcohol group as the moderating factor. The third set of analyses, i.e., the Exploratory analysis, was a statistical exploratory analysis conducted using repeated measures ANOVA tests and Tukey HSD tests on the ImPACT cognitive scores at the three time intervals as the dependent variables with the alcohol group variable and substance related variables as moderating factors. These are considered exploratory because of the small sample sizes (see Table 9 in Section 1.5 in the Methodology chapter for the group distributions and sizes).

The results discussed are significant and approaching significant interaction effects and alcohol group effects. Approaching significant effects are those effects that have a p value that is less that .10 but higher than .05. For the Cognitive and Symptom analyses, the interaction effects are the effects related to the combination between time and alcohol group and are shown in the repeated measures ANOVA results (Alcohol group and time interaction effects). These interaction effects show that either a particular alcohol group changed their mean scores over the time intervals (time interaction effect), or that, at a specific time interval, one alcohol group scored significantly differently than another (alcohol group interaction effect). These specific interaction effects are shown in a Tukey HSD test.

The Alcohol group effect is where the alcohol group variable had an effect on the overall mean scores for the dependent variable, that is, the mean score obtained from all of the variable's scores from the year. These effects are shown in the repeated measures ANOVA results.

For the Exploratory analysis, the interaction effects are shown in the repeated measures ANOVA results. These effects are an interaction between the substance related variable, alcohol group variable and time (Substance related variable, alcohol group and time interaction effect) or an interaction between the alcohol group variable and the substance related variable (Substance related variable and alcohol group interaction effect). The substance related variable and alcohol group interaction effect involves the means of the whole year's scores for each group.

Section 1: ImPACT Cognitive Score Analysis

1.1 Alcohol group and time Interaction effects

From the cognitive scores' repeated measures ANOVA tests, there were significant interaction effects between alcohol group and time for the following scores: Visual Motor Speed, Reaction Time and Cognitive Efficiency Index. The repeated measures ANOVA results and the mean and standard deviation values are shown in Table 13.

Table 13: ImPACT Composite score Interaction effects

ImPACT/ Alcohol group	*n*	*M (SD)* Interval 1	Interval 2	Interval 3	*F*(2, 168)	*p*
Verbal Memory						
Safe/ Hazardous	74	87.61 (9.72)	88.93 (9.34)	91.84 (8.96)	1.80	.168
Harmful/ Dependent	12	93.42 (7.14)	95.25 (7.63)	92.83 (7.32)		
Visual Memory						
Safe/ Hazardous	74	71.65 (12.68)	73.69 (12.81)	75.57 (12.82)	0.60	.550
Harmful/ Dependent	12	69.92 (9.20)	73.17 (12.84)	77.83 (12.04)		
Visual Motor Speed						
Safe/ Hazardous	74	38.06 (7.01)	39.23 (6.49)	41.65 (7.03)	3.18	*.044
Harmful/ Dependent	12	40.30 (4.40)	40.24 (6.25)	40.16 (5.88)		
Reaction Time						
Safe/ Hazardous	74	0.60 (0.09)	0.59 (0.10)	0.57 (0.08)	6.44	**.002
Harmful/ Dependent	12	0.64 (0.12)	0.60 (0.08)	0.67 (0.12)		
Cognitive Efficiency Index						
Safe/ Hazardous	74	0.34 (0.15)	0.37 (0.15)	0.43 (0.13)	9.49	**<.001
Harmful/ Dependent	12	0.27 (0.30)	0.36 (0.20)	0.20 (0.31)		

Note: *$p < .05$, **$p < .01$; In all instances high scores are in the direction of good performance, with the exception for Reaction Time where a low score is in the direction of good performance.

The Visual Motor Speed significant results had a low power value (.60) which did not meet Cohen's cut-off of .80 and had a low Partial eta-squared (η_p^2) effect size value of .04. The Reaction Time effects had a high power value that was above Cohen's cut-off (.90) and a medium effect size ($\eta_p^2 = .07$). The power value for the Cognitive Efficiency Index is high at .98 and surpassed Cohen's cut-off, while the effect size ($\eta_p^2 = .10$) is considered medium to large.

Further analyses with the Tukey HSD test for the Visual Motor Speed composite scores showed a significant time interaction effect. The Visual Motor Speed mean scores for the Safe/Hazardous group increased significantly from interval 1 to interval 3 ($p < .001$) and from interval 2 to interval 3 ($p < .001$). These values can be seen in Table 13. The Harmful/Dependent group did not change their mean scores significantly over the time intervals. This shows that the Safe/Hazardous group improved their performance on Visual Motor Speed tests over time and that the Harmful/Dependent group did not.

A Tukey HSD test performed on the significant Reaction Time interaction effects showed a significant alcohol group interaction effect. At interval 3, the Safe/Hazardous group had significantly shorter mean Reaction Time scores when compared to the Harmful/Dependent group ($p = .012$) (i.e., performance was better for the Safe/Hazardous drinkers that for the Harmful/Dependent drinkers). The Tukey HSD test also showed a significant time interaction effect where the Harmful/Dependent group increased their mean Reaction Time scores significantly from interval 2 to interval 3 ($p = .037$). This shows that the Harmful/Dependent group's performance declined over time.

A Tukey HSD test performed on the significant Cognitive Efficiency Index interaction effects showed a significant alcohol group interaction effect. At interval 3, the Safe/Hazardous drinkers scored significantly higher than the Harmful/Dependent drinkers ($p < .001$) (i.e., performance was better for the Safe/Hazardous drinkers than for the

Harmful/Dependent drinkers). The Tukey HSD test also showed a significant time interaction effect where the Safe/Hazardous group increased their scores significantly from interval 1 to interval 3 ($p < .001$) and from interval 2 to interval 3 ($p = .012$) while the Harmful/Dependent drinkers decreased their mean scores significantly from interval 2 to interval 3 ($p = .018$). This shows that the Safe/Hazardous group improved their performance over time while the Harmful/Dependent group's performance declined.

1.2 Alcohol group effects

The composite scores' repeated measures ANOVA tests showed significant alcohol group effects for Reaction Time and Cognitive Efficiency Index. The Verbal Memory results approached significance where the *p* value was just over .05. The repeated measures ANOVA results and the mean and standard deviation values are shown in Table 14.

Table 14: ImPACT Composite score Alcohol group effects

ImPACT/ Alcohol group	*n*	*M (SD)*	Common *SD*	*F*(1, 84)	*p*
Verbal Memory					
Safe/Hazardous	74	89.46 (9.47)	7.07	3.95	.050
Harmful/Dependent	12	93.83 (7.23)			
Visual Memory					
Safe/Hazardous	74	73.64 (12.81)	10.52	0.00	.999
Harmful/Dependent	12	73.64 (11.61)			
Visual Motor Speed					
Safe/Hazardous	74	39.65 (6.98)	6.08	0.10	.757
Harmful/Dependent	12	40.23 (5.41)			
Reaction Time					
Safe/Hazardous	74	0.59 (0.09)	0.08	4.04	*.048
Harmful/Dependent	12	0.64 (0.11)			

ImPACT/ Alcohol group	*n*	*M (SD)*	Common *SD*	*F*(1, 84)	*p*
Cognitive Efficiency Index					
Safe/Hazardous	74	0.38 (0.15)	0.14	6.26	*.014
Harmful/Dependent	12	0.27 (0.27)			

Note: $*p < .05$, $**p < .01$; In all instances high scores are in the direction of good performance, with the exception for Reaction Time where a low score is in the direction of good performance.

The Reaction Time results had a power value that did not reach Cohen's cut-off (.51) and had a small to medium effect size ($\eta_p^2 = .05$). The Cognitive Efficiency Index results had a power value which failed to pass Cohen's cut-off (.70) and a medium effect size value ($\eta_p^2 = .07$).

Table 14 shows that the overall mean Reaction Time scores were significantly lower for the Safe/Hazardous drinkers than for the Harmful/Dependent drinkers (i.e., performance was better for the Safe/Hazardous drinkers than for the Harmful/Dependent drinkers). The Cognitive Efficiency Index mean score was significantly higher for the Safe/Hazardous group than for the Harmful/Dependent group (i.e., performance was better for the Safe/Hazardous drinkers than for the Harmful/Dependent drinkers).

The Verbal Memory relationship that approached significance had a low power value (.50) and a small effect size ($\eta_p^2 = .04$). As this relationship did not reach significance, no further analyses were done. However, it was observed that the Safe/Hazardous group had a lower overall mean score when compared with the Harmful/Dependent group (i.e., performance was worse for the Safe/Hazardous drinkers than for the Harmful/Dependent drinkers).

Section 2: ImPACT Symptom Score Analysis

2.1 Interaction effects

From the symptom scores' repeated measures ANOVA tests, there were significant interaction effects between alcohol use and time only for the Numbness/Tingling symptom. The Nausea, Vomiting and Visual problems symptom score results approached significance. The significant and approaching significant repeated measures ANOVA results, along with the Total symptom score results, are shown in Table 15. The mean and standard deviation values for these results are also shown in Table 15.

Table 15: ImPACT Symptom score Interaction effects

ImPACT/ Alcohol group	*n*	*M (SD)* Interval 1	Interval 2	Interval 3	*F*(2, 168)	*p*
Total						
Safe/ Hazardous	74	19.66 (14.34)	18.62 (19.57)	18.07 (16.95)	0.32	.728
Harmful/ Dependent	12	22.92 (23.88)	22.92 (15.47)	25.25 (25.76)		
Nausea						
Safe/ Hazardous	74	0.39 (1.06)	0.30 (0.84)	0.09 (0.44)	2.73	.068
Harmful/ Dependent	12	0.50 (0.80)	0.33 (0.89)	0.83 (1.75)		
Vomiting						
Safe/ Hazardous	74	0.03 (0.23)	0.04 (0.35)	0.05 (0.33)	2.55	.081
Harmful/ Dependent	12	0.08 (0.29)	0.00 (0.00)	0.42 (1.44)		

ImPACT/ Alcohol group	n	*M (SD)* Interval 1	Interval 2	Interval 3	F(2, 168)	p
Numbness/ Tingling						
Safe/ Hazardous	74	0.11 (0.46)	0.14 (0.51)	0.09 (0.50)	3.12	*.047
Harmful/ Dependent	12	0.50 (1.00)	0.17 (0.39)	0.75 (1.76)		
Visual problems						
Safe/ Hazardous	74	0.65 (1.30)	0.54 (1.24)	0.31 (0.86)	2.91	.057
Harmful/ Dependent	12	0.92 (1.56)	0.00 (0.00)	0.58 (1.16)		

Note: *p < .05, **p < .01; In all instances high scores are in the direction of poor health.

The Numbness/Tingling symptom effects power value did not meet Cohen's cut-off (.59) and the effect size was small (η_p^2 = .04). The approaching significant Nausea, Vomiting and Visual problem symptom score results all had low power values (.53, .50 and .56 respectively) and had the same value for effect size which is considered small (η_p^2 = .03).

A Tukey HSD test for the interaction effects on the Numbness/Tingling symptom mean scores showed a significant alcohol group interaction effect. At interval 3, the Safe/Hazardous group had a significantly lower mean score than the Harmful/Dependent group (p = .010) (i.e. the Safe/Hazardous group had less severe symptoms than the Harmful/Dependent group). There were no other relevant significant differences in the Tukey HSD test.

From Table 15, it can be seen that the Total symptom scores did not differ significantly between the groups or over the time intervals, thus no further analyses were performed on this relationship. The Nausea, Vomiting and Visual problem symptom score results only approached significance, and thus no further analyses were performed on these relationships. However, it was observed that at all of the time intervals the Harmful/Dependent groups had higher Nausea symptom mean scores than the Safe/Hazardous group (p = .068). The Safe/Hazardous group was observed to have decreased their Nausea symptom scores from interval 1 to interval 3 but the Harmful/Dependent group increased their mean scores from interval 1 to interval 3 (p = .068). In terms of the Vomiting symptom mean scores, it was observed that the Harmful/Dependent group's interval 3 mean score was higher than all of the other mean scores, especially the Safe/Hazardous group's interval 3 mean score (p = .081). The Visual problem symptom mean scores for the Safe/Hazardous group were observed to have steadily decreased from interval 1 to interval 2 to interval 3 while the Harmful/Dependent group had a higher interval 1 mean score, a very low interval 2 mean score and then a higher interval 3 mean score (p = .057).

2.2 Alcohol group effects

The symptom scores' repeated measures ANOVA tests also showed significant alcohol group effects for the Balance problems, Dizziness and Numbness/Tingling symptom scores. The Vomiting symptom results approached significance. The repeated measures ANOVA results and the mean and standard deviation values are shown in Table 16.

Table 16: ImPACT Symptom score Alcohol group effects

ImPACT/ Alcohol group	*n*	*M (SD)*	Common SD	*F*(2, 168)	*p*
Total					
Safe/Hazardous	74	18.78 (17.02)	18.78	1.09	.300
Harmful/Dependent	12	23.69 (21.54)			
Vomiting					
Safe/Hazardous	74	0.04 (0.31)	0.24	2.89	.093
Harmful/Dependent	12	0.17 (0.85)			
Balance problems					
Safe/Hazardous	74	0.17 (0.63)	0.49	5.61	*.020
Harmful/Dependent	12	0.53 (1.11)			
Dizziness					
Safe/Hazardous	74	0.29 (0.83)	0.64	6.84	*.011
Harmful/Dependent	12	0.81 (1.24)			
Numbness/Tingling					
Safe/Hazardous	74	0.11 (0.49)	0.42	7.56	**.007
Harmful/Dependent	12	0.47 (1.18)			

Note: $*p < .05$, $**p < .01$; In all instances high scores are in the direction of poor health.

The observed power value for the Balance problems symptom (.65) did not reach Cohen's cut-off and the effect size is considered medium ($\eta_p^2 = .06$). The observed power value for the Dizziness symptom was just below Cohen's cut-off (.73) and the effect size is considered medium ($\eta_p^2 =.08$). The observed power value for the Numbness/Tingling symptom did not meet Cohen's cut-off (0.59) and the effect size was small ($\eta_p^2 = .04$). The approaching significant results for the Vomiting symptom score had a low power value (.39) and a small effect size ($\eta_p^2 = .03$).

Table 16 shows that the Safe/Hazardous group had significantly lower overall mean Balance problems, Dizziness and Numbness/Tingling scores symptom scores when compared the Harmful/Dependent group (p = .020, .011, and .007, respectively). The Vomiting symptom score results only approached significance and no further analyses were performed on that data. However it was observed that the Vomiting symptom mean scores were higher for the Harmful/Dependent group than for the Safe/Hazardous group, but not significantly (p = .093).

Section 3: Statistical Exploratory Analysis

A series of exploratory statistical analyses were done with each of the ImPACT composite scores (Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Cognitive Efficiency Index) over time with the alcohol use group variable and each of the moderating factors (First drunk, First age, Drink, Hangover, Pass out, Parental alcohol use, Smoking and Substance use). These analyses are considered exploratory because of the small sample sizes. All of the moderating factors, except Hangover, had a significant effect on at least one of the composite scores. However, there were no significant results in terms of Verbal Memory in these exploratory analyses. Only the significant results will be discussed.

3.1. Visual Memory

Visual Memory mean scores were only significantly affected by an interaction effect between Parental alcohol use, alcohol group and time ($F(2, 162) = 4.61$, $p = .011$). The power value for the significant Parental alcohol use, alcohol group and time interaction effects was just below Cohen's cut-off (.77) and the effect size was medium ($\eta_p^2 = .05$). A Tukey HSD test on the interaction effect did not find any significant results. The repeated measures ANOVA result and mean and standard deviation values for this effect are shown in Table 17.

Table 17: Visual Memory and the Parental alcohol use, alcohol group and time Interaction effect

Parental alcohol use	**Alcohol group**	***n***	***M (SD)*** Interval 1	Interval 2	Interval 3	***F* (2,162)**	***p***
Abstain	**Safe/ Hazardous**	18	69.89 (13.94)	68.61 (9.89)	70.61 (11.06)		
Abstain	**Harmful/ Dependent**	3	67.00 (6.00)	82.33 (2.52)	89.67 (7.77)		
Light	**Safe/ Hazardous**	52	72.00 (12.59)	75.38 (13.56)	77.02 (13.34)	4.61	*.011
Light	**Harmful/ Dependent**	9	70.89 (10.15)	70.11 (13.53)	73.89 (10.68)		
Heavy	**Safe/ Hazardous**	4	75.00 (8.76)	74.50 (10.50)	79.00 (9.06)		

Note: $^{*}p < .05$, $^{**}p < .01$; For Visual Memory instances, high scores are in the direction of good performance

3.2 Visual Motor Speed

Visual Motor Speed was significantly affected by the interaction between the Smoking, alcohol group and time variables ($F(2, 164) = 4.84$, $p = .009$) and the Pass out and alcohol group variables ($F(2, 77) = 4.92$, $p = .010$). These significant interaction effects will be discussed individually below.

3.2.1 Substance related variable, alcohol group and time interaction effect

a) Smoking

The interaction between Smoking, alcohol group and time significantly affected the Visual Motor Speed mean scores as can be seen in Table 18 which shows the repeated measure ANOVA results along with the mean and standard deviation values.

Table 18: Visual Motor Speed and the Smoking, alcohol group and time Interaction effect

Smoking	Alcohol group	*n*	*M (SD)* Interval 1	Interval 2	Interval 3	*F* (2,164)	*p*
Yes	Safe/ Hazardous	15	38.88 (7.15)	39.35 (5.90)	41.32 (5.17)	4.84	**.009
Yes	Harmful/ Dependent	7	38.84 (3.17)	41.04 (7.35)	42.07 (6.80)		
No	Safe/ Hazardous	59	37.85 (7.02)	39.20 (6.67)	41.73 (7.46)		
No	Harmful/ Dependent	5	42.34 (5.41)	39.13 (4.86)	37.48 (3.24)		

Note: *$p < .05$, **$p < .01$; For Visual Motor Speed instances high scores are in the direction of good performance

The power value for this effect was just below Cohen's cut-off (.79) and the effect size value is considered medium ($\eta_p^2 = .06$). A Tukey HSD test showed that the Safe/Hazardous group who do not smoke increased their mean scores significantly from interval 1 ($p < .001$) and interval 2 ($p = .003$) to interval 3.

3.2.2 Substance related variable and alcohol group interaction effect

a) Pass out

The Visual Motor Speed scores were significantly affected by the interaction between the Pass out and alcohol group variables as can be seen in Table 19 which shows the repeated measure ANOVA results along with the mean and standard deviation values.

Table 19: Visual Motor Speed and the Pass out and alcohol group Interaction effect

Pass out	***n***	***M (SD)***	**Common *SD***	***F*(2, 77)**	***p***
Abstain					
Safe/Hazardous	7	42.27 (8.96)			
Harmful/Dependent	0	-			
Never					
Safe/Hazardous	52	39.90 (6.64)			
Harmful/Dependent	4	40.04 (7.69)			
Not in year					
Safe/Hazardous	2	42.64 (7.07)			
Harmful/Dependent	0	-	5.80	4.92	*.010
Less than monthly					
Safe/Hazardous	12	35.44 (4.45)			
Harmful/Dependent	5	42.22 (3.38)			
Monthly					
Safe/Hazardous	1	52.83 (1.41)			
Harmful/Dependent	2	35.93 (2.03)			
Weekly					
Safe/Hazardous	0	-			
Harmful/Dependent	1	39.71 (3.18)			

Note: **p* < .05, ***p* < .01; For Visual Motor Speed instances high scores are in the direction of good performance

The power value for this relationship was just below Cohen's cut-off at .79 and had a large effect size value ($\eta_p^2 = 0.11$). A Tukey HSD test on this relationship showed no significant results.

3.3 Reaction Time

Reaction Time was significantly affected by the interaction between alcohol group and time and the following variables: First drunk ($F(4, 158) = 4.14$, $p = .003$), First age ($F(2, 162) = 3.93$, $p = .022$), Drink ($F(4, 154) = 2.64$, $p = .036$) and Substance use ($F(2, 164) = 4.12$, $p = .018$). Reaction Time was also significantly affected by the interaction between the alcohol group variable and the Pass out variable ($F(2, 77) = 5.48$, $p = .006$). These will be discussed under separate headings below.

3.3.1 Substance related variable, alcohol group and time interaction effects

Table 20 shows the significant repeated measures ANOVA results for Reaction Time with the interaction between alcohol group and time with First drunk, First age, Drink and Substance use. These will be discussed below under headings for each of the substance related variables.

