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Degree of Master of Arts (Neuropsychology)

Investigating history of concussion and data from head impact telemetry (xPatch) in relation  
to neuropsychological outcomes in a sample of adult rugby players in

Cape Town

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## Table of Contents

|   |    |
|---|----|
| <b>List of Figures</b> .....  | 9  |
| <b>List of Tables</b> .....   | 10 |
| <b>Abstract</b> .....   | 11 |
| <b>Introduction</b> .....   | 13 |
| Classification and Diagnosis of Concussion.....   | 15 |
| Defining concussion.....  | 15 |
| Subconcussive head injuries.....  | 18 |
| Epidemiology of Concussion.....   | 19 |
| Concussion in rugby.....  | 20 |
| Pathophysiology of Concussion.....  | 22 |
| Neuropathology of Concussion.....   | 23 |
| Clinical Symptoms in the Acute Post-Concussion Phase.....   | 24 |
| Assessment of clinical symptoms .....   | 27 |
| Cognitive impairment in the acute post-concussion phase .....   | 29 |
| <i>Association between cognitive outcomes and neuropathological consequences in the acute post-concussion phase</i> ..... | 30 |
| Investigating the course of cognitive recovery post- concussion with formal neuropsychological assessment.....            | 31 |
| Persisting Neuropathological Consequences Associated with Recovery Post-Concussion ....                                   | 32 |
| Persistent cognitive symptoms post-concussion .....   | 33 |
| Possible reasons for inconsistencies.....   | 35 |

|   |    |
|---|----|
| Neuropsychological Effects of Multiple Concussions.....   | 36 |
| The Neuropsychological Effects of Cumulative Subconcussive Head Injuries .....  | 39 |
| Chronic Traumatic Encephalopathy (CTE) .....  | 42 |
| Measuring Concussive and Subconcussive Head Injuries using Head Impact Telemetry (HIT)<br>.....   | 43 |
| <b>Summary and Rationale</b> .....  | 46 |
| <b>Study 1: Investigating the relationship between cognitive performance and concussion<br/>history in a sample of rugby players and non-contact sports players</b> ..... | 48 |
| Aims and Hypothesis .....   | 49 |
| Methods.....  | 50 |
| Design and setting .....  | 50 |
| Participants .....  | 50 |
| <i>Exclusion criteria</i> .....   | 52 |
| Materials.....  | 53 |
| <i>Screening measures</i> .....   | 53 |
| <i>Symptom and cognitive testing measures</i> .....   | 55 |
| Procedure.....  | 57 |
| Data analysis .....   | 58 |
| <i>Sample characteristics</i> .....   | 58 |
| <i>Effect of concussion history on cognitive performance</i> .....  | 58 |
| Ethical considerations .....  | 60 |
| <i>Informed consent</i> .....   | 60 |

|  |    |
|--|----|
| <i>Confidentiality</i> .....   | 61 |
| <i>Referrals</i> .....   | 61 |
| <i>Debriefing</i> .....  | 61 |
| <i>Risks and benefits</i> .....  | 61 |
| Results.....   | 62 |
| Between-groups analysis: Participant characteristics .....   | 62 |
| Between-groups analysis: Cognitive outcomes .....  | 65 |
| Correlational analyses: Cognitive outcomes and indicators of a history of concussion severity .....  | 66 |
| Discussion .....   | 67 |
| Summary of results.....  | 68 |
| Limitations and future directions .....  | 74 |
| Conclusion.....  | 75 |
| <b>Study 2: A novel method investigating the use of the xPatch (X2Biosystems Inc.), a means of head impact telemetry (HIT), to determine the number of subconcussive head injuries in a sample of university rugby players</b> ..... | 76 |
| Aims and Hypothesis .....  | 78 |
| Methods.....   | 78 |
| Design and setting .....   | 78 |
| Participants .....   | 78 |
| Materials.....   | 79 |
| <i>Head impact telemetry (HIT)</i> .....   | 79 |

|  |     |
|--|-----|
| Procedure.....   | 80  |
| <i>Recruitment</i> .....   | 80  |
| <i>Protocol for data collection</i> .....  | 80  |
| Data analysis .....  | 80  |
| Ethical considerations .....   | 82  |
| Results.....   | 83  |
| Linear and rotational acceleration forces .....  | 83  |
| Location of impacts to the cranium.....  | 87  |
| Discussion.....  | 89  |
| Summary of results.....  | 89  |
| <i>Mean number of head impacts</i> .....   | 89  |
| <i>Forward vs. Backline players</i> .....  | 93  |
| <i>Impact locations</i> .....  | 94  |
| Limitations and future directions .....  | 95  |
| Conclusion.....  | 96  |
| <b>Study 3: Investigating the relationship between the xPatch (X2Biosystems Inc.) data, and potential subconcussive head injuries, and cognitive outcomes, in a sample of university rugby players</b> ..... | 97  |
| Aims and Hypothesis .....  | 99  |
| Methods.....   | 99  |
| Design and setting .....   | 99  |
| Participants .....   | 100 |

|  |            |
|--|------------|
| Materials.....   | 100        |
| <i>Head impact telemetry (HIT)</i> .....   | 100        |
| <i>Screening measures</i> .....  | 100        |
| <i>Symptom and cognitive testing measures</i> .....                              | 100        |
| Procedure.....   | 100        |
| Data analysis. ....  | 101        |
| <i>Correlational analyses: Cognitive outcomes and head impact exposure</i> ..... | 101        |
| <i>Reliable Change Index (RCI)</i> .....   | 101        |
| Ethical considerations.....  | 102        |
| Results.....   | 102        |
| Correlational analyses: Cognitive outcomes and head impact exposure. ....        | 102        |
| <i>Total head impacts</i> .....  | 102        |
| <i>Total impact frequency burden (IFB)</i> .....                                 | 103        |
| Individual comparisons: RCI analyses.....  | 104        |
| Discussion.....  | 107        |
| Summary of results.....  | 107        |
| <i>Correlations</i> .....  | 108        |
| <i>RCI results</i> .....   | 109        |
| Limitations and future directions.....   | 111        |
| Conclusion.....  | 111        |
| <b>General Summary and Conclusion</b> .....                                      | <b>112</b> |

|  |     |
|--|-----|
| <b>References</b> .....  | 119 |
| <b>Appendix A: Concussion definition and symptoms</b> .....                            | 157 |
| <b>Appendix B: Demographic and medical history questionnaire (Control group)</b> ..... | 158 |
| <b>Appendix C: Demographic and medical history questionnaire (Rugby group)</b> .....   | 160 |
| <b>Appendix D: The ImPACT neuropsychological test battery</b> .....                    | 162 |
| <b>Appendix E: Informed consent document (Control group)</b> .....                     | 163 |
| <b>Appendix F: Informed consent document (Rugby group)</b> .....                       | 168 |
| <b>Appendix G: Debriefing form</b> .....   | 173 |
| <b>Appendix H: Rugby playing positions</b> .....                                       | 175 |

### List of Figures

- Figure 1:* A breakdown of the eligible participants recruited from each rugby club.....52
- Figure 2:* Example of the data display provided by the IMS, with data including, but not limited to, xPatch player assignment and charge levels, and number, location, and severity of impacts recorded per xPatch (X2Biosystems Inc., 2013).....82
- Figure 3.* Rugby union positions and set-up from a scrum.....175

### List of Tables

|  |     |
|--|-----|
| Table 1. <i>Points Calculation of the Duration of Loss of Consciousness Composite Score</i> .....  | 60  |
| Table 2. <i>Between-group Differences: Participant Characteristics per Group (Control vs. Rugby Not Concussed vs. Rugby Concussed)</i> .....                     | 63  |
| Table 3. <i>Participant Characteristics: Concussion History per Group (Control vs. Rugby Not Concussed vs. Rugby Concussed)</i> .....                            | 64  |
| Table 4. <i>Cognitive Outcomes Scores per Group (Control vs. Rugby Not Concussed vs. Rugby Concussed)</i> .....  | 66  |
| Table 5. <i>Correlations Between Cognitive Outcomes and Indicators of a History of Concussion Severity for Rugby Players</i> .....                               | 67  |
| Table 6. <i>Resultant Linear and Rotational Acceleration Forces, and the Total Impact Frequency Burden (IFB) for Forwards and Backline Players</i> .....         | 85  |
| Table 7. <i>Average Resultant Linear and Rotational Acceleration Forces, and the Total Impact Frequency Burden (IFB) for Forwards and Backline Players</i> ..... | 86  |
| Table 8. <i>Head Impacts (&gt; 10g) by Location of Impact</i> .....  | 87  |
| Table 9. <i>Average Head Impacts (&gt; 10g) by Location of Impact for Forwards and Backline Players</i> .....  | 88  |
| Table 10. <i>Head Impacts (&gt; 10g) and Resultant Linear and Rotational Acceleration Forces by Injury Tolerance Level and Impact Severity Limits</i> .....      | 88  |
| Table 11. <i>Correlations Between Head Impact Exposure and Cognitive Outcomes</i> .....  | 104 |
| Table 12. <i>RCI Analyses for Cognitive Outcome Changes from time 1 to time 2 with the Internal Rugby group</i> .....  | 106 |

### Abstract

While Rugby Union has worldwide popularity, with over 5 million registered and non-registered players participating every year, the game lends itself to a high incidence of concussion among players. Rugby players, more so than that recorded for any other contact sport, including American Football, are also more frequently exposed to head collisions not resulting in concussion (i.e., subconcussive head injuries). Despite some evidence for a potential association between such injuries and acute neurological and neuropsychological difficulties, which may at times persist among some players, overt symptoms still guide the initial on-field response for further concussion management to be initiated. The aim of this study was threefold: 1) to investigate the relationship between rugby players' history of concussion and neuropsychological outcomes, 2) to explore the use of a head impact telemetry (HIT) device in describing high-impact head collisions (and potentially subconcussive injuries), and 3) to explore the relationship between that HIT data and neuropsychological outcomes. Study 1 investigated differences between non-contact sport participants ( $n = 23$ ) and rugby players with (Rugby Concussed;  $n = 31$ ), and without a history of concussion (Rugby Not Concussed;  $n = 26$ ) in a baseline cognitive assessment. Results showed that at the beginning of the rugby season there were no differences in cognitive abilities at a group level; a more severe concussion history was largely not associated with a poorer performance on these cognitive outcomes. Study 2 was a pilot study utilising the xPatch to objectively capture a rugby player's exposure to head impacts in an amateur rugby team (UCT IRL team;  $n = 8$ ). Although the majority of impacts captured were of a 'mild' severity, there were many acceleration forces, particularly rotational accelerations, recorded above an injury threshold potentially implicated with concussion. Following from this, Study 3 used a prospective and repeated-measures design with the same UCT IRL team, to evaluate a means for investigating a player's neuropsychological vulnerability to high-

impact subconcussive head injuries. Using correlational analyses, the Reliable Change Index (RCI) and head collision data from Study 2, there was a lack of evidence to indicate that player's increased exposure to repeated high-impact head collisions results in a generally poorer neuropsychological performance. However, a number of test practice effects are noted. Combined, these findings suggest that (a) identifying possible enduring neuropsychological difficulties retrospectively is limited, and issues such as test-practice effects and test sensitivity should be considered in future, preferably prospective studies, (b) rugby players are vulnerable to sustaining multiple high-impact subconcussive head injuries and the data suggests utility in including HIT like the xPatch, and (c) that implementing a multi-faceted protocol for monitoring rugby players' that negates a reliance on concussion diagnosis is necessary to better understanding individual recovery trajectories.

*Keywords:* concussion, sports concussion, subconcussive, mild traumatic brain injury, mTBI, neuropsychological, cognitive, rugby.

