RESEARCH

Cumulative Mild Head Injury (CMHI) in contact sports: An Evaluation of the Pre and Post Season Cognitive Profiles of Rugby Players Compared with Non-Contact Sport Controls at the University of Limpopo (Turfloop Campus)

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DECLARATION

I, Mokgadi J. Rapetsoa, declare that this mini-dissertation hereby submitted to the University of Limpopo for the degree Masters of Arts in Clinical Psychology, has not been previously submitted by me for a degree at this or any other University ; that it is my own in design and in execution, and that all material contained herein has been duly acknowledged.

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RAPETSOA MJ (200815335) **DATE**

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ABSTRACT

The effect of Cumulative Mild Head Injury (CMHI) in contact sports, such as rugby, is seen increasingly at school level where more and more injuries are reported. Research on CMHI in contact sport is needed specifically amongst previously disadvantaged groups where little or no research has taken place. The research is thus intended to seek a better understanding of CMHI in the contact sport of rugby specifically amongst amateur players. A quantitative research approach was utilised with a quasi-experimental research design. A sample of 18 student rugby players and 18 volleyball (non-contact sport) controls was used. In terms of mean performances the tests did not reveal a consistent pattern of deficits which is typically associated with the effects of Cumulative Mild Head Injuries. There were significant results however, in terms of variability that suggests potential deficits in attention among the rugby group. The results are therefore indicative of a poorer overall cognitive profile for the rugby playing group. It is concluded that the increased variability may be displayed in individuals who suffer CMHI at an earlier age.

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CHAPTER ONE: INTRODUCTION

1.1 Introduction

The study is relevant to the socio-cultural political context in South Africa which aims to promote sport as a unifying agent. The South African Rugby Football Union (SARFU) encourages boys from the age of 6 years old to participate in the sport as it is a team sport which promotes teamwork. The knowledge that head injury in contact sports may cause neuropsychological deficits however is a cause for concern as these cognitive deficits may have a cumulative effect (Shuttleworth-Jordan, Balarin&Pucheret, 1993; Shuttleworth-Edwards & Whitfield, 2007).

Mild Traumatic Brain Injury (MTBI), also known as Mild Head Injury (MHI) can be defined as an immediate and transient impairment of neural functioning such as alteration of consciousness, disturbance of vision, equilibrium or other similar symptoms (Wilberger, 1993). MTBI is further sub-classified as mild, moderate and severe, depending on the length of the loss of consciousness and duration of Post-Traumatic Amnesia (PTA) which is dependent upon the period of time for which there is no recollection of events. Mild Traumatic Brain Injury (MTBI) can occur in contact sports without the player experiencing loss of consciousness, thus it is sometimes difficult to recognise sports related MTBI. The immediate management of MTBI requires serial observation (Satz, Alfano, Light, Morgenstern, Zaucha, Asarnow & Newton, 1997). According to Wilberger (1993) immediately after MTBI in the sporting arena the majority of athletes report a variety of disturbances including headaches, dizziness, memory problems, irritability, difficulty sleeping and lack of concentration. Such symptoms are typically short-lived and resolve spontaneously. However, in a small number of athletes, problems may become persistent, resulting in a post-concussion.

There is a suggestion that indicates that cumulative and more permanent neuropsychological impairments arise from repeated Mild Head Injury (MHI), as a result of neural abrasion, which may be harmful to an athlete's well-being (Gronwall, 1989; Rutherford, Stephens, Potter & Fernie, 2005). A study on neuro-trauma indicated that repeated head injuries result in cumulative damage to the brain and cause cumulative damage to hippocampal cells (Packard, 2008). On-going research suggests that repeated MHI or Cumulative Mild Head Injury (CMHI) which occurs over an extended period of time (weeks to months to years) is likely to

result in cumulative neurological and cognitive deficits (Silver, McAllister &Arciniegas, 2009).

1.2 Research Problem

The impact of CMHI in contact sports, particularly rugby, is increasingly prevalent in the school sports setting (Nel, Grace & Nel, 2011). CMHI is prevalent amongst all rugby players, irrespective of their race; as such, the current research was undertaken at a predominantly black University for the purpose of research convenience and as such, the demographic of the research implies that there will a large proportion of black students as participants. The research is thus intended to seek a better understanding of CMHI in the contact sport of rugby specifically amongst amateur players.

1.3Purpose of the study

1.3.1 Aim of the study

To evaluate if CMHI in rugby (contact sport) affects the cognitive profiles of rugby players (contact sports), compared with a non-contact sport (volleyball) controls pre and post season at the University of Limpopo (Turfloop campus).

1.3.2 Objectives of the study

- To examine the extent of cognitive deficit amongst the rugby playing experimental group and the volleyball control group pre and post season.
- To compare the cognitive profiles of the experimental (rugby playing) group with the control (volleyball group) pre and post season.

1.4 Significance of the research

The importance of this study is to create more awareness and provide knowledge on how sport practices influence a player's cognitive, emotive and behavioural functioning. This study will look at the impact CMHI (caused by contact sport) has on the cognitive functioning of rugby players at the University of Limpopo (Turfloop Campus).

The findings of the current study will help to broaden research, particularly at previously disadvantaged universities, on CMHI in rugby. Furthermore the recommendations that have

arisen out of the investigation will help to guide future research at the university in terms of sport and head injury in contact sports (See chapter 8).

1.5 Summary

The definition of CMHI includes aspects such as loss of consciousness, impairment of neural functioning, disturbance of vision and equilibrium. Repetitive MTBI can cause short lived symptoms such as headaches, dizziness, memory problems, irritability, difficulty sleeping and lack of concentration. Research has revealed that CMHI may cause permanent neuropsychological impairments.

CHAPTER TWO: THEORETICAL FRAMEWORK FOR THE STUDY

2.1 Introduction

The following chapter gives a theoretical framework for the study. It utilises two well-known models that, although theoretical in nature, can be applied to studies on brain injury to enhance a practical understanding of the effect to cognitive processes within the brain.

2.2 Operational Definitions

The Glasgow Coma Scale (GCS) - the GCS is a system for determining consciousness levels using a numeric value for each part of the rating scale. The degree of consciousness is assessed numerically by the best response. The numeric values for each parameter are added up and can be used as an overall objective measurement with 15 suggestive of no impairment, 3 compatible with brain death, and 7 as a parameter for a state of coma (Lezak, Howieson and Loring, 2004)

Mild Head Injury (MHI) also known as Mild Traumatic Brain Injury (MTBI) – the type of injury focused on in this research is mild closed head injury, which will subsequently be termed Mild Head Injury or MHI. The term Mild Traumatic Brain Injury (MTBI) may be used interchangeably with MHI in this study depending on the researcher being quoted. Reasonable criteria for MHI is loss of consciousness (LOC) for 30 minutes or less, being dazed without LOC, an initial Glasgow Coma Scale Score of 13 – 15 without evidence of depressed skull fractures, intracranial haematomas or other neurological pathology (Evans, 1992).

Cumulative Mild Head Injury (CMHI) – this type of injury is related to continuing symptomology after an incidence of MHI has supposedly resolved. It is associated with a longer recovery period and evidence suggest that repeated MHI which isnow termed Cumulative Mild Head Injury (CMHI) is associated with the increased risk of permanent cognitive deficits (Larrabee, 1999).

2.3Satz's (1993) Brain Reserve Capacity Threshold Theory (BRC)

Satz's (1993) Brain Reserve Capacity Threshold Theory (BRC) and Jordan's (1997) model of inter-individual variability attempts to explain how the use of theoretical concepts allow for the identification of cognitive patterns that might not otherwise be apparent. BRC refers to a threshold factor within each human being which represents a crucial point at which normal functioning is sustained, preceding the expression of symptoms caused by injury or disease to the brain. Furthermore, inside this model is the notion that there are individual differences that exist with regard to BRC that account for variable instances of vulnerability and symptom onset. The model holds that BRC thresholds differ between individuals. Educational levels represent an indirect measure of an individual's BRC threshold.

According to the theory, a higher BRC will likely act as a protective factor for an individual, thus decreasing the risk of functional impairment and the likelihood of an individual exhibiting symptoms of neurological impairment. Individuals with lower BRC acts as a vulnerability factor creating greater risk of functional impairment. In terms of this theory any reduction in BRC due to neurological pathology is likely to increase the chances of an individual to have functional impairment. An individual with less BRC will more likely show neuropsychological impairment or symptom onset (Jordan, 1997).

2.4Jordan's (1997) model of inter-individual variability

Jordan (1997) developed a *Shuttle* model of variability within the context of BRC. She postulates that owing to different levels of pre-existing cerebral reserve in association with the onset of neural attrition, due to normal ageing, the presentation of symptomatology in brain damaged groups will occur differently between individuals. This will be reflected in an increased variability of cognitive scores associated with the ageing process and declining raw scores. Specifically, due to protective factors which raise the threshold of symptoms onset, a notable percentage of individuals will not present with much cognitive fall-off. Alternatively, due to vulnerability factors which lower the threshold of symptom onset, a notable percentage of individuals will show a marked fall-off. This will result in a substantially expanded distribution of scores.

As the brain ages, protective factors which cause high BRC thresholds in some individuals become less effective, and previously good scores will perform closer to the norm. This results

in marked variability in symptom presentation between individuals. The distribution of scores will narrow again and be reflected in the reduced variability of cognitive test scores in association with the continued lowering of mean scores (Jordan, 1997).

2.5 Summary

This chapter contextualizes the theoretical framework in this study; it shows a theoretical understanding of Cumulative Mild Head Injury (CMHI) in terms of Brain Reserve Capacity (BRC) and Threshold Concept theory as a theoretical basis the study.

CHAPTER THREE: LITERATURE REVIEW

3.1 Introduction

Traumatic brain injuries (TBIs) from participation in sports and recreational activities have received increased public awareness. Prior to the 1980s mild brain injuries received relatively little attention, while moderate to severe brain injuries dominated the neurological and neuropsychological literature. Patients with mild brain injuries were thought to recover quickly and completely in the vast majority, if not all cases (Lovell, Echemendia, Barth, & Collins, 2004).

According to Partington (2001) incidence of concussion in the field of contact sports is high, not only for boxing, but also for soccer, football and especially rugby. An overview of studies investigating persistent, harmful cognitive and symptomatic outcomes following increasing sports MTBI, suggests that individuals may be at risk for permanent neurological damage following participation in a contact sport. Established sequelae of Traumatic Brain Injury (TBI) typically involving frontal systems include cognitive decline, behavioural changes such as diminished self-regulation and aggression, and increased risk for Alzheimer's disease (Brown, 2011; Shuttleworth-Edwards & Whitefield, 2007).

This section provides a context for mild head injuries (MHI) in general as there is lack of knowledge and scientific study about the topic in South Africa.

3.2 Traumatic Brain Injury (TBI)

According to Lezak et al., (2004), Traumatic Brain Injury (TBI) refers to injury involving the brain. Head trauma is classified as either open or closed injury. An open or penetrating TBI is associated with a skull fracture (Richardson, 1990) and includes all injuries from any source in which the skull and dura are penetrated. The consequence of this type of TBI is a confined focal lesion, which may result in predictive and limited cognitive losses. The degree of injury is linked to the force and depth of penetration and resultant loss of brain tissue (Lezak et al., 2004).

Closed brain injuries are referred to as blunt head injuries that is, no penetration or compressed fracture of the skull (Lezak et al., 2004).

Closed head injuries refer to a sudden acceleration and/or deceleration of the head which has come into contact with motionless or slowly moving object or as a result of impact from a faster moving object (Richardson, 1990). The severity of closed head injury depends upon how much diffuse damage is present, and can cover a range of severity from mild to moderate to severe (Lezak et al., 2004).