Table 20: Reaction Time and substance related variable, alcohol group and time Interaction effects

Substance related variable	**Alcohol group**	***n***	***M (SD)*** Interval 1	**Interval 2**	**Interval 3**	***F*(*df*)**	***p***
First drunk						***F*(4,158)**	***p***
Never	Safe/ Hazardous	12	0.58 (0.08)	0.56 (0.09)	0.56 (0.07)		
Primary School	Safe/ Hazardous	3	0.64 (0.11)	0.63 (0.15)	0.59 (0.13)		
Primary School	Harmful/ Dependent	2	0.70 (0.22)	0.61 (0.12)	0.75 (0.14)		
High School	Safe/ Hazardous	38	0.60 (0.10)	0.59 (0.09)	0.57 (0.08)	4.14	**.003
High School	Harmful/ Dependent	8	0.60 (0.05)	0.58 (0.08)	0.66 (0.13)		
Over 18	Safe/ Hazardous	21	0.58 (0.07)	0.61 (0.11)	0.57 (0.08)		
Over 18	Harmful/ Dependent	2	0.77 (0.19)	0.64 (0.04)	0.60 (0.06)		

Substance related variable	Alcohol group	*n*	*M (SD)* Interval 1	Interval 2	Interval 3	*F*(*df*)	*p*
First age						***F*(2,162)**	***p***
Never	Safe/ Hazardous	15	0.58 (0.10)	0.58 (0.12)	0.57 (0.10)		
High School	Safe/ Hazardous	14	0.62 (0.11)	0.59 (0.11)	0.58 (0.09)		
High School	Harmful/ Dependent	5	0.66 (0.12)	0.64 (0.10)	0.76 (0.14)	3.93	*.022
Over 18	Safe/ Hazardous	45	0.59 (0.08)	0.60 (0.09)	0.57 (0.07)		
Over 18	Harmful/ Dependent	7	0.63 (0.12)	0.57 (0.06)	0.60 (0.04)		
Drink						***F*(4,154)**	***p***
Never	Safe/ Hazardous	7	0.63 (0.15)	0.58 (0.12)	0.58 (0.10)		
Wine	Safe/ Hazardous	23	0.63 (0.09)	0.62 (0.09)	0.60 (0.09)		
Wine	Harmful/ Dependent	1	0.68 (-)	0.61 (-)	0.88 (-)		
Spirits	Safe/ Hazardous	5	0.56 (0.07)	0.54 (0.04)	0.55 (0.04)		
Beer	Safe/ Hazardous	4	0.59 (0.17)	0.52 (0.04)	0.52 (0.08)	2.64	*.036
Beer	Harmful/ Dependent	5	0.70 (0.17)	0.65 (0.09)	0.71 (0.13)		
Mixture	Safe/ Hazardous	26	0.57 (0.07)	0.60 (0.11)	0.56 (0.06)		
Mixture	Harmful/ Dependent	6	0.60 (0.04)	0.55 (0.04)	0.60 (0.03)		
Other	Safe/ Hazardous	9	0.58 (0.07)	0.56 (0.07)	0.58 (0.05)		

Substance related variable	Alcohol group	*n*	*M (SD)* Interval 1	Interval 2	Interval 3	*F*(*df*)	*p*
Substance use						***F*(2,164)**	***p***
Yes	Safe/ Hazardous	5	0.57 (0.03)	0.60 (0.08)	0.55 (0.06)	4.12	*.018
Yes	Harmful/ Dependent	1	0.68 (-)	0.61 (-)	0.88 (-)		
No	Safe/ Hazardous	69	0.60 (0.09)	0.59 (0.10)	0.57 (0.08)		
No	Harmful/ Dependent	11	0.64 (0.12)	0.59 (0.08)	0.65 (0.10)		

Note: $^*p < .05$, $^{**}p < .01$; For Reaction Time a low score is in the direction of good performance

a) First drunk

The First drunk, alcohol group and time interaction effect ($F(4, 158) = 4.14$, $p = .003$) significantly affected the mean Reaction Time scores. This interaction effect had a power value that reached Cohen's cut-off at .91 and had a medium to high effect size ($\eta_p^2 = .09$). However, the Tukey HSD test for this effect showed no significant results for Reaction Time. The mean and standard deviation values are shown in Table 20 above.

b) First age

The First age, alcohol group and time interaction effect ($F(2, 162) = 3.93$, $p = .022$) significantly affected Reaction Time. This interaction effect had a power value that did not reach Cohen's cut-off (.70) and had a small to medium effect size value ($\eta_p^2 = .05$). The Tukey HSD test showed that, at interval 3, the Harmful/Dependent drinkers who started drinking regularly in high school had a significantly higher mean Reaction Time than the Safe/Hazardous drinkers who do not drink alcohol regularly ($p = .003$), who started drinking regularly in high school ($p = .012$) and who started drinking regularly over the age of 18 ($p =$

.001). This result indicates that, specifically at interval 3, the Harmful/Dependent drinkers who started drinking regularly in high school performed worse than the Safe/Hazardous drinkers who do not drink regularly or who started drinking regularly in high school and over the age of 18. The Tukey HSD test also showed that the Harmful/Dependent drinkers who started consuming alcohol regularly in high school increased their mean Reaction Time scores significantly from interval 2 to interval 3 (p = .047). This result indicates that the performance of the Harmful/Dependent drinkers who started drinking regularly in high school declined over the year. The mean and standard deviation values are shown in Table 20 above.

c) Drink

The Drink, alcohol group and time interaction effect ($F(4, 154) = 2.64$, $p = .036$) significantly affected Reaction Time. The interaction effect had a power value that was just below Cohen's cut-off (.73) and had a medium effect size ($\eta_p^2 = .06$). The Tukey HSD test for this relationship showed no significant results. The mean and standard deviation values are shown in Table 20 above.

d) Substance use

The Substance use, alcohol group and time interaction effect ($F(2, 164) = 4.12$, $p = .018$) significantly affected Reaction Time. The power value did not reach Cohen's cut-off (.72) and had a small to medium effect size value ($\eta_p^2 = .05$). The Tukey HSD test for this relationship showed that at interval 3, the Harmful/Dependent drinker who uses substances scored significantly higher than the Safe/Hazardous drinkers who use substances ($p = .045$) and who do not use substances ($p = .038$). This result indicates that at interval 3 the Harmful/Dependent drinker who uses substances performed worse on the tests than the

Safe/Hazardous drinkers who do and do not use substances. The Harmful/Dependent drinker who uses substances increased their Reaction Time score significantly (i.e., their performance got worse) from interval 2 to interval 3 (p = .034). This result indicates that the performance of Harmful/Dependent drinker who uses substances declined over the time intervals. The mean and standard deviation values are shown in Table 20.

3.3.2 Substance related variable and alcohol group interaction effect

a) Pass out

Reaction Time was significantly affected by the interaction between the Pass out and alcohol group variables as can be seen in Table 21 which shows the repeated measure ANOVA results along with the mean and standard deviation values.

Table 21: Reaction Time and the Pass out and alcohol group Interaction effect

Pass out	***n***	***M (SD)***	**Common *SD***	***F*(2, 77)**	***p***
Abstain					
Safe/Hazardous	7	0.57 (0.11)			
Harmful/Dependent	0	-			
Never					
Safe/Hazardous	52	0.59 (0.09)			
Harmful/Dependent	4	0.59 (0.04)			
Not in year					
Safe/Hazardous	2	0.58 (0.05)	0.08	5.48	**.006
Harmful/Dependent	0	-			
Less than monthly					
Safe/Hazardous	12	0.60 (0.07)			
Harmful/Dependent	5	0.65 (0.12)			
Monthly					
Safe/Hazardous	1	0.40 (0.01)			
Harmful/Dependent	2	0.73 (0.13)			

Pass out	***n***	***M (SD)***	**Common *SD***	***F*(2, 77)**	***p***
Weekly					
Safe/Hazardous	0	-			
Harmful/Dependent	1	0.57 (0.07)			

Note: $*p < .05$, $**p < .01$; For Reaction Time a low score is in the direction of good performance

The power value for this relationship was high and passed Cohen's cut-off (.84) and had a large effect size value ($\eta_p^2 = .12$). A Tukey HSD test showed that the Safe/Hazardous group who reported passing out monthly had lower overall mean Reaction Time scores compared to the Harmful/Dependent group who reported passing out monthly ($p = .02$) (i.e., performance was worse for the Harmful/Dependent drinkers than for the Safe/Hazardous drinkers).

3.4 Cognitive Efficiency Index

The Cognitive Efficiency Index scores were significantly affected by the interaction between alcohol group and time and the following variables: First drunk ($F(4, 158) = 4.66$, $p = .001$), First age ($F(2, 162) = 4.28$, $p = .015$), Drink ($F(4, 154) = 3.62$, $p = .007$) and Substance use ($F(2, 164) = 6.50$, $p = .002$). The Cognitive Efficiency Index scores were also significantly affected by the interaction between the alcohol group variable and the following variables: First drunk ($F(1, 81) = 16.83$, $p < .001$), Drink ($F(2, 77) = 3.64$, $p = .031$), Pass out ($F(2, 77) = 5.83$, $p = .004$), Smoking ($F(1, 82) = 8.35$, $p = .005$) and Substance use ($F(1, 82) = 4.41$, $p = .039$). These significant interaction effects will be discussed individually below.

3.4.1 Substance related variable, alcohol group and time interaction effects

Table 22 shows the significant repeated measures ANOVA results for Cognitive Efficiency Index with the interaction between alcohol group and time with First drunk, First age, Drink and Substance use. These will be discussed below under headings for each of the substance related variables.

Table 22: Cognitive Efficiency Index and the substance related variable, alcohol group and time Interaction effects

Substance related variable	Alcohol group	*M (SD)*					
		n	**Interval 1**	**Interval 2**	**Interval 3**	***F*(*df*)**	***p***
First Drunk						***F*(4,158)**	
Never	Safe/ Hazardous	12	0.34 (0.16)	0.43 (0.16)	0.47 (0.11)		
Primary School	Safe/ Hazardous	3	0.41 (0.13)	0.34 (0.05)	0.47 (0.12)		
Primary School	Harmful/ Dependent	2	0.02 (0.68)	0.27 (0.31)	-0.11(0.28)		
High School	Safe/ Hazardous	38	0.33 (0.17)	0.36 (0.15)	0.43 (0.12)	4.66	**.001
High School	Harmful/ Dependent	8	0.36 (0.16)	0.36 (0.21)	0.21 (0.31)		
Over 18	Safe/ Hazardous	21	0.34 (0.12)	0.35 (0.16)	0.41 (0.16)		
Over 18	Harmful/ Dependent	2	0.13 (0.33)	0.44 (0.01)	0.46 (0.09)		
First Age						***F*(2,162)**	
Never	Safe/ Hazardous	15	0.36 (0.16)	0.40 (0.19)	0.46 (0.12)		
High School	Safe/ Hazardous	14	0.37 (0.17)	0.37 (0.14)	0.46 (0.12)		
High School	Harmful/ Dependent	5	0.12 (0.36)	0.24 (0.21)	-0.08(0.29)	6.50	**.002
Over 18	Safe/ Hazardous	45	0.33 (0.15)	0.36 (0.15)	0.42 (0.14)		
Over 18	Harmful/ Dependent	7	0.37 (0.22)	0.44 (0.15)	0.40 (0.10)		

Substance related variable	Alcohol group	*M (SD)*					
		n	**Interval 1**	**Interval 2**	**Interval 3**	***F*(*df*)**	***p***
Drink						***F*(4,154)**	
Never	Safe/ Hazardous	7	0.30 (0.23)	0.36 (0.24)	0.42 (0.19)		
Wine	Safe/ Hazardous	23	0.34 (0.12)	0.33 (0.15)	0.42 (0.14)		
Wine	Harmful/ Dependent	23	0.19 (-)	0.32 (-)	-0.40 (-)		
Spirits	Safe/ Hazardous	5	0.33 (0.13)	0.37 (0.12)	0.33 (0.15)		
Beer	Safe/ Hazardous	4	0.28 (0.38)	0.38 (0.15)	0.48 (0.14)	3.62	**.007
Beer	Harmful/ Dependent	5	0.11 (0.39)	0.28 (0.24)	0.12 (0.34)		
Mixture	Safe/ Hazardous	26	0.35 (0.13)	0.37 (0.15)	0.46 (0.10)		
Mixture	Harmful/ Dependent	6	0.41 (0.16)	0.42 (0.17)	0.37 (0.09)		
Other	Safe/ Hazardous	9	0.36 (0.12)	0.43 (0.14)	0.44 (0.12)		
Substance Use						***F*(2,164)**	
Yes	Safe/ Hazardous	5	0.43 (0.05)	0.41 (0.12)	0.49 (0.08)		
Yes	Harmful/ Dependent	1	0.19 (-)	0.32 (-)	-0.40 (-)		
No	Safe/ Hazardous	69	0.33 (0.16)	0.36 (0.16)	0.43 (0.13)	6.50	**.002
No	Harmful/ Dependent	11	0.27 (0.31)	0.36 (0.21)	0.25 (0.26)		

Note: $*p < .05$, $**p < .01$; For Cognitive Efficiency Index scores a high score is in the direction of good performance

a) First drunk

The Cognitive Efficiency Index scores were significantly affected by an interaction between the First drunk, alcohol group and time factors ($F(4, 158) = 4.66$, $p = .001$). The power value reached Cohen's cut-off at .94 and the effect size is considered large ($\eta_p^2 = .11$). The Tukey HSD test showed that, at interval 3, the Harmful/Dependent drinkers who first got drunk in primary school had significantly lower mean scores than the Safe/Hazardous drinkers who had never gotten drunk ($p = .001$), who first got drunk in primary school ($p = .024$), who first got drunk in high school ($p = .002$) and who first got drunk over the age of 18 ($p = .005$). This result indicates that at interval 3 the Harmful/Dependent drinkers who first got drunk in primary school scored worse than the Safe/Hazardous drinkers who have never gotten drunk and who first got drunk in primary school, high school and over the age of 18. The Tukey HSD test also showed that the Safe/Hazardous drinkers who first got drunk in high school increased their mean scores significantly from interval 1 to interval 3 ($p = .046$). This result indicates that the Safe/Hazardous drinkers who first got drunk in high school improved their performance over the year. The mean and standard deviation values are shown in Table 22 above.

b) First age

The Cognitive Efficiency Index scores were significantly affected by the First age, alcohol group and interaction effects ($F(2, 162) = 4.28$, $p = .015$). The power value was just below Cohen's cut-off (.74) and the effect size was small to medium ($\eta_p^2 = .05$). A Tukey HSD test showed that at, interval 3, the Harmful/Dependent drinkers who started drinking regularly in high school had significantly lower mean scores compared to the Harmful/Dependent drinkers who started drinking regularly over the age of 18 ($p < .001$), as well as the Safe/Hazardous drinkers who do not drink alcohol regularly ($p < .001$), who started drinking regularly in high school ($p < .001$) and who started drinking over the age of 18 ($p < .001$). This result indicates that at interval 3 the Harmful/Dependent drinkers who started drinking regularly in high school scored worse than the Harmful/Dependent drinkers who started drinking regularly over the age of 18 and the Safe/Hazardous drinkers who do not consume alcohol regularly and who started drinking regularly in high school and over the age of 18. The Tukey HSD test also showed that the Safe/Hazardous drinkers who started drinking regularly over the age of 18 increased their mean scores significantly from interval 1 to interval 3 ($p = .032$). This result indicates that the Safe/Hazardous drinkers who started drinking regularly over the age of 18 improved their performance over the year. The Tukey HSD test showed that the Harmful/Dependent drinkers who started drinking regularly in high school decreased their mean scores significantly from interval 2 to interval 3 ($p = .002$), thus their performance declined. The mean and standard deviation values are shown in Table 22 above.

c) Drink

The Cognitive Efficiency Index scores were significantly affected by the Drink, alcohol group and time interaction effects ($F(4, 154) = 3.62$, $p = .007$). The power value exceeded Cohen's cut-off (.87) and had a medium to large effect size value ($\eta_p^2 = .09$). The Tukey HSD test showed that at interval 3, the Safe/Hazardous drinkers who consumed a mixture of alcoholic beverages most often had a significantly higher mean score than the Harmful/Dependent drinkers who consumed beer most often ($p = .005$) (i.e. the Safe/Hazardous drinkers who consumed a mixture of alcoholic beverages scored better than the Harmful/Dependent drinkers who consumed beer). Also at interval 3, the Harmful/Dependent drinker who consumed wine had a significantly lower Cognitive Efficiency Index score compared to the Harmful/Dependent drinkers who consumed a mixture of alcohol beverages ($p = .004$), as well as the Safe/Hazardous drinkers who do not consume alcoholic beverages ($p < .001$), who consumed wine ($p < .001$), who drank spirits ($p = .011$), who consume a mixture of alcoholic beverages (p < .001), who consumed beer ($p < .001$) and who consumed other types of alcoholic beverages ($p < .001$). This result indicates that at interval 3 the Harmful/Dependent drinker who consumed wine scored worse than the Harmful/Dependent drinkers who consumed a mixture of alcoholic beverages and the Safe/Hazardous drinkers who do not consume alcoholic beverages and who consume wine, spirits, a mixture, beer and other types of alcoholic beverages most often. The Tukey HSD test also showed that the Harmful/Dependent drinker who consumed wine decreased their score significantly from interval 2 to interval 3 ($p = .003$). This result indicates that the performance of the Harmful/Dependent drinker who consumed wine decreased over time. The mean and standard deviation values are shown in Table 22 above.

d) Substance use

The Cognitive Efficiency Scores were significantly affected by the Substance use, alcohol group and time interaction effect ($F(2, 164) = 6.50$, $p = .002$). The effect had a high power value that surpassed Cohen's cut-off (.90) and a medium effect size ($\eta_p^2 = .07$). The Tukey HSD test showed that at interval 3, the Harmful/Dependent drinker who uses substances scored significantly lower than the Harmful/Dependent drinkers who do not use substances ($p = .009$), the Safe/Hazardous drinkers who do not use substances ($p < .001$) and the Safe/Hazardous drinkers who do use substances ($p < .001$). This result indicates that at interval 3 the Harmful/Dependent drinker who uses substances scored worse on the test than the Safe/Hazardous drinkers who do and do not use substances. The Tukey HSD test also showed that the Safe/Hazardous drinkers who do not use substances significantly increased their mean scores from interval 1 to interval 3 ($p < .001$). The Harmful/Dependent drinker who uses substances decreased their score significantly from interval 1 (p = .017) and interval 2 (p = .001) to interval 3. These results indicate that the performance of the Safe/Hazardous drinkers who do not use substances improved while the performance of the Harmful/Dependent drinker who uses substances declined. The mean and standard deviation values are shown in Table 22 above.

3.4.2 Substance related variable and alcohol group interaction effect

Table 23 shows the significant repeated measures ANOVA results for Cognitive Efficiency Index with the alcohol group variable and First drunk, Drink, Pass out, Smoking and Substance use. These will be discussed below under headings for each of the substance related variables.

Table 23: Cognitive Efficiency Index and the Substance related variable and alcohol group Interaction effects

Substance related variable	**Alcohol group**	***n***	***M (SD)***	**Common *SD***	***F(df)***	***p***
First drunk					***F*(1,81)**	
Never	Safe/Hazardous	15	0.40 (0.16)			
High school	Safe/Hazardous	14	0.40 (0.15)			
High school	Harmful/Dependent	5	0.09 (0.30)	0.99	16.83	**<.001
Over 18	Safe/Hazardous	45	0.37 (0.15)			
Over 18	Harmful/Dependent	7	0.40 (0.16)			

Substance related variable	Alcohol group	*n*	*M (SD)*	Common *SD*	*F(df)*	*p*
Drink					***F*(2,77)**	
Never	Safe/Hazardous	7	0.36 (0.22)			
Wine	Safe/Hazardous	23	0.36 (0.14)			
Wine	Harmful/Dependent	1	0.04 (0.38)			
Spirits	Safe/Hazardous	5	0.34 (0.12)			
Beer	Safe/Hazardous	4	0.38 (0.24)	0.93	3.64	*.031
Beer	Harmful/Dependent	5	0.17 (0.32)			
Mixture	Safe/Hazardous	26	0.39 (0.13)			
Mixture	Harmful/Dependent	6	0.40 (0.14)			
Other	Safe/Hazardous	9	0.41 (0.13)			
Pass out					***F*(2,77)**	
Abstain	Safe/Hazardous	7	0.40 (0.19)			
Never	Safe/Hazardous	52	0.37 (0.15)			
Never	Harmful/Dependent	4	0.43 (0.14)			
Not in year	Safe/Hazardous	2	0.41 (0.03)			
Less than monthly	Safe/Hazardous	12	0.37 (0.12)	0.95	5.85	**.004
Less than monthly	Harmful/Dependent	5	0.18 (0.29)			
Monthly	Safe/Hazardous	1	0.59 (0.09)			
Monthly	Harmful/Dependent	2	0.14 (0.33)			
Weekly	Harmful/Dependent	1	0.36 (0.23)			
Smoking					***F*(1,82)**	
Yes	Safe/Hazardous	15	0.39 (0.14)			
Yes	Harmful/Dependent	7	0.18 (0.29)	1.01	8.38	**.005
No	Safe/Hazardous	59	0.38 (0.15)			
No	Harmful/Dependent	5	0.41 (0.19)			

Substance related variable	**Alcohol group**	***n***	***M (SD)***	**Common *SD***	***F(df)***	***p***
Substance use					***F*(1,82)**	
Yes	Safe/Hazardous	5	0.44 (0.09)			
Yes	Harmful/Dependent	1	0.04 (0.38)	1.00	4.41	*.039
No	Safe/Hazardous	69	0.38 (0.15)			
No	Harmful/Dependent	11	0.29 (0.26)			

Note: *$p < .05$, **$p < .01$; For Cognitive Efficiency Index scores a high score is in the direction of good performance

a) First age

The Cognitive Efficiency Index scores were significantly affected by the First age and alcohol group interaction effects ($F(1, 81) = 16.83$, $p < .001$). The effect had a high power value that exceeded Cohen's cut-off (.98) and a large effect size value ($\eta_p^2 = .17$). The Tukey HSD test showed that the Harmful/Dependent drinkers who started drinking regularly in high school had a significantly lower mean Cognitive Efficiency Index score when compared to the Harmful/Dependent drinkers who started drinking regularly over the age of 18 ($p = .001$), as well as the Safe/Hazardous drinkers who have never started drinking regularly ($p < .001$), who started drinking regularly in high school ($p < .001$) and over the age of 18 ($p < .001$). This result indicates that the Harmful/Dependent drinkers who started drinking regularly in high school scored worse than the Harmful/Dependent drinkers who started drinking regularly over the age of 18 and the Safe/Hazardous drinkers who have not started drinking regularly and who started drinking regularly in higher school and over the age of 18. The mean and standard deviation values are shown in Table 23 above.

b) Drink

The Cognitive Efficiency Index scores were significantly affected by the Drink and alcohol group interaction effects ($F(2, 77) = 3.64$, $p = .031$). The power value was below Cohen's cut-off (.66) and had a medium to large effect size value ($\eta_p^2 = .09$). The Tukey HSD test showed that the Harmful/Dependent drinkers who consume beer had a significantly lower mean Cognitive Efficiency Index score when compared to the Safe/Hazardous drinkers who consume a mixture of alcoholic drinks ($p = .024$) and who consume other kinds of alcoholic drinks ($p = .040$). This result indicates that the Harmful/Dependent drinkers who consume beer most often scored worse than the Safe/Hazardous drinkers who consume a mixture of alcoholic beverages and who drink other types of alcoholic beverages. The mean and standard deviation values are shown in Table 23 above.

c) Pass out

The Cognitive Efficiency Index scores were significantly affected by the Pass out and alcohol group interaction effects ($F(2, 77) = 5.83$, $p = .004$). The power value was above Cohen's cut-off (.86) and the effect size was large ($\eta_p^2 = .13$). The Pass out and alcohol group interaction effects Tukey HSD test showed no significant results. The mean and standard deviation values are shown in Table 23 above.

d) Smoking

The Cognitive Efficiency Index scores were significantly affected by the Smoking and alcohol group interaction effects ($F(1, 82) = 8.35$, $p = .005$). The power value passed Cohen's cut-off (.81) and the effect size was medium to large ($\eta_p^2 = .09$). A Tukey HSD test showed that the Harmful/Dependent drinkers who smoke had significantly lower Cognitive Efficiency Index mean scores than the Harmful/Dependent drinkers who do not smoke ($p =$

.017), as well as the Safe/Hazardous drinkers who do smoke (p = .003) and who do not smoke (p = .001). This result indicates that the Harmful/Dependent drinkers who smoke scored worse than all of the other categories. The mean and standard deviation values are shown in Table 23 above.

e) Substance use

The Cognitive Efficiency Scores were significantly affected by the Substance use and alcohol group interaction effect ($F(1, 82) = 4.41$, p = .039). The effect had a low power value (.55) and a medium effect size (η_p^2 = .05). The Tukey HSD test showed that the Harmful/Dependent drinker who uses substances had a significantly lower overall Cognitive Efficiency Index mean score than the Safe/Hazardous drinkers who use substances (p = .038). This result indicates that the Harmful/Dependent drinker who uses substances scored worse than the Safe/Hazardous drinkers who use substances. The mean and standard deviation values are shown in Table 23 above.