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Cape Town

Mild traumatic brain injury (mTBI), also referred to as concussion (Collins et al., 1999; Levin & Diaz-Arrastia, 2015; Shuttleworth-Edwards & Radloff, 2008; Stewart, McNamara, Lawlor, Hutchinson, & Farrell, 2015), has over time received heightened focus, as a unique and complex aspect of TBI, and with regards to resultant neuropsychological and behavioural sequelae, which may or may not persist in different individuals. The concern over the impact of concussion has been widespread, demanding the attention of large, multi-centre research groups and international sport and health organisations, and has for a while become a priority research area for the World Health Organisation (WHO; Cassidy et al., 2004; von Holst & Cassidy, 2004). Due to collaborative efforts to collect large and heterogeneous data sets from a general and sporting population suffering from concussion, relevant research can be implemented on a continual basis with reference to such resources (Cancelliere, Donovan, & Cassidy, 2016; King, Hume, Brughelli, & Gissane, 2014; Levin & Diaz-Arrastia, 2015; Stewart et al., 2015; Talavage et al., 2014).

In addition to a large institutional focus, over the past ten years or so, there has been a surge of public opinion and concern regarding the potential deleterious short- and long-term effects of concussion, particularly among contact sports players. Public opinion, as indicated by various media reports (Belson, 2015; Croatto, 2014; Schwarz, 2010), has been put forward especially in response to a few case studies indicating the delayed presentation of neuropsychological and behavioural changes later on in life, in individuals presenting with a history of multiple concussions and potential subconcussive head injuries (impacts that do not result in an observable, diagnosed, or reported concussion (Bazarian, Zhu, Blyth, Borrino, &

Zhong, 2012; Breedlove et al., 2012; King et al., 2014; Talavage et al., 2014). The concern over concussion is especially relevant to contact sports players because they have been shown to be at an increased risk for high exposure to repeated head injuries, involving concussion and such subconcussive head injuries (Coronado, McGuire, Faul, Sugerman, & Pearson, 2012; King et al., 2014; Spiotta, Shin, Bartsch, & Benzel, 2011; Talavage et al., 2014).

There is a growing awareness with regard to the association between exposure to repeated concussions and the increased risk of long-term neuropsychological and neurological consequences (Baugh et al., 2012; Levin & Diaz-Arrastia, 2015; McKee et al., 2013). However, there exists much debate surrounding these potential short- and long-term sequelae of concussion and the implications of this for developing appropriate return-to-play guidelines (Carroll et al., 2014; McCrea, 2008; McCrory et al., 2013a). Diagnosing concussion and identifying appropriate guidelines to manage sport players who are at increased risk for these injuries has proved somewhat problematic. There is inconsistency with operationalising and diagnosing concussion, and with the knowledge about the short-term neuropsychological impact of concussion and the relationship of this to the possible development of long-term neuropsychological impairment.

With regards to diagnosing concussion in sport, the clinical care and management of athletes and research protocols have traditionally relied on the recognition of signs and symptoms associated with a concussive event (Holm, Cassidy, Carroll, & Borg, 2005; National Center for Injury Prevention and Control, 2003; Stein, 1996). Despite advances in research toward identifying objective markers of concussion (Henry, Tremblay, & De Beaumont, 2016a; King et al., 2014; Orr et al., 2016; Talavage et al., 2014), a reliance on symptom detection from the side line still remains, and currently comprehensive symptom and self-report checklists do exist that are regarded as being suitable for the immediate on-field identification of concussion (Bailes, Petraglia, Omalu, Nauman, & Talavage, 2013;

McCrory et al., 2013a; McCrory et al., 2013b). However, objective markers of injury would likely serve to lessen the challenges related to reliable concussion and potential subconcussive diagnoses.

In sum, in lieu of increased exposure of contact sport players to repeated head injury conditions, including concussion, and potential long-term outcomes, research targeted at the observation and the understanding and management of players in a contact sport environment, as well as objective measures of injury, is critical.

## Literature Review

### Classification and Diagnosis of Concussion

The classification of TBI severity is made on a continuum from mild to moderate to severe (Stein, 1996), and concussion is categorised as a subset of mTBI. Concussion has historically been known as an injury resulting from low velocity forces to the cranium consequently causing the brain to shake, resulting in clinical symptoms that do not necessarily relate to any pathological injury (McCrory et al., 2013a). The terms mTBI and concussion are often used synonymously in the literature (Abbas et al., 2015; Levin & Diaz-Arrastia, 2015; Stein et al., 2016). For the purpose of this thesis, I will use the term *concussion*.

**Defining concussion.** In a consensus statement on concussion in sport, McCrory et al. (2013a) defined concussion as a brain injury induced by biomechanical forces to the head resulting in a complex set of pathophysiological consequences affecting the brain. Furthermore, it was noted that with concussion there is an alteration in mental status that may or may not include a loss of consciousness, and which is characterised by a graded set of clinical symptoms (e.g., headache, feeling in a fog, loss of consciousness, amnesia, and irritability). The consensus statement also suggested that while concussion may result in

neuropathological changes, the acute<sup>1</sup> clinical symptoms largely reflect a functional disturbance rather than a structural injury; hence a reliance on clinical symptomology for a diagnosis of concussion. Thus, with these types of injuries one should not necessarily expect to find abnormalities on standard structural neuroimaging studies and diagnoses can be made in the absence of such structural evidence (see Appendix A for a more detailed description of this definition). Despite some variation in symptomology, this recent consensus statement is for the most part in keeping with previous and current bodies of research that have set out to characterise concussion within the domain of TBI (American Academy of Neurology (AAN), 1997; Aubry et al., 2002; Carroll et al., 2004; Holm et al., 2005; Kristman et al., 2014; McCrory et al., 2009; Menon, Schwab, Wright, & Maas, 2010; National Center for Injury Prevention and Control, 2003).

As would be expected given the work of different collaborations in characterising concussion and the consequent methodological variation in this research, classifying and diagnosing concussion presents a unique challenge within the TBI severity spectrum (Carroll et al. 2014). There are some classification systems, such as the Glasgow Coma Scale (GCS; Jennett & Teasdale, 1981) and The Mayo Classification System for Traumatic Brain Injury Severity (Malec et al., 2007), and single indicators, such as loss of consciousness and posttraumatic amnesia, that can be used to attempt to characterise head injuries on this TBI severity continuum. However, these grading scales identified as accurate diagnostic assessments following moderate and severe TBI, may be of limited utility when diagnosing concussions given their lack of sensitivity to key, yet subtle associated diagnostic signs and symptoms (McCrea, 2008; Petraglia, Maroon, & Bailes, 2012). For example, while the GCS is one of the most recognised and widely used measures for grading TBI severity (Davis et al., 2006), it relies on gross symptoms of severe neurologic dysfunction. On this point, it has

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<sup>1</sup> Although the acute phase of recovery has no specific cut-off, it is understood to be within three months post-concussion (Henry et al., 2011).

been recognised that sports-related concussion is associated with LOC in a minority of patients (10%) and that other symptoms, such as confusion, amnesia, dizziness, visual disturbances, and headache are more characteristic thereof (Bailes et al., 2013; Petraglia et al., 2012).

More recent research has turned to using a multifaceted approach to diagnosing concussion that aims to capture a broad spectrum of concussion-specific clinical signs and symptoms. In doing so, this approach focuses on more subtle indicators of concussion, thus alleviating some of the limitations that are inherent in these crude conventional classification methods (McCrory et al., 2009; McCrory et al., 2013a). Furthermore, classification systems, such as the Sports Concussion Assessment Tool V.3 (SCAT3; McCrory et al., 2013b), that have been developed specifically for the diagnosis and management of sports-related concussion have since been provided.

However, these criteria are still only guidelines for practitioners and researchers. Although they provide a general framework for the clinician in diagnosing concussion, there is no perfect diagnostic tool and there is often uncertainty, debate and criticism regarding when to ignore or when to act on more subtle signs and symptoms associated with concussion. The confusion often occurs given that some of these symptoms (e.g., headaches, confusion, dizziness), are not concussion-specific. The uncertainty surrounding what constitutes a concussion is problematic for clinicians, coaches, and players having to make immediate decisions regarding the severity of a head injury in sport, and regarding return-to-play management. Further drawbacks to the process of diagnosing concussion are that symptoms may only manifest several hours or days post-injury and are often underreported or not identified by sports players and on-field medical staff, particularly for competitive and fast-paced contact sports such as rugby (Bailes et al., 2013; McCrory et al., 2013a; McKee et al., 2009). Further, research with rugby players has indicated that players may also not report

their symptoms for fear of being excluded from further team selection (Shuttleworth-Edwards, Border, Reid, & Radloff, 2004), or likely due to wanting to stay on the field during games (Gregory, 2010).

**Subconcussive head injuries.** To add to this complexity, contact sports players may also be exposed to multiple head injuries during a game that are not as severe as a concussion; what researchers refer to as subconcussive head injuries (Bailes et al., 2013; King et al., 2014). Subconcussive head injuries are generally thought to involve similar but smaller impact forces than concussion, and are thought to operate below the minimum concussion threshold necessary to produce any overt symptoms that may draw medical attention. Importantly, *concussion* and *subconcussive head injuries* are two distinct terms (Bailes et al., 2013; Bazarian et al., 2012; Beckwith et al., 2013; Breedlove et al., 2012; Coronado et al., 2012; King et al., 2014; Martini, Eckner, Kutcher, & Broglio, 2013; Spiotta et al., 2011; Talavage et al., 2014).

Despite the apparent absence of concussion-like symptoms, research findings suggest that these head collisions associated with subconcussive head injuries, particularly when cumulative over time, may be associated with short- and long-term neuropsychological impairment, and thus are cause for precautionary intervention (Gavett, Stern, & McKee, 2011; McKee et al., 2009; Stern et al., 2011; Talavage et al., 2014).

The reported impact of these head injuries on neuropsychological performance is however mixed, owing largely to limited research and sample sizes in these studies (Bailes et al., 2013; McAllister et al., 2012; Talavage et al., 2014). Nevertheless, the repeated nature of these often undetected injuries and the resultant cumulative effect thereof over time, is thought to potentially have a significant long-term deleterious neuropsychological impact. This point is especially pertinent given the sub-clinical nature of such injuries, which may consequently not be appropriately identified or managed and therefore the incidence and

management of these repeated injuries may be underestimated (Bailes et al., 2013; Baugh et al., 2012; Gavett et al., 2011; McKee et al., 2009; Stern et al., 2011; Talavage et al., 2014).

Bearing in mind that many concussion injuries are underreported, along with the repeated nature of potential subconcussive injuries, the following section provides evidence for the reported epidemiology of concussion, globally, and in South Africa.

### **Epidemiology of Concussion**

TBIs ranging from mild to severe, are considered to be a public health concern, both in South Africa (Cywes, 1990; Levin, 2004; Roozenbeek, Maas, & Menon, 2013) and globally (Coronado et al., 2012; Feigin et al., 2013; Langlois & Sattin, 2005; Maas, Stocchetti, & Bullock, 2008; Roozenbeek et al., 2013; Tagliaferri, Compagnone, Korsic, Servadei, & Kraus, 2006). According to the Centers for Disease Control (CDC), there was an estimated total of 2.5 million mortalities, hospital admissions, and Emergency Department (ED) visits attributed to a TBI in 2010 (CDC, 2016). However, these figures are considered to be an underestimation given that only individuals who presented for clinical care and received a relevant diagnostic code were included in this count. Furthermore, it was reported that 75% of these reported TBI's were characterised as mild (Voss, Connolly, Schwab, & Scher, 2015).

While concussion is considered to be a prevalent neurotrauma within the general population, it is potentially twice as prevalent among athletic personnel (Cassidy et al., 2004; Coronado et al., 2012; Coronado et al., 2015). The results of large studies conducted in the United States have shown that the overall incidence of concussion in collegiate athletes is 0.25 per 1000 (Gessel, Fields, Collins, Dick, & Cornstock, 2007; Hootman, Dick, & Agel, 2007).