3.3 Mild Traumatic Brain Injury (MTBI)

Mild Traumatic Brain Injury (MTBI), also known as Mild Head Injury (MHI) can be defined as an immediate and transient impairment of neural functioning such as alteration of consciousness, disturbance of vision, equilibrium or other similar symptoms (Wilberger, 1993). Herring, Cantu, Guskiewicz, Putukian and Ben-Kibler (2006), developed the following definition seeking to transcend any limitations to previous descriptions this is stated as: a complex pathophysiological process affecting the brain, induced by traumatic biochemical forces which include a) concussion may be caused by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head b) concussion typically results in the rapid onset of short lived impairment of neurological function that resolves spontaneously c) concussion may result in neurological changes but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury, d) concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course and e) concussion is typically associated with grossly normal structural neuroimaging studies. MTBI is further sub-classified as mild, moderate and severe, depending on the length of the loss of consciousness and duration of Post-Traumatic Amnesia (PTA) which is dependent upon the period of time for which there is no recollection of events. In the sports arena where concussion tends to be the favoured term for MTBI, a series of definitions has evolved. However, there has been a lack of an accepted definition to the numerous limitations in accounting for the common symptoms present. In addition relatively minor impact injuries that result in either persistent physical or cognitive symptoms have not been included in these definitions. Mild Traumatic Brain Injury (MTBI) can occur in contact sports without the player experiencing loss of

consciousness thus it is sometimes difficult to recognise sports related MTBI. The immediate management of MTBI requires serial observation.

Elson and Ward (1994) stated that MTBI, similar to moderate and severe head injury, can also lead to diffuse axonal injury (DAI). DAI is caused by shearing forces generated by sudden brain acceleration or deceleration Alexander (1995). DAI is the primary neuropathology of head injury (HI). Alexander (1995) supported one of the first researchers Holbourn (1943) in finding that DAI is the main causal mechanism for injury and ensuring behavioural dysfunction in MTBI. In the acute phase after MTBI, a period of one to three months post injury, a range of complications from mild to severe can occur. The three areas in which MTBI is associated with problems in the acute phase post injury are cognitive deficits; such as difficulty in concentrating, memory problems and impaired problem solving (Binder, 1986); Post-Traumatic Amnesia (PTA) (Anderson, 1996) and physical symptoms; including dizziness, fatigue, sleep difficulty, nausea, headache, blurred vision and insensitivity to intense light and sound (Evans, 1992).The same symptoms that are present under physical symptoms are also present in Post Concussive Syndrome (PCS) (Anderson, 1996). Emotional sequelae have also been identified as an MTBI problem area (Evans, 1992; Szymanski & Linn, 1992) symptomology includes irritability, anger outbursts, depression, anxiety and poor social functioning (Szymanski & Linn, 1992).

According to Wilberger (1993) immediately after MTBI in the sporting arena the majority of athletes report a variety of disturbances including headaches, dizziness, memory problems, irritability, difficulty sleeping and lack of concentration. Such symptoms are typically short-lived and resolve spontaneously. However, in a small number of athletes, problems may become persistent, resulting in a post-concussion.

At an Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine meeting (1993) the spectrum of MHI was broadened to include different grades of injury severity. It was recommended that MHI be defined by the measure of at least one of the following: (a) any period of Loss of Consciousness (LOC) for less than 30 minutes, with Glasgow Coma Scale (GCS – used to measure the degree of consciousness an individual is experiencing) of 13 to 15 following the LOC; (b) any loss of memory for events immediately before or after the accident with PTA of less than 24 hours; (c) any alteration in mental state at the time of the accident (for instance, double vision, loss of

balance, taste or smell) that may or may not be transient. This definition encompassed a broader range of injury severity than was traditionally used (Satz et al., 1997).

3.4 Incidence of MHI

The exact statistics pertaining to the prevalence of closed head injuries are relatively difficult to obtain. Terms such as mild, minor, moderate, and minimal are applied to head injuries without exact or universal definitions in hospitals, trauma rooms and medical practices. The causes of MHI are similar to those that are more severe. In the early 1990's in the United States of America (USA), it was reported that Motor Vehicle Accidents (MVAs) caused 46% of MHI, whereas sports, accidents, falls and assaults caused 28%, 10% and 23% respectively. This was supported by Cassidy et al. (2004), who reported that the majority of causes of MHI globally are MVAs, accidents, falls and assaults while sports injuries comprise the bulk of the remainder. It is also notable that death rates are elevated amongst young adults who suffer severe or moderate to MHI with an incidence of 15% per 1000 per year contrasted with 2% per 1000 per year in other age groups (McMillan, Teasdale, Weir & Stewart, 2011).

There are many reasons why the exact incidence of MHI is difficult to determine, for instance:

- Most health surveys concentrate on patients who have been hospitalised. However, many patients who sustain MHI are not hospitalised but are evaluated in hospital emergency rooms or doctors' offices;
- The International Classification of Diseases (ICD, 2010) and other descriptive tools are often applied ambiguously to injuries (like maxillofacial fractures and scalp lacerations) by including them in the overall statistics for injuries to the head;
- Patients who have sustained multiple injuries that include a MHI may be classified according to their most severe or complex injury and the occurrence of MHI injury may be ignored (Dacey, Vollmer &Dikmen, 1993).

Wrightson and Gronwall (1980), carried out a study on the incidence of MHI in New Zealand, estimating the incidence to be 1 769 per 100 000 population per year for people aged 15 and beyond. For those under 15 years of age, the incidence was put at 2 929 per

100 000 population per year. These figures were supported by a review completed by Van der Naalt (2001). It has been estimated that 80% to 95% of all head injuries experienced in Europe can be considered mild (Vos et al., 2002). According to Cassidy et al., (2004) in their overview of the incidence of MHI in Europe, the incidence amongst hospital treated adults for MHI was between a 100 and 300 per 100 000. However, since the majority of MHI are not documented in emergency or medical departments, these figures probably underestimate the true incidence. They postulate that a more plausible estimate exceeds 600 out of 100 000. The authors note that in Sweden, MHI ranged from 191 out of 100 000 to 718 out of 100 000. Accident data in Sweden indicates that 50% - 60% of MHI patients admitted to a hospital were injured in a fall and 25% received their injury while operating a motor vehicle. It must be noted that MVAs in Sweden are relatively low as compared to the rest of Europe.

A study conducted by Isik, Gökyar, Yildiz, Bostanci and Ozdemir (2011) in Eastern Europe analysed 851 patients younger than 14 years old with head injuries incurred between January 2003 and June 2008. Five hundred and fifty participants were male and 301 were female. According to the GCS scores, 74% of patients had an MHI with a score of 13-15 on the scale.

An additional factor confounding estimates of the incidence of MHI injury is lack of a universal definition for this type of head injury. This is illustrated by Ryu, Feinstein, Colantonio, Streiner and Dawson (2009) who conducted a study on the variability in diagnosis of MTBI. They wanted to determine the incidence of MTBI in Canada. Potential MTBI cases were identified through reviewing three months of Emergency Department (ED) and Family Physician (FP) health records. Potential cases were selected from ED records using the International Classification of Disease, 10th revision (ICD, 2010). Documented diagnoses of MTBI were compared to expert reviewer diagnosis. Incidence of MTBI was determined using the documented diagnosis and data from hospital catchment areas and the population census. The results reported that 876 potential MTBI cases were identified with 25 cases from FP records. Key indicators of MTBI were missing on many records and only 308 of 876 patients reviewed had GCS scores. The expert reviewer disagreed with the documented diagnosis in 380 of the 876 cases. The expert calculated incidence rates of hospital treated MTBI as 426 or 545 out of a 100 000. Family physician cases increased the rate to 653 cases of MTBI out of 100 000. The research concluded that

health record documentation of key indicators for MTBI is often lacking and some patients with MTBI appear to be missed or misdiagnosed by primary care physicians. The study yielded a more comprehensive case definition that resulted in estimated incidence rates higher for MTBI's than previous reports or research.

The Congress Report on MTBI in the USA (Maroon et al. 2000), reported that more than 1.5 million people experience (TBI) each year. Of the 1.5 million who survive a TBI, 392 000 are hospitalised, 543 000 are treated in emergency departments and released inside several hours and 221 000 are treated in clinics and physicians' offices. The remaining 381 000 do not receive medical care. Of those who were hospitalised, 146 000 stayed in hospital for only one night. These data suggests that as many as 75% of people who sustain a brain injury incur a MHI. In 2001, the TBI Surveillance Programme in the state of South Carolina Department of Health, USA (SCDOH, 2001) identified 56 780 cases of TBI from 1996 to 2000. From the data that was collated 86% or 49 099 were MHI's. Of these 85% were identified through ED surveillance. However, it was postulated that overall national surveillance systems underestimate the occurrence of MTBI because they do not include injured people who received medical care in other facilities for instance, outpatient clinics, family physicians or those who receive no medical care for their injuries.

According to Menascu (2011) the United States National Coma Bank Document reports that about 85% of all head injuries requiring medical treatment are mild in nature, about 8% are moderate and the remaining 6% are severe. The incidence of closed head injuries varies significantly according to different demographic factors including gender, age and socio-economic status. They also state that most MHI do not come to the attention of health care personnel. The incidence is thus likely to be underestimated rather than overestimated. Additional difficulty in determining an accurate incidence of MHI is confounded by the inclusion of contusions to the face and scalp in these statistics which do not necessarily constitute an MHI. The ratio of boys to girls rises from approximately 1.5 to 1 for pre-school children to approximately 2 to 1 for school-age children and adolescents. These changes appear to reflect the sharp increase in head injuries among males and a gradual decrease among females (possibly because of sport's participation and other more extreme activities that traditionally males not females usually participate in). The incidence of closed head trauma also varies with age. Data demonstrates that the incidence is relatively stable from birth to age 5 years, with injuries occurring in about 160 per 100 000 children in

this age group. After age 5 the overall incidence gradually increases until early adolescence and then shows rapid growth, reaching a maximal peak of 290 per 100 000 by the age of 18 years. It is also reported that incidence of MHI varies with familial socio-economic status. It seems that more middle class families are likely to report MHI than the working classes and/or that they have more access to medical facilities.

Research carried out by Brown and Nell (1991) in South Africa reported an average of 316 per 100 000 incidents of brain injuries per year. In this study Black and Coloured men were more at risk of brain injury than White males. Black males in the 25 to 44 year age groups were most at risk of brain injury. More recent research reports that 89 000 cases of brain injuries are reported annually in South Africa (Durban Hospital, Department of Neurosurgery, 2010). The report also states that in South Africa the three most common causes of head injury are MVAs, motorbike, or vehicle-pedestrian accidents (more than 50%). Approximately 25% of head injuries are due to falls and other types of violence. These contribute to nearly 20% of the total head injuries reported per year.

3.5 Cumulative Mild Head Injury (CMHI)

There is a theory that indicates that cumulative and more permanent neuropsychological impairments arise from repeated or Cumulative Mild Head Injury (CMHI), as a result of neural abrasion, which may be harmful to an athlete's well-being (Gronwall, 1989; Rutherford, Stephens, Potter & Fernie, 2005). A study on neuro-trauma indicated that repeated head injuries result in cumulative damage to the brain and cause cumulative damage to hippocampal cells (Packard, 2008). On-going research suggests that repeated MHI or Cumulative Mild Head Injury (CMHI), which occurs over an extended period of time (weeks to months to years), is likely to result in cumulative neurological and cognitive deficits (Silver, McAllister & Arciniegas, 2009).

Players of contact sport run the risk of head injury. Shuttleworth-Edwards and Whitfield (2007) state that sports injuries, especially contact sport, fall into the category of mild head injury. As long ago as the eighties Barth et al. (1989), in the United States of America, noted that there were approximately three hundred thousand (300,000) sports injuries a year

and eighteen per cent (18%) to twenty per cent (20%) of these injuries are head). American football is the most notable where cerebral concussions occur frequentlyand more than two hundred and fifty thousand (250 000) concussions occur in this sport alone. One in five high school American football players suffers a concussion annually and physicians have become more conservative in treating such patients because the ability to process information is reduced after concussion. The duration and severity of functional impairment is greater with repeated concussions and Cantu (1995), suggested that the damaging effects of concussion are cumulative. Rugby Union is a sport where impact injuries to the neck, back and head are common and where MHI can occur as a result thereof (Shuttleworth-Jordan, Balarin & Pucheret, 1993; Nel, Grace & Nel, 2011). Research indicates that MHI frequently occurs in Rugby Union (Partington, 2001). Schoolboy rugby players suffer damage to the muscle and concussive injuries. Seward, Orchard, Howard and Collinson (1993), concur that concussions are high amongst this group and suggests that such incidents are higher than in adult Rugby Union players.

