The impact of repeated mild traumatic brain injuries (concussions) on the cognitive and academic functioning of early adolescent rugby union players: A controlled, longitudinal, prospective study.

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To my husband, Grant

Without you little would be possible.

Even less would be worthwhile.

To my children, Candace, Keaton and Laine

You serve as a constant source of inspiration for me.

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ABSTRACT

This study investigated, within the context of Brain Reserve Capacity (BRC) theory, whether repeated concussions resulted in residual deficits in cognitive and academic functioning of early adolescent rugby players relative to non-contact sports controls. Participants were 150 grade 7 preparatory school boys tested pre and post three winter sport seasons using neuropsychological and academic tests. Participants were matched in terms of age, sex, grade, school, language, participation in sport, socio-economic status and estimated IQ. Chi-square analysis revealed that the rugby group reported significantly more concussions than controls ($p< 0.01$). Wechsler Intelligence Scale for Children **WISC III Vocabulary Subtest VOC** scores (estimate of BRC) were significantly higher for controls versus rugby players ($p< 0.01$) (*d = 4.5)* at baseline. Correlational analysis revealed a strong positive relationship between **VOC** and **Academic Aggregate AGG** ($r = 0.52$) and **Similarities SIM** ($r=0.55$) a measure of abstract thinking. Group mean comparisons, using repeated measures ANOVA, across the battery of tests for the rugby and concussion group participants' responses showed significant time effects ($p < 0.01$) for some measures, a significant group effect for **SIM** $p = 0.03$ (*d* =3)(rugby), with controls performing better than the rugby group. Analysis of the percentages of deficits for **SIM** (non-compromised participants) revealed that at baseline 12.5 % of the non rugby group performed below the appropriate norm in contrast to 17.4% of the rugby group. Three years later the outcome was 8.3% and 28.2% respectively. When **VOC** was factored into the analysis there was a significant difference between the scores of the control group with no concussions and the rugby group with multiple concussions, over time for **AGG** $p=0.02$ (*d*= 2.1)(combined). These findings suggest that: 1. early adolescent rugby players experience more concussions than controls; 2. they may be more vulnerable in terms of BRC; 3. they may be compromised in terms of the development of abstract thinking and over time following repeated concussion there is a decline in their academic scores.

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CHAPTER 1: INTRODUCTION

1.1. Research Context

On 19 May 2006 an article in the Weekend Argus read; “Tommie Watson played rugby for the last time on Saturday. Hours later he was declared brain dead… The 18 year old sustained a head injury during the match but that might have been prevented if he had access to a new method of testing” (Editorial, 2006, p.17). Nearly eight years earlier to the day on 16 May 1998 Morne Prins died after injuring his head in a rugby tackle during a match. The article in the Eastern Province Herald referred to the incident as a “freak occurrence” (Editorial, 1998, p.6). In July 2005 a 15 year old boy presented to the Head Injury Clinic having sustained a head injury from a tackle in a rugby match in April of that year (Clinical Interview, 2005). Following the head injury he suffered a cerebral vascular accident and although he lived to tell the tale, he bears the effects of the injury and its sequelae. His admission to hospital immediately following the injury was short lived. “He was told that he just sustained a concussion but that he would be fine and was discharged. His condition deteriorated at home and hours later he returned to hospital having had a stroke” (Personal Communication with parent in Clinical Interview, 2005).

Each of the three cases referred to brings with it its own message regarding concussion management. The second case highlights ignorance at the time surrounding concussion and the consequences. The first suggests some progress in terms of knowledge about acute management, while the third illustrates that although some progress has been made it is not nearly enough as that outcome may have been prevented. Nevertheless all three case

examples demonstrate severe consequences and represent just the tip of the iceberg in the traumatic brain injury from playing rugby debate. The remainder of the iceberg, like mild traumatic brain injury (MTBI), i.e. concussion, especially in the contact sports such as Rugby Union, remains largely submerged. The reason concussion is not observed is due to the apparent lack of overt signs and symptoms. Coaches fail to notice, monitor and recognize the signs and symptoms of concussion and to remove players from the field and players fail to report symptoms for fear of being removed from the game. Few players report to hospitals and when they do, health care professionals in busy trauma units often do not take the concussion seriously because of the lack of overt signs and symptoms, as was the case of the injured 15 year old referred to above.

Whilst currently a large chunk of the research conducted in the area of sports concussion/MTBI focuses on acute effects and return to play decisions of high school, collegiate and professional players participating in contact sports (Collins, Echemendia and Lovell, 2004), research on players of contact sports such as boxing, soccer, American football, Australian Rules football (AFL), Rugby League (RL) and Rugby Union (RU) shows growing evidence to support the harmful, chronic effects of concussion/MTBI (Shuttleworth-Edwards, Border, Reid and Radloff, 2004), especially injuries incurred within a short space of time from each other (Grindel, Lovell & Collins, 2001). Given the nature of contact sports, exposure to multiple concussions is a characteristic feature; increasing the risk of the player developing long-term or permanent cognitive deficits, typically associated with diffuse brain damage (Shuttleworth-Edwards et al., 2004). The longer the exposure to the game or prior cognitive vulnerability, the greater the potential for sub-clinical effects as well as potentially devastating

consequences under conditions of increased task challenge such as school and university examinations (Shuttleworth-Edwards et al., 2004).

RU is played in over 100 countries world wide, including South Africa (Macleod, 1993). In South Africa RU commonly known as "rugby" is particularly popular at school level; commencing in some schools as early as grade 1 and as late as year 2001 the game was still compulsory in a number of schools. Cause for concern regarding the participation in rugby at school level is strengthened by the knowledge that: head injury is regarded as one of the common causes of acquired disability during childhood with mild head injury the most prevalent in 5-14 year age range (Asarnow, Satz, Light, Zaucha, Lewis & McCleary, 1995, p. 705); multiple concussions are a characteristic feature of contact sports; cognitive development is impeded more by early than late injury despite the immature brain being more plastic than the mature brain, (Nass, 2002); and the fact that the game of RU is so popular and so competitive, even at junior school level.

The first study to examine the effects of MTBI on Rugby Union players was conducted in South Africa by Shuttleworth-Jordan, Balarin and Puchert (1993). This paved the way for numerous other studies under the leadership of Shuttleworth-Jordan/Edwards. These studies focused on adults and late adolescent RU players and results showed deficits for cognitive test data for rugby players versus controls. The research also indicated that the incidence of concussion amongst schoolboys (late adolescence) may in fact be higher than adults (Seward, 1993; Border, 2000). To date, no research has included early adolescent RU players. The concepts of early and late adolescence described by Erikson (Newman & Newman, 1987) will be discussed later under the heading developmental debate. Furthermore there have been no

controlled, prospective, longitudinal studies conducted in the contact sports, including early adolescent rugby union players, to assess the impact of repeated mild traumatic brain injuries (concussions) on cognitive as well as academic functioning.

The current research was motivated firstly by the witnessing of repeated concussions sustained by a number of early adolescent RU players playing the game as a compulsory school sport; secondly by the awareness of the level of competitiveness amongst young players and their parents and coaches; thirdly the varying levels of physical maturation in early adolescence and fourthly the vulnerability of the developing brain with regard to cognitive deficits due to insult or injury. The South African research findings from studies conducted on adult and late adolescent RU players and the paucity of studies conducted on early adolescents, strengthened the motivation.

1.2. Research Questions

The questions posed by the current research are as follows:

- Do differences exist between early adolescent rugby union players and non-contact sport controls (hockey, squash, cross-country, fencing) in terms of brain reserve capacity (BRC), cognitive and academic functioning?
- Do early adolescent boys having sustained concussions show a decline in cognitive and academic functioning relative to non-concussed controls?
- Do compromised participants show cognitive and academic deficits relative to non-compromised participants?

- Do concussed compromised participants show cognitive and academic deficits relative to non-compromised participants with concussion?
- Do concussed compromised/non-compromised participants show cognitive and academic deficits relative to non-concussed compromised/non-compromised participants matched for estimated IQ?
- Do compromised/non-compromised rugby players show cognitive and academic deficits relative to compromised/non-compromised non-contact sport controls matched for estimated IQ?
- Do compromised/non-compromised rugby players with concussion matched for estimated IQ show cognitive and academic deficits relative to non-contact sport matched controls with no reported concussion over the three year period?
- Do non-compromised rugby players with two or more concussions matched for estimated IQ show cognitive and academic deficits relative to non-contact sport matched controls with no reported concussion over the three year period?

The introduction has presented the reader with a glimpse of the research context, the motivation behind the research, and the questions the research will attempt to answer. The full extent of the concerns raised and the rationale for the research will be more clearly appreciated by reviewing the literature.

CHAPTER 2: LITERATURE REVIEW

The thesis reviewed the following literature: Mild Traumatic Brain Injury (MTBI) in terms of types of head injury, classification and indicators of severity of head injury, classification and definition of MTBI (concussion), incidence of MTBI, MTBI across age groups and epidemiology of MTBI; MTBI in contact sport in terms of epidemiology, boxing, American football, soccer, AFL, RL and RU; Sequelae of MTBI in terms of neuropathology/pathophysiology and neuropsychology as well as cumulative mild head injury; BRC Theory; Rugby research in South Africa; International MTBI policies in contact sports; RU MTBI policy; Rationale for the current study and the research hypotheses.

2.1. Traumatic Brain Injury (TBI)

2.1.1. Types of TBI

Brain functioning can be seriously disturbed by physical injury to the head. Injuries are varied and can occur as a result of any of the following: Crushing; head struck by objects in motion; small missiles at high velocity; large objects with low velocity, penetrating injuries; blunt trauma; falls on feet or buttocks and rotational forces (Parker, 2001).

Crushing injuries refer to injuries sustained as a result of compression of the head by unyielding objects e.g. the use of forceps during birth (Pang, 1989). The head struck by objects in motion is more common in sports injuries such as repeated sharp blows to the head which occurs in heading the ball in the game of soccer. Any high velocity projectile resulting in a penetrating wound, such as a bullet or a piece of shrapnel, would be considered a small missile at high velocity while a

large object with high velocity would include falling or windblown objects. Penetrating injuries occur when edged weapons, for example knives or missiles, have caused the injury. The former example usually occurs with moderate force and low velocity (Parker, 2001).

Blunt trauma usually occurs when the head in motion strikes an immobile solid object. This type of injury can combine an acceleration/deceleration injury as in a motor vehicle accident where the head hits the windscreen or in a fall where the head hits the ground (Parker, 2001). Falls on the feet or the buttocks, which may occur in a falling elevator, could result in the brain impacting onto the skull when the feet or buttocks hit the ground (Parker, 2001). Rotational forces, Parker (2001) reports, can be generated by trivial accidents that do not necessarily result in contusions on the brain.

Head injuries such as those mentioned above are generally classified as either open or closed. Open involves penetrating injuries which cause specific and direct loss of neural tissue (Harris, 1998). These injuries do not necessarily cause the victim to lose consciousness and the neurological signs are usually highly specific (Kolb & Whishaw, 2003). Closed head injuries involve no penetration of the skull but rather acceleration and deceleration of the brain within the skull resulting in contusion of the brain and possible subarachnoid hemorrhage (Harris, 1998). Closed head injuries usually result from a blow to the head subjecting the brain to a number of mechanical forces. The site of damage is to a large extent dependent on the mechanical force taking place. Both the site of damage and the mechanical forces in play shall be discussed later in this chapter (see 2.2.1).

2.1.2. Classification and Indicators of Severity of TBI

A review of this aspect of the literature was deemed essential given the concerns raised in the introduction regarding dismissal of MTBI as inconsequential by not just lay persons but also professionals.

Severity classifications range from mild bumps to the head to severe blows resulting in prolonged coma. The Glasgow Coma Scale (GCS) (Jennet & Bond, 1975; Rimel, Giordani, Barth, & Jane, 1982) is a classification system that is used both internationally and locally to assess the severity of the injury for both treatment and prognostic purposes. Three response dimensions eye opening, verbal speech and motor response, are tested to assess the level of consciousness (Teasdale & Jennett, 1974). The severity in terms of this scale depends on the assessment of the depth as well as the length of altered consciousness. A GCS score ranging from 3-8 or a coma duration of more than six hours after admission is considered severe; a score between 9 and 12 and a coma of less than six hours after admission, moderate and a score of 13 and above with twenty minutes or less coma after admission, mild (Bond, 1986).

In severe injuries the duration of coma is a good predictor of outcome but not in milder forms of injury (Lezak, Howieson & Loring, 2004; Ormond Brown, 2006). The GCS is therefore not always an appropriate measure and, when used, it should always be used in conjunction with other pertinent variables at the time of assessment (Lezak et al., 2004). A single score can give an inaccurate indication of the severity of the injury as can a good score due to little or no loss of consciousness (Lezak et al., 2004). An example by Ormond Brown (2006) of the latter is an

individual given a GCS of 15/15 when admitted to a casualty ward, fully orientated following a fall. The injury was considered mild and the person was discharged. However, within hours the person returned to hospital with a GCS of 6/15 due to a haematoma. On further investigation it was discovered that the person had a base of skull fracture, raised intracranial pressure and a midline shift. "The difficulty with a GCS score of 15/15, is that it implies no long-term neuropsychological sequelae" (Ormond Brown, 2006, p. 34).

The duration of posttraumatic amnesia (PTA) is another means of measuring injury severity and is reportedly a better predictor of head injury severity (Ormond Brown, 2006). Posttraumatic amnesia is defined as the time between the actual head injury and the return of normal continuous memory. In PTA there is a dysfunction of both consciousness and memory and it can include confusion, disorientation, agitation, lack of sustained attention and confabulation (Parker, 2001). In terms of *acute* anterograde amnesia, PTA is classified as a problem of consciousness. PTA is classified as *chronic* when memory deficits remain after consciousness and the ability to remember events returns (Parker, 2001, p 153).

In terms of injury severity, a PTA duration of less than five minutes is considered very mild; from 5-60 minutes mild; 1-24 hours moderate; 1-7 days severe; 1-4 weeks very severe and more than four weeks extremely severe (Bigler, 1990a). The difficulty with assessing PTA is that patients are often fully orientated and can coherently respond to questions but at a later stage are not able to remember the occasion (Parker, 2001; Ormond Brown, 2006). Assessment of the duration of PTA as well as eliciting collateral information from caregivers should therefore be done retrospectively (Ormond Brown, 2006).

The assessment of severity referred to above is routinely accompanied by a thorough neurological examination to identify neurological anomalies that could assist with localizing brain damage (Anderson, Northam, Hendy & Wrennall, 2001). By assessing motor power, sensation and coordination of individual limbs one is given an indication of brain functioning (Anderson et al., 2001). Some clinicians disagree about the usefulness of the neurological examination especially with regard to MTBI because it is argued that it is not designed to detect diffuse brain damage but rather to diagnose neurological illness (Parker, 2001, p. 43). The use of the focal neurological examination, as one of the criteria used to establish whether a concussive brain injury has occurred, is therefore questioned (Parker, 2001).

Another form of assessment that can be used to gauge the severity of a head injury is the use of radiological information. Here again there is disagreement about the value of these tests when evaluating differing degrees of TBI. Computerized tomography (CT scan) is used to assess cerebral pathology and, where further complications are expected, use is made of magnetic resonance imaging (MRI) (Anderson et al., 2001, p. 7). The CT scan involves scanning the head with a narrow X-ray beam (Walsh, 1994). The beam is passed through the brain at many different angles, creating different images which when computed results in a three-dimensional image of the brain. This beam allows the transmission of X-ray protons in the layer to be measured. The computer processes the photon data and converts the density information to a visual image of the brain's internal structure (Walsh, 1994, p. 83). CT scans are sensitive to haematomas and intracerebral abnormalities (Parker, 2001, p. 42). They are however, not sensitive enough to detect diffuse axonal injury, particularly in the early stages and patients have recorded normal scan results in the presence of severe TBI (Ormond Brown, 2006).

MRI scans, on the other hand, are reportedly more sensitive and can detect diffuse axonal injury (Anderson et al., 2001). Mittl, Grossman, Hiehle, Hurst, Kauder, Gennarelli & Alburger (1994) show, in one of their studies, that MRI's provide evidence of diffuse axonal injury in patients with mild TBI's and normal CT scans. MRI's reportedly also have greater power for predicting outcome from TBI especially when scans are done after the acute recovery phase (Wilson, Wiedemann, Hadley, Condon, Teasdale & Brooks, 1988).

An MRI scan does not employ X-rays and no injection of contrast media is required. In MRI, gradient magnetic fields in three directions allow spatial detection of signal data as well as the formation of a two-dimensional image (Walsh, 1994, p. 88). Images of the brain using MRI are often superior to CT scans and the greater sensitivity as well as the ability to produce images in the different planes (horizontal, sagittal and coronal) makes it the preferred investigation (Walsh, 1994). The use of MRI scanning of MTBI patients is, however, limited due to the cost and the length of the examination (Lezak et al., 2004).

There are differences of opinion amongst clinicians about the usefulness of the CT scan and the MRI in detecting diffuse brain damage and the role of X-ray or MRI in detecting concussive traumas, particularly in the absence of haemorrhage (Parker, 2001, p. 42). It is argued that some intracellular damage does not show up on neuroimaging despite the cell being dysfunctional due to cytoskeletal abnormalities (Bigler, 2001a). More sensitive neuroimaging techniques than MRI reveal lesions which were not detected on MRI. Single proton emission computed tomography (SPECT) examinations of 43 patients with MTBI revealed cerebral abnormalities in 53% of them whereas the MRI revealed abnormalities in only 9% (Kant, Smith-Seemiller, Isaacs & Duffy, 1997). These findings were supported by MR spectroscopy studies (Friedman, Brooks, Jung, Hart

& Yeo, 1998; Garnett, Blamire, Corkill et al., 2000a; Garnett, Blamire, Rajagopalan et al., 2000b) and magnetization transfer imaging (Bagley et al., 1999; Sinson et al., 2001) which showed that abnormalities in mild TBI cases, which were considered normal on standard structural neuroimaging scans. There were similar findings with the use of PET (positron emission computed tomography) and SPECT (Hofman et al., 2001; Kesler et al., 2000).

The SPECT and PET examinations both require the administration of intravenous radiopharmaceuticals (Bigler & Orrison, 2004, p. 81). Amongst other things, SPECT evaluates blood brain barrier breakdown and regional cerebral blood flow/volume, while PET imaging involves detection of positron-emitting radionuclides (Bigler & Orrison, 2004). The SPECT was developed as an alternative imaging technique for routine use in clinical settings because of the operational complexity and installation cost of PET (Walsh, 1994). PET is similar to the CT in that it provides a cross-sectional image of the brain without the static nature of the CT (Walsh, 1994). It was the first post-CT development in imaging (Kolb & Whishaw, 2003, p. 161). PET provides dynamic information about cerebral functioning such as local cerebral blood flow, oxygen and glucose metabolism as well as blood volume. (Walsh, 1994, p. 85). Following the intravenous injection of radioactively labeled water, the radioactive molecules break quickly releasing particles that are detected by the PET camera (Kolb & Whishaw, 2003). Variations in the density of the flow of particles, from different locations in the brain, are reconstructed to produce and image of a section of the brain (Kolb & Whishaw, 2003).

In the SPECT examination use is made of a gamma camera to record emissions following the injection of a radiotracer into the blood stream. The tracer is taken up differently by normal and pathological tissue thereby allowing the recording of regional differences such as cerebral blood

flow (Walsh, 1994, p. 85). Some correlation between SPECT findings and other measures is suggested, particularly MRI, EEG and neuropsychological testing (Walsh, 1994, p. 85). Given the differences of opinion in this regard one needs to bear in mind the types of conditions examined when correlations have been found. Although SPECT/PET are less likely to be used in severe TBI because structural imaging techniques provide sufficient evidence of lesions, functional imaging has a role to play in more subtle injuries such as providing information about areas of the brain where cerebral metabolism may have been affected due to an injury (Nedd et al., 1993; Newton et al., 1992). PET scans have shown abnormalities in children with mild TBI and these results have been consistent with neuropsychological data (Anderson et al., 2001).

Neuroimaging is typically not performed in the mildest form of sports-related head injuries. In these cases medical decisions are made on the individual's clinical presentation (Bigler & Orrison, 2004). Neuroimaging such as CT or MRI or both may be considered for a more significant mild head injury which is longer in the duration of unconsciousness and more persistent in terms of neurocognitive symptoms. These measures, however only provide structural information which will be insensitive to pathophysiological changes characteristic of MTBI (Bigler & Orrison, 2004). Neuroimaging done acutely in sports-related injuries usually serves to rule out treatable lesions (Mattiello & Munz, 2001). Imaging done in the chronic phase is usually to assess lesions in the context of persistent symptoms and neurocognitive or behavioural deficits (Bigler & Orrison, 2004).

Functional magnetic resonance imaging (fMRI) produces images of activated regions of the brain by detecting indirect effects of neuronal processes on local blood flow, volume and oxygen saturation (Bigler & Orrison, 2004). Despite this technology being available for more than a

decade, investigations using the technology are still in their infancy (Bigler & Orrison, 2004). The value of fMRI was demonstrated in the study conducted on MTBI patients, by McAllister, Saykin, Flasman et al., (1999) where they found that the MTBI patients required greater activation of brain regions when executing working memory tasks than controls. An explanation for this difference, according to Bigler and Orrison (2004), is that the injury disrupts the neuronal network involved with working memory resulting in greater recruitment of neurons to maintain the same function. The neuropsychological measures between the two groups were, however, surprisingly the same. fMRI in terms of measuring functioning is more appropriate for children because unlike the other functional techniques PET & SPECT, it is not invasive (Anderson et al., 2001).

The electroencephalograph/gram (EEG) is the most widely used investigative technique in neurology (Walsh, 1994). This technique records the electrical activity of the brain by means of electrodes placed on the skull (Walsh, 1994). The EEG has, however, been thought to have little diagnostic value following TBI (Anderson et al., 2001). In paediatric samples, however, there was evidence indicating that a combination of EEG with CT scan, during the acute phase following TBI, can predict neurological outcome and educational success following mild to moderate injuries (Ruijs, Gabreels & Thijssen, 1994). Furthermore, it was claimed that evoked potentials appear to be helpful in determining injury severity as well as prognosis (Anderson et al., 2001). A contrasting finding, though, was that in a cohort of relatively homogenous MTBI patients the EEG was insensitive to abnormalities detected by other procedures such as MRI and neuropsycological tests (Parker, 2001).

TBI can also be classified in terms of how it relates to behavioural and neuropsychological outcomes (Jennet & Bond, 1975). This classification system is known as the Glasgow Outcomes Scale. The scales range from *good recovery* to *vegetative state*. *Good recovery* refers to persons who are socially fully functional with only minor physical or cognitive deficits; *moderate disability* refers to persons who can live independently but are restricted in one or more major activity area; *severe disability* includes conscious patients who have varying degrees of dependence on others in terms of their daily activities and the *vegetative state* refers to individuals with not apparent cortical functioning (Jennet & Bond, 1975).

In summary this section explored the ways in which traumatic brain injuries can be classified as well as the indicators of severity. Severity was rated in terms of mild, moderate and severe. The manner in which the severity was established and the injury classified was done in a variety of ways. Numerous measures were alluded to and explored and each elicited their own debate. In this regard Anderson et al. (2001) argued that a number of measures needed to be employed to determine the severity of an injury, particularly with paediatric patients. Furthermore it was argued that no single indicator proved to have sufficient predictive power in isolation (Fletcher, Ewing-Cobbs, Francis & Levin, 1995c). This discussion served to illustrate the challenges in detecting deficits as well as the importance of knowing the limitations of the various measures so that clinicians can make informed decisions when managing clients. The focus on Neuropsychological assessment was limited as this will be discussed again under 2.1.6.; 2.2.2.; 2.4 and 3.5 in the text.

2.1.3. Classification and Definition of MTBI

A number of terms are used interchangeably with mild head injury. Concussion is thought to be a convenient synonym (Wrightson, 2000) as is mild brain injury, mild traumatic brain injury (Bender, Barth & Irby, 2004), minor head injury, minor traumatic brain injury (Matser, Kessels, Lovell, 2004; Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Hinton-Bayre & Geffen, 2004), postconcussive syndrome (Bennet & Raymond, 1997) and lesser levels of brain injury (Parker, 2001). Concussion is derived from the Latin *Concussus*, meaning "to shake violently" (Echemendia & Cantu, 2004, p. 479).

MTBI is defined as a traumatically induced alteration in mental status /traumatically induced cerebral dysfunction (Kraus, McArthur, Silvermand & Jayaraman, 1996) which may or may not involve loss of consciousness (Quality Standards Subcommittee, American Academy of Neurology (AAN), 1997). The definition was endorsed by Lezak et al. (2004) who referred to it as the characteristic injury of mild TBI but they added that concussion can also occur without direct impact to the head e.g. in whiplash injuries where the head is jerked rapidly and forcefully and damage is caused to cerebral and soft tissue structures. One of the hallmarks of concussion is that without any prominent physical damage, the brain is rendered dysfunctional (Giza & Hovda, 2004, p. 45).

The presence or absence of loss of consciousness and the presence of symptoms within or outside of a 15 minute time frame determines one of three grades used to classify the severity of the injury (Maroon, Lovell, Norwig, Podell, Powell & Hartl, 2000). The AAN established guidelines for the grading of cerebral concussion (Maroon, et al., 2000; Bender et al., 2004). The guidelines

were as follows: A *Grade 1* concussion was defined as transient confusion with no loss of consciousness and symptoms which resolve in less than 15 minutes. A *Grade 2* concussion presented with transient confusion and no loss of consciousness but symptoms persisted for more than 15 minutes. A *Grade 3* concussion was defined as any loss of consciousness, either brief or prolonged (Maroon, et al., 2000). These guidelines are widely accepted as the appropriate guidelines to inform current sports concussion management (Bender et al., 2004).

Cantu also established guidelines for the grading of cerebral concussion. In terms of these guidelines a grade 1 concussion referred to no loss of consciousness and post traumatic amnesia of less than 30 minutes; grade 2 loss of consciousness of less than five minutes and post traumatic amnesia between 30 minutes and less than 24 hours; grade 3 loss of consciousness for more than five minutes and a post traumatic amnesia for more than 24 hours. Both sets of definitions are used to inform return to play decisions (Bender et al., 2004, p. 9).

For the purposes of this study the terms concussion and MTBI were used interchangeably and the AAN guidelines for grading concussion, as discussed above, were used as it was simpler for players and coaches to come to terms with.

2.1.4. Epidemiology of MTBI

In the United States, epidemiological studies indicated that one to two million new cases of MTBI were reported each year (Jennet & Frankowski, 1990; Thurman & Guerrero, 1997). Bender et al. (2004), however, argued that many more never come to the attention of health care professionals. It was further argued that because so many individuals do not present to hospitals

or health care facilities, studies often underestimate the incidence of MTBI (Bender et al., 2004) insinuating that the numbers quoted are the minimum. The suggested "underestimate" should be born in mind when considering the incidences reported on in this section. Notwithstanding the underestimation associated with post-incident reporting, an additional stumbling block to reporting MTBI relates to the sports arena where players are prone to underreporting injuries because they want to remain in the game (Bender et al., 2004, p. 6). A review of the literature conducted by Mc Allister (1994) suggested that 50 -70% of all patients hospitalized with a brain injury was attributable to mild brain injury.

Annual estimates of the incidence of concussion world-wide from a variety of causes ranged from 152-430 cases per 100 000 population (Jennet & Macmillan, 1981) with the percentage of injuries related to sport in the range of 3% to 25%. In a National Health Interview Survey (cited in Parker, 2001, p. 13) for the 12 months prior to 1991 it was noted that approximately 20% of 1.54 million brain injuries occurring in the U.S. were attributable to sport or other physical activity; an incidence of 124 per 100 000. The findings were divided between competitive sport (111 000) and recreation (105 000). One hundred thousand of those injuries were concussive (Lovell & Collins, 1998).

In terms of the Catastrophic Sports Industry Registry (C.S.I.R.) the biggest incidence of catastrophic head injury was associated with football, gymnastics, ice hockey and wrestling (Cantu, 1990b cited in Parker, 2001, p 14). Rugby football was cited as an additional sport with a high risk of head injury and the most common head injury victim in American sport was the teenage male football player (Cantu, 1990b cited in Parker, 2001, p 14).

The highest incidence of mild brain injury in the U.S. reportedly occurred between the ages of 15 and 34 (Marshall, 1989). The cumulative incidence of mild brain injury was found to be 130.8 per 100 000 representing 72% of all brain trauma and 82% of all hospitalized cases. The rate for males was 174.7 per 100 000 whereas the rate for females was 85.2 (Kraus & Arzemanian, 1989; Kraus & Nourjah, 1988). Injury rates for both sexes peaked between ages 15-19 (Kraus & Nourjah, 1988). The proportion of mild to moderate brain injuries by external forces was as follows: motor vehicle accidents 42%; falls 24%; firearm assault 14%; sport 12% and other 8% (Parker, 2001). The head injury rate in a study done in Norway was 236/100 000 with a ratio of 307 males to 164 females (Nestvold, Lundar, Blikra & Lennum, 1998). The highest incidence in the male population was for the age range 10-19. 57.6% of the accidents were as a result of traffic accidents; 4.5% occupational; 4.1% sport. The remainder was made up from home, outdoor, indoor and other accidents (Nestvold, Lundar, Blikra & Lennum, 1998).

In the South African context, a national study of the epidemiology of head injuries has never been undertaken (Ormond Brown, 2006). The best indication of this type of information was a survey done in Johannesburg between 1986 -1990. It was part of a broader project which investigated fatal and nonfatal injuries due to external causes (Ormond Brown, 2006). The sample comprised 1181 adults who were diagnosed with TBI. Injuries reported ranged from motor vehicle accidents to assault. The annual incidence of new cases in the non-fatal TBI category in the Johannesburg area was 316 per 100 000 population. This figure was 58% higher than the U.S. incident rate estimate of 200 per 100 000. Males were at greater risk with an incidence of 4.8: 1 (male to female ratio) (Ormond Brown, 2006, p. 34).

In summary, based on the various studies, the average incidence of concussive sports injuries was 49 per 100 000. The average cumulative incidence of MTBI (from any cause) of the various studies covered in this report (excluding SA studies) was 229 per 100 000. The Ormond Brown article on South African head injuries indicated a cumulative incidence of head injury of 316 per 100 000, which was a significant 38% higher than the global average. In terms of the incidence of MTBI, it was clear that the numbers reported each year were substantial despite the reported high incidence of non-reporting. The numbers related to sport, especially in football and particularly in the age range of 10 to 19 years for males are concerning. Concussion related to the various sports disciplines is discussed later in this section and of particular interest will be the South African RU findings.

2.1.5. MTBI across Age Groups

There is a great deal of debate surrounding sequelae following MTBI in children and adolescents as opposed to adults. Some authors (Asarnow et al., 1995 & Polissar, Fay, Jaffe, Liao, Martin, Shurtleff, Rivara & Winn, 1994) claim that children and adolescents have good outcomes and suffer no residual effects while others differ, reporting neurological, cognitive and behavioural consequences (Mira, Tucker & Tyler 1992). Some experts wrongly assume that children suffer less functional impairment if the brain injury occurs early in life (Hart & Faust, 1988), whilst Levin, Ewing-Cobbs and Eisenberg (1995) suggested that there is support in the TBI literature for greater deficits after insults to the brain during infancy and early childhood than in adolescence and adulthood and that deficits are worse in adults than in adolescence. In this regard Mira et al. (1992) refuted the claim that the child's brain is more plastic and recovers readily and completely. These authors argued that memory and attention difficulties persist

following TBI and result in long-term academic and /or behavioural problems if not dealt with. Furthermore, Mira et al. (1992) also argued that TBI in adolescence affects a brain that is still changing and maturing and that children experience greater effects as a result of TBI than adults. In addition they reported that the effects on younger children are greater than on older children because the ability to acquire new learning is disrupted. Mira et al. (1992) stated that even in mild injuries where there is no loss of consciousness, damage is diffuse and can be permanent. They claimed that early rapid recovery (within six months) is not indicative of good prognosis and that less obvious deficits may not be elicited in one on one testing. Furthermore, these authors suggested that individual achievement tests such as cognitive and neuropsychological tests do not give an indication of the child's ability to function in a class room because the tests are undertaken in ideal conditions (Mira et al., 1992).

TBI is the most common cause of brain damage in children and adolescents/young adults (Collins, 1990; Grady & McIntosh, 2002; Thurman, Alverson & Browne, 1999) with the peak incidence falling between the ages 15-24 years (Bennett & Raymond, 1997). It was argued that younger people are more at risk in terms of sustaining brain injuries because of risk taking behaviour, experimenting with substances, participation in contact sports as well as the belief that they are invincible (Bennett & Raymond, 1997).

It was estimated that in the USA alone over one million children sustain closed head injuries annually (Yeates, Luria, Bartkowski, Rusin, Martin & Bigler, 1999). Approximately 100 000 children below the age of 15 years were hospitalized for acute traumatic head injury (Burgess et al., 1999, cited in Parker, 2001) and approximately 10 of every 100 000 children died from head

trauma compared with 1.9 deaths in the next leading cause of death, leukemia (Parker, 2001). The most common cause of injury sustained by children was mild head injury (Ward, 1989).

The difficulty with children sustaining mild head injuries is that often the occurrence of an accident is unknown and this is particularly so when children are not adequately supervised as a brief loss of consciousness is not observed (Parker, 2001). It is sometimes difficult to assess altered states of consciousness and, retro and anterograde amnesia in children even when an injury is known (Parker, 2001).

Estimates showed that as many as 250: 100 000 children sustained a TBI in any one year. Half did not seek medical intervention, 5-10% experienced temporary or permanent neuropsychological sequelae and 5-10% suffered fatal injuries (Goldstein & Levin, 1987). In terms of head injuries reported 85% were classified as mild and 44% as having no loss of consciousness (Kraus, Fife, Cox, Ramstein & Conroy, 1986). Generally the incidence, cause and nature of paediatric TBI varied with respect to age gender and the child's psychosocial context (Anderson, et al. 2001).

One study evaluating whether age plays a role in recovery from sports-related concussion found that high school athletes (mean age 15.9 years) took longer to recover following a concussion than college athletes (mean age 19.9 years) suggesting that age-related differences exist between high school and college athletes (Field, Collins, Lovell & Maroon, 2003) . Seven days post concussion, high school athletes performed significantly worse than age-matched control participants, while college athletes displayed performances similar to matched controls three days post concussion despite having had more severe concussions. Control subjects were matched for

sport, age, history of previous concussions, diagnosed learning disabilities, grade (high school) and college board exams (college subjects) (Field et al., 2003) .

2.1.6. MTBI in Contact Sports

The occurrence of mild concussion in sports is generally due to rapid deceleration resulting from impact with a stationary or an opposing force which is translated to the brain as either a linear and/or rotational force (Bender et al., 2004). The occurrence of MTBI in a number of contact sports is explored in this section and although the major focus is on ball games, boxing is included in the discussion because it was previously a school sport at the school where the research was conducted.

2.1.6.1. Epidemiology

In a study conducted by Segalowitz and Lawson (1995) it was found that 30-37% of their sample of high school and university students had sustained a mild brain injury. Of the millions of high school students who participated in sport each year approximately 300 000 sustained mild brain injuries or concussions. In a study of high school athletes, Powell and Barber-Foss (1999) found that 5.5% of reported injuries were MTBI and of those 63.4% were due to football injuries, 10.5% due to wrestling, 6.2% as a result of girls' soccer and 5.7% as a result of boys' soccer.

2.1.6.2. Boxing

Inherently boxing is regarded as one of the most dangerous sports (Parker, 2001). The danger with boxing is most likely due to the number of bouts. Repeated concussions within a short space of time can be fatal because the brain is predisposed to vascular congestion from autoregulatory dysfunction, by these concussions (Kelly, Nichols, Filley, Lillehei, Rubenstein & Kleinschmidt-DeMasters, 1991). SPECT studies showed more cerebral perfusion deficits and psychometric performance was worse for boxers with increasing number of bouts (Kemp, Housten, Macleod & Pethybridge, 1995).

Cantu (1998b) reported that professional boxing had the highest number of deaths recorded in American sport. Heilbronner and Ravdin (2004), on the other hand, reported that the number of deaths from boxing was surprisingly low. Because the number of amateur and professional boxers world wide is not know, fatality rates for boxing are virtually unknown (Heilbronner & and Ravdin, 2004). However, an estimate of fatality rates of 0.13 deaths per 1 000 participants per year has been reported and was found to be lower than college football at 0.3 per 1 000 (Council of Scientific Affairs, 1983). Fewer deaths were reported amongst amateurs as opposed to professionals (Heilbronner & Ravdin, 2004).

In terms of acute neurological injuries the majority of amateur studies showed that permanent and irreversible dysfunction was rare (Estwanick, Boitano & Ari, 1984). Two studies conducted on professional boxers showed that of the 325 knockouts and 789 technical knockouts among 11 173 boxers only ten boxers required hospitalization. The authors claimed that rarely did the head injury result in neurological dysfunction on neurological examination (Mc Cown, 1959). Use was

not made of neuropsychological assessment in those studies. There are relatively few studies (approximately eight) with amateur boxers that examine the neuropsychological and neurobehavioural effects of blows to the head and only a few published studies have been done on professional boxers (Heilbronner & Ravdin, 2004). Of the studies conducted, although findings were often not significant, clinical neuropsychology proved to be the most sensitive measure of brain dysfunction (Heilbronner & Ravdin, 2004).

With regard to multiple blows to the head and chronic consequences, studies found a positive relationship between the number of bouts and the chances of developing neurological sequelae (Corsellis, Bruton & Freeman-Browne, 1973; Critchley, 1957; Isherwood, Mawdsley & Furgeson, 1966; Ross, Cole, Thompson & Kim, 1983). Studies in this sport group also showed that chronic brain damage is the likely result of cumulative effects of multiple subconcussive head blows (Jordan, Jahre & Hauser et al., 1992a).

2.1.6.3. American Football

Gerberich, Priest, Boen, Staub and Maxwell (1983) reported that there were approximately 200 000 incidences of concussion in high school football games each season. This translates to 20% of all high school football players sustaining at least one concussion each season. The chance of a college football player experiencing concussion was as high as 19% with the possibility of cumulative concussions as well (Lovell & Collins, 1998). Barth, Alves, Ryan, Macciocchi, Rimel, Jane and Nelson (1989) reported that of a sample of college football players surveyed in 1989, almost half had had a concussion by the time they reached college. Of all the sports-related injuries, 63% occurred in football (American) and tackling and being tackled appeared to cause

the most injuries; approximately 60% (Bender et al., 2004, p. 6). The risk of injury was greater during games than during practices; the reason for this was reportedly the motivation to win and the heightened intensity of the game. With regard to the incidence of concussion, Dick (1997) suggested that there was an increase in the frequency of concussion in the game. Although the increase could be attributed to the size and speed of athletes resulting in increased force when making contact, it was questioned whether the increase was rather due to improved reporting of incidences.

In terms of monitoring and managing the above mentioned process, Barth and his colleagues were pioneers in the field of sports neuropsychology and were the first to initiate a prospective study with footballers in the 1980's (Collins, Echemendia & Lovell, 2004). In a four year prospective study investigating the neuropsychological deficits of injured college American football players results showed measurable deficits in attention, concentration and information processing, at 24 hours post-injury, whereas recovery occurred between 5 to 10 days (Barth et al., 1989). The study involved 2350 football players and a control group of 107 individuals comprising patients and students. Neuropsychological testing took place at 24 hours, five and ten days post injury and post season. Other research findings suggested rapid resolution of symptoms following a single uncomplicated head injury (Barth et al., 1989).

Later research conducted on American college football players examined the effects of multiple mild head injuries (Collins, Grindel, Lovell, Dede, Moser, Phalin, Nogle, Wasik, Cordry, Daugherty, Sears, Nicolette, Indelicato & McKeag, 1999). These showed that players with two or more mild head injuries exhibited significant long-term deficits in speed of information processing and executive functioning. In summary, research amongst American football players

suggested that recovery is relatively rapid following an isolated uncomplicated mild head injury but following multiple mild head injuries the effects are additive (Giai-Coletti, 2002, p. 29).

2.1.6.4. Soccer

The biggest risk with soccer lies in "heading" the ball, as well as the missile like effect of a kicked ball (Parker, 2001). Further, a common cause of injury was contact of the head with other body parts of the opponent and or the ground (Matser et al., 2004). Soccer, unlike sports such as American football, is played by males and females and studies suggested comparable incidences of concussion in both sexes (Powell & Barber-Foss, 1999). As in boxing and American football, deaths due to neurological injury have occurred, albeit rare (American Academy of Paediatrics, 1998). Amongst English soccer players between 1931 and 1976 there were 26 fatal traumatic brain injuries reported of which eight were attributed to heading the ball (Hughes, 1974). In the United States 18 fatal accidents occurred during the period 1979 to 1993 as a result of players running into goalposts (DeMarco & Reeves, 1994). During the period 1980 and 1988 four deaths as a result of soccer accidents were reported amongst high school players (Mueller & Cantu, 1999)

In concussion studies conducted on soccer players in Scandinavia (Tysvaer & Lochen, 1991; Sortland & Tysvaer, 1989; Tysvaer, Storli & Bachen, 1989; Tysvaer & Storli, 1981) and the Netherlands (Matser, Kessels, Lezak & Troost, 1998; Matser, Kessels, Lezak & Troost, 1999) the findings were that blows to the head resulted in cognitive impairment. The Dutch studies specifically showed compromised functioning of visual abilities, planning, attention, verbal and visual memory. Furthermore, findings showed that soccer players who practiced and played for many hours each season sustain cognitive deficits commensurate with the nature and the

frequency of the knocks to the head (Matser et al., 2004). The players who headed the ball the most had more difficulties in terms of visual and verbal memory, visual analysis and planning as well as slowed focused attention and visual scanning (Matser et al., 2004).

2.1.6.5. Australian Rules Football (AFL)

Although this game is unique to Australia, it was originally developed by Irish settlers hence the many similarities with Gaelic football (Hinton-Bayre & Geffen, 2004). It is the most popular contact sport in the country with over 2% of the 1993 population playing the game (National Health and Medical Research Council of Australia, 1994). The game is played by two teams of 21 players with 18 players on an oval field at one time. An oval ball is moved around the field by running with it, kicking or punching it. Scoring occurs at any of four posts when the ball passes between the outer posts. Players contest the ball by violently bumping or tackling each other. They are therefore at a high risk of sustaining mild head trauma (Cremona-Meteyard & Geffen, 1994). During the game concussions occur as a result of direct player contact or the players' head striking the ground or the scoring post (Hinton-Bayre & Geffen, 2004). A retrospective study showed that of the active professional players 60% had a history of concussion (Maddocks, Saling & Dicker, 1995).

A preliminary study of 13 concussed AFL players using neuropsychological tests showed that 24 hours post injury players completed significantly fewer items on the Digit Symbol test than their preseason performance. However, five days post injury their performance was equivalent to preseason performance (Dicker & Maddocks, 1988). The difficulty with these findings was the susceptibility of the tests to practice effects (Hinton-Bayre, Geffen, Geffen, McFarland & Friis,

1999). In a follow-up study age-matched controls were used and significant differences were found between AFL players and controls on the Digit Symbol test five days post trauma. The AFL players' performance on the test was comparable to preseason functioning but the controls were significantly better. Concussed players therefore did not show the improvement that one would expect had they not been injured (Maddocks & Saling, 1996).

An earlier study by Cremona-Meteyard and Geffen (1994) investigated the acute and long term effects of concussion on professional AFL players who had sustained a mild head injury. Players were assessed using cued reaction time tasks, at two weeks and again at one year post injury. An elite group of sportsmen with no previous history of concussion were used as the control group. Results revealed no test differences between players and controls at two weeks but at one year post-injury the mild head injury group showed reduced benefit of valid cueing relative to controls. It was therefore concluded that evidence exists of the persistent impairment in the ability to direct visuo-spatial attention following mild head trauma in AFL players.

2.1.6.6. Rugby League (RL)

This game originated in England and is played in approximately 21 countries (Hinton-Bayre & Geffen, 2004); it is the fourth most popular game in Australia after AFL, soccer and RU (National Health and Medical Research Council of Australia, 1994). There are 17 players on a side with 13 on the field at any one time. The game is similar to RU, played on a rectangular field with 'H' posts, with tries, conversions and goals being scored. The games differ in terms of number of players, point scoring, change of play which occurs after six tackles, notwithstanding a penalty, and the ball is passed backwards while running forwards.

Most head injuries incurred in this game are as a result of tackling (Hinton-Bayre & Geffen, 2004). Tackling, an intrinsic part of the game, occurs when the player carrying the ball is brought to the ground by another player (http://ca.encarta.msn.com, 2007). Players are often hit on the head in a tackle or a player's head hits another player or the ground when tackled (Hinton-Bayre, 2000). Injury has also been blamed on dirty play and poor attitudes amongst players and supporters. Inadequate supervision and control due to inadequate rules or inadequate application of good rules have also been named responsible (De Villiers, 1987, p. 165).

The incidence of concussion amongst elite players in RL (8.5%) was twice the incidence for AFL (3.6%) (Seward, Orchard, Hazard & Collinson, 1993). More RL players than any other contact sports, presented to hospital with mild head trauma in the Queensland region (Epidemiological and Health Information Branch, Queensland Health, 1993).

A study was conducted by Hinton-Bayre, Geffen, Geffen, McFarland and Friis (1999) as a follow-up on their 1997 report on the sensitivity of tests of speed of information processing to impairment following a mild head injury in professional RL players. The study aimed to account for practice effects as well as to assess individual variations following a concussion. A Reliable Change Index (RCI) was used as an impairment criterion of decline to achieve the latter. The following measures comprised the neuropsychological test battery: Speed of Comprehension, Digit Symbol Substitution and Symbol Digit Modalities Test. Players were assessed pre-season and up to three times post-injury: 1-3 days, 1-2 weeks and 3-5. Matched controls were re-assessed at similar intervals towards the end of the season. Results revealed a decline in the players' performance on each test 1-3 days post-injury, but a return to baseline functioning by 1-2

weeks post-injury. None of the controls were impaired. During this study it also became apparent that using only one test to measure impairment was insufficient. Using RCI on any one of the three tests improved the tests' sensitivity to concussion. This study was limited by the small sample size, by varying amounts of baseline and post injury data across players, and by an impairment criterion also based on a small sample (Hinton-Bayre et al., 1999). Notwithstanding the limitations, the study did reveal that, when using tests sensitive to the effects of mild head trauma, residual symptoms become apparent, especially in those individuals who are exposed to multiple mild head injuries, such as in RL (Giai-Coletti, 2002).

2.1.6.7. Rugby Union (RU)

RU is another one of the rugby football games. The game has it's origins in soccer and came into existence when in 1823 at Rugby school in England a schoolboy picked up the ball. RU, although not regarded as dangerous as boxing and American football, is considered amongst one of the most dangerous sports in the world due to the body collisions (Wekesa, Asembo & Njororai, 1996). Seward, Orchard, Hazard & Collinson (1993) reported that the incidence of head and neck injuries was 37.3% for RU compared with 28% for RL and 14.4% for AFL.

Seward et al. (1993) reported that, in the game of RU, injuries to the head and neck are the most common, with concussion accounting for about 5% of these injuries. This figure is lower than the numbers quoted for concussion in the earlier studies mentioned here. For example a Stellenbosch study conducted by Roy (1974) reported that 20.5% of injuries were to the head and neck, with 10% of these players either unconscious or suffering from concussion. Results of a follow-up prospective study in Stellenbosch supported these figures, with 20.9% of all rugby injuries to the

head and neck and 13.8% of these injuries involving concussion (Van Heerden, 1976). Although the above mentioned injuries were cited as the most common, mild closed head injuries, joint dislocations and fractures were cited as the more serious injuries sustained in rugby union (Bird, Waller, Marshall, Alsop, Chalmers & Gerrard, 1998, p. 319). In the game of RU injuries are reportedly more common during the tackling phase of play followed by rucks and mauls. Rucks and mauls occur during a formalized contest for possession of the ball called scrummage, during which players assemble in a tight-knit formation and push forward together against their opponents (http://ca.encarta.msn.com, 2007). During rucks players may use only their feet to gain possession of the ball while during mauls they may use their hands (www.rfu.com, 2004). Garraway and Macleod (1995, p. 1486) found that tackling was responsible for 49% of injuries suffered during play of RU. Roy (1974), Nathan, Goedeke and Noakes (1983) and Roux, Goedeke, Visser, Van Zyl, & Noakes, (1987) reported similar findings. Results showed 49% (Roy, 1974), 47% (Nathan et al., 1983) and 55% (Roux et al., 1987) of all injuries occurred during the tackle. This was followed by the ruck (15%), lineout (12%), scrum (8%) and the maul (6%) (Garraway & Macleod, 1995).

Position of play also appeared to be linked to the degree of risk of mild head injury (Bird et al., 1998). In their study, Shuttleworth-Jordan, Balarin and Puchert (1993) noted that forward players exhibited greater impairment than backline players. This finding was associated with the nature of play in the scrum which exposes forwards to a greater potential for cumulative brain injury. Findings from a study by Ryan and McQuillan (1992, p. 73) further supported this with forwards receiving 54.1% of the injuries and backs receiving 45.9 %.

Size of players, speed of the game and level of competitiveness are associated with an increase in dangerousness (Jakoet & Noakes, 1998). Since the introduction of professionalism into RU, injury rates have risen markedly, particularly amongst adolescent players (Garraway, Lee, Hutton, Russel & Macleod, 2000).

Research conducted on 320 high school teams recorded 353 injuries during matches and 142 during practices. Incidences of injury were reportedly low in the early adolescent groups but rose in late adolescence, especially in under-19. Most injuries were to the lower limbs (37%) followed by the head and neck (29%). Concussion accounted for 12% - 22%. The authors argued that the lower percentage was due to under reporting of concussion injuries (Roux, Goedeke, Visser, Van Zyl & Noakes, 1987).

Notwithstanding the argument of under reporting, there appeared to be little consensus regarding the incidence of concussion. Nathan et al. (1983) reported the percentage of concussion during a single rugby season at a South African high school to be 21.5%. On the basis of self reports, Shuttleworth-Edwards et al. (2001a) reported an average historical incidence of 2.3 (range 0-7) concussions per rugby playing schoolboy (effectively 230%), in a survey of three South African schools' top teams, compared with an average incidence of only 0.4 (range 0-1) for an equivalent group of field hockey players. Garraway and Macleod (1995), however, reported a concussion incidence among amateur RU players of 0.62 per 1000 playing hours (effectively 62%). This incidence was double that found among major league soccer players (Green & Jordon, 1998).

Although rugby is a popular sport in a number of countries such as the UK, New Zealand, Australia and South Africa as yet only South Africa has produced research on the effects of

traumatic brain injury in this sport (Ancer, 1999). Shuttleworth-Jordon, Balarin and Puchert (1993, p. 41) were pioneers in this field with their research on concussion in university rugby players. The results of their testing revealed a pattern of impairment associated with diffuse brain damage in the concussed players compared to those in the non-contact sport control group.

This research inspired further study and a three phase research initiative was launched at Rhodes University by Shuttleworth-Jordan in collaboration with the South African Rugby Football Union (SARFU) and SA Sports Science Institute. Each phase consisted of three separate studies which were conducted using the same sample but slightly differing test batteries and different methods of data analysis. Phase one was conducted by Reid (1998), Dickinson (1998) and Ancer (1999). The sample consisted of 26 professional RU players and a control group of 21 professional cricket players. Methodological flaws in some of these studies may have lead to inconsistent findings but patterns in the observed outcomes as a whole suggested a trend of TBI related neuropsychological deficits (Ancer, 1999). Ancer (1999) suggested that these deficits were, at least in part, due to the cumulative effects of concussive and subconcussive injuries. See *2.6. South African Research* for further discussion in this regard.

In summary, section 2.1 explored TBI, the types, classification and indicators of severity as well as definitions of MTBI. The AAN definition of MTBI was identified as the definition that will inform this research. Epidemiology was discussed in general as well as findings across ages and in different sporting disciplines.