In addition, the frequency of sports-related concussion can also be calculated on a per athlete exposure (AE) basis; the incidence of concussion for each practice or competition

hour. College athletes enrolled in the National Collegiate Athletic Association (NCAA) Injury Surveillance Program (ISP) during the 2009/2010 to 2013/2014 academic years, were shown to have an average injury rate of 5.56 per 10000 AE's (practice and competition), with a competition rate of 14.59 AE's per 10000 across different sports, for males and females (Wasserman, Kerr, Zuckerman, & Covassin, 2015).

Regarding concussion prevalence among 452 college American football players specifically, from a period spanning January 2006 to January 2015, 26.1% of players were diagnosed with a concussion, with concussion reported as significantly more prevalent during games compared to practices (Houck et al., 2016). Wasserman et al. (2015) also reported that male American football players reported the most sport-related concussions (36.1% of all reported concussions). Clay, Glover, and Lowe (2013) summarised concussion incidence data in sport in the USA, and advised that caution should be exercised when interpreting such data given that studies used different sports codes, different methods of reporting, and different times of data collection.

**Concussion in rugby.** Rugby Union, hereon referred to as rugby, is the most widely played of the rugby football sports (including rugby, Rugby League, American football, and Australian Rules football). According to World Rugby, the world governing and law-making body for rugby, in 2015 there were 4.91 million registered and non-registered male and female adults, adolescents participating annually in rugby, across more than 117 countries. It was also reported that more than a million children were introduced to the game of rugby in 2015 (World Rugby, 2016). However, whilst the popularity of rugby-related sports is growing on a global scale, an increasing number of players involved in these full contact and collision team sports are now consequently more vulnerable to repetitive instances of concussion (Clay et al., 2013; Gardner, Shores, & Batchelor, 2010; Shuttleworth-Edwards et al., 2008). This is particularly true for rugby; researchers report a higher rate of concussion

than for Rugby League (a faster, more simplified form of rugby with 13, as opposed to 15 players per side), American football and soccer. Furthermore, research has also shown that subconcussive head injuries occur as frequently in rugby as in American Football (Datalys Center for Sports Injury Research and Prevention, 2013; Gardner, Iverson, Huw Williams, Baker, & Stanwell, 2014; King et al., 2014).

In a systematic review and meta-analysis of men's 15 a-side rugby, based on 37 studies across many rugby-playing nations including Australia, England, Ireland, New Zealand, and South Africa, the overall incidence of concussions in rugby during match play was reported to be 4.73 per 1000 player match hours (Gardner et al., 2014).

More specifically, in South Africa, over the course of one rugby season it was reported that between 3% and 23% of adult rugby players experienced a concussion (Shuttleworth-Edwards et al., 2008). In comparison, a higher 30% for adult club rugby players and 23% for university rugby players in New Zealand reported a concussion over the course of one season (Wills & Leathem, 2001), and 14% of male Australian community rugby players, aged 15 to 48 years, experienced a concussion over the course of twenty match playing hours in a season (Hollis et al., 2011). With the high frequency of head collisions and concussions in rugby compared to other contact sports, rugby players thus provide an ideal sample by which to assess neuropsychological deficits associated with concussion and repeated subconcussive injuries.

Despite efforts at reporting epidemiological data, it is considered that variable case definitions, player motivation, and a lack of physician observation at games, has resulted in many sports-related concussions being unrecognised and underreported (Belanger, Spiegel, & Vanderploeg, 2010; Cusimano, Chipman, Volpe, & Donnelly, 2009; Echlin et al., 2012; McCrory et al., 2013a; Shuttleworth-Edwards et al., 2008). Herein lies the problem; medical professionals, coaches, and players are not consistently able to correctly identify and

diagnose on-field concussions during a sporting activity and furthermore, the role of subconcussive head injuries are not accounted for, even though the associated pathophysiology may potentially give rise to both types of injury.

### **Pathophysiology of Concussion**

In terms of its pathophysiology, concussion is defined as any transient neurologic dysfunction resulting from a biomechanical force (McCrea, 2008). In the case of head collisions or impacts sustained during a contact sport, there is a resulting acceleration and deceleration of the brain. These include linear acceleration, rotational acceleration, and impact deceleration forces.

Linear acceleration forces to the brain are associated with more focal brain injuries. For example, impact decelerations occur when the head rapidly decelerates after striking a solid object or fixed structure, such as the playing field or the body/head of an opponent player. This impact can potentially lead to a coup and contrecoup injury of the cerebral cortex, such that the brain is subject to focal contusions in the frontal and posterior regions. However, linear acceleration forces, as a result of the brain moving in the anterior-posterior direction, may also produce gliding contusions in the parasagittal regions of the cerebral cortex and under these conditions axonal injury is particularly vulnerable in the brainstem (Jordan, 1987; Jordan, 2013; Namjoshi et al., 2013).

Regarding rotational forces, these render the brain more vulnerable to diffuse injuries, more commonly resulting in the stretching and tearing of neuronal axons (McKee, Daneshvar, Alvarez, & Stein, 2014; Meaney et al., 1995; Meehan & Bachur, 2009). As a result of these mechanical forces being transduced through the brain and skull, there is a consequent deformation of the brain resulting in the stretching of neural, glial, and vascular tissue (Povlishock, 1992). The implication of these mechanical forces may lead to a consequent neurophysiological cascade, and secondary injury pathways.

This cascade is predominantly characterised by the release of neurotransmitters, ionic shifts, altered metabolism, impaired connectivity, or changes in neurotransmission; these changes in the brain are what underlie the clinical manifestation of concussion (Bigler & Maxwell, 2012; Dimou & Lagopoulos, 2014; Giza & Hovda, 2001; Jordan, 2013; McKee et al., 2014). Although some evidence suggests that there is a rapid reversal of these changes and a return to normal neuro-metabolic function within several days post-injury (Giza & Hovda, 2001; Yoshino, Hovda, Kawamata, Katayama, & Becker, 1991), more recent research indicates that despite the resolution of clinical symptoms and a return to baseline cognitive performance, neuronal functioning may remain abnormal for up to a few weeks to years, post-injury (Lovell et al., 2007; Pearce et al., 2014; Pearce et al., 2015; Prichep, McCrea, Barr, Powell, & Chabot, 2013). In light of this, researchers suggest that ongoing neurophysiological changes in the brain may underlie evidence for persisting neurological and neuropsychological impairment following a history of concussion and subconcussive head injuries, and the potential increased vulnerability of individuals presenting with these injurious conditions to secondary insults (McKee et al., 2014; Stein, Alvarez, & McKee, 2014).

### **Neuropathology of Concussion**

Regarding the neuropathological consequences of concussion, early research provided objective evidence for diffuse axonal injury (DAI), microhaemorrhaging, and microglial activation in the cortex following concussion injuries (Blumbergs et al., 1994; Oppenheimer, 1968). Specifically, Blumbergs et al. (1994) demonstrated that multifocal axonal injury was prevalent in the fornices, nerve fibre bands considered to have a primary role in learning and memory. Given the resulting physiological forces acting on the brain, damage to white matter is considered to be a primary pathophysiological consequence of concussion.

More recent neuroimaging using diffusion tensor imaging (DTI) in patients with concussion, have enabled researchers to investigate white matter microstructure, integrity and connectivity (Aoki & Inokuchi, 2016; Aoki, Inokuchi, Gunshin, Yahagi, & Suwa, 2012; McKee et al., 2014). In a meta-analysis of DTI research, researchers found abnormal structural connectivity between the thalamus and prefrontal cortex, as well as abnormal intra- and inter-hemispheric connectivity, particularly involving the prefrontal cortex (Aoki & Inokuchi, 2016). Other neuroimaging research with sports-related concussion have shown evidence for acute diffusivity changes in white matter (Henry et al., 2011; Meier, Savitz, Singh, Teague, & Bellgowan, 2016; Orr et al., 2015), changes in EEG brain activity (Haneef, Levin, Frost, Mizrahi, 2013; Henry et al., 2016a), abnormal motor changes in motor cortex inhibition (Pearce et al., 2015), and functional changes as a result of collision events recorded by fMRI, particularly in the frontal medial and superior orbital, and hippocampus regions (Talavage et al., 2014). Henry et al. (2011) also reported acute diffusivity changes in the white matter following sports-related concussion. Further, patients presenting with mixed-mechanism concussion have also demonstrated lower fractional anisotropy and higher diffusivity in various neural networks associated with working memory, in comparison with matched healthy control subjects (Grossman et al., 2013; Messé et al., 2011).

In light of these pathological consequences of concussion, the following section will aim to give an account of the clinical manifestation of these changes in the acute post-concussion phase and the assessment thereof.

### **Clinical Symptoms in the Acute Post-Concussion Phase**

The diagnosis and prognosis of concussion traditionally relies heavily on both medical health professionals identifying clinical symptoms, and the subjective reporting of symptoms by patients. Similarly, for sports-related concussion, clinicians rely on these symptoms for diagnostic purposes and to guide return-to-play decisions. Signs and symptoms

related to concussion, as defined by McCrory et al. (2013a; see Appendix A), predominantly include loss of consciousness, amnesia, somatic symptoms (i.e., headaches and dizziness), sleep disturbances, behavioural changes, cognitive impairment, and/or emotional symptoms.

In an extensive review of the literature in 2004, the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury reported that following mixed-mechanism concussion (i.e., not specifically sports-related), symptoms in both children and adults are transient, with a resolution of symptoms within a few days to weeks post-injury; there was limited evidence to suggest that these symptoms persist beyond three months post-injury (Carroll et al., 2004).

Specifically regarding concussion in sport, the National Collegiate Athletic Association (NCAA) Concussion Study, Project Sideline, and the Concussion Prevention Initiative were launched as large multicentre prospective projects to incorporate a standardised approach to assessing symptom recovery following a sport-related concussion in the United States (McCrea, 2008). For the majority of cases in these studies, favourable recovery in the acute post-concussion phase is reported, from approximately one to two weeks post-concussion. Concussion symptoms were noted to be the most severe immediately post-concussion, with evidence for initial symptom recovery already reported within two hours post-injury. Repeated assessments in the post-concussion phase of recovery showed a gradual pattern of symptom recovery over the first few days, such that by seven days post-injury no significant differences between the injured group and normal controls were evident. Overall, across a wide data set, 85% and 97% of injured subjects reported a full symptom recovery within a week and one month, respectively. A consistent pattern of recovery has been shown for high school, university, and adult sports players (Belanger & Vanderploeg, 2005; Benson, Meeuwisse, Rizos, Kang, & Burke, 2011; Broglio & Puetz, 2008; Guskiewicz et al., 2003; Marshall, Guskiewicz, Shankar, McCrea, & Cantu, 2015; McCrea et al., 2003; McCrea, 2008; Randolph et al., 2009).

Despite the majority of the literature on post-concussion recovery reporting transient symptomatology, there are still studies, however limited in number, that show a subset of athletes with a prolonged period of symptom recovery in the acute post-concussion phase (e.g., Guskiewicz et al., 2003; Lau, Collins, & Lovell, 2011; Lau, Lovell, Collins, & Pardini, 2009; McCrea et al., 2003; Pellman, Viano, Tucker, Casson, & Waeckerle, 2003). This research indicates that 10% to 15% of collegiate and professional American football players have symptoms beyond 10 days post-concussion. Prolonged recovery (i.e., more than 10 days) was also shown in over 30% of cases following concussion with male professional ice hockey players (Benson et al., 2011). Furthermore, in their studies, Lau and colleagues (2011) reported that half of all high school athletes took longer than 14 days to recover from concussive symptoms. Covassin and colleagues reported a similar prolonged recovery from sports-related concussion lasting approximately three to four weeks, which is longer than a typical recovery of one to two weeks (Colvin et al., 2009; Covassin et al., 2006).

When symptoms persist longer than three months, a diagnosis of Post-Concussion Syndrome, as defined by the DSM-IV-TR (American Psychiatric Association (APA)), may be made. Among the symptoms typically reported, are headache, depression, difficulty concentrating, fatigue, difficulty sleeping, and feeling 'in a fog' or 'slowed down' (Cantu, Guskiewicz, & Register-Mihalik, 2010; Makdissi et al., 2010; McCrea et al., 2003). These symptoms are however non-specific and are also prevalent in non-concussed athletic populations and patients with other injuries, or illnesses at baseline (Alla, Sullivan, & McCrory, 2012; Meares et al., 2011). Hence, it may be difficult at times to distinguish between post-concussive symptomatology specifically, versus other possible mechanisms for players' presentation.