Early research into repeated MHI by Gronwall and Wrightson (1975) indicated that individuals who incur two or more concussions exhibit a decreased rate in information processing and slower reaction times than individuals who have suffered only one concussion. The same authors conclude that cognitive deficits are more persistent in patients with a history of multiple head injuries. A later study by Gronwall (1989) using participants with MHI corroborates his earlier findings in older individuals and individuals with previous head injuries. These individuals displayed impairment in speed of information processing and took longer to recover than the group with a single MHI. It is evident though studies that the sequelae following MHI may be cumulative even if an individual has recovered clinically and, in fact, that the course of recovery is prolonged after successive injuries. It is thus reasonable to conclude that there is a risk of increasingly negative consequences from subsequent head injuries (Shuttleworth-Edwards et al., 2008). The most dangerous of these consequences is referred to in the literature as second impact syndrome (SIS). This refers to when a minor second impact is sustained before full resolution of the symptoms of the first concussion and which can result in fatal brain swelling (Echemendia & Julian, 2001; Shuttleworth-Edwards et al., 2008). As a serious consequence of head injuries, Enicker & Madiba (2014) emphasized the importance of thorough cleaning and proper suturing of the scalp wounds by the emergency centre doctor who has first contact with the patient can never be over-emphasized as it minimizes

infection and progression of neurological deficits. The wound should be inspected for any foreign bodies and loose bone fragments and referred urgently for neurosurgical assessment (Enicker & Madiba, 2014).

Despite various physical health benefits, rugby carries a high risk for injury, especially head injury, and consequently has a high incidence of concussion (Alexander, 2009; Laubscher, 2006). It is common for 12 to 13 per cent of adolescent rugby players to report mild traumatic brain injury or concussion per season (Laubscher, 2006) and this has been reported to have an effect of the academic achievement of the adolescent. There is still a lack of consensus regarding the outcome of mild traumatic brain injuries. However, recent studies have shown little significance in performances in the pre- and post-season games in adolescent rugby players. For instance Nel, Grace, Nel & Govender (2015) found that the results of their study did not support findings from previous research that used standard neuropsychological test batteries. Kriel (2012) investigated whether there is a significant difference in academic achievement within and between two groups of adolescents that had either played rugby and sustained multiple concussions, or had not played rugby nor sustained any concussions, when measured at four points in time over six years. The finding that the Rugby/Concussed group did not display statistically significant intra-group differences in academic achievement when measured over time, but that academic achievement followed a downward trend, is difficult to substantiate in the literature. It may be that concussive injuries in adolescent boys do not have a cumulative effect as previously postulated or that the proper monitoring and treatment of mild head injuries or concussions and rules that make playing the game safer (Nel, et al., 2015).

The prevention of concussions should be a priority, as athletes will always be exposed to head impacts. The incidence can be mitigated to some degree by education of athletes, coaches and team management, family members and health professionals (King, Brughelli, Hume & Gissane, 2013). Rehabilitation is a crucial part of head injury management (Enicker & Madiba, 2014). Development of up-to-date educational resources is a priority to enable dissemination of appropriate up-to date information for all people involved in the identification and management of a concussion (King et al., 2013). The underreporting of concussion makes identifying the true incidence difficult, and makes diagnosis and management of concussion a challenge.

3.6 Neuropsychological functioning

Individuals who sustain severe and moderate head injuries are more likely to experience significant deficits in neuropsychological functioning than individuals who sustain MHI (Shuttleworth-Edwards et al., 2008). Research has consistently shown that the frontal and temporal lobe structures typically sustain the most focal damage following a severe head injury, although functional deficits are rarely restricted to these particular regions and loss of functioning can be observed across several systems, including memory, visual-spatial (perceptual organisation), motor, and language abilities. The aggregate of these findings raises interesting questions pertaining to the loss of specific cognitive processes as well as broad cognitive systems, post head injury, given the inter-relations of various brain regions (Lezak et al., 2004).

According to Lezak et al. (2004), visio-perceptual processing deficits and problems with perceptual organization are also commonly cited problems in individuals with head injuries. Research has shown a direct correlation between visual perceptual organisation disruption and injury severity. Furthermore, individuals with head injuries often demonstrated difficulty copying complex figures and integrating forms and figures. Disruptions within this domain appear to be particularly prevalent among children under twelve years of age, as children start contact sports as early as six years old this is particularly relevant to the concept of CMHI (Nel, Grace & Nel, 2011). Other cognitive deficits affected by TBI are seen in processing speed, researches have repeatedly demonstrated that individuals with severe head injuries are impaired compared to healthy children on timed neuropsychological tests (Gordon, 2006).

Cassidy et al. (2004), state that literature shows that developmental, structural, and decline of critical cognitive processes such as memory has been widely studied in children, adolescents, adults, and the elderly population. In particular, research has examined various aspects of memory (for example, long-term memory, short-term or working memory, subjective memory, explicit memory, implicit memory, episodic and semantic memory) among a number of clinical pathologies (for instance, clinical depression, schizophrenia, attention-deficit problems) including TBI (Lezak et al., 2004). In TBI, the majority of the studies focused on persistent deficits in verbal memory which are often evidenced post-injury; verbal memory can be pervasive following a head injury and is readily observable a

full year after the initial trauma. Findings suggest that memory deficits are typically most problematic for younger children because of the ostensible relationship between these abilities and impaired learning in school-aged children (Gordon, 2006). Although MTBI injuries are not as catastrophic as TBI injuries there is still the suggestion that, particularly with ageing and exposure to contact sports, memory will be affected (Shuttleworth-Edwards & Whitfield, 2007).

3.7 Pathophysiology of MHI

3.7.1 Diffuse Axonal Injury (DAI)

According to Lezak et al. (2004), acceleration-deceleration trauma, particularly when the brain twists or rotates within the skull (rotational acceleration), may cause axonal strain and tensile stress which may be focally diffuse. This process was referred to in the 1940's by Holbourn(1943) as shear-strain or more recently Diffuse Axonal Injury (DAI). This is one of the most common and devastating types of injury in brain trauma. It is reported as being one of the major causes of unconsciousness and persistent vegetative states after a serious head trauma (Edlow & Wu 2012). The researcher Strich (1970) was the first to comprehensively describe DAI after researching the post-mortems of people who had died after severe head injury. Diffuse Axonal Injury (DAI) is the most consistent pathology reported after moderate to severe head injury (Mureriwa, 1997). However, MTBI also involves distortion, stresses and strains of the vascular brain system and neural brain systems. These stresses and strains are typically caused by acceleration injuries resulting from movement of the head and skull which result in stretching and shearing of nerve fibres causing axonal damage (Lezak et al., 2004). It has been postulated that axonal fibres are stretched in mild brain injury occurrences, resulting in fibres becoming swollen, beaded and varicose. This effect renders the neuron dysfunctional, even though the neuron is still alive (Echemendia& Julian, 2001; Edlow& Wu, 2012; Lezak et al., 2004). These strains are often higher at the surface, than at the depth of the brain, and decrease drastically toward the centre of the brain. Autopsy investigations have indicated that the mass of the cerebral hemisphere above the cerebella structures and the stalk of the midbrain are especially vulnerable to these rotational shear strains. This type of diffuse, non-localised injury is typical of MTBI. Alexander (1995) and Holbourn (1943) noted that diffuse axonal damage

is the main causal mechanism for injury and ensuring behavioural dysfunction in MTBI. Gennarelli, Thibault and Graham (1998), describes three grades of DAI. Grade one is widespread axonal damage in the corpus callosum, the white matter of the cerebral hemisphere and the brain stem. In Grade two DAI, the focal abnormalities in the corpus callosum are often associated with small tissue tear haemorrhages and exist in addition to the injuries in Grade 1. In Grade 3 DAI, the injuries noted in Grade 2 are present with additional axonal abnormalities, commonly in the rostral brain stem, resulting from tissue tear haemorrhages. Grade 1 DAI occurs most often with MBTI and does not result in LOC but, as noted, there is axonal damage.

Diffuse Axonal Injury (DAI) alters numerous executive functions including the speed of information processing, working memory, attention span and interference control (McAllister,2005; Niogi et al., 2008; Turner & Levine, 2008). It is also likely that DAI contributes to persistent post-concussive symptomology and attentional deficits following MTBI (Niogi et al., 2008). They also note that frontal-temporal areas and anterior and inferior regions of the brain are highly susceptible to focal damage which contributes to attentional, executive and emotional control difficulties.

3.7.2 Post- Concussion Syndrome (PCS)

After a relatively minor impact to the head a common pattern of complaints termed Post-Concussion Syndrome (PCS) is found (Parker, 2009). A cluster of self-reported or subjective symptoms may persist long after the injury, even after neuropsychological testing has indicated that there has been resolution. These clusters are referred to in the literature as PCS (Sundström, 2006). This cluster of symptoms may be both acute and long term and they fall into three broad categories namely cognitive, physical and psychological symptoms.

Mild Head Injury (MHI) is described as a multi-dimensional and multi-factorial disorder and the sequelae following damage caused by such an injury will vary from person to person depending on educational level, age, premorbid neuropsychological integrity, injury characteristics and psychological reaction to the injury (Arciniegas et al., 2005). Generally, individuals recover from these symptoms without any residue within one to three months (Alexander, 1995; Ponsford, et al., 2000). Several studies however, report persistent symptomology months (sometimes years) after the initial injury (Alexander, 1995; Ryan

&Warden, 2003). The estimated prevalence of symptoms lasting months or longer is unclear. Binder, Rohling and Larrabee (1997), report prevalence of 7% to 8%, Ponsford et al. (2000), at 10% to 25% and Bohnen, Jolles and Twijnstra (1992) at 80%. When symptoms persist a PCS exists. According to King (1997) such symptomatic reactions to MHI are mediated by a number of issues from concerns about compensation claims to emotional reactions. Broadly, many persistent post-concussive symptoms are an interaction between organic and psychological factors. They usually start on an organic basis and persist and are experienced by the individual on a psychological level.

During the 1970's it was argued, within medical and neurological circles, whether or not the different post-concussive indicators could constitute a syndrome. These symptoms did not appear to form clear-cut set of clusters and therefore, according to some pundits, could not really constitute a syndrome (Rutherford, Merrett & Mcdonali, 1977). However, research, particularly factor analytic studies, has demonstrated symptom clusters within Post-Concussion Syndrome. Two clusters of complaints were identified. The first group of complaints was categorised as impairments and consisted of forgetfulness, slowness, poor concentration and inability to divide attention between two concurrent activities. These complaints were found to be related to the severity of brain injury. The second cluster of complaints consisted of all other symptoms for instance, headaches and anxiety which were labelled intolerances as they were not related to the severity of brain injury (Van Zomeren, Brouwer & Deelman, 1984).

Jacobson (1995) classified post-concussive indicators into somatic and psychological symptoms. The somatic symptoms included headaches, dizziness and fatigue. He further suggested that psychological symptoms are sub-classified into cognitive (poor memory and concentration) and psychological or affective symptoms (irritability, emotional lability, depression and anxiety). Mureriwa (1997) noted that there is a strong correlation between headaches and dizziness and between poor concentration and memory. He suggests that this demonstrates that there are strong correlations between post-concussive symptomology. Bohnen et al. (1992), identified two sub-groups of patients with MHI. They categorised these into two sub-groups after administering a 26 item questionnaire which included post-concussive, cognitive and emotional symptoms. Principal component analysis with various rotations was carried out on the responses of 71 patients 10 days after they received a head trauma. The results of the analysis indicated that the symptoms of headache, dizziness and

intolerance to environmental stimuli resulted in decreased cognitive and work performance. This group of symptoms is referred to as post-concussive or cognitive complaints. The second subgroup was categorised as emotional. It consists of emotional lability and depression as well as specific symptoms like heart palpitations and clammy hands. Boulind (2005) conducted a meta-analysis of research that focused exclusively on the acute phase of PCS in the sports context. He noted a number of physical and neurological symptoms experienced by athletes. These included headaches and dizziness, difficulty concentrating and memory problems plus poor problem solving ability and feelings of mental fogginess.