2.2. Sequelae of MTBI

A concern previously discussed in 2.1 with regard to the management of MTBI was that many MTBI patients do not present to hospitals. Those that do are often not taken seriously by health care professionals in busy trauma units because of the apparent lack of overt signs and symptoms and sufficient evidence. MTBI research showed that of an estimated five million individuals sustaining head injuries in the U.S. annually, only 200 000 are hospitalized (Thurman, Branche, & Sniezek, 1998). In terms of contact sport players, MTBI patients often do not present to hospitals because coaches fail to notice, monitor and recognize the signs and symptoms of concussion and to remove players from the field and players fail to report symptoms for fear of being removed from the game. Yet, serious sequelae can follow MTBI and, just because the individual is not hospitalized, it does not mean that the injury is not serious. There was evidence that the effects following mild brain injury can be a permanent, even if it is mild, reduction of an individual's potential prior to the injury (Bennett & Raymond, 1997). Symonds (1962) (cited in Bennett & Raymond, 1997) similarly questioned whether, despite being mild, the effects of concussion can ever be reversed. This section addresses the sequelae debate by reviewing the physiological as well as the neuropsychological sequelae of MTBI under the following headings: Neuropathology and Pathophysiology of MTBI and Neuropsychological Sequelae of MTBI.

2.2.1. Neuropathology and Pathophysiology of MTBI

Because the brain is shaken around in the skull cavity in closed head injuries the result is usually multiple injury sites as well as diffuse axonal damage (Anderson, Northam, Hendy & Wrennall, 2001). These injuries are commonly accompanied by a coma (Kolb & Whishaw, 2003) but can

occur without loss of consciousness (Lezak et al., 2004). Closed head injuries usually result from a blow to the head subjecting the brain to a number of mechanical forces. The first damage occurs as a contusion beneath the site of the blow (coup) (Walsh, 1994). The second site of damage (contre coup) occurs at a distance from the site of impact (Walsh, 1994). The pressure that produced the coup forces the brain to the opposite side of the skull (Kolb & Whishaw, 2003). The third site of damage occurs when the movement of the brain causes twisting and shearing of fibers resulting in microscopic lesions. The lesions can occur throughout the brain but mostly occur in the temporal and frontal lobes. Major fiber tracts of the brain e.g. those crossing the midline such as the corpus callosum can also be damaged because of the twisting and shearing. The fourth type of damage may be a haematoma caused by blood trapped from a haemorrhage due to the impact (Kolb & Whishaw, 2003). Cerebral oedema may occur as a fifth type of damage. This may occur as a failure of the autoregulatory mechanism of cerebral blood flow due to hypoxia, hypercapnia or obstruction to cerebral circulation. It may also occur due to increased fluid in the brain as a result of obstruction of cerebrospinal fluid flow or an accumulation of fluid within the cells (Anderson et al., 2001).

When reviewing the biophysics of injury, Gennarelli, Thibault & Graham (1998) found that when the brain is moved laterally it is most vulnerable. It is the least vulnerable with sagittal movement and is affected only intermediately when moved on the horizontal plane. They also found that the highest degree of widespread axonal damage takes place with lateral movement (Gennarelli, Thibault & Graham, 1998). These studies were conducted in a controlled environment and the authors caution that rarely will an accident have the same controlled outcome as a laboratory study (Gennarelli, Thibault & Graham, 1998). Furthermore, in an accidental brain injury, rarely does the brain move in a single plane, instead there are multiple

movement vectors which are often diametrically opposed, resulting in considerable axonal strain (Bigler, 2000).

The primary neuropathology of MTBI/concussion is diffuse axonal injury (DAI) (Lezak, 1995; Bigler, 1990a, 2001b; Gennarelli, 1986; Y.K. Liu, 1999; Sohlberg & Mateer, 2001). The term diffuse axonal injury (DAI) resulted from collaborative work by Gennarelli, Thibault, Adams, Graham, Thompson and Marcincin (1982) and Adams, Graham and Gennarelli (1985) cited in Bigler (2000). They defined DAI as "a traumatic injury that involves a prolonged traumatic coma that is not associated with mass lesions or ischemic damage and forms a continuous spectrum of increasing severity associated with increased numbers of damaged axons" (Bigler, 2000, p.5). Although this definition emphasizes traumatic coma, this is not necessarily the case. Like concussion, DAI is also graded in terms of severity from least (grade 1) to most severe (grade 3) and the mild cases do not necessarily involve coma (Bender et al., 2004). Bender et al., (2004) reported that in rare cases axonal shearing can result in haemorrhage and subsequent coma. In the light of DAI one needs to bear in mind that each patient brings their own set of circumstances and anatomy.

The force of an injury along with the individual's anatomy, physiology and genetics is unique to each injury as is the patient's metabolic response and vascular reactions. No two individuals present with the same sequelae despite being subjected to the same accident scenario (Bigler, 2000). Brain injury results in a common axonal pathology but it differs with regard to the amount, location and severity (Gennarelli, Thibault & Graham, 1998), implying the injury is viewed on a continuum with greater deficits increasingly involving more axonal damage. From a continuum perspective, brain injury includes concussion at the beginning of the continuum and

severe types of brain injuries at the end. The presence of DAI at the less severe end of the continuum has been detected in the post-mortem brain of an individual with reportedly less than 60 seconds of loss of consciousness (Blumberg et al., 1994, cited in Bigler, 2000).

DAI can occur without any impact to the head; it requires only a condition of rapid acceleration/deceleration such as takes place in whiplash injuries (Lezak, 1995) sustained in motor vehicle accidents or contact sports when players are tackled (Lishman, 2002) or in rucks and mauls (Garraway, Lee, Hutton, Russel & Macleod, 2000). In DAI damage or destruction of axons occurs as a result of acceleration/deceleration forces acting on the axonal bundles and intracranial blood vessels (Bigler, 1990a, 2001b; Gennarelli, 1986; Y.K. Liu, 1999; Sohlberg & Mateer, 2001).

Shearing forces from sudden acceleration (Peerless & Rewcastle, 1967; Gennarelli, Seggawa et al., 1982; Schneider, Kennedy et al., 1985) and deceleration (Alexander, 1995) of the head, results in axonal tearing and subsequent neural degeneration (Anderson, 1996). The degree of the damaging effects from the shearing of nerve fibres and neurons is proportional to the degree of acceleration (Peerless & Rewcastle, 1967; Gennarelli, Seggawa et al., 1982; Schneider, Kennedy et al., 1985) and deceleration of the head. The more force in terms of the sudden deceleration, the greater the injury will be (Alexander, 1995). Although the acceleration-deceleration effect is reportedly milder with mild head injury, there is evidence to suggest permanent damage in the form of microscopic lesions imposed on the brain (Gentilini, Nichelli, Schoenhuber, Bortolotti, Falasca & Merli, 1985).

It was originally thought that DAI only produced a temporary disturbance of brain function as a result of neuronal, chemical and/or neuroelectrical changes in the absence of gross structural changes (Bigler, 1990a, 2001b; Gennarelli, 1986; Y.K. Liu, 1999; Sohlberg & Mateer, 2001). It is now known that structural damage with loss of brain cells occurs in some concussions (Echemendia and Cantu, 2004). Studies showed that in minutes to days post concussive brain injury, that if brain cells are not irreversibly destroyed they remain in a vulnerable state. The cells are more sensitive to changes in cerebral blood flow, increased intracranial pressure and anoxia (Echemendia and Cantu, 2004). Any minor reduction in blood flow, that is usually well tolerated, during that first vulnerable week, produces extensive neuronal cell loss (Lee, Lifschitz et al. 1995; Lifschitz, Pinanong et al., 1995; Jenkins, Marmarou et al., 1986; Jenkins, Moszynski et al, 1989; Sutton, Hovda et al., 1994).

The sequelae of a DAI are not always evident immediately despite the fact that the effects of the pathology are immediately triggered. The pathological changes can take hours to days for full expression at an ultrastructural level. Using an electron microscope, a DAI can only be identified within an hour following an injury. Axotomy is often delayed; the onset can take 6-12 hours and may only reach full pathological change at 24-72 hours (Gennarelli, Thibault & Graham, 1998). Following an injury it is not just mechanical, stretch and compression consequences but also complex biochemical abnormalities that are immediately triggered which similarly have detrimental effects on both structure and function (Povlishock & Christman, 1995; Povlishock, 1996, cited in Bigler, 2000). Gennarelli et al. (1998) proposed four stages of axonal injury.

Stage I refers to a situation in which the axon in not torn instead it is rapidly but minimally stretched and it undergoes a biochemical alteration which may be transient. To produce a

transient disruption of a neuronal membrane which allows Na+, Ca2+ influx and Cl– and K+ efflux, a minimum of 5% strain load of the axon is required. Strain load refers to an increase in axon length from its resting length. During the transient change, the neuron's ability to propagate an action potential is blocked but neural functioning can reoccur within minutes.

Stage II is referred to as Cytoskeletal Damage which occurs when a minimum of 5-10% strain load leads to swelling and enlargement of the damaged axon which may negatively influence axoplasmic transport (Povlishock & Christman, 1995; Povlishock, 1996, cited in Bigler, 2000). The degree to which cytoskeletal changes takes place is relative to strain load i.e. lesser strain leaves the cytoarchitecture more intact while greater strain load results in greater structural damage to the neuronal architecture (Gennarelli et al. 1998).

Stage III axonal injury is referred to as secondary axotomy and occurs with strains equal to or greater than 15%. This stage encompasses both the biochemical and structural abnormalities of stages I and II and brings with it a much bigger chance of permanent damage. The injury in this stage is not self-reparative (Gennarelli et al. 1998).

Primary axotomy comprises the fourth stage in which immediate structural disruption of the axon occurs at the time of injury. The strain load is generally greater than 20% and consequently results in immediate irreversible damage (Gennarelli et al. 1998). Over and above the effects alluded to in the stages of injury various excitotoxic actions also occur (Shah, Yoon Xu & Broder, 1997, cited in Bigler, 2000).

Neurochemical changes that accompany mechanical changes to the brain following MTBI involve an increase in the demand for glucose when cerebral blood flow is reduced. Excess

glutamate is released and the action potential is inhibited by the subsequent influx of extracellular potassium. (The reason athletes only collapse when they reach the sideline is because it may take several seconds for the potassium to reach suprathreshold levels). Rapid glucose loss occurs because of the state of hyperglycolysis and this is then followed by a decrease in cerebral blood flow and hypoglycolysis which leaves the cells either vulnerable or damaged (Bender et al., 2004). The duration of decreased cerebral blood flow, even a brief interruption, can lead to ischemia and neurological and neurocognitive deficits. In PET studies of cerebral glucose metabolism conducted on children up to age 15 years notable differences between the levels of 1CMRgluc in the children compared with the adults were found (Chugani, Phelps, & Mazziotta, 1987). Giza and Hovda (2004) argued that it is reasonable to assume that during brain development diffuse injury can have persistent effects on the complex neurochemical and anatomic events occurring at that time.

Axonal degeneration has a deleterious effect on cortical arousal and consequently on cognitive performance (Gentilini, Nichelli, Schoenhuber, Bartolotti, Tonelli, Falasca & Merli, 1985). Changes as a result of the damaging effects could be cumulative (Echemendia & Cantu, 2004) and with repeated mild head injuries, a progressive, cumulative loss of tissue and of nervous function" is anticipated (Oppenheimer, 1968, p 306).

2.2.2. Neuropsychological Sequelae of MTBI

Recovery following MTBI can be uneventful but if not, sequelae can persist and it is argued that multiple concussions can present further consequences. Neuropsychological sequelae following MTBI are elicited via both subjective and objective routes. Subjective refers to information

reported by an individual or collateral, whilst objective information is observed or elicited via assessment techniques. This section addresses post concussive symptoms which can be elicited both subjectively and objectively. In terms of symptom presentation one is, however, often more reliant on the former as opposed to the later. Neuropsychological functioning, either recovery or the presence of persistent deficits, on the other hand, is largely determined via objective means using a neuropsychological assessment.

2.2.2.1. The Postconcussive Syndrome (PCS)

In contexts other than sport, symptoms following MTBI are often exaggerated. Sportsmen, on the other hand, in order to return to the game, minimize subjective complaints of physical symptoms (Hinton-Bayre & Geffen, 2004). Individuals sustaining head injuries who do report symptoms usually complain of headaches, blurred vision, dizziness, confusion, disorientation and forgetfulness shortly after the injury (Bennett & Raymond, 1997). The symptoms are generally consistent from patient to patient. What varies greatly is the severity thereof, the duration and the extent to which the individual experiences the symptoms as disabling (Fisher, 1985). The more significant the TBI (moderate or severe), the more prolonged symptoms may be, different symptoms may present and the prognosis may be worse (Jennet, 1990). Alves, Macciocchi and Barth (1993) reported contrasting findings in their study of 587 patients following uncomplicated mild TBI. In follow-up of the patients they reported a linear decrease in symptoms for a year post injury with headache, the most frequent symptom.

Other opinions are that following mild brain injury post concussive symptoms do not present immediately but from the time they do, they can take three months to resolve provided the patient

rests (Mittenberg, Zielinski & Fichera, 1993). Some studies conducted on soccer players, however, showed evidence of persistent post-concussive symptoms displayed by the players (Abreau, Templer, Schuyler, & Hutchison, 1990; Tysvaer, 1992).

A number of rugby studies focused on the post concussive syndrome and the findings generally were that there was a correlation between reported symptoms following a concussion and results on neuropsychological tests (Maroon et al., 2000). It was noted that players could remain symptomatic even after cognitive symptoms appeared to have resolved and that the converse could also occur (Maroon et al., 2000). There also appeared to be a correlation between the degree and extent of post concussive symptoms and the number of previous head injuries (Wilberger, 1993). Due to the extensive research already conducted on post concussive syndrome, little emphasis will placed on this aspect in the current research.

2.2.2.2. Objective Neuropsychological Sequelae

In 1986 Ruff, Levin and Marshall suggested that neuropsychological assessment following minor head injuries focus on attention, psychomotor speed, information processing and anterograde memory functioning. Research conducted later on college and professional RU players focused mainly on speed of information processing and memory. The areas of neuropsychological functioning repeatedly showing deficits following mild TBI were speed of information processing, immediate and delayed verbal and non-verbal memory (Shuttleworth-Edwards et al., 2004).

Prospective studies conducted in American football, AFL and RL, also found speed of information processing to be most sensitive to the effects of concussion (Cremona-Meteyard & Geffen, 1994). Mild TBI studies conducted by Binder, Rohling and Larrabee (1997) had findings consistent with the general population in terms of deficits in speed of information processing. Speed of information processing was considered the hallmark of closed head injury due to acceleration/deceleration forces (Cremona-Meteyard & Geffen, 1994). Lovell and Collins (1998) generally acknowledged that cerebral concussion affects information processing speed and memory, however, they cautioned that each individual athlete could display a different pattern of impairment.

Although a large number of studies conducted on college and adult athletes showed deficits in terms of information processing and memory, child and adolescent studies showed memory and attention deficits following mild head injuries (Anderson, Catroppa, Morse & Haritou, 1999; Satz, 2001). In studies of adolescents only, objective cognitive deficits were in areas of abstraction and reasoning, verbal memory, learning, language, motor speed and visuospatial functioning (Leathem & Body, 1997; Levin & Eisenberg, 1979).

2.2.2.3. Neuropsychological Recovery Following MTBI

Although it was reported that individuals who sustain mild brain injuries often have uneventful recoveries (Bennett & Raymond, 1997), there are studies that showed that approximately 25-30% of persons with a mild TBI three to six months post injury exhibited emotional and cognitive difficulties when reevaluated (Bennett & Raymond, 1997). An argument against the latter was that the injuries were misclassified and could have been moderate as opposed to mild (Bennett &

Raymond, 1997). Whether an injury can be considered mild when there are cognitive impairments lasting more than a few months was questioned. It was argued that if a person has been diagnosed as having a mild brain injury but difficulties persist, the difficulties could be related to preexisting problems or the injury was not in fact a mild one (Bennett & Raymond, 1997).

In a four year prospective study investigating the neuropsychological deficits of injured college American football players results showed measurable deficits in attention, concentration and information processing, at 24 hours post-injury, whereas recovery occurred between five to ten days (Barth et al., 1989). In terms of the recovery time frame, MTBI can be either acute (immediate) or chronic (long term) (Grindel et al., 2001). Acute effects, according Barth et al. (1989) referred to a resolution of cognitive sequelae up to and within three months post injury. According to Mc Crory, Bladin and Berkovic (1997) recovery from acute effects is measured as a return to pre-season functioning. Chronic effects on the other hand are reportedly the objectively observable deficits one year post injury (Winogron, Knights & Bawden, 1984). In both the acute and chronic stages, though, objective testing revealed cognitive deficits in information processing, attention and memory (Shuttleworth-Edwards et al., 2004).

In a study involving 10 universities (n=2350), Barth et al. (1989) examined the effects of mild brain injury using a test-retest design to assess college football players before and after mild brain injury. They confirmed, using neuropsychological tests that the majority of players returned to baseline performance within 10 days. At 24 hours and five days post concussion, injured players still showed neurocognitive deficits but some recovered to better than their preseason baseline, in line with controls by the 10 days (Barth et al., 1989).

Other sport related concussion studies showed that recovery on cognitive test functioning following a concussion generally leveled off by three months and consequences beyond that time frame were considered persistent (Barth et al., 1989; Hinton-Bayre et al., 1999; Maddocks & Saling, 1996; Shuttleworth-Jordan, Puchert & Balarin, 1993; Wilberger, 1993). In prospective studies in American football, Australian Football League and Rugby League performance on standard psychometric measures suggested that, on average, recovery occurred within 5-10 days of sustaining a concussion (Hinton-Bayre & Geffen, 2004).

In a Norwegian study neuropsychological tests were conducted 4-8 months after concussion on 56 children, 9-13 years of age. The participants were matched with a control group on variables of school grade, sex and academic performance. The results of the control group were superior to the experimental group on 29 of the 32 test variables. Analyses of Variance (ANOVA) revealed that the concussion variable explained most of the differences between the groups. Furthermore, as age increased the differences decreased but as the complexity of the tests increased the differences also increased. The results of this study also indicated that neuropsychological sequelae may be evident even in the presence of few subjective complaints and no perceptible lags in academic achievement (Gulbrandsen, 1984).

In terms of neurobehavioural functioning, numerous studies reported that of the individuals who sustained mild TBI two-thirds returned to previous occupational and personal functioning within the first 3 to 6 months following the accident (Binder, 1997; Gentilini, Nichelli et al., 1985; Klonoff & Lamb, 1998; Levin, Mattis, Ruff et al., 1987). Levin, Mattis et al. (1987) reported that after a minor head injury, neurobehavioural impairment generally resolved within the first 3

months while Wrightson and Gronwall (1999) reported complete recovery from mild head injury within a month or two.

In summary, bigger studies (n = 2350) and prospective studies (4 years) cited recovery in 5 – 10 days whilst others referred to resolution over three months and more. Sequelae still present after some months were dismissed as incorrect classification

2.2.2.4. Persistent Neuropsychological Deficits

In terms of persistent neuropsychological deficits, Long and Williams (1988) argued that the assumption that individuals recovered from minor head injuries without any major problems is incorrect as is the view that physiological processes are reversible. Courville (1953) cited in Blakely & Harrington (1993) argued that long term impairment was common following mild head injury. Although neurological recovery appeared to be rapid, recovery from cognitive deficits was prolonged with permanent residual deficits (Gulbrandsen, 1983; Walker and Erculei, 1969 cited in Long & Williams, 1988). Long term chronic difficulties only presented weeks or months post injury and 25-40% of individuals never fully recover. If there was some recovery it was usually complete by two years with very little additional improvement between two to seven years (Oddy, Coughlan, Tyerman & Jenkins, 1985 cited in Long & Williams, 1988). At two years post injury 50% of children remained impaired on more than 30% of the tasks given to them (Klonoff & Low, 1974 cited in Long & Williams, 1988; Klonoff, Low & Clark, 1977).

In contrast to the above, large scale, carefully controlled research conducted by Dikmen, Machamer, Winn and Temkin (1995) showed that there was little difference in terms of neuropsychological functioning at one year post head injury of mildly injured participants

compared with controls. In AFL there was also limited support for persistent deficits following concussion (Cremona-Meteyard & Geffen, 1994). McCrory, Bladen & Berkovic (1997) reported that within days of concussions, players reported no symptoms and there was no evidence of MRI and EEG abnormalities or neuropsychological impairment on neuropsychological tests.

An earlier AFL study of concussed players, however, showed impairment present at one year post injury. With regard to this study, six of the nine players in the study had had previous concussions and it was thought that the deficits at one year suggested cumulative effects of repeated concussions. But, it was also argued that the deficits may have been as a result of concussion incurred during the follow-up period, which were never reported (Cremona-Meteyard & Geffen, 1994).

In a retrospective study where 55 of 198 players had sustained two or more concussions there was no significant difference on performance on Digit Symbol between the two groups and no players had sustained a concussion 6 months prior to the study suggesting no clear evidence of cumulative effects (Maddocks, Saling, & Dicker, 1995). Although it was argued that the data was based on subjective reports and that the study was done retrospectively, a similar study conducted on soccer players revealed impaired performance (Matser et al., 1998, 1999).

Shuttleworth-Edwards et al. (2004) reported unremitting neuropsychological deficits amongst contact sports players in studies conducted on RU players. Other studies that showed similar persistent deficits were in the areas of American football (Collins et al., 1999; Wilberger, 1988), AFL (Cremona-Meteyard & Geffen, 1994) and soccer in the USA, the Netherlands and Scandinavia (Abreau, Templer, Schuyler, & Hutchison, 1990; Matser, Kessels, Jordan, Lezak &

Troost, 1998; Matser, Kessels, Lezak, Jordan & Troost, 1999; Tysvaer & Lochen, 1991, Tysvaer, 1992).

Studies conducted by Rimel, Giordani, Barth, Boll and Jane (1981) and Barth, Macciocchi, Giordani, Rimel, Jane and Boll (1983) relating to duration of cognitive deficits following mild head injuries respectively reported persistent pathology in 66% of 70 patients tested three months post a single mild head injury as well as significant cognitive impairment involving attention-concentration, complex information processing and memory in a substantial number of the same cohort of patients. These studies were unfortunately not well controlled for age and failed to include matched control groups.

In summary a number of researchers reported neuropsychological deficits amongst adult contact sports players. There were, however, also conflicting views regarding the presence of persistent neuropsychological deficits. In instances where deficits were present one year post injury, the deficits were associated with cumulative effects of repeated concussions or attributed to flaws in the study design. In children deficits were reported two years post injury on 30% of tasks given to them.

2.2.2.5. Neuropsychological Sequelae of Cumulative MTBI

In terms of cumulative MTBI, Parker (2001) reported that the rate of a subsequent head trauma following an initial head trauma is double and that in one sample alone the incidence of head injuries and accidents in the year following a head trauma was 23%. Other studies (Gerberich et al., 1983; Zemper, 1994; Guskiewicz et al., 2000) found that once a player has sustained a

cerebral concussion the chances of a second incident are three to six times greater than an athlete who has never had a concussion.

In a retrospective concussion history study of 119 RL players, (Hinton-Bayre & Geffen, 2004) 88% had a history of concussion, while 60% reported multiple concussions. There was however no significant difference in performance on tests sensitive to brain injury between players with no concussions, one concussion and two or more concussions. The arena of cumulative mild traumatic brain injury resulting in long term difficulties was described as controversial by Hinton-Bayre and Geffen (2004). They reported that to date (2004) no AFL or RL studies had found strong evidence for chronic or cumulative effects from concussion despite taking into account previous injuries, the severity and recency of the injuries. No long-term effects were reported or demonstrated on performance measures. These studies, however, lacked prospective data as well as data relating to learning disorders, genetic vulnerability or advancing age (Hinton-Bayre & Geffen, 2004).

In contrast, in a study comparing football players with baseline scores, with controls, a significant interaction between the diagnosis of learning disability and two prior concussions was noted. This finding suggested an additive effect of learning disorders and multiple concussions on lowered functioning (Collins et al., 1999a). Collins et al. (1999a) found that individuals with learning disabilities and numerous concussion performed in the brain-impairment range on tasks of attention and concentration (Trails B) and information processing speed (symbol digit modalities). They suggested an association between two or more concussions and lessening of cognitive skills which, when combined with learning disorder deficits, compromises functioning even further. Athletes in this category would have difficult achieving academically (Collins et al., 1999a).

Gronwall and Wrightson (1975) reported prolonged recovery following a repeat concussion in active players with a history of concussion. The results, however, showed no long-term impairment. In a prospective study of 100 professional American Football players 43 concussions were recorded over three years and there were only six repeat incidents noted (Collins et al., 1999a). These numbers were viewed as an underestimate by the researchers and they cited one of the reasons for inaccurate figures, players moving to other clubs. The difficulty with prospective studies is the attrition rate and in terms of monitoring cumulative effects the researchers call for more longitudinal prospective studies (Collins et al., 1999a).

With reference to neuropsychological sequelae of cumulative mild Traumatic Brain Injury, DeFord, Wilson, Rice, Clausen, Rice, Barabnova, Bullock and Hamm (2002) argued that even without overt structural damage, long-term cognitive dysfunction could be a consequence of repeated mild head injuries. Although Lovell and Collins (1998) placed emphasis on more severe consequences such as "second impact syndrome" which occurs when a patient, still symptomatic from the previous concussion, receives another blow to the head, they also cautioned against milder but significant consequences such as impairment of cognitive processes, mood and behaviour.

In a study comparing baseline scores of football players with controls, the group with no concussions recorded fewer symptoms than those who had single or multiple concussions. The group with two or more concussions performed worse on tasks of attention and concentration (Trails B) and information processing speed (Symbol Digit Modalities) (Collins et al., 1999a). Collins et al. (1999a) concluded that a history of concussion is both significantly and

independently associated with deficits of executive functioning, information processing speed as well as increased self reported symptoms, in the long-term.

The ability to process information is reduced following a concussion and with repeated concussion the severity and duration of the functional impairment is much greater (Symonds, 1962; Gronwall & Wrightson, 1974; Gronwall & Wrightson, 1975). Research conducted on American college football players to examine the effects of multiple mild head injuries (Collins et al., 1999a) showed that players with two or more mild head injuries exhibited significant long-term deficits in speed of information processing and executive functioning.

In summary, not all researchers appeared to be in agreement regarding the sequelae of cumulative MTBI. Lovell and Collins (1998) suggested that the long term and cumulative impact of concussions from contact sports needed further investigation through systematic neuropsychological evaluation of athletes and that this kind of investigation could eventually lead to the development of more sensitive evaluation strategies. Most of the current research has, however, focused on return to play decisions. The difficulty with the further investigation suggested above (i.e. the follow-up of athletes over a number of years) is the natural attrition rate in sport in general as athletes move between clubs, change sports and pursue other interests. Long term evaluation also raises ethical questions when, despite multiple injuries with significant deficits, athletes remain in the game and in a study. Nevertheless, when considering the neuropsychology of MTBI a number of factors have to be taken into consideration. The first is the individual's subjective report of PCS, strengthened by collateral information. The second more reliable factor is objective neuropsychological assessment. The third is to assess whether recovery of the first two has taken place and if not the fourth factor is to assess what the deficits are. The fifth factor is to monitor repeated injuries and the consequences and, when dealing with

children and adolescents, the sixth is practice effects and lastly one needs to consider the developmental debate.

2.3. Developmental Debate

The implications of cognitive deficits following contact sports head injuries in children should be considered within the framework of psychological and neurological development. From a psychological perspective two theories that could be relevant to the debate are Erikson's Psychosocial Theory (Erikson, 1963, 1968) and Piaget's Cognitive Developmental theory (Piaget, 1952b, 1970).

An issue in developmental psychology is the extent to which development is viewed as continuous versus discontinuous. Zimbardo (1992) suggested that some researchers are of the view that development is continuous. That is, that it occurs through the continuous process of an accumulation of qualitative changes. For example, it is believed that we become more skillful in thinking and talking in the same way that we become taller. In contrast, Zimbardo (1992) argued that other psychologists see a succession of re-organisations as development. They view behaviour as qualitatively different in different age-specific life stages. This view holds that particular aspects of development are discontinuous although development per se is continuous. These psychologists theorized about developmental stages qualitatively in different levels of development (Zimbardo, 1992).

Developmental psychologists believe that different behaviours appear at different life stages/ ages because of the operation of different underlying processes. The term stage implies: (i) an interval

of time in which observed qualitative differences occur in physical, cognitive and behavioural functioning; (ii) a progression toward an unexpected end state; (iii) that the stages occur in a sequence (a sequence of separate events); (iv) and that each stage is a building block for the next stage. All individuals progress through the same stages but at different rates (Zimbardo, 1992). Both Erikson's Psychosocial Theory and Piaget's Cognitive Developmental theory fall into the category of stage theories.

Erikson's stages in psychosocial development are as follows: Infancy, Toddlerhood, Early School age, Middle School age, Adolescence, Early Adulthood, Middle Adulthood and Late Adulthood (Newman & Newman, 1987). Newman and Newman, (1987) however added the stages: Prenatal and Very old age and they divided adolescence into Early and Late Adolescence. In terms of this theory a stage of development is a period of life that is characterized by some underlying organization or emphasis. Many different behaviors are viewed as an expression of the underlying stage structure. At each stage there are some characteristics that differentiate if from preceding and succeeding stages. These characteristics are referred to as developmental tasks and define what is considered to be healthy, normal development at each age, in a particular society. The tasks have a sequential nature, which means that success or failure in learning the tasks of one stage leads to greater chances of success or failure in learning those of later stages. Each new stage incorporates the gains made during earlier stages (Newman & Newman, 1987). According to this theory, if the formal operational task of Early Adolescence is not adequately negotiated, the appropriate gains may not be made in the next stages and concrete thinking associated with Middle School Age will continue as the dominant cognitive functioning.

Piaget's stages in cognitive development are as follows: 0-2 years is associated with sensory motor development (infancy); 2-7 years with the preoperational stage (early childhood); 7-11 years with the concrete operational stage i.e. more concrete thinking- implying that the individual cannot reason abstractly (middle childhood); 11 years and above is associated with the formal operational stage (adolescence). The formal operational stage is the final stage of cognitive growth. At this stage thinking becomes abstract (Zimbardo, 1992). For the purposes of this study the stage theories discussed above are utilized.

Non-biological development such as the development discussed above could also be explained by biological growth periods (see page 57-60). In terms of formal operational thought "it is generally agreed that the frontal lobes are hierarchically organized, with processes such as myelination progressing through a number of stages, from primary and sensory areas to association areas and finally frontal regions" (Anderson et al., 2001, p. 94). In this regard, Fuster, (1993); Hudspeth and Pribram, (1990); Kennedy et al., (1982); Klinberg et al., (1999); Staudt et al, (1993); Thatcher, (1991), (1997); Uemura and Hartman, (1978) (as cited in Anderson et al., 2001, p. 94) claimed that the final growth spurt takes place in late adolescence. In terms of injury or insult to the brain Stuss and Benson, (1987) and Walsh, (1978) (as cited in Anderson et al., 2001, p. 93) reported that one of the consequences of the resultant executive dysfunction is concrete thought processes. Whilst Walsh (1994, p. 143) referred to the executive dysfunction as a Frontal Lobe Syndrome and one of the consequences "a loss of the capacity to think in abstract terms." If one considers both biological and non-biological development in relation to time frames, and the impact of injury on functioning it would be difficult to ignore the impact of injury on development.

Related to the concept of developmental stages is the concept of a critical period. A critical period refers to a time during development when an organism is optimally ready, given an optimal environment and optimal stimuli, to acquire particular behaviours. If behaviours are not acquired during critical stages, it will be difficult to do so later on (Zimbardo, 1992). Given this concept, concussion due to sports injury is of particular concern in children not just because of the impact on psychological, especially cognitive development as discussed above but also because of continued neurological development (Giza & Hovda, 2004). The implication of cognitive deficits following a head injury is therefore different for children compared to adults because their cognitive skills are in the process of development. Following a head injury, there is the possibility of children losing what had previously been acquired as well as opportunities for further intellectual development being compromised (Lishman, 2002). It was also argued that the neural apparatus of children is more resilient to damage than that of adults and that residual deficits are different in the two groups. Generally, in children, cognitive deficits depend on the development stage reached (Lishman, 2002).

With regard to the resilience argument and a generally lower incidence of sequelae in children as opposed to adults, Lishman (2002) attributed the resilience in children to the pliability of the skull and intracranial structures in childhood which allows blows to the head to be better absorbed and raised intracranial pressure to be better accommodated (Lishman, 2002). Van De Graaff (2002), however, challenged the pliability argument by suggesting that ossification of the fontanels is completed by the time the child is twenty four months thereby rendering the skull less pliable beyond that time frame. Notwithstanding this resilience argument, Lishman (2002) conceded that during development certain functions are particularly vulnerable to development and this line of thinking follows in the discussion below.

In children the dynamics of cerebral organization change during development; functions that are crucial at one point can be supplemented at another and deficits which were not blatant previously can later come to the fore (Lishman, 2002). One way of looking at changes in children's thinking as they grow older is to look at changes taking place in the brain (Kolb & Whishaw, 2003). Brain changes can be explored by looking at the relative rate of brain growth. Growth spurts in the brain occur consistently between 3-10 months, 2-4 years, 6-8 years, 10-12 years and 14-16 years; the first four spurts coinciding with Piaget's stages of cognitive development (Kolb & Whishaw, 2003) as noted above. Despite the growth spurt ceasing at age 16, the process of brain maturation continues until approximately 18 years. Brain weight is said to increase by approximately 5-10% in each of the two years of growth spurts. When the brain grows there is not an increase in neurons, so the growth is most likely due to growth of glial cells and synapses. The synapses themselves do not add much weight to the brain but their growth goes hand in hand with increased metabolic demands resulting in neurons becoming bigger, blood vessels forming and new astrocytes being produced. An increase in the complexity of the cortex will undoubtedly generate more complex behaviours and one can intimate that significant changes in cognitive functioning will take place during the growth spurts (Kolb & Whishaw, 2003).

Just as Piaget's stages of cognitive development correlate with growth spurts, so do neuropsychological measures of cognitive development correlate with changes in the structure of the brain in the basal ganglia and cerebral cortex (Kolb & Whishaw, 2003). The brain's capacity to change is referred to as plasticity and normal development and learning constitute forms thereof (Nass, 2002). Experience has a huge impact on normal development of the brain and in

this regard sensory input is necessary for development to occur (Kolb & Whishaw, 2003). The manner in which experience impacts on brain development is through modification of existing neural circuitry by the creation of novel circuitry (Nass, 2002). In the same way normal experiences influence brain development, so do abnormal experiences change brain structure and behaviour (Kolb & Whishaw, 2003). Although the immature brain's ability to change is important for both learning and development, it should be noted that the brain is most vulnerable during that critical change period where it becomes more sensitive to injury or environmental stressors. In a review of studies, Gronwall, Wrightson and McGinn (1997) found evidence to support this claim. They established that if head injury occurs at an important developmental age the children may fail to develop a skill as quickly as non-head-injured children (Gronwall, Wrightson and McGinn (1997). Metabolic insults such as anoxia, trauma and malnutrition will therefore have less harmful effects on the mature nervous system as opposed to the immature system. The improved plasticity of the immature nervous system alluded to above contradicts it's vulnerability to insult. How well the individual will recover following an insult will mirror a balance between plasticity and vulnerability (Nass, 2002).

The mechanisms of plasticity involve normal development which include synaptogenesis and myelination; as well as regeneration; diaschisis and release from diaschisis, crowding; reorganization; redundancy and compensation (Nass, 2002). Synaptogenisis includes the formation of synaptic connections, growth of axons and dendrites and more effective synaptic interactions (Nitkin, 2000). This process reaches its peak during late infancy and decreases substantially during childhood. In this instance, increasing as well as destroying synapses is pivotal to learning. The process of myelination takes much longer than synaptogenesis and persists beyond adolescence (Nass, 2002). Myelination refers to the encasement of axons by

myelin; this process is important for normal adult functioning and gives a rough index of cerebral maturation. Areas myelinating in the earlier years control simple movements or sensory analysis whereas areas myelinating later control higher mental functioning (Kolb & Whishaw, 2003).

The central nervous system (mature or immature) does not generally regenerate. Release from diaschisis, crowding, reorganization, redundancy and compensation do however take place. Diashisis refers to "functional impairment at an anatomically connected site, remote from the area of injury" as a result of "loss of afferent input to the remote site" (Nass, 2002, p. 30). This situation is believed to be reversed over time (release from diaschisis) and it is argued that because the immature injured brain is subjected to longer follow up than the mature brain the process of release from diaschisis is partly an artifact of the duration of follow up time (Nass, 2002). Crowding according to Teuber (1974) is a form of plasticity in which there is "competition for the intact neural space with priority to and resultant sparing of Verbal IQ and language at the expense of ordinarily right hemisphere mediated functions – Performance IQ".

In terms of plasticity, reorganization refers to either saving/sparing (pre-acquisition injury) or regaining/recovery (post-acquisition injury) of skills normally performed by the area now damaged. The process of reorganization, with sparing to the intact hemisphere is generally more prominent for language functions and less so for somatosensory and motor functions (Muller, Chugani, Muzik et al., 1998a; Muller, Rothermel, Behen et al., 1998b). However, following early injury there is more sparing of memory than language functions. The extent to which reorganization takes place depends on, amongst other factors, the specific cognitive skills, age at the time of injury, time since the injury, size, location and type of tissue damage as well as the condition of the brain areas around and contralateral to the damage (Nass, 2002).Redundancy

either takes the form of equipotentiality or "extra" neural space. The former refers to the equivalent potential of cognitive functions of the hemispheres whilst the latter refers to additional neural space. This situation, however, does not mean that the function takes place in the same way or to the same extent. Compensation, another form of plasticity, refers to strategies used to perform a task differing from usual strategies for that task, following an injury (Nass, 2002). Plasticity becomes more limited, the greater the hemispheric specialization particularly in terms of mediating a specific cognitive task. It also becomes more limited with age, for specific cognitive skills. The general principle is that recovery is reportedly better after early injury as opposed to late. However, although the immature nervous system is more plastic than the mature, cognitive development will be impeded more by early than late injury as skills are already acquired in the latter making the mature brain more resilient. Although the immature nervous system can develop new neural networks, the mature system can refashion existing networks (Nass, 2002). With regard to plasticity and the immature brain, a decline in abilities following recovery from early injury has been reported and in addition it is argued that capacities of a compromised nervous system may eventually be taxed with the increasing demands that accompany ageing. Plasticity, therefore, has limits even in the immature nervous system (Nass, 2002).

In summary, the impact of experience or injury on the brain varies depending on the time of the impact as well as the stage of brain development and the region of the brain impacted upon, at that time. Following an injury modification of remaining circuits or generation of new circuits, neurons and glia may assist in functional restoration. Factors that play a role in recovery from early cortical injury are not just experience but also hormones and neurotrophic factors (Kolb & Whishaw, 2003).

In terms of TBI in children cognitive recovery can continue for up to five years following an accident, much longer than proven in adults (Klonoff, Low & Clark, 1977). The difficulty with children is that the nature and limits of recovery are not known. As the child matures, the nature of demands increased and it is only then that underlying deficits are revealed (Bowman et al., 1974 cited in Brooks, 1984). Furthermore, impaired cognitive functioning has a cumulative effect on new learning and developmental lags appear when compared with peers (Chadwick, Rutter, Brown, Shaffer & Traub, 1981). Boll (1983) suggested that psychosocial, academic and economic hardships caused by disruption in coping capacity and usual behaviour due to mild head injury had not received the acknowledgement that it should. Children with mild TBI often experienced a host of reported and unreported academic and social disturbances which were exacerbated by failure to identify the underlying cause. It was argued that even mild and transient impairment of attention, memory and information processing produced altered patterns of achievement and self confidence (Boll, 1983). When cognitive deficits persisted it hampered education, behaviour was affected and it in turn disrupted progress at school resulting in underachievement even when no intellectual loss was evident (Lishman, 2002). The late manifestation of learning and behavioural difficulties was most evident in the frontal lobe development of adolescents (Welsh & Pennington, 1988) where development could be incomplete and limited functions exacerbated by secondary degeneration, which is a common late effect of head injury (Johnson, 1992, pg. 405).

In their review, Gronwall, Wrightson and McGinn (1997) called for well controlled prospective studies, designed within a developmental framework, with long-term follow-up of children with MTBI relative to those non-head-injured controls. They suggested that one year follow up would be adequate for adolescents. Information obtained from such studies would help to answer some

of the questions posed by those people responsible for identifying, managing and treating children with head-injury related difficulties. It was also suggested that information from studies on children with MTBI of the frontal lobe might assist with the prediction of anti-social behaviour in the years following the injury (Johnson, 1992).

2.4. Neuropsychological Assessments

Research suggested that neuropsychological testing is an effective way to obtain useful data on the short-term and long-term effects of mild traumatic brain injury (Maroon et. al., 2000). Neuropsychological tests have been shown to be sensitive to even subtle changes in attention/concentration, memory, information processing, and motor speed or coordination (Lovell & Collins, 1998, pg. 10). This type of testing is valuable as it provides information on the injured individual's functional status (Lovell & Collins, 1998).

Rimel et al. (1981) provided some of the first evidence of the importance of neuropsychological testing for patients with MTBI. They found that 55% of all closed head injuries were classified as MTBI and that 59% of the patients had memory problems with 34% subsequently reporting unemployment. A more recent study by Matser, Kessels, Lezak, Jordan and Troost (1999) used neuropsychological tests to assess the impairment of 33 amateur soccer players. Results from these tests indicated impairment in cognitive functioning in memory and planning. South African studies on Rugby Union players conducted between 1993 and 2002 (Shuttleworth-Jordan, Balarin & Puchert, 1993; Reid, 1998; Dickinson, 1998; Ancer, 1999; Bold, 2000; Border, 2000; Finkelstein, 2000; Ackerman, 2000; Beilinsohn, 2001; Giai-Coletti, 2002) also indicated impairment of performance on neuropsychological test. The extent of impairment ranged from

significant to increased variability of scores across a variety of cognitive functions such as attention and concentration, verbal new learning, verbal and visual memory, working memory, speed of information processing and hand-motor dexterity at three days post injury. The performance outcomes were influenced by the time at which testing took place, deficits were greater three days post concussion as opposed to at one or two months. Nevertheless, testing still revealed that at three months the concussed group did not exhibit the same degree of practice effect as the controls suggesting that recovery was not fully complete.

From the results of studies it is apparent that neuropsychological testing provides a sensitive guide to ongoing and possibly cumulative problems after athletic MTBI (Maroon et al., 2000). Information obtained from neuropsychological tests cannot, however, be used in isolation and should in the best interest of the player be supplemented by the evaluation of the player's reports of post concussive symptoms (Maroon et al., 2000). In addition, the effectiveness of neuropsychological testing as a measure of MTBI damage and of recovery post MTBI should be reliant on the use of baseline testing.

The first study to emphasize the usefulness of baseline testing of athletes was conducted by Jeffrey Barth in the 1980's at the University of Virginia (Lovell & Collins, 1998). This study involved baseline assessments of more than 2,300 athletes prior to the beginning of the season. Repeat testing was performed if the athlete had suffered a concussion during the season and follow up testing was performed at 24 hours, 5 days and 10 days consecutively following the concussion. The baseline testing allowed researchers to pick up subtle difficulties on tests sensitive to information processing where no severe disturbances in cognition were identified (Lovell & Collins, 1998). Baseline testing helps to solve the problem of variability among study

participants. Individual players may vary greatly in terms of levels of performance on tests of memory, attention/concentration, mental processing speed, and motor speed (Maroon et al., 2000). Knowing how an individual performed on the tests prior to concussion allows the researcher to identify those deficits which are due to the effects of concussion rather than secondary unrelated factors such as test anxiety or attention deficit disorder (Lovell & Collins, 1998). Appropriate histories regarding previous concussions and their severity also provide necessary information when integrated with baseline test results (Maroon et al., 2000). Unfortunately, very few studies to date have evaluated the usefulness of neuropsychological testing in sports related injuries and even fewer have considered their usefulness in assessing children (Lovell & Collins, 1998).

Data in adolescents is, however, limited and the available data show great variability in this age group (Baker & Patel, 2000). This lack of data has made it difficult to monitor the long term effects of MTBI in adolescents based on neuropsychological tests (Baker & Patel, 2000). The process of ongoing acquisition may confound the interpretation of results. Baker and Patel (2000, pg. 318) reported that changes in test results due to learning may occur within three months. They found in their study, however, that reaction time, executive function and spatial processing were not affected by learning and could therefore be more reliable functions to measure recovery following head injury (Baker & Patel, 2000).

More recent meta-analytic studies confirm the paucity of literature in the field of neuropsychological impact of sports-related MTBI on children and early adolescents. Belanger, Curtiss, Demery, Lebowitz and Vanderploeg (2005) conducted a meta-analysis of relevant literature to determine the impact of MTBI across nine cognitive domains (Global Cognitive

Ability; Attention; Executive Functions; Fluency; Memory Acquisition; Delayed Memory; Language; Visuospatial Skill and Motor Functions). The analysis involved 39 studies which included 1463 cases of MTBI and 1191 control cases. The overall effect of MTBI on neuropsychological functioning was moderate (d = .54). In unselected or prospective studies the overall analysis revealed that by three months there was no residual neuropsychological impairment post injury (d = *.04).* The study claims that it provides compelling evidence that MTBI has little or no effect on neuropsychological functioning by three months or more. This study, however, excluded studies of sports-related injuries (participant had to have sought medical attention) because participants in this category do not necessarily seek medical attention. Children were also excluded although some of the studies in the meta-analysis included adolescents but no reference was made to the ages. Children were specifically excluded because it was felt that they may have different cognitive sequelae following MTBI.

In a meta-analysis study of the neuropsychological impact of sports-related concussion conducted by Belanger and Vanderploeg (2005) on 21 studies, they reported and overall effect size of concussion (d = 0.49) which was comparable to the effect found in the non-sports related MTBI population. Of the 21 studies 16 studies involved multiple assessments, l4 involved pre-post within-subjects comparisons and 12 had a control group. No studies conducted on children were included in this study, high school athletes were but no ages were reported. Children were excluded for the same reason as the exclusion referred to in the study above.

Frenchman, Fox and Maybery (2005) also conducted a meta-analysis of neuropsychological studies of MTBI. Their analysis is a follow up to the meta-analysis conducted by Binder, Rohling and Larrabee (1997) in which they established a relationship between MTBI and small

reductions in cognitive functioning of individuals three months post injury. Frenchman, Fox and Maybery (2005) investigated research published between 1995 and 2003. Seventeen suitable studies were used and of those only five incorporated longitudinal designs and only two of those involved four test sessions, three studies involved sportspeople whilst being followed up in prospective studies. The findings showed that measures of processing speed, attention, working memory, memory and executive functioning were the most sensitive to dysfunction in individuals following MTBI. The authors found the effects of MTBI on neuropsychological functioning to be small though and reduced to levels comparable with controls at three months. This study also excluded children.

A study of the literature that included MTBI and children was conducted by Carroll, Cassidy, Peloso, Borg; Von Holst, Holm, Paniak and Pepin (2004). They critically reviewed 120 of 428 studies and concluded that there was consistent and methodologically sound evidence that the prognosis of children following MTBI is good, with little evidence of residual cognitive, behavioural and academic deficits. In this review 30 studies pertained to children and only two were in terms of this research, considered phase II (study described in a more extensive explanatory manner using comparisons with control groups or multivariable approaches). Of the longitudinal studies, two of five years, one of three years and one of a year none had baseline scores, two had controls matched on gender and injuries, one had controls matched on classroom, age gender, teacher-rated behaviour and academic performance and one had no controls. Ages ranged from birth to fifteen.

In terms of neuropsychological assessment, MTBI and children or early adolescents it is evident that the paucity of literature presents a challenge. Another challenge is the inclusion of

neuropsychological tests as part of concussion management policies in schools and colleges because of the time- and labour-intensive nature of these tests. In addition, it may also be necessary, especially in a South African context, to take into account the fact that not all players in any given team may be first language English speakers. In such cases, the neuropsychological test battery would have to be appropriately modified (Maroon et al., 2000). The latter challenge is taken into consideration in the current research by including only English language speakers from a high socio-economic setting.

In terms of the types of tests, "paper and pencil" neuropsychological tests have been widely used, and have played an important role in the evaluation of sports related concussions (Makdissi et al., 2001). Collie, Darby and Maruff (2001) suggested however that such tests are not ideal for sporting settings, as they are designed for the detection of gross cognitive impairments at a single assessment, not for the identification of mild cognitive deficits on repeated assessment. It is also suggested that "Paper and pencil" neuropsychological procedures are limited in their usefulness due to practice effects (Maroon et al., 2000) and due to the requirement that test administration and interpretation be undertaken by trained personnel which, as mentioned above, results in this form of assessment becoming a time consuming and costly exercise (Collie & Maruff, 2003).

At present in the South African context the 'paper and pencil' tests still appear to be the most contextually appropriate form of neuropsychological testing. This can be attested by the fact the majority of even the most recent studies on the concussion of athletes in South Africa have successfully used paper and pencil tests as oppose to computerized tests to evaluate participants (Shuttleworth-Jordan, Balarin & Puchert, 1993; Reid, 1998; Dickinson, 1998; Ancer, 1999; Bold, 2000; Border, 2000; Finkelstein, 2000; Ackerman, 2000; Beilinsohn, 2001; Giai-Coletti, 2002). In their study on the additive effects of concussion in the South African Rugby Union,

Shuttleworth-Edwards et al. (2004) used updated versions of Wechsler subtests in conjunction with a demographic questionnaire and a self-reported postconcussive symptom questionnaire. The Wechsler Adult Intelligence Scale (WAIS) subtests included (i) a measure of general intellectual functioning, and (ii) neuropsychological measures within five modalities known to be sensitive to the non-specific effects of diffuse brain injury in association with closed head injury (Shuttleworth-Edwards et al., 2004).

Giai-Coletti (2002) used 'paper and pencil' based neuropsychological assessments to evaluate cumulative effects of concussion in high school athletes. The neuropsychological test battery consisted of the following tests: WAIS-III Vocabulary Subtest, WAIS-III Picture Completion Subtest, Wechsler Memory Scale (WMS), Association Learning Subtest (Immediate and Delayed Recall), South African Wechsler Adult Intelligence Scale (SAWAIS), Digit Symbol Substitution Subtest (Incidental and Delayed Recall), SAWAIS Digit Span Subtest, and Trail Making Test (Parts A and B). These tests measured the functioning across major cognitive modalities, including general intelligence, visuoperceptual tracking, verbal memory, and visual memory (Giai-Coletti, 2002).

In terms of the neuropsychological assessment of sports-related MTBI in early adolescents it is evident that the paucity of literature presents a challenge for new researchers. It would appear that studies conducted on young participants will have to be informed by the lessons learnt in the late adolescent/adult research regarding neuropsychological assessment and research design. As with the research discussed above, the current research also used measures that tested Global Cognitive Ability; Executive Functioning (Abstract thinking); Verbal and Visual Memory (Immediate and Delayed) Processing Speed in terms of Visuoperceptual tracking and Motor Functions. Domains that were not tested were Attention and Fluency. Tests used in keeping with

other researches included The Vocabulary subtest (VOC) of the Wechsler Intelligence Scale (WISC III); The Coding subtest (COD) (WISC III) including the Incidental Recall – Immediate (CI) and Delayed (CD) memory tasks; the Wechsler Memory Scale III (WMS III) Verbal Paired Associates Immediate (VPAI) and Delayed (VPAII) memory tasks and the Similarities subtest (SIM) (WISC III) (Belanger and Vanderploeg 2005; Belanger, et.al. 2005; Shuttleworth-Edwards et al., 2004; Giai-Coletti 2002; Shuttleworth-Jordan, Balarin & Puchert, 1993; Reid, 1998; Dickinson, 1998; Ancer, 1999; Bold, 2000; Border, 2000; Finkelstein, 2000; Ackerman, 2000; Beilinsohn, 2001). The Detroit Motor Speed and Precision test (DET) had not been used before and there have been minimal reports relating to Academic functioning. For further discussion relating to neuropsychological measures see 3.5.

2.5. Brain Reserve Capacity (BRC)

Despite research, such as the studies discussed above, supporting the existence of residual cognitive deficits, some researchers disregard the long term effects of mild head trauma citing "no robust evidence of permanent traumatic brain dysfunction" (Binder, 1997, p.443). Shuttleworth-Edwards et al., (2004), however, argued that statements such as these dampen health concerns around mild head injury leaving an impression that recovery has occurred, whilst Satz (1993) suggested that one cannot disregard the long term effects of MTBI. Satz's comment is, no doubt, framed within his preliminary theoretical framework of a threshold theory for acquired brain injury in which he addressed the concept of Brain Reserve Capacity (BRC) (1993, p. 273).