Further, given the mixed evidence for prolonged recovery in the acute post-concussion phase, McCrea et al. (2013) set out to investigate some of the factors influencing

prolonged recovery amongst some high-school and college athletes. Their research identified that only 10% of athletes had a prolonged symptom recovery (beyond seven days), and that this was associated with more severe acute symptoms post-concussion, such as loss of consciousness. This data also showed that 2.3% of the entire sample reported elevated symptoms of recovery from 6 to 12 weeks post-concussion, however, there were no significant deficits on objective measures of neuropsychological or postural stability testing during this interval.

There is also research to suggest that persisting symptoms of concussion beyond the typical recovery period of several days or weeks post-concussion may be attributable to other secondary factors. These include demographics (e.g., being female and older age), psychosocial (pre-existing psychiatric disorder, lack of social support), medical (prior history of TBI's, neurological disorder) and situational factors (litigation, motivation to play; Carroll et al., 2004; Carroll et al., 2014; McCrea, 2008). In light of these potential confounding factors and the relative non-specificity of concussion symptoms, beyond the assessment of traditional signs and symptoms associated with concussion, neuropsychological testing has been shown to detect persisting cognitive deficits in the absence of concussion symptoms (Broglia, Macciocchi, Ferrara, 2007a; Makdissi et al., 2010; Talavage et al., 2014).

**Assessment of clinical symptoms.** Much of the research investigating clinical outcomes and recovery in the acute post-concussion phase have analysed data from large multi-centre projects that have utilised symptom scales and brief cognitive assessment tools that have traditionally been used to assist with the diagnosis and management and return-to-play decisions following sports-related concussions. In an effort to collaborate the most representative of symptom data following concussions, Randolph et al. (2009) derived the Concussion Symptom Inventory (CSI) – it was subsequently recommended as a research and clinical tool for monitoring recovery from sport-related concussion (Broglia et al., 2014).

Despite the advent of such measures, specific to sports-related concussion, research suggests that the utility of symptom reports in concussion management is minimised given its limitations for being susceptible to low specificity. Furthermore, concussion-like symptoms are often reported by dehydrated athletes and those who have been involved in strenuous activity (Patel, Mihalik, Notebaert, Guskiewicz, & Prentice, 2007), and results may be skewed given that athletes may be motivated to continue their activity after injury (Gregory, 2010; McCrea, Hammeke, Olsen, Leo, & Guskiewicz, 2004). Consequently, players may underreport or minimise symptoms.

The use of standardised neuropsychological testing is considered to be the cornerstone of concussion assessment, but should be used in conjunction with immediate, on-field symptom and motor-control assessments (Broglia et al., 2014). This is due to comprehensive neuropsychological assessments often being time consuming, and that they should ideally be administered in an optimal testing environment, free of distraction. As a result of this, administering these comprehensive test batteries immediately post-injury is uncommon. Hence, some research has focused on the development of cognitive assessment tools tailored for sports-related concussion and for its immediate and on-field use. These include, but are not limited to, the Standardised Assessment of Concussion (SAC; McCrea, Randolph, & Kelly, 2000) and the Sports Concussion Assessment Tool – 3<sup>rd</sup> Edition (SCAT3; McCrory et al., 2013b).

However, evidence suggests that while the SAC and other brief cognitive screening instruments are sensitive measures of cognitive impairments in the acute post-injury phase, these measures are less accurate for detecting subtle long-term neuropsychological impairments that may persist beyond the acute phase of recovery. In this regard, it is posited that more comprehensive neuropsychological test batteries may be better placed to detect subtle cognitive impairments in sports players with concussion who had otherwise reported a

complete symptom recovery (Giza et al., 2013; Karr, Areshenkoff, Garcia-Barrera, 2014; McCrea et al., 2005; Talavage et al., 2014). By using a comprehensive neuropsychological and cognitive assessment, clinicians can negate many of the moderating variables that accompany on-field assessments, such as fatigue, dehydration, and motivation to continue playing. More importantly though, this type of assessment allows for an informed and relevant assessment of domains pertinent and vulnerable to the neuropathological consequences of concussion.

**Cognitive impairment in the acute post-concussion phase.** There is huge variability regarding the effect of concussion on cognitive domains in the acute post-concussion phase. This variation is likely attributable to the heterogeneous operational definitions for concussion and cognitive domains, and the inherent variability regarding sample populations and research methodologies (Carroll et al., 2014; Karr et al., 2014).

Studies investigating the acute neuropsychological effects of concussion (mixed-mechanism) using more subtle and detailed neuropsychological measures of cognitive functioning reveal that the cognitive sequelae typically include the following difficulties: attention and working memory, two modalities associated with executive control (Heitger et al., 2006; Keightley et al., 2014; Talavage et al., 2014), learning and memory (Clark et al., 2016; Heitger et al., 2006; Karr et al., 2014; Mathias, Beall, & Bigler, 2004), and processing speed and reaction time (Gardner et al., 2010; Heitger et al., 2006; McCauley et al., 2014; Rohling et al., 2011). Furthermore, Rohling et al. (2011) indicate that in the acute post-concussion phase working memory showed the most significant decline compared to verbal memory, visual memory, processing speed, verbal comprehension, and perceptual reasoning. Working memory has previously been referred to as a hallmark cognitive deficit of mTBI (McAllister, Flashman, Sparling, & Saykin, 2004).

Regarding memory, neuropsychological impairment has been reported for both visual and verbal memory (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Comerford, G. Geffen, May, Medland, & L. Geffen, 2002; Heitger et al., 2006; Landre, Poppe, Davis, Schmaus, & Hobbs, 2006; Mathias et al., 2004) following mixed-mechanism concussion. Results of a meta-analysis on cognitive domains affected following sports-related concussion also show large effect sizes for memory acquisition ( $d = .78$ ) and delayed memory ( $d = .60$ ) on verbal and visual memory tasks (Belanger & Vanderploeg, 2005; Rohling et al., 2011).

Comparatively, visuospatial and motor skills have been found to be the least effected domains of functioning post-concussion (Belanger et al., 2010; Rohling et al., 2011; Talavage et al., 2014).

*Association between cognitive outcomes and neuropathological consequences in the acute post-concussion phase.* In light of the evidence reviewed, the cognitive domains mostly implicated with concussion appear to be consistent with neuropathological and neurological changes in the acute post-concussion phase. Taking this into account, the functioning of the prefrontal cortex relies heavily on cerebral connectivity, a function of white matter in the brain. In general, association fibres, commissural fibres, and projection fibres comprise the core of white matter in the cerebral hemisphere (Haines, 2013). In doing so, the prefrontal cortex is able to synthesise a multitude of information, forming a primary role in cognitive control, notably involving attention and working memory processes that are critical for the support of the functioning of the frontal executive system (Niendam et al., 2012). In turn, although these processes are not directly involved with the neural systems for learning and memory, they do act as a gateway cognitive aid that enables this process to take place. Furthermore, thalamo-cortical fibres play a distinct function in memory formation and consolidation, and this is particularly relevant given the findings reported by Aoki and

Inokuchi (2016) regarding abnormal connectivity between the thalamus and prefrontal cortex. Furthermore, speed of information processing is dependent on the optimal functioning of white matter in the brain. In light of this information, the vulnerability of white matter and impaired connectivity in the brain, particularly in the prefrontal cortex, as a result of the physiological forces acting on the brain due to concussion, validates the predominant research indicating impaired attention and working memory, memory, and processing speed abilities.

**Investigating the course of cognitive recovery post- concussion with formal neuropsychological assessment.** Regarding mixed-mechanism concussion, including for military personnel, there is a consensus for the presence of initial cognitive deficits in adults up to 2 weeks post-concussion, again with these not persisting beyond three months (Boyle et al., 2014; Cancelliere et al., 2014; Carroll et al., 2014; Fazio et al., 2007; Majerske et al., 2008; Schretlen & Shapiro, 2003). This was further corroborated for both global cognitive functioning and for specific cognitive domains following mixed-mechanism concussion (Brenner et al., 2010; Frenchman, Fox, & Maybery, 2005; Tellier et al., 2009; Vasterling et al., 2012).

The course of cognitive recovery from sports-related concussion has generally been shown to be similarly quick and favourable (Broglia & Puetz, 2008; Cancelliere et al., 2014; Echemendia, Putukian, Mackin, Julian, & Shoss, 2001; Guskiewicz et al., 2003; Henry, Elbin, Collins, Marchetti, & Kantos, 2016b; Lovell, et al., 2003; McClincy, Lovell, Pardini, Collins, & Spore, 2006; McCrea, 2008; Nelson et al., 2016). Meta-analytic reviews illustrate that should cognitive difficulties be present in the acute post-concussion phase, these do not persist (Belanger & Vanderploeg, 2005; McCrea et al., 2013; Rohling, et al., 2011; Schretlen and Shapiro (2003).

Other research regarding mixed-mechanism concussions replicate these findings, illustrating that following the acute post-injury phase (i.e., 1-3 months post-concussion), concussion has no significant effect on cognitive functioning (Brenner et al., 2010; Frenchman et al., 2005; Tellier et al., 2009; Vasterling et al., 2012). These results are for both global cognitive functioning and for specific cognitive domains following mixed-mechanism concussion. Furthermore, McCrea et al. (2013) reported that even in athletes who showed prolonged clinical symptoms of concussion (according to the SAC, Graded Symptom Checklist (GSC), and Balance Error Scoring System (BESS) scores) from 45 to 90 days post-concussion, cognitive deficits had resolved by this time.

In summary, the majority of literature concerning the neuropsychological recovery following concussion, mixed mechanism and sports-related, demonstrates favourable recovery over the course of several days to weeks post-concussion and that there appears to be no residual effects by three months post-injury. However, even though the majority of the literature points to the transiency of concussion and related difficulties, there is also some research suggesting persistent neuropathological and cognitive impairment beyond the acute phase.

### **Persisting Neuropathological Consequences Associated with Recovery Post-Concussion**

Recent and sophisticated investigations have identified objective evidence, such as vestibule-ocular dysfunction, for neuropathological changes beyond the acute post-concussion phase (Ellis et al., 2015). Henry et al. (2011) demonstrated neurometabolic differences between concussed college athletes and non-contact healthy controls in the acute phase of testing, as well as the delayed phase of testing. In their study, concussed athletes were scanned at five days and six months post-concussion, with controls also being scanned on two occasions. Furthermore, concussed children, adolescents and adults who were

assessed more than six months post-concussion, also showed lower EEG activity compared to non-concussed athletes (Baillargeon, Lassonde, Leclerc, Elleberg, 2012).

What this research shows is objective evidence for neurological changes persisting beyond the acute post-concussion phase, despite an abundance of evidence for a complete neuropsychology recovery within weeks of injury. Such research may potentially be correlated with delayed neuropsychological recovery, evidenced with a minority of cases, but also demonstrate a possible vulnerability to severe long-term outcomes. Moreover, what this evidence suggests is that despite an apparent full symptom recovery, one may still be vulnerable on a neurological and potentially, neuropsychological level.