Section 4: Summary

This section displayed the results from the statistical analyses of the data collected in this research. Three separate sets of repeated measures ANOVA tests were carried out to address the three research questions. The first set of analyses, that is the Cognitive analysis, included the ImPACT Cognitive mean scores over time with the alcohol group variable. These analyses revealed a significant alcohol group and time interaction effect for the Visual Motor Speed, Reaction Time and Cognitive Efficiency Index mean scores. The analyses also found that the Reaction Time and Cognitive Efficiency Index mean scores for the whole year were significantly related to the alcohol group variable without time as a factor.

The second set of analyses, that is the Symptom analysis, included the ImPACT Symptom mean scores over time with the alcohol group variable. These analyses revealed a significant alcohol group and time interaction effect for Numbness/Tingling symptom scores. The Balance problems, Dizziness and Numbness/Tingling symptom scores for the whole year revealed a significant effect for alcohol group without time as a factor.

The third set of analyses, that is the Exploratory analysis, included the ImPACT Composite scores over time with the alcohol group and substance related variables. This set of analyses, that is the Exploratory analysis, showed that Verbal memory was not significantly affected by any of the substance related variables in conjunction with the alcohol group variable and time. Visual memory was significantly affected by the interaction between Parental alcohol use, alcohol group and time. Visual motor speed was significantly affected by the interaction between Smoking, alcohol group and time, as well as an interaction between the Pass out and alcohol group variables. Reaction time was significantly affected by the interaction between alcohol group and time with the First drunk, First age, Drink and Substance use variables. Reaction time was also significantly affected by an interaction between the Pass out and alcohol group variables. The Cognitive Efficiency Index was significantly affected by the interaction between alcohol group and time with the First drunk, First age, Drink and Substance use variables. The Cognitive Efficiency Index was also significantly affected by the interaction between the alcohol group variable and the First drunk, Drink, Pass out, Smoking and Substance use variables. The only substance related variable that did not have a significant interaction effect with alcohol group, with or without time, was the Hangover variable.

Chapter 4: Discussion

This chapter is a discussion of the results that were reported in the previous chapter. It begins with a broad outline of the current study covering the aims, hypotheses, methodology and findings. The separate sets of analyses are considered in detail, followed by a synthesis of the discussion. The strengths and limitations of this study are also discussed, along with suggestions for further research.

Section 1: Broad outline of this study

This research aimed to ascertain whether there was a relationship between the alcohol use patterns of Rhodes University students and their cognitive and symptomatic functioning (the Cognitive and Symptom analyses, respectively), over a period of one academic year. An additional aim was to investigate whether any of the substance-related factors gathered from the relevant literature (First drunk, First age, Drink, Hangover, Pass out, Parental alcohol use, Smoking and Substance use) affected the suspected relationship between the university students' alcohol use patterns and their cognitive functioning (Exploratory analysis).

The data collection began with Psychology 2 and 3 Rhodes University students completing (i) a test to establish an estimate of initial intelligence/ cognitive reserve , the Peabody Picture Vocabulary Test version 4 (PPVT-4), and (ii) an alcohol use questionnaire, the Alcohol Use Disorders Identification Test (AUDIT), at the beginning of the 2012 academic year. The students' initial intelligence scores were used to ensure that the students were matched in terms of initial intelligence. The AUDIT assesses alcohol use patterns of the previous 12 months and gives a score out of 40 which is categorised into one of four groups: Safe, Hazardous, Harmful and Dependent. The students were split into two groups according to their alcohol use; Safe/Hazardous and Harmful/Dependent (see Section 2.1.1 p. 112 in the Methodology chapter). The Safe/Hazardous group included the range of scores of the lighter drinking AUDIT categories (Safe and Hazardous) while the Harmful/Dependent group included the range of scores of the two heavier drinking AUDIT categories (Harmful and Dependent). The sample was split into these two groups as the first includes all participants who are, by definition, not yet experiencing alcohol-related harms while the AUDIT scores of the second group suggest that they are experiencing such harms.

An initial sample of 216 Psychology 2 and 3 students completed the initial intelligence and alcohol testing for Phase 1. Following the Phase 1 exclusion criteria set out in Section 1.2.1 p. 94 in the Methodology chapter, 37 students were excluded. Students were excluded if they were too old, did not consent to Phase 2 or scored below an initial intelligence cut-off point that indicated borderline impaired functioning. The remaining students were then invited to be tested using the Immediate Postconcussion Assessment and Cognitive Test (ImPACT) battery at three different time intervals over the year (May, August, October), to measure their cognitive and symptomatic functioning over the year. The ImPACT battery gives cognitive scores for Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time, Impulse Control and Cognitive Efficiency Index. The battery also gives symptom scores for Headache, Nausea, Vomiting, Balance problems, Dizziness, Fatigue, Difficulty falling asleep, Sleeping more than usual, Sleeping less than usual, Drowsiness, Light sensitivity, Noise sensitivity, Irritability, Sadness, Nervousness, Emotional, Numbness, Mental slowness, Fogginess, Difficulty concentrating, Difficulty remembering and Visual problems, as well as a Total Symptom Score.

A total of 92 students completed the three ImPACT testing intervals in Phase 2. Following the Phase 2 exclusion criteria set out in Section 1.2.2 p. 95 in the Methodology chapter, six students were excluded before analyses began. The students were excluded if their alcohol use was erratic over the year, if they scored above the cut-off on the Impulse Control composite score or if they received former DET/Township education. As was discussed previously in the Literature Review chapter (Section 7.4, p.76), research has indicated that there is a lack of homogeneity within the African first language individuals in South Africa, with those who have been exposed to relatively disadvantaged former DET/ Township schooling performing poorly on cognitive tasks developed in America, whereas those exposed to more advantaged schooling performed at the American standardized levels (Shuttleworth-Edwards et al., 2004; Shuttleworth-Edwards, Gaylard et al., 2013; Shuttleworth-Edwards et al., 2009). The poor performance is confirmed in this study by the PPVT-4 raw and standard scores, and is considered to be an underestimate of true ability in that population. Therefore it was considered necessary to exclude these former DET/Township schooled individuals from the present study as their performance might contribute to deleterious outcomes on the ImPACT composite scores due to socio-cultural disadvantage, rather than inherent ability. The final sample consisted of 86 students. The Safe/Hazardous group consisted of 74 students and the Harmful/Dependent group of 12.

The two drinking groups were analysed using Chi-squared and *t*-tests for distribution of age, gender, English first language, race groups, history of neurological or psychiatric vulnerability (ADD/Hyperactivity, Learning disability, Psychiatric disorder, Grade repeated, Neurological disorder, Dyslexia and Concussion), years at Rhodes, a measure of initial intelligence/cognitive reserve (PPVT-4) (Dunn & Dunn, 2007) and for a measure of test-taking validity (ImPACT Impulse Control). It was considered that the alcohol groups were matched for the demographic, neurological and psychiatric variables as there were no significant differences between the groups on any of the variables on the tests that were run ($p > 0.05$ in all instances, see Sections 1.3 and 1.4 on p. 98 and p.100 respectively in the Methodology chapter).

Following on from the research aims, the methodology and the literature reviewed, it was hypothesised that the Safe/Hazardous drinkers would perform better on the ImPACT cognitive tests than the Harmful/Dependent drinkers. It was also hypothesised that practice effects would be seen in the Safe/Hazardous drinkers but not to the same extent in the Harmful/Dependent drinkers. It was expected that this differential practice effect between the two groups would be revealed if the Safe/Hazardous drinkers improved their performance significantly over the time intervals where the Harmful/Dependent drinkers did not. It was further hypothesized that the Safe/Hazardous drinkers would score lower on the symptom scores when compared to the Harmful/Dependent drinkers. In terms of the third aim, it was hypothesised that the substance-related moderating variables would have a significant effect on the alcohol use and cognitive functioning relationship.

The Cognitive and Symptom analyses were conducted using repeated measures ANOVA tests with the ImPACT Cognitive and Symptom scores over the three time intervals as dependent variables and the alcohol group variable as the dependent variable. The Exploratory analysis was conducted using repeated measures ANOVA tests with the ImPACT Cognitive scores over the three time intervals as dependent variables and the alcohol group and substance-related variables as dependent moderating variables. Significant results were further analysed using Tukey HSD tests.

The current chapter discusses the results that were reported in the previous chapter. Briefly, higher scores in the Verbal Memory, Visual Memory, Visual Motor Speed and Cognitive Efficiency Index composite scores indicate better performance on the tests. Higher scores for Reaction Time indicate worse performance on the tests. Higher scores on all of the Symptom scores indicate worse health.

The results from the Cognitive analysis showed that Verbal Memory and Visual Memory were not significantly affected by the alcohol group variable, either alone or combined with the time variable. The results from the Symptom Analysis showed that there were no significant results for Total Symptom Score, Headache, Nausea, Vomiting, Fatigue, Difficulty falling asleep, Sleeping more than usual, Sleeping less than usual, Drowsiness, Light sensitivity, Noise sensitivity, Irritability, Sadness, Nervousness, Emotional, Mental Slowness, Fogginess, Difficulty concentrating, Difficulty remembering and Visual problems. The results from the Exploratory analysis showed no significant results for Verbal and Visual Memory and no significant interaction effects with the Hangover variable. The Parental alcohol use variable, in combination with heavy alcohol use, significantly negatively affected the Visual Memory composite score, however, further analyses with a Tukey HSD test found no significant results.

The Cognitive analysis showed that heavier alcohol use was related to worse scores on the Visual Motor Speed, Reaction Time and Cognitive Efficiency Index scores. The Symptom analysis showed that heavier alcohol use was related to worse symptom scores for Numbness/Tingling, Balance problems and Dizziness. The Exploratory analysis showed that the First drunk, First age, Drink, Pass out and Substance use variables, in combination with heavier alcohol use, significantly negatively affected the Reaction Time and Cognitive Efficiency Index composite scores. The Smoking variable, in combination with heavy alcohol use, significantly negatively affected the Cognitive Efficiency Index score. The Pass out and Smoking variables, in combination with heavy alcohol use, significantly negatively affected the Visual Motor Speed composite score. These findings are discussed in more detail in the sections below.

Section 2: Cognitive Analysis

2.1 Cognitive Score Outcomes

The cognitive analysis showed that at the beginning of the research period, the alcohol groups did not differ significantly on any of the ImPACT cognitive scores. However, at the end of the research period, the alcohol groups differed significantly where the

Safe/Hazardous drinkers scored better than the Harmful/Dependent drinkers on the Reaction Time and Cognitive Efficiency Index composite scores. The Safe/Hazardous drinkers significantly improved their mean Visual Motor Speed and Cognitive Efficiency Index composite scores over the year. The Harmful/Dependent drinkers, conversely, significantly declined their performance on the Reaction Time and Cognitive Efficiency Index composite scores over the year. In terms of overall scores, the Safe/Hazardous drinkers performed significantly better than the Harmful/Dependent drinkers on the Visual Motor Speed, Reaction Time and Cognitive Efficiency Index composite scores.

Generally, the significant cognitive findings provide support for the hypothesis that the Harmful/Dependent group performed worse on the cognitive measures, especially relating to attentional aspects, compared to the Safe/Hazardous group and that there is a deleterious relationship between heavy alcohol use and cognitive functioning. The improvements seen in the Safe/Hazardous drinkers' mean scores and the declines in the Harmful/Dependent drinkers' mean scores are also supportive of the hypothesis concerning practice effects implicating neurocognitive vulnerability in the Harmful/Dependent drinkers that is not in evidence for the Safe/Hazardous drinkers.

The Cognitive Efficiency Index is a measure of effort as a combination of response speed and memory accuracy, while Reaction Time is an average response speed (ImPACT, 2007). Visual Motor Speed is a combination of visual motor response speed, processing, learning and memory. Thus, taken together, the results reported suggest that heavier patterns of alcohol use affect the visual aspects of response speed, memory, learning and processing, rather than the verbal aspects. The verbal aspects of learning, memory, processing and attention were not significantly affected in this sample and this time frame. As neither the Verbal Memory nor the Visual Memory composite scores were significantly affected by alcohol use patterns over time, it can be deduced that, in this sample and time frame, response speed and visual processing are more affected by heavier drinking than aspects of memory.

2.1.1 Response speed

Findings on reaction time or response speed are not consistent in previous alcohol-related cognitive research. For example, Sher et al. (1997) and Corley et al. (2011) found that heavy alcohol users scored worse than lighter drinkers. K. L. Hanson et al. (2011) found no difference between those with an alcohol use disorder and those without. Some researchers that measured response speed over time in alcohol use research did not explicitly state the findings regarding response speed, such as the research by Rodgers et al. (2005) and Smith and Fein (2010). This exclusion may indicate that no significant differences were found in those studies.

The current research showed that heavier alcohol use was related to worse scores for response speed, as measured by the ImPACT Reaction Time and Cognitive Efficiency Index scores. Some alcohol-related research has shown similar results, that response speed is slower in those who consume more, or those who have alcohol use disorders, compared to those who do not consume as much (Ferrett et al., 2010; Glass et al., 2009; Kopera et al., 2012; Rosenbloom et al., 2004; Sher et al., 1997; Thoma et al., 2011). Kopera et al. (2012) suggest that response speed deficits seen in alcohol-related cognitive functioning research could be due to the trade-off between accuracy and speed. Those who drink heavily may perform tests slower in order to maximise their accuracy. This can also be seen in the Harmful/Dependent group's poorer performance on the Cognitive Efficiency Index. The Cognitive Efficiency Index, as described in the ImPACT manual (2007), is an indicator of the amount of effort put in when taking the test and lower scores indicate less effort.

As the response speed findings in alcohol-related cognitive research are either missing or not discussed as much as other cognitive domains (for example memory and attention), the scope of this discussion was increased to include the broader aspects of response speed deficits in diffuse brain injury. Previous research on different types of brain injury has shown that visual processing speed is sensitive to diffuse brain injury such as TBI and concussion (Lezak et al., 2004). Concussion research has shown that response speed is increased, that is worsened, in those who have concussions, especially in the days after the concussion (Gardner et al., 2012; Makdissi et al., 2001). Particularly in testing using the ImPACT battery, response speed is worsened in rugby players, both those who are diagnosed with concussions and those who are not (Clark, 2010; Shuttleworth-Edwards et al., 2008; Shuttleworth-Edwards et al., 2014). It is well documented that processing speed is a particularly sensitive indicator of diffuse brain injury in general, which has been shown in concussion research using the ImPACT battery (Lezak et al., 2004; Shuttleworth-Edwards et al., 2014). This gives strength to the current findings that response speed is negatively affected by heavier alcohol use and that heavier alcohol use causes deleterious changes in association with diffuse brain injury.

2.1.2 Memory

In terms of memory deficits, the current research did not find any significant relationship between alcohol use patterns and memory test performance. In contrast, a body of previous research has found poorer performance in memory for those who have an alcohol use disorder (Ferrett et al., 2010; Kopera et al., 2012; Rosenbloom et al., 2004; Thoma et al., 2011). Conversely, commensurate with the null findings in the present study, a group of researchers including K. L. Hanson et al. (2011), Glass et al. (2009) and Sher et al. (1997) found no difference between those who have a history of alcohol use disorders and those who do not, for verbal learning and memory. However, K. L. Hanson et al. (2011) found that verbal memory and learning were significantly affected by alcohol use during the 10 year research period. This might suggest that heavier alcohol use over a longer period of time could cause damage to memory and that memory damage takes much longer than the time period of the present study. Similarly, it might be that the duration, rather than just the amount or pattern of alcohol use, affects the memory domains (Hermens et al., 2013; Tapert et al., 2005). The memory problems associated with alcohol use are generally attributed to alcohol's effects on the Glutamate neurotransmitter (Marshall et al., 2010) and the hippocampal region in the brain (Brown et al., 2009; Tapert et al., 2005; White, 2003). Thus, it could be that extended use of alcohol may cause problems with the hippocampal region and Glutamate neurotransmitter production over time.

It is also possible that the memory tests applied in this study were not sufficiently stringent to identify memory problems in this analysis. Clark (2010) also suggested this in research on visual motor speed and concussions. Clark (2010) used traditional paper and pencil memory tests in addition to the ImPACT battery to gain a more complete picture of memory damage in concussions.

If the isolated, and contrary, approaching-significance results are taken into account, the difference between the alcohol groups is in contrast to the rest of the current research findings, as the Harmful/Dependent drinkers scored better than the Safe/Hazardous drinkers on the Verbal Memory composite score overall. While the Verbal Memory composite findings in the current research approached significance, they did not have a large effect size. Also, the mean scores for the heavier drinkers were erratic, as they increased between the first and second intervals and then decreased in the third interval to below the mean score from the first interval. This suggests that this finding does not have the consistency to provide meaningful exploratory discussion of possible implications. This finding could also suggest that the heavier drinkers had the ability to perform better but did not put in the effort to do so, which is supported by the significantly lower overall Cognitive Efficiency Index scores for the Harmful/Dependent drinkers.

2.2 Implications of Cognitive Score Findings

In summary, the above findings suggest that heavier alcohol use has a significant effect on response speed in this particular sample but does not affect memory substantially, at least over a period of a single academic year. It also suggests that alcohol use has a minor effect on visual learning, processing, memory and attention. The findings on the present study are generally comparable with such prior research about the sensitivity of response speed and visual processing in the initial stages of incipient brain pathology, and it can be anticipated that if heavy drinking continues, damage will become clinically evident on testing in the areas of verbal memory and learning (K. L. Hanson et al., 2011).

2.2.1 Cognitive Implications

As previous research has suggested, diffuse brain injury produces deficits in response speed. Previous research has not covered the mechanisms behind these changes in response speed as the mechanisms are not as easily traced as, for example, those for certain memory types (hippocampal region). It could be suggested that the response speed effects seen in this research could be either a cognitive slowing, as suggested by Clark (2010) or a physical motor slowing. Alternatively, it is due to a combination of both of these factors since cognitive response speed is tested by means of the motor response required by clicking the mouse. In other words, either there is a mental slowing in the information processing speed in the brain itself, or there is a slowing in the movement of the body, such as the hands, in making the response. From the current research it is not possible to distinguish between the contributions of these two aspects to the results. It is possible that the use of paper and pencil tasks to measure response speed would be more susceptible to measuring the physical aspect, whereas this research used a computerised task which may serve to minimize the motor aspect and be more attuned to measuring the cognitive aspect. Therefore, in the current research, it seems likely that the cognitive aspect of response speed, rather than the physical aspect, was substantively negatively affected by heavier alcohol use.

2.2.2 Academic Implications

Previous research has suggested that alcohol use at university may impact academic work, for example Park (2004) and Rickwood et al. (2011) stated that between 11% and 13% of their samples had reported academic problems because of alcohol use. McGee and Kypri (2004) reported that their sample of university students reported being late for class, missing a class, not concentrating in class and not completing assignments. These are self-report findings. Aertgeerts and Buntinx (2002) performed analyses on alcohol abuse and dependence diagnoses, and pass rates for first year university students. The researchers found that there was a 25% increased risk of failing the academic year for those who fit the DSM-IV diagnosis for alcohol dependence. This previous research does not include what aspects of cognitive dysfunction may be the cause of academic problems. As a cautionary note, the current research does not specify causality, as students who perform poorly academically may be more inclined to consume more alcohol as a way of coping, which was also suggested by Hayatbakhsh et al. (2011), Karlamangla et al. (2006) and Peltzer and Ramlagan (2009). A similar argument was discussed by Lyvers (2000) who stated that it is unclear which comes first, the alcohol use or the damage in the brain. The current research, though, suggests that the damage worsens over time when heavy alcohol use is present. Moreover, a recent study on diffuse brain injury effects in association with participation in the contact sport of rugby reveals an associated decline in academic performance over the high school years (Alexander, Shuttleworth-Edwards, Kidd and Malcolm, in press). Diffuse brain impairment in association with heavy alcohol use in university learners over time might similarly be expected to show decline in academic achievement over time.