The concept of reserve emerged "following repeated observations that, across individuals , there is not a direct relationship between the severity of the factor that disrupts performance (such as degree of brain pathology or brain damage) and the degree of disruption in performance" (Stern, 2003, p.589). Stern (2003) argues that the natural variability across individuals in cognitive reserve (CR) could be translated into varying degrees of susceptibility to factors that impact on performance. Alternatively, he argues that there could be individual differences in how people compensate once pathology disrupts the brain networks that underlie performance. The latter parallels the plasticity debate (discussed on pages 58-62) and focuses on compensation as opposed to a threshold. Stern (2003) proposes two modules that encompass the two arguments reported above. The first is the threshold model, described as passive, which incorporates his first argument and the second is a more active model which incorporates his second argument (Stern, 2002 & 2003). The threshold model is associated with Satz's (1993) BRC theory.

The concept of BRC is linked to a threshold factor which is in existence prior to the presentation of symptoms resulting from dysfunction in the central nervous system. Individual differences exist in terms of BRC that account for variable instances of protection from or vulnerability to symptom onset (Satz, 1993). In terms of this theory IQ is equated with BRC levels and it suggests that an individual with a low IQ will have a lower level of BRC. If one starts with a lower BRC one's threshold in terms of protective factors will be lower and the threshold will also be reached easier than an individual with a higher IQ. This was demonstrated through studies which indicated that premorbid intellectual functioning and/or development of education may serve as protective or vulnerability factors in the development of functional morbidity associated with Alzheimer's dementia or with HIV infection (Satz, 1993). Schmand, Smit, Geerlings and Lindeboom (1997) argued that BRC was better reflected by intelligence than by education.

Results of their study on the effects of intelligence and education on the development of dementia showed that low pre-morbid intelligence is an important risk factor for cognitive decline and dementia. Giai-Coletti (2002) suggested that this principle could be extended to the morbidity associated with cumulative mild head injury.

Satz (2001) argues that the acquisition of cognitive skills in different rates of development, will at the time of a head injury, be vulnerable to negative effects due to a future lowered critical threshold for impairment. An assumption associated with this theory of BRC is that general intelligence and educational level are indirect, although imprecise, indexes of cognitive reserve (Satz, 1993). There is also an aggregation effect of lowered brain reserve capacity increasing vulnerability to functional impairment because of the combination of premorbid vulnerability factors and either single or multiple episodes of neurological damage (Satz, 2001). Shuttleworth-Jordan (1999) used the BRC theory to demonstrate how null effects after a MTBI may be misleading as a sign of full recovery. In order to provide evidence of MTBI in the face of null effects Shuttleworth-Jordan (1999) introduced the concept of variability of results used as an indication of the presence of cognitive dysfunction. Variability invalidated the "null" indications of average effects (Shuttleworth-Jordan, 1999).

Shuttleworth-Edwards et al. (2004) argued that the BRC theory had implications for research on the effects of TBI: acute, persistent and cumulative. They suggested that differences in IQ and education level were of particular relevance in South Africa where the range of level of education could vary significantly among players in a rugby team. Lack of homogeneity in the sample made it essential that differences in individual outcomes were accounted for (Shuttleworth-Edwards et al. 2004). Shuttleworth-Edwards et al. (2004) suggested that the differences in individual

outcomes could be accounted for through either an examination of standard deviations and distribution scores between groups or analysis of the percentage of deficit across measures for the research sample group compared to the percentage of deficit of the control group. Shuttleworth-Edwards et al. (2004) further argued that the latter method could have more immediate clinical relevance than the statistical comparison of group effects. Both methods were used in the three phase studies in South Africa on MTBI in rugby. These different methods of analysis could account for some of the inconsistencies in the finding of these studies.

In summary, although Satz (1993, p. 275) argued that the concept of BRC could only be "operationally defined (and measured) in terms of overall brain size or in terms of specific functional relations" as opposed to it's relation to adaptive behaviour, he alluded to indirect measures of the psychosocial factors intelligence and education and their association "with adaptive (protective) as well as maladaptive (vulnerability) outcomes" (Satz, 1993, p. 281). The South African Rugby Union research (Shuttleworth Edwards et al., 2004) subsequently contributed to the body of literature relating to psychosocial factors. The current study investigates, within the context of (BRC) theory, whether repeated concussions result in residual deficits in the cognitive and academic functioning of early adolescent rugby players relative to non-contact sports controls. In terms of protective and vulnerability factors, attention is given to participants that are compromised and those that are not, as well as the role of VOC as a baseline measure.

2.6. Rugby Union Research in South Africa

Although it is acknowledged that a discussion of South African research is not relevant within the context of an international audience, this section comprising previous RU research is included in the literature review because it acted as a stepping stone for the current research. The seed for the current research was planted by the limitations, claims and recommendations made following the research projects discussed below. Furthermore, the approach to the data analysis of the current study was influenced by the varying outcomes obtained from the different types of analyses used across the different studies.

A prospective study by Shuttleworth-Jordan et al. (1993) appears to be the first study in South Africa to examine the effects of mild head injury in RU players. This study compared 60 university RU players to non-contact sport controls on measures of hand dexterity (Denkla Finger Tapping and Perdue Pegboard), short-term verbal memory (Digit Span Forwards), verbal new learning (Digit Supraspan Test) and working memory (Trail Making Test Parts A & B and Digit Span Backwards). Two levels of analysis were performed: (i) pre- and post-season differences between non-head injured players and matched controls, and (ii) test differences between players with a reported mild head injury sustained during the season and matched controls at pre-season, five days, two months and three months post-season Giai-Coletti's (2002).

Results of the former analysis indicated deficits in the scores of non-head injured rugby players compared to the control group in verbal new learning, working memory and hand motor dexterity (Shuttleworth-Jordon et. al., 1993). It was also noted that the rugby players showed less capacity than the controls for practice effects between pre- and post-season testing. Positional variation

was also reported with forwards scoring lower on the tests than backs. Relative to controls, those rugby players that reported sustaining mild head injury displayed significant dysfunction in the areas of new verbal learning, working memory, attention and hand motor dexterity at three days post-injury (Shuttleworth-Jordan et al., 1993). Considerable recovery was evident at one month, two month and again at three months post-injury. However, the mild head injury group did not demonstrate a practice effect to the same degree as the controls on Digits Backwards, Digit Difference, Digit Supraspan and Finger Tapping. This indicates that full recovery had not yet been reached. The Shuttleworth-Jordan et al. (1993) research acted as the spring-board for further research in this area in South Africa.

The further concussion research in South Africa following on from the Shuttleworth-Jordan's (1993) study focused on top adult and late adolescent RU rugby players, tested individually, with an emphasis on persistent rather than acute or sub-acute effects (Shuttleworth-Edwards et al., 2004, p.3). Three phases of the research on the national rugby squad, under 21 university teams and top senior schoolboy teams (n=26; n=19; n=47) versus cricket and field hockey equivalents (n=21; n=21; n=34) were completed. The data from these studies were analyzed in 3 ways namely individual analysis of the percentage of deficits, comparisons of group means and comparisons of group means with existing norms (Table 2.1). The research conducted by Ancer (1999), Dickinson (1998) and Reid (1998) comprised the first phase of the study and compared professional rugby and cricket players. The rugby group was assessed pre-season whereas the cricket group was assessed post-season. The second phase of the study conducted by Bold (2000), Border (2000) and Finkelstein (2000) incorporated the rugby data of the first phase plus additional data from an U21 rugby group. The control group comprised hockey players and both groups were tested preseason. In the third phase Ackerman (2000), Beilinsohn (2001) and Giai-

Coletti (2002) compared high school rugby and hockey groups and both groups in each study were tested pre-season. Across the high school studies no significant differences between the rugby and control groups were reported but Ackerman (2000) and Giai-Coletti (2002) reported tentative indications of difference. In each phase the data were analyzed in three different ways (Table 2.1). Across the phases Reid (1998), Bold (2000) and Ackerman (2000) compared group means with existing norms and only Bold's (2000) data indicated significance between the rugby and control group. Dickinson (1998), Border (2000) and Beilinsohn (2001) analyzed the data in terms of percentage of deficits and Border (2000) and Dickinson (1998) reported significant deficits for rugby players. Ancer (1999), Finkelstein (2000) and Giai-Coletti (2002) used group mean comparisons and only Finkelstein reported significant differences between the groups.

Table 2.1 Summary of South African Research

Date	Author/s	Type of contact sport	Type of analysis	Results
1993	Shuttleworth-Jordon, Balarin and Puchert	University RU players (n=60) Non-contact sport controls (n=25)	Group means	Pre- and post- season comparison of non-concussed rugby players and controls revealed a pattern of impairment associated with diffuse brain damage. Relative to the controls, the five players out of 60 who did sustain a mild head injury during the season showed significant impairment on their performance on tests of attention, verbal new learning, working memory and hand-motor dexterity at three days post injury. At one month however, substantial recovery had occurred with further recovery at two months. It was noted that at three months that the concussed group did not exhibit the same degree of practice effect as the controls suggesting that recovery

				was not fully complete.
1998	Reid (phase 1)	Professional RU players (Springboks) (n=26); Professional cricket players as control group (n=21)	Group means and standard deviations to existing norms	No significant differences in performance on tests sensitive to diffuse brain damage between the rugby and cricket players relative to norms were found. An increased variability in the rugby playing group relative to the control group on the tasks sensitive to diffuse brain damage was noted however.
1998	Dickinson (phase 1)	Professional RU players (Springboks) n=26; Professional cricket players as control group n=21	Percentage of deficits	Significant impairment indicated by the performance of the rugby players on tests of speed information processing, reduced mental flexibility, attention and concentration, sustained attention, verbal and visual memory and new learning. Forwards performed significantly worse on tests sensitive to deficits associated with mild head injury compared to backs.
1999	Ancer (phase 1)	Professional RU players (Springboks) n=26; Professional cricket players as control group n=21	Group means comparison	No significant evidence to support the presence of brain damage effects in the rugby group. However, significantly increased variability scores for the rugby players compared with cricket players on tests particularly sensitive to cognitive deficit associated with mild head injury. Forwards performed significantly worse than backs.
2000	Bold (phase 2)	Professional RU (Springboks) n=26; U21 rugby players n=19; Non-contact sport control of national hockey players n=21	Group means and standard deviations to existing norms	Significant difference in the direction of poorer performance relative to norms for the total rugby group and for the forwards group versus controls on tests sensitive to diffuse brain damage.
2000	Border (phase 2)	Professional RU (Springboks) n=26; U21 rugby players n=19; Non-contact sport	Percentage of deficits	Significantly higher impairment indicated by the performance of rugby players compared to hockey (non-contact sport) players on tests sensitive to

		control of national hockey players n=21		diffuse brain damage. Also significantly higher impairment indicated by performance of forwards compared to backs. The most sensitive test was the Digit Symbol Substitution Test.
2000	Finkelstein (phase 2)	Professional RU (Springboks) n=26; U21 rugby players n=19; Non-contact sport control of national hockey players (n=21)	Group means comparison	Significantly poorer performance across all rugby groups relative to the performance of the controls on tests highly sensitive to diffuse brain damage. Forwards performed significantly worse on than backs for both groups (springboks and U21).
2000	Ackerman (phase 3)	Top level high school rugby players (n=47); Non-contact sport control of top level high school hockey players (n=34)	Group means and standard deviations correlation analysis	Tentative indication of deleterious effects of cumulative concussion in rugby group i)Total rugby group versus total hockey comparison provided tentative indications of initial stages of diffuse damage associated with mild head injury in the rugby group ii) backs versus forwards comparison provided some evidence of for impairment of verbal learning and memory in the rugby forward group number of head injuries and cognitive test performance revealed no significant relationship between the two.
2001	Beilinsohn (phase 3)	Top level high school rugby players (n=47); Non-contact sport control of top level high school hockey players (n=34)	Percentage of deficits	No substantial evidence of a higher level of cognitive/neuropsychological impairment in i) rugby players and hockey players; ii) rugby forward and rugby backline players iii) rugby forward and hockey players and, iv)rugby backline and hockey players.
2002	Giai-Coletti (phase 3)	Top level high school rugby players (n=79); Non-contact sport	Group means	Overall results provide only tentative support for hypothesis. In a comparison between:

		control of top level high school hockey players (n=58)		i) rugby players and hockey players, performance by rugby players revealed a significant impairment on two tests of visual tracking (in another visual tracking test there was a tendency by the rugby players to be slower than the hockey players but this difference was not significant) and, ii) rugby forward and rugby backline players, one assessment of memory showed significantly impaired performance by the backline players while another assessment of learning revealed significantly poorer performance by forward players

In terms of the percentage of deficit analysis, directed across the three phases, there was a higher percentage of deficits for cognitive test data for rugby players versus controls (national and U21), and forwards versus backs. At school level only one result indicated deficits for rugby versus controls and there were no significant differences between forwards and backs (Shuttleworth-Edwards et al., 2004). More specifically, results of the first phase revealed poorer performance of forwards in paper and pencil tests sensitive to the effects of diffuse damage than backs (Ancer, 1999 Dickinson, 1998 & Reid, 1998). This result was also evident in the second phase study, however in this phase of the study Bold (2000), Border (2000) and Finkelstein (2000) found that the rugby group performed poorer than the control group on tests sensitive to the effects of diffuse brain damage. In the third phase, as with the professional players, the high school results provided less evidence for the presence of cognitive deficits in rugby players versus controls (Ackerman, 2000; Beilinsohn, 2001; Giai-Coletti, 2002). The absence of significant findings in terms of cognitive deficits as measured by percentage of deficits in professional rugby players versus controls was attributed to the small sample size (Ancer, 1999; Dickinson, 1998; Reid, 1998). The absence in schoolboy rugby, Shuttleworth-Edwards et al. (2004) argued, suggested

that the effects of mild head injury are additive, explaining why older players show more deficits than younger players. Adding to this suggestion Shuttleworth-Edwards et al. (2004) concluded that the apparent absence of deficits at school level requires further investigation.

Giai-Coletti's (2002) group mean comparison results, not included in the abovementioned Shuttleworth-Edwards et al. (2004) analysis, however, provided some support in terms of senior schoolboy rugby players (n=79) being more susceptible to the effects of cumulative concussions than non-contact controls (n=58). The support, she argued, was strengthened by the results being obtained from the Digit Symbol Substitution Subtest, a test sensitive to diffuse brain damage. This researcher, however, found no significant difference between rugby forwards and backs. Shuttleworth-Edwards et al. (2004) suggested that this finding may be due to positional stances being less entrenched at school level than older level players. In general, limitations of the abovementioned research studies include: sample size, the cross sectional nature of the studies as opposed to longitudinal/prospective, exclusion of early adolescents and players and controls from lower level teams as well as academic results. More specifically, with the exception of the original Shuttleworth-Jordan et al. (1993) study, all the other studies only tested participants once. Furthermore, the studies did not control for age, previous and no rugby participation. Given the South African school context a large number of controls may well have previously played rugby. In one study research participants and controls were tested at different times in different settings.

Not only have the above mentioned researchers, suggested the use of larger samples, the need for further investigation at school level to monitor the additive effects of mild head injuries on cognition as well as the impact of different positions on cognitive performance of players,

recommendations were also made for longitudinal/prospective studies to investigate the long term effects of repeated concussions (Ackerman, 2000; Border, 2000; Beilinsohn 2001; Giai-Coletti's 2002).

Despite their recommendations, the focus of other newer South African research has shifted to acute effects and return to play decisions as well as the use of computerized neuropsychological measures. The merits in this are acknowledged in terms of managing acute effects as well as ease of administration. However, in the broader South African schools context access to information technology remains with the privileged few and research in this area should focus on finding tools that work and that can be adapted for use in all settings.

2.7. Concussion Management Policy

In terms of the South African context there is no policy regarding concussion management. The South African Rugby Football Union (SARFU) has, however, published guidelines for the clinical management of concussion in rugby. The most recent posting on the SARFU website regarding this issue was on 16 May 2006 and was titled, *Step-by-step concussion management: A Guide to coaches and administrators (www.sarfu.org.za, 2006).* These guidelines were written by Drs. Ryan Kohler and Ismail Jakoet in 2003.

There were guidelines for acute management as well as long term management. For the purpose of this proposal, only the long term management of concussion is addressed. SARFU recommended that: "Should a player suffer two concussions in a season he should not play for the rest of the season. Should he suffer three concussions he should be excluded from contact or

collision sport permanently" *(www.sarfu.org.za, 2006)*. They acknowledged that although their recommendations used the precautionary principle they were not evidence based. They also argued that there was no evidence to support the contention that sustaining several concussions over a sporting career will necessarily result in permanent damage. More importantly they argued that should the recommendations be implemented, it would mean the end of a professional player's career and the player could institute a legal challenge to the decision. In their guideline publication they also claimed that the exact pathophysiology of concussion was not known. Furthermore, SARFU defined concussion as a clinical syndrome characterized by immediate and transient post-traumatic impairment of neural function, such as alteration of consciousness, disturbance of vision and/equilibrium due to cerebral or brain stem involvement (Jakoet, 2003). There was no reference to grading of concussion in terms of severity. The above mentioned, non-referenced definition (Jakoet, 2003) was an old definition recommended by Maroon (1980) and upgraded by Cantu (1986) (Bender et al., 2004). This definition was revised in 1999 by the Quality Standards Subcommittee of the American Academy of Neurology (Maroon et al., 2000) and is the one referred to in 2.2 above. The definition of concussion used by a sporting body has major implications with respect to the treatment of concussed athletes, because definitions often determine management protocols (Maroon et al., 2000). SARFU's lack of policy with respect to concussion could be attributed to their lack of clarity in terms of defining and understanding concussion.

2.8. Relevance of the Current Study

The relevance of the current study "The impact of repeated mild traumatic brain injuries (concussions) on the cognitive and academic functioning of early adolescent rugby union players:

A longitudinal prospective study" with a particular focus on residual deficits, is discussed within the framework of contrary and supportive findings in this field. In terms of contrary findings, it was argued by some that sporting injuries do not involve the same degree of acceleration/deceleration force on the brain as do motor vehicle accidents and that in most cases this type of injury was not considered severe enough to warrant hospitalization. Consequently, these injuries are under-represented in the current research literature (Maddocks, 1995).

It was also argued, by Machiocchi, Barth and Littlefield (1998), from their review of the research, that the frequency of head injury was relatively low in sports; that the overwhelming majority of single grade 1 injuries had few persisting symptoms and that in the short term morbidity appeared low. They, however, reported that multiple injuries (>3), especially grade 2 or grade 3, could have long term irreversible consequences and that athletes with apparently equivalent injuries by clinical standards may have different outcomes. Moreover, they went on to state that mild sports head injury ought to receive increased research attention (p. 34).

Preliminary findings on concussive injuries in American football suggested that information-processing deficits were evident within 24 hours of the injury and that neuropsychological function returned to normal within five to ten days (Maddocks, 1995). Similar results were obtained in Maddocks' (1995) prospective study on AFL players. Maddocks (1995) reported mild concussive injuries, involving disturbance or loss of consciousness for only a few seconds. Post Concussive symptoms such as headache, nausea and fatigue resolved within the first few days following injury. Comparison with baseline measure revealed however, that reaction times and speed processing may be reduced for up to two weeks following injury (Maddocks, 1995). The findings suggested minimal evidence of residual deficits.

According to Machiocchi, Barth and Littlefield (1998, pg. 32) most existing studies examined short term symptoms or neuropsychological functioning with minimal regard for more extended or broader aspects of outcome. For example, no studies addressed quality of life, academic performance (in the case of high school and college athletes) and general psychological well-being or for that matter, athletic or job performance. These components may be critical when establishing morbidity following MTBI. Notwithstanding, the focus of the abovementioned studies on acute or short term effects of MTBI, the results continued to yield outcomes that raised concerns for players in the long term.

One such study, conducted by Field et al. (2003), focused on acute response to injury. In comparing the recovery of high school and collegiate athletes following sports-related concussion, the authors reported that despite high school athletes performing significantly worse than age matched controls seven days after injury in comparison to collegiate athletes, they hypothesized that in terms of long-term effects of mild concussion (six months to one year post injury) high school athletes would show a more complete recovery than collegiate athletes given the theories of cortical plasticity. The study showed, however, that the college athletes had a better acute outcome than the high school athletes even though only 11% of the high school players had LOC after the injury compared with 34% college athletes (Field et al., 2003). This outcome raised concerns as it was in contrast to traditional assumptions since return to play decisions were graded in terms of severity on the basis of LOC.

A second concern raised by Field et al's. (2003) research was that there was the assumption that the recovery from concussion was a linear phenomenon and that sequelae were worse

immediately post injury. Their findings showed that high school athletes presented with significant memory deficits seven days post concussion whereas when compared with control data their performance was comparable at day three and day five (Field, Collins, Lovell and Maroon, 2003). The data coincided with data from a similar study on college athletes (Lovell & Collins, 1998). These findings indicated delayed development of cognitive deficits and hence the suggestion of repeated ongoing monitoring of concussed players as well as caution in terms of return to play decisions based on self report data and symptom based profiles (Field, Collins, Lovell and Maroon, 2003). The study indicated that although symptoms had resolved within 3 days, significant cognitive deficits remained at seven days. Furthermore, both college and high school concussed athletes reported fewer symptoms than their baseline scores at five to seven days post injury. These findings raised concerns about underreporting of symptoms, naivety by the players of the potential severity and players' belief that toughness is displayed by playing through the injury (Field, Collins, Lovell and Maroon, 2003).

The biggest concerns with regard to players of contact sport and concussion are the long term consequences. The work by Shuttleworth-Edwards et al. (2004) showed more support for the presence of residual deficits following repeated concussions. Shuttleworth-Edwards et al., (2004) showed that repeated concussions in late adolescent and adult RU players resulted in cognitive deficits that were additive. Despite their findings and the findings of international research on late adolescent and adult players of contact sport including rugby union showing the chronic effects of repeated concussions, these risks have not been considered in the rugby playing community at large and little attention is paid to it by members of the health team. There appears to be a few individuals involved with RU training who have become concerned and have begun to take

preventative steps as a result of the findings regarding the acute and chronic effects of MTBI. These 'converts' are, however, few and far between.

Most of the recent and current research conducted in the area of sports concussion/MTBI has focused, and is focusing, on acute effects and return to play decisions for high school (mean age of 15,9 years), collegiate and professional players of contact sport. There has been little focus on younger players and the residual deficits associated with repeated injuries on such players. Currently in South Africa, at school level, acute concussion is managed by the provision of first aid at most games and at some schools general practitioners are on standby. Boys identified as having sustained a concussion are referred to their general practitioners or to the nearest hospital. "Return to play" decisions are generally made by the treating practitioner in conjunction with the school coach. A handful of schools have recently implemented computerized concussion testing. The current schools' policy recommends, in line with SARFU's guidelines, a three week break from the game following a concussion even though the practitioner gives the all clear. The all clear is generally given on symptom presentation with little emphasis on cognitive or neuropsychological functioning. Unfortunately, as alluded to earlier, mild traumatic brain injury is generally viewed as a minor incident by medical practitioners, sports coaches and managers because they are unaware of the impact on cognitive, academic, behavioural en emotional functioning. A common comment is: "Oh ! it's just a concussion, he'll be fine!" .

The current study was thought to be relevant bearing in mind the concerns raised above and in particular the findings from previous South African research studies conducted on adult and late adolescent RU players. To date no research has included early adolescent RU players. Furthermore there have been no controlled, longitudinal/prospective studies done with early

adolescent RU players to assess the impact of repeated mild traumatic brain injuries (concussions) on cognitive as well as academic functioning (Appendix R).

More specifically, in terms of the relevance of the research, this doctoral study sought to investigate the possible neuropsychological/cognitive and academic effects of repeated concussions on early adolescent junior and high school Rugby Union players. The aims were to investigate, over a period of three years, within the context of BRC theory, using paper and pencil cognitive/neuropsychological tests sensitive to diffuse damage as well as mark reports, whether differences existed between early adolescent rugby union players and non-contact sport controls (hockey, squash, cross-country, fencing) in terms of cognitive and academic functioning. It also aimed to investigate, whether players having sustained repeated concussions showed residual deficits as evidenced by sustained impaired performance on tests sensitive to diffuse brain damage and academic tests relative to non-concussed sport controls.

Participants with a history of prior moderate to severe head injury or learning disability would not be excluded from the study. Because the BRC of these individuals was already vulnerable putting them more at risk for the deleterious effects of repeated mild head injuries, they would be analyzed as a separate group relative to similarly compromised non-contact sport controls. The HYPOTHESES were that in terms of the **time effect** across all the levels of analysis – the scores (the averages) over time of the groups combined would remain the same. In terms of the **group effect,** the hypothesis was one of no group differences e.g. (**concussion versus non-concussion; rugby versus non-rugby; compromised versus non-compromised).** In terms of the **combined effect (of groups and time)** the hypothesis was that there would be no interaction between time and groups. Therefore there would be:

(i) No differences between early adolescent rugby union players and non-contact sport controls (hockey, squash, cross-country, fencing) in terms of BRC, cognitive and academic functioning.

(ii) No difference between early adolescent concussed participants and non-concussed controls in terms of cognitive and academic functioning.

(iii) No difference between compromised participants and non compromised in terms of cognitive and academic functioning.

(iv) No difference between compromised participants and non compromised in terms of cognitive and academic functioning when the concussion variable is added.

(v) No difference between compromised/non-compromised concussed participants showing cognitive and academic deficits relative to compromised/non-compromised non-concussed participants matched for estimated IQ.

(vi) No difference between compromised/non-compromised rugby players showing cognitive and academic deficits relative to compromised/non compromised non-contact sport controls matched for estimated IQ.

(vii) No difference between compromised/non-compromised rugby players with concussions showing cognitive and academic deficits relative to non-contact sports matched controls with no reported concussions, matched for estimated IQ.

(viii) No difference between non-compromised rugby players with two or more concussions showing cognitive and academic deficits relative to non-contact sport matched controls with no reported concussions, matched for estimated IQ.

CHAPTER 3: METHODOLOGY

3.1. Participants

During 2002 and 2003, two consecutive grades of 12-13 year old grade 7 adolescent boys from a Cape Town boys' preparatory school were tested pre and post winter sport season (N=150, 75 boys in 2002 and 75 in 2003). The total grade 7 sample, excluding absentees, comprised rugby (n=115) and non-contact sports controls (n=33). The latter included predominantly hockey plus cross country, fencing and squash players. All boys proceeding to grade 8 from the two consecutive grade 7 classes of the original sample were tested post season in 2003 and 2004. The 2003/4 post season sample comprised rugby (n=73), (n=35) previous rugby and non-contact sports controls (n=30). The shift in the distribution between rugby versus non-contact sports controls (predominantly hockey players) was because boys at the preparatory school could play both rugby and hockey, whereas from grade 8 they were compelled to choose between the two. All boys from the two consecutive classes were tested again at the end of grade 9 (2004, 2005). The total grade 9 sample, excluding absentees, comprised rugby (n=66), (n=40) previous rugby and non-contact sports controls (n=30). Testing further than grade 9 would have been complicated by the ages of the boys as a number of the cognitive tests have ceilings in terms of age. The ceiling is 16 years 11 months and grade 10 boys fall in the age range 15+ to 16.11 years.

3.1.1. Demographic Data

In terms of the demographics, all the boys were English speaking grade 7 learners from a preparatory school attached to a high school catering for high functioning, high SES learners.

They were all between the ages of 12 and 13, and English was the medium in which they were taught. The school had very few socio-economically disadvantaged learners and all boys participated in sport. On commencement of the study the age range of the participants (N = 148) was 12.1 – 13.8 years with a mean age of 12.7 years and a standard deviation of 0.40. On completion of the study the mean age of (N = 136) participants was 15.2 years with a standard deviation of 0.48. When considering each grade, which for the purposes of this study spanned two years, 30 boys in grade 7 reported playing for the 1st team in year one, 26 for the second team, 35 for the third team and 24 for the fourth/fifth team (Table 3.1). The number of team participants changed in the second and third year. At the college/high school the boys were joined by boys from other schools who filled positions on the respective teams, they however, did not form part of the study. The numbers of participants that played in the various positions are indicated in Table 3.2 below. There was virtually no shifting between playing in the forward or backline positions. Forwards tended to remain forwards and backline players remained in the backline.

Table 3.1 Rugby players per team per year

Team	Year 1	Year 2	Year 3
1st team	30	14	13
2nd team	26	22	17
3rd team	35	16	20
4th team	21	14	16
5th team	3	5	00

Table 3.2 Number of players in each position per year

Positions	Year 1	Year 2	Year 3
Forwards	N=65	N=40	N=40
Backs	N=50	N=31	N=26
No Rugby	N=33	N=67	N=70

3.1.2. Selection Criteria

All 150 grade 7 boys, age 12-13 years in their final year at preparatory school, progressing to the same high school for grades 8-12, were selected. Participants with a history of prior moderate to severe head injury, learning, neurological and/or psychiatric disorder were excluded from the first level of analysis but included in subsequent levels of analysis in this study. Because the BRC of these individuals is already vulnerable, putting them more at risk for the deleterious effects of repeated mild head injuries, they were to be analyzed as a separate group relative to similarly compromised non-contact sport controls. Participants with previous mild head injuries were not excluded because this would exclude a large number of rugby players and furthermore the aim of the study was to investigate the effects of repeated concussions (MTBI) on cognitive and academic functioning.

3.1.3. Exclusion Criteria

To control for potentially confounding variables, participants with a history of prior moderate to severe head injury, learning, neurological and/or psychiatric disorder were excluded from the first level of analysis of this study. Because post-season testing took place towards the end of November, approximately ten weeks after the season ended, players concussed at the end of the season (last two weeks in August) were excluded from the study because the study was researching residual/chronic as opposed to acute effects. The latest that post season testing could take place was at the end of November because the school closed in December.

3.2. Sample Size

The study commenced with a sample size of 150 participants. In year one, (n=115) rugby and (n=33) non contact sport controls. In year two, (n=73) rugby, (n=35) previous rugby and (n=30) non-contact sport controls and in year three, (n=66) rugby, (n=40) previous rugby and (n=30) non-contact sport controls.

Previous rugby research studies using group mean comparisons, used (N=47) professional players (n=26) rugby & (n=21) cricket (Reid, Shuttleworth-Jordan, Ancer, Dickinson, Radloff & Jakoet, 1999; Ancer, 1999); (N=66) professional and u21 players (n= 45) rugby & (n=21) national hockey (Finkelstein, 1999); (N=81) high school players (n=47) rugby & (n=34) hockey (Shuttleworth-Edwards, Ackermann, Beilinsohn, Border & Radloff, 2001; Ackerman, 2000) and (N=137) high school players (n=79) rugby & (n=58) hockey (Giai-Coletti, 2002). Significant differences at $p<0.05$ and $p<0.01$ levels were revealed in a variety of areas in all of the above mentioned research projects.

Senior statisticians at the University of Cape Town and the University of Stellenbosch gave professional opinions that the current sample size was sufficient from a statistical perspective. Power is maximized through our designs, through our statistical procedures it allows and the data it produces (Heiman, 1995). In terms of power the general rule is to look for big effects, to use large samples or to employ sensitive experimental designs such as those that involve the use of repeated measurements and the same subjects reducing experimental error and thus making small differences translate into large effect sizes (Howell, 1995). With a power >1 there is more than 100% chance of rejecting the null hypothesis of no difference between the groups.

The current sample size was restricted by the scope prospective, controlled, longitudinal nature of the study. This was an unfunded doctoral thesis. Limitations in terms of the sample size were as follows: The study was prospective in nature and was aimed at early adolescents. Early adolescence commences at age 12-13 years and this therefore included grade 7 learners. Grade 7 learners were in their final year at preparatory school and needed to move to a high school for grades 8-12. There are very few schools where boys remain in the same schooling system i.e. where they attend the same preparatory and high schools. In terms of this study it was necessary for boys to remain in the same school and fortunately most learners did. It would have been very difficult to include participants from preparatory schools feeding a wide variety of high schools because of the logistics of arranging test situations for small rugby and non-contact sport control groups at schools. Not only would the task have been almost impossible, there would have been too many variables impacting on the data. This particular school only catered for 75 learners in grade 7 and for that reason an additional group was assessed the following year, bringing the total number of participants to 150. The addition of the second group added a year to the data collection and any additional groups would have had the same effect. In terms of the various specific levels of analyses planned it was estimated that a perfect sample size would be in the region of 500 participants which would have equated to another 5 years of 75 participants per year, each group tested for 3 years resulting in an additional 15 years added on to the existing 4 years of data collection. This scenario would have been beyond the scope of both a funded and unfunded doctoral thesis. However, in terms of the sample size needed, when there is a large effect size e.g. of 3.3 and a power of 0.95 is needed, a small sample size will be required e.g. 2.4 participants, in a repeated measures design. Alternatively with a medium effect size e.g. 0.50 at a power of 0.80 a sample size of 126 would be needed for a two-sample *t* (Howell, 1995). The equation used to calculate the effect size was $d = M_1 - M_2 / \sigma_{pooled}$ and $\sigma_{pooled} = \sqrt{[(\sigma_1^2 + \sigma_2^2) / 2]}$

(Cohen,1988). This formula was used to calculate the effect size for the groups and it is argued that for the time effect sizes the same formula can be used (web.uccs.edu/lbecker/Psy590/es.htm#Cohen, 2008). Below is an example of 2 graphs for power calculations based on the CD from the "any concussion" data. One is for the group effect (Fig 3.1) and one for the time effect (Fig 3.2) that shows for example when comparing groups, for a "medium effect" of 0.5, one obtains a power of between 70% and 75%. For a "large effect" of 0.8, there is a power in excess of 90%. What this means is that if a medium effect was present in the data, one would have had a 70-75% chance of picking it up, or >90% for a large effect. For the time effect the sample is even bigger and the power better (>90%).

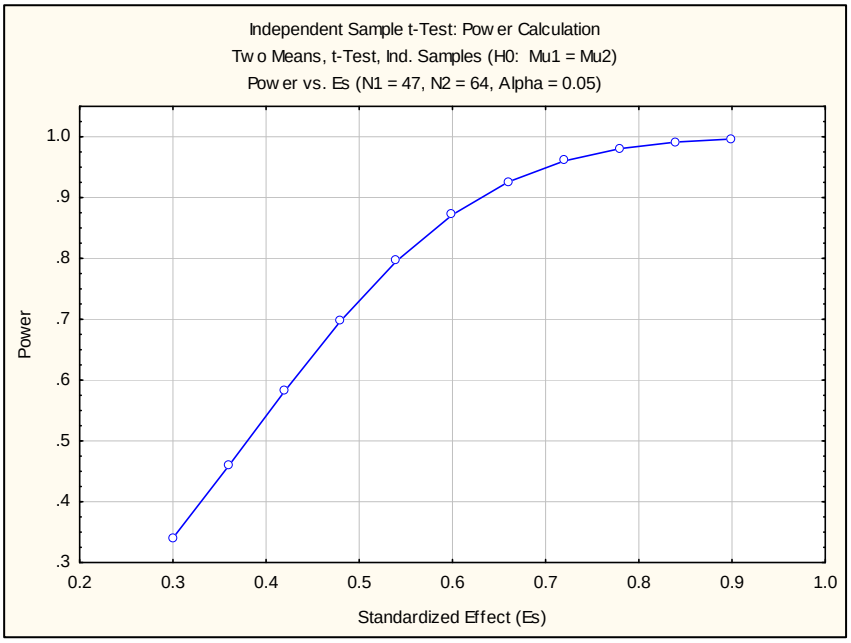

Figure 3.1 Power calculation for 'any concussion' on CD group effect size

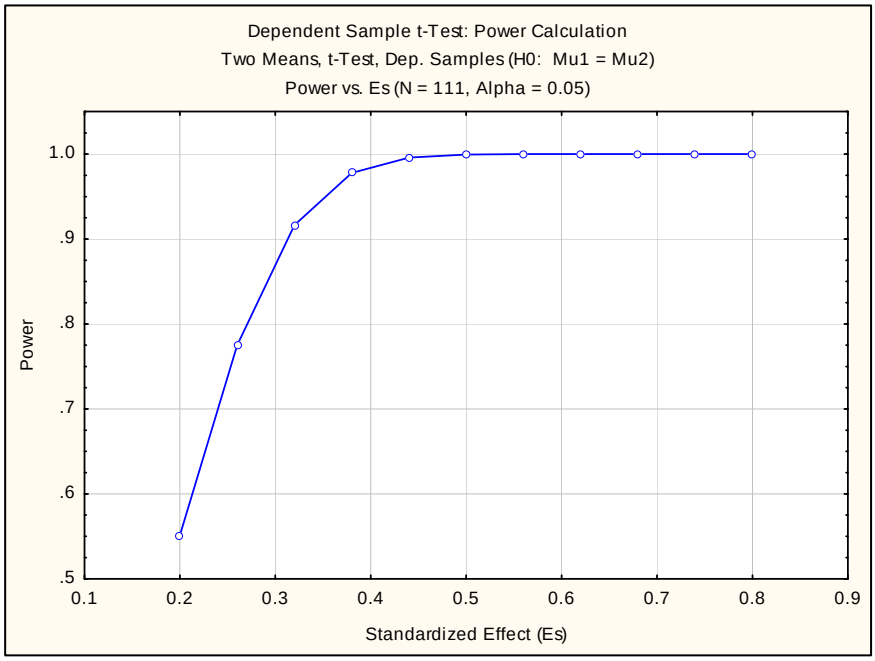

Figure 3.2 Power calculation for 'any concussion' on CD time effect size

3.3. Procedure

All boys completed self-report questionnaires and paper and pencil tests at the initial pre-test (March 2002 & 2003) and three post winter sport season tests (2002/3; 2003/4; 2004/5). The baseline, pre-test was conducted seven months after the previous rugby season. Post-season testing took place towards the end of November, approximately 10 weeks after the end of the season, to target residual rather than acute effects. Therefore, players concussed at the end of the season (last two weeks of August) would have been excluded from the study. Players reported concussions to their coaches who documented the incidents in the school injury record book along with all other injuries. In terms of the study, concussions were reported when completing

the questionnaire. The value of the injury book at the junior school was that reported or non-reported concussion could be verified.

In the first year(s) at the preparatory school boys completed the baseline testing in their respective classes. There were three grade 7 classes per year. At post-test occasions some classes were tested in their classrooms whilst others were tested in a lecture theatre. Class teachers were present on all occasions. In subsequent years the group was split into two groups with approximately 30-35 boys in each group and testing took place in a lecture theatre with a teacher and student assistants present. Generally, testing took on average one hour, however, in groups where learners were slower, testing time was slightly longer. The tests were administered during school time by one researcher, a clinical psychologist who received her training at Rhodes University where a course in neuropsychology formed part of the criteria necessary for obtaining a clinical masters degree in psychology. The same test battery (Appendix D-M) was used at each test occasion. Standardized test instructions taken from either the original manuals or Lezak's (1995) guidelines, appeared on each test protocol.

All participants spoke English, were taught in English and the testing was conducted in English. Prior to testing participants were informed of the nature and purpose of the study and consent, withdrawal and confidentiality were addressed (see Ethical Considerations 3.8.6). Academic data (Aggregate, English and Mathematics scores) for each boy was obtained from the master in charge of academics or the headmaster.

3.4. Consent Forms and Questionnaire

Prior to the commencement of the study meetings were held with the head of sport, coaches and parents to discuss the project. Information regarding the study as well as the consent forms were forwarded to the headmaster and head of sport (Appendix A), parents (Appendix B) and boys (Appendix C) prior to testing. Consent was obtained from the headmaster of the school (Appendix A) and parents (Appendix B). Participants were informed about the nature of the study and invited to give free informed assent should they wish to participate (Appendix C). In terms of consent within a school setting and the influence on the consent procedure, the researcher was very aware of the ethical implications of parents and the school consenting but that the child may not give his assent. The child's decision was always respected and at no point was any participant stopped from leaving the room when they chose to do so.

Participants were informed that participation was voluntary and they could withdraw from the study at any stage and such withdrawal was without penalty. Furthermore the school and the parents could withdraw the boys at any stage without adverse consequences and without having to state a reason. Such free informed consent and withdrawal was consistent with the revised Guidelines on Ethics for Medical Research (4^{th} edition) presented by the Medical Research Council of South Africa Ethics Committee as well as the Department of Health (RSA) (2005), Draft Guidelines for Good Practice in the Conduct of Clinical Trials in Human Participants in South Africa. Furthermore, all parties were assured of strict confidentiality and anonymity. They were assured that no boy's name would be mentioned in the thesis or any publication. Confidentiality about individual participants would be preserved, through the use of coding

throughout the research. Information would only be made available to the boy, his parents and/or a medical practitioner on request with the boy and the parents' permission.

The self-report questionnaire comprised demographic information regarding age, grade, sport, length of participation in the sport, previous concussions, head injuries, learning difficulties, medical and psychiatric history as well as recent concussion and symptoms experienced (Appendix D).

3.5. Neuropsychology Test Battery

The assessment battery contained the cover sheet (Appendix E) and the tests. The tests included: (i) a measure of general intellectual functioning and (ii) neuropsychological measures within 6 modalities known to be sensitive to the non-specific effects of diffuse brain damage (see 2.6 p. 66). The tests most likely to show the effects of brain damage are tests that require immediate memory, concentration, response speed and abstract concept formation whereas tests of previously learned information and verbal associations tended to be less affected (Lezak, 1995). VOC (Appendix F) was used to provide an estimate of premorbid IQ. Tests sensitive to MTBI included the following: SIM (Appendix L); COD (Appendix H) including CI (Appendix I) and CD (Appendix M) memory tasks; The VPAI (Appendix G) and VPAII (Appendix K) memory tasks; DET (Appendix J).

The WISC III and the WMS III have standardized norms. The WISC III has outstanding reliability. Full, Verbal and Performance Scales have internal consistency reliability co-efficients of 0.89 or above over the entire range covered in the standardization group (Sattler, 2001). A

number of studies have investigated the criterion validity of the WISC III by correlating the test with other intelligent tests, measures of achievement and school grades. The validity studies show that the WISC III has satisfactory criterion validity (Sattler, 2001). Furthermore, it has adequate construct, concurrent and predictive validity for many types of children with and without disabilities in the age range covered by the test (Sattler, 2001). The WISC III subtests are suitable for use with adolescent boys because it is has norms for ages 6 years to 16 years 11 months. Furthermore, these subtests were deemed suitable given that participants attended a school catering for relatively high functioning English speaking children from a high socio-economic background.

3.5.1. General Intellectual Functioning

VOC was used to provide an estimate of premorbid IQ. The Vocabulary subtest provides a useful index of general mental ability (Sattler, 2001). It is expected to "hold" following a brain injury (Golden, Espe-Pfeifer & Wachsler-Felder, 2000). Performance on the subtest has been found to be stable over time and relatively resistant to neurological deficits and psychological disturbance (Sattler, 2001).

In an attempt to ensure group homogeneity, Lezak (1995) condoned the use of a single subtest score in isolation as an adequate estimate of premorbid IQ. Ancer (1999) highlighted a major omission in many previous studies by noting the lack of an attempt to build 'hold' tests into the battery (pg. 74).

VOC is not a timed test it contains 30 words arranged in order of difficulty. The test requires the child to listen as each word is read aloud and to explain the meaning of each word (Sattler, 2001). In the current study children were asked to write their responses. Items were scored 2, 1, or 0 (Sattler, 2001).

3.5.2 Tests sensitive to MTBI

3.5.2.1. Verbal Memory (immediate and delayed)

VPA I is an untimed task of verbal cued recall and learning (Wechsler, 1997); It includes a delayed task (VPA II) to assess delayed recall and retention of verbally learned material. Previous head injury studies indicated problems for schoolboy rugby players on the delayed recall task (Shuttleworth-Edwards et al., 2004).

WMS III is the most popular memory test battery in the field. The psychometric and clinical characteristics are clearly superior to early versions of the test. The reliability data surrounding the VPA subtest improved quite drastically from prior versions. The split half reliability of this subtest rose from an average of .60 to an r of .92 for VPA I and from an average of .40 to .86 for VPA II (Mitrushina, Boone & D'elia, 1999). Wechsler (1997) reported that changes to this test were made on the basis of research which showed that performance on difficult associations had been found to be more highly correlated with other measures of verbal memory and was more sensitive to cerebral dysfunction. All the items are of the more difficult type and are therefore more likely to be assessing the same construct. Reliability estimates of both VPAI and II subtests on test-retest reliability fell within the 0.81- 0.82 range (Golden et al., 2000).

Administration of the test VPA I involved reading eight difficult word pairs in succession, to the participants. Each word pair was read at the rate of one pair of words every three seconds; that is the words were spoken about 1 second apart, with two seconds separating each pair. Five seconds after the pairs had been read the examiner cued the participants with the first word of each pair and the participant was expected to respond by writing the associated word (Golden et al., 2000). The first trial assesses recall of verbally cued material. The first trial was followed by three more trials with the same set of word-pairs in different orders. The order is randomized so that learning of word-pairs takes place (Golden, et al., 2000). As with the first trial, after each trial the participants were cued and responded by writing the associated word. Subsequent repeated trials assess cued learning of verbal material (Golden et al., 2000).

To be able to do this test participants required language abilities at a basic and fundamental level, allowing the test to be administered to individuals who do not have strong language skills i.e. English. With language skills intact immediate recall is best shown by the first recall (Golden et al., 2000).

Scoring of VPA I comprised the sum of the totals across all four trials. Improvement in the scores from the first to the last trial suggests an effective learning strategy when memory is poor (Golden et al., 2000). VPA II is a 30 minute delayed recall of the last learning trial. This test measures delayed recall and short-term retention of verbally learned material. It provides an assessment of the participant's ability to encode, store and retrieve information (Golden et al., 2000).

In terms of the individual analysis, norms were available from studies conducted on primary and high school students (Hulicka, 1966 and Ivinskis, Allen & Shaw, 1971) as the standardized sample included only individuals ranging in age from 16 – 89 years.

3.5.2.2. Processing Speed (Visuoperceptual Tracking)

Processing speed measures the ability to process visually perceived nonverbal information quickly, with concentration and rapid eye-hand coordination being important components (Sattler, 2001, p. 233). COD is consistently more sensitive to brain damage than all of the other WISC III subtests (Sattler, 2001). It is a good indicator of diffuse brain damage commonly associated with head injury. Performance on this test is affected regardless of the locus of the lesion and even when brain damage is minimal (Shuttleworth-Jordan & Bode, 1995).

COD is a complex, timed, fine motor test. In addition to measuring visuoperceptual tracking it measures fine motor control, speed, memory, sustained attention, visuomotor coordination and complex scanning (Golden et al., 2000). This test is a poor measure of crystallized intelligence but it contributes to the Processing Speed Index (Sattler, 2000). The test consists of 6 rows, containing, in total, 126 blank squares. Above each square is a number from 1-9 in random order. Seven of the squares at the beginning of the first row are sample squares. At the top of the page is a printed key. The numbers 1-9 are each paired with different symbols. The participants were instructed (as per the WISC III manual) to fill in the symbols in the blank squares below each number as quickly and as accurately as possible. Before commencing the test participants were given an opportunity to complete the sample squares. Participants were requested to work from left to right and not to omit any squares. The test time was two minutes (120 seconds). On

completion of the test participants were asked to circle their last entry. If they had not completed the test to the end of the fourth line they were asked to do so immediately for the purpose of the recall tasks.

3.5.2.3. Visual Memory (immediate and delayed)

CI and CD tasks are part of the test battery due to their demonstrated sensitivity to the effects of diffuse brain damage (Walsh, 1985). Delayed memory is reportedly more sensitive in this regard than immediate memory. The outcome of the recall tasks gives an indication of the participant's visual memory (Lezak, 1995). A recall of six of the nine symbol pairs indicates a performance in the low range of normal (Kaplan, Fein, Morris & Delis, 1991).

CI

The short form of the Incidental Recall (Shuttleworth-Jordan & Bode, 1995) described by (Kaplan, Fein et al. 1991) was used in this study. The immediate recall was presented to the participants once they had completed to the end of the forth row of the COD task. Completion to the end of the forth row ensured sufficient exposure to all the numbers. Participants were requested to fill in on a separate sheet of paper the symbols they could recall in the blocks under the numbers 1-9 that correspond with the numbers.

CD

The delayed recall was presented to the participants after twenty to thirty minutes. Participants were again requested to fill in on a separate sheet of paper the symbols they could recall in the blocks under the numbers 1-9 that correspond with the numbers.

3.5.2.4. Abstract Thinking

SIM is a test measuring abstract thinking/verbal concept formation and long term memory. The abstract verbal skills are thought to reflect higher cortical functions of the brain (Golden et al., 2000). SIM is one of the tests more sensitive to brain injury, regardless of localization, than any other Wechsler Intelligence Scale tests (Hischenfang, 1960b). Unlike VOC, it is not considered a "Hold" test. Concrete thinking or the inability to think abstractly often appears with any kind of brain damage, this outcome may be reflected in lowered scores on SIM (Lezak, 1995). Lezak (1995, p. 697) suggested that lowered SIM scores may be the most pronounced residual cognitive deficit of a bright person that has had a mild brain injury. The SIM subtest requires participants to answer questions about how objects or concepts are alike. They are asked to state the similarity between two items in each of the 19 word pairs (Sattler, 2001). Items 1-5 are scored as one or zero and items 6-19 as two, one or zero.

3.5.2.5. Motor Functioning

DET is a test measuring motor speed and precision (Baker and Leland, 1967). This test was administered for three minutes and it involved participants making an "x" in a circle. The instruction was that this task should be done as quickly and as accurately as possible working from left to right, not missing any circles and not recording outside of the circle. The scores in terms of the norms which were chronologically based were expected to increase with age.

The difficulty with neuropsychological testing is that the data is dependent on the person being assessed. The outcome is influenced by the person's motivation and whether they are honest in

their responses. The individual may also exaggerate their difficulties which will show more dysfunction than is present (Bennett & Raymond, 1997). For the purposes of the current research the terms neuropsychological tests and cognitive tests will be used interchangeably.

3.6. Academic Battery

Academic measures were obtained from the participant's overall academic, mathematics and English examination scores. Mathematics and English were the only two subjects that remained constant for all students over time during junior and high school years and were therefore the most appropriate means of monitoring differences in scores over time. Scores were taken from final examination results of the year prior to pre-testing and from the final examination results at the end of each year post-testing. Results were reported in percentages by the schools.

English scores were expected to remain relatively stable whereas it was anticipated that mathematics scores may be influenced by concussions. English language skills assessment includes assessment of vocabulary, concept formation, language development, comprehension, knowledge of conventional behaviour and social judgment. English scores were expected to remain stable because assessment was of old acquired knowledge that remains stable following an insult to the brain, except of course if the insult is to the left side of the brain. Mathematics on the other hand includes an element of attention and concentration, working memory, mathematical reasoning, logical thinking, identifying relationships, understanding probability as well as processing speed. Studies on neuropsychological sensitivity of Wechsler tests have shown that tests reliant on immediate memory, concentration, response speed and abstract concept formation are most likely to show effects of brain damage (Lezak, 1995)

3.7. Data Processing and Statistical Analysis

Following each pre and post-test all participants' booklets were marked, scored and scaled scores obtained where applicable. The data were marked and scored by the researcher and an assistant, a Business Science graduate, who was trained by the researcher in the scoring of the tests. Both strictly adhered to the scoring procedure to ensure uniformity of scoring. Scoring was counterchecked to ensure consistency of scoring.

At the pre-test phase each individual's demographic data, concussion medical, psychiatric and educational history, cognitive and academic scores were captured on an excel spreadsheet. This procedure was repeated at each post-test phase. All data captured was counterchecked to ensure accuracy.

The quantitative and qualitative data analyses in the main comprised four parts: (i) correlational analysis (ii) group mean comparisons (iii) descriptive analysis in the form of percentage of deficits and (iv) case-study analysis. A statistical package, Statistica was used to perform the cross tabulations, correlational analysis and group mean comparisons.

(i) Correlational analyses were performed to establish the strength and direction of the relationship between the VOC score an estimate of BRC and the other subtest scores which included the neuropsychological tests (SIM, VPA1, VPA2, CODING, CI, CD, DT) and academic tests (AGG, ENG, MATHS) of all participants. Correlations are used in predictions. The higher a correlation between two variables the more accurate one can predict the value of one variable when one knows the value of the other. A correlation above 0.50 either positive or

negative suggests a moderate to strong relationship between two variables (Sattler, 2001). The hypothesis is that there will be no relationship between the VOC score and the other measures and the correlation will therefore equal zero.