**Persistent cognitive symptoms post-concussion.** Despite the results of studies on concussion reflecting a mostly favourable cognitive recovery, there is evidence, at least in some studies, for a slightly delayed cognitive recovery in the acute post-concussion phase. Researchers have conjectured that persisting cognitive impairments post-concussion, could be related to ongoing neuropathological consequences (Breedlove et al., 2012; Henry et al., 2016a; Talavage et al., 2014). Using the ImPACT and a brief interview and clinical examination to assess vestibular symptoms and impairment, including dizziness, imbalance, and oculomotor components, Henry et al. (2016a) reported a delayed recovery of between 21 and 28 days following sports-related concussion for most athletes, across all outcome measures. More specifically, clinical symptoms, as measured by the Post-Concussion Symptom Scale (PCSS) of the ImPACT battery, resolved the quickest, over the first two weeks, with neurocognitive impairment persisting across all domains up to 28 days post-concussion. Regarding cognitive abilities assessed by Henry et al. (2016b), there was some variability, indicating specific domains of functioning may be more vulnerable to prolonged recovery. Specifically, verbal memory showed a slower, gradual recovery trajectory, with no impairment evident after four weeks only. However, it should also be noted that Henry et al.

(2016b) and the studies cited in validation of a slightly longer cognitive recovery tended to use a young, adolescent sample, compared to studies evidencing quicker recovery times, which report primarily on male college athletes (Colvin et al., 2009; McCrea et al., 2005; Prichep, et al., 2013).

With a cohort of 2000 high-school athletes, Covassin, Elbin, and Nakayama (2010) tested players prospectively at baseline, followed up by five repeated testing sessions up to 30 days post-concussion. The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) battery was used for the assessment of cognitive functioning. The data for this study showed a predominantly favourable cognitive recovery by 14 days for verbal memory and motor processing speed, however, with reaction time performances only comparative to baseline testing scores after 21 days post-injury.

Further, evidence for a limited research pool demonstrating enduring cognitive difficulties was also corroborated by the results of a systematic review by Carroll et al. (2014). Combined, 21 studies, all with a minimum of 30 concussion cases with an objective measure of cognitive outcomes, mostly revealed the presence of cognitive deficits within two weeks post-concussion, but with some evidence suggesting that complete recovery may take six months to a year.

Regarding specific cognitive domains of functioning, a limited number of studies indicate that in some individuals, certain cognitive deficits do persist for longer than three months (Bigler et al., 2013; Carroll et al., 2014). Attention (Catale, Marique, Closset, & Meulemans, 2009; Maruta, Suh, Niogi, Mukherjee, & Ghajar, 2010), memory (Catale et al., 2009; Vanderploeg, Curtiss, & Belanger, 2005), some executive functions (Erez, Rothschild, Katz, Tuchner, & Hartman-Maeir, 2009) and information processing (Kinnunen et al., 2011) abilities have all been implicated in terms of persisting post-concussive cognitive effects. Furthermore, in their year-long prospective study, Heitger et al. (2006) indicated that deficits

in verbal learning had not resolved by 3 or 6 months, with full recovery only shown at a 12 month assessment. Given some evidence for delayed recovery for cognitive functions beyond the acute post-concussion phase, it is considered that research investigating long-term recovery trajectories do provide evidence for persisting impaired post-concussive cognitive outcomes.

**Possible reasons for inconsistencies.** In light of these apparent inconsistencies with regards to research on cognitive recovery post-concussion, Dean and Sterr (2013) indicate that this is likely due to a variety of different tasks and distinct samples, of varying ages, sex, included in the various studies. Shum, Levin, and Chan (2011) also comment on the problematic issue of inherent heterogeneity between individuals with different severity and mechanisms of head injuries. With such an abundance of research aimed at testing numerous cognitive domains, inconsistent operational definitions for these is especially problematic, particularly for meta-analytic reviews attempting to corroborate these results. This is especially relevant for heterogeneous outcomes for executive functions, for example (Karr et al., 2014).

In addition, other moderating variables associated with individual characteristics have been reported to impact on concussive symptoms, thus blurring recovery trajectories. Rather than as a consequence of concussion, Vasterling et al. (2012) report that PTSD and depression are associated with more enduring cognitive deficits post-concussion and thus controlling for these variables amongst others is important. Moderating variables that have predominantly been identified include, but are not limited to, demographic (younger age, female sex; Baker et al., 2016; Corwin et al., 2014; Makdissi, Cantu, Johnston, McCrory, & Meeuwisse, 2013a; Miller et al., 2016; Scopaz & Hatzenbuehler, 2013), head injury severity (loss of consciousness, posttraumatic amnesia, increased symptom burden; Kontos et al., 2013; Lau, Kontos, Collins, Mucha & Lovell, 2011; Makdissi et al., 2013a; McCrea et al.,

2013;), a prior concussion history (Makdissi et al., 2013b; Scopaz & Hatzenbuehler, 2013), medical (ADHD and other mood disorders; Corwin et al., 2014; Miller et al., 2016), and situational factors (i.e., litigation, motivation to play, delayed reporting of symptoms; Carroll et al., 2014; McCrea, 2008). Nevertheless, evidence is extremely varied regarding the influence of possible secondary factors on a prolonged recovery post-concussion, and further research is necessary to collaborate and validate these findings.

Within contact sports environments, players are often highly motivated to continue playing, despite the fact that a concussion may have in fact occurred. This type of scenario, whereby symptoms of a potential concussion are either not reported, not recognised, or diagnosed after the fact with a lack of precaution taken to return-to-play decisions, creates a situation in which athletes are vulnerable to sustaining multiple concussions without the appropriate management and care thereof. In this regard, post-hoc investigations of patients and retired sports players who have experienced multiple concussions have illustrated that long-term neuropsychological consequences may be attributed to the repeated nature of these ‘mild’ (sometimes undiagnosed) head injuries (Baugh et al., 2012; Belson, 2015; Croatto, 2014; Lehman, Hein, Baron, & Gersic, 2012; McKee et al., 2009; Stewart et al., 2015).

### **Neuropsychological Effects of Multiple Concussions**

While the evidence is mixed, contrary to expectation, some research has shown that there is in fact also no enduring neuropsychological impairments beyond the acute post-concussion phase for sports players with a history of multiple concussions (Brooks et al., 2013; Collie, McCrory, & Makdissi, 2006; Iverson, Brooks, Lovell, & Collins, 2006; Mannix et al., 2014). In a more recent study Brooks et al. (2013) reported that there was no association with having a history of multiple concussions and poorer neuropsychological performance using the ImPACT battery, although subjective concussion symptoms were reported at baseline. Brooks et al. (2013) used adolescent hockey players between the age of

13 and 17 years, and these findings were further corroborated by Mannix et al. (2014) who reported that in a sample of student athletes having a history of two or more concussions was associated with a higher symptom burden; however, there was no strong or consistent association with cognitive abilities. Furthermore, it was indicated that baseline symptom reporting was more associated with clinical and demographic factors (i.e., mental health history, headache/migraine history, gender, developmental and/or learning problems) than with a history of multiple concussions; these factors combined accounted for only 10% of the variance in total symptoms scores. Although these two more recent studies provide evidence for a young population of contact sports players, they are consistent with a lack of cognitive effects after multiple concussions found with collegiate, elite, and adult contact sports players using the ImPACT for baseline neuropsychological assessments (Broglio, Ferrara, Piland, & Anderson, 2006; Collie et al., 2006; Thornton, Cox, Whitfield, & Fouladi, 2008).

This evidence is nevertheless based on retrospective research assessing baseline neuropsychological performance, rather than monitoring recovery trajectories. As result of this, although no between-group differences are evident, such research fails to investigate the extent of delayed recovery in concussed athletes presenting with a history of concussion.

Once again, however, there is evidence to suggest that players with a history of concussion may yet still show some neurological and neuropsychological vulnerability. Research has shown that high school and college athletes with a history of concussion may be more susceptible to sustaining a repeat concussion in the future (Covassin, Stearne, & Elbin, 2008; Covassin et al., 2013; Guskiewicz et al., 2003; Harris, Voaklander, Jones, & Rowe, 2012), and worse outcomes on tests of cognitive functioning in the acute stage following the last known concussion (Belanger & Vanderploeg, 2005; Covassin, Moran, & Wilhelm, 2013; Stephens, Rutherford, Potter, & Fernie, 2010; Terry et al., 2012). More specifically, sports players with a history of cumulative concussions performed worse on tasks of attention,

memory and reaction times, and reported more migraine- cognitive-fatigue symptom clusters than did athletes with only a single concussion in the acute post-concussion phase, or with no history of concussion (Covassin et al., 2013; Iverson, Gaetz, Lovell, & Collins, 2004; Stephens et al., 2010). This more severe outcome following repeat concussions may relate to what researchers refer to as Second-impact Syndrome (SIS): an increased vulnerability to a more severe secondary insult in the post-concussion recovery phase, given the resulting neuropathological consequences of concussion (Ford, Giovanello, Guskiewicz, 2013; Hart et al., 2013; Henry et al., 2016a; Randolph, Karantzoulis, & Guskiewicz, 2013).

This increased vulnerability of players in the acute post-concussion phase, especially following repeat concussion, may potentially relate to a slower recovery period. Recent experimental animal models also indicate the presence of long-term functional deficits following a history of multiple concussive head injuries (Mannix et al., 2013; Meehan, Zhang, Mannix, & Whalen, 2012). Enduring functional impairment due to a frequent history of concussion are also suspected in human subjects given that slower cognitive recovery times were associated with a history of multiple concussions in college American football players, particularly for those with three or more concussions (Guskiewicz et al., 2003). However, delayed recovery in this study was only defined as persisting beyond seven days following the last known concussion. More recently, Castile et al. (2011) reported that clinical symptoms took between a week and a month to resolve in 20.9% of those with recurrent concussions, compared with this rate of recovery in 13.8% of participants presenting with their first concussion. Concussion symptoms reported included, but are not limited to, headache, dizziness/unsteadiness, concentration difficulty, confusion/disorientation, amnesia, loss of consciousness. Further, 6.5% of players with recurrent concussions took longer than a month to resolve, compared to 0.6% of players presenting with their first concussion.

Nevertheless, research on the cumulative effects of multiple concussions is somewhat lacking. One idea for this is the relative low number of concussions sustained by any one player, particularly with a youth or college sample of athletes (Iverson et al., 2006). Hence, there may be a threshold number of concussions at which differences in self-reported and cognitive symptoms between control participants and concussed sports-players are regarded as statistically significant.

Other ideas regarding a lack of strong evidence for potential enduring neuropsychological outcomes post-concussion, including multiple concussions, relates to methodological limitations. For example, Carroll et al. (2014) and Karr et al. (2014) highlighted some of the methodological flaws with prior retrospective research and prior meta-analyses, and the difficulty collaborating these findings to provide a more consistent and reliable estimation of the overall long-term neuropsychological impact regarding a history of multiple concussions. In addition to some of the other moderating variables mentioned in this report, such as participant characteristics and operational definitions of concussion and cognitive domains, the severity of each concussion, the time since the last concussion, the nature of the comparison group, and the type of assessment used, are just some of the variables that can potentially impact on a lack of consistency in being able to detect long-term neuropsychological outcomes. Further, a lack of sufficient evidence of cumulative concussions may also be due to limited research in which neuropsychological outcomes beyond the acute post-concussion phase are investigated. There is thus a demand for research that investigates delayed recovery trajectories post-concussion (Karr et al., 2014).

### **The Neuropsychological Effects of Cumulative Subconcussive Head Injuries**

Relative to the evidence for the effects of cumulative concussions, the evidence for potential repetitive subconcussive head injuries is even more scant. Further, within the

limited literature on the topic, the reported impact of subconcussive head injuries on neuropsychological performance is mixed, owing largely to limited research and sample sizes in these studies (Bailes et al., 2013; Gysland et al., 2012; Talavage et al., 2014).

Despite the apparent absence of concussion-like symptoms, some research findings suggest that these head collisions, particularly when cumulative over time (Baugh et al., 2012), may be associated with short- and long-term neuropsychological and neurological impairment, and thus are cause for precautionary intervention (Bazarian et al., 2012; Breedlove et al., 2012; Gavett et al., 2011; McAllister et al., 2012; McKee et al., 2009; Stern et al., 2011; Talavage et al., 2014). Given the sub-clinical nature of such injuries, which are consequently not appropriately identified or managed, the incidence and management of these repeated injuries are likely underestimated.