In terms of the current research findings, problems with response speed may affect how the students process and understand their academic work. The students may also take longer to complete assignments because it takes longer for them to process the topic and understand, firstly, what is required for the assignment and, secondly, the resources needed to complete the assignment. This reduced response speed and processing speed may also impact their exams because they may take longer to understand the questions and to form answers.

Even though the memory domains may be affected in later stages of heavy alcohol use, problems with memory may still occur during a student's academic career. The current results indicated that deficits appeared over a one year period, which suggests that deficits are likely to worsen as the students continue to consume alcohol at heavy levels. This will affect how they study for tests and exams and also remember previous years' work that may pertain to their current work.

Section 3: Symptom Analysis

3.1 Symptom Score Outcome

The Symptom analysis showed that the Harmful/Dependent drinkers reported significantly more severe symptoms relating to Numbness/Tingling, specifically at the end of the research. Overall, the Harmful/Dependent drinkers reported significantly worse symptoms relating to Numbness/Tingling, Balance problems and Dizziness. The symptom scores did not change significantly over the year for either group. In terms of the results that are approaching significance, the Harmful/Dependent drinkers experienced more severe symptoms than the Safe/Hazardous drinkers, particularly with the Nausea, Vomiting and Visual problem symptoms. The Harmful/Dependent group had higher Nausea symptom mean scores at all of the time intervals and increased their mean scores from interval 1 to interval 3. The Safe/Hazardous group had lower Nausea symptom mean scores at all of the time intervals and decreased their mean scores from interval 1 to interval 3. The Harmful/Dependent group had a higher Vomiting symptom mean score at interval 3 compared to the Safe/Hazardous group. The Safe/Hazardous group decreased their mean Visual problem mean scores from interval 1 to interval 3. In contrast, the Harmful/Dependent group changed their scores erratically and had a high interval 1 mean score, low interval 2 mean and a higher interval 3 score. These trends are consistent with the above significant findings and the hypothesis that the Harmful/Dependent drinkers experience more severe symptoms compared to the Safe/Hazardous group.

The significantly higher symptom scores for Dizziness, Balance problems and Numbness/Tingling shown by the Harmful/Dependent drinkers were also found in previous research (Barclay et al., 2008; McIntosh & Chick, 2004; Schuckit, 2009) (see Section 5.2.3 p. 38 of the Literature Review). These researchers suggested that the symptoms are caused by nervous system degeneration which could be caused by long-term heavy alcohol use. These symptoms are also similar to those experienced by those suffering from Wernicke-Korsakoff's syndrome (Kopelman et al., 2009; Lezak et al., 2004; Royal College of Psychiatrists, 1979; Zubaran et al., 1997). At this early stage the symptoms are fairly minor and may not lead to Wernicke-Korsakoff's syndrome, especially without the nutrition deficiency, specifically thiamine. As it is an early stage of the students' drinking careers, it is difficult to predict what sort of damage the alcohol may cause, that is, whether it may eventually lead to Wernicke-Korsakoff's syndrome or to another alcohol-related condition. As it stands, even though this student population is young and still relatively new in their drinking careers, their heavier patterns of alcohol use could be related to early signs of alcohol-related damage in the brain which are presented by the symptoms.

3.2 Implications of Symptom Score Findings

According to Swift and Davidson (1998), and Wiese et al. (2000), nausea, vomiting, dizziness, balance problems and visual problems are symptoms of hangover, which can last for several hours. Even though the students were requested not to consume alcohol in the 24 hours before the tests, they could still have been experiencing and reporting residual hangover symptoms from their previous alcohol consumption experience. This could indicate that the Harmful/Dependent drinkers' hangover symptoms last longer than the Safe/Hazardous drinkers' symptoms; that is longer than 24 hours. It may also suggest that Safe/Hazardous drinkers are less likely to experience hangovers. This indicates that the Harmful/Dependent drinkers may require more time to recover from their alcohol use.

The symptoms reported above may detract from the students' learning. Such symptoms are likely to affect concentration and, in more severe cases, reduce the likelihood that students will attend their lectures, study for tests or complete assignments. All of these symptoms may impede students in their academic efforts, resulting in poorer academic outcomes. These symptoms, and similar, could be contributing factors to the academic implications found in previous research, such as that by Park (2004) and Rickwood et al. (2011) which were discussed in the previous section (Section 2.2.2, p.172).

Section 4: Exploratory Analysis

4.1 Exploratory Analysis Outcomes

The results here are separated into two groups. One is the duration of alcohol use, which encompasses the First drunk and First age variables. The second group comprises the substance-related variables, which includes the Pass out, Drink, Substance use and Smoking variables. These will be discussed under the relevant sections below.

4.1.1 Duration of alcohol use

The current research findings showed that those Harmful/Dependent drinkers who first started drinking regularly in high school scored significantly worse on both the Reaction Time and Cognitive Efficiency Index composite scores. The Safe/Hazardous drinkers who started drinking regularly over the age of 18 scored significantly better and improved their scores significantly over the year on the Cognitive Efficiency Index composite scores. The Harmful/Dependent drinkers who were first intoxicated in primary school scored significantly worse on the Cognitive Efficiency Index composite score at interval 3 while the Safe/Hazardous drinkers who were first intoxicated in high school significantly improved their Cognitive Efficiency Index scores over the year.

These results are supportive of the hypothesis that the moderating factors will have an effect on the relationship between alcohol use and cognitive functioning. The results suggest that the length of alcohol use, as measured by the First drunk and First age variables, affects cognitive functioning. This is linked to the suggestion above (p. 171) that the duration of alcohol use along with the amount of alcohol consumed is linked to cognitive deficits (Hermens et al., 2013; Tapert et al., 2005). Previous and current research has found that the younger one starts drinking regularly, and the younger one gets intoxicated, the more one experiences cognitive damage; for example, consequences include lower levels of serotonin in the brain (Fils-Aime et al., 1996), neurological damage and cognitive deficits (Hermens et al., 2013) and smaller hippocampal regions (Tapert et al., 2005). The likely explanation is that the earlier a person starts drinking, the more they will have consumed compared to other people of the same age and same drinking habits who started drinking later. This supports the theory, mentioned above, that the reason that the present findings do not include memory impairments seen in other research, is because the current participants are young and their drinking histories still relatively short. Also the participants have not been diagnosed in the past with an alcohol use disorder. Thus their drinking is neither at a high enough level to be diagnosed as a disorder nor long enough to produce the damage seen in similar previous research.

4.1.2 Substance-related habits

The substance-related variables that were significantly related to cognitive functioning in the research were Pass out, Drink, Substance use, and Smoking.

Pass out:

A significant finding in this research is that those Harmful/Dependent drinkers who pass out monthly exhibited worse Reaction Time composite scores than the Safe/Hazardous drinkers. This is supportive of the hypothesis that the moderating factors will have an effect on the relationship between alcohol use and cognitive functioning. No research could be found that included an examination of the effects of multiple pass out incidents due to alcohol use on cognitive functioning, where a pass out is losing consciousness due to alcohol use. Rickwood et al. (2011) researched the harms associated with alcohol use, which included pass out incidents, but did not look at whether these were associated with cognitive impairment. The current research reported that almost three quarters of the sample had not passed out due to alcohol use or abstained from alcohol consumption. This indicates that almost a quarter had passed out at one point or another, which is higher than the figure of 7.6% presented in the research by Rickwood et al. (2011). Losing consciousness due to excess alcohol use is suggested to be due to alcohol’s effects on the GABA neurotransmitter.

Alcohol is said to increase GABA's inhibitory actions which enhances alcohol's sedentary actions (Barclay et al., 2008). According to the literature, a person will lose consciousness or pass out due to alcohol use when their BAC level is between 0.3 and 0.5 grams of alcohol per 100 millilitres of blood (Barclay et al., 2008). This suggests that almost a quarter of the current sample had BAC levels which reached at least 0.3 grams of alcohol per 100 millilitres of blood, which is a high BAC level. If the BAC level increases further, death may occur as the hind-brain areas, those that are responsible for the respiratory and circulatory systems, are depressed (Fort, 1973).

<u>Drink type</u>

The results indicated that at interval 3, the Harmful/Dependent drinkers who consumed beer and wine most often scored significantly lower on the Cognitive Efficiency Index composite score than the Safe/Hazardous and Harmful/Dependent drinkers who consumed a mixture of alcoholic beverages. The Harmful/Dependent drinker who consumed wine most often scored lower than those who consume other beverage types and the Safe/Hazardous drinkers. The Harmful/Dependent wine drinker also decreased their Cognitive Efficiency Index score over the time period. In terms of the overall group scores, the Harmful/Dependent beer drinkers scored lower than the Safe/Hazardous drinkers who consumed a mixture or other types of alcohol. These results indicate that the Harmful/Dependent drinkers who consumed wine and beer performed worse on the tests than those who consumed other beverage types and the Safe/Hazardous drinkers. These results are supportive of the hypothesis that the moderating factors will have an effect on the relationship between alcohol use and cognitive functioning.

Mortensen et al. (2001) found similar low scores for the beer drinkers, but opposite findings for the wine drinkers. Previous research found that wine had beneficial cognitive, and physical, effects (de Lorimier, 2000; Mortensen et al., 2001; Peregrin, 2005). The current research found that the Harmful/Dependent wine drinker scored significantly lower on some of the cognitive tests. As there was only one student in this category and that previous research has found contrary results, it suggests that the current finding is not strong and is not representative of the whole population.

While the cognitive and physical effects of wine are commonly researched, the cognitive effects of beer are not. Research reviewed by de Lorimer (2000) showed that while wine was related to less cognitive and physical decline due to aging, beer produced no significant cognitive effects, protective or harmful. Mortensen et al. (2001) and Corley et al. (2011) conversely found that beer was related to cognitive deficits as shown by lower cognitive test scores, which is supported by the current research findings.

Substance use and smoking

Smoking cigarettes significantly negatively affected both the Visual Motor Speed and Cognitive Efficiency Index composite scores. The Safe/Hazardous drinkers who do not smoke significantly improved their Visual Motor Speed composite scores across the time intervals. The Harmful/Dependent smokers scored significantly lower on the Cognitive Efficiency Index composite score compared to the Harmful/Dependent non-smokers and both Safe/Hazardous groups. The results of this research also showed that those Harmful/Dependent drinkers who use other substances scored worse in both composite scores. The Safe/Hazardous drinkers who do not use other substances scored better and improved their scores over the year, particularly on the Cognitive Efficiency Index composite scores. Again, these results are in support of the hypothesis that the moderating factors will have an effect on the relationship between alcohol use and cognitive functioning.

The findings are similar to the finding by Glass et al. (2009), that smoking and substance use in combination with alcohol use is related to a decrease in cognitive functioning, especially functions linked to response speed. However, the result that showed that the use of substances and the heavy use of alcohol together affect response speed is tenuous in this research as there was only one student who was in the Harmful/Dependent group and who reported using substances. Even though this research found a tenuous relationship, other research by Glass et al. (2009), Thoma et al. (2011) and Tapert and Brown (1999) also found that substance use in general was related to cognitive deficits. The current research only studied whether or not the students used substances, and not which substances they may be using. Also, substance use is illegal and as such, the students may have been reluctant to indicate that they were using illegal substances for fear of possible repercussions. Even given these considerations, a significant result was found that was in support of previous research findings and the hypothesis concerning the substance-related variables. Thus, poly-substance use, for example the use of alcohol, tobacco and illegal substances together, is more harmful than using just one substance.

4.2 Implications of Exploratory Analysis Findings

Overall, the implication of these findings is that longer life-time drinking duration, along with current heavy drinking practices, are related to more severe cognitive deficits. The past cannot be changed, however, the implication of the findings on the Pass out, Drink, Substance use and Smoking variables, is that the students may be able to alter the way that alcohol damages their brains. They can achieve this by changing their frequency and amount of alcohol use and their substance-related habits. By decreasing their overall alcohol consumption, the students could avoid pass out incidents, and improve their overall cognitive function. Previous research has shown that during abstinence, there is some improvement in overall cognitive functioning (Kopera et al., 2012; Manning et al., 2008; Rosenbloom et al., 2004). Aside from decreasing the amount that they drink, the students who do consume alcohol could improve their cognitive functioning by avoiding any other substances, including tobacco. The students could also avoid consuming beer and try other alcohol types.

Section 5: Synthesis and Implications

The findings suggest that response speed is the most affected at this stage while memory does not seem to be affected at this stage. It seems quite likely that the decline in memory, as seen in previous longer-term research, takes longer to develop, whereas the response speed and processing deficits develop quicker. This could explain why, over the period of a year, the memory composites were not significantly affected but the scores based on response speed and processing were. In light of literature that suggests that memory problems tend to develop later with alcohol use when compared to problems with processing speed (Glass et al., 2009; Sher et al., 1997), it can be assumed that if the students continue with their current drinking patterns, worse symptoms and cognitive functioning deficits could result, including impairment to the memory domains. It cannot be ruled out, however, that the memory tests on the ImPACT battery were not sensitive enough to detect incipient memory problems and that more probing memory tests might identify these. A longer duration of alcohol use, along with heavier alcohol use patterns, also seems to be related to cognitive deficits, as suggested by the First drunk and First age variable findings.

As mentioned above, the students’ cognitive functioning is not static. Even though their current cognitive status may be in danger, if they decrease their consumption and employ healthier drinking and substance-related habits, there is support from previous research to indicate that they may be able to regain some of the lost functions, reduce their symptom severity and improve on their cognitive functioning (Kopera et al., 2012; Manning et al., 2008; Rosenbloom et al., 2004). This will help them in their academic endeavours, as well as in their future professional careers. However, if they continue to consume alcohol at harmful levels, their symptoms may get worse and their cognitive deficits may increase and may lead to more severe neuro-degeneration and eventually more severe alcohol-related health problems.

In summary, this research indicates that the students' heavy drinking patterns may be related to cognitive deficits in response speed and processing, as well as certain physical symptoms, even at this early stage of drinking. It also indicates that the cognitive damage related to heavy drinking later on in life, may start at this stage. These cognitive deficits may have an effect on the students' academic work. As the students are at university to better themselves, the students should be made aware of any damage that they may be related to to their brains. This will hopefully enable them to make better choices in their alcohol use.

Students are unlikely to stop drinking entirely; however they may be persuaded to decrease the amount and frequency of their alcohol use and change some of their habits, such as smoking and substance use. They may be willing to ensure that they do not ever consume alcohol to the point of losing consciousness, if they know that this seriously affects the neurocognitive damage associated with drinking. They may also be willing to stop using other substances if they are made aware of the cognitive damage that substance use in combination with heavy alcohol use is likely to cause.

Section 6: Strengths of the study

This study, unlike most previous studies, involved a carefully controlled longitudinal design, an initial intelligence control and a sophisticated computerised test to achieve its aims. Previous research has not included all of these particular design features and control factors in one research study. The research also included practice effects as a diagnostic tool, which previous similar research has not.

Previous research has not reported or used values that indicate statistical power, for example, effect sizes and observed power values. The current research has reported these power values to increase the strength of the findings. As stated in the Methodology chapter (see Section 3.2, p. 128), statistical power values provide an additional way to interpret statistical analyses. In combination with significance values, observed power values and effect sizes provide more certainty to the clinical significance of the analyses and interpretation.

This research controlled for multiple factors, thus reducing the chance that they could exert confounding effects on the cognitive functioning and alcohol use relationship. These factors were an estimate of initial intelligence/ cognitive reserve (as measured by the PPVT-4 raw and standard scores), gender, race and first language. The research also included students who are normally excluded from similar research because they have a history of any one of the following: ADD/hyperactivity, learning disability, psychiatric disorder, repeated grade, neurological disorder, dyslexia or concussion. By controlling these factors, that is ensuring there was an even distribution of each factor in each alcohol group, the research sample was larger and included a diverse range of students. In terms of cognitive reserve theory all of these factors would serve as risk factors to increase the likelihood of additional brain injury with alcohol use (see Literature Review Section 6, p. 45). Therefore the ability to include these at risk individuals in the sample in a controlled manner across the comparative groups, allowed for capturing signs of brain impairment more effectively.

Various substance-related factors were included in this research (First drunk, First age, Drink, Hangover, Pass out, Parental alcohol use, Smoking and Substance use). Previous research had not included all of them in one study. These factors were gained from a multitude of research studies on alcohol use and its effects on cognitive functioning. Previous research that was reviewed also had not investigated the effects of pass out incidents on cognitive functioning, where the current research did.

This study included symptom outcomes using the ImPACT Symptom scale as well as the cognitive composite measures. By investigating both the cognitive and symptom functioning of the students, a more integrated view of alcohol-related diffuse brain changes can be seen. This comprehensive view of the functioning of the students strengthens the study and its findings.

Section 7: Limitations of the study

The limitations for this study include sample choice and time frame, among others. Even though the distribution of the alcohol groups was similar to previous research conducted at Rhodes University, there were very few Harmful/Dependent drinkers in the sample (12 students). If more students were included, maybe stronger relationships could have been found.

Importantly, as there were very small numbers of participants in the subgroups when investigating the various moderating factors, it is accepted that in many instances this would preclude the assumption of normality being met. Therefore, the investigation of these aspects must be considered exploratory and cautiously interpreted, with a view to providing pointers for possible further investigation.

The sample pool only consisted of second and third year Psychology students. This is highly focused and could be expanded to include other faculties at Rhodes University as well as students from other universities around South Africa. By using a more diverse range of students, the results could be generalised to the university student population in South Africa.

Previous similar research was conducted over multiple years. As such, this research period is considered quite short, only spanning a single academic year. Perhaps if the research period was longer, more changes and differences could have been detected. Also, memory deficits could possibly be detected in research with a longer duration.

This research demonstrated more significant results on the Visual Motor Speed, Reaction Time and Cognitive Efficiency Index composite scores than in the Verbal and Visual Memory composite scores. Thus, the memory tests in the ImPACT battery may not have been sufficiently sensitive to find subtle memory impairments in this sample. The Verbal memory tasks in the ImPACT are also visual, as the test takers see the words on the screen. Thus, visual recall may play a part in the recall of the words, therefore rendering the test less sensitive to verbal memory deficits. Therefore, in future research, additional memory tests could be utilised in order to gain a greater understanding of the memory impairments that may be present in this sample. The test developers may also wish to develop the memory tests further if they wish to expand the test's usage beyond that of concussion management.

This research did not use Bonferroni's adjustment on the level of significance. This adjustment is usually used in multiple analyses studies to control for increased rates of Type I errors (Tredoux & Durrheim, 2002). It was not used in this research because, as argued in Clark's (2010) research, it is likely to increase the risk of committing Type II errors.

A criticism of similar research comparing different groups could be that the differences found in the cognitive and symptomatic functioning may be the result of pre-existing differences, that is, other than exposure or non-exposure to harmful alcohol use. However, this is partly eliminated in this study by (i) implementing a baseline assessment for each student, and (ii) statistically matching the two alcohol groups on variables of gender, race groups, English first language, neurological vulnerability presence, initial intelligence/

cognitive reserve (as measured by the PPVT-4 raw and standard scores), age and years at Rhodes University.

Section 8: Future research

This particular sample was used in an exploratory analysis of how the substance-related variables impacted the alcohol use and cognitive functioning relationship. Future research could expand on this by including more participants in those categories where there was only one participant or very few participants or where a category had no students. A larger sample would also give strength to the current findings if similar results were found. Additionally, the sample could be expanded to include other faculties and other universities.

This study was conducted over a single academic year, while others studied participants over a longer period of time. An extended study could be conducted by contacting the students who participated in this research. The students could complete the AUDIT and ImPACT battery again to ascertain if their alcohol use has changed since leaving the university and if their cognitive functioning scores have changed due to either time or alcohol use. Additionally, a new sample could be studied and tested at the beginning of every university year while they are in university attendance.

Another relevant aspect to consider is that the changes in cognitive functioning may have an effect on the students' academic performance. Further research could be done to see whether there were any changes in the students' academic grades that correlated with their alcohol use over the year and their cognitive functioning scores. Their academic performance from previous years could also be analysed to see if their performance has improved or declined.

In terms of the statistical analyses, further analyses could be conducted using the Bonferroni's correction. This would be achieved by dividing the significance value by the number of comparisons (Tredoux & Durrheim, 2002). This would aid in avoiding committing Type I errors, but would increase the risk of committing Type II errors.

Future research could include traditional paper and pencil tests which may be more sensitive in measuring the memory domains. For example, Clark's (2010) research included the ImPACT battery, along with the Visual Reproduction and Verbal Paired Associates subtests from the WMS-III. Other research that included the ImPACT and traditional paper and pencil cognitive tests include Conklin et al. (2013), Shuttleworth-Edwards et al. (2008) and Tsushima et al. (2013). Including both computerised and paper and pencil tests may give a more sensitive view of the memory domains, especially verbal memory.

Chapter 5: Final Word

Young adulthood is a period of great adjustment, where young adults mature cognitively, socially and physically. University students, in particular, are expanding their minds by learning new subjects and expanding their social circles by moving from a school environment to a university environment. Through this social expansion period, students experience reduced parental influence and increased social pressure, which creates more situations for alcohol consumption, which in turn may lead to problematic drinking habits.

These problematic alcohol use habits may in turn hamper young adult development, especially cognitively as previous research has shown. The current results reinforce findings of the previous research, broadly confirming concerns that heavier drinking patterns among university students implicate deleterious cognitive and symptomatic consequences that have distressing implications for their academic achievements and long term neurological health. The particular results in this research suggest that response speed and visual, rather than verbal processing tasks are negatively affected by heavier alcohol use in a one year period, while memory domains seem to take longer to be affected. These cognitive problems may present in students failing to complete assignments or tests on time as well as taking longer than lighter drinking students to understand course topics. The symptomatic problems may also affect the students negatively by distracting them during study times and preventing them from going to lectures.