(ii) Group mean comparisons were conducted between rugby players versus controls, concussed versus non-concussed players, compromised versus non compromised controls (e.g. those boys with a history of prior moderate to severe head injury or learning, neurological or psychiatric disorder) for all neurocognitive/psychological (SIM, VPA1, VPA2, CODING, CI, CD, DT) and academic measures (AGG, ENG, MATHS), across repeated test occasions. The next level of analysis focused on the non compromised group where the concussed rugby group was compared with the non-concussed, non-rugby groups for all neurocognitive and academic measures, across repeated test occasions, taking into consideration the effect of VOC on the scores. This level of analysis was repeated for vulnerable/compromised players. This data analyses provided statistical comparisons between the groups using Analysis of Variance, Repeated Measures Analysis of Variance and Analysis of Covariance. As with the recommendation of Cohen (1994) and Cicchetti (1998) use will be made of graphical methods, reporting of effect sizes and descriptive statistics.

(iii) The third level of analysis involved descriptive measures. Analysis was undertaken to explore within group differences in light of the between group findings. A sole reliance on tests of statistical significance (e.g. group mean comparisons) in the understanding of neuropsychological data could confound conclusions drawn from such research regarding brain functions Donders (2000) (as cited in Bielensohn, 2001) and Zakzanis (1998). To avoid drawing fallacious conclusions with respect to brain-behaviour relations via sole reliance on quasi-

experimental group comparisons and to produce clinically relevant data (Bielensohn, 2001), percentages of deficits were indicated.

The analysis of the descriptive data involved comparisons of percentages of deficits on test scores of participants from comparative groups for all neurocognitive and academic measures, across repeated test occasions, to investigate pre and post season effects. In terms of the percentages of deficits, analysis included baseline and third post year data of non compromised participants. The number of participants of each comparative group 'non-rugby', 'previous rugby' and 'current rugby' that showed deficits on neuropsychological and academic tests was calculated. As with the Shuttleworth- Edwards et al., (2004) study, deficits on the individual tests were determined according to the degree to which the test scores differed significantly from the most appropriate norm in the direction of poor performance. According to Shuttleworth- Edwards et al., (2004) no deficit was determined by a test score within one standard deviation from the norm. A mild deficit was determined by a test score equal to or greater than 1 standard deviation from the norm but less than two standard deviations and a moderate to severe deficit was a test score equal to or greater than two standard deviations from the norm. For the purposes of this study, any deviation from the norm was noted. As with the Shuttleworth- Edwards et al. (2004, p. 158) study the most appropriate available normative data for each of the subtests was used (Appendix Q). The norms in this study were determined from the group of participants that were not compromised, that did not play rugby and had reported no concussions. The norms obtained were in keeping with the population norms obtained for the particular cognitive tests. The norms for the academic measures were calculated on the percentages for the different measures.

(iv) Case-study analyses were conducted on rugby players reporting three or more concussions and those with fewer or no concussions and previous and no rugby. In terms of the choice of three or more concussions Shuttleworth-Edwards, Ackermann, Beilinsohn, Border and Radloff (2001a) reported an average historical incidence of 2.3 (range 0 – 7) concussions per South African schoolboy rugby player versus an average incidence of 0.4 (range 0 – 1) for an equivalent group of hockey players. The reason for this level of analysis was to prevent a small population of individuals with perceived or reported deficits from going undetected and to gain a better understanding of the individual as opposed to their test scores within or between groups. By grappling with the individual, his history and his/her performance on estimates of BRC, Neuropsychology and academic functioning, one brings a measure of clinical relevance to the research context. The norms used for the case studies were the same norms used in the descriptive measures above.

3.8. Ethical Considerations

Permission to conduct the study was obtained from the headmasters of both the preparatory and senior schools (Appendix A). Informed consent was obtained from the parents and assent from the boys participating in the study (Appendix B & C). Parents who did not wish their sons to participate could withdraw their sons from the study at any time without penalty. On the day of testing boys were also informed that their participation was strictly voluntary and should they not wish to participate they could withdraw then or at any stage. Furthermore they were reassured that withdrawal would not prejudice any boy in any way in terms of his position in the sport team. Participants were assured of strict confidentiality and anonymity at all times and that information would only be available to parents and/or a medical practitioner on request and with

the boy and the parents' permission. It was emphasized that the project was neither invasive nor harmful to the child's physical, mental and or emotional well-being. Data arising from the research project would be used for thesis and scientific publication purposes only. The results of the study would be made available to the participants, their parents and the participating school.

It is important to note that the study took place prior to and on completion of the rugby season not during the season and that the aim of the study was to investigate chronic/residual effects of repeated concussions. Acute effects did not form part of the study and were managed by coaches, first aiders, medical practitioners and parents. The school involved in the study had a full medical and first aid service available at all games. They also had a general practitioner who was involved with a concussion management programme. When a boy was concussed he was assessed at the school by the medical staff and referred to either his general practitioner or the hospital. In appendices A-C staff, parents and boys were informed that the study would not interfere with or substitute good medical practice. The policy regarding return to play was one that the school used and was recommended by SARFU. Since the inception of this study the preparatory school has altered its policy from compulsory rugby for all boys, to boys being given a choice to play the game. Furthermore, by the time the study was completed the high school had introduced a computerized concussion management programme to monitor acute concussions and to inform return to play decisions.

This research did not get involved in return to play decisions; it merely monitored whether there were residual/chronic effects from repeated concussions at the end of each season. However, if and when, at the end of the season, there was a significant decline in the boys scaled scores either following a concussion or not, the researcher informed the player and his parents of the risk.

Some boys stopped playing the game of rugby as a result of the feedback whereas others continued knowing the risks. Please note that this intervention did not form part of the study, it was merely implemented because of a personal, clinical and ethical responsibility towards the well being of the boys. In terms of this responsibility the researcher remained mindful of the limited neuropsychology knowledge regarding the impact of MTBI on brain functioning of first-aiders, coaches, parents, general practitioners, sports medicine physicians, neurosurgeons and neurologists. The practitioners generally advise the patient and his family in terms of the symptom presentation and other neurological investigations, with little or no focus on neuropsychological functioning. Furthermore, CT and MRI scanning usually show no brain abnormalities following MTBI and consequently the individual is given a clean bill of health bar a few residual symptoms. This, however, does not mean that the individual's cognitive/neuropsychological functioning is intact.

The methodology of the current study provided for a prospective, longitudinal well controlled design. Participants were matched in terms of age, sex, grade, school, language, participation in sport, socio-economic status and BRC (estimated IQ). The analysis provided for between group and within group comparisons and these findings were further supported by case studies.

CHAPTER 4: RESULTS

The results chapter consists of four major levels of analysis: *Level 1* includes the *Subjective Measures* which are the results of data analyzed from the participants' subjective reports of concussions sustained. *Level 2* includes the *Objective Measures* which include both global and more specific analysis: The global level refers more broadly to the all inclusive (all participant) analysis which involves the *Relationship Measures* i.e. the strength and direction of the relationship between subtests as well as the *Analysis of Variance* of the major groups i.e. "Concussion", "Rugby" and "Compromised" participants. The more specific results are those results obtained from the analysis of all above mentioned groups (in the global analysis) combined. *Level 3* refers to *Descriptive Measures* in which the number of players in each comparative group that shows deficits on the cognitive and academic tests is calculated. *Level 4* is the *Individual Results Analysis*, alternatively referred to as '*Case Study'*.

The results are presented below as subjective, objective and descriptive measures as well as in a 'case study' format.

4.1. Subjective Measures

This section (level 1) presents the outcomes of the subjective reports of concussion from rugby union players and non-contact sport controls. The data which included responses from all participants were analyzed by means of cross tabulation using a Chi-square test. The results were of participants' subjective reports of the number of concussions sustained prior to and over the three year research period. The comparison groups were rugby players (n= 66) versus non-

rugby/previous rugby players (n =70). The results p = <0.01 revealed that (Fig 4.1) 32% of rugby players reported no concussion whilst 68% responded positively to sustaining one/more concussions. In the non-rugby and previous rugby playing group 64% reported no concussion whilst 36% responded positively to sustaining one/more concussions (Fig 4.1).

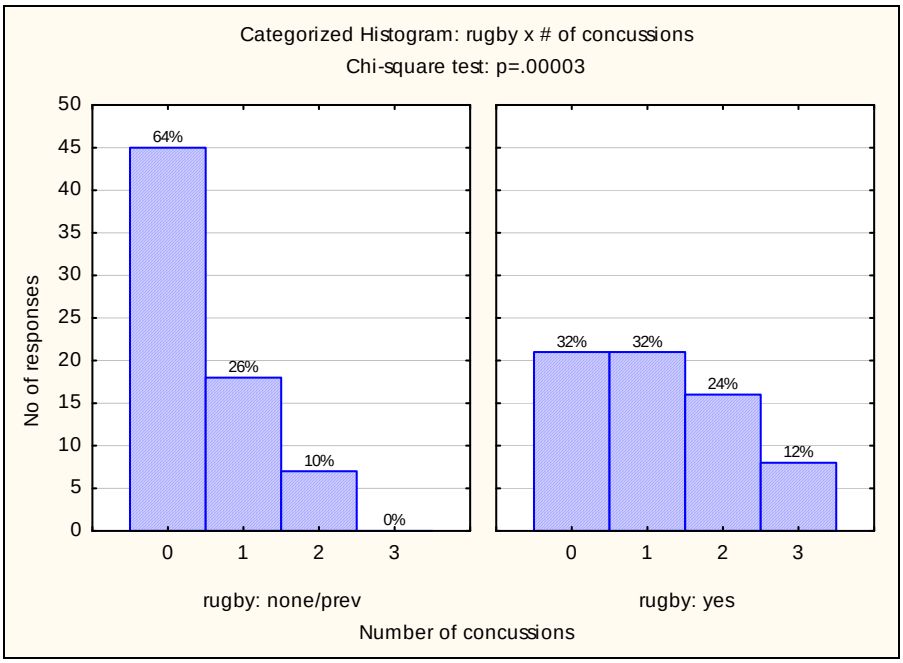

Figure 4.1 Number (#) of concussions reported by groups

Of the concussion scores in the previous and no rugby group 76% was accounted for by the previous rugby group and 24% by the non rugby group. Of the 30 participants representing the non-rugby playing group four reported having had one concussion (13.3% of this group), two, two concussions (6.6% of this group) and 24 no concussions (80% of this group). Of the 40 participants that comprised the previous rugby group 14 participants reported one concussion (35% of this group), five participants two concussions (12.5% of this group), and 21 participants zero concussions (52.5% of this group) (Table 4.1)

Table 4.1 Percentages of participants' reports of number of concussions.

Concussions	No Rugby N=30	Previous N=40	Rugby N=66
0	80%	52.5%	32%
1	13.3%	35%	32%
2	6.6%	12.5%	24%
3	0	0	12%

However, when each reported concussion incident (the actual number) is tallied for the different groups as opposed to the numbers reported by individual participants, the actual number of concussions for the rugby playing group (n=66) is 77 (Table 4.2). When these figures are recalculated as actual incidences, the percentage of concussions reported by non rugby players is 20%, previous players 65% and rugby players 117%.

Table 4.2 Actual number/incidence of concussions reported.

Concussions	No Rugby N=30	Previous N=40	Rugby N=66
0	24	21	21
1	4	14	21
2	2 (x2)	5 (x2)	16 (x2)
3	0	0	8 (x3)
Totals	8	24	77

Twenty two participants reported having had concussions (rugby and other) prior to commencement of the study. Of the 22 participants 14 had had grade 1 concussions and eight grade 3 concussions (p. 16). Of those eight, three were again concussed during the study period. No boys reported grade 2 concussions. During the *first year* of the study a total of 51 participants reported concussions. 14 grade three concussions were reported in total (Table 4.3) three in the first year, nine in the second year and two in the third year. Of the three referred to in the first year of the study, two boys reported two concussions in the same year. The total number of participants reporting concussions during the *second year* were 38 and during the *third year* 23.

Grade 1 concussions were the most commonly experienced during the research period, however, reporting thereof diminished over time from 44 reports in year one to 17 in the third year.

Table 4.3 Number of concussions per concussion grade reported prior to and during the study

	Grade	Yes/ concussion
Prior to testing	grade 1	n= 14
	grade 2	N= 0
	grade 3	N= 8
Year 1	grade 1	n= 44
	grade 2	N= 4
	grade 3	N= 3
Year 2	grade 1	n 25
	grade 2	N= 4
	grade 3	N= 9
Year 3	grade 1	n= 17
	grade 2	N= 4
	grade 3	N= 2

Over the three years, the symptoms reported most frequently by concussed participants were dazed/dizzy (n=85), headache (n=41), disorientation and "seeing stars" (n=35). Eleven participants were reportedly hospitalized, 12 reported loss of memory and 19 reported nausea (Table 4.4). The figures for hospitalization are not in keeping with the reports of grade 3 injuries given that the general public perception is that one has to be unconscious before serious intervention is required. The hospitalization figures of four, three and four respectively, do not coincide with grade 3 injuries of three, nine and two reported in years one, two and three.

Table 4.4 Concussion related symptoms reported during the study

Symptoms	Year	Numbers
Dazed / dizzy	year 1	n= 38
	year 2	n= 31
	year 3	n= 16
		Total= 85

Headache	year 1	n= 15
	year 2	n= 16
	year 3	n= 10
		Total= 41
Loss of memory	year 1	n= 3
	year 2	n= 7
	year 3	n= 2
		Total= 12
Nausea	year 1	n= 4
	year 2	n= 5
	year 3	n= 10
		Total= 19
Seeing stars / disorientation	year 1	n= 14
	year 2	n= 6
	year 3	n= 15
		Total= 35
Hospitalization	year 1	n= 4
	year 2	n= 3
	year 3	n= 4
		Total= 11

In summary, results of subjective measures indicated that concussion is more prevalent in the rugby playing group versus the non-rugby and previous rugby playing group. Rugby players experience more repeated concussions than non rugby players, with most of the concussions falling in the grade one category. Notwithstanding the frequency of grade 1 concussions, participants reported more grade 3 injuries than grade two injuries over the study period. The symptom reported as experienced most often was feeling dazed or dizzy.

4.2. Objective Measures

The objective global (Level 2 analysis) measures firstly comprised the correlational analysis between the IQ estimate and the other subtest scores (neuropsychological and academic data) to establish the presence of a relationship between the two groups. Secondly it included the analysis

of variance of "Concussion", "Rugby" and "Comprised" measures and thirdly the outcome of the more specific analysis was the analysis of all the global measures combined.

4.2.1. Relationship Measures

To establish the strength and direction of the relationship between the VOC and the other subtest scores, the Pearson Product Moment Correlation Coefficient (r) was used. In terms of the results, the relationship between VOC baseline and the other measures of the neuropsychological tests SIM, VPA1, VPA2, COD, CI, CD, DT and academic tests AGG, ENG and MATHS of all participants was analyzed across the four test situations to determine the strength of the relationship at the outset and over time.

In terms of the analysis from baseline through to year three the moderate to strong positive relationships were between the **VOC** and **SIM** with the following ($r = 0.51, 0.55, 0.44, 0.40$; $p < 0.01$) respectively; **VOC** and **AGG** with the following $r = (0.47, 0.52, 0.52, 0.46$; $p < 0.01$) respectively (Fig 4.2); **VOC** and **ENG** with the following ($r = 0.47; 0.57; 0.47; 0.61$; $p < 0.01$) respectively (Fig 4.3). The only mild to moderate positive relationships were between the **VOC** and the **MATHS** with the following ($r = 0.42; 0.47; 0.44; 0.43$; $p < 0.01$) respectively and **VOC** and the **VPAI** with the following ($r = 0.37; 0.30; 0.30; 0.34$; $p < 0.01$) respectively. No significant relationship was evident between the **VOC** and the **CD; CI; COD; DET; VPAII** suggesting that performance on these subtests is not dependant on performance on VOC. Therefore a low or high VOC would have no bearing on the outcome of performance on these tests which measure delayed and immediate visual memory, processing speed, motor functioning and delayed verbal memory respectively.

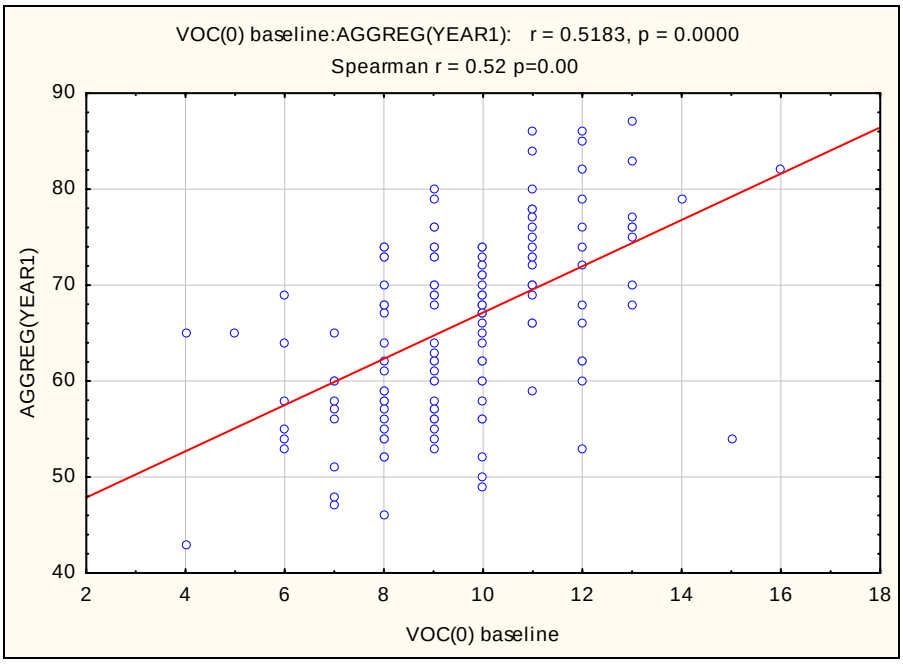

Figure 4.2 WISC III Vocabulary Subtest and the Academic Aggregate Correlation

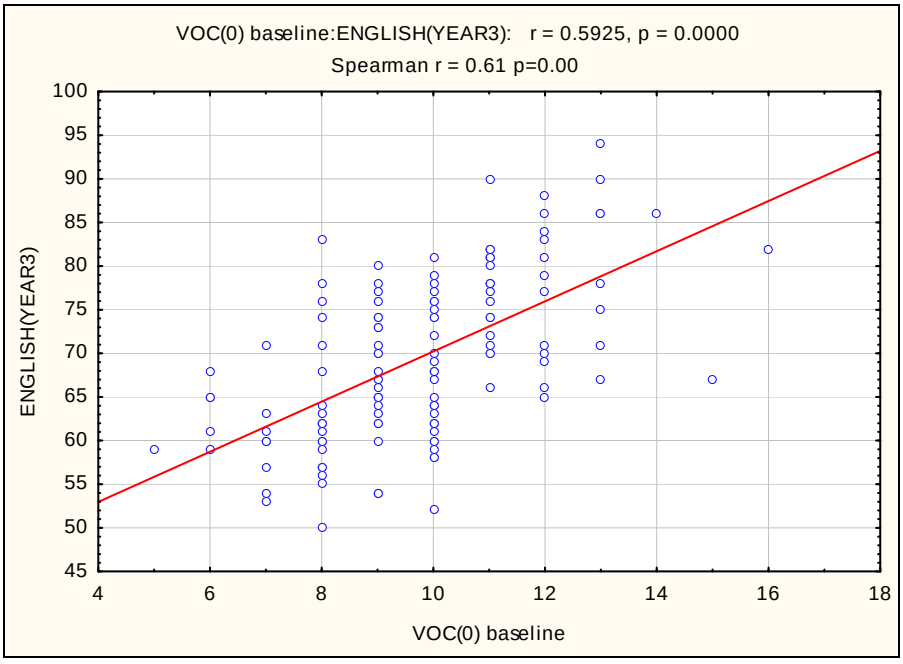

Figure 4.3 WISC III Vocabulary Subtest and the English Correlation

4.2.2. REPEATED MESURES ANOVA

The second set of objective measures are presented as group mean comparisons across all groups on all neuropsychological data and academic data for *total concussed players versus total non-concussed players; total rugby players versus total non-contact sport controls; and for compromised versus non compromised participants over time*. Further levels of analyses also investigated the role of the VOC as a covariate in the analyses to examine the outcome within the context of brain reserve capacity.

4.2.2.1. Concussion Measures

This included analyses across the entire group to establish the impact of MTBI's on cognitive and academic measures. The data were examined across the levels: Any concussion; Concussion 1 (0-1 versus 2-3 concussions) and the number (#) of concussions i.e. (0, 1, 2, 3). The numbers of participants falling in the different concussion categories across the cognitive and academic subtests (Table 4.5) were as follows:

Table 4.5 Number of participants falling into each concussion category in terms of cognitive and academic assessment

Levels of Analyses	Cognitive (N=111)	Academic (N=136)
Any concussion		
Yes	n= 64	n= 70
No	n= 47	n= 66
Concussion 1		
0-1	n= 81	n= 105
2-3	n= 30	n= 31
# of concussions		
0	n= 47	n= 66

1	n= 34	n= 39
2	n= 22	n= 23
3	n= 8	n= 8

The cognitive column in Table 4.5 represents the participants that completed the neuropsychological test battery and the academic group represents the participants that completed the regular compulsory school assessments.

The impact of **Any Concussion** on the participants' performance on a range of cognitive and academic test scores was analyzed using Repeated Measures ANOVA. The measures included all the participants as indicated in Table 4.5.

Comparisons were made between **the group with no reported concussion and the group with reported concussions** across the subtests CD, CI, COD, DET, SIM, VPAI, VPAII and Academic Test scores AGG, ENG and MATHS over time from baseline through 3 post-test years.

Table 4.6 Results of Concussions versus Controls

	n Concussed	**n Controls**	**Time**	**Group**	**Combined**	***d* Group**
CD	64	47	0.6	0.4	0.3	1.2
CI	64	47	0.3	0.2	0.05	1.95
COD	64	47	<0.01*	0.7	0.7	0.6
DET	64	47	<0.01*	0.8	0.1	-0.46
SIM	64	47	0.1	0.1	0.8	2.2
VPAI	64	47	<0.01*	0.7	0.03**	0.39
VPAII	64	47	<0.01*	0.7	0.04**	0.42
AGG	70	66	<0.01*	0.8	0.8	0.5
ENG	70	66	<0.01*	0.3	0.5	1.6
MATHS	70	66	<0.01*	0.6	0.4	0.76

Cognitive = (CD, CI, COD, DET, SIM, VPAI, VPAII); Academic = (AGG, ENG, MATHS)
* denotes significance $p<0.05$
** denotes significance $p<0.05$ in the opposite direction.

Subtests that showed no significance in terms of combined and group effect but rather in time effects were **COD** (F_{05} (3, 327) = 9.43; p < 0.01 (time))**,** DET (F_{05} (3, 327) = 190.55; p <0.01(time)) (Fig 4.4), **AGG** (F_{05} (3, 402) = 5.29; p <0.01(time))**, ENG** (F_{05} (3, 402) = 55.80; p<0.01 (time)) and **MATHS** (F_{05} (3, 402) = 3.76; p=0.01 (time))**.** In terms of effect size for time see Section 3.2 and Fig 3.2.

The **VPAI** subtest (Fig 4.5), showed a statistically significant **time effect VPAI** (F_{05} (3,327) = 60.94; p <0.01(time)) as well as a **combined effect** for time and concussion**,** p=0.03(interaction) but no group effect. A statistically significant **combined effect** for **VPAII** (F_{05} (3,327) = 2.87; p=0.04 (interaction)) and **time effect** (F_{05} (3,327) = 9.83; p< 0.01 (time)) was noted but no **group effect** p=0.77 (any concussion). Subtests that showed no significance in terms of combined, time and group effects were **CD, CI** and **SIM.**

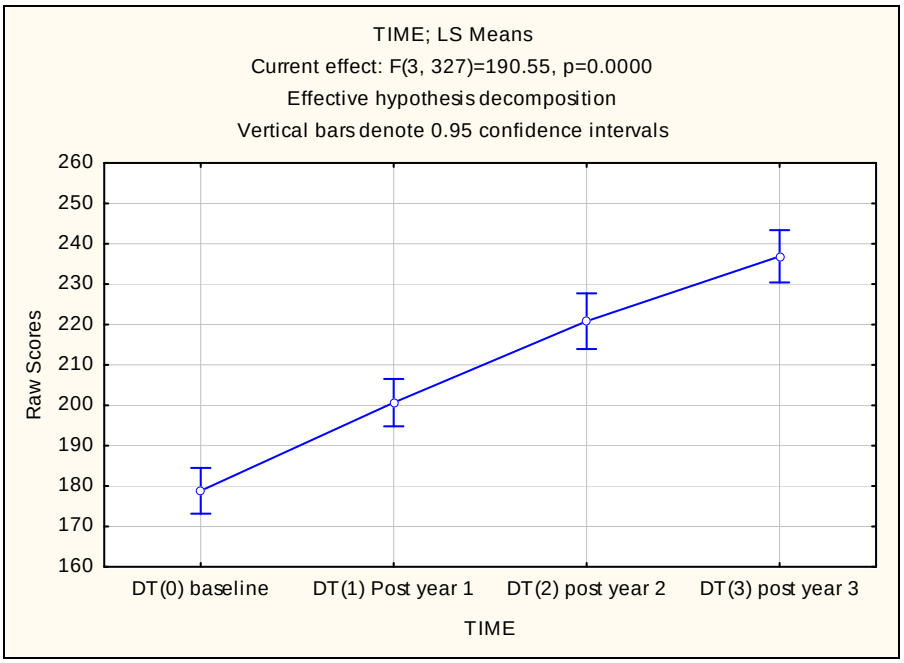

Figure 4.4 Detroit Motor Speed and Precision Test time effect

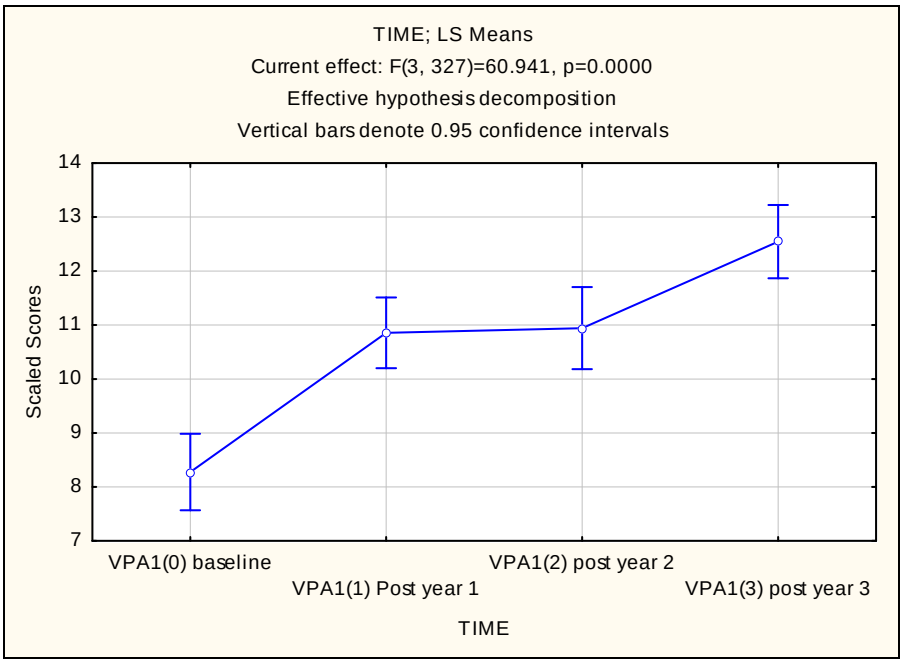

Figure 4.5 Wechsler Memory Scale Verbal Paired Associates Immediate Recall time effect

As with the analyses for "Any concussion" the same process was followed for **0-1 versus 2-3 Concussions** termed "Concussion 1" on the participants' performance on a range of cognitive and academic test scores using Repeated Measures ANOVA. Results in this section comprised comparisons between the group with zero to one reported concussion and the group with 2-3 or multiple reported concussions on the various subtests. The number of participants (n) in the cognitive analyses were 0-1 concussions (n=81) and 2-3 concussions (n=31). The participant numbers for the academic categories were 0-1 concussions (n=105) and 2-3 concussions (n=31). Subtests that showed no significant combined or group effect but time effects were the **COD** p<0.01 (time); **DET** p <0.01 (time)**; VPAI** p <0.01 (time)**, VPAII** p <0.01 (time)**; AGG** p <0.01 (time)**, ENG** p = 0.01 (time) **and MATHS** p = 0.01 (time). The findings were similar to the first set of analysis, "any concussion" but unlike that analysis VPAI and VPAII had no combined

effect. Analyses also showed the three subtests CD, CI and SIM as not significant at all three levels of analysis (combined, group and time).

Additional analyses investigated the impact of **0 versus 1 versus 2 versus 3 Concussions** "number (#) of concussions" on the participants' performance on cognitive and academic test scores using Repeated Measures ANOVA. These results were similar to those already presented and discussed above. The group with three concussions consisted of eight participants as opposed to zero concussions (n=66), one concussion (n=39), two concussions (n=23). The subtests that showed no significance in terms of combined, time and group effects were **CD, CI** and **SIM.** Subtests that showed no significant combined or group effect but a time effect were, as with the previous analyses, the **COD** $p<0.01$ (time); **DET** $p<0.01$ (time)**; VPAI** $p<0.01$ (time)**, VPAII** $p<0.01$ (time)**; AGG** $p<0.01$ (time)**, ENG** $p<0.01$ (time) and **MATHS** $p=0.01$ (time).

An investigation of the difference in **VOC** scores between participants with three concussions and those with zero concussions revealed a significant difference between three concussions ($n = 8$, ($M = 8.6$, $SD = 1.19$)) and zero concussions $n = 47$ ($M= 10.5$, $SD = 1.92$), F_{05} $(1, 54) = 6.92$; ($d = 1.26$) with a power (using the harmonic mean = 13.67) of 0.90 at significance level $p = 0.05$.

In **summary** in terms of the 'Concussion' analyses the results that were significant were for the time effects in **COD, DET, AGG, ENG, MATHS, VPA I and VPA II** subtests. The only tests not significant in terms of time were CD, CI and SIM. There was also a significant difference in the VOC scores between participants with no concussions and those with 3 or more concussions.

4.2.2.2. Rugby Measures

These measures included an analysis of the VOC scores between the rugby and the non-rugby groups and a global level of analysis of scores on individual subtests (cognitive and academic) of the rugby groups across time. A one way ANOVA analysis of the **VOC** data scores at baseline between rugby players (n= 66, M(SD) = 9.2(0.2)) and those who previously played/never played, (n= 70, M(SD) = 10.2(0.2)) revealed (VOC F_{05} (1, 134) = 9.53; p =.002), (d = 4.5) with a power > 1 at .05 significance Alpha for Two-Tailed Test) suggesting a significant difference between the scores of the two groups (Fig 4.6) with the rugby players scoring significantly lower than players that had never played rugby and players that had previously played rugby.

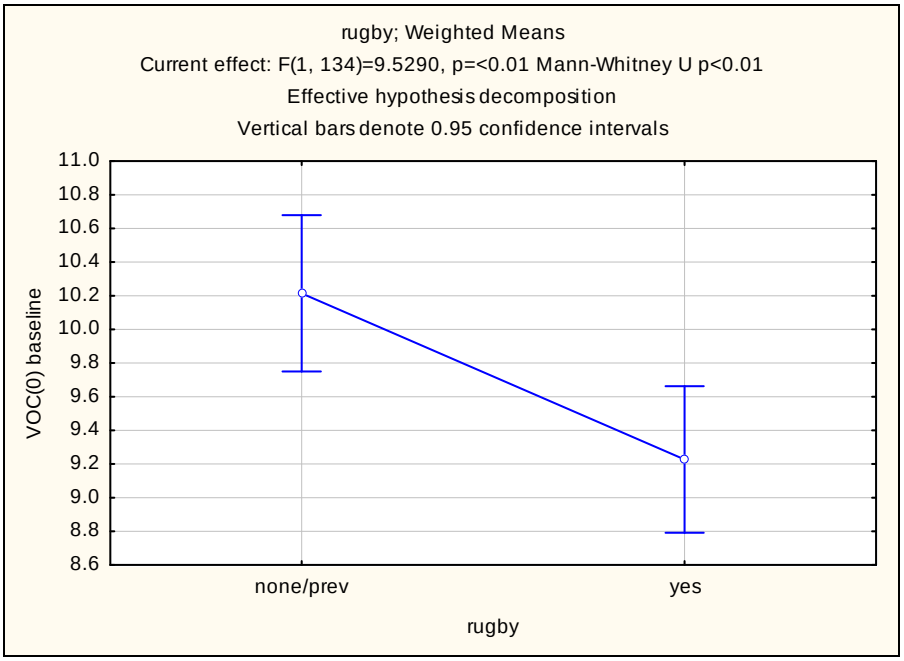

Figure 4.6 WISC III Vocabulary subtest group effect

In terms of the global analysis, the **no rugby and previous rugby groups were compared with the current rugby group,** (rugby) on a range of cognitive and academic test scores using

Repeated Measures ANOVA. The numbers of participants for the cognitive tests category were no rugby and previous rugby (n=55) and (n=56) for current rugby whilst the academic numbers were no rugby and previous rugby (n=70) and (n=66) for current rugby.

The results show a statistically significant difference between the combined previous and no rugby scores (n=55, M (SD) = 10.2(0.21)) and the current rugby group (n= 56, M (SD) = 9.5(0.21)), for **SIM** (F_{05} (1,109) = 4.6; p=0.03 (rugby)) (*d* = 3.0) (Table 4.7) (Fig 4.7). Subtests that showed no significant combined or group effect but time effects were the **COD** (F_{05} (3,327) = 10.28; p<0.01 (time)); **DET** (F_{05} (3,327) = 250.80; p <0.01 (time))**; VPAI** (F_{05} (3,327) = 66.01; p <0.01 (time))**, VPAII** (F_{05} (3,327) = 11.47; p <0.01 (time))**; AGG** (F_{05} (3,402) = 5.50; p <0.01 (time))**, ENG** (F (3,402) = 55.59; p = 0.01 (time)) and **MATHS** (F (3,402) = 3.99; p = 0.01 (time)) (Table 4.7). There was a trend between the current rugby group and the control group albeit not statistically significant for **VPAII** (F_{05} (1, 109) = 3.46; p = 0.06) and **AGG**$_{05}$ (F (1, 134) = 2.93; p=0.08 (rugby)) (Fig 4.8) (See discussion for comment of inclusion of the latter).

Table 4.7 Results of Rugby versus Controls

	N Rugby	**n Controls**	**Time**	**Group**	**Combined**	***d* Groups**
CD	56	55	0.5	0.1	0.2	2.2
CI	56	55	0.2	0.06	0.06	2.6
COD	56	55	<0.01*	0.9	0.9	0.16
DET	56	55	<0.01*	0.9	0.9	0.06
SIM	56	55	0.07	0.03*	0.9	3.0
VPAI	56	55	<0.01*	0.2	0.1	2
VPAII	56	55	<0.01*	0.06	0.2	2.9
AGG	66	70	<0.01*	0.08	0.1	2.4
ENG	66	70	<0.01*	0.1	0.5	2.17
MATHS	66	70	<0.01*	0.2	0.2	1.85

Cognitive = (CD, CI, COD, DET, SIM, VPAI, VPAII); Academic = (AGG, ENG, MATHS)
*denotes significance p <0.05

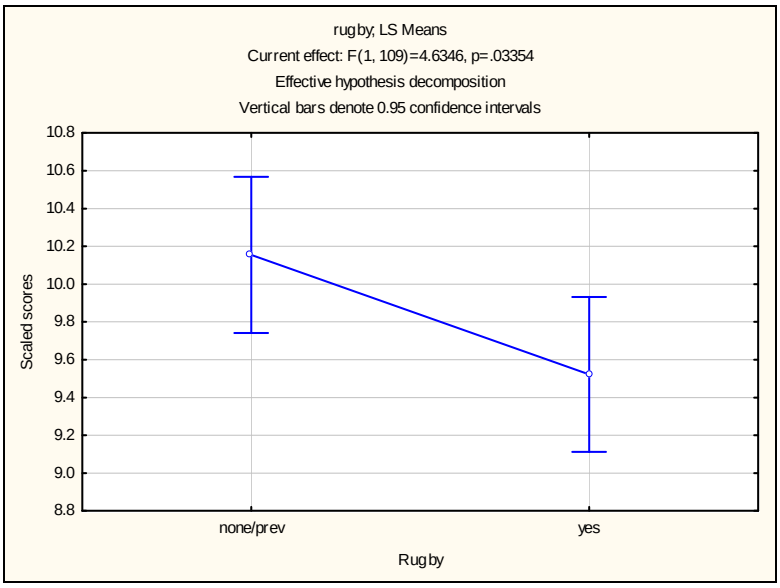

Figure 4.7 WISC III Similarities Subtest group effect (two 'rugby' groups – current rugby/previous versus no rugby)

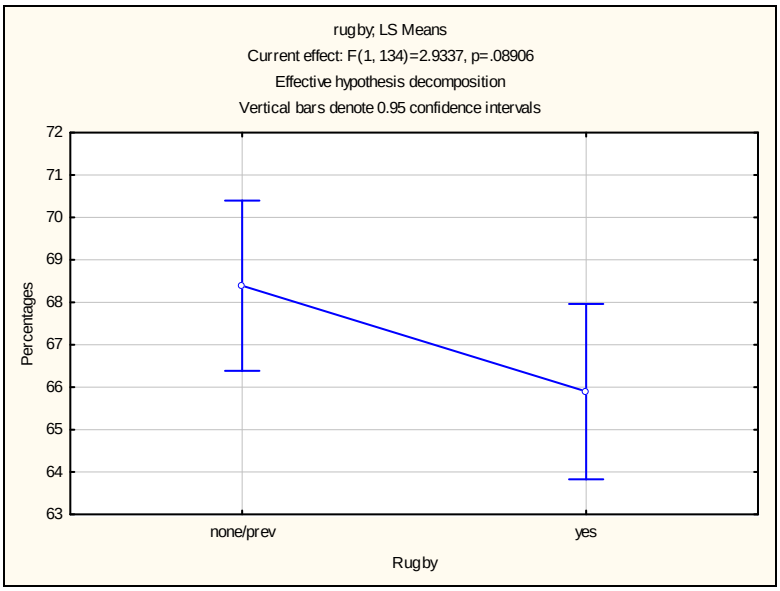

Figure 4.8 Academic Aggregate group effect (two 'rugby' groups – current rugby/previous versus no rugby)

Analysis of **<u>0 rugby; previous rugby and current rugby,</u>** (rugby 3) on cognitive and academic test scores respectively using Repeated Measures ANOVA with (n=24) no rugby; (n=31)

previous rugby and (n=56) for current rugby cognitive measures and (n=30) no rugby; (n=40) previous rugby and (n=60) for current rugby academic measures revealed no significant difference between the groups in terms of the combined effect (interaction), time effect (time) and group effect (rugby 3) for **CI and CD, MATHS and SIM** scores (See Appendix O for graphic presentation on non significant combined effects). However, on further scrutiny there was unlike the previous analysis only a trend for **SIM** (F (2,108) = 2, 74); p=0.07 (rugby 3) for current players to score lower than both none and previous players (Fig 4.9) albeit not significant.

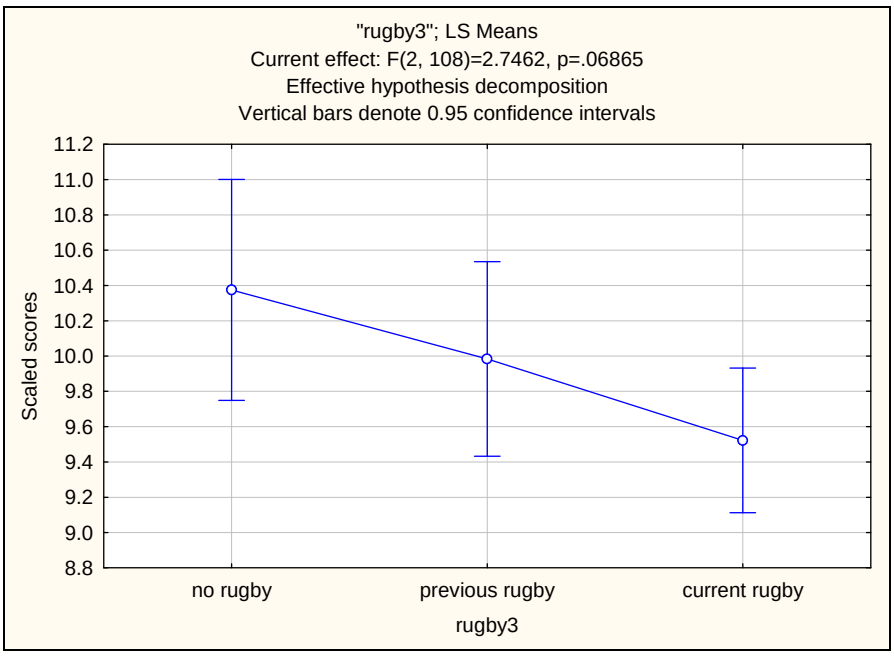

Figure 4.9 WISC III Similarities Subtest group effect (three 'rugby' groups – continuous rugby, previous and no rugby)

As with the previous analyses, subtests that showed no significant combined or group effect but a time effect were the **COD, DET, VPAI, VPAII, AGG and ENG** ($p < 0.01$). The only cognitive tests not significant in terms of time were CD, CI, and SIM.

In **summary**, results showed that the analysis of baseline VOC scores for rugby players as opposed to non rugby and previous players yielded significant results VOC $p<0.01$ showing that rugby players scored significantly lower than controls. Significant time effects were evident on **COD, DET, VPAI, VPAII, AGG and ENG** analysis. The non-statistically significant finding in terms of time effects for SIM was considered significant in the light of expected practice effects. Analysis combining the rugby and previous rugby group showed SIM $p=0.03$ (rugby) (Fig 4.7)

4.2.2.3. Compromised Measures

Under the heading of Compromised Measures there were two phases to the analysis. The **first** phase, the **global** approach, examined compromised measures under the heading **'Learning Difficulty' versus 'Non-Learning Difficulty'** as well as the impact of concussion on this group. The **second** more **specific** phase examined compromised measures under the heading 'P**roblems' versus 'Non-Problems'** which included the learning and non-learning difficulty groups and incorporated 'concussion', 'rugby' 'rugby and concussion' and 'rugby and multiple concussions' groups. Analysis of the 'problem' data, the **second** phase of the analysis provides more **specific** analysis and is discussed later under the headings **Combined Compromised and Concussion Measures, Combined Compromised and Rugby Measures, Combined Compromised Concussions and Rugby Measures, Combined Compromised Multiple Concussions and Rugby Measures**

The 'learning difficulty' (n=23) analysis included four participants reportedly with diagnosed learning disabilities, thirteen with diagnosed Attention Deficit (with or without) Hyperactivity Disorder (ADHD/ADD) and six reported grade failures. The data revealed eight participants in

the learning disability group however, four of those were also accounted for in the ADHD group. Of the thirteen participants with reported ADHD, twelve were on medication and two reported co-morbid anxiety/depression. Of the six participants that reported grade failure zero reported learning disabilities or ADHD.

The **learning difficulty** (n=23) versus **non-learning difficulty** (n=88) cognitive and (n=26 & n=110) academic analysis was the third arm of the global level of analysis that examined the impact of learning difficulties on participants' scores on cognitive and academic tests over time. The data was analyzed using Repeated measures ANOVAS.

Table 4.8 Results of Compromised versus Controls

	n Compromised	**n Controls**	**Time**	**Group**	**Combined**	***d* Group**
CD	23	88	0.4	0.3	0.8	1.6
CI	23	88	0.4	0.6	0.9	0.68
COD	23	88	<0.01*	0.4	0.9	1.16
DET	23	88	<0.01*	0.7	0.6	0.6
SIM	23	88	0.1	0.8	0.8	0.38
VPA1	23	88	<0.01*	0.03*	0.6	3.3
VPA11	23	88	<0.01*	0.3	0.4	1.5
AGG	26	110	0.03	0.002*	0.5	4.6
ENG	26	110	<0.01*	0.01*	0.4	3.9
MATHS	26	110	<0.01*	0.002*	0.3	4.6

Cognitive = (CD, CI, COD, DET, SIM, VPAI, VPAII); Academic = (AGG, ENG, MATHS)
*denotes significance p <0.05

Results showed no significant combined, time and group effects for the **CD, CI and SIM** scores and no significant combined and group effects but significant time effects for **COD** (F_{05} (3,327) = 6.41; p<0.01 (time)); **DET** (F_{05} (3,327) = 156.17; p <0.01 (time))**; VPAII** (F_{05} (3,327) = 5.47; p <0.01 (time))**;** (time)) There was no significant combined effect of time and learning difficulties for **VPAI, AGG, ENG** and **MATHS** but significant group effects for **VPAI** (F_{05} (1,109) = 5.01; p=0.03 (learning difficulty)) (Fig 4.10); **AGG** (F_{05} (1,134) = 9.47; p<0.01 (learning difficulty))

(Fig 4.11); **ENG** (F_{05} (1,134) = 6.63; p=0.01 (learning difficulty)) (Fig 4.12); **MATHS** (F_{05} (1,134) = 9.24; p<0.01 (learning difficulty)) (Fig 4.13) (Table 4.8) as well as time effects for (**VPAI** (F_{05} (3,327) = 66.01; p<0.01 (time); **AGG** (F_{05} (3,402) = 2.99; p=0.03 (time); **ENG** (F (3,402) = 39.56; p< 0.01 (time); and **MATHS** (F (3,402) = 3.84; p< 0.01 (time).

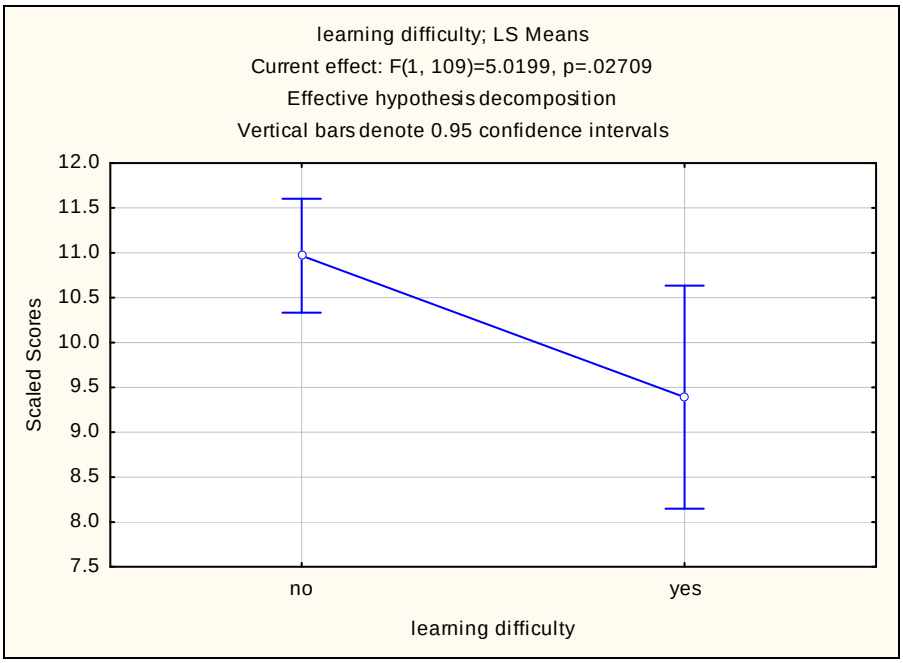

Figure 4.10 Verbal Paired Associates Immediate Recall difference between the learning difficulty group and the non-learning difficulty group

In terms of the impact of **learning difficulties versus non-learning difficulties and concussion** on the participant's performance on a range of cognitive and academic test scores over time, the analysis using Repeated measures ANOVA showed similar findings to the results discussed above under the heading **learning difficulties versus non-learning difficulties** in particular the test categories **VPAI, AGG, ENG and MATHS.** There was however no combined effect between difficulties and concussion. In terms of the concussion groups only a trend was visible on the **SIM** (F (1,107) = 3.2; p=0.07) subtest. On the whole reports of concussions sustained by

the different groups did not significantly impact on performance across the range of subtests and academic scores.

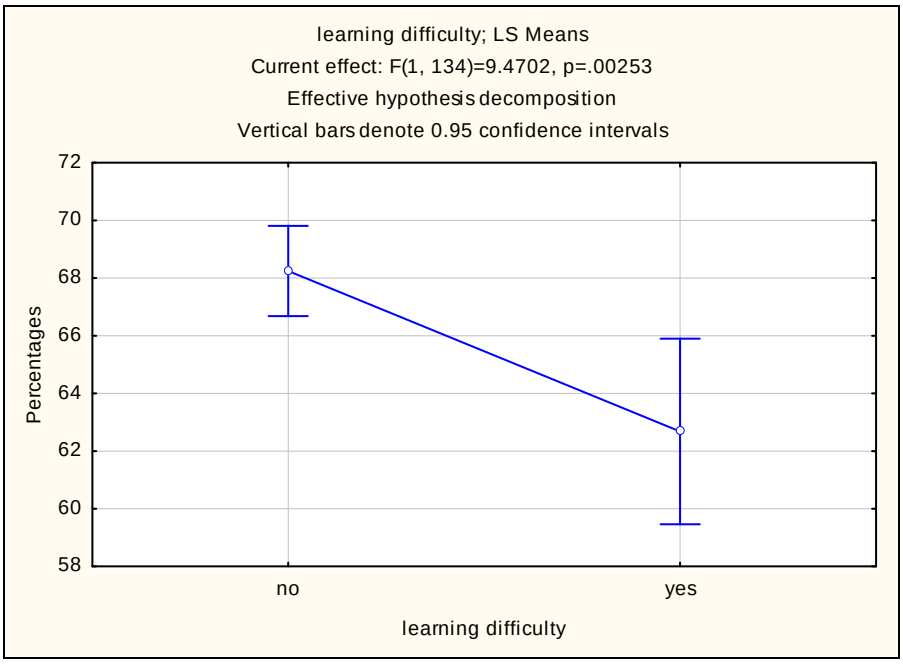

Figure 4.11 Academic Aggregate differences between the learning difficulty group and the non-learning difficulty group

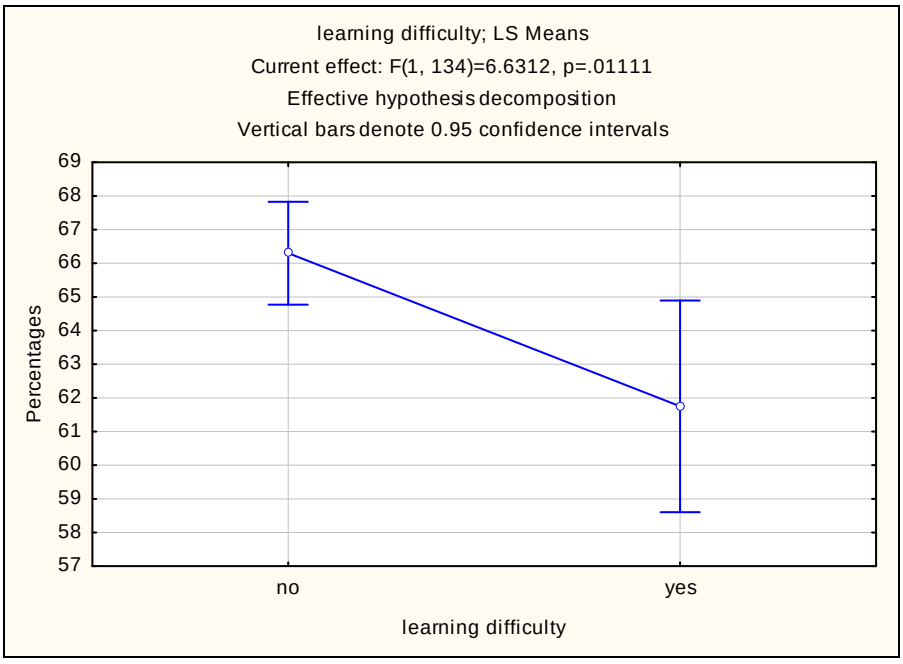

Figure 4.12 English differences between the learning difficulty group and the non-learning difficulty group

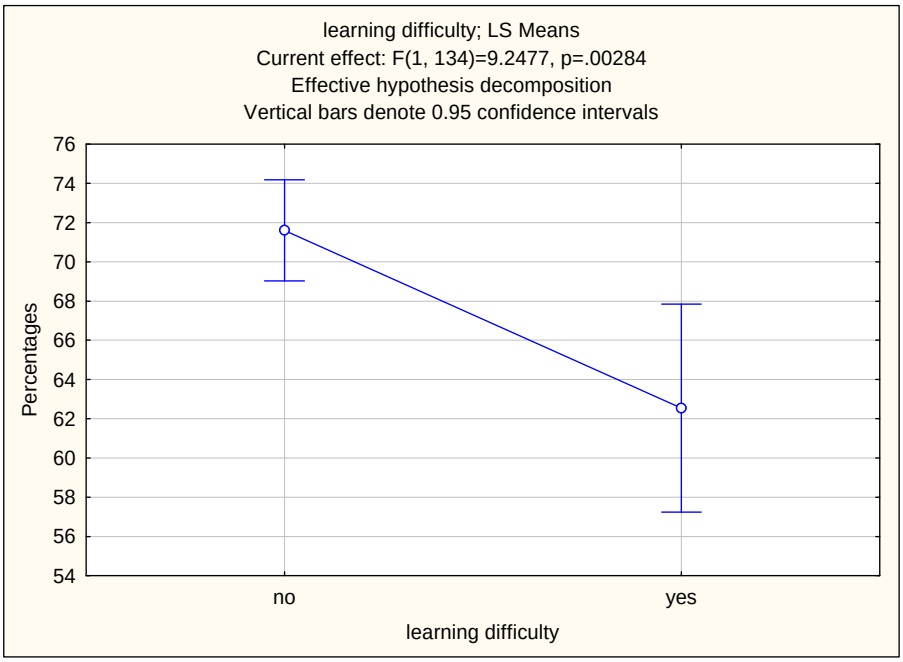

Figure 4.13 Mathematics difference between the learning difficulty group and the non-learning difficulty group

4.2.2.4. Combined Compromised and Concussion Measures

This analysis compared the compromised group (problems) versus non-compromised groups in terms of concussion versus non-concussion across time, taking into consideration the effect of the co-variant **VOC** on the data.