Animal studies have provided good evidence for the long-term accrual of neurological damage following repetitive subconcussive head injuries. Furthermore, there is biophysical and advanced neuroimaging data to suggest some short-term decline in neuropsychological and neurological functioning following subconcussive head injuries (Abbas et al., 2015; Bazarian et al., 2012; Breedlove et al., 2012; Spiotta et al., 2011; Talavage et al., 2014). Further, there is also evidence for anisotropic and diffusional white matter changes in the brain following increased exposure to repetitive subconcussive impacts, in the absence of any clinical symptoms of concussion (Beckwith et al., 2013; Dashnaw, Petraglia, & Bailes, 2012; Martini et al., 2013; Spiotta et al., 2011). However, as noted, there is limited research to substantiate these results, especially given the lack of evidence to establish causality between repeated subconcussive impacts and these changes. Furthermore, there is also some evidence to suggest that there is in fact no clinically meaningful effect of subconcussive head injuries on neuropsychological outcomes (Gysland et al., 2012; Miller, Adamson, Pink, & Sweet, 2007).

However, more recently, Talavage et al. (2014) reported on one of the few studies to combine neuropsychological and neurological data for subconcussive head injuries. Comparing the potential impact of subconcussive and concussive head injuries on those outcomes, Talavage et al. (2014) identified an adolescent group of American football players who demonstrated no clinically observed impairment (COI-), but who had deviant scores (from baseline) on a measure of cognitive functioning (functionally-observed impairment: FOI+). Players in this group were identified as COI-/FOI+. The label for this group indicates that these players who were exposed to repeated head injuries, by participation in American football, presented with no clinical signs of concussion (i.e., no concussion diagnosis was made), but presented with comparatively lower neuropsychological performance scores from their baseline performance. Based on fMRI results, it was found that there existed significant changes in signal amplitude in working memory structures that were statistically more frequent in the COI-/FOI+ subjects. Thus, it is suspected that this group of subjects experienced neurologic trauma from repeated, subconcussive head injuries, potentially putting sub-clinical stress on neural tissue as previously described (Geddes, Vowles, Nicoll, & Revesz, 1999). While bearing in mind these results, it is important to also note that the study conducted by Talavage et al. (2014) had a limited sample size – twenty-one subjects participated in each aspect of the study. Thus, further research incorporating different contact sports, and for different age groups should be further explored to support or refute these findings.

In sum, the implications of these results for post- concussive outcomes are that, in the absence of observable or identifiable concussion symptoms, despite their vulnerability, players may continue to participate in practices and games (contact and non-contact). However, with respect to the neurophysiological changes associated with multiple concussions and/or subconcussive head injuries, it is not clear as to the amount of force per

impact, or cumulative total force, which renders an individual more vulnerable to cumulative brain trauma, and with regards to the relationship of this to potential subsequent long term impairment. Chronic Traumatic Encephalopathy (CTE) has been proposed as one such potential long term outcome.

### **Chronic Traumatic Encephalopathy (CTE)**

CTE refers to a neurodegenerative disease that is characterised by the accumulation of hyperphosphorylated tau (p-tau) in the brain. Although all confirmed cases of CTE report a history of repeated brain trauma, it is clear that not all individuals exposed to repeated brain trauma develop CTE; genetics has been identified as a likely risk factor in this regard (Gandy & DeKosky, 2012). However, in a recent review, Jordan (2013) argued that the strongest risk factors for CTE are increased exposure to head injuries in general (i.e., within a contact sports environment), and age. To support this claim, it was reported that in a subset of American football players with CTE (confirmed by autopsy), there was a positive correlation between its severity, amount of exposure to American football, years since retirement, and age at death (McKee et al., 2013). Furthermore, the amount of concussions (family-reported) were not significantly correlated with the severity of CTE in these patients. This point is extremely noteworthy and interesting as it illustrates that it may *not* be necessarily pertinent to only investigate, treat and manage contact sportsmen with concussions or more severe TBI's. Instead it suggests that contact sport players should be managed according to their exposure to high-impact head collisions, regardless of whether a player is diagnosed with a concussion (bearing in mind the inherent limitations of this). It was further noted by Baugh et al. (2012), Dashnaw et al. (2012), Gavett et al. (2011), and Stewart et al. (2015), that repetitive concussion *and* subconcussive head injuries may be linked to the development of CTE. These points are particularly relevant given the increasing vulnerability of contact sport players to

repeated concussion and subconcussive head injuries, in conjunction with no obvious enduring effects in the post-injury phase.

Given that clinicians are more aware of these outcomes following concussion and subconcussive head injuries, and given that these outcomes may occur without the correct identification and management of players, it is important to investigate means to detect when such injuries occur, and monitor player's neuropsychological and neurological functioning even in the absence of obvious signs of concussion. One method of doing so is to use objective markers of concussion.

### **Measuring Concussive and Subconcussive Head Injuries using Head Impact Telemetry (HIT)**

New technology that records head collision events to provide biomechanical data on head accelerations is known as head impact telemetry (HIT; Simbex, NH). By equipping athletes with this technology to quantify head collisions, research can attempt to identify the threshold of force needed to produce concussion-like symptoms, or to render a subject more vulnerable to experiencing neuropsychological or neurological impairment. Furthermore, such research can be used to investigate whether related symptoms are always present following a substantial head collision, or whether they present as a result of multiple subconcussive head injuries. However, it may also be true that sports players who sustain significant blows to the head may not necessarily present with a concussion clinically. As such, the force or cumulative forces sustained to the brain may be a better prediction of neuropsychological and/or neurological impairment, especially in the absence of these symptoms. Regarding the use of HIT, given the comparative high-risk of concussion in contact sport, this environment offers an ideal opportunity to assess the biomechanical forces involved with a concussion.

While research for collecting biomechanical data for head collisions in contact sport is ongoing, there is much variation as to what investigators use as a minimal limit at which recorded data is analysed. This minimal threshold is what is referred to as a data acquisition limit, and is a necessary limit so as to exclude 'noise' that is not related to actual head collisions, but general knocks that the athlete may incur. While impacts under 10g have been identified as noncontact events (Crisco et al., 2011; Ng, Bussone, & Duma, 2006), there has been limited research to identify a data acquisition limit for subconcussive head injuries (King et al., 2014), and prior research using data acquisition limits greater than 10g may be excluding subconcussive impacts from their dataset (Beckwith et al., 2013; Broglio et al., 2011). Nevertheless, the majority of research reports use a data acquisition limit of 10g (Crisco et al., 2012; Daniel, Rowson, & Duma, 2012; Hanlon & Bir, 2012; King et al., 2014; Mihalik et al., 2012; Reynolds et al., 2016). This acquisition limit was further recommended by X2Biosystems, a large developer and provider of HIT, which has previously been used in research with rugby (Cleary, 2015; Guinness, 2015; King et al., 2014).

Furthermore, studies have attempted to remodel concussion incidents to develop injury-risk curves for such injuries. In a study using American football players, a peak linear acceleration of 98g and peak rotational acceleration of 6432 rad/s<sup>2</sup> were the estimated values at which a concussion may occur. However, these values are considered to be an overestimation given that their analysis did not account for impact collisions not resulting in concussion (Baugh et al., 2012; King et al., 2014; Pellman et al., 2003). For Australian football (AFL) and rugby players that sustained a concussion, reconstruction of these real life events, reported mean linear acceleration values to be 103g and 8022 rad/s<sup>2</sup> for linear and rotational acceleration forces respectively (Fréchède & McIntosh, 2009).

In light of such research that attempts to remodel concussion type injuries, the following values have been identified as indications of an increased risk for sustaining a

concussion, referring to these values as injury tolerance levels:  $> 95g$  for linear acceleration forces and  $> 5500 \text{ rad/s}^2$  for rotational acceleration forces. Furthermore, studies have also established impact severity limits for linear acceleration (mild,  $<66g$ ; moderate,  $66-106g$ ; severe,  $>106g$ ) and rotational acceleration forces (mild,  $<4600 \text{ rad/s}^2$ ; moderate,  $4600-7900 \text{ rad/s}^2$ ; severe,  $>7900 \text{ rad/s}^2$ ; Harpham, Mihalik, Littleton, Frank, & Guskiewicz, 2014; Ocwieja et al., 2012; Zhang, Yang, & King, 2004).

Nevertheless, despite these forces being identified as having a risk for a concussive head injury, this research can be somewhat misleading. McCrea and Powell (2012) report that in an ongoing study using HIT with college football players an average magnitude of  $95g$  for concussion impacts was observed, with a range of  $60-120g$ . However, while six of the nine players who sustained a concussion recorded a concussive impact greater than  $95g$ , the concussive impacts for these six players account for less than 1% of the 27000 non-concussive impacts that were recorded above  $95g$ . These results are in support of the fact that an overwhelming majority of sports-players that sustain a force to the brain capable of producing concussion-like symptoms, do not present clinically as such (i.e., they present with subconcussive head injuries). In light of this information, it is concerning that these players exposed to high-impact subconcussive head injuries, in spite of having endured a substantial impact, are vulnerable to repeat injuries of this same magnitude. The short- and long-term impact of such injuries, recurring repeatedly without appropriate interventions, is concerning.

In terms of other literature in this area, in a recent study that set out to investigate subconcussive head injuries in a non-helmeted contact sport, King et al. (2014) recorded 20687 impacts greater than  $10g$ . Their research was conducted with amateur rugby players in New Zealand. Although the results show that the majority of impacts were recorded in the 'mild' category of impact severity ( $< 66g$  and  $<4600 \text{ rad/s}^2$ ), as proposed by Zhang et al. (2004), mean linear accelerations measured over the season of matches were similar to mean

linear accelerations reported in studies of American high school (Broglia et al., 2010; Eckner, Sabin, Kutcher, & Broglia, 2011; Urban et al., 2013) and collegiate American football players (Beckwith et al., 2013; Rowson, Brolinson, Goforth, Dietter, & Duma, 2009). Mean rotational accelerations measured were higher than those for American youth (Daniel et al., 2012), high school (Broglia et al., 2010; Urban et al., 2013), and collegiate (Beckwith et al., 2013; Rowson et al., 2009) American football players. With this research being novel, there is no standardised HIT device that is used. For example, King et al., (2014) used a moulded instrumented mouth guard (X2Biosystems Inc.), whereas Talavage et al. (2014) used HIT sensors that were fitted to a helmet worn by American football players.

Talavage et al. (2014) demonstrated that while peak acceleration forces were not predictive of neurological trauma (i.e., structural changes), there is a significant correlation between the number and severity of head collisions, as measured by a Riddell Revolution helmet (Riddell, Elyria, OH) fitted with a sensor array (HIT System), and performance on neurocognitive testing. These results showed that players who were not diagnosed with a concussion but who demonstrated significantly lower scores on cognitive testing had a significantly greater number of head collision events. For collisions between 20g and 80g, this group experienced the most number of head collisions to each region of the helmet, and for collisions greater than 80g, this group experienced significantly more collisions to the top-front of the helmet.

### **Summary and Rationale**

In summary, there is sufficient evidence to suggest that in the acute post-concussion phase of recovery, a contact sports player may experience some neuropsychological impairment. However, the short-term neuropsychological recovery and prognosis of these injuries are relatively favourable and players are mostly quick to return to play. It is nevertheless important to pay attention to research suggesting that a minority of contact

sports players present with slightly delayed symptom recovery; this has been shown to be true for more observable concussive symptoms and subtle cognitive abilities. Furthermore, there is also some evidence to suggest that contact sports players presenting with multiple concussions also show a vulnerability to subsequent concussion, more severe neuropsychological outcomes in the acute post-concussion phase, and a prolonged recovery trajectory. Despite the fact that these cases rarely persist beyond the acute post-concussion phase (i.e., more than three months post-injury), objective neurological evidence does indicate persisting neurological changes beyond this period even if only for a minority of sporting individuals.