On a positive note, previous research has indicated that alcohol-related cognitive damage may be reversible if the students decrease their drinking levels and change some of their substance-related habits, for example reducing or discontinuing their smoking and substance use. Given these findings, and that students are at university primarily to learn, it seems imperative to encourage the development of intervention programmes with a view to persuading and assisting students to reduce their drinking levels and change their drinking habits. The deleterious findings from the present study have prime relevance for the purposes of future interventions of this type. The provision of research-based, reality tested outcomes from the present study, in conjunction with that of other studies implicating similar concerns, can act as a powerful deterrent while helping students to make more appropriate choices in terms of their social habits.

References

Abels, M. D. (2007). Assessing the high-risk behaviour of first year students entering the University of the Western Cape. (Unpublished Master's thesis). University of the Western Cape, Cape Town.

Aertgeerts, B., & Buntinx, F. (2002). The relation between alcohol abuse or dependence and academic performance in first-year college students. *Journal of Adolescent Health, 31*(3), 223-225. doi:10.1016/S1054-139X(02)00362-2

Alant, E., & Beukes, S. M. (1986). The application of the Peabody Picture Vocabulary Test-Revised (PPVT-R) to non-mainstream children. *The South African Journal of Communication Disorders. Die Suid-Afrikaanse Tydskrif Vir Kommunikasieafwykings*, 33, 7-14.

Alexander, D.G., Shuttleworth-Edwards, A.B., Kidd, M., & Malcolm, C.M. (In press). Mild traumatic brain injuries in early adolescent rugby players: Long-term neurocognitive and academic outcomes. *Brain Injury*.

Allen, J. P., Litten, R. Z., Fertig, J. B., & Babor, T. (1997). A review of research on the alcohol use disorders identification test (AUDIT). *Alcoholism: Clinical and Experimental Research, 21*(4), 613-619. doi:10.1111/j.1530-0277.1997.tb03811.x

American Psychiatric Association. (2013). Substance-related and addictive disorders [electronic]. *American Psychological Association,* 9 October 2013. Retrieved from http://www.dsm5.org/Documents/Substance%20Use%20Disorder%20Fact%20Sheet.pdf

American Psychiatric Press. (2000). *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR* (4th ed.). Washington, DC: Author.

Anderson, S., & Dawson, J. (1999). Neuropsychological correlates of alcoholic hangover. *South African Journal of Science, 95*(3), 145-147.

Andersson, C., Johnsson, K. O., Berglund, M., & Ojehagen, A. (2007). Alcohol involvement in Swedish university freshmen related to gender, age, serious relationship and family history of alcohol problems. *Alcohol and Alcoholism, 42*(5), 448-455. doi:10.1093/alcalc/agm008

Arnett, J. J. (2001). Conceptions of the transition to adulthood: Perspectives from adolescence through midlife. *Journal of Adult Development, 8*(2), 133-143. doi:10.1023/A:1026450103225

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). *The Alcohol Use Disorders Identification Test: Guidelines for use in primary care* (2nd ed.). Switzerland: World Health Organisation.

Barclay, G. A., Barbour, J., Stewart, S., Day, C. P., & Gilvarry, E. (2008). Adverse physical effects of alcohol misuse. *Advances in Psychiatric Treatment*, *14*(2), 139-151. doi:10.1192/apt.bp.105.001263

Bava, S., & Tapert, S. F. (2010). Adolescent brain development and the risk for alcohol and other drug problems. *Neuropsychology Review*, *20*(4), 398-413. doi:10.1007/s11065-010-9146-6

Becker, J. T., Dew, M. A., Aizenstein, H. J., Lopez, O. L., Morrow, L., & Saxton, J. (2011). Concurrent validity of a computer-based cognitive screening tool for use in adults with HIV disease. *AIDS Patient Care and STDs*, *25*(6), 351-357. doi:10.1089/apc.2011.0051

Bednarski, S. R., Erdman, E., Luo, X., Zhang, S., Hu, S., & Li, C. R. (2012). Neural processes of an indirect analog of risk taking in young nondependent adult alcohol drinkers: An fMRI study of the stop signal task. *Alcoholism: Clinical and Experimental Research*, *36*(5), 768-779. doi:10.1111/j.1530-0277.2011.01672.x

Berger, K. S. (2001). *The developing person through the life span* (5th ed.). New York: Worth.

Betz, S. K., Eickhoff, J. R., & Sullivan, S. F. (2013). Factors influencing the selection of standardized tests for the diagnosis of specific language impairment. *Language, Speech & Hearing Services in Schools*, *44*(2), 133-146. doi:10.1044/0161-1461(2012/12-0093

Birch, A. (1997). *Developmental psychology: From infancy to adulthood* (2nd ed.). Basingstoke: Macmillan.

Blakemore, S., & Choudhury, S. (2006). Development of the adolescent brain: Implications for executive function and social cognition. *Journal of Child Psychology & Psychiatry, 47*(3), 296-312. doi:10.1111/j.1469-7610.2006.01611.x

Blanco, C., Okuda, M., Wright, C., Hasin, D. S., Grant, B. F., Liu, S. M., & Olfson, M. (2008). Mental health of college students and their non-college-attending peers: Results from the national epidemiologic study on alcohol and related conditions. *Archives of General Psychiatry, 65*(12), 1429-1437. doi:10.1001/archpsyc.65.12.1429

Blanton, C. A., Green, M. W., & Kretsch, M. J. (2013). Body iron is associated with cognitive executive planning function in college women. *British Journal of Nutrition, 109*(5), 906-913. doi:10.1017/S0007114512002620

Borovsky, A., Elman, J. L., & Fernald, A. (2012). Knowing a lot for one's age: Vocabulary skill and not age is associated with anticipatory incremental sentence interpretation in children and adults. *Journal of Experimental Child Psychology, 112*(4), 417-436. doi:10.1016/j.jecp.2012.01.005

Borsari, B., & Carey, K. B. (2003). Descriptive and injunctive norms in college drinking: A meta-analytic integration. *Journal of Studies on Alcohol, 64*(3), 331-341.

Borsari, B., Murphy, J. G., & Barnett, N. P. (2007). Predictors of alcohol use during the first year of college: Implications for prevention. *Addictive Behaviors, 32*(10), 2062-2086. doi:10.1016/j.addbeh.2007.01.017

Bowman, B., Seedat, M., Duncan, N., & Burrows, S. (2006). 'Race', social transformation and redress in the South African social and health sciences. In G. Stevens, V. Franchi & T. Swart (Eds.), *A race against time: Psychology and challenges to deracialisation in South Africa* (pp. 91-103). Pretoria: Unisa Press.

Britton, A., Singh-Manoux, A., & Marmot, M. (2004). Alcohol consumption and cognitive function in the Whitehall II study. *American Journal of Epidemiology, 160*(3), 240-247. doi:10.1093/aje/kwh206

Broglio, S. P., Macciocchi, S. N., & Ferrara, M. S. (2007). Neurocognitive performance of concussed athletes when symptom free. *Journal of Athletic Training, 42*(4), 504-508.

Brown, S. A., McGue, M., Maggs, J. L., Schulenberg, J. E., Hingson, R., Swartzwelder, S., . . . Murphy, S. (2009). Underage alcohol use: Summary of developmental processes and mechanisms: Ages 16–20. *Alcohol Research & Health, 32*(1), 41-52.

Brown, S. A., Tapert, S. F., Granholm, E., & Delis, D. C. (2000). Neurocognitive functioning of adolescents: Effects of protracted alcohol use. *Alcoholism: Clinical and Experimental Research*, *24*(2), 164-171. doi:10.1111/j.1530-0277.2000.tb04586.x

Casey, B. J., Giedd, J. N., & Thomas, K. M. (2000). Structural and functional brain development and its relation to cognitive development. *Biological Psychology, 54*(1–3), 241-257. doi:10.1016/S0301-0511(00)00058-2

Castaneda, R., & Galanter, M. (1988). Ethnic differences in drinking practices and cognitive impairment among detoxifying alcoholics. *Journal of Studies on Alcohol and Drugs, 49*(4), 335-339.

Chastain, G. (2006). Alcohol, neurotransmitter systems, and behavior. *Journal of General Psychology, 133*(4), 329-335. doi:10.3200/GENP.133.4.329-335

Clark, S. B. (2010). Neurocognitive and symptom profiles of concussed and nonconcussed provincial rugby union players over one season. (Unpublished Doctoral dissertation). Rhodes University, Grahamstown.

Clausen, T., Rossow, I., Naidoo, N., & Kowal, P. (2009). Diverse alcohol drinking patterns in 20 African countries. *Addiction, 104*(7), 1147-1154. doi:10.1111/j.1360-0443.2009.02559.x

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155-159. doi:10.1037/0033-2909.112.1.155

Colby, S. M., Colby, J. J., & Raymond, G. A. (2009). College versus the real world: Student perceptions and implications for understanding heavy drinking among college students. *Addictive Behaviors, 34*(1), 17-27. doi:10.1016/j.addbeh.2008.07.023

Collie, A., Darby, D., & Maruff, P. (2001). Computerised cognitive assessment of athletes with sports related head injury. *British Journal of Sports Medicine, 35*(5), 297-302. doi:10.1136/bjsm.35.5.297

Collie, A., Maruff, P., Darby, D. G., & McStephen, M. (2003). The effects of practice on the cognitive test performance of neurologically normal individuals assessed at brief test–retest intervals. *Journal of the International Neuropsychological Society, 9*(03), 419-428. doi:10.1017/S1355617703930074

Conklin, H. M., Ashford, J. M., Di Pinto, M., Vaughan, C. G., Gioia, G. A., Merchant, T. E., . . . Wu, S. (2013). Computerized assessment of cognitive late effects among adolescent brain tumor survivors. *Journal of Neuro-Oncology, 113*(2), 333-340. doi:10.1007/s11060-013-1123-5

Cooper, M. L. (1994). Motivations for alcohol use among adolescents: Development and validation of a four-factor model. *Psychological Assessment, 6*(2), 117-128. doi:10.1037/1040-3590.6.2.117

Corley, J., Jia, X., Brett, C. E., Gow, A. J., Starr, J. M., Kyle, J. A. M., . . . Deary, I. J. (2011). Alcohol intake and cognitive abilities in old age: The Lothian birth cohort 1936 study. *Neuropsychology, 25*(2), 166-175. doi:10.1037/a0021571

Corral, M., Rodríguez, M., Amenedo, E., Sánchez, J. L., & Díaz, F. (2006). Cognitive reserve, age, and neuropsychological performance in healthy participants. *Developmental Neuropsychology, 29*(3), 479-491. doi:10.1207/s15326942dn2903_6

Cox, W. M., & Klinger, E. (1988). A motivational model of alcohol use. *Journal of Abnormal Psychology, 97*(2), 168-180. doi:10.1037/0021-843X.97.2.168

Crawford, J. R., Parker, D. M., & McKinlay, W. W. (Eds.). (1992). *A handbook of neuropsychological assessment*. United Kingdom: Lawrence Erlbaum Associates Ltd.

Cysique, L. A. J., Maruff, P., Darby, D., & Brew, B. J. (2006). The assessment of cognitive function in advanced HIV-1 infection and AIDS dementia complex using a new computerised cognitive test battery. *Archives of Clinical Neuropsychology, 21*(2), 185-194. doi:10.1016/j.acn.2005.07.011

Darke, S. (1988). Anxiety and working memory capacity. *Cognition and Emotion, 2*(2), 145-154. doi:10.1080/02699938808408071

Dawson, K. S., Batchelor, J., Meares, S., Chapman, J., & Marosszeky, J. E. (2007). Applicability of neural reserve theory in mild traumatic brain injury. *Brain Injury, 21*(9), 943-949. doi:10.1080/02699050701553171

de Lorimier, A. A. (2000). Alcohol, wine, and health. *The American Journal of Surgery, 180*(5), 357-361.

de Witte, P., Pinto, E., Ansseau, M., & Verbanck, P. (2003). Alcohol and withdrawal: From animal research to clinical issues. *Neuroscience & Biobehavioral Reviews, 27*(3), 189-197. doi:10.1016/S0149-7634(03)00030-7

Demetriou, A., & van Lieshout, C., F.M. (1998). *Life-span developmental psychology* (2nd ed.). New York: John Wiley.

Dick, D. M., & Foroud, T. (2006). Candidate genes for alcohol dependence: A review of genetic evidence from human studies. *Alcoholism: Clinical and Experimental Research*, *27*(5), 868-879.

Duff, K. (2012). Evidence-based indicators of neuropsychological change in the individual patient: Relevant concepts and methods. *Archives of Clinical Neuropsychology : The Official Journal of the National Academy of Neuropsychologists, 27*(3), 248-261. doi:10.1093/arclin/acr120

Duff, K., Beglinger, L. J., Schultz, S. K., Moser, D. J., McCaffrey, R. J., Haase, R. F., . . . Paulsen, J. S. (2007). Practice effects in the prediction of long-term cognitive outcome in three patient samples: A novel prognostic index. *Archives of Clinical Neuropsychology, 22*(1), 15-24. doi:10.1016/j.acn.2006.08.013

Duff, K., Callister, C., Dennett, K., & Tometich, D. (2012). Practice effects: A unique cognitive variable. *The Clinical Neuropsychologist*, *26*(7), 1117-1127. doi:10.1080/13854046.2012.722685

Dunn, L. M., & Dunn, D. M. (2007). *Peabody Picture Vocabulary Test, fourth edition, manual*. San Antonio, Texas: Pearson.

Eisenberg, M. A., Andrea, J., Meehan, W., & Mannix, R. (2013). Time interval between concussions and symptom duration. *Pediatrics, 132*(1), 8-17. doi:10.1542/peds.2013-0432

Elbin, R., Kontos, A. P., Kegel, N., Johnson, E., Burkhart, S., & Schatz, P. (2013). Individual and combined effects of LD and ADHD on computerized neurocognitive concussion test performance: Evidence for separate norms. *Archives of Clinical Neuropsychology, 28*(5), 476-484. doi:10.1093/arclin/act024

El-Guebaly, N. (2007). Investigating the association between moderate drinking and mental health. *Annals of Epidemiology, 17*(5), S55-S62.

Elias, P. K., Elias, M. F., D'Agostino, R. B., Silbershatz, H., & Wolf, P. A. (1999). Alcohol consumption and cognitive performance in the Framingham Heart study. *American Journal of Epidemiology, 150*(6), 580-589.

Falleti, M. G., Maruff, P., Collie, A., & Darby, D. G. (2006). Practice effects associated with the repeated assessment of cognitive function using the CogState battery at 10-minute, one week and one month test-retest intervals. *Journal of Clinical and Experimental Neuropsychology, 28*(7), 1095-1112. doi:10.1080/13803390500205718

Fazio, V. C., Lovell, M. R., Pardini, J. E., & Collins, M. W. (2007). The relation between post concussion symptoms and neurocognitive performance in concussed athletes. *Neurorehabilitation, 22*(3), 207-216.

Fils-Aime, M., Eckardt, M. J., George, D. T., Brown, G. L., Mefford, I., & Linnoila, M. (1996). Early-onset alcoholics have lower cerebrospinal fluid 5-hydroxyindoleacetic acid levels than late-onset alcoholics. *Archive of General Psychiatry, 53*, 211-216.

Finnigan, F., Hammersley, R., & Cooper, T. (1998). An examination of next-day hangover effects after a 100 mg/100 ml dose of alcohol in heavy social drinkers. *Addiction, 93*(12), 1829-1838. doi:10.1080/09652149834360

Fisher, K. (2009). *Working memory in South Africa.* (Unpublished Master's thesis). University of Witwatersrand, Johannesburg.

Fleming, M. F., Barry, K. L., & MacDonald, R. (1991). The alcohol use disorders identification test (AUDIT) in a college sample. *International Journal of Addictions, 26*, 1173-1185.

Fort, J. (1973). *Alcohol: Our biggest problem*. New York: McGraw-Hill.

Fox, B. A., & Cameron, A. G. (1970). *Food science: A chemical approach* (2nd ed.). London: Hodder & Stoughton.

Fray, P. J., & Robbins, T. W. (1996). CANTAB battery: Proposed utility in neurotoxicology. *Neurotoxicology and Teratology, 18*(4), 499-504. doi: 10.1016/0892-0362(96)00027-X

Friedman, T. W., Robinson, S. R., & Yelland, G. W. (2011). Impaired perceptual judgment at low blood alcohol concentrations. *Alcohol, 45*(7), 711-718. doi:10.1016/j.alcohol.2010.10.007

Gardner, A., Shores, E. A., Batchelor, J., & Honan, C. A. (2012). Diagnostic efficiency of ImPACT and CogSport in concussed rugby union players who have not undergone baseline neurocognitive testing. *Applied Neuropsychology: Adult, 19*(2), 90-97. doi:10.1080/09084282.2011.643945

Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2009). *Cognitive neuroscience: The biology of the mind* (3rd ed.). New York: Norton.

Giedd, J. N. (2008). The teen brain: Insights from neuroimaging. *Journal of Adolescent Health, 42*(4), 335-343. doi:10.1016/j.jadohealth.2008.01.007

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., . . . Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2*(10), 861-863.

Gill, J. S. (2002). Reported levels of alcohol consumption and binge drinking within the UK undergraduate student population over the last 25 years. *Alcohol and Alcoholism, 37*(2), 109-120.

Glass, J. M., Buu, A., Adams, K. M., Nigg, J. T., Puttler, L. I., Jester, J. M., & Zucker, R. A. (2009). Effects of alcoholism severity and smoking on executive neurocognitive function. *Addiction, 104*(1), 38-48. doi:10.1111/j.1360-0443.2008.02415.x

Glenwright, M., & Agbayewa, A. S. (2012). Older children and adolescents with high-functioning autism spectrum disorders can comprehend verbal irony in computer-mediated communication. *Research in Autism Spectrum Disorders, 6*(2), 628-638.

Goble, D. (2013). The impact of low to moderate alcohol consumption on different types of human performance. (Unpublished Master's thesis). Rhodes University, Grahamstown.

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., . . . Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences of the United States of America, 101*(21), 8174-8179.

Granville-Chapman, J. E., Yu, K., & White, P. D. (2001). A follow-up survey of alcohol consumption and knowledge in medical students. *Alcohol and Alcoholism, 36*(6), 540-543. doi:10.1093/alcalc/36.6.540 540-543

Grattan-Miscio, K., & Vogel-Sprott, M. (2005). Effects of alcohol and performance incentives on immediate working memory. *Psychopharmacology, 181*(1), 188-196. doi:10.1007/s00213-005-2226-2

Gray, S., Plante, E., Vance, R., & Henrichsen, M. (1999). The diagnostic accuracy of four vocabulary tests administered to preschool-age children. *Language, Speech & Hearing Services in Schools, 30*(2), 196.

Guillot, C. R., Fanning, J. R., Bullock, J. S., McCloskey, M. S., & Berman, M. E. (2010). Effects of alcohol on tests of executive functioning in men and women: A dose response examination. *Experimental and Clinical Psychopharmacology, 18*(5), 409-417. doi:10.1037/a0021053

Guthrie, J. T., & Seifert, M. (1977). Letter–sound complexity in learning to identify words. *Journal of Educational Psychology, 69*(6), 686-696. doi: 10.1037/0022-0663.69.6.686

Halperin, J. M., Trampush, J. W., Miller, C. J., Marks, D. J., & Newcorn, J. H. (2008). Neuropsychological outcome in adolescents/young adults with childhood ADHD: Profiles of persisters, remitters and controls. *Journal of Child Psychology and Psychiatry, 49*(9), 958-966. doi:10.1111/j.1469-7610.2008.01926.x

Halstead, M. E., Walter, K. D., & The Council on Sports Medicine and Fitness. (2010). Sport-related concussion in children and adolescents. *Pediatrics, 126*(3), 597-615. doi:10.1542/peds.2010-2005

Ham, L. S., & Hope, D. A. (2003). College students and problematic drinking: A review of the literature. *Clinical Psychology Review, 23*(5), 719-759.

Hanson, G., Venturelli, P. J., & Fleckenstein, A. E. (2008). *Drugs and society*. Burlington, MA: Jones and Bartlett Publishers.

Hanson, K. L., Medina, K. L., Padula, C. B., Tapert, S. F., & Brown, S. A. (2011). Impact of adolescent alcohol and drug use on neuropsychological functioning in young adulthood: 10-year outcomes. *Journal of Child & Adolescent Substance Abuse, 20*(2), 135-154. doi:10.1080/1067828X.2011.555272

Hartshorne, C. (2000). *The effects of alcoholic hangover on human performance.* (Unpublished Master's thesis). University of Natal, Pietermaritzburg.

Hayatbakhsh, M. R., Najman, J. M., Bor, W., Clavarino, A., & Alati, R. (2011). School performance and alcohol use problems in early adulthood: A longitudinal study. *Alcohol, 45*(7), 701-709. doi:10.1016/j.alcohol.2010.10.009

Health Professions Council of South Africa. (2010). *The professional board for psychology health professions council of South Africa list of tests classified as being psychological tests.*. Unpublished manuscript. Retrieved 1 August 2011, Retrieved from http://www.hpcsa.co.za/downloads/psychology/psychom_form_207.pdf

Hermens, D. F., Lagopoulos, J., Tobias-Webb, J., De Regt, T., Dore, G., Juckes, L., . . . Hickie, I. B. (2013). Pathways to alcohol-induced brain impairment in young people: A review. *Cortex, 49*(1), 3-17. doi:10.1016/j.cortex.2012.05.021

Herting, M. M., Fair, D., & Nagel, B. J. (2011). Altered fronto-cerebellar connectivity in alcohol-naive youth with a family history of alcoholism. *Neuroimage, 54*(4), 2582-2589.