The American Psychological Association (APA, 2000) encouraged more research and communication among researchers with regards to PCS, which were included in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (APA, 1994) but not in the 5th edition(2012). The criteria which are subject to change as research is on-going (See criteria list 2, p. 40) include a history of head trauma, cognitive problems plus symptomology that lasts 3 months or longer and which doesn't improve. These symptoms must impair day-to-functioning (APA, 2000). According to Arciniegas et al. (2005), PCS or Post-Concussion Disorder in the nosology of the DSM-IVas generally signifies the development of a constellation of physical, cognitive, and emotional or behavioural post-concussive symptoms. Since there are differences in the symptomology across patients it has been suggested that PCS is a group of illnesses rather than one illness (Arciniegas et al., 2005). Diagnosing the syndrome is quite difficult because virtually all symptoms listed in PCS criteria have a high basal rate in the general population (Dikmen & Levin, 1993; Satz et al., 1999). These symptoms are also exhibited in other conditions, not involving injury to the head, including: a) Post-Traumatic Stress Disorder or PTSD (Hickling, Gillen, Blanchard, Buckley & Taylor, 1998); b) anxiety and/or depressive disorders (King, 1997; McCauley, Boake, Levin, Contant& Song, 2001) and c) chronic pain (Gasquoine, 1997; Smith-Seemiller, Fow, Kant & Franzen, 2003). It must be noted that the problems experienced by individuals with MHI are more precisely understood as post-concussive symptoms rather than Post- Concussion Syndrome per se (Niogi et al., 2008).

Females may be more at risk for developing PCS, even though men are more likely to sustain head injury (Gasquoine, 1997; McCauley et al., 2001). A small but significant effect size was noted in a meta-analysis of eight studies on mild to severe head injuries, finding an overall outcome for women are worse than men (Farace & Alves, 2000). One explanation

of this might be related to differences in the mechanisms of injury. In particular, a greater percentage of females sustain head injuries in MVAs 57% versus 22%, whereas a greater percentage of males sustain head injuries whilst participating in sports, 33% versus 9%. Research also indicates that sports injuries exhibit less persistent sequelae than MVAs (Bazarian, Wong, Harris, Leahey, Mookerjee & Dombovy, 1999). This difference might also be due to under-reporting of sports related MHI because participants do not want to be removed from play (Carroll et al., 2004). The older the individual is the more susceptible they are to persistent sequelae. The risk of persistent sequelae post-head injury is twice as high at age 40 than at age 30 (Arciniegas et al., 2005; Kashluba et al., 2004). Injuries tend to differ with age for instance; younger adults are more likely to be injured in MVAs whereas older adults are more likely to be injured in falls (Thurman, Alverson, Dunn, Guerrero & Sniezek, 1999). Pre-injury factors may also play a role in any age-related differences because the elderly injured in falls are more likely to have other ailments impacting on PCS outcomes (Lange, Iverson & Rose, 2010). However, some studies did not identify a significant relationship between age and outcomes after head injuries (Breed, Flanagan & Watson, 2004). There are no clear explanations for the inconsistent relationships between age and PCS. Some studies suggest that differences are related to the level of injury severity which impacts on positive PCS outcomes. The relationship between pre-injury personality characteristics and MHI has been suggested as an explanation for persistent symptomology. Parker (2009) suggests that personality change can be a consequence of MHI. Other studies believe poor coping skills may explain prolonged PCS (Bohnen et al., 1992; Ponsford, et al., 2000). Persistent symptoms after MHI can be associated with malingering especially if litigation is involved (Kashluba et al., 2004). This is underpinned by a study by Binder et al. (1997), who performed a meta-analysis to assess the relationship between potential financial gain and clinical outcomes after head injury. They concluded that financial incentives had a positive effect on the development of persistent PCS, especially in patients with MHI.

3.7.2.1 Acute symptoms and symptom recovery from Post- Concussion Syndrome (PCS)

McCrea (2007), reports that the diagnosis of concussion or MTBI is to a large extend based on the subjective symptoms reported by the patient. It is the same for the recovery after MTBI, as most often this is determined by the patient's self-reported resolution of concussion symptoms. The diagnosis of PCS is reserved for those patients with persistent

complaints after MTBI. The importance of symptoms diagnosis and prognosis highlights the need to establish empirically defined parameters for the expected course of recovery. Some studies have generated evidence that is based on the natural history of symptom recovery after MTBI which guides the clinician in interpreting a patient's persistent complaint at the individual level.

The World Health Organization (WHO), in 2004 collaborated on a study of MTBI which published a detailed review of literature on the prognosis after a mild brain injury (Carroll et al., 2004). One hundred and twenty studies of best-evidence on prognosis after MTBI met their criteria for inclusion in the review. There was consistent and methodologically sound evidence that children's prognosis after MTBI is good, with quick the resolution of symptoms and with little evidence of residual cognitive, behavioural or academic deficits. They found that adults' cognitive deficits and symptoms were common in the acute stage and many of the studies reported that recovery for most adults is within 3 to 12 months post-injury. Where compensation and litigation were reported as factors in sustained symptomology however, there was very little consistent evidence for other predictors of long term effects. The report concluded that symptoms after MTBI are typically temporary in nature for both children and adults, with quick or gradual resolution within days to weeks post-injury in the majority of patients. MTBI symptoms are highly non-specific and often are the same to those reported after other types of injury for example, orthopaedic injury. The World Health Organisation (WHO, 2004) task force summarised the results of several studies on self-reported symptoms on MTBIand noted that headaches, blurred vision, dizziness, subjective memory problems and sleep problems were the most commonly experienced symptoms.

3.8 Neuropsychological sequelae of MHI

Individuals who sustain MTBI often report a group of adverse events, usually referred to as post-concussive symptoms. As previously stated post-concussive symptoms are commonly divided into three categories physical, emotional and cognitive. The latter include problems with memory, concentration, initiation, planning, and problem solving as well as difficulties with attention and slowed information processing speed (Ruff, 2005). A total of 10-20% of MTBI patients experience post-concussive problems or symptoms that persist beyond the recognised recovery period of approximately 6-12 months (Alexander, 1995; Ruff, 2005).

Physical symptoms include headache, fatigue, dizziness, blurred vision, light and sound sensitivity as well as sleep disturbance (Paré, Rabin, Fogel & Pépin, 2009; Ziino & Ponsford, 2006). Fatigue is one of the most reported symptoms after a MHI. Severe fatigue may affect cognition interfering with return to work, exercise and sports programmes and it may also limit social interactions (Borgaro, Baker, Wethe, Prigatano & Kwasnica, 2005; Sundström, 2006). Immediately after an MHI nearly 50% of individuals report that they become fatigued easily, this impacts on their quality of life (Carroll et al., 2004). Fatigue may be the primary effect of Central Nervous System (CNS) dysfunction, or a secondary effect of sleep disturbance or depression post MHI, and may also confound neuropsychological assessments (Arciniegas et al., 2005). Psychological and emotional symptoms such as depression are also commonly reported after a MHI (Busch & Alpern, 1998; Holsinger et al., 2002). Other symptoms frequently reported after all types of head injury are anxiety and irritability (Alexander, 1995; Pare, et al., 2009). As these symptoms often co-exist they can be difficult to assess and separate (Van der Linden et al., 1999).

Interestingly, these symptoms are similar to patients who experience TBI. For instance, an investigation in the USA by Englander, Bushnik, Oggins and Katznelson (2010), attempted to define any association between fatigue and abnormalities in sleep, mood, cognitive, physical and hormonal functioning after TBI. The study was conducted in a rehabilitation centre hospital. There were 119 participants who had experienced a head injury at least one year before the investigation. The findings of the study noted that 53% of the sample reported fatigue on the Multidimensional Assessment of Fatigue (MAF), while one third reported it on the Fatigue Severity Scale (FSS). Sixty five percent (65%) were found to have moderate to severe growth hormone deficiency and 15% of the men had a testosterone deficiency. The study concluded that fatigue after brain injury is linked to gender, depression, pain, memory problems, hormonal deficiencies and motor dysfunction. It was suggested that investigation of post TBI fatigue should include the screening for depression, pain and sleep disturbances.

Kerr (2010) conducted a pilot study into the cognitive and neuropsychological sequelae of MHI in children, aged between 6 and 12 years old, with a GCS of 15. They were assessed using the CANTAB or Cambridge Neuropsychological Test Automated Battery (See appendix F) to examine their neuropsychological performance after sustaining MHI. The outcome of the study was that children with MHI had a significantly poorer performance

with regards to problem-solving on the CANTAB. It was postulated that this played a part in their impulsive tendencies and poor planning ability. The results suggested that children with MHI may be at risk of developing problems with high-level cognitive functioning post injury.

The term plasticity refers to the brain's ability to learn, remember and forget as well as its capacity to reorganize and recover from injury. Brain insults cause rapid cell death, and a disruption of functional circuits, in the affected regions. As the injured tissue recovers from events associated with cell death, regenerative processes are activated that over months lead to a certain degree of functional recovery. Factors produced by new neurons and glia, axonal sprouting of surviving neurons, and new synapse formation help to re-establish some of the lost functions. The timing and location of such events is crucial in the success of the regenerative process (Wieloch & Nikolich, 2006). Children have enhanced capacity for learning and memory compared to adults as reflected in their ability to learn a second language, play musical instruments or become proficient in complicated sports such as golf or tennis. Children also have remarkable ability to recover from early brain injuries as demonstrated by their ability to recover receptive language after left hemispherectomy performed for epilepsy as late as the second decade (Kulak & Sobaniec, 2004). Mechanisms of plasticity include: first, a change in the balance of excitation and inhibition; second, a long-term potentiation (LTP) or long-term depression (LTD); third, a change in neuronal membrane excitability; fourth, the anatomical changes, which need a longer period of time. Specific anatomical changes include formation of new axon terminals and new synapses.

3.9 Neuropsychological assessment

Lovell (2008) states that the use of neuropsychological testing in sports medicine was developed in the mid-1980's when professional sport became the international money making entity it is today. Lovell (2008) states that Barth et al. (1989), and his colleagues at the University of Virginia (USA) demonstrated the usefulness of neuropsychological test procedures to document cognitive recovery within the first week of concussion. A series of events transpired in the early 1990's and that shifted the use of neuropsychological testing into clinical sports medicine. A number of high profile professional athletes in American

Football were injured and did not recover timeously, costing their franchises much money. This resulted in the implementation of baseline neuropsychological testing by several American Football clubs in the mid 1990's. The National Hockey League (NHL), also in the USA, mandated baseline neuropsychological assessments for all athletes for similar reasons. A number of large scale studies of collegiate athletes in America were completed, in addition to the increased use of neuropsychological testing in sports. These studies provided further evidence that neuropsychological tests yielded useful clinical information. Neuropsychological testing has specifically allowed a baseline and post-injury analysis of the subtle cognitive functions likely to be affected by concussive injury. This has provided objective data to help compile return-to-play and treatment protocols for all kinds of professional sports. The author further stated that the use of traditional neuropsychological testing (pencil and paper testing) and later computerised testing has resulted in the rapid expansion of knowledge regarding concussion.

According to McCrory et al. (2009), the application of neuropsychological (NP) testing in concussion is of clinical value and contributes important information in contemporary evaluations of concussion. It has been demonstrated that cognitive recovery may occasionally precede, or more commonly follow, clinical symptom resolution. This indicates that the assessment of cognitive function is an important part in any return to play protocol in professional sport. It should be emphasised that NP testing should not be the only basis for the management of return-to-play decisions. It should be seen as a part of the clinical decision making process in conjunction with a range of various clinical domains and investigations. In most cases, NP testing is used to assist return-to-play decisions, and is not carried out until an individual is symptom free. Neuropsychologists are in the best position to interpret NP test results by virtue of their background and training. In instances where a neuropsychologist is not available other medical professionals, particularly neurologists, perform or interpret NP screening tests. All-inclusive concussion management protocols have been advocated in a number of consensus statements and should include baseline cognitive and postural stability (utilising a bio-kineticist) testing, especially for athletes in high-risk sports (Tommasone & McLeod, 2006).