Outcomes are predominantly only investigated with players with diagnosed concussion(s). It has nevertheless been demonstrated in some studies that sports players suffering from multiple head injuries, in the absence of obvious clinical symptoms (i.e., subconcussive impacts), may experience neuropsychological decline from baseline. Also, it has been demonstrated that players who had experienced cognitive decline in the absence of a concussion, did so to the same degree as those who had been diagnosed with a concussion. These players also experienced a greater number of head collisions, and collisions of a similar or greater force to players who were diagnosed with a concussion (Talavage et al., 2014). Combined, the implication of this research thus suggests that with, or without a concussion diagnosis, contact sport players may be returning to a contact sports environment prematurely. Consequently, these players are thus vulnerable to cumulative brain trauma and the related sequelae, despite the relative absence of enduring concussive symptoms. Moreover, as a result of an increased exposure to repeated 'mild' brain trauma in the absence of any symptoms to alert medical attention, these players may be at risk of more serious long-term neuropsychological difficulties, such as CTE (Broglio et al., 2014; Gavett et al., 2011; Lehman et al., 2012).

Given the high incidence rate of concussion and highly probable subconcussive head injuries in rugby, the first of three studies presented in this dissertation, Study 1, aims to investigate the relationship between neuropsychological outcomes and history of concussion following repeated exposure to these injury conditions. Given the inherent difficulties with relying on a concussion diagnosis and given the potential deleterious impact of subconcussive head injuries, the focus of Study 2 will be to pilot the use of an objective measure of head collisions in rugby (HIT xPatch sensor) with a sample of club rugby players in South Africa. In Study 3, the relationship between the HIT data and neuropsychological outcomes is explored. In sum, such research aims to provide a measure of a player's vulnerability to having been exposed to repeated high-impact head collisions, and thus potential, cumulative brain trauma. Furthermore, the studies presented here will contribute to a larger prospective investigation into the short- and long-term neuropsychological and neurological effects of subconcussive and concussion head injuries among South African rugby players.

**Study 1: Investigating the relationship between cognitive performance and concussion history in a sample of rugby players and non-contact sports players**

Research has demonstrated at least some evidence, albeit limited, for a slightly delayed cognitive and symptom recovery in the acute post-concussion phase for some sporting individuals (Benson et al., 2011; Carroll et al., 2014; Covassin et al., 2010; Guskiewicz et al., 2003; Henry et al., 2016b). It has also been shown in a small cohort of contact sport athletes and with some animal experimental models that neurophysiological changes post-concussion may persist beyond the acute-post concussion phase (Baillargeon et al., 2012; Ellis et al., 2015; Henry et al., 2011; Mannix et al., 2013; Meehan et al., 2012).

There is also some evidence to suggest that having a history of multiple concussions can be associated with a slower recovery time for post-concussion symptoms and an increased vulnerability to clinical and cognitive impairment in the acute post-concussion phase, rather than reflecting any enduring clinical consequences (i.e., after three months; Belanger & Vanderploeg, 2005; Castile et al., 2011; Covassin et al., 2008; Covassin et al., 2013; Guskiewicz et al., 2003; Stephens et al., 2010). Furthermore, individuals are more likely to be susceptible to a repeat concussion if they have a history of prior concussions (Covassin et al., 2013; Guskiewicz et al., 2003; Harris et al., 2012). Although unfavourable cognitive and clinical outcomes have been described in the acute post-concussion phase, evidence for enduring neuropsychological difficulties following a history of concussions, is somewhat limited (Covassin et al., 2013; Karr et al., 2014).

Combined, research investigating the cumulative impact of multiple concussions in rugby on neuropsychological performance is especially scant, particularly with adult rugby players who may be at risk of a higher previous exposure to concussive and subconcussive head injuries, given the longer history of participation compared to younger players (Gardner et al., 2014).

Further, given even some variation in the literature, however limited, about the relationship between a history of concussion and the possibility of persistent symptomatology and unfavourable neuropsychological effects, this warrants the continued investigation of this relationship particularly in sporting individuals.

### **Aims and Hypothesis**

The aim of Study 1 is thus to investigate the relationship between rugby players with a history of exposure to concussive head injuries (with the last concussion not being closer than three months prior to testing) compared to rugby players without a history of previous

concussions and to non-contact sportsmen. In this regard, I will test the following hypothesis:

Rugby players with a history of concussion (with the last concussion not being closer than three months prior to testing) will perform worse on neuropsychological outcomes than rugby players without a history of concussion. A dose response relationship is expected such that players with a more severe concussion history are expected to perform worse.

Further, non-contact sport controls will show better scores on neuropsychological outcomes than rugby players.

## **Methods**

**Design and setting.** Study 1 is retrospective and quantitative. Using descriptive analyses, I investigated the relationship between prior concussions and measures of cognitive and self-report symptom outcomes among rugby players and non-contact sport participants. I divided the rugby players into two groups: rugby players with previously reported concussions (Rugby Concussed group) and rugby players without previously reported concussions (Rugby Not Concussed group). The non-contact sport participants formed the Control group, which served to control for the effect of sport involvement and exercise, without the impact of concussion.

With the assistance of a co-researcher, I conducted baseline assessments that involved the completion of screening, and cognitive and self-report measures, with all participants. These testing sessions took place in a private computer room at the University of Cape Town (UCT).

**Participants.** I recruited rugby playing participants that constituted the two Rugby groups ( $N = 57$ ) from the University of Cape Town Rugby Football Club (UCTRFC), Western Province Rugby Academy (WPRA), Villager Football Club (VFC), and from a rugby team participating in the UCT Internal Rugby League (referred to here as the UCT IRL

team), using purposive sampling techniques (see Figure 1 for a breakdown of eligible participants from each rugby club). The commitments and requirements for each rugby club were outlined and subsequently agreed upon by the club management prior to completing any testing or recruitment procedures with prospective players. Each of the rugby clubs involved assured me that they would encourage participation in the study. To recruit rugby players, with the agreement and consent from the team support staff, I attended several practices with each rugby club to introduce the aim of the study and the subsequent procedures should these players want to participate. Participants and team support staff were then provided with my contact details by which testing sessions could be arranged for groups of players at a suitable time. These participants were all fluent in English, male, and aged 18 to 27 years.

In terms of the Rugby Concussed group, I included players that had received at least one formal diagnosis of a concussion, not within three months of testing, either by an independent medical health professional, or on-field medical staff (i.e., paramedics, or team doctor). For the Rugby Not Concussed group, I included players who had not formally been diagnosed with a concussion by relevant medical personnel previously. I gleaned this information from two self-report demographic and medical history questionnaires, with these adapted for the Control group and the two Rugby groups, respectively (see Appendices B and C).



Figure 1: A breakdown of the eligible participants recruited from each rugby club.

Note. U20 refers to an age group for players aged 18-20 years; UCT RFC = University of Cape Town Rugby Football Club; WPRA = Western Province Rugby Academy; VFC = Villagers Football Club; UCT IRL (University of Cape Town Internal Rugby League team).

I allocated participants to the Control group ( $n = 23$ ) that were, at the time of the study, involved in regular non-contact sport or exercise activity, had not played rugby at a university level, and who had no previously diagnosed concussion(s)<sup>2</sup>. Control group participants were recruited using convenience sampling. I made use of the Student Research Participation Programme (SRPP)<sup>3</sup> run by the UCT Department of Psychology and personal communication with the non-contact sport clubs at UCT.

**Exclusion criteria.** For all three groups, these exclusion criteria included: (a) being of the female sex, (b) scoring 21 or more on the Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Brown, 1996), and (c) prior or current diagnosed psychiatric

<sup>2</sup> Participants who had previously played rugby in school were not excluded from participation in the Control group given their lack of a recent exposure to possible rugby-related head injuries.

<sup>3</sup> The SRPP was introduced within the Department of Psychology to assist researchers with recruitment of undergraduate participants and also to encourage undergraduates to participate in research within the department. Undergraduate students are required to obtain a certain number of SRPP points per semester for each Psychology undergraduate course.

illnesses, learning disabilities, or neurological disease. In addition, (d) prior diagnosed concussion(s) was used as an exclusion criterion for the Control and Rugby Not Concussed group participants, only.

### **Materials.**

*Screening measures.* These measures were completed by all the participants as a source of demographic and psychological information, and a history of current alcohol use for exclusion and matching purposes.

*Demographic and medical history questionnaire.* To compile this questionnaire (Appendices B and C, for Control and Rugby groups, respectively) I used some aspects from the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT; Lovell, Collins, Podell, Powell, & Maroon, 2000) demographic form and incorporated additional questions that I felt relevant to obtaining a more detailed description of a participant's medical history and prior head injury complaints and diagnoses. I used this measure to obtain information about participants' age, height, weight, language proficiency, learning disabilities, psychiatric disorders, current and/or previous concussions, suspected undiagnosed concussions, and current and/or previous exposure to contact sport.

*Alcohol Use Disorders Identification Test (AUDIT).* The AUDIT (Saunders, Aasland, Babor, De La Fuente, & Grant, 1993) has been developed from a cross-national data set as a screening instrument for current and lifetime hazardous and harmful alcohol consumption. Importantly, Saunders et al. (1993) posit that this instrument is sensitive to identifying individuals with less severe drinking problems; a sample that in fact forms a larger proportion of the general population. Participants respond to each item on a 5-point Likert scale; higher scores indicate more damaging alcohol consumption. In this regard, Saunders et al. (1993) reported that among those diagnosed as having hazardous or harmful alcohol use, 92% had an AUDIT score of 8 or more, and 94% of those with non-hazardous consumption had a score of

less than 8. A score of 8 is the most commonly recommended cut-off score to indicate hazardous and harmful alcohol consumption (Babor, Higns-Biddle, Saunders, & Monteiro, 2001; Conigrave, Hall, & Saunders, 1995; Saunders et al., 1993). Given the high level of alcohol usage associated with rugby players in general (Quarrie et al., 1996; Sekulic, Bjelanovic, Pehar, Pelivan, & Zenic, 2014) and amongst South African rugby players and university students (Potgieter, et al., 2014), a cut-off score and high levels of alcohol use was not used as an exclusion criteria, but rather as a moderating variable that could be used to investigate any between-group differences.

The AUDIT has high validity and it has been identified as both sensitive and specific cross-culturally, for males and females (Blank, Connor, Gray & Tustin, 2015; Saunders et al., 1993; Seth et al., 2015; Skipsey, Burleson, & Kranzler, 1997). For studies that have used a cut-off score of more than or equal to a score of 8, sensitivity ranged from .61 to .96, and specificity ranged from .84 to .96 (Bohn, Babor, & Kranzler, 1995; Conigrave et al., 1995; Johnson, Lee, Vinson, & Seale, 2013; Lundin, Hallgren, Balliu, & Forsell, 2015; Seppä, Mäkelä, & Sillanaukee, 1995). The instrument has proven to be accurate in detecting alcohol dependence in university students (Blank et al., 2015; Fleming, Barry & MacDonald, 1991), has previously been used successfully in South Africa (Adams, Savahl, Isaacs, & Zeta Carels, 2013; Myer et al., 2008), and in concussion research (Straume-Naesheim, Andersen, Dvorak, & Bahr, 2005).

*Beck Depression Inventory-Second Edition (BDI-II)*. The BDI-II (Beck et al., 1996) is a 21-item self-report questionnaire that measures levels of depressive symptomatology in individuals aged 13 to 80 years. Each item has four possible responses; the participant is required to choose the response that best suits how s/he has felt for the past two weeks. Higher scores indicate greater levels of depression; scores greater than 20 (the cut-off in this

study) indicate that the person is dysphoric or moderately depressed (Beck et al., 1996; Dozois, Dobson, & Ahnberg, 1998).

This measure is highly internally consistent ( $\alpha = .93$ ; Dozois et al., 1998), and has good test-retest reliability ( $\alpha = .93$ ; Beck et al., 1996). The BDI-II has been used in sports-related concussion research in a South African sample of adolescents and young adults. Higher self-reported symptom scores for the BDI-II and other self-report measures have been shown to be consistent for South African compared to matched American athletes (Shuttleworth-Edwards, Whitefield-Alexander, Radloff, Taylor, & Lovell, 2009).