The "problems" group (n=36) incorporated the learning difficulty group (n=23) which included learning disability, ADHD and previous grade failures as well as participants that reported encephalitis/ meningitis (n=6), epilepsy/ seizures (n=2), depression and or anxiety (n=1 participant treated for depression) of the eight recorded because seven of the eight participants were already accounted for by five no medication and two ADHD where medication taken by the

latter two participants were for ADHD and not depression, previous grade 3 concussions (n= 4) (the other four accounted for by one grade failure, one ADHD, one meningitis and one depression) and those reportedly on medication. In terms of the medication where (n=15), twelve participants were already accounted for in the ADHD segment of the learning disability group. Two participants received medication for epilepsy/seizures and one for depression/anxiety.

Examination of the non-compromised group's data according to VOC categories showed that in the above average group (n = 23), (VOC ≥ 12) four of the "current" rugby players (n=6) reported zero concussions and two, two concussions. Amongst the non rugby players (n=10) nine reported zero concussions and one, one concussion whereas three of the "previous" players (n=7) reported one concussion and four zero concussions (Figure 4.14). Only the current rugby players reported multiple concussions.

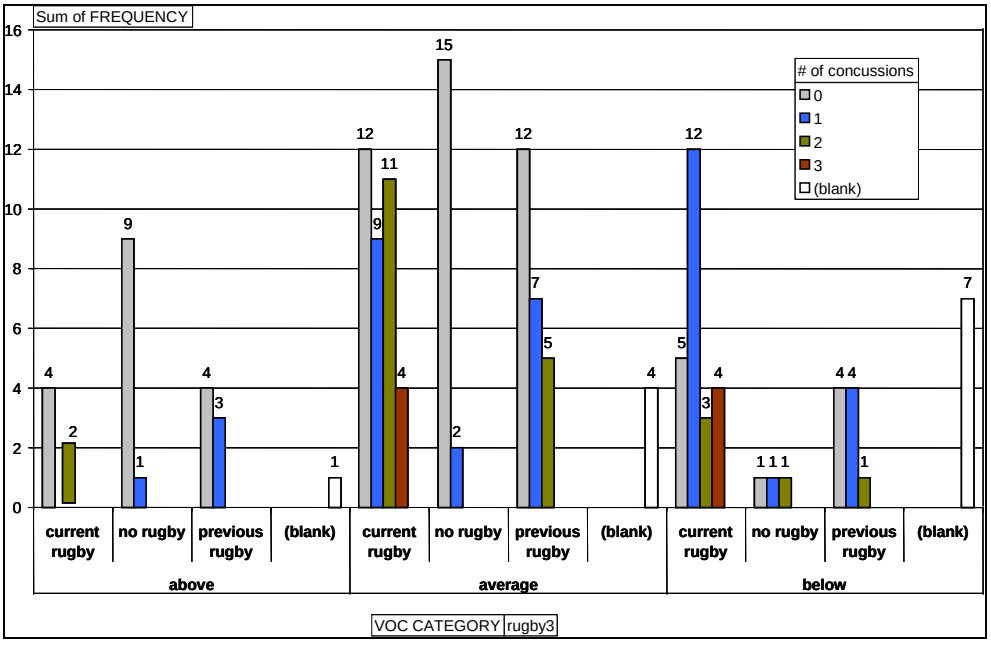

Figure 4.14 Non-compromised group's data in terms of the IQ equivalent VOC scores, showing numbers of reported concussions for 'rugby' playing groups

In the average category (VOC 9-11), (n= 77) of the "current" players (n=36), twelve reported zero concussions, nine one concussion, eleven two concussions and four players three or more concussions. In the non rugby group (n= 17) fifteen participants reported zero concussions and two one concussion and in the "previous" group (n=24) twelve reported zero concussions, seven one concussion, five two concussions (Figure 4.14).

In the below average group (n=35), (VOC ≤ 8) five of the "current" group (n=24) reported zero concussions, twelve one concussion, three two concussions and four three or more concussions. In the non-rugby group (n=3) there was one person that reported zero concussion, one one concussion and one two concussions and there were seven blank responses (See Figure 4.14). In the "previous' group four persons reported one concussion and one person, two concussions.

The data of the compromised group (problems) versus non-compromised groups in terms of concussion versus non-concussion across time, taking into consideration the effect of the co-variant **VOC** was analyzed in terms of **VOC**; "concussion" "problems" and "time" as well as the interaction between "concussion" and "problems" (see Table 4.9); "time" and "concussion"; "time" and "problems"; "time", "concussion" and "problems" and between "time" and **VOC**.

Table 4.9 Number of participants with any concussion- with or without "problems"

	Problems: yes	Problems: no
Any concussion: yes	N= 19	N= 45
Any concussion: no	N= 17	N= 30

Results of the analysis of the effect of VOC on participants' responses showed significant VOC effects for: **CD** (F_{05} (1,106) = 5.63**;** p=0.02 (VOC (0) baseline)) and **SIM** (F_{05} (1,106) = 44.43)**,** **VPA I** (F_{05} (1,106) = 18.47)**, VPA II** (F_{05} (1,106) = 9.91)**, AGG** (F_{05} (1,131) = 46.34)**, ENG**

(F_{05} (1,131) = 74.24) and **MATHS** (F_{05} (1,131) = 32.94) where for all subtests p <0.01 (VOC (0) baseline). The significant CD effect is not in keeping with the findings from the correlation analysis discussed earlier whilst the effect for the other subtest is i.e. SIM, AGG and ENG showed a strong positive relationship and MATHS, VPA I and VPA II a moderate relationship. There were significant group effects for: **VPA1** (F_{05} (1,106) = 4.08; p=0. 04 (problems)) where compromised (n=36) and non-compromised (n=75); **AGG** (F_{05} (1,131) = 4.87; p=0.03 (problems)); **ENG** (F_{05} (1,131) = 4.72; p= 0.03 (problems)) and **MATHS** (F_{05} (1,131) = 6.56 p=0.01 (problems)) where compromised (n=40) and non-compromised (n=96) with the effect of the co-variant VOC on the data, factored in. With the effect of the co-variant **VOC** on the data factored in, no significant effects were evident when the concussion variable was added only a trend albeit not significant was observed for the subtest **AGG** (F_{05} (1,131) = 3.08; p=0.08 (concussion)). In this concussion group analysis "problems" i.e. the comparisons between the compromised and non compromised groups, was a significant effect but there was no significant interaction between "time", "concussion" and "problems". Significant time effects were in keeping with previous analysis already discussed with the exception of SIM (F= (3,318) 2.78; p=0.04(time)).

4.2.2.5. Combined Compromised and Rugby Measures

More specific analysis compared the compromised group versus the non-compromised group (problems) across three levels of rugby (no; previous; current) taking into consideration the effect of the co-variant **VOC** on the data. The data were analyzed in terms of **VOC**; "rugby 3" "problems" and "time" as well as the interaction between "rugby 3" and "problems"; "time" and

"rugby 3"; "time" and "problems"; "time", "rugby 3" and "problems" and between "time" and **VOC**.

In keeping with previous analyses reported above, results of the analysis of the effect of VOC on participants' responses showed significant VOC effects for the subtests: **CD** p =0.03 (VOC (0) baseline); **SIM, VPA 1, VPA 2, AGG, ENG** and **MATHS** where for all subtests p<0.01 (VOC (0) baseline). Similar to previous findings, there were significant effects for comparisons of the compromised versus non compromised groups "Problems" for: **ENG** (F (1,131) = 5.87; p=0.01 (problems)) and **MATHS** (F (1,131) = 8.32; p <0.01 (problems)) **VPA1** (1,106) = 4.95; p=0.02 (problems) and **AGG** (F (1,131) = 8.79 p <0.01 (problems). Significant time effects were in keeping with previous analysis already discussed with the exception of **SIM** (F= (3,318) = 3.13; p=0.02 (time)).

4.2.2.6. Combined Compromised Concussion and Rugby Measures

This level of analysis compared the **compromised** group and the **non compromised** groups "problems" in terms of **"no rugby" and "no concussion"** versus **"current rugby" and "concussion"** across **time** in the context of **VOC as the covariant**. The data at this level was analyzed in terms of VOC; "problems", "time" and "rugby/concussion groups" as well as the interaction between "rugby/concussions" and "problems"; "time" and rugby/concussions"; "time" and "problems"; "time" and VOC and "time", rugby/concussions" and "problems". There were significant outcomes for **VPA II** p=0.04 (VOC (0) baseline); **SIM, VPA I, AGG, ENG** and **MATHS** where for all subtests p<0.01 (VOC (0) baseline). These findings were similar to findings mentioned earlier in terms of covariance except for the VPA II findings and the lack of

CI significant results. There were significant time effects for **SIM** (F (3,168) =3.2; p = 0.03 (time)).

4.2.2.7. Combined Compromised Multiple Concussions and Rugby Measures

This analysis compared the **compromised** group (problems) and the **non-compromised** group in terms of the **no rugby and no concussion** and **rugby with two or more concussions** across **time** in the context of **VOC as the covariant**. Of the eight boys that had a grade 3 concussion prior to the study there were none that fell into the three or more concussion category. Results of analysis of the compromised group versus the non-compromised group across the no rugby and no concussion and rugby with two or more concussions levels over time with **VOC** as the covariant, showed significant findings for: **SIM, VPA I, AGG** and **ENG** where for all subtests $p<0.01$ (VOC (0) baseline). These findings were similar to findings mentioned earlier in terms of covariance with the exception of **VPA II, MATHS** and **CI** significant results. Results of the interaction between compromised, rugby/many concussion groups and their "non-rugby non - concussion" counterparts were not significant. Results showed that when **VOC** was factored into the analysis of the non-compromised group there was a significant difference between the scores of the control group with no concussions and the rugby group with many concussions over time **AGG** (F (3.132) = 3.6; p= 0.02) (*d* = 2.1)(Time* rugby/ many concussions groups) (Figure 4.15). The number of participants for the no rugby/ no concussion group for academic measures (**AGG**) was (n=25) and for the current rugby/ 2-3/ more concussions group (n=24). In terms of academic performance the results show that in this cohort the rugby players had a higher score at baseline but after three years the scores were below the level of the controls. There were no significant

results for time effects across any of the cognitive and academic measures and the scores of the controls had increased.

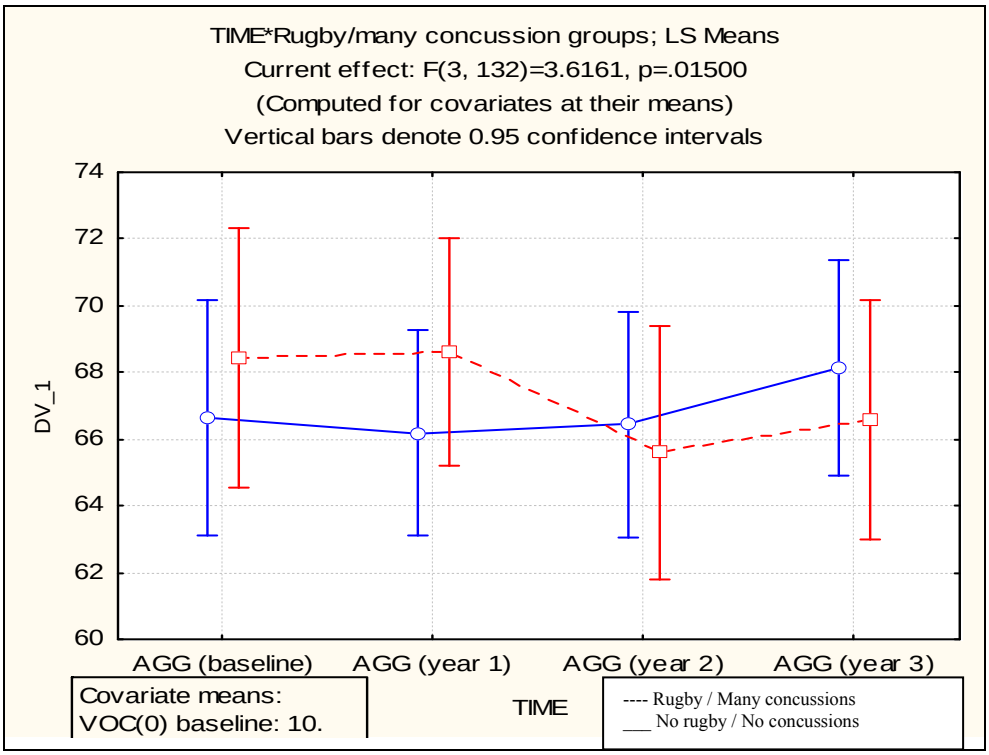

Figure 4.15 Time* rugby/ many concussions groups

In terms of the interaction of the 'Time* rugby/ many concussions' groups analysis there were no significant findings for MATHS, ENG, VPAII, SIM, DET, CODING, CD, CI but a trend for VPAI albeit not significant.

In **summary,** in terms of the *global* analysis of 'learning difficulties' results showed significant group effects for **VPAI** p=0.03 (learning difficulty) (Fig 4.19); **AGG** p<0.01 (learning difficulty) (Fig 4.20); **ENG** p=0.01 (learning difficulty) (Fig 4.21); **MATHS** p<0.01 (learning difficulty) (Fig 4.22) but when the concussion variable was added there were no significant differences between the groups. In terms of the more *specific* analysis the 'compromised' analysis with VOC as the covariate across the 'Concussion', 'Rugby', 'Concussions and Rugby' and 'Multiple Concussions and Rugby' measures across time, significant findings between the groups were for

VPAI, AGG, MATHS and ENG. These results were similar to the results obtained in the 'learning difficulty' analysis. Results supporting a significant relationship between the VOC and neuropsychological/cognitive test and academic scores were supported by significant results $p<0.01$(VOC (0) baseline) for SIM, VPAI, AGG, ENG and MATHS at the combined 'rugby and concussion' level but at this level there was also a significant result for VPAII $p=0.04$ (VOC (0) baseline) which is contrary to the earlier global analysis. In the separate 'concussion' and 'rugby' analysis CD $p =0.03$ (VOC (0) baseline) also showed a significant result which is not in keeping with the previous global findings which showed no relationship with VOC. At this level of analysis with VOC as covariate the SIM result was significant in terms of results over time for **SIM** $p=0.04$ (time) 'concussion', **SIM** $p=0.02$ (time) 'rugby' and **SIM** $p = 0.03$ (time) combined 'rugby and concussion'. The 'concussion' and 'rugby' results contradict the results obtained at a global level of analysis. In terms of the interaction between time, rugby and many concussions with the effects of the covariate VOC taken into account, the only significant result was **AGG** $p=0.02$ ($d = 2.1$) (Time* rugby/ many concussions groups) (Figure 4.15).

4.3. Descriptive Measures

In the functional areas of visual memory, abstract thinking and academics, the objective cognitive and academic testing showed consistent support for greater numbers of rugby players showing deficits than previous rugby players and non-playing controls (Table 4.10,4.11,4.12,4.13). On tests of visual memory CI, CD, the CD test (Table 4.10) showed that at baseline the number of non rugby players showing deficits were fewer than for previous and current rugby players and that by the third year the number of previous players showing deficits had also decreased in relation to the rugby players but the numbers showing deficits remained more than the number of

the non rugby players. On the CI test (Table 4.11) the number of previous players at baseline showing deficits was more in keeping with the non rugby players where fewer numbers showed deficits compared with rugby players. These results were similar after three years.

Table 4.10 Percentages of cognitive deficits (-1SD in the direction of poor performance). Results reflecting Rugby> Controls
Coding: Incidental Recall - Delayed

CD	-1 std dev	std dev	+1 std dev
baseline non rugby	11.1%	88.9%	0.0%
3rd year non rugby	7.1%	92.9%	0.0%
baseline current rugby	26.1%	73.9%	0.0%
3rd year current rugby	25.6%	74.4%	0.0%
baseline previous rugby	22.6%	77.4%	0.0%
3rd year previous rugby	13.0%	87.0%	0.0%

Table 4.11 Percentages of cognitive deficits (-1SD in the direction of poor performance). Results reflecting Rugby> Controls
Coding: Incidental Recall - Immediate

CI	-1 std dev	std dev	+1 std dev
baseline non rugby	11.1%	88.9%	0.0%
3rd year non rugby	7.1%	92.9%	0.0%
baseline current rugby	28.3%	71.7%	0.0%
3rd year current rugby	23.1%	76.9%	0.0%
baseline previous rugby	12.9%	87.1%	0.0%
3rd year previous rugby	8.7%	91.3%	0.0%

The SIM test (Table 4.12) showed that at baseline the numbers of non-rugby and previous players showing deficits were less than for rugby players. Although for the former two groups the numbers of participants showing deficits decreased by the third year more so for non rugby as

opposed to the previous rugby group, the deficits for the rugby players increased. These findings need to be considered in the context of cognitive development and in particular development of executive functioning at a critical stage of development. In terms of scores more than 1 standard deviation above the mean, results showed that the percentages of non rugby players was much higher than the percentages of rugby and previous rugby players.

Results of AGG showed that although the numbers of previous players showing deficits at baseline were lower than the non-rugby and current rugby groups, after three years they were in line with the numbers of non rugby participants showing deficits and the numbers of the rugby group showing deficits remained much higher than the numbers showing deficits for both groups (Table 4.13). Furthermore, in terms of the numbers of participants performing better than the norm group (more than one standard deviation above the norm), the percentage of non-rugby players remained higher than both the rugby and previous rugby groups.

Table 4.12 Percentages of cognitive deficits (-1SD in the direction of poor performance). Results reflecting Rugby> Controls
WISC III Similarities

SIM	-1 std dev	std dev	+1 std dev
Baseline non rugby	12.5%	75.0%	12.5%
3rd year non rugby	8.3%	75.0%	16.7%
Baseline current rugby	17.4%	78.3%	4.3%
3rd year current rugby	28.2%	64.1%	7.7%
Baseline previous rugby	9.7%	87.1%	3.2%
3rd year previous rugby	8.7%	82.6%	8.7%

Table 4.13 Percentages of academic deficits (-1SD in the direction of poor performance). Results reflecting Rugby> Controls
Academic Functioning

AGGREGATE	-1 std dev	std dev	+1 std dev
baseline non rugby	12.5%	62.5%	25.0%
3rd year non rugby	12.5%	75.0%	12.5%
baseline current rugby	26.1%	63.0%	10.9%
3rd year current rugby	23.9%	73.9%	2.2%
baseline previous rugby	9.7%	71.0%	19.4%
3rd year previous rugby	12.9%	83.9%	3.2%

4.4. Individual Results / Case Study

The intention of this fourth level of analysis was to compare individual rugby players that sustained three or more concussions with controls that played no rugby and had no concussions, matched in terms of estimated BRC. However, investigation of the rugby group with three or more concussions revealed that eight participants fell into the continuous rugby i.e. "current" and three or more concussions group. Of the eight, three were in the below average IQ equivalent range, four in the average range and one in the "compromised" and below average range. There were no participants in the above average range. When no rugby and no concussion participants were matched in terms of IQ equivalent the following numbers emerged: one below average and compromised, 15 average of which six were classified as compromised and nine above average of which two were reportedly compromised. This left only nine average and seven above average non-compromised participants in that group. Furthermore participants could only be matched in terms of the average range of intellectual functioning because the non-rugby group had no below

average candidates and the rugby group had no above average candidates. This left nine no rugby no concussion participants in the average group and four rugby players in the three or more concussion group.

Despite the limitations of matching participants for further analysis, the intention of the individual analysis to prevent a small population of individuals with deficits from going undetected was pursued. Data of reported concussed rugby/previous players over the three year study period were therefore presented for further discussion. These participants either presented for intervention following a concussion or were identified via the research process as participants at risk given their score profiles. To complete the picture reports of non-contact sport controls with no reported concussions matched in terms of estimated IQ were also presented. Given the nature of the study compromised participants were not excluded from this level of discussion either. For research purposes participants were identified in alphabetical terms from A to G.

Two participants (B and C) fell in the below average category, two (E and F) in the above average category and (A, D and G) in the average category. All boys were from advantaged backgrounds and all except one had both parents at home. The boy with the single parent nevertheless had a good supportive extended family. The language of instruction was English and with one exception the home language was English. The "exception", of Asian descent, nevertheless posted the highest score for the VOC subtest. All boys were in the same grade and age range and none of these boys had previously failed a grade. One boy was not involved in rugby, two played in the backline and four were forwards. Three of the players had slots in either the first or second team whilst three had third or fourth team slots. Two boys were categorized in terms of the compromised category.

Participant A (Table 4.14) a rugby player came to the researcher's attention because his low subtest scores were inconsistent with what one would expect from a participant with an average intellect (VOC = 9), no reported difficulties and only two reported concussions. On further investigation it became apparent that the participant had indeed had more than two concussions and furthermore that he had an Attention Deficit Disorder for which he was, at the time of intervention, receiving medication. He had not to date informed the school of his difficulty for fear of being labeled and was reluctant initially to divulge this information to the researcher. This information only came to light because the researcher raised concerns with the boy's parents regarding his inconsistent scores. The parents reported that the boy was not initially on medication for the disorder but had recently started taking it to manage his difficulties. In addition he had received remedial intervention and additional academic support. Participant A played in the back line position in either the first or second team depending on the selection process. See his scores below (Table 4.14) in comparison with D (Table 4.15) and G (Table 4.16). When compared with norms (indicated in brackets c= control group norm and s.d. = standard deviation) for the control group his scores for the cognitive tests were consistently lower with the exception of the DET subtest where initially his scores were higher than the control group but after three years the score was below the norm as well. His third year academic (AGG) and English scores although largely in keeping with the norm were arguably due to remedial and pharmaceutical intervention. This however had little influence on his mathematic scores.

Table 4.14 Participant A scores versus normative data

	Pre Test	y1	Post Test 1	y2	Post Test 2	y3	Post Test 3
Concussion		Yes		No		Yes	
VOC	9 c(10.9) s.d.(1.5)						
SIM	9 c(10) s.d.(2.3)		8 c(10.6) s.d.(1.9)		9 c(10.5) s.d.(1.6)		8 c(10.7) s.d.(1.5)
VPA I	5 c(9.4) s.d.(3.1)		8 c(10.8) s.d.(3.3)		4 c(11.8) s.d.(3.3)		5 c(12.5) s.d.(2.6)
VPA II	4 c(11) s.d.(2.2)		7 c(11.4) s.d.(1.4)		2 c(12) s.d.(0.8)		2 c(11.7) s.d.(0.8)
COD	10 c(11.8) s.d.(3)		8 c(12.1) s.d.(3.1)		8 c(11.8) s.d.(3.6)		7 c(12.7) s.d.(4)
CI	5 c(7.8) s.d.(1.6)		4 c(7.1) s.d.(1.9)		5 c(7.8) s.d.(1.2)		3 c(7.8) s.d.(1.8)
CD	7 c(7.8) s.d.(1.9)		2 c(6.8) s.d.(2)		4 c(8.1) s.d.(1.1)		3 c(7.7) s.d.(1.6)
DET	189 c(167.2) s.d.(28.8)		201 c(186) s.d.(29)		218 c(216.4) s.d.(29.8)		206 c(237.5) s.d.(33.1)
AGG	60 c(68.4) s.d.(7.8)		60 c(68.1) s.d.(8.5)		62 c(68.5) s.d.(9.1)		66.9 c(70.3) s.d.(10.4)
ENG	60 c(65.5) s.d.(6.6)		55 c(66) s.d.(7)		66 c(66.6) s.d.(9.6)		67 c(72.8) s.d.(10.3)
MATHS	35 c(71.2) s.d.(10.2)		45 c(74.6) s.d.(13.9)		46 c(72.5) s.d.(13.4)		54 c(73.7) s.d.(15.2)

Participant D (Table 4.15) reported no concussions, was not compromised in any way and reported no rugby but had had some exposure to the game at age ten. He also recorded an average intellect (VOC = 9) but his scores over the years were higher than that of A. He however did not complete his participation in the third year of study as there was arguably no incentive for him to do so. This response could also tie in with a lack of motivation which is evident in his lowered

academic scores in the third year. On the VOC score D's score was lower than the control norm. His SIM subtest scores were within the norm. On the VPAI subtest he initially performed lower than the norm but then scored within the norm and for VPAII although initially lower he performed within the norm by the next test. His higher VPAII score by the second test period versus the VPAI score indicates slowed processing for auditory material but nevertheless that the information is learned and retained. The above and average performance on the COD subtest suggests an intact processing speed for visual material. Retrieval of visual information is confirmed by the performance on CI and CD in the average range indicating intact immediate and delayed recall. Performance on the DET was in the average range. D's ENG, AGG and MATHS scores were all in the normal range. See scores for D below (Table 4.15).

Table 4.15 Participant D scores versus normative data

	Pre Test	**y1**	**Post Test 1**	**y2**	**Post Test 2**	**y3**	**Post Test 3**
Concussion		No		No		No	
VOC	9 c(10.9) s.d.(1.5)						
SIM	9 c(10) s.d.(2.3)		10 c(10.6) s.d.(1.9)		11 c(10.5) s.d.(1.6)		c(10.7) s.d.(1.5)
VPA I	6 c(9.4) s.d.(3.1)		7 c(10.8) s.d.(3.3)		11 c(11.8) s.d.(3.3)		c(12.5) s.d.(2.6)
VPA II	6 c(11) s.d.(2.2)		10 c(11.4) s.d.(1.4)		12 c(12) s.d.(0.8)		c(11.7) s.d.(0.8)
COD	15 c(11.8) s.d.(3)		17 c(12.1) s.d.(3.1)		15 c(11.8) s.d.(3.6)		c(12.7) s.d.(4)
C1	9 c(7.8) s.d.(1.6)		9 c(7.1) s.d.(1.9)		8 c(7.8) s.d.(1.2)		c(7.8) s.d.(1.8)
CD	9 c(7.8) s.d.(1.9)		9 c(6.8) s.d.(2)		8 c(8) s.d.(1.1)		c(7.7) s.d.(1.6)
DET	183 c(167.2) s.d.(29)		185 c(186) s.d.(29)		202 c(216.4) s.d.(29.8)		c(237.5) s.d.(33.1)

AGG	69 c(68.4) s.d.(7.8)		68 c(68.1) s.d.(8.4)		65.8 c(68.5) s.d.(9.1)		64.8 c(70.3) s.d.(10.4)
ENG	63 c(65.5) s.d.(6.6)		70 c(66) s.d.(7)		60 c(67) s.d.(9.6)		70 c(73) s.d.(10.3)
MATHS	70 c(71.2) s.d.(10.2)		74 c(75) s.d.(13.9)		75 c(73) s.d.(13.4)		68 c(74) s.d.(15.2)

Participant G (Table 4.16) was a backline player and played either in the second or first team depending on the team's requirements. His VOC score also fell in the average intellect range (VOC = 9) it, like the other previously mentioned scores was also lower than the norm for the control group. During the research period participant G, categorized as a non compromised participant, had multiple concussions including a grade 3 concussion. Although his cognitive test scores (Table 4.16) declined following the grade three concussion, they improved a year later relative to his baseline. SIM scores however remained for the most part of the three years within the norm whereas his processing speed measured by the COD subtest remained below the norm over time suggesting slowed information processing. This result may have accounted for the decline in overall academic and mathematic scores (see comment below). VPAI, VPAII, CD, CI and DET scores within the norm suggested intact verbal and visual memory as well as motor functioning. Although the AGG and MATHS scores were within the normal range at baseline, within that range they improved in the first year but then dropped following the concussions and although at year three they were in the range they were lower than his own previous scores. Although G's ENG score increased from baseline to year three and was in the norm it remained at the low end of the norm range. An important consideration in terms of G's performance on the cognitive and academic tests is that following the grade 3 concussion he was exposed to extensive neuropsychological assessment and remedial intervention. The additional exposure to

neuropsychology test batteries would have impacted, in terms of practice effects, on his performance on the cognitive tests.

Table 4.16 Participant G scores versus normative data

	Pre Test	y1	Post Test 1	y2	Post Test 2	y3	Post Test 3
Concussion		Yes		Yes		Yes	
VOC	9 c(10.9) s.d.(1.5)						
SIM	9 c(10) s.d.(2.3)		8 c(10.6) s.d.(1.9)		10 c(10.5) s.d.(1.6)		10 c(10.7) s.d.(1.5)
VPA I	12 c(9.4) s.d.(3.1)		13 c(10.8) s.d.(3.3)		9 c(11.8) s.d.(3.3)		15 c(12.5) s.d.(2.6)
VPA II	12 c(11) s.d.(2.2)		12 c(11.4) s.d.(1.4)		7 c(12) s.d.(0.8)		12 c(11.7) s.d.(0.8)
COD	6 c(11.8) s.d.(3)		8 c(12.1) s.d.(3.1)		6 c(11.8) s.d.(3.6)		8 c(12.7) s.d.(4)
C1	8 c(7.8) s.d.(1.6)		7 c(7.1) s.d.(1.9)		8 c(7.8) s.d.(1.2)		9 c(7.8) s.d.(1.8)
CD	8 c(7.8) s.d.(1.9)		7 c(6.8) s.d.(2)		9 c(8.1) s.d.(1.1)		8 c(7.7) s.d.(1.6)
DET	181 c(167.2) s.d.(28.8)		178 c(186) s.d.(29)		205 c(216.4) s.d.(29.8)		238 c(237.5) s.d.(33.1)
AGG	72 c(68.4) s.d.(7.8)		76 c(68.1) s.d.(8.5)		70 c(68.5) s.d.(9.1)		68 c(70.3) s.d.(10.4)
ENG	60 c(65.5) s.d.(6.6)		72 c (66.) s.d.(7)		68 c(67) s.d.(10)		66 c(73) s.d.(10.3)
MATHS	68 c(71.2) s.d.(10.2)		78 c(75) s.d.(13.9)		75 c(73) s.d.(13.4)		64 c(74) s.d.(15.2)

Participant B (Table 4.17) played rugby in the first year of the research project and played for a total of 5 years before joining the previous rugby group in the second year of the study. B played for the 1st/2nd teams as a forward. B fell in the below average VOC category but reported no

learning difficulties only encephalitis prior to commencement of the study. On the SIM subtest, he initially and during the second year scored within the mean for the test, other than that his scores were much higher than the norm especially at year three. VPAII and COD scores were in the average range whilst VPAI subtest scores were initially below the mean but improved to average and above. CI scores were in the average range at the end of the 1st and 3rd year but not at baseline and in the second year. CD scores were across the board below the norm suggesting delayed visual memory difficulties. DET scores with the exception of baseline scores were in the average range but at the bottom of the range suggesting possible motor difficulties.

On completion of the research, in a follow-up assessment, it emerged that B (Table 4.17) had previously had remedial intervention and that he had been diagnosed with a language disorder hence his initial low VOC score. B also had motor slowing due to poor wrist flexion. A full educational assessment later revealed a very superior verbal scale and a superior performance scale with a significant difference between the two scales and only a high average working memory and processing speed index. The assessment revealed difficulty with receptive (auditory processing) and expressive language, working memory, fine motor control and vision. His performance on visual-motor tests was unsatisfactory but it was thought that this might have been influenced by the fine motor control difficulties. The minimal influence on the test scores following a concussion may be explained by the boy's high brain reserve capacity, however when one views the scores of participant F (Table 4.20) it is clear that given the high brain reserve capacity, the scores of participant B should indeed be higher e.g. COD. This deficit when compared with participants in the same range is accounted for by the difficulties alluded to above. When one compares these scores with the scores of participant A that fell in the average

range of intellectual functioning one can clearly see that A had less of a buffer than B in terms of resilience.

Table 4.17 Participant B scores versus normative data

	Pre Test	Y1	Post Test 1	y2	Post Test 2	Y3	Post Test 3
Concussion		Yes		No		No	
VOC	8 c(10.9) s.d.(1.5)						
SIM	10 c(10) s.d.(2.3)		14 c(10.6) s.d.(1.9)		11 c(10.5) s.d.(1.6)		18 c(10.7) s.d.(1.5)
VPA I	6 c(9.4) s.d.(3.1)		12 c(10.8) s.d.(3.3)		16 c(11.8) s.d.(3.3)		16 c(12.5) s.d.(2.6)
VPA II	12 c(11) s.d.(2.2)		12 c(11.4) s.d.(1.4)		12 c(12) s.d.(0.8)		12 c(11.7) s.d.(0.8)
COD	12 c(11.8) s.d.(3)		11 c(12.1) s.d.(3.1)		12 c(11.8) s.d.(3.6)		14 c(12.7) s.d.(4)
C1	4 c(7.8) s.d.(1.6)		7 c(7.1) s.d.(1.9)		6 c(7.8) s.d.(1.2)		6 c(7.8) s.d.(1.8)
CD	3 c(7.8) s.d.(1.9)		4 c(6.8) s.d.(2)		5 c(8.1) s.d.(1.1)		4 c(7.7) s.d.(1.6)
DET	137 c(167.2) s.d.(28.8)		175 c(186) s.d.(29)		238 c(216.4) s.d.(29.8)		220 c(237.5) s.d.(33.1)
AGG	74 c(68.4) s.d.(7.8)		74 c(68.1) s.d.(8.5)		80 c(68.5) s.d.(9.1)		81.3 c(70.3) s.d.(10.4)
ENG	66 c(65.5) s.d.(6.6)		72 c(66) s.d.(7)		73 c(66.6) s.d.(9.6)		78 c(72.8) s.d.(10.3)
MATHS	76 c(71.2) s.d.(10.2)		75 c(74.6) s.d.(13.9)		83 c(72.5) s.d.(13.4)		90 c(73.7) s.d.(15.2)

Participant C (Table 4.18) continued to play rugby throughout the research period as a forward for either the 2nd or 3rd team. He reported one concussion in the first year and although his scores were lower than baseline at the end of the first year they improved by the end of year three.

During the third year the player played very few games due to injury and illness. C's VOC score fell in the average range of intellectual functioning but was just slightly below the study norm. His SIM scores were in the average range; however his baseline SIM score of twelve dropped to nine and remained at ten following a concussion in the first year. This performance is not in keeping with performances of other participants in the average VOC range of functioning.

On both the VPAI and VPAII subtests C performed within the norm at baseline and in the third year but below the norm for the middle two years. COD scores were initially outside of the range but were within the range in the second and third year. With the exception of the CD score in the third year, CI and CD scores remained below the scores of the norm groups throughout the three years. DET, AGG, ENG and MATHS scores were all within the study norms but in the second year were lower than the previous year despite no report of concussion. The scores nevertheless increased from baseline to year three. The lowered scores in the second year relative to C's own performance as well as lowered scores on some of the other cognitive tests leads one to question whether an injury incurred in the second year that was not reported or whether the scores were showing residual deficits. The participant's scores however improved by year three, a year in which the participant had minimal contact due to reduced game time as a result of injury.

Table 4.18 Participant C Scores versus normative data

	Pre Test	**Y1**	**Post Test 1**	**y2**	**Post Test 2**	**y3**	**Post Test 3**
Concussion		Yes		No		No	
VOC	8 c(10.9) s.d.(1.5)						
SIM	12 c(10) s.d.(2.3)		9 c(10.6) s.d.(1.9)		10 c(10.5) s.d.(1.6)	`	10 c(10.7) s.d.(1.5)
VPA I	8 c(9.4) s.d.(3.1)		6 c(10.8) s.d.(3.3)		7 c(11.8) s.d.(3.3)		12 c(12.5) s.d.(2.6)

VPA II	10 c(11) s.d.(2.2)		6 c(11.4) s.d.(1.4)		4 c(12) s.d.(0.8)		12 c(11.7) s.d.(0.8)
COD	8 c(11.8) s.d.(3)		5 c(12.1) s.d.(3.1)		8 c(11.8) s.d.(3.6)		12 c(12.7) s.d.(4)
C1	3 c(7.8) s.d.(1.6)		3 c(7.1) s.d.(1.9)		3 c(7.8) s.d.(1.2)		3 c(7.8)\ s.d.(1.8)
CD	1 c(7.8) s.d.(1.9)		2 c(6.8) s.d.(2)		3 c(8.1) s.d.(1.1)		6 c(7.7) s.d.(1.6)
DET	197 c(167.2) s.d.(28.8)		201 c(186) s.d.(29)		205 c(216.4) s.d.(30)		247 c(237.5) s.d.(33.1)
AGG	66 c(68.4) s.d.(7.8)		73 C(68.1) s.d.(8.5)		65.5 c(68.5) s.d.(9.1)		70 c(70.3) s.d.(10.4)
ENG	67 c(65.5) s.d.(6.6)		72 c(66) s.d.(7)		61 c(66.6) s.d.(9.6)		74 c(72.8) s.d.(10.3)
MATHS	74 c(71.2) s.d.(10.2)		76 c(74.6) s.d.(13.9)		67 c(72.5) s.d.(13.4)		79 c(73.7) s.d.(15.2)

The two participants in the above average VOC category were E (Table 4.19) and F (Table 4.20). E played rugby throughout and reported a concussion due to rugby prior to commencement of the study. During the research period he had two concussions in one year and one in the following year. He played second and eventually in the third team as a forward. F played rugby initially as a forward for the third team for a period of 5 years. After the first year of the study he ceased to play and reported no concussions during his rugby career.

Participant E (Table 4.19) recorded an above average VOC score, with a SIM within the study norms over time. This SIM outcome is not what one would expect given the higher IQ as is evident in the VOC score of 14. VPAI and VPAII scores were within the norm with VPAI scores on the higher side of the range. COD scores at baseline and year two were below the norm group but in the normal range in the first and third year. In contrast participant F's scores on this subtest

increased substantially from the baseline over time. E's scores on this subtest cannot be attributed to slow motor functioning as his scores were far superior on the DET subtest than that of the norm group's and in keeping and better in year two and three than F's scores. CI and CD scores were within the range with the exception of the CI score lower than the norm at the end of the second year. In the second year CD scores also dipped relative to E's previous scores along with the other scores. DET improved over time and was above the norm scores, suggesting minimal impact from deficits in motor speed on coding scores. ENG and MATHS scores increased by the third year and were generally above or within the norm scores. Although the AGG scores were mostly above the norm scores relative to E's baseline his scores decreased suggesting the possibility of increased complexity and workload impacting on overall academic functioning. This participant was a prolific reader from a very young age which will account for the high VOC scores as well as the preservation of ENG scores; in addition he received regular additional mathematics input.

Table 4.19 Participant E scores versus normative data

	Pre Test	**y1**	**Post Test 1**	**Y2**	**Post Test 2**	**y3**	**Post Test 3**
Concussion		Yes		Yes		No	
VOC	14 c(10.9) s.d.(1.5)						
SIM	11 c(10) s.d.(2.3)		12 c(10.6) s.d.(1.9)		9 c(10.5) s.d.(1.6)		11 c(10.7) s.d.(1.5)
VPA I	11 c(9.4) s.d.(3.1)		13 c(10.8) s.d.(3.3)		15 c(11.8) s.d.(3.3)		15 c(12.5) s.d.(2.6)
VPA II	12 c(11) s.d.(2.2)		12 c(11.4) s.d.(1.4)		12 c(12) s.d.(0.8)		12 c(11.7) s.d.(0.8)
COD	8 c(11.8) s.d.(3)		10 c(12.1) s.d.(3.1)		8 c(11.8) s.d.(3.6)		11 c(12.7) s.d.(4)
C1	7 c(7.8) s.d.(1.6)		9 c(7.1) s.d.(1.9)		4 c(7.8) s.d.(1.2)		8 c(7.8) s.d.(1.8)

CD	9 c(7.8) s.d.(1.9)		9 c(6.8) s.d.(2)		7 c(8.1) s.d.(1.1)		9 c(7.7) s.d.(1.6)
DET	207 c(167.2) s.d.(28.8)		228 c(186) s.d.(29)		253 c(216.3) s.d.(29.8)		270 c(237.5) s.d.(33.1)
AGG	83 c(68.4) s.d.(7.8)		79 c(68.1) s.d.(8.5)		77.5 c(68.5) s.d.(9.1)		72.9 c(70.3) s.d.(10.4)
ENG	79 c(65.5) s.d.(6.6)		78 c(66) s.d.(7)		74 c(66.6) s.d.(9.6)		86 c(72.8) s.d.(10.3)
MATHS	86 C(71.2) s.d.(10.2)		85 c(74.6) s.d.(13.9)		92 c(72.5) s.d.(13.4)		88 c(73.7) s.d.(15.2)

F (Table 4.20) also scored well above the norm over time on MATHS and COD, AGG and ENG were also above the norm with the exception of the third year where they were in the norm. CI and CD scores were consistently in the higher part of the norm range as was VPAI. VPAII was above the norm except at baseline and DET scores for the most part were above the norm. Although SIM scores were in the normal range, the scores in the second and third year declined from the baseline. Just like E, AGG and SIM scores fail to increase over time and one needs to consider the impact of more complex tasks and an increased workload on the cognitive and academic functioning of boys that have been exposed to the game of rugby at a critical phase in the development of executive functioning.

Table 4.20 Participant F scores versus normative data

	Pre Test	**y1**	**Post Test 1**	**y2**	**Post Test 2**	**y3**	**Post Test 3**
Concussion		No		No		No	
VOC	16 c(10.9) s.d.(1.5)						
SIM	12 c(10) s.d.(2.3)		12 c(10.6) s.d.(1.9)		10 c(10.5) s.d.(1.6)		10 c(10.7) s.d.(1.5)
VPA I	8 c(9.4) s.d.(3.1)		16 c(10.8) s.d.(3.3)		15 c(11.8) s.d.(3.3)		16 c(12.5) s.d.(2.6)

VPA II	12 c(11) s.d.(2.2)		12 c(11.4) s.d.(1.4)		12 c(12) s.d.(0.8)		12 c(11.7) s.d.(0.8)
COD	16 c(11.8) s.d.(3)		19 c(12.1) s.d.(3.1)		18 c(11.8) s.d.(3.6)		19 c(12.7) s.d.(4)
C1	9 c(7.8) s.d.(1.6)		9 c(7.1) s.d.(1.9)		9 c(7.8) s.d.(1.2)		9 c(7.8) s.d.(1.8)
CD	9 c(7.8) s.d.(1.9)		9 c(6.8) s.d.(2)		9 c(8.1) s.d.(1.1)		9 c(7.7) s.d.(1.6)
DET	227 C(167.2) s.d.(28.8)		213 c(186) s.d.(29)		238 c(216.4) s.d.(29.8)		243 c(237.5) s.d.(33.1)
AGG	81 c(68.4) s.d.(7.8)		82 c(68.1) s.d.(8.5)		83 c(68.5) s.d.(9.1)		79 C(70.3) s.d.(10.4)
ENG	78 c(65.5) s.d.(6.6)		84 c(66) s.d.(7)		81 c(66.6) s.d.(9.6)		82 c(72.8) s.d.(10.3)
MATHS	90 c(71.2) s.d.(10.2)		92 c(74.6) s.d.(13.9)		97 c(72.5) s.d.(13.4)		95 c(73.7) s.d.(15.2)

When reflecting on the performances of the above mentioned participants it was evident that scores on subtests cannot just be considered at face value. Each "case" is a unique individual with a unique set of circumstances and a unique response to injury. Although group norms provide guidelines to inform decision making with regard to management, they have to be considered within a clinical framework taking into consideration the individual's history, collateral information and previous level of cognitive, academic, behavioural and emotional functioning. Nevertheless considering all the case studies, the scores of A appeared to be the most sensitive to cumulative concussion.

CHAPTER 5: DISCUSSION

5.1. Context of the Study

The current study was conducted in the context of MTBI sustained by children/early adolescents participating in contact sports. MTBI was defined, using The AAN guidelines (see p. 16), as a traumatically induced alteration in mental status with or without loss of consciousness (Maroon, et al., 2000; Bender et al., 2004). The focus was on the impact of MTBI as well as repeated MTBI, sustained during the game of rugby in the RU framework, on neuropsychological/cognitive and academic functioning. Matters considered relevant within the context of the current study included the impact of MTBI on the developing brain, especially injuries occurring at a critical stage of cognitive development and whether, over time, repeated injuries resulted in residual deficits. A concern that served as a further motivating factor to conduct the study was the minimal attention given to MTBI by clinicians, coaches, players and the general public. It is believed that the dismissal by clinicians of the sequelae of MTBI is largely fuelled by experiences with screening measures that have limited sensitivity for MTBI effects as well as the perception around the protective function of plasticity. The perceptions of coaches and players are in turn influenced by the actions of medical practitioners.

The theoretical framework underpinning the current study was Satz's (1993) BRC theory (discussed under 5.3.2) in relation to protective and vulnerability factors following an insult to the brain. Erikson's Psychosocial Theory (Erikson, 1963, 1968) provided the platform for the developmental stage of the participant group as well as the developmental tasks of physical maturation, formal operations, emotional development, membership of peer groups and heterosexual relationships, to be negotiated by individuals in the Early Adolescent stage. For the

purposes of the study only the formal operational task of early adolescence was explored in relation to physical maturation. The other theoretical influence came from Piaget's Cognitive Developmental theory (Piaget, 1952b, 1970) in the form of development of formal operational thought in early adolescence. Both Erikson and Piaget emphasized the development of this higher cortical functioning at this stage of development. The concept of higher and lower cortical functioning is associated with hierarchical theories of brain functions and mental ability factors. "Higher" functioning refers to more complex mental operations whilst "lower" refers to simple operations. The degree to which concepts are abstract or concrete determines their place on the scale. Abstract concepts represent a higher level of thinking than concrete concepts (Lezak et al., 2004). From a psychosocial perspective, Erikson, (1963, 1968) stressed that tasks at each developmental stage had to be successfully negotiated for gains to occur at the subsequent stage. For example if in early adolescence one did not successfully negotiate formal operational thought, one would continue to respond in a concrete manner in late adolescence.

Biologically, growth spurts reportedly occur between 3-10 months, 2-4 years, 6-8 years, 10-12 years and 14-16 years; with the process of brain maturation continuing until approximately 18 years and beyond (Kolb & Whishaw, 2003). Nass (2002) contributed to the developmental debate from a biological perspective suggesting that cognitive functioning was more impeded following early as opposed to late injury because in the former functioning had not yet adequately developed. Kolb & Whishaw, (2003) argued that higher mental functioning, important for normal adult functioning was controlled by areas that myelinate later. The process of myelination usually takes much longer than synaptogenesis and persists beyond adolescence (Nass, 2002). Abstract thinking alternatively referred to as verbal concept formation (Lezak, 1995), which is assessed by amongst others, the Similarities subtest (Lezak, 1995; Sattler, 2001), is associated with the frontal

lobes and executive functioning (Walsh, 1994). Although, both Piaget and Erikson associated the development of formal operational thought, the stage subsequent to concrete thinking, with early adolescence, Piaget's early adolescence began at age 11 whilst Erikson's began at 12 through to 18 years (Erikson, 1963, 1968; Piaget, 1952b, 1970). In relation to development, it is suggested that Goldberg (2001, p.3) may concur with his suggestion that "it is more than coincidence that the biological maturation of the frontal lobes takes place at the age that has been codified in virtually all developed cultures as the beginning of adulthood." The frontal lobes are hierarchically organized, with processes such as myelination progressing through a number of stages, from primary and sensory areas to association areas and finally frontal regions," with the final growth spurt taking place in late adolescence (Fuster, 1993; Hudspeth & Pribram, 1990; Kennedy et al., 1982; Klinberg et al., 1999; Staudt et al, 1993; Thatcher, 1991, 1997; Uemura & Hartman, 1978 as cited in Anderson et al., 2001, p. 94). If we consider, in the light of the bio-psychosocial and cognitive development discussion, the claim made by Nass (2002) above, one can understand the following argument made by Goldberg (2001): He argues that, "the prevalence of lasting impairment following "mild" head trauma, is much higher than previously thought and that the impairment invariably affects frontal lobe functioning. Furthermore, he argues that "the frontal lobes have an exceptionally low 'functional breakdown threshold', and that frontal lobe dysfunction is to the brain disease what fever is to bacterial infection" (Goldberg, 2001, p.115).

From the initial review we saw that head injury was regarded as one of the common causes of acquired disability during childhood with mild head injury the most prevalent in 5-14 year age range (Asarnow, Satz, Light, Zaucha, Lewis & McCleary, 1995, p. 705). A Norwegian study showed that the results of neuropsychological tests conducted 4-8 months after concussion on 56

children; 9-13 years of age matched with a control group on variables of school grade, sex and academic performance were superior for the control group when compared with the experimental group on 29 of the 32 test variables. ANOVA revealed that the concussion variable explained most of the differences between the groups. We also saw that as age increased the differences decreased but as the complexity of the tests increased the differences increased. The results of this study also indicated that neuropsychological sequelae were evident even with few subjective complaints and no perceptible lags in academic achievement (Gulbrandsen, 1984).

A review of contact sports such as boxing (Jordan, Jahre, Hauser et al., 1992a,b), soccer (Matser et al., 2004), AFL (Maddocks, Saling & Dicker, 1995), RL (Hinton-Bayre & Geffen, 2004), American Football (Collins et al., 1999) and RU (Shuttleworth Edwards et al., 2001a) showed a strong relationship between multiple concussions and contact sport, albeit in older participants.

Contrary findings (Machiocchi, Barth & Littlefield, 1998, pg. 34), however, showed that the frequency of head injury was relatively low in sport, that the overwhelming majority of single grade 1 injuries had few persisting symptoms and that in the short term, morbidity appeared low. Hinton-Bayre and Geffen, (2004) also reported no significant difference in performance on tests sensitive to brain injury between players with no concussions, one concussion and two or more concussions. They reported that to date (Hinton-Bayre & Geffen, 2004) no AFL or RL studies found strong evidence for chronic or cumulative effects from concussion. These studies, however, lacked prospective data as well as data relating to learning disorders, genetic vulnerability or advancing age (Hinton-Bayre & Geffen, 2004).

The literature review also showed that there was little consensus regarding 'time frames' such as 'acute', 'chronic' and 'residual' when referring to deficits or effects. It is unclear whether acute refers to short term and whether chronic and residual imply long term and what exactly these time frames are. Acute effects, could refer to a resolution of cognitive sequelae up to and within 3 months post injury (Barth et al., 1989) or return to pre-season functioning (McCrory et al., 1997) whilst chronic effects could be the objectively observable deficits one year post injury (Winogron, Knights & Bawden, 1984). Persistent or residual effects are reported to be those consequences that extend beyond the three months time frame (Barth et al., 1989; Hinton-Bayre, Geffen, Geffen, McFarland & Friis, 1999; Maddocks & Saling, 1996; Shuttleworth-Jordan, Puchert & Balarin, 1993; Wilberger, 1993). It was suggested that long term chronic difficulties often presented weeks or months post injury and 25-40% of individuals never fully recover. These findings were however in sharp contrast to the findings in American football, AFL and RL of an average recovery within 5-10 days of sustaining a concussion (Hinton-Bayre and Geffen, 2004). In the current study assessment to identify residual deficits occurred 10 weeks after completion of a rugby season and more than 12 weeks following a reported concussion. Deficits were also monitored over a period of three years.