Hessen, E., Nestvold, K., & Anderson, V. (2007). Neuropsychological function 23 years after mild traumatic brain injury: A comparison of outcome after paediatric and adult head injuries. *Brain Injury, 21*(9), 963-979. doi:10.1080/02699050701528454

Hingson, R. W., Zha, W., & Weitzman, E. R. (2009). Magnitude of and trends in alcohol-related mortality and morbidity among US college students ages 18-24, 1998-2005. *Journal of Studies on Alcohol and Drugs. Supplement,* (Supplement no. 16), 12-20.

Horsman, M. (2010). Concussion in contact sport: Investigating the neurocognitive profile of Afrikaans adolescent rugby players. (Unpublished Master's thesis). Rhodes University, Grahamstown.

Humensky, J. L. (2010). Are adolescents with high socioeconomic status more likely to engage in alcohol and illicit drug use in early adulthood? *Substance Abuse Treatment, Prevention, and Policy, 5*(19), 1-10.

Ilan, A. B., & Gevins, A. (2001). Prolonged neurophysiological effects of cumulative wine drinking. *Alcohol, 25*(3), 137-152. doi:10.1016/S0741-8329(01)00191-4

ImPACT. (2007). *ImPACT 2007 (6.0) clinical interpretation manual [electronic]*. Unpublished manuscript. Retrieved 1 August 2011, Retrieved from http://www.impacttest.com/pdf/ImPACT_Clinical_Interpretation_Manual.pdf

Iverson, G. L. (2006). Sensitivity of computerized neuropsychological screening in depressed university students. *The Clinical Neuropsychologist*, *20*(4), 695-701. doi:10.1080/138540491005857

Iverson, G. L., Lovell, M. R., & Collins, M. W. (2002). *Immediate Postconcussion Assessment and Cognitive Testing (ImPACT) normative data version 2.0*. Pittsburgh, USA: ImPACT Applications Incorporated.

Jackson, K. M., Sher, K. J., Gotham, H. J., & Wood, P. K. (2001). Transitioning into and out of large-effect drinking in young adulthood. *Journal of Abnormal Psychology*, *110*(3), 378-391. doi:10.1037/0021-843X.110.3.378

Karlamangla, A., Zhou, K., Reuben, D., Greendale, G., & Moore, A. (2006). Longitudinal trajectories of heavy drinking in adults in the United States of America. *Addiction, 101*(1), 91-99. doi:10.1111/j.1360-0443.2005.01299.x

Keith, M. S., Stanislav, S., & Wesnes, K. (1998). Validity of a cognitive computerized assessment system in brain injured patients. *Brain Injury, 12*(12), 1037-1043. doi:10.1080/026990598121945

Kersel, D. A., Marsh, N. V., Havill, J. H., & Sleigh, J. W. (2001). Neuropsychological functioning during the year following severe traumatic brain injury. *Brain Injury, 15*(4), 283-296. doi:10.1080/02699050010005887

Kheswa, J. G. (2006). *A descriptive analysis of alcohol and drug use amongst adolescents in Soweto.* (Unpublished Master's thesis). University of Johannesburg, Johannesburg.

Kokotailo, P. K., Egan, J., Gangnon, R., Brown, D., Mundt, M., & Fleming, M. (2004). Validity of the Alcohol Use Disorders Identification Test in college students. *Alcoholism: Clinical and Experimental Research, 28*(6), 914-920.

Kopelman, M. D., Thomson, A. D., Guerrini, I., & Marshall, E. J. (2009). The Korsakoff syndrome: Clinical aspects, psychology and treatment. *Alcohol and Alcoholism, 44*(2), 148-154. doi:10.1093/alcalc/agn118

Kopera, M., Wojnar, M., Brower, K., Glass, J., Nowosad, I., Gmaj, B., & Szelenberger, W. (2012). Cognitive functions in abstinent alcohol-dependent patients. *Alcohol, 46*(7), 665-671. doi:10.1016/j.alcohol.2012.04.005

Kuntsche, E., Knibbe, R., Gmel, G., & Engels, R. (2005). Why do young people drink? A review of drinking motives. *Clinical Psychology Review, 25*(7), 841-861. doi:10.1016/j.cpr.2005.06.002

Kypri, K., Langley, J. D., McGee, R., Saunders, J. B., & Williams, S. (2002). High prevalence, persistent hazardous drinking among New Zealand tertiary students. *Alcohol and Alcoholism, 37*(5), 457-464. doi:10.1093/alcalc/37.5.457

Lehohla, P. (2012). Census 2011: Census in brief. [electronic] (No. 03-01-41). Pretoria: Statistics South Africa. Retrieved from http://www.statssa.gov.za/census2011/Products/Census_2011_Census_in_brief.pdf

Leroi, I., Sheppard, J., & Lyketsos, C. G. (2002). Cognitive function after 11.5 years of alcohol use: Relation to alcohol use. *American Journal of Epidemiology, 156*(8), 747-752.

Levenson, R. W. (1987). Alcohol, affect, and physiology: Positive effects in the early stages of drinking. In E. Gottheil, K. A. Druley, S. Pashko & S. P. Weinstein (Eds.), *Stress and addiction* (pp. 173-196). New York: Brunner/Mazel.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.

Lobo, E., Dufouil, C., Marcos, G., Quetglas, B., Saz, P., Guallar, E., & Lobo, A. (2010). Is there an association between low-to-moderate alcohol consumption and risk of cognitive decline? *American Journal of Epidemiology, 172*(6), 708-716.

Loeber, S., Duka, T., Welzel, H., Nakovics, H., Heinz, A., Flor, H., & Mann, K. (2009). Impairment of cognitive abilities and decision making after chronic use of alcohol: The impact of multiple detoxifications. *Alcohol and Alcoholism, 44*(4), 372-381. doi:10.1093/alcalc/agp030

Louw, D. A., van Ede, D. M., & Louw, A. E. (1998). *Human development* (2nd ed.). Pretoria: Kagiso Tertiary.

Lovinger, D. M. (1999). The role of serotonin in alcohol's effects on the brain. *Current Separations, 18*(1), 24-28.

Luhtanen, R. K., & Crocker, J. (2005). Alcohol use in college students: Effects of level of self-esteem, narcissism, and contingencies of self-worth. *Psychology of Addictive Behaviors, 19*(1), 99-103. doi:10.1037/0893-164X.19.1.99

Lyvers, M. (2000). " Loss of control" in alcoholism and drug addiction: A neuroscientific interpretation. *Experimental and Clinical Psychopharmacology, 8*(2), 225-245. doi:10.1037/1064-1297.8.2.225

Makdissi, M., Collie, A., Maruff, P., Darby, D. G., Bush, A., McCrory, P., & Bennell, K. (2001). Computerised cognitive assessment of concussed Australian rules footballers. *British Journal of Sports Medicine, 35*(5), 354-360. doi:10.1136/bjsm.35.5.354

Mann, K., Ackermann, K., Croissant, B., Mundle, G., Nakovics, H., & Diehl, A. (2005). Neuroimaging of gender differences in alcohol dependence: Are women more vulnerable? *Alcoholism: Clinical and Experimental Research, 29*(5), 896-901.

Manning, V., Wanigaratne, S., Best, D., Hill, R. G., Reed, L. J., Ball, D., . . . Strang, J. (2008). Changes in neuropsychological functioning during alcohol detoxification. *European Addiction Research, 14*(4), 226-233. doi:10.1159/000156479

Marshall, E. J., Humphreys, K., & Ball, D., M. (2010). *The treatment of drinking problems: A guide for the helping profession* (5th ed.). Cambridge, UK: Cambridge University Press.

Martens, M. P., Dams-O'Connor, K., & Beck, N. C. (2006). A systematic review of college student-athlete drinking: Prevalence rates, sport-related factors, and interventions. *Journal of Substance Abuse Treatment, 31*(3), 305-316. doi:10.1016/j.jsat.2006.05.004

McCambridge, J., McAlaney, J., & Rowe, R. (2011). Adult consequences of late adolescent alcohol consumption: A systematic review of cohort studies. *PLoS Medicine, 8*(2), 1-13. doi:10.1371/journal.pmed.1000413

McCrady, B. S., & Epstein, E., E. (Eds.). (1999). *Addictions: A comprehensive guidebook.* New York: Oxford University Press.

McGee, R., & Kypri, K. (2004). Alcohol-related problems experienced by university students in New Zealand. *Australian and New Zealand Journal of Public Health, 28*(4), 321-323. doi:10.1111/j.1467-842X.2004.tb00437.x

McIntosh, C., & Chick, J. (2004). Alcohol and the nervous system. *Journal of Neurology, Neurosurgery & Psychiatry, 75*(Supplement 3), iii16-iii21. doi:10.1136/jnnp.2004.045708

McKinney, A., & Coyle, K. (2006). Alcohol hangover effects on measures of affect the morning after a normal night's drinking. *Alcohol and Alcoholism, 41*(1), 54-60. doi:10.1093/alcalc/agh226

McShane, K. E., & Cunningham, J. A. (2003). The role of relevancy in normative feedback for university students' drinking patterns. *Addictive Behaviors, 28*(8), 1523-1528. doi:10.1016/S0306-4603(03)00074-1

Meyer, A., & Sagvolden, T. (2006). Fine motor skills in South African children with symptoms of ADHD: Influence of subtype, gender, age, and hand dominance. *Behavioral and Brain Functions, 2*(1), 33-45. doi:10.1186/1744-9081-2-33

Mhlongo, M. M. (2008). African university students, the Five Factor Model, and parental bonding: Prediction of alcohol use. (Unpublished Master's thesis). University of Limpopo, Turfloop.

Mikhailovich, K., George, A., Rickwood, D., & Parker, R. (2011). A duty of care: Non-drinkers and alcohol related harm among an Australian university sample. *Journal of Higher Education Policy and Management, 33*(6), 595-604.

Miles, H., Winstock, A., & Strang, J. (2001). Identifying young people who drink too much: The clinical utility of the five-item Alcohol Use Disorders Identification Test (AUDIT). *Drug and Alcohol Review, 20*(1), 9-18. doi:10.1080/09595230123211

Montgomery, C., Fisk, J. E., Murphy, P. N., Ryland, I., & Hilton, J. (2012). The effects of heavy social drinking on executive function: A systematic review and meta-analytic study of existing literature and new empirical findings. *Human Psychopharmacology: Clinical and Experimental*, *27*(2), 187-199.

Moore, D. W., Ashman, T. A., Cantor, J. B., Krinick, R. J., & Spielman, L. A. (2010). Does gender influence cognitive outcome after traumatic brain injury? *Neuropsychological Rehabilitation, 20*(3), 340-354. doi:10.1080/09602010903250928

Moreira, M. T., Smith, L., & Foxcroft, D. (2009). Social norms interventions to reduce alcohol misuse in university or college students. *Cochrane Database of Systematic Reviews, 3* doi:10.1002/14651858.CD006748.pub2.

Moriyama, Y., Muramatsu, T., Kato, M., Mimura, M., & Kashima, H. (2006). Family history of alcoholism and cognitive recovery in subacute withdrawal. *Psychiatry & Clinical Neurosciences, 60*(1), 85-89. doi:10.1111/j.1440-1819.2006.01464.x

Mortensen, E. L., Jensen, H. H., Sanders, S. A., & Reinisch, J. M. (2001). Better psychological functioning and higher social status may largely explain the apparent health benefits of wine: A study of wine and beer drinking in young Danish adults. *Archives of Internal Medicine, 161*(15), 1844-1848.

Moser, R. S., & Schatz, P. (2002). Enduring effects of concussion in youth athletes. *Archives of Clinical Neuropsychology, 17*(1), 91-100. doi: 10.1016/S0887-6177(01)00108-1

Mulia, N., Ye, Y., Greenfield, T. K., & Zemore, S. E. (2009). Disparities in alcohol-related problems among white, black, and Hispanic Americans. *Alcoholism: Clinical and Experimental Research, 33*(4), 654-662.

Neighbors, C., Lee, C. M., Lewis, M. A., Fossos, N., & Larimer, M. E. (2007). Are social norms the best predictor of outcomes among heavy-drinking college students? *Journal of Studies on Alcohol and Drugs, 68*(4), 556-565.

Norris, R. C., Hottel, J. V., & Brooks, S. (1960). Comparability of Peabody Picture Vocabulary Test scores under group and individual administration. *Journal of Educational Psychology, 51*(2), 87-91. doi: 10.1037/h0046136

Notebaert, A. J., & Guskiewicz, K. M. (2005). Current trends in athletic training practice for concussion assessment and management. *Journal of Athletic Training, 40*(4), 320-325.

O'Hare, T., & Sherrer, M. V. (1999). Validating the Alcohol Use Disorder Identification Test with college first-offenders. *Journal of Substance Abuse Treatment, 17*(1–2), 113-119. doi:10.1016/S0740-5472(98)00063-4

Olivier, J., M. (2009). *Investigating literacy development among learners with a second language as medium of education – the effects of an emergent literacy stimulation program in grade R.* (Unpublished Doctoral dissertation). Stellenbosch University, Stellenbosch.

Olwagen, A. C. J. (2003). *Transgenerational patterns of substance abuse.* (Unpublished Master's thesis). University of Johannesburg, Johannesburg.

O'Malley, P. M. (2004). Maturing out of problematic alcohol use. *Alcohol Research and Health, 28*(4), 202-204.

Ozonoff, S., Cook, I., Coon, H., Dawson, G., Joseph, R. M., Klin, A., . . . Pennington, B. F. (2004). Performance on Cambridge Neuropsychological Test Automated Battery subtests sensitive to frontal lobe function in people with autistic disorder: Evidence from the collaborative programs of excellence in autism network. *Journal of Autism and Developmental Disorders, 34*(2), 139-150. doi:10.1023/B:JADD.0000022605.81989.cc

Pakendorf, C., & Alant, E. (1997). Culturally valid assessment tools: Northern Sotho translation of the Peabody Picture Vocabulary Rest-Revised. *The South African Journal of Communication Disorders. Die Suid-Afrikaanse Tydskrif Vir Kommunikasieafwykings, 44*, 3-11.

Pally, R. (2000). *The mind-brain relationship*. New York: Karnac Books.

Parada, M., Corral, M., Mota, N., Crego, A., Rodríguez Holguín, S., & Cadaveira, F. (2012). Executive functioning and alcohol binge drinking in university students. *Addictive Behaviors, 37*(2), 167-172. doi:10.1016/j.addbeh.2011.09.015

Park, C. L. (2004). Positive and negative consequences of alcohol consumption in college students. *Addictive Behaviors, 29*(2), 311-321. doi:10.1016/j.addbeh.2003.08.006

Parlesak, A., Billinger, M. H., Bode, C., & Bode, J. C. (2002). Gastric alcohol dehydrogenase activity in man: Influence of gender, age, alcohol consumption and smoking in a Caucasian population. *Alcohol and Alcoholism, 37*(4), 388-393.

Parry, C. D. H., Plüddemann, A., Steyn, K., Bradshaw, D., Norman, R., & Laubscher, R. (2005). Alcohol use in South Africa: Findings from the first demographic and health survey (1998) [Abstract]. *Journal of Studies on Alcohol, 66*(1) 91-97.

Patrick, M. E., & Schulenberg, J. E. (2011). How trajectories of reasons for alcohol use relate to trajectories of binge drinking: National panel data spanning late adolescence to early adulthood. *Developmental Psychology, 47*(2), 311-317. doi:10.1037/a0021939

Patrick, M. E., Collins, L. M., Smith, E., Caldwell, L., Flisher, A., & Wegner, L. (2009). A prospective longitudinal model of substance use onset among South African adolescents. *Substance use & Misuse, 44*(5), 647-662. doi:10.1080/10826080902810244

Pedersen, S. L., & McCarthy, D. M. (2013). Differences in acute response to alcohol between African Americans and European Americans. *Alcoholism: Clinical and Experimental Research, 37*(6), 1056-1063. doi:10.1111/acer.12068

Peele, S., & Brodsky, A. (2000). Exploring psychological benefits associated with moderate alcohol use: A necessary corrective to assessments of drinking outcomes? *Drug and Alcohol Dependence, 60*(3), 221-247. doi:10.1016/S0376-8716(00)00112-5

Peltzer, K. (2000). Health behaviour in black South African university students. *South African Journal of Psychology, 30*(4), 46-49.

Peltzer, K., & Ramlagan, S. (2009). Alcohol use trends in South Africa. *Journal of Social Science, 18*(1), 1-12.

Peltzer, K., Davids, A., & Njuho, P. (2011). Alcohol use and problem drinking in South Africa: Findings from a national population-based survey. *African Journal of Psychiatry, 14*(1), 30-37.

Peregrin, T. (2005). Wine—A drink to your health? *Journal of the American Dietetic Association, 105*(7), 1053-1054. doi: 10.1016/j.jada.2005.05.016

Perkins, H. W. (2003). The emergence and evolution of the social norms approach to substance abuse prevention. In H. W. Perkins (Ed.), *The social norms approach to preventing school and college age substance abuse: A handbook for educators, counselors, and clinicians* (pp. 3-18). San Francisco, CA: Jossey-Bass.

Piechatzek, M., Indlekofer, F., Daamen, M., Glasmacher, C., Lieb, R., Pfister, H., . . . Schütz, C. G. (2009). Is moderate substance use associated with altered executive functioning in a population-based sample of young adults? *Human Psychopharmacology: Clinical and Experimental, 24*(8), 650-665. doi:10.1002/hup.1069

Poskitt, E. (1984). Foetal alcohol syndrome. *Alcohol and Alcoholism, 19*(2), 159-165.

Presley, C. A., Meilman, P. W., & Leichliter, J. S. (2002). College factors that influence drinking. *Journal of Studies on Alcohol and Drugs, Supplement 14,* 82-90.

Ramchandani, V., Bosron, W., & Li, T. (2001). Research advances in ethanol metabolism. *Pathologie Biologie, 49*(9), 676-682.

Rapoport, S. I. (1976). *Blood-brain barrier in physiology and medicine*. New York: Raven Press.

Reinert, D. F., & Allen, J. P. (2007). The Alcohol Use Disorders Identification Test: An update of research findings. *Alcoholism: Clinical and Experimental Research, 31*(2), 185-199. doi: 10.1111/j.1530-0277.2006.00295.x

Rhodes University. (2013). *Digest of statistics 2012 [electronic]*. Unpublished manuscript. Retrieved 10 October 2013, Retrieved from http://www.ru.ac.za/static/intranet/dmu/digest12.pdf

Richardson, J. T. (2011). Eta squared and partial eta squared as measures of effect size in educational research. *Educational Research Review, 6*(2), 135-147. doi:10.1016/j.edurev.2010.12.001

Rickwood, D., George, A., Parker, R., & Mikhailovich, K. (2011). Harmful alcohol use on campus: Impact on young people at university. *Youth Studies Australia, 30*(1), 34-40.

Rodgers, B., Windsor, T. D., Anstey, K. J., Dear, K. B. G., Jorm, A.,F., & Christensen, H. (2005). Non-linear relationships between cognitive function and alcohol consumption in young, middle-aged and older adults: The PATH through life project. *Addiction, 100*(9), 1280-1290. doi:10.1111/j.1360-0443.2005.01158.x

Rosenbloom, M. J., Pfefferbaum, A., & Sullivan, E. V. (2004). Recovery of short-term memory and psychomotor speed but not postural stability with long-term sobriety in alcoholic women. *Neuropsychology, 18*(3), 589-597. doi:10.1037/0894-4105.18.3.589

Royal College of Psychiatrists. (1979). *Alcohol and alcoholism*. London: Tavistock.

Rushton, J. P., Skuy, M., & Fridjhon, P. (2003). Performance on Raven's Advanced Progressive Matrices by African, East Indian, and white engineering students in South Africa. *Intelligence, 31*(2), 123-137. doi:10.1016/S0160-2896(02)00140-X

Saults, J. S., Cowan, N., Sher, K. J., & Moreno, M. V. (2007). Differential effects of alcohol on working memory: Distinguishing multiple processes. *Experimental and Clinical Psychopharmacology, 15*(6), 576-587. doi:10.1037/1064-1297.15.6.576

Scarmeas, N., & Stern, Y. (2003). Cognitive reserve and lifestyle. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 625-633. doi:10.1076/jcen.25.5.625.14576

Schatz, P., & Putz, B. O. (2006). Cross-validation of measures used for computer-based assessment of concussion. *Applied Neuropsychology, 13*(3), 151-159. doi:10.1207/s15324826an1303_2

Schatz, P., & Sandel, N. (2013). Sensitivity and specificity of the online version of ImPACT in high school and collegiate athletes. *The American Journal of Sports Medicine, 41*(2), 321-326. doi:10.1177/0363546512466038

Schnirring, L. (2001). How effective is computerized concussion management? *The Physician and Sportsmedicine, 29*, 11-16.

Schuckit, M. A. (2009). Alcohol-use disorders. *The Lancet, 373*(9662), 492-501. doi:10.1016/S0140-6736(09)60009-X

Schulenberg, J. E., & Maggs, J. L. (2002). A developmental perspective on alcohol use and heavy drinking during adolescence and the transition to young adulthood. *Journal of Studies on Alcohol, 63*(SUPPL. 14), 54-70.

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. doi:10.1037/1064-1297.16.3.240

Sher, K. J., Martin, E. D., Wood, P. K., & Rutledge, P. C. (1997). Alcohol use disorders and neuropsychological functioning in first-year undergraduates. *Experimental and Clinical Psychopharmacology, 5*(3), 304-315. doi:10.1037/1064-1297.5.3.304

Shields, A. L., Guttmannova, K., & Caruso, J. C. (2004). An examination of the factor structure of the Alcohol Use Disorders Identification Test in two high-risk samples. *Substance use and Misuse, 39*, 1161-1182.

Shuttleworth-Edwards, A. B., Gaylard, E. K., & Radloff, S. E. (2013). WAIS-III test performance in the South African context: Refinement of an existing cross-cultural normative database. In K. Cockcroft, & S. Laher (Eds.), *Psychological assessment in South Africa: Research and applications* (pp. 17-32). Johannesburg: Wits Press.