3.10 Sporting codes

Studies have shown that CMHI can also be seen in other contact sporting codes such as American Football and boxing. In a study conducted by The North Carolina Center for the Study of Retired Athletes (2007) it was reported that of the 595 retired National Football League (NFL) players who recalled sustaining 3 or more concussions on the American Football field, 20.2% said that they had suffered depression (this is three times the rate of players who had not sustained concussion). The New York Times (2011) reported a study that was conducted in 2000 from a sample of former and retired NFL players. The study surveyed 1 090 former NFL players and found that around 60% had suffered at least one concussion in their career and 26% suffered three or more concussions. Those who had suffered more concussions reported more problems with memory, concentration, speech impediments, headaches and other neurological problems than those who did not suffer any (or only one concussion).

Heilbronner et al. (2009), report that researchers have become aware that the most important factor contributing to the severity and long term consequences of head trauma in boxing is not necessarily the number of knock outs, but rather the subtle and chronic cumulative effect of multiple blows sustained over a period of time. He does, however, note that other researchers have reported null outcomes when examining the neurocognitive effects of amateur boxing. Furthermore, the author states that studies investigating the neuropsychological effects of amateur boxing have revealed few, possibly negligible, neurocognitive deficits. It was noted that the majority of studies on amateur boxing indicate that it does not lead to the extent and degree of deficits found in professional boxers.

3.11. Rugby Union

Rugby is a form of contact sport played by two teams consisting of fifteen players each. The objective of the game is to score as much points against the opposing team. Rugby football is divided into the rugby league and rugby union where Rugby union is the more common type played in the world. Rugby union is dominantly played by first tier countries such as Australia, New Zealand, Argentina, France, Scotland and South Africa. The majority of research on CMHI in the sport has been conducted in South Africa by Shuttleworth-Jordan (1999) with various other researchers at Rhodes University, Grahamstown. Rugby union is a collision sport that exposes players to cervical spinal

injuries, with permanent disabling injuries being the most serious and highly publicised complication (Viljoen & Patricios, 2012). Rugby is a fast-moving and high-intensity team sport that result in numerous injuries to the players; on average, players perform 20-40 tackles per match and as a result, the prevalence of injury is high. Almost 25% of neck injuries occur when there is a mismatch in experience between the two opposing front rows. Over 40% of the injuries are muscular strains or bruising, 30% are strains, followed by dislocations, fractures, lacerations and overuse injuries. From the inception of the game of Rugby Union, which is speculated to have been between the start of the 1600s and the mid-1800s, the game has been regarded as a violent sport and the formation of the Rugby Football Union in 1871 was necessitated, in part, by the need for laws to reduce this violence (Richards, 2007).

The laws of Rugby Union have developed in both number and complexity from these amateur beginnings to the game's professional present. Subsequent law development has not only had the motive of reducing unnecessary injury (Burry & Calcinai, 1988) but also to distinguish Rugby Union from other codes of rugby and from simpler games that were thought to be for the common working classes (Richards, 2007). Despite this exclusivity, Rugby Union, like all other sports, was forced to compete with other sports for spectators. As a result, from as early as the 1876 - 1877 season, in which the Rugby Football Union (RFU) reduced the number of players on the field from 300 to 30, there have been numerous examples of law changes that were brought about to enhance spectator gratification. The result has been a faster, more open, running game which is believed to be associated with an increased risk of injury due to the greater speed and frequency of physical contacts (Noakes & Du Plessis, 1996).

This concern about catastrophic injuries resulted in the RugbySmart intervention programme being adapted, with permission, for the South African context. The South African programme, BokSmart, was launched in July 2009. The BokSmart programme has four main elements (Viljoen & Patricios, 2012):

• BokSmart Rugby Safety Workshops – a compulsory DVD facilitated course that all coaches and referees in SA attend on a biennial basis

• BokSmart Rugby Medic Programme – an entry-level rugby first-aid short course aimed at training members of underprivileged rugby-playing communities

• A toll-free BokSmart Spine-line number, which assists in the management and road transport of head-, neck- and spine injured rugby players to the nearest appropriate medical facility

• A freely accessible online educational resource (http://www.boksmart.com), which provides researched documentation and practical advice on a variety of rugby-related topics.

The elements of the programme demonstrate the practical implementation of an injury prevention and rugby safety programme using an evidence-based approach, yet keeping in mind the rugby landscape within a South African context in an attempt to maximise adoption and impact of the programme. The ultimate result is aimed at safer rugby, and fewer catastrophic injuries. Key to maximising compliance and uptake is on-going research, keeping current with the injury patterns of the game, regular interaction and collaboration with all rugby stakeholders, constant education of all role players, and fluid and modifiable implementation strategies that are moulded to the needs of the times (Viljoen & Patricios, 2012).

3.12 Summary

The chapter discussed appropriate literature relevant to this study.A range of relevant literature describing head injury neuropsychological functioning, neuropsychological assessment and sporting codes that deal with CMHI, as well as Rugby Union were discussed.

CHAPTER FOUR: RESEARCH METHODOLOGY

4.1 Introduction

This chapter focuses on the research methodology used in this study, which includes the research design and hypotheses, sampling, data collection and data collection tools which includes questionnaires and a neuropsychological test battery, data analysis, reliability, variability and bias as well as ethical considerations.

4.2 Research design

The research used a quantitative approach. A quasi-experimental research design was utilised by the study. Quasi-experimental designs can be used to provide preliminary support for verifying potentially important treatments (Bless, Higson-Smith, & Kagee, 2006). In this research an experimental group (rugby players) and a control group (volleyball players) were utilised.

4.3 Sampling

4.3.1 Area of the study

The study was conducted at the University of Limpopo (Turfloop Campus). The University of Limpopo is situated in Limpopo Province, South Africa. Formerly known as the University of the North, the University of Limpopo was established in 1959 under the apartheid regime for previously disadvantaged students. It is located 40km east of the Polokwane town.

4.3.2 Sample

According to Welman, Kruger and Mitchell (2012) purposive sampling allows the researcher to gain an initial understanding of phenomena and allows the research to identify and differentiate the needs of one or more relevant groups. It produces a sample where the included groups are selected according to specific characteristics that are considered to be important, as related to the variables that are going to be studied.

The study sample therefore consisted of all male rugby players (contact sport) at the University of Limpopo (Turfloop Campus) and a control group sample of all male volleyball players (non-contact sport). As the study is neuropsychological in nature and

investigates CMHI, the sample was treated as a clinical sample in line with studies conducted at Rhodes University, Grahamstown (Shuttleworth-Jordan, Balarin & Pucheret, 1993; Shuttleworth-Edwards & Whitfield, 2007). This allowed for the use of inferential statistics in the analysis of data. The final sample consisted of 18 rugby players (9 forwards and 9 backline players) and 18 volleyball players.

Exclusions from the study included a) any rugby or volleyball player who reported to drinking large amounts of alcohol or taking recreational drugs or who has been taken prescribed medication. The rationale is that any drug (including alcohol in large amounts) will impede function on psychological testing; b) any rugby or volleyball player who suffered a TBI as a result of an MVA or any other accident; c) any rugby or volleyball player who suffered MTBI or CMHI and been admitted to hospital within 2 years of the research study; d) students who were female were excluded because they were limited in number in the targeted sporting codes and e) any prospective participant who suffered an illness at the time of the investigation and/or suffered any illness in the last 2 years likely to have impacted on his ability to complete neuropsychological testing, for instance meningitis. The biological and pre-assessment questionnaire elicited this information (see appendix 1).

4.4 Data collection

The data was collected pre and post-season. It was collected six weeks pre-season and six weeks after the seasons ended (post-season). The data was collected by the researcher who was shown how to administer the test battery which was used in previous studies (Ancer, 1999; Border, 1999; Partington, 2001; Reid, 1998). A quiet room on Campus was found where the psychological tests were administered without the participant being distracted. The participant was made comfortable, and as testing took over an hour for each individual, water was offered. The participants were tested on Saturday mornings so that they would not be tired after classes or rugby or volleyball practice.

4.5 Research hypotheses

The study hypotheses:

1. Rugby players show greater cognitive deficit, in terms of significant difference in mean scores, than the non-contact sport group both pre and post season due to the effects of CMHI.
2. Rugby players will exhibit poorer cognitive profiles than non-contact sport group both pre and post season.

4.6 Data collection tools

Tools used in this study were specifically selected to measure factors that may contribute to the hypothesis.

4.6.1 Questionnaires

The two questionnaires that were used were developed by researchers involved in the continuing studies into MHI in Rugby Union players at Rhodes University Psychology Clinic (Ancer, 1999; Border, 1999; Partington, 2001; Reid, 1998). Questionnaire 1 (Appendix 1) was used to obtain the participant's demographic information such as age, years of schooling, sports played and previous sports related head injuries acquired. Questionnaire 2 (Appendix 2) was used to obtain self-reported information about any post-concussive or sub-concussive injuries suffered.

4.6.2 Neuropsychological Test Battery

As all the rugby players and volleyball players are studying at the University of Limpopo (Turfloop Campus) there was no need to test Intelligence Quotient (IQ) as all have matriculation exemptions and can be inferred to have a normal IQ. A modified version of a neuropsychological test battery utilising specific tests on The Wechsler Adult Intelligence Scale (WAIS IV, 2013) was used in line with previous studies at Rhodes University, Grahamstown (Ancer, 1999; Border, 1999; Partington, 2001; Reid, 1998). The tests are too big to add as appendices and are therefore described in this chapter. The tests employed in this investigation are:

a) Tests for visuo-perceptual tracking:

Digit Symbol Substitution Subtest: this subtest consists of 67 digits in three rows with an open block below each digit. The examiner first demonstrates how the test is completed by filing in the sample section at the top of the sheet. The participant has to copy the symbol corresponding to the digit into the open block; the key is provided at the top of the sheet and is numbered from 1-9. Instructions are given and followed according to the WAIS IV manual. Even when brain damage is minimal, this test has been shown to be sensitive to diffuse brain injury (WAIS, 2013).

Trail Making Test Part A: The test consists of a number of circles enclosing numbers on a sheet of paper. The participant has to connect up the circles sequentially, that is, the numbers in the circles must be followed in order of 1, 2, 3, etc. The researcher has to watch carefully and if the subject makes an error it must be corrected immediately. The time taken to complete the test is recorded. The instructions and administration are followed according to the WAIS IV manual.

Trail Making Test Part B: This test is administered in the same way. It has a number of circles enclosing letters of the alphabet as well as numbers. The subject is required to connect the circles but this time the letters of the alphabet must follow in the correct order. That is, A, B, C, etc. this requires a shift in response set (Lezak et al., 2004) and involves complex Visio-perceptual tracking skills. It also requires the subjects make use of working memory function.

b) Visual memory tests

Digit Symbol Incidental Recall: The testuses the Digit Symbol Substitution test previously described above. It is used in this instance to test recent memory function, which has been shown to be susceptible to the effect of diffuse brain injury (Shuttleworth-Jordan & Bode, 1995). The participant completes the Digit Symbol Substitution test and when finished is required to fill in as many symbols as he can remember on an empty grid, numbered 1-9, on another test sheet. This test is not timed.

Digit Symbol Delayed Recall: the test requires the participant to fill in as many symbols from 1-9 as can be recalled in the correct order; it takes place after a 20-minute delay. Delayed memory tends to be more susceptible to the effect of diffuse brain damage that immediate memory (Lezak, et al., 2004).

c) Verbal memory tests

Digit Span Subtest: this particular test examines verbal memory. The subject is required to repeat, after the researcher, a set of digit forward and then on a separate test a set of digit backwards. The score is the longest series of numbers that the participant can remember correctly. The test engage differing cognitive processes and are therefore analysed individually. Digit forwards tests immediate verbal memory and is primarily a test of attention or freedom from distraction. The digit backwards tests working memory function; this test is very sensitive to diffuse brain damage which may occur after closed head injury (Lezak et al., 2004).