Shuttleworth-Jordan et al. (1993) found deficits in the scores of non-head injured rugby players compared to a control group of non-rugby players. Rugby players showed less capacity than controls for practice effects between pre- and post-season testing. Relative to controls, those rugby players with MTBI displayed significant dysfunction on neuropsychological tests at three days post-injury (Shuttleworth-Jordon et al., 1993). Although considerable recovery was evident at one month, two months and again at three months post-injury, the mild head injury group did

not demonstrate a practice effect to the same degree as the controls suggesting that full recovery had not yet been reached.

In contrast to the recovery reported in adults above, cognitive recovery in children with TBI could continue for up to five years following an accident (Klonoff et al. 1977). With children, residual deficits are often only revealed as the child matures due to the increase in the complexity of demands (Bowman et al., 1974, cited in Brooks, 1984). It was noted that when cognitive deficits persisted it hampered education, behaviour and progress at school resulting in underachievement even when no intellectual loss was evident (Lishman, 2002). In relation to residual deficits, although the immature nervous system is reportedly more plastic than the mature, cognitive development is impeded more by early than late injury because skills are already acquired in the latter making the mature brain more resilient. Plasticity in the immature nervous system therefore has limits with the brain being most vulnerable during critical change periods (Nass, 2002). Insults such as trauma will therefore have more harmful effects on the immature nervous system as opposed to the mature system. The improved plasticity of the immature nervous system alluded to above, therefore contradicts its vulnerability to insult. How well the individual recovers following an insult mirrors a balance between plasticity and vulnerability (Nass, 2002).

Aspects relating to issues of vulnerability are addressed in Satz's (1993) BRC theory (See 2.5 & 5.3.2), in which IQ is equated with BRC. A low BRC is associated with a low threshold in terms of protective factors and the lower the threshold, the easier it is reached making the individual more vulnerable to insults, than individuals with a higher IQ

5.2. Aims, Research Questions and Hypothesis of Study

The aim of the research was to assess the impact of repeated mild traumatic brain injuries (concussions) on the cognitive and academic functioning of early adolescent RU players. It aimed to investigate, over a period of three years, within the context of BRC theory, using paper and pencil cognitive/neuropsychological tests sensitive to diffuse damage as well as academic mark reports, whether early adolescent RU players having sustained repeated concussions showed signs of residual deficits as evidenced by sustained impaired performance on tests sensitive to diffuse brain damage and whether they showed a decline in cognitive and academic functioning relative to non-concussed sport controls (hockey, squash, cross-country, fencing). This approach differed from previous RU studies because it included baseline testing followed by three post tests over a three year period and a control group matched for age, sex, grade, sport participation, SES and level of education. It was felt that results, obtained form the one test occasion utilized in many of the previous RU studies, showing residual deficits, could be questioned given the cross-sectional nature of a single test occasion where previous exposure to rugby was not taken into consideration.

The HYPOTHESES were that in terms of the **time effect** across all the levels of analysis – the scores (the averages) over time of the groups combined would remain the same. In terms of the **group effect,** i.e. the group differences e.g. (**concussion versus non concussion; rugby versus non rugby; compromised versus non-compromised),** the hypothesis was that the average score between the groups would be the same. In terms of the **combined effect (interaction of groups and time)** the hypothesis was that there would be no interaction between time and groups.

5.3. Discussion of Results

The AAN concussion guidelines (Maroon et al., 2000; Bender et al., 2004) were the guidelines used in this study (see p. 16). These guidelines were used as they are widely accepted as the appropriate guidelines to inform current sports concussion management (Bender et al., 2004). Participants and coaches were informed and advised accordingly and concussion reporting and recording was done within this framework. Of the 150 participants that commenced the study at the pre-test phase only 136 remained in the study from the academic perspective and only 111 from the group that completed the neuropsychological test perspective (Table 4.5). Therefore, of the 136 boys remaining at the school 25 chose not to continue with the neuropsychological testing. In terms of consent procedures this information is most encouraging because it shows that participants were not afraid within the school context to exercise free will. Participants that chose not to continue with the study were members of the control group who saw no reason to continue and compromised rugby players with multiple concussions who felt that negative results could influence further inclusion in the team.

The results included subjective measures which comprised participants' reports of MTBI; objective measures which comprised quantitative data obtained from test scores; descriptive measures which examined the qualitatively aspects of the data and case studies which explored individual differences. Although the measures provided valuable data they were not without their challenges. The challenge of the first measure was the subjective nature of the responses but this was tempered by the use of repeated response questionnaires and feedback from parents and coaches. The first challenge faced in the pursuit of objective data was to determine the most

suitable tests for use in the South African context. Ordinarily, given the past inequalities in access to education for a large portion of the population, choices would be limited to tests standardized on the South African population. However, given the high functioning, high SES, English speaking research population and the use of the WAIS and WMS subtests on similar populations in the previous RU studies (Shuttleworth-Edwards et al., 2004), use of the WISC III and WMS III subtests were considered appropriate. The next challenge was the use of individual subtests as group tests. This challenge was overcome by, firstly, selecting particular subtests to test the hypothesis, of no difference between groups. Selection of tests followed the precedent set by Shuttleworth-Jordan et al., (1993), Shuttleworth-Edwards (2001) and Shuttleworth-Edwards et al., (1995, 1999, 2001 & 2004) in previous RU MTBI studies. Secondly, these tests were administered in groups, by exposing both the control group and the research group to the same tests at the same time at each test occasion and statistically determining the differences between the groups. At all times, in this group setting, all conditions pertaining to optimal test conditions were adhered to. It should be noted that what was required for this study was not performance on a full IQ test or neuropsychological battery for diagnostic purposes but performance on selected cognitive tests, under optimal conditions, following which individual scores are added to provide group scores. Test conditions were considered vital in conducting this study. Selecting of the tests were done with the research question in mind and in this instance the question was specifically related to group differences as opposed to individuals versus population norms. Test had to be of such a nature that they could be conducted in groups. If the group was very diverse in terms of abilities or if there were children with special needs individual testing may have been considered. However, the participant group was relatively 'homogenous'. Given that the sampling of the experimental group and the control group was controlled in such a way that they were derived from the same parent population, the research assumption is implicit in that there is

no difference between groups with respect to SES, education, school, age, grade, participation in sport, gender, language, and at the final levels of analysis medical, psychiatric and psychological factors, the only tangible difference is that one group is exposed to the game of rugby whilst the other group participates in different sporting codes. There is nothing to suggest at all that the group that play rugby and the group that engage in other sporting disciplines are from different parent populations. Therefore the study proceeded on the no difference assumption. In the analysis of between group differences one is not interested in individual scores versus a predetermined norm but in a statistical difference between the means of the different groups. From a statistical perspective this type of analysis is what is required as well as useful but the clinical relevance is lost. The challenge in terms of clinical relevance was overcome by the incorporation of descriptive analyses and case studies. The challenge faced when using these types of analyses was the qualitative nature of the results. One would therefore refrain from using this type of analysis in isolation. In terms of this thesis, despite the limitations of the individual analyses, it was felt that it complemented the other levels of analyses and by taking the findings of all three levels into account one would have a more holistic view of the outcome of the research.

5.3.1. MTBI's

Twenty two participants (15%) of the sample reported having had concussions (rugby and other) prior to commencement of the study (Table 4.3). Concussions were ascribed to a range of causes from falling out of a tree, falling off a skateboard or bicycle, rugby, cricket and motor vehicle accidents spanning a time frame from toddlerhood to the current time. Participants with previous grade 3 concussions were, in conjunction with other compromised participants, excluded from

some aspects of the study. In terms of the presentation of the concussions the most frequently reported symptom was feeling dazed or dizzy followed by complaints of headache (Table 4.4). These reports were consistent with what one would expect from grade 1 and 2 MTBI's.

A total of 51 participants reported concussions in the first year of the study, 38 during the second year and 23 during the third (Table 4.3). The decline in these numbers could be attributed to the decline in the number of rugby players from 115 in the first year to 66 in the third year. It could, however, also be attributed to the reluctance by players to report concussions for fear of being prevented from participating in the game. Scrutiny of self-report questionnaires showed participants denying concussions in the year of the concussion but reporting it in the following year as an incident that occurred the previous year. There were also anecdotal reports of participants advising those that had sustained concussions not to report the incident as it could influence further participation in the game. Nevertheless a cross tabulation analysis of reported concussions using a Chi-square test showed that rugby players (n= 66) reported significantly more concussions (p= .00013) than controls (n= 70) and more multiple concussions than controls (Fig. 4.1) and (Table 4.1). It is important to note that these figures represent the number of participants that reported concussions irrespective of whether they report one, two or three concussions and it was these figures used in the between group analysis. If one considers these figures in terms of incidences of concussion, the figure for the rugby only playing group would be 68% for individuals that reported concussions and when the results are annualized, an average of 22.7% of participants reported concussions. This figure excludes concussions of previous players. However, when one calculates every single concussion incident as opposed to the participant a very different picture emerges (Table 4.2). In terms of concussion, six incidences (20%) were reported by non-rugby players (n= 30), twenty six (65%) by previous players (n=

40) and seventy seven (117%) by rugby players (n= 66) When these results are annualized, it shows that 36.8% of rugby playing participants reported concussions. These results exclude previous players and they are substantially higher than the figures linked to participants.

This latter figure is higher that the figure reported by Nathan et al. (1983) who reported the percentage of concussions during a single rugby season at a South African high school to be 21.5%. It however does not support the incidence reported by Shuttleworth-Edwards et al. (2001a) who reported an average historical incidence of 2.3 (range 0-7) concussions per rugby playing schoolboy (effectively 230%), in a survey of three South African schools' top teams, compared with an average incidence of only 0.4 (range 0-1) for an equivalent group of field hockey players. The presence of differences in the cognitive and academic scores of concussed versus non-concussed participants is discussed in section 5.3.3.

5.3.2. BRC within the Research Context

The research commenced with a null hypothesis of no difference within the framework of BRC theory. With respect to the BRC theory that underpinned this research, **VOC** scores were used to determine an estimated threshold factor in terms of protection from or vulnerability to symptom onset. ANOVA results showed significant group effects for baseline scores on the (VOC F_{05} (1, 134) = 9.53; p =.002), (d =4.5) with a power > 1 at .05 significance level between rugby players (n= 66, M (SD) = 9.2(0.2)) and those that previously played/never played (n= 70, M (SD) = 10.2(0.2)). The rugby players scored significantly lower than players that had never played rugby and players that had previously played rugby thereby suggesting that non-rugby players performed better on the estimated IQ equivalent test than rugby players and therefore should this

theory hold one might argue that individual differences existed at baseline between the two groups possibly accounting for variable instances of protection from or vulnerability to insult. BRC theory holds that a threshold factor is in existence prior to the presentation of symptoms resulting from dysfunction in the central nervous system (Satz's, 1993). Furthermore an investigation of the difference in VOC scores between rugby playing participants with three concussions and those with zero concussions revealed that participants with three or more concussions (n = 8, (M = 8.6, SD = 1.19)) scored significantly lower than participants with zero concussions n = 47 (M= 10.5, SD = 1.92), F_{05} (1, 54) = 6.92; (d = 1.26) with a power (using the harmonic mean = 13.67) of 0.90 at significance level p = 0.05.

In terms of this discussion one needs to be mindful of the fact that the study commenced with the no difference assumption between the two groups given that all participants were considered to be from a high SES, high level of education parent group and would in all probability fall in a high BRC category. One also needs to consider: firstly that the participants in the rugby group had already been playing rugby, some from age 7 and others from age 10 years, prior to the commencement of the study; secondly, that given the numbers of participants reporting concussions in the current study, the likelihood, that they sustained concussions during previous years, might be high. The VOC results showing significant differences between the two groups could be explained in a number of ways. Some may argue that: "bright children chose not to play rugby", whilst others may argue that early exposure to rugby and concussion impacted on the cognitive functioning of the rugby playing participants. It could also be argued that the rugby groups were less motivated which could be explained by the decrease in drive and initiative seen in patients following MTBI (Goldberg, 2001) or their poor performance could be attributed to boredom, distractibility or fatigue (Sattler, 2001) but then these behaviours could equally apply

to the control group. In general rugby players that participated in the testing were found to be more motivated than controls to perform well because they had more to loose in terms of being allowed to continue playing the game, than controls.

When considering the group differences and the arguments alluded to above one needs to consider Satz's (2001) assertions: firstly of a threshold factor that is in existence prior to an insult to the brain; secondly, that the acquisition of cognitive skills at different rates of development, will at the time of a head injury, be vulnerable to negative effects due to a future lowered critical threshold for impairment; and thirdly, that the aggregation effect of lowered brain reserve capacity increases vulnerability to functional impairment because of the combination of premorbid vulnerability factors and either single or multiple episodes of neurological damage (Satz, 2001). Given that participants in this study represent a generally high functioning community and despite significant differences the rugby players' scores remain in the average range albeit lower than scores of controls, one could argue for the presence of the protective factor associated with a higher BRC. This kind of argument assumes that these participants were previously in the high average range of functioning and that because of the protective factor they still remain in that range but only at a lower level. Alternatively, one could argue, given the significant differences, that BRC was compromised because of insults to the brain from repeated concussions during critical stages of development, in other words that these children may have been compromised in terms of reaching their full potential which may have been above as opposed to below average. One could also argue that future BRC could be compromised by playing rugby and being subjected to multiple concussions. From this perspective, the argument for aggregation of lowered brain reserve capacity increasing vulnerability to functional impairment (Satz, 2001) could hold and could support the proposition that cumulative MTBI

results in long term difficulties. Alternatively, the argument for plasticity not affording protection, because of the impact of insults to the brain during a crucial stage of cognitive development, could hold. In other words the opportunity for protection could be limited when the functions are inadequately established at the outset.

Relationship measures

Results of the Correlational analyses performed on the measure of **VOC** scores and the neuropsychological/ cognitive and academic test measures to determine the relationship, revealed that there were significant moderate to strong positive relationships $p<0.01$ between **VOC** and **SIM** with the following ($r = 0.51, 0.55, 0.44, 0.40$) respectively; **VOC** and **AGG** with the following ($r = 0.47, 0.52, 0.52, 0.46$) respectively (Fig 4.2); **VOC** and **ENG** with the following ($r = 0.47; 0.57; 0.47; 0.61$) respectively (Fig 4.3). According to Sattler (2001) a correlation above 0.50 either positive or negative suggests a moderate to strong relationship between two variables. He reported a significant relationship between VOC and SIM of Vocabulary ($r = .69$). Despite the hypothesis of no relationship between VOC and the other measures the results showed that there is a relationship between VOC and the subtests referred to above and the null hypothesis of no relationship is therefore rejected. Since correlations are used in predictions, the higher a correlation between two variables the more accurate one can predict the value of one variable when one knows the value of the other.

These significant relationships therefore suggest that by knowing the value of VOC one can predict the value of AGG, SIM, ENG MATHS and VPAI. It can be argued that this suggestion is supported by results in this study that show significant group effects for rugby players versus controls on the both the VOC and SIM subtest as well as a strong trend with regard to group

effects for AGG. On all three occasions the rugby players scored lower than controls and one could therefore, given the discussion above regarding relationships, predict the value of SIM and AGG knowing the value of VOC for rugby players and controls. The reverse, in terms of lowered AGG, SIM and ENG lowering IQ, however, does not hold true from a statistical perspective. Furthermore, there are claims that VOC falls into the crystallized category of intelligence and is considered a "Hold" test whereas Similarities is more fluid and considered a "Don't Hold" test (Lezak, 1995) suggesting that the latter is more likely to show the effects of a brain insult. Given the discussion of "Hold" and "Don't Hold" tests in the context of VOC, IQ, BRC and SIM, it is argued that from Satz's BRC perspective, perhaps it is the "Hold" aspect that provides the protective factor and the "Don't Hold" the vulnerability factor.

5.3.3. Between Group Differences

In terms of this research one could argue that a protective factor in terms of BRC and plasticity exists given that between group analysis for the rugby, concussion and the combined rugby and concussion groups showed no significant difference between the group means on tests of visual and verbal memory, processing speed and motor functioning.

Group effects

There were significant group effects for the no rugby and previous rugby groups combined (n =55) versus rugby (n = 56) **SIM** (F_{05} (1,109) = 4.6; p=0.03 (rugby)) (*d* = 3.0) (Table 4.7) (Fig 4.7). It appears that when the group that never played rugby is compared with the group that was exposed to rugby both previously and currently there is a significant difference in performance on

this subtest. The non-rugby players perform better than the rugby group on the measure of abstract thinking.

Results between the rugby (n = 66) and previous group combined with no rugby (n = 70) showed a trend for **VPAII** (F (1, 109) = 3.46; p = 0.06) and **AGG** (F (1, 134) = 2.93; p=0.08 (rugby) (*d* = 2), (Fig 4.8)) albeit non significant. It is argued that it is not appropriate to present trends, however in terms of performance on VPAII a tendency towards a difference may suggest that for the rugby players there is a tendency towards difficulties with delayed memory tasks and in terms of AGG although the statistics are not significant, in a school setting, this difference between the groups equates to a difference between a C and a B symbol on the school report card at the end of year 3. Please see Appendix O for graphic presentation as well as discussion on Combined Effects below in relation to further AGG findings.

The results showed no significant group effects for the concussion versus control groups. One would expect the concussion scores to be significant versus the non-concussion scores. However, as there is no significance, it may imply that the concussed controls perform better than the concussed rugby players, hence, neutralizing the combined concussion group's scores. Conversely or similarly, it may imply that the non-concussed rugby players' scores are poor relative to non-concussed controls, which neutralizes the non-concussed group's scores. This argument needs to be considered in the light of the significant difference between the concussions reported by the rugby group versus controls; the significant differences in VOC and SIM scores of the rugby versus control groups; the significant difference in VOC scores between participants with multiple concussions compared with zero concussions and the significant differences in AGG scores for rugby players with multiple concussions versus controls with no concussions.

Results of groups effects for **'Compromised'** data showed significant group effects for compromised participants (n = 23) versus non-compromised participants (n = 88) on the cognitive test **VPAI** p=0.03 (learning difficulty) F = 5.02(1,109) (d = 3.3), (Fig 4.10); and (n = 26) and (n = 110) respectively for the academic tests **AGG** p<0.01 (learning difficulty) F = 9.5(1,134) (d = 4.6) (Fig 4.11); **ENG** p=0.01 (learning difficulty) F = 6.6(1,134) (d = 3.9), (Fig 4.12); **MATHS** p<0.01 (learning difficulty) F = 9.2(1,134) (d = 4.6), (Fig 4.13). The significant difference between the groups for VPAI which was not observable for VPAII may suggest that in terms of the latter there was no difference between the learning that took place for the two groups whilst for the former slowed processing may have influenced the learning process for the compromised participants. Slowed processing speed, it could be argued, could in turn impact on the participant's performance on tasks such as ENG, AGG and MATHS when complexity and demands are increased as grade challenges increase. The non significant VPAII scores between the groups may also have been influenced by a scoring ceiling on this test which would have meant that no more improvements could be monitored if indeed they had occurred.

When the concussion variable was added to the above analyses there were no significant differences between the groups. These results suggest that compromised individuals perform poorer on these tests than controls regardless of whether they have been concussed. Given the significant relationship between VOC and these subtests, the link between estimated BRC and performance on these tests by learners with learning difficulties cannot be ignored i.e. there is the assumption that learners with a lower VOC will perform poorer on these subtests. However, this argument is contradicted by the absence of a significant decline in scores by compromised participants on the SIM subtest p=0.8 (learning difficulty).

A low BRC is therefore not necessarily associated with all learning difficulties as was illustrated in Case "B" (Table 4.17) – where the participant scored lower on VOC due to a language disorder rather than a low BRC. In this instance the participant's score for the SIM subtest improved from a scaled score of 10 to 18 over time. One therefore needs to be mindful firstly that language disorders such as "Mixed Receptive-Expressive disorders are diagnosed on the basis of a delay in language capabilities in the presence of a significantly higher IQ" (Hawkridge, Keyter & Steyn, 2005, p. 196) and that there may be exceptions particularly as it relates to the use of IQ scores as a predictor of performance for all individuals. Furthermore, this example highlights the need for each case to be considered on its merits and in order to do that vigorous neuropsychological assessment, which includes a thorough history taking and collateral input, as opposed to mere 'concussion testing' is required.

There were no significant results at the more specific level of analysis for the 'compromised' group in terms of rugby and concussion analysis. In contrast, in a study comparing football players with baseline scores, with controls, a significant interaction between the diagnosis of learning disability and two prior concussions was noted. This finding suggested an additive effect of learning disorders and multiple concussions on lowered functioning (Collins et al., 1999a). The authors suggested an association between two or more concussions and lessening of cognitive skills which when combined with learning disorder deficits compromised functioning even further (Collins et al., 1999a).

There were, however, significant group effects for compromised measures: **VPA1** (F_{05} (1,106) = 4.08; p=0. 04 (problems)) where compromised (n=36) and non-compromised (n=75); **AGG** (F_{05}

(1,131) = 4.87; p=0.03 (problems)); **ENG** (F_{05} (1,131) = 4.72; p= 0.03 (problems)) and **MATHS** (F_{05} (1,131) = 6.56 p=0.01 (problems)) where compromised (n=40) and non-compromised (n=96) with the effect of the co-variant VOC on the data, factored in. These results were similar to group results from earlier analysis (Table 4.8) that did not take into consideration the effect of the covariance i.e. **VPAI** p=0.03 (learning difficulty); **AGG** p<0.01 (learning difficulty); **ENG** p=0.01 (learning difficulty); **MATHS** p<0.01(learning difficulty) where at this level of analysis the compromised group differed significantly from the non-compromised group without the interaction with the concussion variable. With the effect of the co-variant **VOC** on the data factored in, no significant effects were evident when the concussion variable was added only a trend albeit not significant was observed for the subtest **AGG** (F_{05} (1,131) = 3.08; p=0.08 (concussion)). In terms of the 'Compromised' and 'Rugby' analyses, results were similar to previous findings with significant effects for comparisons of the compromised versus non compromised groups "Problems" for: **ENG** (F (1,131) = 5.87; p=0.01 (problems)) and **MATHS** (F (1,131) = 8.32; p <0.01 (problems)) **VPA1** (1,106) = 4.95; p=0.02 (problems) and **AGG** (F (1,131) = 8.79 p <0.01 (problems). Participants with problems scored significantly lower than controls on the academic tests and the test measuring verbal cued recall and learning.

Combined effects

In terms of the combined effect for VPAI (F_{05} (3,327) = 2.93; p=0.03 (interaction)) on 'Any Concussion" measures it was noted that at baseline the concussion group scored lower than the non-concussed group but in subsequent post-tests there was no difference between the groups. This result suggests that for both groups learning took place. Similarly, there was also a statistically significant **combined effect** for **VPAII** (F_{05} (3,327) = 2.87; p=0.04 (interaction)) suggesting that for both groups learning was retained.

In terms of academic scores AGG, there was a trend for the rugby players to score lower than the combined previous and no rugby group and a similar 'trend' picture was evident for SIM when compared across 3 separate groups (rugby 3) of rugby, previous and no rugby. However, when the effect of VOC was factored into the analysis comparing the non-compromised groups in terms of rugby and multiple concussions (n = 24) with no rugby and no concussions (n = 25) over time, there was a significant interaction effect between time, rugby and many concussions for **AGG** (F_{05} (3.132) = 3.6; p=0.02) (d = 2.1) (Time* rugby/ many concussions groups) (Figure 4.15). In terms of academic performance the results showed that in this specific cohort, the rugby players had a higher score at baseline but after three years the scores were below the level of the controls and the scores of the controls had increased. Because the obtained F = 3.6 exceeded $F_{.05}$= 3.06, the H_o of no difference between controls with no reported concussions and rugby players with multiple concussions was rejected. Given that the effect of VOC is accounted for, the differences between the groups can possibly be explained by the rugby and many concussion variables. At this point one needs to be mindful of the fact that the concussion measures were calculated on the number of participants reporting multiple concussions and not the total number of concussion for those participants. The significant outcome reported above may suggest that in terms of BRC theory, for this specific cohort, there was a measure of protection at baseline which would be in keeping with the assumption of no difference in BRC between the groups given the parent population. However, after three years of playing rugby and multiple concussions, in the face of changes in the volume and complexity of the academic work, and a significantly poorer performance than controls, one may consider Satz's (1993) arguments relating to aggregated effects and vulnerability factors.

The current findings suggest some support for the findings reported by Moser, Schatz and Jordan (2005) that there seemed to be subtle yet significant prolonged neuropsychological effects in youth athletes with a history of two or more previous concussions. Similarly, they reported that cumulative academic grade point averages were significantly lower, not only for youth athletes with two or more previous concussion groups, but for youth athletes who experienced recent concussions, suggesting that athletes with lower grade point averages may be more prone to concussion (Moser, Schatz & Jordan, 2005).

Time effects

In terms of the concussion versus control groups, subtests that showed no significance in terms of combined and group effect but rather in time effects were **COD** (F_{05} (3, 327) = 9.43; $p < 0.01$ (time))**,** DET (F_{05} (3, 327) = 190.55; $p < 0.01$(time)) (Fig 4.4), **AGG** (F_{05} (3, 402) = 5.29; $p < 0.01$(time))**, ENG** (F_{05} (3, 402) = 55.80; $p<0.01$ (time)) and **MATHS** (F_{05} (3, 402) = 3.76; p=0.01 (time))(Table 4.6)**.** In terms of the effect sizes for time, Howell (1995) suggested that if a medium effect size e.g. 0.50 at a power of 0.80 is required a sample size of 126 participants would be needed for a two-sample *t*. The n sizes, because of the repeated measures and longitudinal design, for the cognitive (n = 327) and academic (n = 402) measures above are clearly larger than the number suggested by Howell (1995). To illustrate the power as it relates to time in this study, the effect size of one of the subtests was calculated using the following equation: $d = M_1 - M_2 / \sigma_{pooled}$ and $\sigma_{pooled} = \sqrt{[(\sigma_1^2 + \sigma_2^2) / 2]}$ (Cohen, 1988). Please see Section 3.2 for a discussion regarding medium and large effects and (Fig 3.2) for the graphical presentation of the time effects, that show the power in relation to effect size with a much smaller N, than reported above.

It is argued that significant time effects suggest practice effects. In terms of this debate an increase in the scores over time is expected if one considers the participants' development, previous experience and familiarity with the tests. For example, in terms of the norms for DET, performance improves with age and this result is what one would expect over time. Another illustration is the statistically significant **time effect** for **VPAI** (F_{05} (3,327) = 60.94; p <0.01(time)) showing a change in the combined averages from the baseline to the third post-test (Fig 4.5). It is argued that the increase in the scores could have been attributed to cognitive development or maturation or test familiarization, however, the test was designed for persons 16 years and older and was first administered to the boys at age 12 to 13. One would therefore expect lower scores initially.

Results of the study across the analysis of rugby presented as 'rugby (Table 4.7) and compromised data presented as 'learning disability' (Table 4.8) also showed significant time effects for a number of cognitive and academic measures but similarly as for the concussion scores above not for SIM, CI and CD. In terms of no significance, the results of the global 'rugby' and 'rugby 3' analysis showed SIM F_{05} (3.327) = 2.3; p=0.07 (time) and SIM F_{05} (3,324) = 2.02; p=0.11 (time) respectively and the 'concussion' analysis SIM F_{05} = (3.327) = 2.0; p=0.11 (time). The lack of significant time effects in this respect is significant because if practice effects arguably played a role in the changes in scores of the other subtests over time one needs to question why there was no significant change in the SIM scores. Like all the other tests these tests were also presented at each test occasion. If scores improved on the other tests arguably because of practice effects, would one not expect the same pattern to apply to performance on these tests? These are relatively simple tests so students should be able to improve scores over time and they do as is evident in the analysis of percentages of deficits of non concussed, non

rugby players as well as in case studies of individuals with a higher estimated BRC. Furthermore, since abstract thinking begins to develop in early adolescence, one could argue that one would expect the scores on the SIM subtest, given the added practice effect, to improve. Notwithstanding this argument one could also argue that these are scaled scores adjusted for age so in fact, ordinarily one should not see huge increases in the scores over time. Nevertheless significant time effects were reported for other tests with scaled scores i.e. COD, VPAI and VPAII. In terms of the SIM subtest, although the range of scores for the non-concussion group was between 9.6 and 10.6, the range for the concussion group was between 9.2 and 10. On the whole the scores across the board were generally low, albeit in keeping with the VOC scores of the no/previous rugby group versus the rugby playing groups (Fig 4.6). When comparing the three rugby groups the current rugby group scores range was between 9.1 and 9.9 and the no rugby group scores range was between 9.7 and 11.

Although the argument regarding practice effects could hold true for the SIM subtest, it cannot hold true for the visual spatial memory tests CI and CD because these tests have a ceiling in terms of how well one can perform. The non-significant time effect for CI and CD showed that scores generally remained at the same level and on viewing the differences between the rugby groups for those subtests it was evident that the rugby group contributed to lowering the overall time effect (Appendix O - rugby 3 groups).

Contrary to the global findings relating to SIM time effects that were discussed above, at the specific level of analysis, when the effect of VOC was factored into the analysis there were significant time effects for SIM. With VOC as covariate the results were **SIM** (F_{05} (3,318) =2.78; p=0.04 (time)) 'concussion', **SIM** (F_{05} (3,318) = 3.13; p=0.02 (time)) 'rugby' and **SIM** (F (3,168)

=3.18; p = 0.03 (time)) 'VOC baseline' in the combined measures. These results contradicted the results obtained at a global level of analysis which did not take into account the effect of VOC on the scores as was evident in the 'rugby' analysis SIM (F_{05} (3.327) = 2.3; p=0.07 (time)); 'rugby 3' analysis SIM (F_{05}(3,324) =2.02; p=0.11 (time)) and the 'concussion' analysis SIM (F_{05}(3.327) = 2.0; p=0.11 (time). Despite the significant results at this level of analysis the increase over time remains minimal when compared with the increase over time of the other subtests.

In terms of making sense of these discrepancies, when one considers, for example, the significant relationship between variables VOC and SIM with a correlation coefficient r = 0.51, 0.55, 0.44, 0.40 respectively from baseline to year 3, one needs to be mindful of the degree to which the two variables vary together. Therefore, when considering the significant SIM (time) analysis, the covariate VOC refers to the amount of variance accounted for in SIM by VOC. The amount of variance not explained by VOC is the residual variance i.e. the true error after controlling for VOC (IQ equivalent). With the VOC effect removed from the data of the significant SIM (time) the true error is more reliable than the true error in the non significant SIM (time) results that do not take into consideration the effect of the covariance VOC, suggesting that the former result is a more reliable reflection of the time effect.

In terms of further discussion in this regard, if the correlational coefficient is big the error is reduced. Significance in this regard, however, refers to the value of (p) i.e. if p=0.5 the probability of making an error is 5% and therefore the higher (p) the less we can believe the chance of an error being made. Given the significant relationships $p<0.01$ between **VOC** and **SIM** with the following r = 0.51, 0.55, 0.44, 0.40 respectively, the correlational coefficient can be considered 'big' implying a reduced error. If the error is reduced *F* becomes bigger as in **SIM**

p=0.02 (time) F_{05}=3.2 (3,312) 'rugby' as opposed to SIM p=0.11 (time) F_{05} = 2.02 (3.324) 'rugby 3'. These findings suggest that one can more reliably reject the H_o of no difference, at the critical value $F_{.05}$= 2.64 (Appendix P) of the former result which factored in the effect of the covariance VOC.

In terms of the *F* distribution, in ANOVA, if the score exceeds the critical value we reject the H_o and conclude the groups are different. From the discussion above regarding error reduction, it would appear that the increase in the size of *F* where the effect of VOC on the SIM score was accounted for suggests that the chance of error had been reduced and that the result was a truer reflection in terms of significant changes in time.

With the influence of VOC on the scores accounted for there was a higher *MS*=5.1 for the significant SIM (time) result as opposed to *MS*=3.23 for the non significant SIM (time), indicating larger and more reliable within group variations. Findings from the percentage of deficits of SIM (Table 4.12), qualitatively, confirmed the large variability within the group, with the non rugby and previous rugby participants' scores declining in terms of deficits over time and increasing in terms of scores above one standard deviation from the mean. In contrast, deficits increased for rugby players and the percentage of scores one standard deviation above the mean were low.

Although the findings with regard to the time significance for SIM contradicted the argument made in relation to practice effects with the non significant time results in the global analysis, it is important to consider the influence of within group differences and the role of the non rugby and previous rugby groups' scores in terms of practice effects. Notwithstanding the above discussion

it should be noted that there were no significant time effects for AGG across any of the *specific* levels of analyses. There were also no significant time effects for any of the cognitive and academic measures when the non compromised participants were compared in terms of no rugby and no concussions versus rugby and multiple concussions suggesting that when the data of these two groups were put together to calculate the time effect there would have been differences between the groups over time. If one considers, that when the effects of VOC are accounted for in relation to AGG that there are no time effects as opposed to when they are not accounted for, one needs to consider the implications bearing in mind the strong correlation between AGG and VOC Fig 4.2). This finding is in contrast to the findings of SIM above. The difference in the two results can possibly be explained by the fact that the SIM test remained constant over the 4 test occasions whereas the AGG results changed from baseline across the post-test occasions due to curriculum and subject changes. Students that struggled could choose more manageable subjects and those that excelled could choose more challenging subjects.

5.3.4. Within Group Differences

To ensure that the research methods had clinical as well as statistical significance, the percentages of deficits were calculated, using the method of previous RU studies, reported by Shuttleworth-Edwards et.al. (2004). This method was also used by researchers in boxing and soccer studies (Casson et.al., 1984; Kaste et.al., 1982; Matser et.al., 1999; Tysvaer & Lochen, 1991). In this regard Bielensohn, (2001) argued that when comparisons of individual players to normative data was done the calculation of individual levels of deficits was more sensitive than analysis that compared group means because it showed individual variation within groups and therefore enriched interpretation of results.

The percentage of deficits were not markedly different for the rugby playing group in relation to the other groups for English, Mathematics, Delayed and Immediate Auditory/Verbal memory test performances (Appendix N). Furthermore on the tests measuring Motor Functioning and Speed of Information Processing the percentage of deficits were in contrast to the findings of greater deficits for rugby players reported in previous South African research. On the Speed of Information Processing test the percentage of rugby players and previous players showing deficits after three years was less than the non-rugby group and the same findings were noted for the percentage of previous players and rugby players on the test of Motor Speed and Precision, with the previous players showing marginally fewer numbers with deficits (Appendix N). One could argue that rather than developing deficits rugby players in terms of their training develop different skills (i.e. they may focus more on motor skills, hand-eye co-ordination which would impact on speed of information processing) from their non-rugby playing counterparts that may acquire other skills such as abstract thinking. In terms of this research one must bear in mind that the non-rugby control group did not imply no sport participation, at all. At the school where the research was conducted it was compulsory for all boys to participate in at least one summer and one winter sport. Therefore, although, the argument above may apply to a handful of boys that for medical reasons did no sport at all or perhaps a few cross-country runners, it would not be applicable to hockey players who would have undergone, from a cognitive perspective, similar speed and motor skills training as rugby players. Furthermore it was evident from the percentage of deficit scores of rugby players versus controls (non and previous players) on visual memory tests (retention of information following the visual processing speed test) that rugby players showed more deficits in terms of immediate and delayed retention than their counterparts.

In the functional areas of abstract thinking and academics, objective cognitive and academic testing showed consistent qualitative support for greater numbers of rugby players showing deficits than previous players and non-playing controls (Tables 4.10; 4.11; 4.12 & 4.13). One could argue that the larger number of rugby players showing residual deficits may be explained by the lower estimated BRC baseline for rugby players versus non rugby players possibly making them more vulnerable to brain insults than the non-rugby players.

Scrutiny of the percentages of participants in the different groups showing deficits on the test of abstract thinking (Table 4.12) showed that at baseline the numbers of non-rugby and previous players showing deficits were less than for rugby players. Although for the former two groups the numbers of participants showing deficits decreased by the third year more so for non rugby as opposed to the previous rugby group, the percentage of players showing deficits increased markedly for the rugby playing group. In addition, the percentage of participants in the more than 1 standard deviation above the mean group was higher for the non rugby group as opposed to the other two groups. Other than providing an explanation for the time effect for SIM when the effect of VOC has been accounted for, these findings could suggest that possibly the non rugby group with minimal insults to the brain, compared with the rugby group, were not hampered in terms of development of abstract thinking and that possibly they were less vulnerable given the protective factors associated with their higher estimated BRC. The findings of more deficits for SIM amongst participants playing rugby and sustaining insults to the brain could also be explained by the statistically significant lower VOC scores and the statistically significant concussion scores suggesting greater vulnerability for these participants in terms of the development of specific cognitive functions. The previous players' performances after three years were not dissimilar from the non-players suggesting that one could again argue for the existence of a protective factor

in terms of BRC as well as a compensation factor in terms of CR. This argument is strengthened by findings that suggest that once participants have ceased playing the game, performance on the test improves.

One could also theorize that the lowered abstract thinking score may also account for the significant decline in the academic functioning of rugby players with multiple concussions compared with non rugby players with no concussions, from a junior to senior school setting. At high school the academic demands increase and tasks become progressively more complex requiring intact abstract thinking skills. This thinking would support reports claiming that as the child matures, the nature of demands increases, revealing underlying deficits (Bowman et al., 1974 as cited in Brooks, 1984) and that when cognitive deficits persist it hampers education, behaviour, and school progress, resulting in underachievement even when no intellectual loss is evident (Lishman, 2002).

One can argue that the abovementioned discussion of the percentages of deficits, particularly the increase in the percentage of rugby players showing deficits; has flowed from descriptive analyses and that it should be viewed with caution. However, one should consider the discussion within the context of the significant findings already discussed. The findings were: a statistically significant difference between the control (n =55) versus rugby groups (n = 56) **SIM** p=0.03 (rugby) F_{05} = 4.6(1,109) (d = 3.5), (Fig 4.7) where the controls performed significantly better than the rugby group in terms of their performance on the abstract thinking test; the vulnerability of rugby players given their statistically significant baseline BRC estimate indicators compared with the no rugby/previous rugby group (**VOC** p<0.01, F_{05} = 9.53(1, 134) (d = 5), in which they performed significantly poorer; the vulnerability of rugby players with multiple concussions (n =

8, M (SD) = 8.6(1.19)) compared with participants with 0 concussions (n = 47, M (SD) = 10.5(1.92)), VOC F_{05} (1, 54) = 6.92; (d = 1.26) (d = 0.989) with a power (using the harmonic mean = 13.67) of 0.74 at significance level p = 0.05; and the significantly poorer academic performance of rugby players with multiple concussions versus controls. It would be difficult to ignore, firstly, the possible role of BRC in terms of protection from and vulnerability to insult and secondly, the possible role of the aggregation effects in terms of vulnerability in relation to the development of abstract thinking at a critical stage of cognitive development and the role of the latter in the future complex functioning and increased task challenge of early adolescents moving from a junior to a high school setting. The current findings possibly support findings by Leathem and Body (1997) and Levin and Eisenberg (1979) of objective cognitive deficits in areas of abstraction and reasoning. Findings of persistent deficits were also supported by Collins et al., (1999a) showing that participants with two or more concussions performed worse on tasks of attention and concentration and information processing speed and that a history of concussion is both significantly and independently associated with deficits of executive functioning. Although the current study could not confirm results in terms of attention and concentration and information processing speed it played a role, through statistical analyses, descriptive analyses and case studies, in highlighting aspects relating to higher cortical functioning such as abstract thinking.

Given the findings reported above, it is suggested that the findings may also support arguments for the limitations of plasticity in keeping with Nass's (2002) account that in the immature brain, a compromised nervous system is eventually taxed by increasing demands that accompany ageing. In terms of this discussion we, however, need to be mindful of results in the current study showing some measure of protection in the form of no significant differences between the groups

on cognitive functions that developed earlier in life. More recent studies have also shown support for the notion of cognitive reserve citing larger pre-morbid brain volume and higher education levels as playing a role in decreasing vulnerability to cognitive deficits following TBI (Kesler, Adams, Blasey, & Bigler, 2003).

In terms of the current research it is tentatively hypothesized that a lowered VOC for rugby players may have made them more vulnerable to negative effects due to a future lowered critical threshold for impairment. Because they were still in a critical stage of cognitive development in terms of abstract thinking this development was possibly impacted on and this impaired functioning may have in turn influenced their academic performance when demands were increased at a later stage.

5.3.5. Individual Differences/Case Studies

The force of an injury along with the individual's anatomy, physiology and genetics is unique to each injury as is the patient's metabolic response and vascular reactions. No two individuals present with the same sequelae despite being subjected to the same accident scenario (Bigler, 2000). In addition no two individuals despite having the same potential for protection from or vulnerability to insult in terms of BRC will exhibit the same performance on tests of cognitive and academic functioning following a grade 1 or 2 MTBI. This argument was highlighted in the current research in the discussion of case studies A, D and G (Tables 4.14; 4.15 & 4.16) where individuals scored in the average range in terms of BRC but their outcomes on cognitive and academic tests were vastly different.

In this regard, given the concept of trans-situational consistency of performance (Shuttleworth-Edwards, 2001), it is argued that one can expect, all things being equal, an individual with an IQ in the average range to perform in that range on a global level. Although the approach is useful, one must, however, also err on the side of caution in terms of making assumptions as each "case" is a unique individual with a unique set of circumstances and a unique response to injury. For example: The case of "A" (Table 4.14) who had a VOC in the average range played rugby and had numerous concussions and although he fell in the compromised group neglected to disclose this information . In the case of A the concept of trans-situational consistency of performance was useful in terms of guiding the researcher to question scores that were consistently below the average range. In contrast case "D" (Table 4.15) played no rugby, was not compromised and reported no concussions. His scores on cognitive and academic tests, in terms of the norms, remained in the average to above average range. Case "G" also played rugby was not compromised but had multiple concussions including a grade 3 concussion. Although his cognitive test scores (Table 4.16) declined following the grade three concussion, they improved a year later relative to his baseline. SIM scores however remained for the most part of the three years within the norm whereas his processing speed measured by the COD subtest remained below the norm over time suggesting slowed information processing. Although group norms provide guidelines to inform decision making with regard to management, the individuals' scores have to be considered within a clinical framework against normative data as well as taking into consideration the individual's history, collateral information and previous level of cognitive, academic, behavioural and emotional functioning.

In the case of "B" the concept of trans-situational consistency of performance and BRC was not useful as the researcher would have been guided by the VOC score in terms of a performance

expectation. Participant B (Table 4.17) played rugby in the first year of the research project and played for a total of five years before joining the previous rugby group in the second year of the study. B fell in the below average VOC category and reported no learning difficulties only encephalitis prior to commencement of the study. On the SIM subtest, his baseline score was within the mean but by the third year his scores were in the superior range and not what one would expect given his VOC score of 8. A full educational assessment later revealed functioning in the superior range with specific cognitive challenges. The minimal influence on the test scores following a concussion may be explained by the boy's high brain reserve capacity, however when one views the scores of participant F (Table 4.20) it is clear that given the high BRC, the scores of participant B should in general indeed be higher e.g. COD. When one compares these scores with the scores of participant A that fell in the average range of intellectual functioning one can argue that A had less of a buffer than B in terms of protection despite both being compromised in some way.

It is important to note specifically as demonstrated in the case of A that deficits were identified as a pattern over time when compared with norms but also within the individual's own expected performance. Over time performances for G and B also revealed changes in the absence of either no concussion and or no or previous rugby. These findings are in keeping with the comments by Giza and Hovda (2004) that it is difficult to identify Neuropsychological sequelae following MTBI in a juvenile brain injury because overt neurological dysfunction is not always prominent. It is also not always feasible to assess the possibility of lost cognitive potential because this may only be apparent at a later period of neurological development. As the child matures, the nature of demands increases and it is only then that underlying deficits are revealed (Bowman et al., 1974 cited in Brooks, 1984). Case A's results are in keeping with the statement by Chadwick et

al. (1981) that impaired cognitive functioning has a cumulative effect on new learning and developmental lags appear when compared with peers. The comment was further supported throughout the research, not just at the individual but also at the between and within group level of analysis, highlighting the need for further longitudinal, prospective studies.

The case studies highlight the importance of not just considering between group differences and within group differences in studies of this nature but also individual differences. Furthermore, although group norms provide guidelines to inform decision making with regard to management, they have to be considered within a clinical framework taking into consideration the individual's history, collateral information and previous level of cognitive, academic, behavioural and emotional functioning.

5.4. Summary

In terms of rejecting and not rejecting the null hypothesis, the hypothesis of no difference between early adolescent rugby union players and non-contact sport controls (hockey, squash, cross-country, fencing) in terms of BRC, cognitive and academic functioning is rejected but the hypothesis of no difference in terms of early adolescent boys having sustained concussions showing a decline in cognitive and academic functioning relative to non-concussed controls is not rejected. The hypothesis of no difference for the non-compromised rugby players with two or more concussions matched for estimated IQ showing academic deficits relative to non-contact sport matched controls with no reported concussion over the three year period is rejected.

The hypothesis of no difference between participants with learning difficulties showing cognitive and academic deficits relative to participants with no learning difficulties is rejected but the hypothesis of no difference when the concussion variable is added is not rejected. Similarly, the researcher does not reject the hypothesis of no difference for concussed compromised/non-compromised participants showing cognitive and academic deficits relative to non-concussed compromised/non-compromised participants' matched for estimated IQ as well as when the rugby variable is added to the compromised/non-compromised data.

The current research shows a statistically significant decline over three years in academic functioning from junior school to high school of a small, high functioning, non-compromised, high SES, cohort of participants who play rugby and have had two or more concussions when compared with non rugby, non concussed participants. There is a statistically significant differences between the performance of controls versus rugby players on tests of abstract thinking, with the rugby players performing significantly worse than controls. It is theorized that difficulties relating to abstract thinking could impact on academic functioning in the long term because of the key role that abstract thinking plays in mediating management of more complex tasks when academic demands are increased. An additional finding is the statistically significant difference in the VOC scores, the score used to estimate BRC, between rugby players versus controls at baseline as well as rugby players with multiple concussions versus controls. In both instances controls performed better than the rugby players. Finally, there is a statistically significant difference between the rugby players reporting concussions versus controls. The rugby players reported more concussions than controls.

Despite the preliminary nature of the results, the results were obtained using a rigorous, well controlled, prospective, longitudinal research design. It is argued that when dealing with the developing brain and in particular insults occurring at critical stages of cognitive development, the use of this type of design is preferable to the use of a cross sectional alone or a combination of cross sectional and prospective designs because the latter design options provide fewer test opportunities within the context of protection from and vulnerability to insults, to assess the impact on cognitive and academic functioning. It is suggested that the current research provided an appropriate methodological vehicle for addressing the question of whether repeated mild traumatic brain injuries result in residual academic and cognitive deficits of early adolescents, taking into consideration that: as they mature, the nature of demands increases (Bowman et al., 1974 cited in Brooks, 1984); and that impaired cognitive functioning has a cumulative effect on new learning and developmental lags appear when compared with peers (Chadwick et al., 1981 cited in Brooks, 1984). Inclusion of the descriptive data and case studies in the research also showed how statistical data could obscure important clinical findings and that research of this nature should include not just group mean comparisons but also descriptive analyses, exploring within group differences, as well as case studies. The implications are that similar research on a much large population needs to be undertaken.

In **conclusion, statistical measures showed the following:** 1. The rugby group experienced significantly more concussions than controls. 2. The BRC estimate at baseline was significantly lower for the rugby group compared with controls. 3. The BRC estimate was significantly lower for the group with three or more reported concussions versus controls with no reported concussions. 4. There was a significant relationship between VOC and some academic and cognitive subtests. 5. The compromised group performed poorer than controls on the academic

tests but not on the cognitive tests. 6. There were significant results for time effects on some subtests and academic measures revealing minimal practice effects for abstract thinking. 7. The score for the abstract thinking subtest SIM was significantly lower for the rugby group compared with controls. 8. Over time when the effect of VOC was accounted for in the analysis of a non-compromised group, there was a significant difference in the overall academic performance between the rugby group with multiple concussions versus the control group with no concussions but not for specific cognitive subtests.

Descriptive measures showed that more rugby players than controls showed deficits after three years on some of the academic and cognitive tests and vice versa. Case studies showed that from a clinical perspective, despite significant test scores, one should not rely on test scores alone. Finally, given the significant results, the research showed that there is some impact on some aspects of cognitive and academic functioning of early adolescent RU players following repeated MTBI's.

5.5. Implications

5.5.1 Theoretical Implications

The study commenced with the hypothesis of no difference, between early adolescent rugby players and controls, in BRC, cognitive and academic functioning. BRC theory was used to underpin the study from the perspective that individuals differed in terms of their cognitive reserve which provided a threshold for vulnerability to and protection from insults to the brain and that in the face of repeated insults there is an aggregated effect (Satz, 1993 & 2001). The

theories of Piaget (1952b & 1970) and Erikson (1963 & 1968) provided the definition for the developmental stage and the development of formal operational thought, one of the tasks to be negotiated during this stage. Both theories, similar to the biological model proposed stages of development and Erikson like the biological proponents placed emphasis on critical stages of development i.e. development interrupted at a critical stage would have lasting consequences for further healthy, normal development. The notion of CR was also explored and it would appear that BRC and CR are, and are not, in agreement. Broadly speaking it would appear that they are saying the same thing but with different emphases. BRC appears to put more emphasis on an individual's innate ability or genetics whilst acknowledging the influence of psychosocial aspects whilst CR acknowledges that difference could be either or in terms of genetics or psychosocial environment (Stern, 2003). BRC is associated with threshold factors termed passive processes, which are in existence prior to the onset of injury or insult whilst CR is associated with compensation factors thus focusing on active aspects post insult (Stern, 2002 & 2003). The arguments of both have merits and are relevant. From a theoretical perspective, to better understand these arguments within the context of the research, the following hypothesis is explored:

The sampling of the experimental group and the control group was controlled in such a way that they were derived from the same high functioning, high SES parent population and there is the assumption, given the parent group status, the school and the education level at the school, that these participants would have relatively high threshold factors associated with BRC. It is argued that the only tangible difference is that one group is exposed to the game of rugby whilst the other group participates in different sporting codes. Considering, the BRC assumption and the research assumption of no difference between the groups with respect to SES, education, school,

age, grade, participation in sport, gender and language, there is the assumption of no difference between the participants in terms of BRC threshold factors.

However, at baseline the control group scores significantly higher than the rugby playing group, on the VOC test used to estimate BRC suggesting that the exposure to rugby and subsequent MTBIs lowered the protective factor for the rugby playing group and made them more vulnerable to further insults to the brain. It also suggests that compensation factors are limited at this stage. However, because their functioning is still within the average range albeit lower than controls one could also argue for some measure of protection. No significant differences between the groups on some cognitive and academic measures, despite the rugby group reporting significantly more concussions than controls, may suggest the presence of either protective factors in terms of BRC or compensation in terms of CR. In this regard it is hypothesized that where cognitive functioning was already established compensation was more likely to take place as was evident with no significant differences mentioned above but then it is also assumed that participants did have a reasonable measure of protection given the assumption of high BRC.