Shuttleworth-Edwards, A. B., Kemp, R. D., Rust, A. L., Muirhead, J. G., Hartman, N. P., & Radloff, S. E. (2004). Cross-cultural effects on IQ test performance: A review and preliminary normative indications on WAIS-III test performance. *Journal of Clinical and Experimental Neuropsychology, 26*(7), 903-920. doi:10.1080/138033904905108

Shuttleworth-Edwards, A. B., Radloff, S. E., & Whitefield-Alexander, V. J. (2014). Vulnerability in speed of visuomotor ability. In A. Niranjan, & L. Lunsford (Eds.), *Concussion* (pp. 213-225). Pittsburgh: Karger. doi:10.1159/000358785

Shuttleworth-Edwards, A. B., Radloff, S. E., Whitefield-Alexander, V. J., Smith, I. P., & Horsman, M. (2013). Practice effects reveal visuomotor vulnerability in school and university rugby players. *Archives of Clinical Neuropsychology : The Official Journal of the National Academy of Neuropsychologists, 29*(1), 86-99. doi:10.1093/arclin/act061

Shuttleworth-Edwards, A. B., Smith, I., & Radloff, S. E. (2008). Neurocognitive vulnerability amongst university rugby players versus noncontact sport controls. *Journal of Clinical and Experimental Neuropsychology, 30*(8), 870-884. doi:10.1080/13803390701846914

Shuttleworth-Edwards, A. B., Whitefield-Alexander, V. J., Radloff, S. E., Taylor, A. M., & Lovell, M. R. (2009). Computerized neuropsychological profiles of South African versus US athletes: A basis for commentary on cross-cultural norming issues in the sports concussion arena. *The Physician and Sportsmedicine, 37*(4), 45-52. doi:10.3810/psm.2009.12.1741

Skogen, J. C., Mykletun, A., Ferri, C. P., Bebbington, P., Brugha, T., Coid, J., . . . Stewart, R. (2011). Mental and personality disorders and abstinence from alcohol: Results from a national household survey. *Psychological Medicine, 41*(4), 809-818. doi:10.1017/S0033291710001388

Skuy, M., Schutte, E., Fridjhon, P., & O'Carroll, S. (2001). Suitability of published neuropsychological test norms for urban African secondary school students in South Africa. *Personality and Individual Differences, 30*(8), 1413-1425. doi: 10.1016/S0191-8869(00)00123-9

Slicker, E. K. (1997). University students' reasons for not drinking: Relationship to alcohol consumption level. *Journal of Alcohol and Drug Education, 42*(2), 83-102.

Slutske, W. S., Hunt-Carter, E., Nabors-Oberg, R., Sher, K. J., Bucholz, K. K., Madden, P. A. F., . . . Heath, A. C. (2004). Do college students drink more than their non-college-attending peers? Evidence from a population-based longitudinal female twin study. *Journal of Abnormal Psychology, 113*(4), 530-540. doi:10.1037/0021-843X.113.4.530

Smit, P., Pretorius, P., & Joubert, G. (2010). University of the Free State medical students' view of at-risk drinking behaviour and psychoactive substance use. *South African Journal of Psychiatry, 15*(1), 13-18.

Smith, S., & Fein, G. (2010). Cognitive performance in treatment-naïve active alcoholics. *Alcoholism: Clinical and Experimental Research, 34*(12), 2097-2105.

Snyder, P. J., Jackson, C. E., Petersen, R. C., Khachaturian, A. S., Kaye, J., Albert, M. S., & Weintraub, S. (2011). Assessment of cognition in mild cognitive impairment: A comparative study. *Alzheimer's & Dementia, 7*(3), 338-355. doi:10.1016/j.jalz.2011.03.009

Sowell, E. R., Thompson, P. M., & Toga, A. W. (2004). Mapping changes in the human cortex throughout the span of life. *Neuroscientist, 10*(4), 372-392.

Spear, L. P. (2002). The adolescent brain and the college drinker: Biological basis of propensity to use and misuse alcohol. *Journal of Studies on Alcohol, 63*(Supplement 14), 71-81.

Squeglia, L. M., Schweinsburg, A. D., Pulido, C., & Tapert, S. F. (2011). Adolescent binge drinking linked to abnormal spatial working memory brain activation: Differential gender effects. *Alcoholism: Clinical and Experimental Research, 35*(10), 1831-1841. doi:10.1111/j.1530-0277.2011.01527.x

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society, 8*(03), 448-460. doi:10.1017.S1355617701020240

Stern, Y. (2009). Cognitive reserve. *Neuropsychologia, 47*(10), 2015-2028. doi:10.1016/j.neuropsychologia.2009.03.004

Stevens, C., Sidener, T. M., Reeve, S. A., & Sidener, D. W. (2011). Effects of behavior-specific and general praise, on acquisition of tacts in children with pervasive developmental disorders. *Research in Autism Spectrum Disorders, 5*(1), 666-669. doi:10.1016/j.rasd.2010.08.003

Steyl, T., & Phillips, J. (2012). Actual and perceived substance use of health science students at a university in the Western Cape, South Africa. *African Health Sciences, 11*(3), 329-333.

Suliman, S., Seedat, S., Williams, D. R., & Stein, D. J. (2010). Predictors of transitions across stages of alcohol use and alcohol-use disorders in South Africa. *Journal of Studies on Alcohol and Drugs, 71*(5), 695-703.

Sullivan, E. V., & Pfefferbaum, A. (2005). Neurocircuitry in alcoholism: A substrate of disruption and repair. *Psychopharmacology, 180*(4), 583-594. doi:10.1007/s00213-005-2267-6

Surujlal, J., Nolan, V., & Ubane, T. (2012). Drinking patterns and related consequences among university student-athletes. *African Journal for Physical, Health Education, Recreation and Dance, 18*(2), 281-292.

Swift, R., & Davidson, D. (1998). Alcohol hangover. *Alcohol Health & Research World, 22*, 54-60.

Tapert, S. F., & Brown, S. A. (1999). Neuropsychological correlates of adolescent substance abuse: Four-year outcomes. *Journal of the International Neuropsychological Society, 5*(6), 481-493. doi:10.1017/S1355617799566010

Tapert, S. F., Caldwell, L., & Burke, C. (2005). Alcohol and the adolescent brain: Human studies. *Alcohol Research and Health, 28*(4), 205-212.

Tapert, S. F., Granholm, E., Leedy, N. G., & Brown, S. A. (2002). Substance use and withdrawal: Neuropsychological functioning over 8 years in youth. *Journal of the International Neuropsychological Society, 8*(7), 873-883. doi:10.1017/S1355617702870011

Terre-Blanche, M., Durrheim, K., & Painter, D. (Eds.). (2006). *Research in practice* (2nd ed.). Cape Town: UCT Press.

Theakston, J. A., Stewart, S. H., Dawson, M. Y., Knowlden-Loewen, S. A. B., & Lehman, D. R. (2004). Big-five personality domains predict drinking motives. *Personality and Individual Differences, 37*(5), 971-984. doi:10.1016/j.paid.2003.11.007

Thoma, R. J., Monnig, M. A., Lysne, P. A., Ruhl, D. A., Pommy, J. A., Bogenschutz, M., . . . Yeo, R. A. (2011). Adolescent substance abuse: The effects of alcohol and marijuana on neuropsychological performance. *Alcoholism: Clinical and Experimental Research, 35*(1), 39-46. doi:10.1111/j.1530-0277.2010.01320.x

Thomas, D. G., Collins, M. W., Saladino, R. A., Frank, V., Raab, J., & Zuckerbraun, N. S. (2011). Identifying neurocognitive deficits in adolescents following concussion. *Academic Emergency Medicine, 18*(3), 246-254. doi:10.1111/j.1553-2712.2011.01015.x

Thomas, S., Benke, G., Dimitriadis, C., Inyang, I., Sim, M. R., Wolfe, R., . . . Abramson, M. J. (2010). Use of mobile phones and changes in cognitive function in adolescents. *Occupational and Environmental Medicine, 67*(12), 861-866. doi:10.1136/oem.2009.054080

Tillinghast Jr, B. S., & Renzulli, J. S. (1968). Reliability of a group form of the Peabody Picture Vocabulary Test. *The Journal of Educational Research, 61*(7), 311-314.

Tlustos, S. J., Chiu, C. Y. P., Walz, N. C., Holland, S. K., Bernard, L., & Wade, S. L. (2010). Neural correlates of interference control in adolescents with traumatic brain injury: Functional magnetic resonance imaging study of the counting Stroop task. *Journal of the International Neuropsychological Society, 17*(1), 181-189. doi:10.1017/S1355617710001414

Tredoux, C., & Durrheim, K. (Eds.). (2008). *Numbers, hypotheses & conclusions: A course in statistics for the social sciences*. Cape Town: UCT Press.

Tsushima, M., Tsushima, W., Tsushima, V., Lim, N., Madrigal, E., Jackson, C., & Mendler, M. H. (2013). Use of ImPACT to diagnose minimal hepatic encephalopathy: An accurate, practical, user-friendly internet-based neuropsychological test battery. *Digestive Diseases and Sciences, 58*(9), 2673-2681. doi:10.1007/s10620-013-2668-z

Twamley, E. W., Hami, S., & Stein, M. B. (2004). Neuropsychological function in college students with and without Posttraumatic Stress Disorder. *Psychiatry Research, 126*(3), 265-274. doi:10.1016/j.psychres.2004.01.008

van Heerden, M. S., Grimsrud, A. T., Seedat, S., Myer, L., Williams, D. R., & Stein, D. J. (2009). Patterns of substance use in South Africa: Results from the South African Stress and Health Study. *SAMJ: South African Medical Journal, 99*(5), 358-366.

van Kampen, D. A., Lovell, M. R., Pardini, J. E., Collins, M. W., & Fu, F. H. (2006). The "value added" of neurocognitive testing after sports-related concussion. *The American Journal of Sports Medicine, 34*(10), 1630-1635.

Wang, Y., Chan, R. C. K., & Deng, Y. (2006). Examination of postconcussion-like symptoms in healthy university students: Relationships to subjective and objective neuropsychological function performance. *Archives of Clinical Neuropsychology, 21*(4), 339-347. doi:10.1016/j.acn.2006.03.006

Wechsler, H., & Kuo, M. (2003). Watering down the drinks: The moderating effect of college demographics on alcohol use of high-risk groups. *American Journal of Public Health, 93*(11), 1929-1933.

Wechsler, H., & Nelson, T. F. (2008). What we have learned from the Harvard School of Public Health College Alcohol Study: Focusing attention on college student alcohol consumption and the environmental conditions that promote it. *Journal of Studies on Alcohol and Drugs, 69*(4), 481-490.

Weingardt, K. R., Baer, J. S., Kivlahan, D. R., Roberts, L. J., Miller, E. T., & Marlatt, G. A. (1998). Episodic heavy drinking among college students: Methodological issues and longitudinal perspectives. *Psychology of Addictive Behaviors, 12*(3), 155-167. doi:10.1037/0893-164X.12.3.155

Weiss, E. M., Kemmler, G., Deisenhammer, E. A., Fleischhacker, W. W., & Delazer, M. (2003). Sex differences in cognitive functions. *Personality and Individual Differences, 35*(4), 863-875. doi: 10.1016/S0191-8869(02)00288-X

Wetherill, R. R., Bava, S., Thompson, W. K., Boucquey, V., Pulido, C., Yang, T. T., & Tapert, S. F. (2012). Frontoparietal connectivity in substance-naïve youth with and without a family history of alcoholism. *Brain Research, 1432*, 66-73.

White, A. M. (2003). What happened? Alcohol, memory blackouts, and the brain. *Alcohol Research and Health, 27*(2), 186-196.

White, H. R., Fleming, C. B., Kim, M. J., Catalano, R. F., & McMorris, B. J. (2008). Identifying two potential mechanisms for changes in alcohol use among college-attending and non-college-attending emerging adults. *Developmental Psychology, 44*(6), 1625-1639. doi:10.1037/a0013855

Whitefield, V. J. (2007). Glory is temporary, brain injury may be forever: A neuropsychological study on the cumulative effects of sports-related concussive brain injury amongst grade 12 school boy athletes. (Unpublished Doctoral dissertation). Rhodes University, Grahamstown.

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

Wilken, J. A., Kane, R., Sullivan, C. L., Wallin, M., Usiskin, J. B., Quig, M. E., . . . Keller, M. (2003). The utility of computerized neuropsychological assessment of cognitive dysfunction in patients with relapsing-remitting multiple sclerosis. *Multiple Sclerosis (Houndmills, Basingstoke, England), 9*(2), 119-127. doi:10.1191/1352458503ms893oa

Williams, A., & Clark, D. (1998). Alcohol consumption in university students: The role of reasons for drinking, coping strategies, expectancies, and personality traits. *Addictive Behaviors, 23*(3), 371-378.

Windle, M. (2003). Alcohol use among adolescents and young adults. *Alcohol Research & Health, 27*(1), 79-85.

Windle, M., & Zucker, R. A. (2010). Reducing underage and young adult drinking: How to address critical drinking problems during this developmental period. *Alcohol Research and Health, 33*, 29-44.

Wolf, R. C., Sambataro, F., Lohr, C., Steinbrink, C., Martin, C., & Vasic, N. (2010). Functional brain network abnormalities during verbal working memory performance in adolescents and young adults with dyslexia. *Neuropsychologia, 48*(1), 309-318. doi: 10.1016/j.neuropsychologia.2009.09.020

Wood, P. K., Sher, K. J., & Bartholow, B. D. (2002). Alcohol use disorders and cognitive abilities in young adulthood: A prospective study. *Journal of Consulting and Clinical Psychology, 70*(4), 897-907. doi:10.1037/0022-006X.70.4.897

World Health Organisation. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.

World Health Organisation. (2011a). *Global status report on alcohol and health.* [electronic]. Retrieved from http://www.who.int/substance_abuse/publications/global_alcohol_report/msbgsruprofiles.pdf

World Health Organisation. (2011b). *South Africa.* [electronic]. Retrieved from http://www.who.int/substance_abuse/publications/global_alcohol_report/profiles/zaf.pdf

World Health Organisation. (2014). *Global status report on alcohol and health.* [electronic]. Retrieved from http://apps.who.int/iris/bitstream/10665/112736/1/9789240692763_eng.pdf?ua=1

Wright, C. B., Elkind, M. S. V., Luo, X., Paik, M. C., & Sacco, R. L. (2006). Reported alcohol consumption and cognitive decline: The Northern Manhattan study. *Neuroepidemiology, 27*(4), 201-207.

Younes, M., Hill, J., Quinless, J., Kilduff, M., Peng, B., Cook, S. D., & Cadavid, D. (2007). Internet-based cognitive testing in multiple sclerosis. *Multiple Sclerosis, 13*(8), 1011-1019. doi:10.1177/1352458507077626

Young, C., & de Klerk, V. (2008). Patterns of alcohol use on a South African university campus: The findings of two annual drinking surveys. *African Journal of Drug and Alcohol Studies, 7*(2), 101-112.

Young, C., & de Klerk, V. (2012). Correlates of heavy alcohol consumption at Rhodes University. *Journal of Child & Adolescent Mental Health, 24*(1), 37-44.

Young, C., & Mayson, T. (2010). The Alcohol Use Disorders Identification Scale (AUDIT) normative scores for a multiracial sample of Rhodes University residence students. *Journal of Child and Adolescent Mental Health, 22*(1), 15-23.

Yudofsky, S. C., & Hales, R. E. (Eds.). (2002). *The American psychiatric publishing textbook of neuropsychiatry and clinical neurosciences* (4th ed.). Washington D.C.: American Psychiatric Publishing.

Zeigler, D. W., Wang, C. C., Yoast, R. A., Dickinson, B. D., McCaffree, M. A., Robinowitz, C. B., & Sterling, M. L. (2005). The neurocognitive effects of alcohol on adolescents and college students. *Preventive Medicine, 40*(1), 23-32. doi:10.1016/j.ypmed.2004.04.044

Zubaran, C., Fernandes, J. G., & Rodnight, R. (1997). Wernicke – Korsakoff syndrome. *The Fellowship of Postgraduate Medicine, 73*, 27-31.

Appendix 1: Initial Testing Booklet

The Neuropsychological Effects of Students' Drinking Patterns

Research Participation Booklet

Researcher: Tamara Mayson (g06m2760)

Your Student number: ______________________

Section 2: Letter of consent

I, ________________________________ (your student number), agree to participate in the research project of Tamara Mayson. By giving my consent, I understand that:

1. The researcher is a student conducting research as part of the requirements for a Masters degree at Rhodes University. The research project has been approved by the relevant ethics committees, and is under the supervision of Dr Charles Young and Professor Ann Edwards in the Psychology Department at Rhodes University.
2. The researcher is interested in the neuropsychological effects of students' drinking patterns over the second year of studies at Rhodes University.
3. My participation in this research is completely voluntary; however I may receive an incentive for participating, including an entry into 1 or 2 random prize draws depending on the stages of the research that I am chosen for and complete.
4. I understand that my consent at this initial stage of the research does not mean that I am obliged to consent to the next phase of the research
5. My participation at this stage involves completing a general abilities test, alcohol use questionnaire and biographical questionnaire truthfully.
6. If I am randomly selected for the next stages of the research, my participation will involve completing a computerised neuropsychological test battery, alcohol use questionnaire and biographical questionnaire on three separate occasions during the year.
7. I may be asked to answer questions of a personal nature, but I can choose not to answer any questions about aspects of my life which I am not willing to disclose.
8. I am invited to voice to the researcher any concerns I have about my participation in the study, or consequences I may experience as a result of my participation, and to have these addressed to my satisfaction.
9. The de-identified data obtained from this research may be used by the Rhodes University researchers or researchers affiliated to ImPACT-USA Inc., for future presentation and publication purposes, and/or may be used together with additional data acquired for the purposes of future research projects.
10. I am free to withdraw from the study at any time.
11. No identifying information will be used in any report or publication.

If you would like to participate in the next phase of research, the neurocognitive testing, please indicate that here: Yes ☐ No ☐

Date: ________________________ Signature: ___________________________

Section 3: General Abilities Test

Training Examples:

A1	
1	2
3	4

A2	
1	2
3	4

B1	
1	2
3	4

B2	
1	2
3	4

Test:

145	
1	2
3	4

146	
1	2
3	4

147	
1	2
3	4

148	
1	2
3	4

149	
1	2
3	4

150	
1	2
3	4

151	
1	2
3	4

152	
1	2
3	4

153	
1	2
3	4

154	
1	2
3	4

155	
1	2
3	4

156	
1	2
3	4

157	
1	2
3	4

158	
1	2
3	4

159	
1	2
3	4

160	
1	2
3	4

161	
1	2
3	4

162	
1	2
3	4

163	
1	2
3	4

164	
1	2
3	4

165	
1	2
3	4

166	
1	2
3	4

167	
1	2
3	4

168	
1	2
3	4

169	
1	2
3	4

170	
1	2
3	4

171	
1	2
3	4

172	
1	2
3	4

173	
1	2
3	4

174	
1	2
3	4

175	
1	2
3	4

176	
1	2
3	4

177	
1	2
3	4

178	
1	2
3	4

179	
1	2
3	4

180	
1	2
3	4

181	
1	2
3	4

182	
1	2
3	4

183	
1	2
3	4

184	
1	2
3	4

185	
1	2
3	4

186	
1	2
3	4

187	
1	2
3	4

188	
1	2
3	4

189	
1	2
3	4

190	
1	2
3	4

191	
1	2
3	4

192	
1	2
3	4

193	
1	2
3	4

194	
1	2
3	4

195	
1	2
3	4

196	
1	2
3	4

197	
1	2
3	4

198	
1	2
3	4

199	
1	2
3	4

200	
1	2
3	4

201	
1	2
3	4

202	
1	2
3	4

203	
1	2
3	4

204	
1	2
3	4

205	
1	2
3	4

206	
1	2
3	4

207	
1	2
3	4

208	
1	2
3	4

209	
1	2
3	4

210	
1	2
3	4

211	
1	2
3	4

212	
1	2
3	4

213	
1	2
3	4

214	
1	2
3	4

215	
1	2
3	4

216	
1	2
3	4

217	
1	2
3	4

218	
1	2
3	4

219	
1	2
3	4

220	
1	2
3	4

221	
1	2
3	4

222	
1	2
3	4

223	
1	2
3	4

224	
1	2
3	4

225	
1	2
3	4

226	
1	2
3	4

227	
1	2
3	4

228	
1	2
3	4

Section 4: Biographical Information

1. General:

1.1. Email address: ____

1.2. Date of birth: ____

1.3. Gender: ____

1.4. Race: ____

1.5. First Language: ____

1.6. Nationality ____

1.7. Do you live in a residence, in digs or at home?____________________

1.8. Are you left or right handed? ______________________________

1.9. Are you colour blind? ________________________________

2. Alcohol related:

2.1. At what age did you first get drunk?

- ☐ I've never been drunk
- ☐ Before age 12 (Primary school)
- ☐ Ages 13-17 (High school)
- ☐ Age 18 and above

2.2. Where did this happen?

- ☐ I've never been drunk
- ☐ Home
- ☐ Work
- ☐ School
- ☐ Friend's house
- ☐ Other

2.3. At what age did you start drinking regularly?

- ☐ Never
- ☐ Before age 12 (Primary school)
- ☐ Ages 13-17 (High school)
- ☐ Age 18 and above

2.4. What do you drink most often now?

- ☐ I don’t drink alcohol
- ☐ Wine
- ☐ Spirits
- ☐ Beer
- ☐ A mixture
- ☐ Other alcoholic beverages

2.5. How often have you had a hangover in the last year?

- ☐ I don’t drink alcohol
- ☐ I haven’t drunk alcohol in the last year
- ☐ Never
- ☐ Less than monthly
- ☐ Monthly
- ☐ Weekly
- ☐ Daily or almost daily

2.6. How often have you passed out when drunk in the last year?

- ☐ I don’t drink alcohol
- ☐ I haven’t drunk alcohol in the last year
- ☐ Never
- ☐ Less than monthly
- ☐ Monthly
- ☐ Weekly
- ☐ Daily or almost daily

2.7. Are your parents:

- ☐ Abstainers from alcohol
- ☐ Light drinkers
- ☐ Moderate drinkers
- ☐ Heavy drinkers
- ☐ Alcoholics

3. Academics:

3.1. Did you complete all of your Matric Exams in English? ______________

3.2. At what age did you complete you Matric Exams? ____________________

3.3. What sort of high school education have you had?

- ☐ South African Model C/ Government School, English Medium
- ☐ South African Model C/ Government School, Afrikaans Medium
- ☐ South African Private school
- ☐ South African Township school
- ☐ Other

3.4. What was the name and address of your school? ___________________

3.5. Please tick the following if they apply:

- ☐ Received Speech Therapy
- ☐ Attended special classes or remedial classes (not including extra lessons)
- ☐ Received Occupational therapy
- ☐ Repeated any grade
- ☐ Have been diagnosed with a learning disability

4. <u>Health:</u>

4.1. Have you been in good health lately? ____________________________

4.2. How many hours sleep on average do you have per night?____________

4.3. Do you smoke, or have you given up smoking recently? _____________

4.4. Do you take any addictive substances, not including alcohol and caffeine?

- ☐ Yes
- ☐ No

4.5. If so, what type of addictive substances?__________________________

4.6. Do you take any chronic medication? ____________________________

4.7. If you are female, have you ever been pregnant? ___________________

4.8. Have you ever had a head injury that resulted in hospitalisation? And if so, how long ago? ____

4.9. Have you ever been diagnosed with and/ or treated for any of the following?

- ☐ ADD/ ADHD
- ☐ Hyperactivity
- ☐ Psychiatric disorder
- ☐ Neurological disorder
- ☐ None of the above

Section 5: Alcohol Survey

This is a survey to measure alcohol consumption. Please read each question carefully and answer as truthfully as you can. Answer each question yourself by marking the relevant answer. These questions refer to the previous 12 months.