Associate Learning subtest- Immediate Recall (Wechsler Memory Scale): this test is made up of a list of 10-paired words, which are divided into 5 easy, and 5 hard pairs. The researcher reads the list and then repeats the list but only reads the first word. The participant then has to recall the paired word. This is repeated three times and the easy pairs rely on old associate learning patterns whereas the hard pairs rely on new learning ability (Lezak et al. 2004), hard pairs are more susceptible to picking up indicators of diffuse brain damage.

WMS Associate Learning Delayed Recall: after a delay of 20 minutes the test is repeated because delayed memory is more susceptible to the effects of diffuse brain damage. This would typically show lower scores in individuals who had suffered MHI against normal controls (Stuss, Ely, Hugenholtz, Richard, LaRochelle, Poire, et al., 1985).

4.7 Data Analysis

A comparison of group means and standard deviation was conducted across all the neuropsychological tests. For every test the mean and standard deviation were conducted for the rugby and volleyball group. Appropriate data was analysed using an independent t-test and the f – statistic (Standard Deviation = SD) will be reported.

4.8 Reliability, validity and bias

As the psychological tests are standardised they measure what they are supposed to measure. The following threats to internal validity may occur (adapted from Cook and Campbell, 1979).

a) History

According to Cook and Campbell (1979) this kind of threat might take place when an effect that is observed might be due to different events that take place between pre and post season testing.

b) Maturation

The time between pre and post testing will be short however, maturation is an effect which can take place due to the participants ageing which assumes growing more knowledgeable or more experienced between pre and post season.

c) Mortality

This type of threat occurs if participants drop out of the experimental and/or control group post-season (leaving fewer participants).

d) Selection maturation

Selection-maturation is when participants amongst the rugby (and volleyball controls) mature at different speeds.

e) Selection-history

This is linked to the rugby and volleyball group participants' different environmental contexts, which is called their distinctive local history. According to Cook and Campbell (1979) this may have an impact on the outcomes of the proposed research. Any threats to internal validity will be noted and reported in the analysis of results and final conclusions.

4.9 Bias

The whole protocol has been used in previous studies undertaken at Rhodes University and is thus appropriate for the study needs. Administrator bias was controlled for by the researcher being taught how to administer the psychological tests by the co-supervisor.

4.10Ethical considerations

Ethical clearance was sought through relevant (See appendix 3) channels at the University of Limpopo (Turfloop Campus). The participants were informed about the research and the intention of the research, which was to evaluate the cognitive profiles of cumulative mild head injury (CMHI) in contact sports in rugby players as compared with non-contact sport controls. This gave participants enough information to be able to make an informed decision in regards to participation. This was carried out through a consent form that entailed information about the study's confidentiality and anonymity. The consent form was filled in by the participant. They were also free to end their participation at any time. No physical or psychological harm was anticipated (nor found) towards the participants. The neuropsychological testing process did not infringe on any of the participants' rights and they were not forced to participate. The participants were also informed about their opportunity to inquire about the results of the study. No vulnerable populations were recruited in this study as they did not fall into the scope of the sample.

4.11 Summary

The chapter aimed to conceptualise the methodology used in the study. The study is a quasi-experimental research. It highlighted the methods of collection, interpretation, validity and ethical considerations for the study.

CHAPTER FIVE: RESEARCH RESULTS

5.1 Introduction

This chapter focuses on the manner in which data were analysed including all the statistical methods used. The group mean comparisons, standard deviations, t-statistics, f-statistics significant difference and comparison of variability across the neuropsychological test battery, between rugby versus volleyball players. All the data were cleaned, coded and before beginning analysis. Before any analysis could commence, all relevant scale items were reverse scored, and missing values were replaced with the mean. The results on the post-concussive symptomology are reported where there is a difference in frequency between the rugby and volleyball playing control group.

5.2 Demographic analysis

Table 1, Table 2 and Table 3 below shows the participants' socio-demographic information. The sample consisted of 36 registered male learners from the University of Limpopo who played rugby and volleyball at the time of the investigation. The university is predominantly Black African in composition but it does comprise of different ethnic groups.

Table 1 below, describes the participants' age in categories where in the age category of 17-20 there were 7 rugby players and 8 volleyball players. In the 20-25 category, both rugby and volleyball presented with the same number of players (10) and only one participant from the rugby players fell into the above 30 category. The average starting age for playing rugby was approximately15 years and the starting age for playing volleyball was 14 years.

Table 1: Description of participants' Age demographics (N= 36)

Age	**Rugby players N**	**Volleyball players N**
17-20	7	8
20-25	10	10
Above 30	1	0
Total	10	18

Table 2: Description of participants' ethnicity demographics (N= 36)

Ethnic	**Rugby players N**	**Volleyball players N**
Sepedi	10	16
Setswana	1	0
Shona	2	0
Siswati	3	0
Xitsonga	2	2
Total	18	18

According to Table 2, most participants were Sepedi speaking (rugby 10, volleyball 16), the Setswana group was made up of 1 rugby player, the Shona and Siswati made up of 2 and 3 rugby players respectively. The Xitsonga group made of 2 rugby players and 2 volleyball players.

Table 3: Description of participants' level of study demographics (N= 36)

Level	Rugby players N	Volleyball players N
First year	4	4
Second year	4	3
Third year	8	8
Fourth year	2	3
Total	18	18

In Table 3, both the rugby and volleyball players reported 4 participants each in the first year. Participants from the second year were 4 rugby and 3 volleyball players. Third year reported 8 players each from the rugby and volleyball teams and the fourth year participants were 2 from the rugby players and 3 from the volleyball players

5.3 Analysis of means and variability across the test battery

The group mean comparisons and comparison of variability across the neuropsychological tests battery, standard deviations, t-statistics, f-statistic and statistical differences ($p<.0.05$) will be presented for rugby versus volleyball players pre and post season. The sample was made up of 9 rugby playing forwards, 9 rugby backline players and 18 volleyball players (non-contact sport). No players of rugby or volleyball were excluded from the research as per exclusion category (see 4.3.2).

5.3.1 Comparisons of means and variability across the neuropsychological test battery

5.3.1.1. Rugby players versus volleyball players

According to the mean scores pre-season (Table 4), there were no significant differences in all of the test scores in both the rugby players and the volleyball players, rugby forwards versus volleyball players, rugby backline players versus the volleyball controls and rugby

forwards versus the rugby backline players. In terms of the mean scores of post-season (Table 5), no significant results were found in these results.

The rugby players showed significantly increased variability on the test of Vocabulary ($p<.041$), and Associate Learning Easy- Immediate Recall ($p<.013$) in comparison with volleyball playing group in the post-season.

Table Legend/Key	
VOCA	Vocabulary
ALE	Association Learning Easy Pairs
ALH	Association Learning Hard Pairs
ALT	Association Learning Total
ALDR	Association Learning Delayed Recall
DST	Digit Symbol substitution Total
TMAT	Trail Making test A Total
TMBT	Trail Making test B Total
PCT	Picture completion test Total
DSubIREC	Digit Substitution Immediate Recall
DSubDRec	Digit Substitution Delayed Recall

Table 4: Pre-season comparison of Means (t-statistic) and Standard Deviation (f-statistic) between rugby players versus volleyball players

TEST	RUGBY			VOLLEYBALL			T-TEST		LEVENE'S	
	z N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	18	28.72	8.050	18	32.33	6.371	-1.492	.145	1.224	.276
ALE	18	18.50	.857	18	18.39	.850	.390	.699	.006	.941
ALH	18	8.11	2.324	18	8.06	2.235	.073	.942	.012	.914
ALT	18	17.306	2.5445	18	17.250	2.4808	.066	.948	.006	.941
ALDR	18	9.50	1.098	18	9.50	1.098	0.000	1.000	0.000	1.000
DST	18	1.89	1.875	18	1.50	1.543	.679	.502	1.476	.233

TMAT	18	28.00	3.447	18	27.28	2.927	.678	.503	.419	.522
TMBT	18	71.11	7.226	18	70.83	4.878	.135	.893	.194	.663
PCT	18	17.72	3.444	18	17.83	3.222	-.100	.921	.136	.714
DSubIREC	18	8.44	.511	18	8.50	.514	-.325	.747	.212	.648
DSubDRec	18	8.22	.943	18	8.28	.958	-.175	.862	.023	.881

Table 5: Post-season comparison of Means (t-statistic) and Standard Deviation (f-statistic) between rugby players versus volleyball players

TEST	RUGBY			VOLLEYBALL			T-TEST		LEVENE'S	
	N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	**18**	**28.61**	**9.140**	**18**	**32.33**	**6.371**	**-1.417**	**.165**	**4.533**	**.041+***
ALE	**18**	**18.78**	**.428**	**18**	**18.39**	**.850**	**1.734**	**.092**	**6.884**	**.013+***
ALH	18	9.06	1.474	18	8.06	2.235	1.584	.122	1.166	.288
ALT	18	18.417	1.4168	18	17.250	2.4808	1.733	.092	2.034	.163
ALDR	18	9.72	.575	18	9.50	1.098	.761	.452	3.083	.088
DST	18	1.33	1.847	18	1.50	1.543	-.294	.771	.420	.521
TMAT	18	26.56	3.650	18	26.33	3.430	.188	.852	.234	.632
TMBT	18	69.83	7.778	18	69.83	7.778	-.259	.797	.546	.465
PCT	18	17.89	3.787	18	17.83	3.222	.047	.962	.230	.635
DSubIREC	18	8.61	.502	18	8.50	.514	.656	.516	.883	.354
DSubDRec	18	8.28	.895	18	8.28	.958	.000	1.000	.000	1.000

NB: Marked effects significant if $p < 0.05$*

5.3.1.2. Rugby forwards versus volleyballplayers

In terms of the mean scores of pre- and post-season (Table 6 and Table 7), no significant differences were found in these results.

There was significant increase in variance by the rugby forwards in the test of Associate Learning –Delayed recall ($p<.005$) and Digit Symbol Substitution- Immediate recall ($p<.011$) as compared to volleyball players in the post-season.

Table 6: Pre-season comparison of means (t-statistic) and Standard Deviation (f-statistic) between rugby forwards versus volleyball players

TEST	RUGBY F			VOLLEYBALL			T-TEST		LEVENE'S	
	N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	9	29.89	7.356	18	32.33	6.371	-.893	.380	.149	.703
ALE	9	18.44	.726	18	18.39	.850	.168	.868	.120	.732
ALH	9	8.33	2.598	18	8.06	2.235	.289	.775	.001	.978
ALT	9	17.500	2.8284	18	17.250	2.4808	.236	.816	.001	.971
ALDR	9	9.56	1.333	18	9.50	1.098	.115	.909	.001	.972
DST	9	1.78	1.922	18	1.50	1.543	.406	.688	.180	.675
TMAT	9	27.22	3.701	18	27.28	2.927	-.043	.966	1.346	.257
TMBT	9	68.11	8.492	18	70.83	4.878	-1.064	.297	2.687	.114
PCT	9	18.33	3.122	18	17.83	3.222	.384	.704	.046	.832
DSubIREC	9	8.56	.527	18	8.50	.514	.262	.795	.208	.652
DSubDRec	9	8.11	1.269	18	8.28	.958	-.382	.705	1.023	.322

Table 7: Post-season comparison of means (t-statistic) and Standard Deviation (f-statistic) between rugby forwards versus volleyball players

TEST	RUGBY F			VOLLEYBALL			T-TEST		LEVENE'S	
	N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	9	28.89	8.753	18	32.33	6.371	-1.169	.254	1.468	.237
ALE	9	18.78	.441	18	18.39	.850	1.281	.212	3.738	.065
ALH	9	9.56	1.130	18	8.06	2.235	1.883	.071	1.676	.207
ALT	9	18.944	1.2105	18	17.250	2.4808	1.924	.066	1.805	.191
ALDR	**9**	**10.00**	**.000**	**18**	**9.50**	**1.098**	**1.353**	**.188**	**9.441**	**.005+***
DST	9	.78	1.481	18	1.50	1.543	-1.161	.257	.405	.530
TMAT	9	25.11	3.822	18	26.33	3.430	-.841	.408	.039	.844
TMBT	9	66.44	9.029	18	70.39	4.692	-1.508	.144	3.765	.064
PCT	9	18.00	3.674	18	17.83	3.222	.121	.905	.003	.955
DSubIREC	**9**	**8.78**	**.441**	**18**	**8.50**	**.514**	**1.383**	**.179**	**7.440**	**.011+***
DSubDRec	9	8.44	1.014	18	8.28	.958	.418	.679	.005	.942

NB: Marked effects significant if $p < 0.05$*

5.3.1.3 Rugby backlineversus volleyball players

In terms of the mean scores of pre- and post-season (Table 8 and Table 9), no significant results were found in these results.