However, where cognitive functioning is still in a critical stage of development as in the case of the development of abstract thinking one could hypothesize that compensation may be hampered. Comparison between the groups showed that controls performed significantly better than rugby players on the test of abstract thinking and that there was very little practice effect for performance on this subtest when compared to significant time performances on all the other subtests. Descriptive data also showed that at baseline more rugby players showed deficits than previous and non rugby groups on the Similarities subtest and that after three years the percentage of rugby players that showed deficits increased whereas the non rugby and previous

rugby group numbers decreased. Given these findings it is hypothesized that: The rugby players were already compromised at baseline and this in turn lowered their reserve threshold in terms of a protective factor, further insults from multiple MTBIs resulted in an aggregated effect in terms of vulnerability which would impact on the development of abstract thinking at a critical stage of development given that early adolescence has been identified as a key stage of development for higher cortical functioning from a biological, psychosocial and cognitive development perspective. Given the many arguments that injury at a critical stage of development may have lasting effects and given the results showing poorer abstract thinking and academic functioning one may argue for support of this claim. However, descriptive analysis and trends for SIM when the three groups non-rugby, previous rugby and rugby are compared, findings show that although there was a greater percentage of deficits for the rugby players after three years, deficits for previously players declined and were not substantially different from non-rugby players after three years. This may suggests that once insults to the brain cease, compensation can take place and that protective factors given one's threshold still have a role to play. Notwithstanding this suggestion, if insults to the brain continue, it is hypothesized that compensation does not take place as in reflected in the decrease in percentages of deficits for the rugby group. This argument is further supported by the significant decline in academic functioning over a period of three years of rugby players with multiple concussions when compared with controls with no reported concussions where both groups are from a non-compromised cohort. In this cohort the rugby players performed better academically than controls at baseline but although the scores of controls, increased, rugby players scores, decreased. One can argue that at baseline despite this cohort having played rugby previously they still had a measure of reserve in terms of protective factors given their relatively high AGG scores, however, after repeated insults over a three year period and a significant decline in academic functioning versus controls one has to question the

role of compensation factors and consider the possibility of Satz (2001) argument that the acquisition of cognitive skills in different rates of development, will at the time of a head injury, be vulnerable to negative effects due to a future lowered critical threshold for impairment. There is also the argument for an aggregation effect of lowered brain reserve capacity increasing vulnerability to functional impairment because of the combination of pre-morbid vulnerability factors and either single or multiple episodes of neurological damage (Satz, 2001). It would appear that pre-morbidly this specific group was not vulnerable in terms of general intelligence or educational levels, however, one can argue that despite the assumption of protection and a high threshold, repeated insults appear to lower the threshold and increase the vulnerability factor.

5.5.2 Research Implications

The practical implications of the research are that, given the research design and the power afforded by the design, the sample was adequate, however, for more detailed levels of analyses at least 500 participants would be needed to provide a better statistical outcome. Larger numbers would be difficult to manage at one school in one grade because junior schools do not cater for such large numbers in one grade. In terms of this type of study it would be difficult to follow the same cohort for a number of years if the junior and high schools are not linked. Furthermore by adding additional schools a whole host of confounding variables would be introduced that would impact on the validity of the data. Although this particular study would have difficulty in terms of generalizability it is tightly controlled with regard to the presence of confounding variables. A further practical consideration would be the cost of the project and for future research of this nature funding would be essential.

From a research design perspective it is evident that the use of repeated measurements and the same subjects reduces experimental error thus making small differences translate into large effect sizes. Large effects, use of large samples and/or the use of sensitive experimental designs all impact on positively on power. There is also the argument, from a methodological perspective, for individual testing as opposed to group testing. However, given the large numbers of participants suggested, this would prove too impractical for one person to manage. Furthermore, increasing the number of researchers will, in turn add another confounding variable to the research process.

Given the statistically significant differences between the groups for SIM and AGG, with the tentative suggestions of insults to the brain during critical stages of development, having an impact on future functioning, there is the recommendation for the inclusion of more executive functioning tests in studies of this nature. It is also acknowledged that despite the significant findings, and Lezak (1995) condoning the use of a single subtest score as an estimate of premorbid IQ, that claims, regarding functioning and conclusions emanating from them, should not be made on the strength of results of one or two tests.

Implications, for future research, are recommendations for more prospective, longitudinal, well controlled large studies, supported by imaging studies to support or refute claims and hypotheses made in this and other studies with similar findings. Future studies should also control for the effect of VOC on the data, given the discussion of the impact of VOC on the data when it was and was not controlled for in terms of the Covariate analyses.

5.5.3 Policy Implications

The implications in terms of the South African context and the game played, are that players, parents, coaches, school management and clinicians should be alerted not only to the acute effects of concussion but also the subsequent risk of cognitive and academic deficits following multiple concussions. The risks of repeated concussions from playing rugby on the developing brain can be minimized by not allowing children and early adolescents to engage in the contact aspect of the game but instead to build on their skills by allowing them to play touch rugby or six down touch instead of the Rugby Union game of rugby. When children are older they can then make informed decisions whether they wish to pursue the game in stead of being forced into it by over zealous parents and coaches.

The Maroon et al. (2000) definition and guidelines used in the current research can be used as a vehicle to encourage debate regarding appropriate definitions, amongst SARFU officials. In terms of concussion management, SARFU may then put appropriate, informed definitions and policies in place for children and late adolescence. Since the school structure makes use of SARFU guidelines, changes at that level will evoke changes at school level.

Should the contact aspect of the game continue in its' current form at this age, on-field medical care should be available at games and practices. All players with the consent of the parents should undergo baseline assessment prior to commencing the sport. The clinical assessment should include the player's relevant history, collateral information and either computer based or paper and pencil testing. The latter may be more appropriate in the South African context.

Assessment of concussion should be conducted by trained personnel, to assess mental status and the concussed player should not return to the field of play. The incident should be followed up with a medical as well as neuropsychological assessment. Furthermore, a log book of concussed players should be kept by the school indicating the date, time and nature of the injury. This information should be passed on to the next school should the boy transfer to another school. Both the boy and his parents should be advised of the risks of residual cognitive and academic deficits following multiple concussions.

5.6. Critique of Current Research

Authors such as Banich et al., (1990), Bishop, (1997), Karmiloff-Smith, (1997), Karmiloff-Smith, (1998) and Thomas & Karmiloff-Smith, (2002) argued that there are huge gaps in the knowledge of functional plasticity due to the paucity of longitudinal data on children with early brain injury. In this regard they raised concerns because it was felt that the severity and nature of deficits in the developing brain are not known. Levine, Kraus, Alexander, Suriyakham & Huttenlocher (2005) also stressed that without longitudinal studies one could not determine whether a decline in IQ actually occurred or whether it was an artifact of cross-sectional sampling bias.

The current research makes some, albeit tentative contribution towards adding to our knowledge of the residual cognitive and academic deficits following numerous MTBI's at a critical stage of cognitive development. It also highlights how as individuals with repeated concussions mature, the demands of more complex tasks impact on their cognitive and academic functioning.

It answers the research questions and either rejects or fails to reject the null hypotheses of no difference. Although the research design is a sound one, it is complicated and makes the sorting and analyzing of such large volumes of data extremely challenging. The analyses, interpretation and reporting of all the different levels of analyses, to ensure transparency, also makes the results section cumbersome, and can be difficult for the reader to digest.

The findings of no significance or just trends in the concussion analysis compared with the rugby and the combined analysis could give one the impression that concussion is not a factor in the difference between rugby players and controls. Although an argument for the non-significance is offered in the discussion it could also be argued that it may suggest underreporting of concussions and that individuals with more serious concussions were not managed accordingly, highlighting dismissal of concussion effects as inconsequential. The study, because of it's longitudinal nature, highlighted the disregard for the effects of concussion by uncovering the drop over the three years in the number of concussion reported. In terms of the research, "rugby" appears to be a more reliable measure with the "concussion" measure possibly less reliable because of the largely subjective nature of reporting, despite coaches (at junior school level) indicating that boys had been concussed. In contrast to Shuttleworth-Edwards et al. (2004) argument for the absence of deficits in schoolboy rugby, the current research shows that in terms of deficits there is some impact on the cognitive and academic functioning of early adolescent rugby players sustaining repeated concussions.

5.7. Limitations of Current Research

Despite the design and the large effect sizes on can argue that a bigger N would have allowed more levels of analyses however, the sample size was restricted by the scope of the study. This was an unfunded doctoral thesis. Limitations in terms of the sample size were as follows: The study was prospective in nature and was aimed at early adolescents. Early adolescence commences at age 12-13 years and this therefore included grade 7 learners. Grade 7 learners were in their final year at preparatory school and needed to move to a high school for grades 8-12. There are very few schools where boys remain in the same schooling system i.e. where they attended the same preparatory and high schools. In terms of this study it was necessary for boys to remain in the same school and fortunately most learners did. It would have been very difficult to include participants from preparatory schools feeding a wide variety of high schools because of the logistics of arranging test situations for small rugby and non-contact sport control groups at schools across the peninsula. Not only would the task have been almost impossible, there would have been too many variables impacting on the data. This particular school only catered for 75 learners in grade 7 and for that reason an additional group was assessed the following year, bringing the total number of participants to 150. The addition of the second group added a year to the data collection and any additional groups would have had the same effect. An increased sample size amounting to 500 participants would have equated to another five years of 75 participants per year, each group tested for three years resulting in an additional 15 years added on to the existing 4 years of data collection. This scenario would have been beyond the scope of both a funded and unfunded doctoral thesis.

The prospective/longitudinal nature of the study could have been viewed as a limitation due to the natural attrition rate of boys leaving the school or decisions to cease participation. Generalizing to other populations is another limitation of this study as this study was done on a specific cohort, with a particular aim in mind in terms of following the same group, in the same context to minimize the influence of extraneous variables. In terms of generalizing the findings it is limited to elite, privileged schools, where all the boys speak English, play sport and where academic standards are high with grades generally in the average to high average range. Nevertheless, because of the above parameters the WISC III and WMS III were suitable assessment instruments in this South African context. Because of the limitation in terms of generalizability it is not possible to determine whether the same pattern of results would be evidenced in less advantaged and or disadvantage schools where academic and cognitive functioning may be more varied and the standard of education perhaps lower due to lack of resources. It could be argued that the research did not adequately deal with concussion data effectively because the incidence of concussion was not the focus in the analyses rather the participants reporting numbers of concussions.

References

Abreau, F., Templer, D. I., Schuyler, B. A., & Hutchison, H. T. (1990). Neuropsychological assessment of soccer players. *Neuropsychology, 4*, 175-181.

Ackerman, T. A. (2000). *Minor "dings"- major effects: A study into the cognitive effects of mild head injuries in high school rugby.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Alexander, M. P. (1995). Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. *Neurology, 45*, 1253-1260.

Alves, W., Macciocchi, S. N., & Barth, J. T. (1993). Postconcussive symptoms after uncomplicated mild TBI. *Journal of Head Trauma and Rehabilitation, 8*(3), 48-59.

American Academy of Pediatrics Policy Statement (AAPPS) (1998). Recommendations for participation in competitive sports. *The Physician and Sportsmedicine, 16,* 165-167.

Ancer, R. (1999). *Cumulative mild head injury in rugby: Cognitive test profiles of professional rugby and cricket players.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Annegers, J. F. (1983). The epidemiology of head trauma in children. In K. Shapiro (Ed.), *Pediatric head trauma* (pp. 1-10). Mount Kisco, NY: Futura.

Anderson, S. J. (1996). Sports-related head injuries: A neuropsychological perspective. *Sports Medicine, September,* 23-27.

Anderson, V. A., Catroppa, C., Morse, S. A., & Haritou, F. (1999). Functional memory skills following traumatic brain injury in young children. *Peadiatric Rehabilitation, 3*(4), 159-166.

Anderson, V. A., Northam, E., Hendy, J., & Wrennall, J. (2001). *Developmental Neuropsychology: A Clinical Approach.* Hove and New York: Psychology Press.

Asarnow, R. F., Satz, P., Light, R., Zaucha, K., Lewis, R., & McCleary, C. (1995). The UCLA study of mild head injury in children and adolescents. In S. Broman & M. Michel (Eds.), *Traumatic Head Injury in Children* (pp. 117-146). NY: Oxford University Press.

Bagley, L. J., Grossman, R. I., Galetta, S. L., Sinson, G. P., Kotapka, M., & Mc Gowan, J. C. (1999). Characterization of white matter lesions in multiple sclerosis and traumatic brain injury as revealed by magnetization transfer contour plots. *American Journal of Neuroradiology, 20*, 977-981.

Baker, R. J., & Patel, D. R. (2000). Sports related mild traumatic brain injury in adolescents. *Indian Journal of Pediatrics, 67*(5), 317-321.

Banich, M.T. Levine, S.C., Kim, H., & Huttenlocher, P. (1990). The effects of developmental factors on IQ in hemiplegic children. *Neuropsychologia 28,* 35-47.

Barth, J. T., Alves, W. M., Ryan, T. V., Macciocchi, S. N., Rimel, R., Jane, J. A., & Nelson, W. E. (1989). Mild head injury in sports: Neuropsychological sequelae and recovery of function. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), *Mild head injury* (pp. 257-275). Oxford University Press.

Barth, J. T., Macciocchi, S. N., Giordani, B., Rimel, R., Jane, J. A., & Boll, T. J. (1983). Neuropsychological sequelae of minor head injury. *Neurosurgery, 13*(5)*,* 529-532.

Belanger, H. G., Curtiss, G., Demery, J. A., Lebowitz, B. K., & Vanderploeg, R. D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society, 11,* 215-227.

Belanger, H. G., & Vanderploeg, R. D. (2005). The neuropsychological impact of sports-related concussion: A meta-analysis. *Journal of the International Neuropsychological Society, 11,* 345-357.

Benatar, S., Bhoola, K. D., Cleaton-Jones, P., De Klerk, W. A., Du Toit, D., & Herman, A., et al.(No date). *Guidelines on ethics for medical research: general principles.* Medical Research Council of South Africa.

Bender, S. D., Barth, J. T., & Irby, J. (2004). *Traumatic brain injury in sports. An international neuropsychological perspective.* The Netherlands: Swets & Zeitlinger Publishers.

Bennett, T. L., & Raymond, M. J. (1997). Mild brain injury: An overview. *Applied Neuropsychology, 4*(1), 1-5.

Beilinsohn, T. (2001). *Cumulative mild head injury in rugby: A comparison of cognitive deficit and postconcussive symptomatology between schoolboy rugby players and non-contact sport controls.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Bigler, E. D. (1990a). Neuropathology of traumatic brain injury. In E. D. Bigler (Ed.), *Traumatic brain injury.* Austin, TX: Pro-ed.

Bigler, E. D. (2000). The lesion(s) in traumatic brain injury: Implications for clinical neuropsychology. In R. Sugaraman (Ed.), *Clinical Neuropsychology Course Manual* (pp. 67-94). Cape Town: Afrox.

Bigler, E. D. (2001a). The lesion(s) in traumatic brain injury: Implications for clinical neuropsychology. *Archives of Clinical Neuropsychology, 16,* 95-131.

Bigler, E. D. (2001b). Quantitative magnetic resonance imaging in traumatic brain injury. *Journal of Head Trauma Rehabilitation, 16*, 117-134.

Bigler, E. D., & Orrison, Jr., W. W. (2004). Neuroimaging in sports-related brain injury. In M. R. Lovell, R. J. Echemendia, J. T. Barth, M. W. Collins (Eds.), *Traumatic brain injury in sports. An international neuropsychological perspective* (pp. 71-93). The Netherlands: Swets & Zeitlinger Publishers.

Binder, L. M. (1997). A review of mild head trauma. Part II: Clinical implications. *Journal of Clinical and Experimental Neuro psychology, 19* (3), 432-457.

Binder, L. M., Rohling, M. L., & Larrabee, G. J. (1997). A review of mild head trauma. Part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology, 19,* 421-431.

Bird, Y. N., Waller, A. E., Marshall, S. W., Alsop, J. C., Chalmers,D. J., & Gerrard, D. F. (1998). The New Zealand rugby injury and performance project: V. Epidemiology of a season of rugby injury. *British Journal of Sports Medicine, 32,* 319-325.

Bishop, D.V.M. (1997). Cognitive neuropsychology and developmental disorders: Uncomfortable bedfellows. *Quarterly Journal of Experimental Psychology: Section A 50,* 899-923.

Blakely, T. A., & Harrington, D. E. (1993). Mild head injury is not always mild. *Medical science and the law, 33*(3), 231-142.

Bold, L. (2000). *Cumulative mild head injuries in contact sport: A comparison of the cognitive profiles of rugby players and non-contact sport controls with normative data.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Boll, T. J. (1983). Minor head injury in children: out of sight but not out of mind. *Journal of Clinical Child Psychology, 12*(1), 74-80.

Bond, M. R. (1986). Neurobehavioural sequelae of closed head injury. In I. Grant & K. M. Adams (Eds.), *Neuropsychological Assessment of Neuropsychiatric Disorders.* NY: Oxford University Press.

Border, M. A. (2000). *Heads and tales - The effect of mild head injuries on rugby players: Cognitive deficits and postconcussive symptoms.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Brooks, N. (1984). Traumatic brain injury & psychological implications for long term recovery. In N. Brooks (Ed.), *Closed Head Injury: Psychological, Social and Family Consequences* (pp. 179-194). Oxford: Oxford University Press.

Cantu, R. C. (1986). Guidelines for return to contact sport after a cerebral concussion. *The Physician and Sportsmedicine, 20,* 55-66.

Cantu, R. C. (1998b). Return to play guidelines after a head injury. *Clinics in Sports Medicine, 17*(1), 45-60.

Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., & Holm, L., et al. (2004). Prognosis for mild traumatic brain injury: results of the WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation. Med., 43,* 84-105.

Casson, I. R., Siegel, O., Sham, R., Campbell, E. A., Tarlau, M., & DiDomenico, A. (1984). Brain Damage in modern boxers. *Journal of the American Medical Association,* 251, 2663-2667.

Chadwick, O., Rutter, M., Brown, G., Shaffer, D., & Traub, M. (1981). A prospective study of children with head injuries: II Cognitive sequelae. *Psychol Med, 11,* 49-61.

Chugani, H. T., Phelps, M. E., & Mazziotta, J. C. (1987). Positron emission tomography study of human brain functional development. *Annals of Neurology, 22*(4), 487-497.

Cicchetti, D. V. (1998). Role of null hypothesis significance testing (NHST) in the design of neuropsychological research. *Journal of Clinical and Experimental Neuropsychology, 20,* 293-295.

Clinical Interview, (2005, July 18). Personal Communication.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Earlbaum Associates.

Cohen, J. (1994). The earth is round (p< .05). *American Psychologist*, *49,* 997-1003.

Collie, A., Darby, D., & Maruff, P. (2001). Computerized cognitive assessment of athletes with sports related head injury. *British Journal of Sports Medicine, 35,* 297-302.

Collie, A., & Maruff, P. (2003). Computerized neuropsychological testing. *British Journal of Sports Medicine, 37(2).*

Collins, J. G. (1990). Types of injuries by selected characteristics: United States, 1985-1987. *Vital Health Statistics,* 175, 1-68.

Collins, M. W., Echemendia, R. J., & Lovell, M. R. (2004). Collegiate and high school sports. In M. R. Lovell, R. J. Echemendia, J. T. Barth, M. W. Collins (Eds.), *Traumatic brain injury in sports. An international neuropsychological perspective* (pp. 111-127). The Netherlands: Swets & Zeitlinger Publishers.

Collins, M. W., Grindel, S. H., Lovell, M. R., Dede, D. E., Moser, D. J., & Phalin, B. R., et al. (1999). Relationship between concussion and neuropsychological performance in college football players. *JAMA, 282*(10), 964-970.

Corsellis, J., Bruton, C., & Freeman–Browne, D. (1973). The aftermath of boxing. *Psychological Medicine, 3,* 270-303.

Council on Scientific Affairs. (1983). Brain injury in boxing. *Journal of the American Medical Association, 249* (2), 254-257.

Cremona-Meteyard, S. L., & Geffen, G. M. (1994). Persistent visuospatial attention deficits following mild head injury in Australian Rules football players. *Neuropsychologist, 32,* 649-662.

Critchley, M. (1957). Medical aspects of boxing, particularly from a neurological standpoint. *British Medical Journal, 1*, 357-362.

Dick, R. W. (1997). A summary of head and neck injuries in collegiate athletics using the NCAA Injury Surveillance System. In E. F. Hoerner (Ed.), *Head and Neck Injuries in Sports.* Philadelphia: American Society for Testing and Materials.

Dicker, G., & Maddocks, D. (1988). An objective measure of recovery from concussion in Australian Rules footballers. *The Australian Journal of Science and Medicine in Sport, December,* 17.

Dikmen, S., Machamer, J. E., Winn, H. R., & Temkin, N. R. (1995). Neuropsychological outcome at 1-year post head injury. *Neuropsychology, 9,* 80-90.

DeFord, S. M., Wilson, M. S., Rice, A. C., Clausen, T., Rice, L. K., & Barabnova, A. (2002). Repeated mild brain injuries result in cognitive impairment in B6C3F1 mice. *Journal of Neurotrauma, 19*(4), 427-438.

DeMarco, J., & Reeves, C. (1993). Injuries associated with soccer goalposts, United States, 1979-1993. *Journal of the American Medical Association, 271,* 1233.

De Villiers, J. C. (1987). Concussion in sport – how little is too much? *Proceedings of the Second South African Sports Medicine Association Congress, April,* pp. 164-168.

Dickinson, A. (1998). *Postconcussive sequelae in contact sport: Rugby versus non-contact sport controls.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Echemendia, R. J., & Cantu, R. C. (2004). Return to play following brain injury. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.). *Traumatic brain injury in sports. An international neuropsychological perspective* (pp. 479-498). The Netherlands: Swets & Zeitlinger Publishers.

Editorial. (2006, May 19). Headstart in handling sport injuries. *Weekend Argus,* p.17.

Editorial. (1998, May 23). A salute to Morne. *Eastern Province Herald,* p.6.

Epidemiology & Health Information Branch. (1993). *Injuries (with particular reference to head injuries)* (Information Circular No. 19E). Brisbane: Queensland Health.

Erikson, E. H. (1963). *Childhood and society* (2nd ed.). NY: Norton.

Erikson, E. H. (1968). *Identity, youth and crisis.* NY: Norton.

Estwanick, J. J., Boitano, M., & Ari, N. (1984). Amateur boxing injuries at the 1981 & 1982 USA/ABF national championship. *Physicians Sports Medicine, 12,* 123-128.

Ewing-Cobbs, L., Fletcher, J. M., & Levin, H. S. (1986). Assessment may fail to detect subtle classroom functioning. *Journal of Head Trauma Rehabilitation, 1,* 57-65.

Field, M., Collins, M. W., Lovell, M. R., & Maroon, J. (2003). Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes. *Journal of Paediatrics, 142,* 546-553.

Finkelstein, M. (1999). *The scrum-down on brain damage effects of cumulative mild head injury in rugby: A comparison of group mean scores between national rugby players and non-contact sport controls.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Fisher, J. (1985). Cognitive and behavioural consequences of closed head injury. *Seminars in Neurology, 5,* 197-204.

Flanagan, S. (1999). Psychiatric management of mild traumatic brain injury. *The Mount Sinai Journal of Medicine, 66*(3), 152-159.

Fletcher, J., Ewing-Cobbs, L., Francis, D., & Levin, H. (1995c). Variability in outcomes after traumatic brain injury in children: A developmental perspective. In S. H. Broman & M. E. Michel (Eds.), *Traumatic head injury in children* (pp. 3-21). New York: Oxford University Press.

Frencham, K. A. R., Fox, A. M., & Maybery, M. T. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology, 27,* 334-351.

Friedman, S. D., Brooks, W. M., Jung, R. E., Hart, B. L., & Yeo, R. A. (1998). Proton MR spectroscopic findings correspond to neuropsychological function in traumatic brain injury. *American Journal of Neuroradiology, 19,* 1879 -1885.

Garraway, W. M., Lee, A. J., Hutton, S. J., Russell, E. B. A. W., & Macleod, D. A. D. (2000). Impact of professionalism on injuries in rugby union. *British Journal of Sports Medicine,* 34, 348-351.

Garraway, M., & Macleod, D. (1995). Epidemiology of rugby football injuries. *The Lancet, 345,* 1485-1487.

Garnett, M. R., Blamire, A. M., Corkill, R. G., Cadoux-Hudson, T. A. D., Rajagopalan, B., & Styles, P. (2000a). Early proton magnetic resonance spectroscopy in normal-appearing brain correlates with outcome in patients following traumatic brain injury. *Brain*, *123*, 2046- 2054.

Garnett, M. R., Blamire, A. M., Rajagopalan, B., Styles, P., & Cadoux-Hudson, T. A. D. (2000b). Evidence for cellular damage in normal-appearing white matter correlates with

injury severity in patients following traumatic brain injury: A magnetic resonance spectroscopy study. *Brain*, *123*, 1403-1409.

Gennarelli, T. A. (1986). Mechanisms and pathophysiology of cerebral concussion. *Journal of Head Trauma Rehabilitation, 1,* 23-29.

Gennarelli, T. A., Seggawa, H. et al. (1982). Physiological response to angular acceleration of the head. In P. L. Gildenberg (Ed.), *Head Injury: Basic and Clinical Aspects* (pp. 129-140). New York: Raven.

Gennarelli, T. A., Thibault, L. E., & Graham, D. I. (1998). Diffuse axonal injury: An important form of traumatic brain damage. *Neuroscientist, 4,* 202-215.

Gentilini, M., Nichelli, P., Schoenhuber, R., Bortolotti, P., Tonelli, L., & Falasca, A. (1985). Neuropsychological evaluation of mild head injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 48,* 137-140.

Gerberich, S. G., Priest, J. D., Boen, J. R., Staub, C. P., & Maxwell, R. E. (1983). Concussion Incidences and Severity in Secondary School Varsity Football Players. *American Journal of Public Health, 73,* 1370-1375.

Giai-Coletti, C. (2002). *Rugby: more than just a game. A study of the cumulative effects of mild head injuries on high school rugby players.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Giza, C. C., & Hovda, D. A. (2004). The pathophysiology of traumatic brain injury. In M. R. Lovell, R. J. Echemendia, J. T. Barth & M. W. Collins (Eds.), *Traumatic brain injury in sports. An international neuropsychological perspective* (pp 45-70). The Netherlands: Swets & Zeitlinger Publishers.

Goldberg, E. (2002). *The executive brain: Frontal lobes and the civilized mind.* NY: Oxford University Press.

Golden, C. J., Espe-Pfeifer, P., & Wachsler-Felder, J. (2000). *Neuropsychological interpretations of objective psychological tests.* NY: Kluwer Academic/Plenum Publishers.

Goldstein, F. C., & Levin, H. S. (1987). Epidemiology of pediatric closed head injury: Incidence, clinical characteristics and risk factors. *Journal of Learning Disabilities,* 20, 518-525.

Grady, M. S., & McIntosh, T. C. (2002). Head Trauma. In A. K. Asbury et al. (Eds.), *Diseases of the Nervous System* (3rd ed.). Cambridge: Cambridge University Press.

Green, G. A., & Jordan, S. E. (1998). Are brain injuries a significant problem in soccer? *Clinics in Sports Medicine, 17*, 795- 809.

Grindel, S. H., Lovell, M. R., & Collins, M. W. (2001). The assessment of sport-related concussion: the evidence behind neuropsychological testing and management. *Clinical Journal of Sports Medicine, 11,* 134-143.

Gronwall, D., & Wrightson, P. (1974). Delayed recovery of intellectual function after minor head injury. *The Lancet, 2*, 605- 609.

Gronwall, D., & Wrightson, P. (1975). Cumulative effect of concussion. *Lancet, 2,* 995-997.

Gronwall, D., Wrightson, P., & McGinn, V. (1997). Effect of mild head injury during the preschool years. *Journal of the International Neuropsychological Society, 3,* 592-597.

Gulbrandsen, G. B. (1984). Neuropsychological sequelae of light head injuries in older children 6 months after trauma. *Journal of Clinical Neuropsychology, 6*(3), 257-268.

Guskiewicz, K. M., Weaver, N. L., Padua, D. A., & Garrett, W. E. Jr. (2000). Epidemeology of concussion in collegiate and high school football players. *American Journal of Sports Medicine, 28*(5), 643-650.

Harris, J. C. (1998). *Developmental neuropsychiatry (Volume II): Assessment, diagnosis and treatment of developmental disorders.* NY: Oxford University Press.

Hart, K. J., & Faust, D. (1988). Prediction of the effects of mild head injury: a message about the Kennard principle. *Journal of Clinical Psychology, 44*(5), 780-782.

Hawkridge S., Keyter L., & Steyn B. (2008). *Developmental Disorders.* In R. A., Emsley & W. P. Pienaar (Eds.) *Textbook of Psychiatry, Department of Psychiatry: Stellenbosch Univeristy (2nd ed.).* Stellenbosch: MHICSA publishers.

Heilbronner, R. L., & Ravdin, L. D. (2004). Boxing. In M. R. Lovell, R. J. Echemendia, J. T. Barth & M. W. Collins (Eds.), *Traumatic brain injury in sports. An international neuropsychological perspective* (pp 45-70). The Netherlands: Swets & Zeitlinger Publishers.

Heiman, G.A. (1995). *Research methods in psychology.* Boston: Houghton Mifflin Company.

Hinton-Bayre, A. D. (2000). Psychometric assessment and the management of concussion in contact sport. *Unpublished doctoral thesis.* University of Queensland, Australia.

Hinton-Bayre, A. D., & Geffen, G. M. (2004). Australian Rules Football and Rugby league. In Lovell, M. R., Echemendia, R. E., Barth, J. T. & Collins, M. W. (Eds.). *Traumatic brain injury in sports. An international neuropsychological perspective* (pp 169-192). The Netherlands: Swets & Zeitlinger Publishers.

Hinton-Bayre, A. D., Geffen, G. M., Geffen, L. B., McFarland, K. A., & Friis, P. (1999). Concussion in contact sports: reliable change indices of impairment & recovery. *Journal of Clinical and Experimental Neuropsychology, 21,* 70-86.

Hirschenfang, S. (1960b). A comparison of WAIS scores of hemiplegic patients with and without aphasia. *Journal of Clinical Psychology, 16*, 351.

Hofman, P., Stapert, S., van Kroooneneburgh, M., Jolles, J., de Kruijk, J., & Wilmink, J. (2001). MR Imaging, single-photon emission CT, and neurocognitive performance after mild traumatic brain injury. *American Journal of Neuroradiology,* 22, 441-449.

Howell, D. C. (1995). *Fundamental statistics for the behavioural sciences (3rd ed.).* USA: Wadsworth Publishing Company.

http://ca.encarta.msn.com (2007) *Encarta Dictionary* [Online]. Retrieved March 25, 2007. Available from http://ca.encarta.msn.com/dictionary_1861709802/scrum.html

Hughes, R. (1974). Head Damage. A warning to all players. *The Sunday Times,* Nov 10.

Hulicka, I. M. (1966). Age differences in Wechsler Memory Scale scores. *Journal of Genetic Psychology, 190,* 135-145.

Isherwood, I., Mawdsley, C., & Ferguson, F.R. (1966). Pneumoencephalographic changes in boxers. *Acta Radiology Diagnosis, 5,* 654-661.

Ivinskis, A., Allen, S., & Shaw, E. (1971). An extension of Wechsler Memory Scale norms to lower age groups. *Journal of Clinical Psychology, 27,* 354-357.

Jakoet, I. (2003). SA Rugby introduces stringent concussion guidelines. *www.sarugby.net.*

Jakoet, I., & Noakes, T. D. (1998). A high rate of injury during the 1995 Rugby World Cup. *South African Medical Journal,* 45-48.

Jenkins, L. W., & Marmarou, A., et al. (1986). Increased vulnerability of the traumatized brain to early ischemia. A. Unterberg, 273-282.

Jenkins, L. W., & Moszynski, K. et al. (1989). Increased vulnerability of the mildly traumatized brain to cerebral ischemia: The use of controlled secondary ischemia as a research tool to identify common or different mechanisms contributing to mechanical and ischemic brain injury. *Brain Research, I477,* 211-224.

Jennet, B., & Bond, M. (1975). Assessment of outcome after severe brain damage. A practical scale. *Lancet, i,* 480-484.

Jennett, B., & Frankowski, R. F. (1990). Epidemiology of head injury. In R. Brinkman (Ed.), *Handbook of Clinical Neurology,* (pp. 1-16). NY: Elsevier.

Jennet, B., & Macmillan, R. (1981). Epidemeology of head injury. *British Medical Journal, 282,* 101-104.

Johnson, D. (1992), Head injured children and education: A need for greater delineation and understanding. *British Journal of Educational Psychology, 62,* 404-409.

Jordan, B., Jahre, C., & Hauser, W. et al. (1992a). Serial computed tomography in professional boxing. *Journal of Neuroimaging, 2*, 181-185.

Kant, R., Smith-Seemiller, L., Isaacs, G., & Duffy, J. (1997). Tc-HM-PAO SPECT in persistent post concussion syndrome after mild head injury. Comparison with MRI/CT. *Brain Injury, 11,* 115-124.

Kaplan, E., Fein, D., Morris, R., & Delis, D. (1991). *WAIS-R as a neuropsychological instrument.* San Antonio, T. X.: The Psychological Corporation.

Karmiloff-Smith, A. (1997). Crucial differences between developmental cognitive neuroscience and adult neuropsychology. *Developmental Neuropsychology 13,* 513-524.

Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences 2,* 389-398.

Kaste, M., Kuurne, T., Vilkki, J., Katevuo, K., Sainio, K., & Meurala, H. (1982). Is chronic brain damage in boxing a hazard of the past? *The Lancet, 27,* 1186-1188.

Kelly, J. P., Nichols, J. S., Filley, C. M., Lillehei, K. O., Rubenstein D., & Kleinschmidt-DeMasters, B. K. (1991). Concussions in Sports: Guidelines for the prevention of catastrophic outcome. *Journal of the American Medical Association, 266*, 2867-2869.

Kemp, P. M., Houston, A. S., MacLeod, M. A., & Pethybridgge, R. J. (1995). Cerebral perfusion and psychometric testing in military amateur boxers and controls. *Journal of Neurology, Neurosurgery, and Psychiatry, 59,* 368-374.

Kesler, S. R., Adams, H. F., & Bigler, E. D. (2000). SPECT, MR & quantitive MR imaging: Correlates with neuropsychological and psychological outcome in traumatic brain injury. *Brain Injury, 14*, 851-857.

Kesler, S. R., Adams, H. F., Blasey, C .M., & Bigler, E. D. (2003). Premorbid Intellectual Functioning, Education, and Brain Size in Traumatic Brain Injury: An Investigation of the Cognitive Reserve Hypothesis. *Applied Neuropsychology, 10*(3), 153-162.

Killam, C., Cautin, R. L., & Santucci, A. C. (2005). Assessing the enduring residual neuropsychological effects of head trauma in college athletes who participate in contact sports. *Archives of Clinical Neuropsychology, 20*(5), 599-611.

Klonoff, P. S., & Lamb, D. G. (1998). Mild head injury, significant impairment on neuropsychological test scores, and psychiatric disability. *The Clinical Neuropsychologist, 12,* 31-42.

Klonoff, H., Low, M. D., & Clark, C. (1977). Head injuries in children: A prospective five year follow-up. *Journal of Neurology, Neurosurgery, and Psychiatry, 40,* 1211-1219.

Kolb, B., & Whishaw, I. Q. (2003). *Fundamentals of Human Neuropsychology(5th ed.).* New York: Worth Publishers.

Kraus, J. F., Fife, D., Cox, P., Ramstein, K., & Conroy, C. (1986). Incidence, severity, and external causes of pediatric brain injury. *American Journal of Epidemiology, 119,* 186-201.

Kraus, J. F., & Norjah, P. (1988). The epidemiology of mild, uncomplicated brain injury. *The Journal of Trauma, 28,* 1637-1643.

Kraus, J. F., & Arzemanian, S. (1989). Epidemiologic Features of Mild & Moderate Head Injury, pp. 9-28. Boston: Blackwell Scientific Publications.

Kraus, J. F., Mc Arthur, D. L., Silverman, T. A., & Jayaraman, M. (1996). Epidemiology of Head Injury. In R. K. Narayan, J. E. Wilberger & J.T. Povlishock (Eds.), *Neurotrauma* (pp. 13-30). San Francisco: Mc Graw-Hill.

Leatham, J. M., & Body, C. M. (1997). Neuropsychological sequelae of head injury in a New Zealand adolescent sample. *Brain Injury, 11*(8), 565-575.

Lee, S. M., Lifshitz, J. et al. (1995). Focal cortical-impact injury produces immediate and persistent deficits in metabolic autoregulation. *Journal of the Cerebral Blood Flow Metabolism, 15,* 722 (Abstract).

Levin, H., Ewing-Cobbs L., & Eisenberg, H. (1995). Neurobehavioural outcome of pediatric closed head injury in children. In Broman, S. H., Michel, M. E. (Eds.), Traumatic Head Injury in Children. New York, NY: Oxford University Press, pp. 70-94.

Levin, H. S., & Eisenberg, H. M. (1979). Neuropsychological impairment after closed head injury in children and adolescents. *Journal of Pediatric Psychology, 4*, 389-402.

Levin, H. S., Mattis, S. Ruff, R. M., Eisenberg, H. M., Marshall, L. F., Tabaddor, K., High, W. M., & Frankowski, R. F. (1987). Neurobehavioural outcome of minor head injury: A three center study. *Journal of Neurosurgery, 66,* 234-243.

Levine, S. C., Kraus, R., Alexander, E., Suriyakham, L. W., & Huttenlocher, P. R. (2005). IQ decline following early unilateral brain injury: A longitudinal study. *Brain & Cognition, 59*(2), 114-123.

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

Lezak, M.D. (1995). *Neuropsychological assessment.* NY: Oxford University Press.

Lifshitz, J., Pinanong, P. et al. (1995). Regional uncoupling of cerebral blood flow and metabolism in degenerating cortical areas following a lateral cortical contusion. *Journal of Neurotrauma, 12*, 129 (Abstract).

Lishman, W.A. (2002). *Organic psychiatry: The psychological consequences of cerebral disorder.* (3rd ed.). Oxford: Blackwell Scientific Publications.

Liu, Y. K. (1999). Biomechanics of "low-velocity impact" head injury. In N. R. Varney & R. J. Roberts (Eds.), *The Evaluation and treatment of mild traumatic brain injury.* Mahwah, N. J.: Erlbaum.

Long, C. J., & Williams, J. M. (1988). Neuropsychological Assessment and the treatment of head trauma. In H. A. Whitaker (ed.). Springer Verlag, New York. 132-160.

Lovell, M. R., & Collins, M. W. (1998). Neuropsychological assessment of the college football player. *Journal of Head Trauma Rehabilitation, 13*(2), 9-26.

Lovell, M. R., Collins, M. W., Iverson, G. L., Field, M., Maroon, J., Cantu, R., Podele, K., Powell, J., Belza, M., & Fu, F. H. (2003). Recovery from mild concussion in high school athletes. *Journal of Neurosurgery, 98,* 296-301.

Lovell, M. R., Echemendia, R. J., Barth, J. T., & Collins, M. W. (2004). *Traumatic brain injury in sports. An international neuropsychological perspective.* The Netherlands: Swets & Zeitlinger Publishers.

Machiocchi, S. N., Barth, J. T., & Littlefield, L. M. (1998). Outcome after head injury. *Clinics in Sports Medicine, 17*(1), 27-36

Macleod, D. A. D. (1993). Risks & injuries in rugby football. In G. R. McLatchie & C. M. E. Lennox (Eds.). *The soft tissues. Trauma & sports injuries* (pp.371-381). London: Butterworth Heinemann Ltd.

Maddocks, D. L. (1995). Is cerebral concussion a transient phenomenon?. *The Medical Journal of Australia 162,*167.

Maddocks, D., & Saling, M. (1996). Neuropsychological deficits following concussion. *Brain Injury, 10,* 99-103.

Maddocks, D., Saling, M., & Dicker, G. (1995). A note on normative data for a test sensitive to concussion in Australian Rules footballers. *Australian Psychologist, 30,* 125-127.

Makdissi, M., Collie, A., Maruff, P., Darby, D. G., Bush, A., McCrory, P., & Bennell, K. (2001). Computerized cognitive assessment of concussed Australian Rules footballers. *British Journal of Sports Medicine, 35,* 354-360.

Maroon, J. C., Lovell, M. R., Norwig, A. T. C., Podell, K., Powe, J. W., & Hartl, R. (2000). Cerebral concussion in athletes: Evaluation & neuropsychological testing. *Neurosurgery, 47*(3), 659-669.

Marshall, L. F. (1989). *A Neurosurgeon's View of the Epidemiology of Minor & Moderate Head Injury,* pp. 29-34. Boston: Blackwell Scientific Publications.

Matser, E. J. T., Kessels, A. G. H., & Lovell, M. R. (2004). Soccer. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports. An international neuropsychological perspective* (pp.193-208). The Netherlands: Swets & Zeitlinger Publishers.

Matser, E. J. T., Kessels, A. G. H., Jordan, B. D., Lezak, M. D., & Troost, J. (1998). Chronic traumatic brain injury in professional soccer players. *Neurology, 51*, 791-796.

Matser, E. J. T., Kessels, A. G. H., Lezak, M. D., Jordan, B. D., & Troost, J. (1999). Neuropsychological impairment in amateur soccer players. *Journal of the American Medical Association. 282,* 971-973.

Mattiello, J. A., & Munz, M. (2001). Four types of acute post-traumatic intracranial hemorrhage. *New England Journal of Medicine, 344,* 580.

Mc Allister, T. W. (1994). Mild traumatic brain injury & the postconcussive syndrome In J. M. Silver, S. C. Yudofsky, & R. E. Hales (Eds.) *Neuropsychiatry of Traumatic Brain Injury,* pp. 357-392. Washington, DC: American Psychiatric Press.

Mc Allister, T. W., Saykin, A. J., Flashman, L. A., Sparling, M. B., Johnson, S. C., Guerin, S. J. et al. (1999). Brain activation during working memory 1 month after mild traumatic brain injury: A functional MRI study. *Neurology,* 53, 1300-1308.

McCown, I. A. (1959). Boxing Injuries. *American Journal Surgery,* 98, 509.

McCrory, P. R., Bladin, P. F., & Berkovic, S. F. (1997). Retrospective study of concussive convulsions in elite Australian rules & rugby league footballers: phenomenology, aetiology and outcome. *British Medical Journal, 314,* 171-174.

McQuillan, R. J. M. (1992). A survey of rugby injuries attending an accident and emergency department. *Irish Medical Journal, 85*(2), 72-73.

Mira, M. P., Tucker, B. F., & Tyler, J. S. (1992). *Traumatic brain injury in children and adolescents.* Texas: Pro-ed.

Mitrushina, M. N., Boone, K. B., & D'Elia, L. F. (1999). *Handbook of normative data for neuropsychological assessment.* NY: Oxford University Press.

Mittenberg, W. Zielinski, R., & Fichera, S. (1993). Recovery form mild head injury: A treatment manual for patients. *Psychotherapy in Private Practice, 12*(2), 37-52.

Mittl, R., Grossman, R., Hiehle, J., Hurst, R., Kauder, D., Gennarelli, T., & Alburger, G. (1994). Prevalence of MR evidence of diffuse axonal injury in patients with mild head injury & normal CT findings. *American Journal of Neuroradiology, 15,* 1583-1589.

Moser, R. S., Schatz, P., & Jordan, B. D. (2005). Prolonged effects of concussion in high school athletes. *Neurosurgery*, *57*(2), 300-306.

Mueller, F. O., & Cantu, R. C. (1999). Catastrophic injuries & fatalities in high school and college sports, 1982-1988. *Medicine & Science in Sports and Exercise, 22,* 737-741.

Muller, R. A., Chugani, H. T., Muzik, O. et al.(1998a). Brain organization of motor and language functions following hemispherectomy: A [(15)O]- water positron emission tomography study. Journal of Child Neurology.

Muller, R. A., Rothermel, R., Behen, M., et al. 1998b. Brain organization of language after early unilateral lesion: A PET study. Brain and Language: *62*, 422-451

Nass, R. (2002). Plasticity: mechanisms, extent & limits. In S. J. Segalowitz & I. Rapin (Eds.), *Handbook of Neuropsychology (2nd ed.) Vol 8 (1), Child Neuropsychology, part 1* (pp. 29-68). Amsterdam: Elsevier.

Nathan, M., Goedeke, R., & Noakes, T. D. (1983). The incidence and nature of rugby injuries experienced at one school during the 1982 rugby season. *South African Medical Journal, 64,* 132-137.

National Health & Medical Research Council. (1994). *Football injuries of the head and neck.* Canberra: Australian Government Publishing Service.

Nedd, K., Sfakianakis, G., Ganz, W., Urriccho, B., Ernberg, D., Villanueva, P., Jabir, A., Bartlett, J., & Keena, J. (1993). 99m Tc-HMPAQ SPECT of the brain in mild to moderate traumatic brain injury patients: Compared with CT-a prospective study. *Brain Injury,* 7, 469-479.

Nestvold, K., Lundar, T., Blikra, G., & Lennum, A. (1988). Head injuries during one year in a central hospital in Norway: A prospective study. *Neuroepidemiology, 7,* 134-144

Newman, B. M. & Newman, P. R. (1987). *Development through life: A psychosocial approach.* California: Brooks/Cole Publishing Company.

Newton, M., Greenwood, R., Briton, K., Charlesworth, M., Nimmon, C., Carroll, M., & Dolke, G. (1992). A study comparing SPECT with CT and MRI after closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 55,* 92-94.

Nitkin, R. (2000). Dendritic mechanisms in brain function and developmental disabilities. Cerebral Cortex: *10*, 925-926.

Oppenheimer, D. R. (1968). Microscopic lesions in the brain following head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 31*, 299-306.

Ormond-Brown, D. S. (2006). Head injury in South Africa. *The Specialist Forum, 6,* 34-38.

Pang, D. (1989).Physics and pathophysiology of closed head injury. In M. Lezak (Ed.) *Assessment of the Behavioiural Consequences of Closed Head Injury* Vol 7. *Frontiers of clinical neuroscience.* New York: Alan R. Less.

Parker, R. S. (2001). *Concussive brain trauma: Neurobehavioural impairment & maladaptation* Florida: CRC Press.

Peerless, S. J., & Rewcastle, N. B. (1967). Shear injuries of the brain. *Canadian Medical Association Journal, 96* (10), 577-582.

Piaget, J. (1970). Piaget's theory. In P.H. Mussen (Ed.). *Carmichaels's Manual of Child Psychology* (3rd ed. Vol 1). New York: Wiley.

Piaget, J. (1952b). *Judgmental reasoning in the child*. New York: Humanities Press. (Original work published in French 1924).

Polissar, N., Fay, G., Jaffe, K., Liao, S., Martin, K., Shurtleff, H., Rivara, J., & Winn, H. (1994). Mild pediatric traumatic brain injury: Adjusting significance levels for multiple comparisons. *Brain Injury, 8,* 249-264.

Powell, J. W., & Barber-Foss, K. D. (1999). Traumatic brain injury in high school athletes. *Journal of the American Medical Association, 282*, 958-963.

Quality Standards Subcommittee, American Academy of Neurology. (1997). Practice Parameter: The management of concussion in sports (summary statement). *Neurology, 48,* 581-585.

Reid, I. (1998). *Tackling mild head injury in rugby: A comparison of the cognitive profiles of professional rugby & cricket players.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Reid, I., Shuttleworth-Jordan, A. B., Ancer, R., Dickinson, A., Radloff, S., & Jakoet, I. (1999). To scrum or not to scrum: First report from the RU-SARFU rugby head injury study. *Journal of the International Neuropsychology Society, 5,* 283. Abstract.

Rimel, R. W., Giordani, B., Barth, J. T., Boll, T. J., & Jane, J. A. (1981). Disability caused by minor head injury. *Neurosurgery, 9*(3), 221-228.

Rimel, R. W., Giordani, B., Barth, J. T., Boll, T. J., & Jane, J. A. (1982). Moderate head injury: Completing the clinical spectrum of brain trauma. *Neurosurgery, 11,* 344-351.

Ross, R., Cole, M., Thompson, J., & Kim, K. (1983). Boxers-computed tomography, EEG and neurosurgical evaluation. *Journal of the American medical Association,* 249, 211-213.

Roux, C., Goedeke, R., Visser, G. R., Van Zyl, W. A., & Noakes, T. D. (1987). The epidemiology of schoolboy rugby injuries. *South African Medical Journal, 71*, 307-313.

Roy, S. P. (1974). The nature and frequency of rugby injuries. *South African Medical Journal, 85,* 2, 2321-2327.

Ruff, R. M., Levin, H. S., & Marshall, L. F. (1986). Neurobehavioural methods of assessment and the study of outcome in minor head injury. *Journal of Head Trauma & Rehabilitation, 1*(2), 43-52.

Ruijs, M., Gabreels, F., & Thijssen, H. (1994). The utility of electroencephalography and cerebral computed tomography in children with mild & moderately severe closed head injuries. *Neuropediatrics, 25,* 73-77.

Runyon, R. P., & Haber, A. (1980). *Fundamentals of behavioural statistics* (4th ed.). Reading, Massachusetts: Addison-Wesley Publishing Company.

Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed.). J. M. Sattler, Publisher, Inc.

Satz, P. (2001). Mild head injury in children and adolescents. *Current Directions in Psychological Science, 10,* 106 – 109.

Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for threshold theory. *Neuropsychology, 7,* 273-295.

Schmand, B., Smit, J. H., Geerlings, M. I., & Lindeboom, J. (1997). The effects of intelligence and education on the development of dementia: A test of the brain reserve hypothesis. *Psychological Medicine, 27*(6), 1337-1344.

Schneider, R. C., Kennedy, J. C. et al. (1985). *Sports Injuries.* Baltimore: Williams & Wilkins.

Segalowitz, S. J., & Lawson, S. (1995). Subtle symptoms associated with self-reported mild head injury. *Journal of Learning Disabilities, 28,* 309-319.

Seward, H., Orchard, J., Hazard, H. & Collinson, D. (1993). Football injuries in Australia at the elite level. *The Medical Journal of Australia, 159,* 298-301.

Sherwood, L. (2007). *Human physiology: From cells to systems* (4th ed.). USA: Thomson Brooks/Cole.

Shuttleworth-Edwards, A. B. (2001). Personal Communication.

Shuttleworth-Edwards, A. B., Ackermann, T., Beilinsohn, T., Border, M., & Radloff, S. (September, 2001a). *A study on the effects of cumulative mild head injury in high school rugby.* Paper presented in Shuttleworth-Edwards, A. B. (Chair), Sports related head injury. Symposium convened at the 8th National Conference of the SA Clinical Neuropsychological Association (SACNA), University of Cape Town.

Shuttleworth-Edwards, A. B., Border, M., Reid, I., & Radloff, S. (2004). Additive effects of concussion in South African rugby union: A case of a little becoming too much. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Sports Neuropsychology* (pp.1-29). The Netherlands: Swets & Zeitlinger Publishers.

Shuttleworth-Edwards, A. B., Border, M., Reid, I., & Radloff, S. (2004). South African Rugby Union. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports. An international neuropsychological perspective* (pp.149-168). The Netherlands: Swets & Zeitlinger Publishers.

Shuttleworth-Jordan, A. B. (1999). When a little becomes too much: A prospective theoretical context for cumulative mild head injury effects. A response to apparently null outcomes. *A paper present at the 22nd mid-year meeting of the International Neuropsychological Society 16th European Conference, Island of Madeira, Portugal.*

Shuttleworth-Jordan, A. B., Balarin, E., & Puchert, J. (1993). *Mild head injury effects in rugby: Is playing the game really worth the cost?* Paper presented at the International Neuropsychological Society's 16th European Conference, Island of Madeira (Portugal).

Shuttleworth-Jordan, A. B., & Bode, S. G. (1995). Taking account of age-related differences on digit symbol and incidental recall for diagnostic purposes. *Journal of Clinical and Experimental Neuropsychology, 17*(3), 439-448.

Shuttleworth-Jordan, A. B., Puchert, J., & Balarin, E. (1993). Negative consequences of mild head injury in rugby: a matter worthy of concern. In R. Plunkett & S. Anderson (Eds.). *Proceedings of the 5th National Neuropsychology Conference.* Durban: South African Neuropsychological Association (SACNA).

Sinson, G. P., Bagley, L. J., Cecil, K. M., Torchia, M., Mc Gowan, J. C., Lenkinski, R. E. et al. (2001). Magnetization transfer imaging and proton MR spectroscopy in the evaluation of axonal injury: Correlation with Clinical outcome after traumatic brain injury. *American Journal of Neuroradiology, 22,* 143-151.

Sortland, O., & Tysvaer, A. T. (1989). Brain Damage in former association football players. An evaluation by cerebral computed tomography. *Neuroradiology, 31,* 44-48.

Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative neuropsychological approach.* New York: Guilford Press.

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

Stern, Y. (2003). The concept of cognitive reserve: A catalyst for research. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 589-593.

Sutton, R. L., Hovda, D. A. et al. (1994). Metabolic changes following cortical contusion: Relationships to edema and morphological changes. *A Ca Neurochir, 6,* 446-448.

Symonds, C. (1962). Concussion and its sequelae. *Lancet, 1*, 1-5.

Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. *Lancet, ii,* 81-84.

Teuber, H. L. (1974). Why two brains? In Schmitt F., Worden F., (Eds.), *The Neurosciences: Third Study Program.* Cambridge, MA: MIT Press, pp. 71-74.

Thomas, M., & Karmiloff-Smith, A. (2002). Is developmental disorder like cases of adult brain damage? Implications from connectionist modeling. *Behavioral and Brain Sciences 25,* 727-787.

Thurman, D., & Guerrero, J. (1997). Trends in hospitalization associated with traumatic brain injury. *Journal of the American Medical Associated, 282*(10), 954-757.

Thurman, D. J., Alverson, C., Browne, D., et al. (1999). Traumatic brain injury in the United States. *A report to Congress*. Atlanta: Centers for Disease Control and Prevention, U.S. Department of Health and Human Services.

Thurman, D. J., Branche, C. M., & Sniezek, J. E. (1998). The epidemiology of sports related traumatic brain injuries in the United States: Recent developments. *Journal of Head Trauma Rehabilitation, 13*(2), 1-8.

Tysvaer, A. T. (1992). Head and neck injuries in soccer. Impact of minor trauma. *Sports Medicine, 14,* 200-213.

Tysvaer, A. T., & Lochen, E. A. (1991). Soccer injuries to the brain. A neuropsychologic study of former soccer players. *The American Journal of Sports Medicine, 19,* 56-60.

Tysvaer, A. T., Storli, O., & Bachen, N. I. (1989). Soccer injuries to the brain. A neurlogic and electroencephalographic study of former players. *Acta Neurologica Scandinavia, 80,* 151-156.

Van De Graaff, K. M., (2002). *Human Anatomy: Skeletal system introduction and Axial skeleton.* New York: McGraw-Hill Companies.

Van Heerden, J. J. (1976). n Ontleding van rugbybeserings. *South African Medical Journal, 50,* 1374-1379.

Van Zijl, S., Johnson, B., Benatar, S., Cleaton-Jones, P., Netshidzivhani, P., & Ratsaka-Mothokoa, M., et al. (2005). *Ethics in health research: principles, structures and processes.* Department: Health Republic of South Africa.

Walsh, K. W. (1985). *Understanding brain damage.* Edinburgh: Churchill-Livingstone.

Walsh, K. W. (1994). *Neuropsychology: A Clinical approach.* Edinburgh: Churchill-Livingstone.

Ward, J. D. (1989). Pediatric Head Injuries: Special Considerations. In D. P. Becker & S. K Gudeman. *Textbook of Head Injury,* pp. 319-325. Philadelphia: Saunders.

Wechsler, D. (1997). *Wechsler Memory Scale-Third Edition.* San Antonio, TX: The Psychological Corporation.

Wekesa, M., Asembo. J. M., & Njororai, W. W. S. (1996). Injury surveillance in a rugby tournament. *British Journal of Sports Medicine, 30,* 61-63.

Welsh, M. C., & Pennington, B. F. (1988). Assessing frontal lobe functioning in children: views from developmental psychology. *Developmental Neuropsychology, 4*(3), 199-230.

Wilberger, J. E. (1993). Minor head injuries in American football. Prevention of long term sequelae. *Sports Medicine, 15,* 338-343.

Wilson, J., Wiedemann, K., Hadley, D., Condon, B., Teasdale, G., & Brooks, D. (1988). Early and late magnetic resonance imaging & neuropsychological outcome after head injury. *Journal of Neurology, Neurosurgery, & Psychiatry, 51,* 391-396.

Winogron, H., Knights, R., & Bawden, H. (1984). Neuropsychological deficits following head injury in children. *Journal of Clinical Neuropsychology, 6,* 269-286.

Wrightson, P. (2000). The development of a concept of mild head injury. *Journal of Clinical Neuroscience, 7*(5), 384-388.

Wrightson, P., & Gronwall, D. (1999). *Mild head injury.* Oxford: Oxford University Press.

Yarnell, P. R., & Lynch S. (1970). Retrograde memory immediately after concussion. *Lancet, 1,* 863-864.

Yeats, K. O., Luria, J., Bartkowski, H., Rusin J., Martin L., and Bigler, E. D. (1999). Postconcussive symptoms in children with mild closed head injuries. *Journal of Head Trauma Rehabilitation, 14*, 337-450.

web.uccs.edu/lbecker/Psy590/es.htm#Cohen (2008). *Effect size.* [Online]. Retrieved June 18, 2008.
Available from
http://web.uccs.edu/lbecker/Psy590/es.htm#Cohen

www.rfu.com (2004). *England Rugby Supporters Club*. [Online]. Retrieved March 25, 2007.
Available from
http://www.rfu.com/ersc/index.cfm/fuseaction/ERSC.Home/storyID/4790/storytypeID/74/

Jakoet, I. (2006). South Africa rugby guidelines on the management of concussion. Retrieved June 30, 2006 from the World Wide Web: *http://www.sarfu.org.za.*

Zakzanis, K. K. (1998). Methodological commentary: Brain is related to behaviour ($p<.05$). *Journal of Clinical and Experimental Neuropsychology, 3,* 419-427.

Zemper, E. (1994). Análisis of cerebral frequency with the most commonly used models of football helmets. *Journal of Athletic Training, 29*(1), *44-50.)*

Zimbardo, P. G. (1992). *Psychology and life.* USA: Harper Collins Publishers.

APPENDIX A

HEADMASTER CONSENT FORM

I, _____ headmaster of _____ have been informed of the concussion in schoolboy rugby research project which will be conducted by Mrs Debbie Alexander, a senior clinical psychologist/lecturer in the Department of Child and Family Psychiatry at Tygerberg Hospital/University of Stellenbosch and University of the Western Cape. I am aware that the research is in fulfillment of the requirements for the degree of PhD (PSYCHOLOGY) at the University of the Western Cape.

I understand that:

1. The research involves assessing the impact of repeated concussions on the cognitive and academic functioning of adolescent boys. Comparisons in terms of that functioning are made between rugby playing and non-rugby playing boys and boys who have been concussed and those who have not.

2. The research was moved from Rhodes University to the University of the Western Cape, by the researcher for logistical reasons.

3. The research involves all volunteering Grade 9 ex-XXXXX preparatory boys, who will be assessed using internationally validated paper and pencil cognitive tests. Their English, mathematics and overall academic scores will form part of the test battery scores. The initial pre and post-season assessments took place at the preparatory school. The assessment (post winter sport season) will take place in November of this year, on the school property. In addition, pupils will be requested to fill out a brief questionnaire with questions relevant to the research.

4. This study does not interfere with or substitute for good medical practice. It is therefore advised that all schoolboys with concussion be seen as soon as possible by their general practitioner or other medical practitioners and should not return to contact sport for at least 3 weeks from the time of injury and thereafter on the advice of the medical practitioner.

5. Participation in the research is strictly voluntary, parents have the right to withdraw their sons from the study at any stage without penalty and boys have the right to withdraw at any stage without penalty. Likewise the school may stop the study temporarily or permanently at any stage without penalty.

6. The research and academic information collected on individual schoolboys will remain **anonymous** and **strictly confidential.** No boy's name will be mentioned in the thesis or any publication. Confidentiality about individual participants will be preserved, through the use of coding throughout the research. Information will only be made available to the boy, his parents and/or a medical practitioner on request with the boy and the parents' permission. This information may form part of the management decision in individual

cases. However, the researchers will not be held accountable for medical decisions made by medical practitioners or parents on the basis of that information.

7. Data arising out of this project will be used for thesis and publication purposes only by the collaborating university.

I hereby give consent for those pupils who will be participating in this research project to be assessed by the above mentioned researcher.

Signed : _____

Dated : _____

APPENDIX B

28th October 2005

CONCUSSION RESEARCH

Dear Parents

In keeping with our goals to maximize the safety of sports players generally, and particularly in the contact sports where there is a known risk of concussion, we plan to continue with the risk prevention strategies for concussion. One of our objectives is to support the concussion study, commenced in 2002/3 at the preparatory school. The study aims to investigate the impact of repeated concussions on the cognitive and academic functioning of early adolescent rugby union players and involves both rugby playing boys and non rugby playing boys.

The research is conducted by Mrs D. Alexander a senior clinical psychologist/lecturer in the Department of Child and Family Psychiatry at Tygerberg Hospital/University of Stellenbosch and University of the Western Cape. The research is in fulfillment of the requirements for the degree of PhD (PSYCHOLOGY) at the University of the Western Cape. The research, as you may recall was registered at Rhodes University but has since been moved to the University of the Western Cape, by the researcher for logistical reasons.

Your sons are the first schoolboys to participate in a prospective study of this nature. As you know, they were assessed at the preparatory school in 2003 pre and post winter sport season and post season at the college in 2004, using paper and pencil tests. Post season paper and pencil screening continue for the last time in the 4th term of this year.

Screening involves the evaluation of cognitive functions such as verbal/auditory and visual skills, attention and concentration, memory, fine motor control and processing speed. Overall academic, English and mathematics scores will also be required to assess the impact on academic functioning. These exercises are usually enjoyed by the boys and take approximately 60 minutes to complete. In addition, pupils will be requested to fill out a brief questionnaire relevant to the research. We wish to emphasize that the project is neither invasive nor harmful to the child's physical, mental and or emotional well-being. Participation is entirely voluntary and parents may withdraw their son at any time without penalty

The data will be examined by researchers at the University of the Western Cape. The results of the research will be used by the collaborating university for scientific publication purposes only. The information collected on individual schoolboys will remain **anonymous** and **strictly confidential.** No boy's name will be mentioned in the thesis or any publication. Confidentiality about individual participants will be preserved, through the use of coding throughout the research. Information will only be made available to boys, parents and/or medical practitioner with parents' consent, on request. This information may form part of the management decision in individual cases. However, the researcher will not be held accountable for medical decisions made by medical practitioners or parents on the basis of that information.

It is important to be aware that this study does not interfere with or substitute for good medical practice. We therefore advise that all schoolboys with concussion be seen as soon as possible by their general practitioners and should not return to contact sport for at least 3 weeks from the time of injury (SARFU guidelines) and thereafter on the advice of the medical practitioner.

Should your son **not participate in any formal sport** activity, his **participation is still of benefit** to the research. This is in order to make comparisons between boys who are exposed to sports concussion, and those who are not exposed.

Your son's participation in the research is **strictly voluntary**, you have the right to withdraw him from the entire project or any part thereof and he can withdraw at any time without penalty. Withdrawal of your son from the project will not prejudice him in any way.

Should you **agree** to your son's participation **please complete the consent form. The form can be handed to Mr XXXXXX before or on 11 November 2005.**

The assessment will take place on 11 November at 09H00.

Should you require further information or wish to discuss the research, please feel free to approach Mrs Alexander. Mrs Alexander is a XXXXXX parent and you can contact her at any time at 021-9384650, 021- 6898221 or 083 2708027.

Yours sincerely

XXXXXXXX
Vice-Principal

CONSENT – FREE INFORMED BY PARENT/GUARDIAN

I understand the nature of the research project as specified above. I understand that my son's participation in the research is strictly voluntary and that I have the right to withdraw him from the study at any stage and that he has the right to withdraw at any stage without penalty.

SIGNED: _____ **DATE:** _____

APPENDIX C

GENERAL INFORMATION FOR PUPILS

I am a senior clinical psychologist/lecturer in the Department of Child and Family Psychiatry at Tygerberg Hospital/University of Stellenbosch and University of the Western Cape. I am conducting research on sport concussion in teenagers as a requirement for a PhD degree at the University of the Western Cape.

The aim of the research is to assess the impact of repeated concussions on the academic and cognitive, functioning of adolescent boys. Comparisons will be made between boys who play rugby and those who do not as well as those who have had concussions and those who have not.

The research involves pre and post season (winter sport) screening initially and then post season screening for the remaining years. Screening involves assessment using internationally validated paper and pencil cognitive tests. You are requested to complete a brief questionnaire relevant to the research. Screening will take place during school hours.

The results of the research will be used by the collaborating university for scientific publication purposes only. Individual information collected on individual schoolboys will be **anonymous and strictly confidential.** No boy's name will be mentioned in the thesis or any publication. Confidentiality about individual participants will be preserved, through the use of coding throughout the research. Information will only be accessed following a head injury and made available to the boy, the parents and/or medical practitioner on request. This data may form part of the management decision in individual cases. However, the researcher will not be held accountable for management or medical decisions made by medical practitioners, sports coaches, sports administrators, parents or other relevant parties on the basis of that information.

Base line data obtained during screening is likely to be valuable in terms of gauging impairment in functioning following concussion. Post concussion assessment data can be compared with baseline information and the degree of impairment or recovery will serve to aid decisions in terms of your concussion management. It is therefore very important to be as accurate and honest as possible when responding.

Should you require further information please feel free to approach me prior to testing. You can contact me at any time at 021-9384650, 021- 6898221 or 083 2708027.

Yours sincerely

Debbie Alexander

Should you wish to participate in the research project please read and complete the PUPIL ASSENT section attached.

FREE INFORMED ASSENT

I understand the nature of the research project as specified above. I understand that my participation in the research is strictly voluntary and that I have the right to withdraw from the study at any stage without penalty.

SIGNED: _____ **DATE:** _____

APPENDIX D

SELF REPORT RESEARCH QUESTIONNAIRE

UNIVERSITY OF THE WESTERN CAPE

NAME:	AGE:
DATE OF BIRTH:	GRADE:
CURRENT WINTER SPORT:	
WHICH TEAM (e.g. U15b) ?	
WHICH POSITION ?	
HOW MANY YEARS HAVE YOU PLAYED RUGBY/HOCKEY ?	
1. WERE YOU CONCUSSED THIS SEASON (2005) ?	YES / NO

IF YOU WERE THEN PLEASE COMPLETE THE TABLE BELOW. PLEASE STATE THE DURATION OF THE SYMPTOMS IN SECONDS, MINUTES, HOURS AND/OR DAYS? ALL OF THIS INFORMATION IS REQUIRED FOR EVERY TIME THAT YOU WERE CONCUSSED.

YEAR	Transient confusion, no loss of consciousness, symptoms resolve in < 15 min	Transient confusion, no loss of consciousness, symptoms last > 15 min	Any loss of consciousness, either brief or prolonged	Any loss of memory	Dazed or dizzy	Disorientated or saw stars	Nausea	Headache	Admission to hospital

2. IF YOU ARE NOT PLAYING RUGBY THIS YEAR, DID YOU PLAY PREVIOUSLY?	YES / NO
If yes, which team ?	
And which position ?	
How many years did you play rugby ?	
3. WERE YOU EVER CONCUSSED IN A RUGBY MATCH PRIOR TO THIS (2005) SEASON?	YES / NO

IF YOU WERE THEN PLEASE COMPLETE THE TABLE BELOW. PLEASE STATE THE DURATION OF THE SYMPTOMS IN SECONDS, MINUTES, HOURS AND/OR DAYS? ALL OF THIS INFORMATION IS REQUIRED FOR EVERY TIME THAT YOU WERE CONCUSSED.

YEAR	Transient confusion, no loss of consciousness, symptoms resolve in < 15 min	Transient confusion, no loss of consciousness, symptoms last > 15 min	Any loss of consciousness, either brief or prolonged	Any loss of memory	Dazed or dizzy	Disorientated or saw stars	Nausea	Headache	Admission to hospital

4. HAVE YOU EVER SUSTAINED A HEAD INJURY OR CONCUSSION THAT WAS NOT RELATED TO RUGBY/SPORT?	YES / NO

IF YES THEN PLEASE COMPLETE THE TABLE BELOW. PLEASE STATE THE DURATION OF THE SYMPTOMS IN SECONDS, MINUTES, HOURS AND/OR DAYS? ALL OF THIS INFORMATION IS REQUIRED FOR EVERY TIME THAT YOU WERE CONCUSSED.

YEAR and cause of injury	Transient confusion, no loss of consciousness, symptoms resolve in < 15 min	Transient confusion, no loss of consciousness, symptoms last > 15 min	Any loss of consciousness, either brief or prolonged	Any loss of memory	Dazed or dizzy	Disorientated or saw stars	Nausea	Headache	Admission to hospital

5. HAVE YOU EVER FAILED A GRADE AT SCHOOL?	YES / NO
If yes, please specify	
6. HAVE YOU EVER EXPERIENCED LEARNING DIFFICULTIES OR REQUIRED REMEDIAL CLASSES?	YES / NO
If yes, please specify	
7. HAVE YOU EVER HAD SEIZURES, FEBRILE CONVULSIONS OR EPILEPSY?	YES / NO
If yes, please specify	
8. HAVE YOU EVER HAD ENCEPHALITIS/MENINGITIS (i.e. infection of the brain)?	YES / NO
If yes, please specify	
9. HAVE YOU EVER RECEIVED TREATMENT FOR DEPRESSION, ANXIETY, ATTENTION DEFICIT OR HYPERACTIVITY?	YES / NO
If yes, please specify	
10. ARE YOU CURRENTLY TAKING ANY FORM OF MEDICATION?	YES / NO
If yes, please specify	

APPENDIX E

ASSESSMENT BATTERY

Code: _____

Name: _____

Age: _____ **Date of Birth: _____**

Grade: _____

Date:

APPENDIX F

WISC III - VOCABULARY

UNTIMED ***Note:*** *This test is not timed so make sure that participants are given sufficient time to complete their answers. All participants must be finished before continuing.*

Instructions:

"I am going to say some words. Listen carefully and then write down the meaning of each word. I do not expect you to know the meaning of all the words but just try and do as many as you can."

1. What is a **COW**?
2. What is a **BICYCLE**?
3. What is a **DONKEY**?
4. What is the **ALPHABET**?
5. What does **ANCIENT** mean?
6. What does **LEAVE** mean?
7. What does **BRAVE** mean?
8. What is an **ISLAND**?
9. What does **ABSORB** mean?
10. What does **NONSENSE** mean?
11. What does **PRECISE** mean?
12. What does **TRANSPLANT** mean?
13. What does **BOAST** mean?
14. What does **MIGRATE** mean?
15. What is a **FABLE**?
16. What does **STRENUOUS** mean?
17. What does **MIMIC** mean?
18. What does **RIVALRY** mean?
19. What does **SECLUDE** mean?
20. What does **UNANIMOUS** mean?
21. What is an **AMENDMENT**?
22. What does **COMPEL** mean?
23. What does **IMMINENT** mean?
24. What does **AFFLICTION** mean?
25. What does **DILATORY** mean?
26. What does **ABBERATION** mean?

VOCABULARY		
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APPENDIX G

WMS III – VERBAL PAIRED ASSOCIATES

UNTIMED **Note:** This test is not timed so make sure that participants are given sufficient time to complete their answers. All participants must be finished before continuing.

Instructions:

"I am going to say a word and then say another word that goes with it. I will say a whole list of words like that. Listen carefully because when I am finished I will say the first word, and I want you to write down the word that goes with it. For example, if the pairs were 'Fruit-West', 'Gold-Walk', and then I say the word 'Fruit', you would write down (pause) 'West'. When I say the word 'Gold', you would write down (pause) 'Walk'."

If someone does not understand the directions, you may repeat them, paraphrasing where necessary.

Read the word pairs at the rate of one pair of words every 3 seconds; that is, the words are spoken about 1 second apart, with 2 seconds separating the pairs.

When you are sure all participants understand the directions, say:

"Now listen carefully to the list of word pairs as I read them."

List A:

Truck-Arrow
Insect-Acorn
Reptile-Clown
Bank-Cartoon
Star-Ladder
Baboon-Paper
Rose-Bag
Elephant-Glass

"Now turn the page in your answer books to the page that looks like this.."
(Place Record sheet for **List A** on the overhead).

After reading List A, pause for 5 seconds and present Recall A. Say:

"Write down your answer when I say the first word of each pair"

Recall A:

1. "Which word goes with **Bank**
2. "Which word goes with **Reptile**
3. "Which word goes with **Star**
4. "Which word goes with **Rose**
5. "Which word goes with **Elephant**
6. "Which word goes with **Truck**
7. "Which word goes with **Insect**

8. "Which word goes with **Baboon**

Read the first word of each pair. Allow a maximum of 10 seconds for the examinee's response.

After completing List A, say:

"Now turn over the page in your answer books."

"Now I will read the same list again, except with the word pairs in a different order. Listen carefully."

Read List B from the form.

List B:

Star-Ladder
Elephant-Glass
Insect-Acorn
Truck-Arrow
Reptile-Clown
Bank-Cartoon
Baboon-Paper
Rose-Bag

After reading List B, present Recall B using the same procedures as with Recall A. You may repeat the question. 'Which word goes with _____? If necessary.

"Now turn the page in your answer books to the page that looks like this…"
(Place Record sheet for **List B** on the overhead).

"Write down your answer when I say the first word of each pair"

Recall B:

1. **Elephant**
2. **Insect**
3. **Reptile**
4. **Rose**
5. **Star**
6. **Baboon**
7. **Bank**
8. **Truck**

"Turn over the page in your answer books."

"I will read the same list again, except with the word pairs in a different order. Listen carefully."

List C:

Rose-Bag
Baboon-Paper
Star-Ladder
Reptile-Clown
Elephant-Glass
Insect-Acorn
Bank-Cartoon

Truck-Arrow
"Now turn the page in your answer books to the page that looks like this…."
(Place Record sheet for **List C** on the overhead).

"Write down your answer when I say the first word of each pair"

Recall C:

1. **Insect**
2. **Star**
3. **Truck**
4. **Rose**
5. **Elephant**
6. **Reptile**
7. **Bank**
8. **Baboon**

"Turn over the page in your answer books."
"I will read the same list again, except with the word pairs in a different order. Listen carefully."

List D:

Baboon-Paper
Truck-Arrow
Star-Ladder
Insect-Acorn
Rose-Bag
Reptile-Clown
Bank-Cartoon
Elephant-Glass

"Now turn the page in your answer books to the page that looks like this…."
(Place Record sheet for **List D** on the overhead).

"Write down you answer when I say the first word of each pair"

Recall D:

1. **Star**
2. **Rose**
3. **Insect**
4. **Baboon**
5. **Elephant**
6. **Bank**
7. **Reptile**
8. **Truck**

Paired Associate Learning

	List A	
1		
2		
3		
4		
5		
6		
7		
8		

Paired Associate Learning

	List B	
1		
2		
3		
4		
5		
6		
7		
8		

Paired Associate Learning

	List C	
1		
2		
3		
4		
5		
6		
7		
8		

Paired Associate Learning

	List D	
1		
2		
3		
4		
5		
6		
7		
8		

APPENDIX H

WISC III – CODING
(Digit Symbol Substitution)

TIMED **120 seconds** (2 minutes)

Instructions:

Showing the Record Form on the overhead say the following:

"Now turn over the page in your answer book to this page and follow what I am going to show you on the overhead."

"Look at these divided boxes. You see, each box has a number in the top part (*sweep your finger along the numbers)* and a special mark in the bottom part (*sweep your finger along the marks).* Each number has its own mark (*point to the number 1 and its mark, then to number 2 and its mark).*

Pointing to the Sample Items, say:

"Now look down here where the boxes have numbers in the top part but the squares on the bottom are empty. You are to put in the empty squares *(point to the first several Sample spaces)* the marks that should go there like this…."

Point to the first Sample Item and say:

"Here is a 2"

Point to the symbol below the 2 in the Key and say:

"The 2 has this mark so I put it in this square, like this." (Draw the symbol)

Point to the second Sample Item and say:

"Here is a 1"

Point to the symbol below the 1 in the Key and say:

"The 1 has this mark so I put it in this square, like this." (Draw the symbol)

Point to the third Sample Item and say:

"This is the number 4"

Point to the symbol below the 4 in the Key and say:

"The 4 has this mark so I put it in this square, like this." (Draw the symbol)

After demonstrating the first three Sample Items, say:

"Now you fill in the rest of these boxes up to this dark line." (Point to the line)

When the Sample exercise has been completed, say:

"When I tell you to start, you do the rest of them."

Point to the first test Item and say:

"Begin here and fill in as many squares as you can, one after the other, without skipping any. Keep going until I tell you to stop. Work as **quickly** as you can without making any mistakes. When you finish this line *(sweep across the first row with your finger)* go on to this one *(point to the first item in row 2).*
Ready? Begin.

Begin timing and at the end of **120 seconds** say:
"STOP"
Once they have stopped, say:

"Now draw a circle around the last item you have completed. If you have not completed to the end of the fourth row (*point it out on the overhead)*, do so now."

Give them time to complete the fourth row

"Good. Now turn over to the next page in your answer books."

APPENDIX I

WISC – CODING: INCIDENTAL RECALL – IMMEDIATE

UNTIMED **Note:** This test is not timed so make sure that participants are given sufficient time to complete their answers. All participants must be finished before continuing.

Instructions:

Place the Record Sheet for the Incidental recall on the overhead and say:

"Now turn over the page to the Record sheet that looks like this."

INCIDENTAL RECALL

1	2	3	4	5	6	7	8	9

"See how many of the symbols used in the previous test you are able to remember. There is not time limit and you can do them in any order you wish."

APPENDIX J

DE TROIT MOTOR SPEED AND PRECISION TEST

TIMED **180 seconds** (3 minutes)

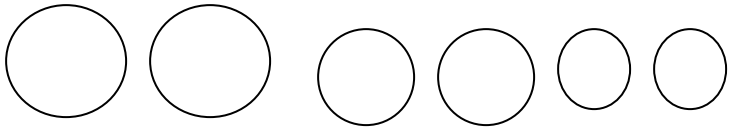
Instructions:

"Turn the page in your answer book to the Record sheet that looks like this.."
(Place the Record sheet on the overhead projector.)

"At the top of the page I want you to indicate whether you are left of right handed. Just make a tick in the correct box."

"There is a page with lots of circles on it. I want you to make a mark like this 'X' in each circle, be careful to keep your mark well within the circle."

Illustrate on circles in the sample, as per the example below:



It is important that the subject does not get the impression that they have to go from edge to edge.

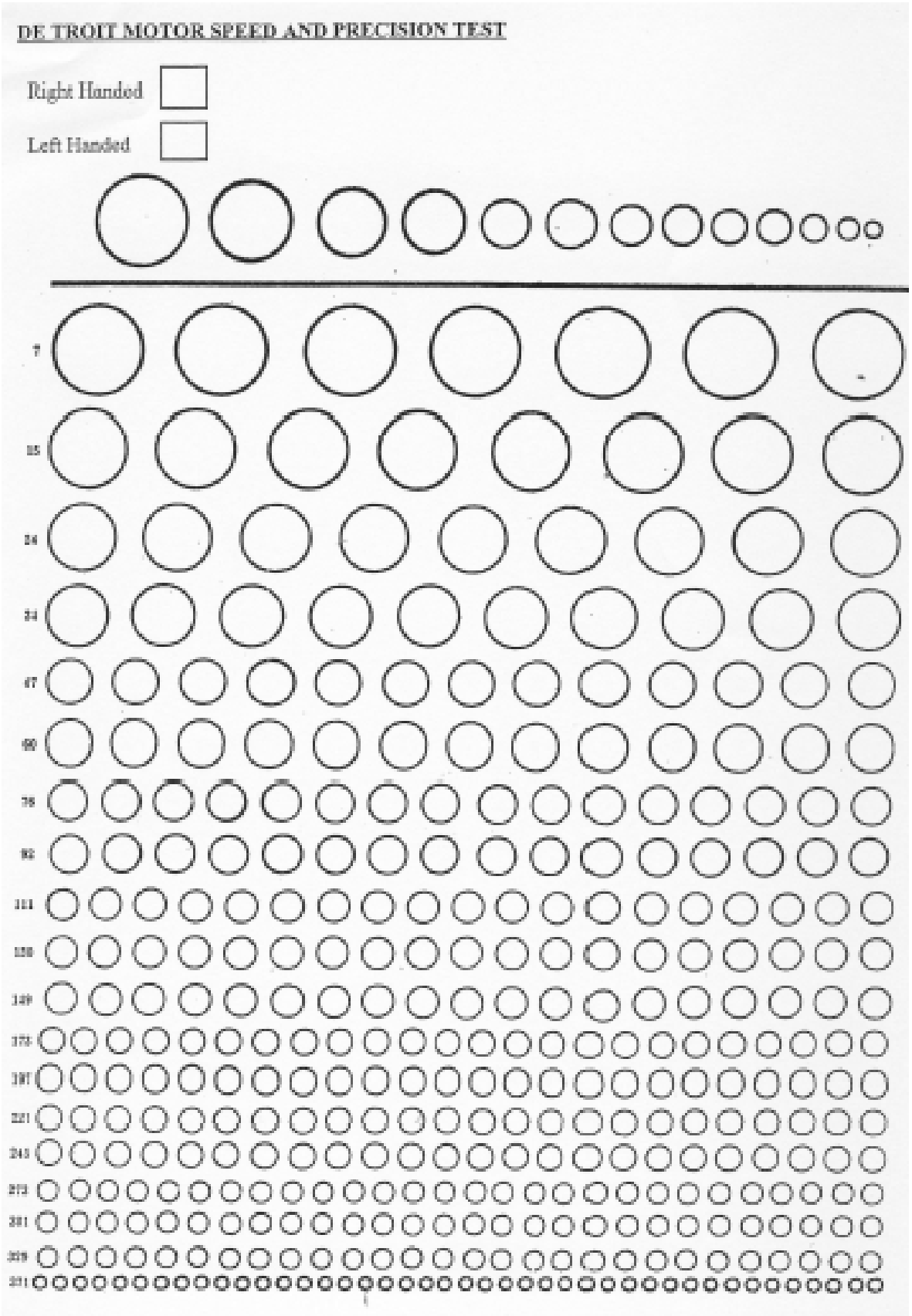
"You are to do as many as you can right after the other. Work as quickly as you can. It does not need to be tidy as long as you stay inside the circle. When you finish this row *(point to the first row)* carry on with the next row *(point to the next row).* Do you understand?

"When I tell you to start you do as many rows as you can and keep going until I say 'STOP'. Ready – Start."

After **180 seconds** (3 minutes) say:
"STOP"

"Turn over the page in your record books."

APPENDIX J continued



APPENDIX K

WMS III – VERBAL PAIRED ASSOCIATES: DELAYED

UNTIMED **Note:** this test is not timed so make sure that participants are given sufficient time to complete their answers. All participants must be finished before continuing.

Instructions:

"Now turn the page in your answer books to the page that looks like this.."
(*Place Record sheet for **Delayed Recall** on the overhead).*

"Remember the word pairs that you learned earlier? I told you a word and gave you another word that went with it. I want you to recall as many of those word pairs as you can remember, one more time. I will say the first word of the pair and you write down the word that goes with it. Ready?

Allow approximately 10 seconds for the examinee to respond.

Delayed Recall:

1. "Which word goes with **Truck**
2. "Which word goes with **Insect**
3. "Which word goes with **Reptile**
4. "Which word goes with **Bank**
5. "Which word goes with **Star**
6. "Which word goes with **Baboon**
7. "Which word goes with **Rose**
8. "Which word goes with **Elephant**

Paired Associate Learning

		Delayed Recall
1		
2		
3		
4		
5		
6		
7		
8		

APPENDIX L

WISC III – SIMILARITIES

UNTIMED **Note:** This test is not timed so make sure that participants are given sufficient time to complete their answers. All participants must be finished before continuing.

Instructions:

> "I am now going to say two words and ask you how they are alike. For example, if I ask. 'How are red and blue alike?' you would write down, 'They are both colours'.

Check to see whether everyone understands what you are asking and then proceed. If someone does not understand provide another example. e.g. in what way are a pool and sea the same? You swim in both.

"Turn over the page in your record books to the one that looks like this…"
(Place the Similarities Record Sheet on the overhead)

Proceed to Item 1 and say:
"Number 1. In what way are a piano and a guitar alike? How are they the same? Write the answer on the sheet provided."

Then proceed to Item 2 and say:
"Number 2. Now tell me, in what way are a candle and a lamp alike?"

Then proceed with the other Items.

3. *In what way are a **SHIRT** and a **SHOE** alike?*
4. *In what way are a **WHEEL** and a **BALL** alike?*
5. *In what way are **MILK** and **WATER** alike?*
6. *In what way are an **APPLE** and a **BANANA** alike?*
7. *In what way are a **CAT** and a **MOUSE** alike?*
8. *In what way are an **ELBOW** and a **KNEE** alike?*
9. *In what way are **ANGER** and **JOY** alike?*
10. *In what way are a **TELEPHONE** and a **RADIO** alike?*
11. *In what way are a **PAINTING** and a **STATUE** alike?*
12. *In what way are a **FAMILY** and a **TRIBE** alike?*
13. *In what way are **ICE** and **STEAM** alike?*
14. *In what way are **TEMPERATURE** and **LENGTH** alike?*
15. *In what way are a **MOUNTAIN** and a **LAKE** alike?*
16. *In what way are **RUBBER** and **PAPER** alike?*
17. *In what way are **FIRST** and **LAST** alike?*
18. *In what way are the numbers **NINE (9)** and **TWENTY-FIVE (25)** alike?*
19. *In what way are **SALT** and **WATER** alike?*

SIMILARITIES

1	A PIANO and a GUITAR:	
2	A CANDLE and a LAMP:	
3	A SHIRT and a SHOE:	
4	A WHEEL and a BALL:	
5	MILK and WATER	
6	An APPLE and a BANANA	
7	A CAT and a MOUSE	
8	An ELBOW and a KNEE	
9	ANGER and JOY	
10	A TELEPHONE and a RADIO	
11	A PAINTING and a STATUE	
12	A FAMILY and a TRIBE	
13	ICE and STEAM	
14	TEMPERATURE and LENGTH	
15	A MOUNTAIN and a LAKE	
16	RUBBER and PAPER	
17	FIRST and LAST	
18	NINE (9) and TWENTY-FIVE (25)	
19	SALT and WATER	

APPENDIX M

WISC III – CODING: INCIDENTAL RECALL – DELAYED

UNTIMED **Note:** This test is not timed so make sure that participants are given sufficient time to complete their answers. All participants must be finished before continuing.

Instructions:

Place the Record Sheet for the Incidental Recall on the overhead and say:

"Now turn over the page to the Record Sheet that looks like this…"

INCIDENTAL RECALL

1	2	3	4	5	6	7	8	9

"I would like to see how many of the symbols used in the earlier test you are still able to remember. There is no time limit and you can do them in any order you wish."

APPENDIX N

PERCENTAGES OF COGNITIVE AND ACADEMIC DEFICITS

Speed of Information Processing

CODING	-1 std dev	std dev	+1 std dev
baseline non rugby	16.7%	77.8%	5.6%
3rd year non rugby	21.4%	57.1%	21.4%
baseline current rugby	17.4%	80.4%	2.2%
3rd year current rugby	7.7%	92.3%	0.0%
baseline previous rugby	19.4%	74.2%	6.5%
3rd year previous rugby	8.7%	82.6%	8.7%

Motor Functioning

DT	-1 std dev	std dev	+1 std dev
baseline non rugby	11.1%	72.2%	16.7%
3rd year non rugby	21.4%	85.7%	0.0%
baseline current rugby	6.5%	80.4%	13.0%
3rd year current rugby	7.7%	84.6%	7.7%
baseline previous rugby	3.2%	67.7%	29.0%
3rd year previous rugby	4.3%	87.0%	8.7%

Verbal Memory (Incidental Recall)

VPA 1	-1 std dev	std dev	+1 std dev
baseline non rugby	5.6%	83.3%	11.1%
3rd year non rugby	7.1%	85.7%	7.1%
baseline current rugby	26.1%	63.0%	10.9%
3rd year current rugby	20.5%	51.3%	28.2%
baseline previous rugby	22.6%	61.3%	16.1%
3rd year previous rugby	4.3%	65.2%	30.4%

Verbal Memory (Delayed Recall)

VPA 2	-1 std dev	std dev	+1 std dev
baseline non rugby	27.8%	72.2%	0.0%
3rd year non rugby	21.4%	78.6%	0.0%
baseline current rugby	37.0%	63.0%	0.0%
3rd year current rugby	17.9%	82.1%	0.0%
baseline previous rugby	32.3%	67.7%	0.0%
3rd year previous rugby	8.7%	91.3%	0.0%

Academic Scores

ENGLISH	-1 std dev	std dev	+1 std dev
baseline non rugby	11.1%	77.8%	11.1%
3rd year non rugby	16.7%	0.7%	16.7%
baseline current rugby	21.7%	65.2%	13.0%
3rd year current rugby	19.6%	73.9%	6.5%
baseline previous rugby	12.9%	71.0%	16.1%
3rd year previous rugby	12.9%	87.1%	0.0%

MATHS	-1 std dev	std dev	+1 std dev
baseline non rugby	16.7%	61.1%	22.2%
3rd year non rugby	22.2%	66.7%	11.1%
baseline current rugby	19.6%	50.0%	30.4%
3rd year current rugby	13.0%	78.3%	8.7%
baseline previous rugby	29.0%	41.9%	29.0%
3rd year previous rugby	9.7%	80.6%	9.7%

APPENDIX O

RUGBY 3 COMBINED EFFECTS

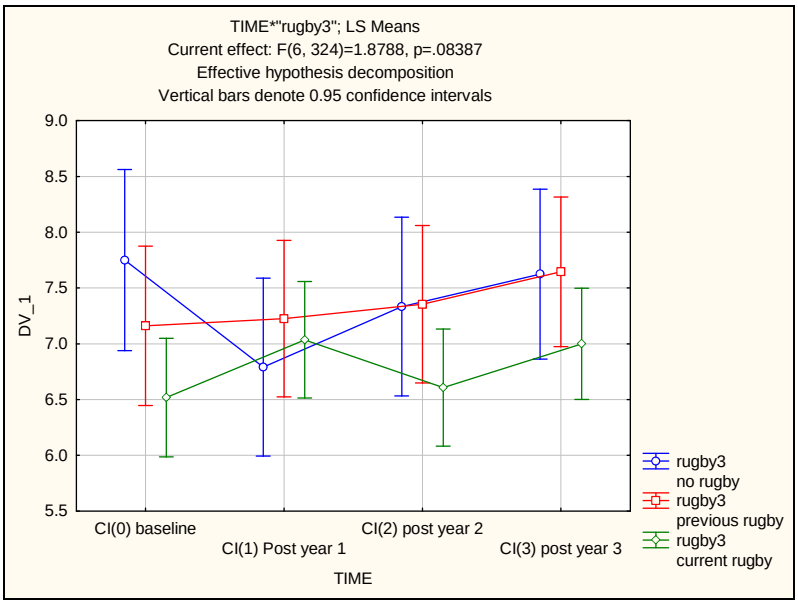


Figure: CI combined effects

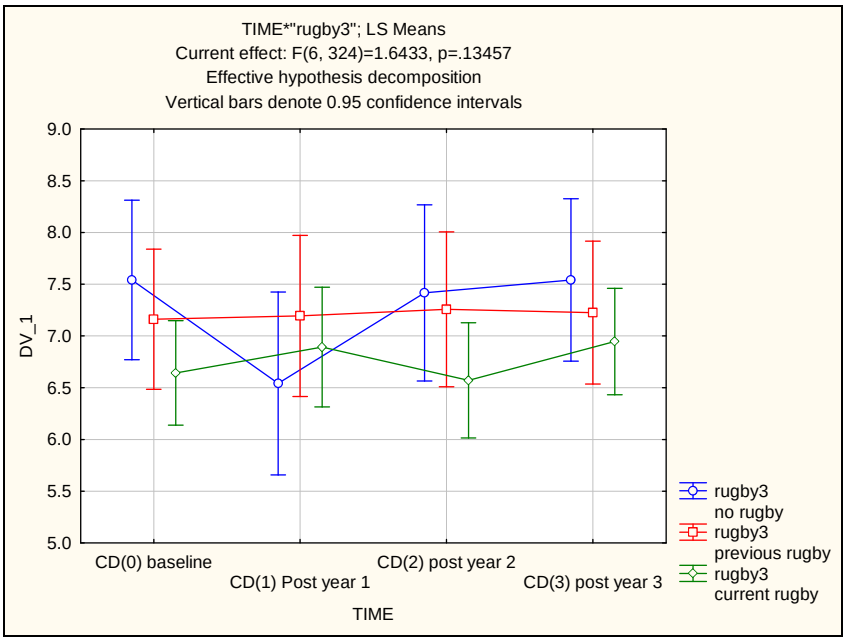


Figure: CD combined effects

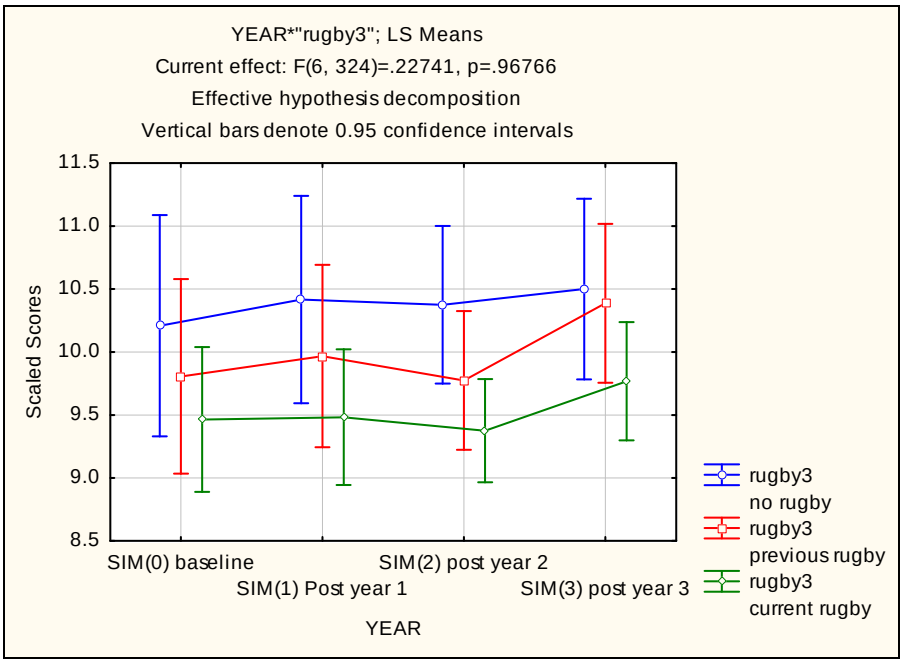


Figure WISC III Similarities Subtest combined effect (? REMOVE)

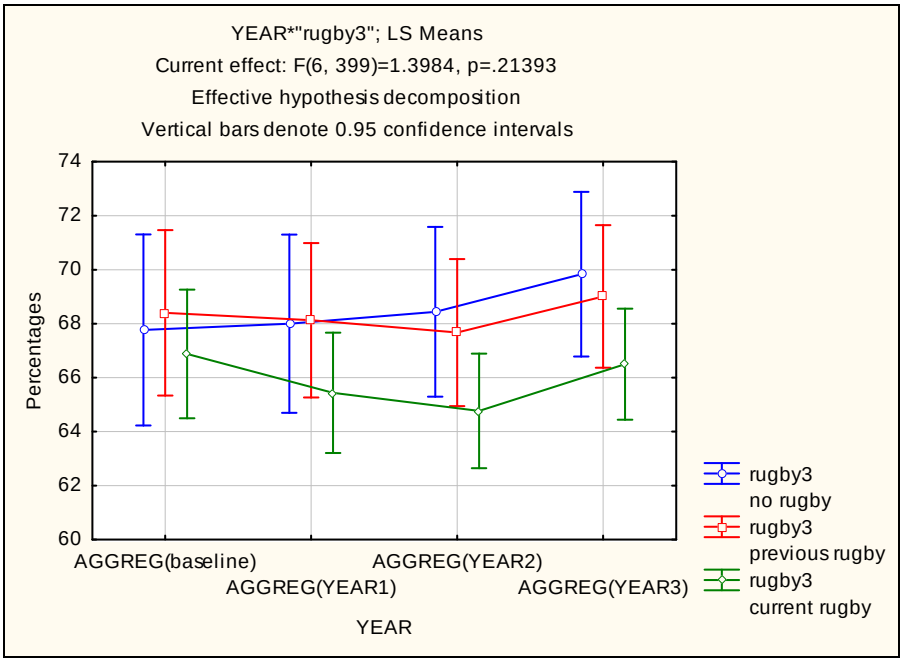


Figure Academic Aggregate combined effect

APPENDIX P

CRITICAL VALUES OF THE *F* DISTRIBUTION

Table 16.5 Abbreviated Version of Table D.3, Critical Values of the *F* Distribution Where $\alpha = .05$

Degrees of Freedom for Denominator	Degrees of Freedom for Numerator										
	1	2	3	4	5	6	7	8	9	10	15
1	161.4	199.5	215.8	224.8	230.0	233.8	236.5	238.6	240.1	242.1	...
2	18.51	19.00	19.16	19.25	19.30	19.33	19.35	19.37	19.38	19.40	...
3	10.13	9.55	9.28	9.12	9.01	8.94	8.89	8.85	8.81	8.79	...
4	7.71	6.94	6.59	6.39	6.26	6.16	6.09	6.04	6.00	5.96	...
5	6.61	5.79	5.41	5.19	5.05	4.95	4.88	4.82	4.77	4.74	...
6	5.99	5.14	4.76	4.53	4.39	4.28	4.21	4.15	4.10	4.06	...
7	5.59	4.74	4.35	4.12	3.97	3.87	3.79	3.73	3.68	3.64	...
8	5.32	4.46	4.07	3.84	3.69	3.58	3.50	3.44	3.39	3.35	...
9	5.12	4.26	3.86	3.63	3.48	3.37	3.29	3.23	3.18	3.14	...
10	4.96	4.10	3.71	3.48	3.33	3.22	3.14	3.07	3.02	2.98	...
11	4.84	3.98	3.59	3.36	3.20	3.09	3.01	2.95	2.90	2.85	...
12	4.75	3.89	3.49	3.26	3.11	3.00	2.91	2.85	2.80	2.75	...
13	4.67	3.81	3.41	3.18	3.03	2.92	2.83	2.77	2.71	2.67	...
14	4.60	3.74	3.34	3.11	2.96	2.85	2.76	2.70	2.65	2.60	...
15	4.54	3.68	3.29	3.06	2.90	2.79	2.71	2.64	2.59	2.54	...
16	4.49	3.63	3.24	3.01	2.85	2.74	2.66	2.59	2.54	2.49	...
17	4.45	3.59	3.20	2.96	2.81	2.70	2.61	2.55	2.49	2.45	...
18	4.41	3.55	3.16	2.93	2.77	2.66	2.58	2.51	2.46	2.41	...
19	4.38	3.52	3.13	2.90	2.74	2.63	2.54	2.48	2.42	2.38	...
20	4.35	3.49	3.10	2.87	2.71	2.60	2.51	2.45	2.39	2.35	...
22	4.30	3.44	3.05	2.82	2.66	2.55	2.46	2.40	2.34	2.30	...
24	4.26	3.40	3.01	2.78	2.62	2.51	2.42	2.36	2.30	2.25	...
26	4.23	3.37	2.98	2.74	2.59	2.47	2.39	2.32	2.27	2.22	...
28	4.20	3.34	2.95	2.71	2.56	2.45	2.36	2.29	2.24	2.19	...
30	4.17	3.32	2.92	2.69	2.53	2.42	2.33	2.27	2.21	2.16	...
40	4.08	3.23	2.84	**2.61**	2.45	2.34	2.25	2.18	2.12	2.08	...
50	4.03	3.18	2.79	**2.56**	2.40	2.29	2.20	2.13	2.07	2.03	...
60	4.00	3.15	2.76	2.53	2.37	2.25	2.17	2.10	2.04	1.99	...
120	3.92	3.07	2.68	2.45	2.29	2.18	2.09	2.02	1.96	1.91	...
200	3.89	3.04	2.65	2.42	2.26	2.14	2.06	1.98	1.93	1.88	...
500	3.86	3.01	2.62	2.39	2.23	2.12	2.03	1.96	1.90	1.85	...
1000	3.85	3.01	2.61	2.38	2.22	2.11	2.02	1.95	1.89	1.84	...

(Howell, 1995, p. 300)

APPENDIX Q

NORMS FOR % OF DEFICITS

	AGGREG (baseline)	AGGREG (YEAR3)	CD(0) baseline	CD(3) post year 3	CI(0) baseline	CI(3) post year 3	CODING (0) baseline	CODING (3) post year 3	DT(0) baseline	DT(3) post year 3
NO RUGBY NON CONCUSSED										
average	68.375	70.250	7.750	7.667	7.750	7.750	11.750	12.667	167.188	237.500
std dev	7.788	10.405	1.880	1.557	1.612	1.765	2.978	4.030	28.764	33.129
sample size	16	16	16	12	16	12	16	12	16	12
lower (-1 STD)	61	60	6	6	6	6	9	9	138	204
upper (+1 STD)	76	81	10	9	9	10	15	17	196	271

	ENGLISH (baseline)	ENGLISH (YEAR3)	MATHS (baseline)	MATHS (YEAR3)	SIM(0) baseline	SIM(3) post year 3	VPA1(0) baseline	VPA1(3) post year 3	VPA2(0) baseline	VPA2(3) post year 3
NO RUGBY NON CONCUSSED										
average	65.500	72.750	71.188	73.688	10.000	10.667	9.438	12.500	11.000	11.667
std dev	6.563	10.299	10.154	15.239	2.251	1.497	3.054	2.646	2.160	0.778
sample size	16	16	16	16	16	12	16	12	16	12
lower (-1 STD)	59	62	61	58	8	9	6	10	9	11
upper (+1 STD)	72	83	81	89	12	12	12	15	13	12

APPENDIX R

PUBMED search 19/06/07

#46	Search **mild concussion and chronic effect and contact sport and prospective study**	10:30:40	1
#45	Search **mild concussion and residual deficits and contact sport**	10:28:50	0
#37	Search **mild concussion and chronic effects and contact sport**	10:28:01	1
#38	Search **mild traumatic brain injury and chronic effects and contact sport**	10:27:25	1
#44	Search **mild traumatic brain injury and residual deficits and contact sport**	10:26:59	0
#42	Search **mild traumatic brain injury and residual deficits and prospective study**	10:14:10	2
#41	Search **mild traumatic brain injury and chronic effects and prospective study**	10:11:14	2
#40	Search **mild concussion and chronic effects and prospective study**	10:10:28	2
#39	Search **mild concussion and chronic effects and prospective study**	10:10:13	0
#36	Search **mild concussion and chronic effects**	10:05:57	49
#35	Search **mild concussion and residual deficits**	10:05:17	8
#34	Search **mild traumatic brain injury and residual deficits**	10:04:18	12
#33	Search **mild traumatic brain injury and residual deficits**	10:03:41	13
#32	Search **mild traumatic brain injury and chronic effects**	10:02:54	67
#31	Search **mild traumatic brain injury and adolescents**	10:01:50	620
#30	Search **mild concussion and adolescents**	10:00:48	977
#29	Search **mild concussion and adolescents and contact sport**	09:59:37	11
#28	Search **mild concussion and adolescents and contact sport and chronic effects**	09:59:19	0
#27	Search **mild concussion and children and contact sport and chronic effects**	09:57:59	0
#26	Search **mild concussion and children and contact sport and residual effects**	09:57:30	0
#25	Search **mild concussion and children and contact sport**	09:56:11	3
#24	Search **mild concussion and children and contact sport and longitudinal study**	09:54:44	2
#23	Search **mild concussion and children and contact sport and prospective study**	09:50:35	1
#22	Search **mild traumatic brain injury and children and contact sport and prospective study**	09:48:05	0
#21	Search **mild traumatic brain injury and children and contact sport and longitudinal study**	09:47:40	0
#20	Search **mild traumatic brain injury and children and contact sport and longitudinal study** Limits: **only items with links to full text, only items with links to free full text, only items with abstracts**	09:47:14	0
#14	**Related Articles** for **PubMed** (Select **12544160**)	08:12:59	721

#13	Search **mild traumatic brain injury and contact sport and prospective studies**	08:12:20	1
#12	Search **mild traumatic brain injury and contact sport and longitudinal studies**	08:10:33	2
#11	Search **mild traumatic brain injury and adolescents and contact sport and chronic effects and longitudinal studies**	08:09:20	0
#10	Search **mild traumatic brain injury and adolescents and contact sport and residual deficits and longitudinal studies**	08:08:46	0
#9	Search **mild traumatic brain injury and children and contact sport and residual deficits and longitudinal studies**	08:07:13	0
#8	Search **mild traumatic brain injury and children and contact sport and residual deficits and prospective studies**	08:06:46	0
#6	Search **mild traumatic brain injury and children and contact sport**	08:03:52	1