1. How often do you have a drink containing alcohol?	Never	Monthly or less	2-4 times a month	2-3 times a week	4 or more times a week
2. How many drinks containing alcohol do you have on a typical day when you are drinking?	0 to 2	3 or 4	5 or 6	7 to 9	10 or more
3. How often do you have six or more drinks on one occasion?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
4. How often have you found that you were not able to stop drinking once you had started?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
5. How often have you failed to do what was normally expected of you because of drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
6. How often have you needed a first drink in the morning to get yourself going after a heavy drinking session?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
7. How often have you had a feeling of guilt or remorse after drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
8. How often have you been unable to remember what happened the night before because of your drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
9. Have you or someone else been injured because of your drinking?	No		Yes, but not in the last year		Yes, during the last year
10. Has a relative, friend, doctor or other health care worker been concerned about your drinking or suggested you cut down?	No		Yes, but not in the last year		Yes, during the last year

Appendix 2: Initial Phase Letter of consent

I, ________________________________ (your student number), agree to participate in the research project of Tamara Mayson. By giving my consent, I understand that:

1. The researcher is a student conducting research as part of the requirements for a Masters degree at Rhodes University. The research project has been approved by the relevant ethics committees, and is under the supervision of Dr Charles Young and Professor Ann Edwards in the Psychology Department at Rhodes University.
2. The researcher is interested in the neuropsychological effects of students' drinking patterns over a single academic year of studies at Rhodes University.
3. My participation in this research is completely voluntary; however I may receive an incentive for participating, including an entry into 1 or 2 random prize draws depending on the stages of the research that I am chosen for and complete.
4. I understand that my consent at this initial stage of the research does not mean that I am obliged to consent to the next phase of the research
5. My participation at this stage involves completing a general abilities test, alcohol use questionnaire and biographical questionnaire truthfully.
6. If I am randomly selected for the next stages of the research, my participation will involve completing a computerised neuropsychological test battery, alcohol use questionnaire and biographical questionnaire on three separate occasions during the year.
7. I may be asked to answer questions of a personal nature, but I can choose not to answer any questions about aspects of my life which I am not willing to disclose.
8. I am invited to voice to the researcher any concerns I have about my participation in the study, or consequences I may experience as a result of my participation, and to have these addressed to my satisfaction.
9. The de-identified data obtained from this research may be used by the Rhodes University researchers or researchers affiliated to ImPACT-USA Inc., for future presentation and publication purposes, and/or may be used together with additional data acquired for the purposes of future research projects.
10. I am free to withdraw from the study at any time.
11. No identifying information will be used in any report or publication.

If you would like to participate in the next phase of research, the neurocognitive testing, please indicate that here: Yes ☐ No ☐

Date: ________________________ Signature: ___________________________

Appendix 3: Phase 2 Letter of consent

I, ________________________________ (your student number), agree to participate in the research project of Tamara Mayson. By giving my consent, I understand that:

1. The researcher is a student conducting research as part of the requirements for a Masters degree at Rhodes University. The research project has been approved by the relevant ethics committees, and is under the supervision of Dr Charles Young and Professor Ann Edwards in the Psychology Department at Rhodes University.
2. The researcher is interested in the neuropsychological effects of students' drinking patterns over the second year of studies at Rhodes University.
3. My participation in this research is completely voluntary; however I may receive an incentive for participating, including an entry into 2 random prize draws.
4. I understand that my consent at this stage of the research does mean that I am compelled, but not forced to continue to the end of the research, completing phase 2, intervals 1 to 3.
5. My participation at this stage will involve completing a computerised neuropsychological test battery, alcohol use questionnaire and biographical questionnaire on three separate occasions during the year, May, August and October.
6. I may be asked to answer questions of a personal nature, but I can choose not to answer any questions about aspects of my life which I am not willing to disclose.
7. I am invited to voice to the researcher any concerns I have about my participation in the study, or consequences I may experience as a result of my participation, and to have these addressed to my satisfaction.
8. The de-identified data obtained from this research may be used by the Rhodes University researchers or researchers affiliated to ImPACT-USA Inc., for future presentation and publication purposes, and/or may be used together with additional data acquired for the purposes of future research projects.
9. I am free to withdraw from the study at any time.
10. No identifying information will be used in any report or publication.

Date: ________________________ Signature: ___________________________

Appendix 4: Information Sheet

I am a Psychology Masters by Thesis student at Rhodes University doing my research on the neuropsychological effects of students' drinking patterns over the second year of studies at Rhodes University. My objective is to draw upon willing volunteers in Psychology II, to whom, I will administer (i) a commonly employed paper and pencil cognitive test, (ii) a biographical questionnaire, and (iii) an Alcohol survey. I will reward those who participate in this first stage with a single random prize draw of R1000. This data will be used only for the next phase of the research which will be to randomly select approximately 50 to 75 students. Those selected will then make up the target groups for the research investigation, which will involve completing a user friendly computerized neuropsychological screening programme, on three repeat test occasions over the academic year: (i) at the beginning of the year, (ii) towards the middle of the year, and (iii) finally towards the end of the year. The programme that will be used tests a spectrum of cognitive functions and also presents a symptom check list to be completed. The surveys and tests from the students will be kept confidential and will be kept in a safe place, until no longer needed and then destroyed. As an incentive to those students who are chosen for the neuropsychological testing stages, a random prize draw will be done at the end of the year. This draw will include two cash prizes of R1000 each. Only those students, who have participated fully, that is, have come to all the testing stages and completed all the necessary sections, will be included in the draw.

It is completely voluntary to participate in this research project and you can withdraw from the research at any time, even after consenting. You may ask, after withdrawing, that any data that we have received from you, be removed and erased from the research. Should it be required, free counseling services will be offered if needed for any reason.

If you have any questions, please don't hesitate to ask me at any time.

Kind Regards

Researcher: Tamara Mayson, g06m2760@ru.ac.za

Supervisors: Dr Charles Young, c.young@ru.ac.za

Prof Ann Edwards, a.edwards@ru.ac.za

Appendix 5: Phase 2 Interval 1 Information Booklet

The Neuropsychological Effects of Students' Drinking Patterns

Research Participation Booklet
Phase 2: Interval 1

Researcher: Tamara Mayson (g06m2760)

Your Participation Name: ______________________

Section 2: Letter of consent

I, _________________________________ (your student number), agree to participate in the research project of Tamara Mayson. By giving my consent, I understand that:

1. The researcher is a student conducting research as part of the requirements for a Masters degree at Rhodes University. The research project has been approved by the relevant ethics committees, and is under the supervision of Dr Charles Young and Professor Ann Edwards in the Psychology Department at Rhodes University.
2. The researcher is interested in the neuropsychological effects of students' drinking patterns over the second year of studies at Rhodes University.
3. My participation in this research is completely voluntary; however I may receive an incentive for participating, including an entry into 2 random prize draws.
4. I understand that my consent at this stage of the research does mean that I am compelled, but not forced to continue to the end of the research, completing phase 2, intervals 1 to 3.
5. My participation at this stage will involve completing a computerised neuropsychological test battery, alcohol use questionnaire and biographical questionnaire on three separate occasions during the year, May, August and October.
6. I may be asked to answer questions of a personal nature, but I can choose not to answer any questions about aspects of my life which I am not willing to disclose.
7. I am invited to voice to the researcher any concerns I have about my participation in the study, or consequences I may experience as a result of my participation, and to have these addressed to my satisfaction.
8. The de-identified data obtained from this research may be used by the Rhodes University researchers or researchers affiliated to ImPACT-USA Inc., for future presentation and publication purposes, and/or may be used together with additional data acquired for the purposes of future research projects.
9. I am free to withdraw from the study at any time.
10. No identifying information will be used in any report or publication.

Date: ________________________ Signature: ___________________________

Section 3: Test Instructions

1. If you have a question please put your hand up. Please do not disturb the other people taking the test.
2. Please go to www.impacttestonline.com/southafrica
3. Click the "Launch Baseline Test" button
4. Click English
5. Read the instructions and select Metric (centimeters, kilograms, etc.) and then click Next
6. There are 3 sections of this program
   - 1. Sport and Health History
   - 2. Current Symptoms and Conditions
   - 3. Neurocognitive Test
7. Section 1: Sport and Health History:
   a. The Clinic Name is automatically South Africa, in the School/Organization box, select "Alcohol Study" and put in your Date of Birth and click next.
   b. Put your participation name in the name fields. If you're not sure what your participation name is, ask.
   c. Put in your height in metres and weight in kilograms. If you're not sure, put in an approximate amount.
   d. Press next and fill in all your information truthfully. When you get to the question about Years of Education, this is your completed level of education, that is, your education up to and including last year.
   e. When you come to the questions about Sport, if you play a sport that might result, or has resulted already in you getting a concussion, please fill it in, otherwise leave blank.
   f. Please fill in the details about your passed concussions, if you have had any. If you have not had any concussions then leave this blank and move on.
   g. The question on current medications does not include vitamins.
   h. Please fill in all the questions that you are able to, and as truthfully as you are able to. Any information that you put in incorrectly or miss out, might confuse the data and damage my research.
8. Section 2: Current Symptoms and Conditions
   a. This section refers to your symptoms in the last 24 hours

9. Section 3: Neurocognitive Test
    a. There are 6 tests with instructions and samples before each test. The instructions are not timed so you can take as long as you want to read them and understand them carefully. The tests themselves are timed, so do them as quickly as possible.
    b. When you get to the Symbol Search test, you are required to count BACKWARDS from 25, it won't be accepted if you count forwards. Do not plan your moves before you start because the timer will start when you press the next button.
10. When you are finished with the test, press the button that says Print.

<u>Section 4: Additional Questions</u>

1. When was your last meal
    - ☐ 1-2 hours ago
    - ☐ 3-4 hours ago
    - ☐ 5-8 hours ago
    - ☐ More than 8 hours ago
2. When did you last have alcohol?
    - ☐ 1-2 hours ago
    - ☐ 3-4 hours ago
    - ☐ 5-8 hours ago
    - ☐ More than 8 hours ago
    - ☐ I don't drink alcohol
3. When did you last have caffeine?
    - ☐ 1-2 hours ago
    - ☐ 3-4 hours ago
    - ☐ 5-8 hours ago
    - ☐ More than 8 hours ago
    - ☐ I don't drink caffeine
4. When did you last smoke?
    - ☐ 1-2 hours ago
    - ☐ 3-4 hours ago
    - ☐ 5-8 hours ago
    - ☐ More than 8 hours ago

Appendix 6: Phase 2 Interval 2 Information Booklet

The Neuropsychological Effects of Students' Drinking Patterns

Research Participation Booklet
Phase 2: Interval 2

Researcher: Tamara Mayson (g06m2760)

Your Student Number: ____________________

Your Participation Name: ______________________

Section 1: Test Instructions

1. If you have any questions please put your hand up. Please do not disturb the other people taking the test.
2. Please open **Mozilla Firefox**, not Internet Explorer and go to: www.impacttestonline.com/customercenter/
3. **WAIT UNTIL I COME AROUND TO LOG YOU IN.**
4. Select English and click OK on the pop-up box labeled Supervised Testing Environment.
5. Read the instructions and select **Metric (centimeters, kilograms, etc.)** and then click Next
6. Make sure that the School/Organisation box says **Alcohol Study** and that your date of birth is correct.
7. **DO NOT CHANGE YOUR NAME!!!!!!!**
8. You have seen the rest of the screens before. Make sure that your data is correct and up-to-date. There are a couple of things that went wrong before, so please make sure that:
   a. Your height is in **METRES**, so for example, 1.65, **not** 5"2 or 165 or 1.
   b. If your Native Language or Second Language is not in the list, put in either the closest language to it, or put in English. Then write the language on this instruction sheet here: Native/First:____________________ Second:_________________________
   c. Your years of education should be above 12. You have completed at least 12 years of school before you came to University. So this number should be 12 plus the number of years you have been at University, excluding this year.
9. The question on current medications does not include vitamins.
10. Please fill in all the questions that you are able to, and as truthfully as you are able to. Any information that you put in incorrectly or miss out, might confuse the data and damage my research.
11. Section 2: Current Symptoms and Conditions
    a. This section refers to your symptoms in the last 24 hours.
    b. 1 is the least severe, 6 is the most severe.
12. Section 3: Neurocognitive Test
    a. There are 6 tests with instructions and samples before each test. **Read the instructions carefully.** The instructions are not timed so you can take as long as you want to read them and understand them carefully. **The tests themselves are timed**, so do them as quickly as possible.

Please turn over the page

b. When you get to the Symbol Search test, you are required to count **BACKWARDS** from 25, it won't be accepted if you count forwards. Do not plan your moves before you start because the timer will start when you press the next button.

13. When you are finished with the test, press the button that says Print.

Section 2: Additional Questions

1) When was your last meal?
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
2) When did you last have alcohol?
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
   - ☐ I don't drink alcohol
3) When did you last have caffeine? (This includes coffee, tea, hot chocolate, cold and flu medications like Med Lemon)
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
   - ☐ I don't drink caffeine
4) When did you last smoke?
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
   - ☐ I don't smoke

Health:

1) Are you in good health today? _________________________

2) Has anything below changed since the last testing interval:

- ☐ Health
- ☐ Sleeping habits
- ☐ Smoking habits
- ☐ (Illegal) Drug habits
- ☐ Chronic medication
- ☐ Pregnancy
- ☐ Head injury

If yes, please explain below:

____
____
____
____
____
____
____

<u>Alcohol Questions:</u>

1) What is the most that you have drunk in one night? (In number of drinks)

____

2) When was this? (Year)

____

3) Have you drunk this much (on average) more than once?

____

4) If so, how often?

____

5) How many drinks, on average, make you tipsy?

____

6) How many drinks, on average, make you drunk?

____

7) Have your alcohol drinking patterns changed since the last testing interval?

____

8) If so, how?

____
____
____

Section 3: Alcohol Survey

This is a survey to measure alcohol consumption. Please read each question carefully and answer as truthfully as you can. Answer each question yourself by marking the relevant answer. These questions refer to the previous 12 months.

1. How often do you have a drink containing alcohol?	Never	Monthly or less	2-4 times a month	2-3 times a week	4 or more times a week
2. How many drinks containing alcohol do you have on a typical day when you are drinking?	0 to 2	3 or 4	5 or 6	7 to 9	10 or more
3. How often do you have six or more drinks on one occasion?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
4. How often have you found that you were not able to stop drinking once you had started?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
5. How often have you failed to do what was normally expected of you because of drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
6. How often have you needed a first drink in the morning to get yourself going after a heavy drinking session?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
7. How often have you had a feeling of guilt or remorse after drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
8. How often have you been unable to remember what happened the night before because of your drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
9. Have you or someone else been injured because of your drinking?	No		Yes, but not in the last year		Yes, during the last year
10. Has a relative, friend, doctor or other health care worker been concerned about your drinking or suggested you cut down?	No		Yes, but not in the last year		Yes, during the last year

Appendix 7: Phase 2 Interval 3 Information Booklet

The Neuropsychological Effects of Students' Drinking Patterns

Research Participation Booklet
Phase 2: Interval 3

Researcher: Tamara Mayson (g06m2760)

Your Student Number: ________________________

Your Participation Name: ________________________

Section 1: Test Instructions

1. If you have any questions please put your hand up. Please do not disturb the other people taking the test.
2. Please open **Mozilla Firefox**, not Internet Explorer and go to: www.impacttestonline.com/customercenter/
3. **WAIT UNTIL I COME AROUND TO LOG YOU IN.**
4. Select English and click OK on the pop-up box labeled Supervised Testing Environment.
5. Read the instructions and select **Metric (centimeters, kilograms, etc.)** and then click Next
6. Make sure that the School/Organisation box says **Alcohol Study** and that your date of birth is correct.
7. **DO NOT CHANGE YOUR NAME!!!!!!!**
8. You have seen the rest of the screens before. Make sure that your data is correct and up-to-date. There are a couple of things that went wrong before, so please make sure that:
   a. Your height is in **METRES**, so for example, 1.65, **not** 5"2 or 165 or 1.
   b. If your Native Language or Second Language is not in the list, put in either the closest language to it, or put in English. Then write the language on this instruction sheet here: Native/First:____________________ Second:__________________________
   c. Your years of education should be above 12. You have completed at least 12 years of school before you came to University. So this number should be 12 plus the number of years you have been at University, excluding this year.
9. The question on current medications does not include vitamins.
10. Please fill in all the questions that you are able to, and as truthfully as you are able to. Any information that you put in incorrectly or miss out, might confuse the data and damage my research.
11. Section 2: Current Symptoms and Conditions
    c. This section refers to your symptoms in the last 24 hours.
    d. 1 is the least severe, 6 is the most severe.
12. Section 3: Neurocognitive Test
    a. There are 6 tests with instructions and samples before each test. **Read the instructions carefully.** The instructions are not timed so you can take as long as you want to read them and understand them carefully. **The tests themselves are timed**, so do them as quickly as possible.

Please turn over the page

b. When you get to the Symbol Search test, you are required to count **BACKWARDS** from 25, it won't be accepted if you count forwards. Do not plan your moves before you start because the timer will start when you press the next button.

13. When you are finished with the test, press the button that says Print.

Section 2: Additional Questions

1) When was your last meal?
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
2) When did you last have alcohol?
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
   - ☐ I don't drink alcohol
3) When did you last have caffeine? (This includes coffee, tea, hot chocolate, cold and flu medications like Med Lemon)
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
   - ☐ I don't drink caffeine
4) When did you last smoke?
   - ☐ 0-2 hours ago
   - ☐ 3-4 hours ago
   - ☐ 5-8 hours ago
   - ☐ More than 8 hours ago
   - ☐ I don't smoke
5) If I have any follow up questions, can I contact you again?
   - ☐ Yes
   - ☐ No

6) Would you like a summary of my overall results?

- ☐ Yes
- ☐ No

7) Would you like a summary of your alcohol use and cognitive functioning over the year?

- ☐ Yes
- ☐ No

Health:

8) Are you in good health today? ______________________

9) Has anything below changed since the last testing interval:

- ☐ Health
- ☐ Sleeping habits
- ☐ Smoking habits
- ☐ (Illegal) Drug habits
- ☐ Chronic medication
- ☐ Pregnancy
- ☐ Head injury

If yes, please explain below:

____

____

____

Alcohol-related Questions:

10) If you don't drink alcohol, why don't you drink alcohol?

____

____

11) If you do drink alcohol, why do you drink alcohol?

____

____

12) Have your alcohol drinking patterns changed since the last testing interval?

- ☐ Yes
- ☐ No

13) If yes, how?

____

____

Section 3: Alcohol Survey

This is a survey to measure alcohol consumption. Please read each question carefully and answer as truthfully as you can. Answer each question yourself by marking the relevant answer. These questions refer to the previous 12 months.

1. How often do you have a drink containing alcohol?	Never	Monthly or less	2-4 times a month	2-3 times a week	4 or more times a week
2. How many drinks containing alcohol do you have on a typical day when you are drinking?	0 to 2	3 or 4	5 or 6	7 to 9	10 or more
3. How often do you have six or more drinks on one occasion?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
4. How often have you found that you were not able to stop drinking once you had started?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
5. How often have you failed to do what was normally expected of you because of drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
6. How often have you needed a first drink in the morning to get yourself going after a heavy drinking session?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
7. How often have you had a feeling of guilt or remorse after drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
8. How often have you been unable to remember what happened the night before because of your drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
9. Have you or someone else been injured because of your drinking?	No		Yes, but not in the last year		Yes, during the last year
10. Has a relative, friend, doctor or other health care worker been concerned about your drinking or suggested you cut down?	No		Yes, but not in the last year		Yes, during the last year