Significant increase in variance was shown by the rugby backline players in the post-season in the test of Vocabulary ($p<.034$) as opposed to the volleyball playing group.

Table 8: Pre-season comparison of means (t-statistic) and Standard Deviation (f-statistic) between rugby backline versus volleyball players

TEST	RUGBY B			VOLLEYBALL			T-TEST		LEVENE'S	
	N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	9	27.56	8.974	18	32.33	6.371	-1.602	.122	1.595	.218
ALE	9	18.56	1.014	18	18.39	.850	.451	.656	.003	.958
ALH	9	7.89	2.147	18	8.06	2.235	-.185	.855	.014	.907
ALT	9	17.111	2.3819	18	17.250	2.4808	-.139	.891	.001	.982
ALDR	9	9.44	.882	18	9.50	1.098	-.132	.896	.019	.892
DST	9	2.00	1.936	18	1.50	1.543	.729	.473	2.947	.098
TMAT	9	28.78	3.193	18	27.28	2.927	1.219	.234	0.000	1.000
TMBT	9	74.11	4.314	18	70.83	4.878	1.707	.100	1.016	.323
PCT	9	17.11	3.822	18	17.83	3.222	-.516	.610	.080	.779
DSubIREC	9	8.33	.500	18	8.50	.514	-.801	.431	2.083	.161
DSubDRec	9	8.33	.500	18	8.28	.958	.162	.873	1.796	.192

Table 9: Post-season comparison of means (t-statistic) and Standard Deviation (f-statistic) between Rugby Backlines versus Volleyball players

TEST	RUGBY B			VOLLEYBALL			T-TEST		LEVENE'S	
	N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	**9**	**28.33**	**10.037**	**18**	**32.33**	**6.371**	**-1.267**	**.217**	**5.045**	**.034+***
ALE	9	18.78	.441	18	18.39	.850	1.281	.212	3.738	.065
ALH	9	8.56	1.667	18	8.06	2.235	.592	.559	.181	.674
ALT	9	17.889	1.4743	18	17.250	2.4808	.708	.485	.749	.395
ALDR	9	9.44	.726	18	9.50	1.098	-.137	.892	.371	.548
DST	9	1.89	2.088	18	1.50	1.543	.549	.588	.995	.328
TMAT	9	28.00	3.000	18	26.33	3.430	1.238	.227	.862	.362
TMBT	9	73.22	4.604	18	70.39	4.692	1.488	.149	.062	.806
PCT	9	17.78	4.116	18	17.83	3.222	-.039	.970	.599	.446
DSubIREC	9	8.44	.527	18	8.50	.514	-.262	.795	.208	.652
DSubDRec	9	8.11	.782	18	8.28	.958	-.451	.656	.317	.578

NB: Marked effects significant if $p < 0.05$*,

5.3.1.4 Rugby forwards versus rugby backline players

In terms of the mean scores of pre- and post-season (Table 10 and Table 11), no significant results were found in these results.

There was significant increase in variance by the Rugby Forwards in the test of Associate Learning –Delayed recall ($p<.005$) than the Rugby Backline in the post-season.

Table 10: Pre-season comparison of means (t-statistic) and Standard Deviation (f-statistic) between rugby forwards versus rugby Backline

TEST	RUGBY F			RUGBY B			T-TEST		LEVENE'S	
	N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	9	29.89	7.356	9	27.56	8.974	.603	.555	.486	.496
ALE	9	18.44	.726	9	18.56	1.014	-.267	.793	.084	.776
ALH	9	8.33	2.598	9	7.89	2.147	.396	.698	.004	.949
ALT	9	17.500	2.8284	9	17.111	2.3819	.316	.756	.000	.989
ALDR	9	9.56	1.333	9	9.44	.882	.209	.837	.018	.896
DST	9	1.78	1.922	9	2.00	1.936	-.244	.810	.557	.466
TMAT	9	27.22	3.701	9	28.78	3.193	-.955	.354	.847	.371
TMBT	9	68.11	8.492	9	74.11	4.314	-1.890	.077	2.837	.112
PCT	9	18.33	3.122	9	17.11	3.822	.743	.468	.143	.711
DSubIREC	9	8.56	.527	9	8.33	.500	.918	.372	.703	.414
DSubDRec	9	8.11	1.269	9	8.33	.500	-.489	.632	4.904	.042

Table 11: Post-season comparison of means (t-statistic) and Standard Deviation (f-statistic) between rugby forwards versus rugby backline

TEST	RUGBY F			RUGBY B			T-TEST		LEVENE'S	
	N	Mean	SD	N	Mean	SD	t-stat	p-value	f-stat	p-value
VOCA	9	28.89	8.753	9	28.33	10.037	.125	.902	.564	.463
ALE	9	18.78	.441	9	18.78	.441	0.000	1.000	0.000	1.000
ALH	9	9.56	1.130	9	8.56	1.667	1.490	.156	1.944	.182
ALT	9	18.944	1.2105	9	17.889	1.4743	1.660	.116	.800	.384
ALDR	**9**	**9.44**	**.726**	**9**	**10.00**	**.000**	**2.294**	**.036**	**34.602**	**.000+*** **
DST	9	.78	1.481	9	1.89	2.088	-1.302	.211	1.374	.258
TMAT	9	25.11	3.822	9	28.00	3.000	-1.784	.093	.778	.391
TMBT	9	66.44	9.029	9	73.22	4.604	-2.006	.062	2.352	.145
PCT	9	18.00	3.674	9	17.78	4.116	.121	.905	.237	.633
DSubIREC	9	8.78	.441	9	8.44	.527	1.455	.165	3.114	.097
DSubDRec	9	8.44	1.014	9	8.11	.782	.781	.446	.315	.582

NB: Marked effects significant if $p < 0.001$***

5.4 Post Concussion Symptomology (PCS) checklist results

Table 12 illustrates the frequency of participants' answers on the Rhodes University Psychology symptom check list for pre and post season testing. The questionnaire was aimed at testing if participants exhibit any concussive or sub-concussive injuries. The questionnaire also includes depressive symptoms.

Table 12: Cross tabulation of PCS results

		Angry			Total
		Never	Sometimes	Often	
Rugby	Count	9	7	2	18
	%of total	25.0%	19.4%	5.6%	50.0%
Volleyball	Count	18	0	0	18
	% of total	50.0%	0.0%	0.0%	50.0%
		Insomnia			Total
		Never	Sometimes	Often	
Rugby	Count	10	8	0	18
	% of total	27.8%	22.2%	0.0%	50.0%
Volleyball	Count	8	10	0	18
	% of total	22.2%	27.8%	0.0%	50.0%

According to Table 12, all(50.0%) of the volleyball participants reported never to the question of being easily angered or hurt whereas the rugby players' responses were distributed amongst the responses, with 19.4% reporting that they are sometimes easily angered and 5.6% reporting to be easily angered. Furthermore, 22.2% of rugby participants reported that they sometimes have problems sleeping whereas 22.2% of the volleyball participants never have problems sleeping and 27.8% sometimes have problems sleeping. It may be that the rugby group report to being overall easier to anger because of the personality types required to play contact sport or it could be an indicator of CMHI. The insomnia could be linked to being more tired in the day, but this is unclear, and linked to CMHI. These results require further investigation.

5.5 Summary

The results of the study revealed no significant difference in the comparison of mean scores across all participants (t-test). However there was significant difference in variability in items such as Vocabulary ($p<.034$), Associate Learning Easy- Immediate Recall ($p<.005$), Associate Learning –Delayed recall ($p<.005$) and Digit Symbol Substitution- Immediate recall ($p<.011$). The results also show a difference in the PCS list between the rugby and volleyball participants in terms of being easily angered and insomnia.

CHAPTER SIX: DISCUSSION OF RESEARCH RESULTS

6.1 Introduction

This study aimed at investigating the effects CMHI amongst male student rugby players versus a non-contact sport control group. It was hypothesized that rugby players would show greater cognitive deficit than the volleyball players both pre and post season due to the effects of CMHI. It was also hypothesized that rugby players would exhibit poorer cognitive profiles than volleyball players both pre and post season. The research also looked at the variability on the rugby players' test scores as compared to the volleyball players. Generally, the mean results showed no significant differences however, there was a pattern of significance in terms of variability. The following discussion considers each set of significant results across groups and sub-groups and any implications arising from them.

The results will be discussed initially in terms of inter-group comparison on tests where significant differences were found in terms of the mean and variability. The implications of these results and conclusions that can be drawn will then be presented.

6.2 Rugby versus volleyball players significant results

There were no significant results were found in these results in terms of mean performances. There was a significantly increased variability in the rugby playing group on the test of Vocabulary, and Associate Learning Easy- Immediate Recall in comparison with Volleyball playing group in the post-season.Vocabulary measures comprehension and expressive abilities and Associate Learning Easy-Immediate Recall measures logical memory. These results indicate that the rugby group may show a deficit in concentration and attention.

6.3 Rugby forwards versus volleyball players significant results

There was significant increase in variance by the rugby forwards in the test of Associate Learning –Delayed recall and Digit Symbol Substitution- Immediate recall as compared to

volleyball players post-season. The Associate Learning- Delayed recall measures delayed memory and Digit Symbol substitution measures attention and mental control. These results indicate that the rugby forward group show a deficit in attention.

6.4 Rugby backline versus volleyball players significant results

Significant increase in variability was shown by the rugby backline players in the post-season in the test of Vocabulary as opposed to the volleyball playing group. Vocabulary measures comprehension and expressive abilities. The results indicate that the rugby backline group show a deficit in the degree to which they learn and comprehend verbally expressed vocabulary.

6.5Rugby forwards versus rugby backline players significant results

There was significant increase in variance by the rugby forwards in the test of Associate Learning – Delayed recall than the rugby backline in the post-season. Essentially, the rugby forwards are more at risk (because of more contact in scrums and tackling) of CMHI and associative impairment than backlineplayers therefore it is expected that the backline players performed better than the forward players in this subtest.

6.6 Implication of the results

In terms of mean performances the tests did not reveal a consistent pattern of deficits which is typically associated with the effects of Cumulative Mild Head Injuries, thus hypothesis 1 is not supported.There were significant results however, in terms of variability that suggestspotential deficits in attention among the rugby group. The results are therefore indicative of a poorer overall cognitive profile for the rugby playing group which supports hypothesis 2.

In terms of Satz's (1993) Brain Reserve Capacity Threshold Theory (BRC) and Jordan's (1997) model of inter-individual variability it is likely that individual differences that exist with regard to BRC are likely to account for individual variability and symptom onset. As educational measures are an indirect measure of BRC threshold it must be noted that both

the volleyball and rugby group share high educational levels as they are attending university. High educational levels have been found to mask symptomology in rugby players, particularly rugby playing forwards (Partington, 2001). As the variability amongst rugby players has shown a significant difference to the non-contact sports controls it is probable that this is the case in the present research. It is likely that high BRC thus acts as a protective factor for individuals which reduces impairment so individual rugby players are less likely to exhibit neurological impairment until they age as postulated by Jordan's (1997) *Shuttle* model of variability within the framework of BRC. This is seen in an increased variability of cognitive scores associated with the ageing process and declining raw scores. However, this increased variability may be displayed in individuals who suffer CMHI at an earlier age.The results of the research appear to support this notion.

CHAPTER SEVEN: RESEARCH IMPLICATIONS AND EVALUATION

7.1 Implications of the research

The study yielded no significant results in terms of significant difference of mean scores however, there was significance difference shown in variability between the rugby forwards and backline players and the rugby and volleyball group. This may be attributed to individual differences that arise as supported by Satz's (1993) Brain Reserve Capacity Threshold Theory (BRC) and Jordan's (1997) Shuttle model of inter-individual variability. Due to the participant's level of education, symptoms may be masked. Symptoms may surface with prolonged exposure to CMHI. Post Concussive Symptomology (PCS) indicating that the rugby group were more easily angered and had more difficult sleeping were also noted. This may be due to various factors and requires more research.

7.2 Research evaluation

The research is evaluated as follows.

7.2.1Methodological strengths of the study

The use a neuropsychological battery ensured reliability and validity as the tests within the battery have been standardized to measure what they are supposed to. The use of the neuropsychological battery pre-season acts a baseline performance of the players so that the researcher could ascertain if there was a difference in the post-season testing.

7.2.2Methodological weaknesses of the study

The time limit of the study may have been a hindrance to the results. The study was conducted 6 weeks pre- and post-season. It may be that due to the time period results were not minimal (as in fewer significant results) whereas over several seasons results may indicate more significance between contact playing rugby players and non-contact sport controls. The sample was also small a bigger sample would have been more useful for plotting statistical significance.

CHAPTER EIGHT: RECOMMENDATIONS

8.1 Recommendations for future research

The study yielded no significant results in terms of significant difference of mean scores however, there was significance difference shown in variability between the rugby forwards and backline players and the rugby and volleyball group. It is evident from this study that greater significance may be achieved in a larger sample size thus future research should attempt to take into account the effect sample size has on the research findings. Furthermore, because the study was undertaken in a university that is predominantly black, the results only account for the black population; however, it should be noted that the research sample was chosen for the convenience of the research and not an attempt to highlight issues on race in the rugby sport. Further research on CMHI should include participants from all participants regardless of race and ethnicity. A study into Post-Concussive Symptomology (PCS) of rugby players all ethnicities at all levels should be investigated.

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Appendix 1 - Biographical and pre assessment questionnaire

This questionnaire is entirely confidential and will be used by Ms Mokgadi to access your potential risk for neurological injury from concussive injury in rugby. Please make a cross (X) next to the correct answer (Yes or No) or answer the question in the space as required**.**

Rugby Research - Pre-assessment questionnaire

NAME:_____

FIRST LANGUAGE:_____

AGE:_____ D.O.B:_____

UNIVERSITY YEAR OR SCHOOL STD PASSED:_____SPORT: RUGBY/VOLLEYBALL

OTHER:_____

TEAM:_____

CURRENT POSITION:_____

MOST FREQUENTLY PLAYED PAST POSITION:_____

GENERAL HISTORY

Question 1

Have you ever failed a standard at school OR UNIVERSITY? Yes No

If **Yes**, when? _____For what reason?

_____

What was your overall grade in when you completed matric?

_____

How many of your subjects did you take on Standard Grade _____ Higher Grade _____

Question 2

Have you ever experienced learning difficulties or required remedial classes? Yes No

If **Yes**, what was the problem?_____

Question 3

Have you ever experienced neurological problem (for example, seizures, tremors, and stroke?).

Yes No

If **yes**, what was the problem?_____

Question 4

Have you ever suffered from a psychological/ psychiatric disorder? (For example, Depression, Anxiety, Attention Deficit or Hyperactivity)? Yes No

If **Yes**, what was the problem?_____

Question 5

Are you currently taking any form of medication? Yes No

If **Yes**, please
specify?_____

Question 6

Do you smoke? Yes No

If **Yes**, how
much?_____

Question 7

Do you drink alcohol when you are training or before or after playing sport?Yes No

If **Yes**, how
much?_____

Have you felt that you should cut down on your drinking when you are involved in playing sports? Yes No

Question 8

Do you use any other substances? Yes No

If **Yes**, specify type and frequency of
use?_____

Question 9

Have you ever sustained a head injury or concussion that was not related to sport (e.g. motor vehicle accident?)

Note to examiner: **DO NOT INCLUDE SPORTS-RELATED INJURIES HERE**.

Yes No

If **Yes**, date/s? Injury 1_____**2**_____

Injury 1

- What caused the injury/concussion?_____
- Did you lose consciousness? Yes No

If **Yes**, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

Were you hospitalized? Yes No

If Yes, for how long?_____

Injury 2

What caused the injury/ concussion?_____

Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

Were you hospitalized? Yes No

If Yes, for how long?_____

SPORTS HISTORY

Question 10

Rugby and volleyball players

- At what age did you first start playing Rugby or volleyball?
- Have you ever played any other sport for a length period of time?

(For rugby players, check whether they have participated in BOXING)

(For volleyball players, check whether they have participated in BOXING and/or RUGBY)

Yes No

If Yes, specify sport and time period played?_____

Question 11 (Rugby players only)

How many times can you remember sustaining a head injury or concussion during a game of rugby, including occasions when you were knocked or "dinged" so hard that you felt dazed, confused and or disoriented, even though you continued to play in the game? (Note to examiners: Try to ascertain the specific incidences of injury, beginning with the most recent, followed by other incidences in as consecutive an order as the subject can recall.)

If **Yes**, specific date/s? Injury 1_____

Injury 2_____

Injury 3_____

Injury 4_____

Injury 5_____

Injury 1

What caused the injury/
concussion?_____

_____

Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how
long?_____

Did you lose consciousness? Yes No

If Yes, for how
long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

Were you taken off the field? Yes No

If **Yes**, for how long?_____

Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 2

What caused the injury/ concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 3

What caused the injury/
concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how
long?_____

- Did you lose consciousness? Yes No

If Yes, for how
long?_____

- Did you lose your memory? Yes No

If **Yes**, for how
long?_____

- Were you taken off the field? Yes No

If **Yes**, for how
long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 4

What caused the injury/ concussion?_____

- Were you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

- Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

- Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 5

What caused the injury/ concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please
specify?_____

Question 12

What other injuries have you sustained while playing rugby (e.g. facial injuries, sprains, fractures)?

Please
specify._____

Question 13

Have you ever sustained a head injury or concussion while playing a sport other than rugby?

Yes No

If **Yes**, specify which sport/s and date/s

Injury1_____

Injury 2_____

Injury 1

What caused the injury/
concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field ? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 2

What caused the injury/
concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how
long?_____

- Did you lose consciousness? Yes No

If Yes, for how
long?_____

Did you lose your memory? Yes No

If **Yes**, for how
long?_____

- Were you taken off the field? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If**Yes**, please specify_____

Appendix 2: Post-Concussion Questionnaire

Please answer the questionnaire honestly indicating the degree to which each symptom affects you now tick either never, sometimes or often (Do not tick more than one answer).

NAME:_____

1	Do you suffer from headaches?	Never	Sometimes	Often
2	Do you have poor eyesight?			
3	Do you have difficulty hearing?			
4	Do you experience weakness in your Limbs?			
5	Are you Clumsy?			
6	Do you have fits or seizures			
7	Do you become dizzy?			
8	Do you become tired easily?			
9	Are you sensitive to noise?			
10	Are you ever felt that you were seeing, hearing or feeling unusual things?			
11	Are you experiencing any sexual problems?			
12	Do you have any problems with your speech?			
13	Do you stumble over your words when you speak?			
14	Do you stutter or stammer?			
15	Do you slur your words?			

16	Do you have memory difficulties?			
17	Do you have problems with attention and concentration?			
18	Does your attention wander while following a conversation or when you are watching TV or reading?			
19	Are you impatient?			
20	Are you irritable?			
21	Do you become easily angry or hurt?			
22	Do you feel sad or down in the dumps' or depressed?			
23	Do you enjoy seeing your friends and having social contact?			
24	Do you suffer from restlessness?			
25	Do you have problems sleeping?			
26	Is there a problem with your appetite?			
27	Do you feel nervous or anxious?			
28	Do you feel worried or on edge?			
29	Are you argumentative?			
30	Are you feeling short tempered?			
31	Do you become aggressive for no apparent reason?			

Appendix 3:FORM B – PART I

Date: 16 April 2013

PROJECT TITLE: Cumulative Mild Head Injury (CMHI) in contact sports: An Evaluation of the Pre and Post Season Cognitive Profiles of Rugby Players Compared with Non-Contact Sport Controls at the University of Limpopo (Turfloop Campus)

PROJECT LEADER: Ms MJ Rapetsoa

DECLARATION

I, the signatory, hereby apply for approval to conduct research described in the attached research proposal and declare that:

1. I am fully aware of the guidelines and regulations for ethical research and that I will abide by these guidelines and regulations as set out in documents (available from the Secretary of the Ethics Committee); and

2. I undertake to provide every person who participates in this research project with the relevant information in Part III. Every participant will be requested to sign Part IV.

Name of Researcher: Rapetsoa Mokgadi Johanna

Signature:……

Date: 16/04/13

For Official use by the Ethics Committee:

Approved/Not approved

Remarks:……

...……..

……

...……

Signature of Chairperson:……..

Date:……

FORM B - PART II

PROJECT TITLE: Cumulative Mild Head Injury (CMHI) in contact sports: An evaluation of the pre and post season cognitive profiles of rugby players compared with non-contact sport controls at the University of Limpopo (Turfloop campus).

PROJECT LEADER: Miss MJ Rapetsoa

Protocol for conducting research using human participants

1. Department: Psychology

2. Title of project: **Cumulative Mild Head Injury (CMHI) in contact sports: An Evaluation of the Pre and Post Season Cognitive Profiles of Rugby Players Compared with Non-Contact Sport Controls at the University of Limpopo (Turfloop Campus**)

3. Full name, surname and qualifications of project leader:
Dr Saraswathie Govender PhD Psychology Kathryn Anne Nel PhD Psychology

4. List the name(s) of all persons (Researchers and Technical Staff) involved with the project and identify their role(s) in the conduct of the experiment:

Name and qualifications or person responsible for doing the research.
Rapetsoa Mokgadi. M 2 Clinical Psychology

5. Name and address of principal researcher: Mokgadi Johanna Rapetsoa. P.O. Box 4076, Ga-Kgapane, 0838

6. Procedures to be followed: Giving out and collection of questionnaires. The administration of test battery.

7. Nature of discomfort: The material in the questionnaires may cause respondents to feel uncomfortable.

8. Description of the advantages that may be expected from the results of the study:

The study may offer a better understanding of CMHI in contact sports particularly amongst amateur players.

Signature of Project Leader:……

Date:……

PART II - INFORMATION FOR PARTICIPANTS

PROJECT TITLE:Cumulative Mild Head Injury (CMHI) in contact sports: An Evaluation of the Pre and Post Season Cognitive Profiles of Rugby Players Compared with Non-Contact Sport Controls at the University of Limpopo (Turfloop Campus)

PROJECT LEADER: Ms MJ Rapetsoa

1. You are invited to participate in the following research project:

Cumulative Mild Head Injury (CMHI) in contact sports: An Evaluation of the Pre and Post Season Cognitive Profiles of Rugby Players Compared with Non-Contact Sport Controls at the University of Limpopo (Turfloop Campus)

2. Participation in the project is completely voluntary and you are free to withdraw from the project (without providing any reasons) at any time.

3. It is possible that you might not personally experience any advantages during the project, although the knowledge that may be accumulated through the project might prove advantageous to others.

4. You are encouraged to ask any questions that you might have in connection with this project at any stage. The project leader and her/his staff will gladly answer your question. They will also discuss the project in detail with you.

5. The administration of the questionnaires and test battery may be time consuming.

6. Should you at any stage feel unhappy, uncomfortable or is concerned about the research, please contact **Ms NokoShai-Ragoboya at the University of Limpopo, Private Bag X1106, Sovenga, 0727, tel: 015 268 2401**.