*Shipley-2.* The Shipley-2 (Shipley, Gruber, Martin, & Klein, 2009) is a brief measure of general intellectual functioning that tests two aspects of cognition: crystallised ability (*Vocabulary Test* subscale) and fluid cognitive ability (*Abstract Thinking Test* subscale or *Block Designs* subscale) in individuals aged 7 to 89 years. For the current study the Vocabulary (Shipley-V) and Abstraction (Shipley-A) scales were used. While each administration must be monitored, the test is self-administered (via computer) and thus can be administered to a group or individually (Shipley et al., 2009).

This measure has been shown to be highly internally consistent for adults (17 to 89 years;  $r = .92$ ), and correlates strongly with other tests of cognitive ability (Dodrill & Warner, 1988; Shipley et al., 2009; Wechsler, 2003). The Shipley-2 has been used in a recent concussion study with undergraduate, English-speaking students (Hill, Womble, & Rohling, 2015).

***Symptom and cognitive testing measures.*** I used the online Sports ImPACT Version 4.12 (ImPACT Applications Inc.), a computerised neuropsychological test battery developed specifically for the evaluation of sports concussion and baseline cognitive testing, to assess baseline cognitive functioning, and as a measure of self-report concussion symptoms. ImPACT has been normed for participants aged 10 to 59 years old.

*ImPACT post-concussion scale (PCS)*. This scale forms a part of the ImPACT test battery and includes questions related to the participants' concussion history, and about the current severity of 22 specific symptoms related to concussion. The participant is required to rate these via a 7-point Likert-type scale. This scale has been used widely by many sport and research teams to document concussion symptoms (Brown et al., 2014; Majerske et al., 2008; Meehan, Mannix, Stracciolini, Elbin, & Collins, 2013).

The ImPACT PCS has been shown to be internally consistent, with reliability ranging from .88 to .94 for samples of high school and college students. For concussed athletes, the internal consistency of the ImPACT PCS is high ( $r = .93$ ; Lovell et al., 2006). The ImPACT PCS has been used in rugby-related concussion research in South Africa, with players being compared successfully to matched American football players (Shuttleworth-Edwards et al., 2009).

*ImPACT neuropsychological test battery*. This part of the battery comprises of cognitive measures designed to assess a range of cognitive domains of functioning, particularly for memory and neurocognitive speed. Test outcomes comprise of five composite scores: Verbal Memory (evaluates attentional processes, learning, and memory within the verbal domain), Visual Memory (evaluates visual attention and scanning, learning, and memory), Visual Motor Processing Speed (evaluates visual processing, learning, and memory, and visual-motor response speed), Reaction Time (evaluates average response speed), and Impulse Control (this is not a core composite measure of cognitive ability, but can be used to determine test validity; see Appendix D for a more detailed description of the computerised tasks used to assess each composite score).

The ImPACT is both a sensitive and a specific instrument for identifying sports-related concussion. There is also good construct validity and convergent and divergent validity with standardised neuropsychological tests using college athletes (Broglio,

Macciochi, Ferrara, 2007b; Maerlender et al., 2013; Schatz, Pardini, Lovell, Collins, & Podell, 2006; Schatz & Sandel, 2013). Of particular usefulness, ImPACT has been designed for the purposes of baseline, and repeated testing and consists of an unlimited array of randomised test-forms with alternating stimuli (Broglio et al., 2006; Covassin et al., 2010; Henry et al., 2016b). It has thus been shown to be resilient to large practice effects (Lovell et al., 2003; Schatz, 2010) and is considered to be a reliable neurocognitive test battery at even 45 days post-baseline assessment (Nakayama, Covassin, Schatz, Nogle, & Kovan, 2014). Schatz and Ferris (2013) demonstrated that over a 4-week interval between test administrations, after participants had completed the ImPACT in a previous study in 2010 (Schatz, 2010), participants only demonstrated a significant improved performance on the Visual Motor Speed Composite. Schatz and Ferris (2013) report that this improved performance may be attributed to practice effects, specifically regarding a familiarity with the physical mechanics of how students interacted with the test of Visual Motor Speed. Nevertheless, it was shown that repeated exposure to the ImPACT over a month does not result in practice effects in memory performance or reaction time.

**Procedure.** Following recruitment, all participants ( $N = 80$ ) were contacted via e-mail to arrange for a suitable time to complete one testing session lasting approximately 1 hr 30 min. The testing session involved completing screening, and symptom and cognitive testing measures. The combined use of these measures is suitable for group testing, and thus the testing sessions were organised in a group format so as to maximise whatever free time the participants had available. Sessions for the Control and the Rugby groups were held separately, and no more than 10 participants per testing session was scheduled. After completion of the testing session, I excluded the data for those participants who met any of the exclusion criteria at that point. Thus, participants involved in the study were not informed directly that they had met the exclusion criteria and that their data would not be used. While it

may have been ideal to arrange two separate sessions for the screening and cognitive measures, given the challenges faced with recruitment and the risk of further attrition, we were reluctant to ask players to come for testing on two separate occasions.

**Data analysis.** I used IBM SPSS version 22 with a threshold significance level set at  $\alpha = .05$  for the analyses. Before the inferential analyses, I made the appropriate steps toward ensuring that the data met the assumptions for each of the parametric tests used. Unless otherwise stated, all of the required assumptions were upheld for each statistical analysis.

**Sample characteristics.** I compared the three study (Control, Rugby Not Concussed, Rugby Concussed) groups in terms of age, general intellectual functioning, years of formal education, levels of depression, and alcohol usage using a one-way ANOVA. By controlling for these variables more accurate inferences could be made regarding the relationship between concussion history and cognitive performance.

**Effect of concussion history on cognitive performance.** Here I performed two analyses. First, I conducted a one-way ANOVA using three groups (Control, Rugby Not Concussed, Rugby Concussed) to examine whether having a concussion history, or lack thereof, impacted on cognitive performance. For the significant outcomes in this analysis, I followed up the ANOVA with a post-hoc analyses using Tukey's HSD to investigate where (i.e., between which groups) the specific differences existed.

Second, I performed a Pearson's correlation analysis using the two rugby groups (Rugby Not Concussed and Rugby Concussed) to determine whether specific characteristics of concussive head injuries were related to the cognitive outcomes measured in this study. This analysis enabled me to assess, separately, the impact of 1) total concussions (number of previous diagnosed and suspected undiagnosed concussions), 2) previous periods of loss of consciousness (LOC), 3) a LOC composite score, and 4) a concussion composite score. I elaborate on these variables below.

The first two variables I used to correlate with the cognitive outcomes are relatively self-explanatory. The first involved using a continuous variable to account for the *total number of diagnosed concussions plus the number of suspected undiagnosed concussions* (i.e., head injuries while participating in rugby for which players reportedly experienced concussion-like symptoms, but were not formally diagnosed with a concussion). The second variable sought to determine whether a participant having sustained a period of *LOC* in any reported head injury due to participation in rugby impacted on the aforementioned cognitive outcomes.

I also created a *LOC composite score*, the third variable, as a continuous variable for each participant as an indication of their prior accumulated duration of LOC. This was necessary because some of the rugby players reported a period of LOC on more than one occasion, and because the precise duration was not uniform. The LOC composite score was computed by allocating points corresponding to the duration of LOC (shown in Table 1) and summing these points together for each participant; the larger the total duration of LOC across number of concussions was, the larger the score would be. So, for example if a participant had sustained two prior concussions, with one involving no LOC (0 points) and the other involving a LOC of 4 minutes (2 points), their LOC composite score would sum to 2 points (0 + 2).

For the final variable used in the correlation analysis I calculated a measure to indicate the overall severity of a rugby player's concussion history and to correlate this with the cognitive outcomes; a *concussion composite score* was determined for each participant. I computed this score by summing the total number of previous concussions (suspected undiagnosed and diagnosed) and the LOC composite score (as described above) for each participant. A higher concussion score would thus indicate a more severe concussion history, and thus potentially a more vulnerable neuropsychological profile.

Table 1  
*Points Calculation of the Duration of Loss of Consciousness  
 Composite Score*

| Duration of LOC          | Points |
|--------------------------|--------|
| No loss of consciousness | 0      |
| > 1 minute               | 1      |
| Unspecified duration     | 1      |
| 2 minutes – 5 minutes    | 2      |
| 6 minutes – 20 minutes   | 3      |

*Note.* LOC = loss of consciousness

**Ethical considerations.** This research was approved by the UCT Faculty of Health Sciences' Human Research Ethics Committee (HREC REF 010/2015) and the Department of Psychology Research Ethics Committee. Furthermore, this research was conducted in a manner that abides by the ethical guidelines and principles of the International Declaration of Helsinki.

**Informed consent.** At the start of each of the testing sessions, my research partner and I gave a brief presentation to groups of prospective participants. The purpose and nature of the study, requirements for participants, and the risks and benefits involved with their participation were covered in this presentation. We also informed prospective participants that their participation in the study was voluntary and that they had the opportunity to withdraw from the study at any point and without any penalty, should they wish to. I also informed each participant that this study formed part of a larger research project that sought to incorporate neuroimaging as well as post-injury assessments, but that this aspect would not form part of my research. After the presentation, I gave each participant the opportunity to read the informed consent document (see Appendices E and F) and to provide informed consent if they agreed to participate.

**Confidentiality.** We kept participants' identities confidential at all times during the study and this information will remain confidential in the event that the study is published. We labelled all data using participant numbers rather than names, so that they cannot be used to directly identify any particular individual. A separate, private record was kept to identify a participant by his number in the event that he needed to be contacted during the study or needs to be contacted in the future. Furthermore, we stored all data in locked filing cabinets in a secure venue (ACSENT laboratory in the Department of Psychology, UCT), and all electronic information was stored on a password protected computer. Only certain people directly involved in this study have access to these research records. Rugby players were informed that information pertaining to their history of concussion would not in any way have any bearing on future team selection.

**Referrals.** As a part of the screening process, demographic information related to a participant's recent mood was obtained using the demographic and medical history questionnaire. We referred one participant to the sports psychologist associated with the study so that counselling could be initiated, if so desired. This participant had recorded a BDI-II score of more than 21, indicating clinically significant concerns, and I thus informed him of his potential psychological vulnerability.

**Debriefing.** I provided each participant with a debriefing form after they had fully completed their role in the study (Appendix G). The debriefing form contains contact information for various parties involved in the study to address any potential questions or concerns about the participant.

**Risks and benefits.** The study had minimal risks. Participants may have experienced some slight discomfort in terms of having to complete a long assessment, which could have resulted in some fatigue and boredom effects. There are no other discomforts or risks associated with participation in this study. Participants received no direct benefits from their

participation in the study. Those who were undergraduate psychology students (part of the Control group) were however awarded 3 SRPP points, which counts towards their course requirements.

## Results

**Between-groups analysis: Participant characteristics.** Table 2 shows the results of the descriptive statistics and between-groups analyses for the demographic variables and for the screening measures for all participants in the baseline assessment phase. There were 23, 26, and 31 participants in the Control, Rugby Not Concussed, and Rugby Concussed groups, respectively.

There were significant differences between the groups in terms of age and intelligence, with Tukey's HSD showing that the Control group was significantly older and scored significantly higher on the general intelligence test than the Rugby Not Concussed group ( $p = .015$ ,  $p = .032$ , respectively). Although there was a significant between-groups difference for years of education, Tukey's HSD showed that the difference, which reflected that the Control group had more years of education than the Rugby Not Concussed group, was just above the significance threshold ( $p = 0.51$ ). Regarding age, the mean of the sample ( $N = 80$ ) was 20.59 years ( $SD = 2.08$ ). All participants in the study sample had a background of at least 11 years of formal education and with none of the participants reporting failing a grade at high school or at a tertiary level institution.

There were also significant differences between the groups in terms of years of playing rugby and alcohol use. Regarding years of playing rugby, as expected, the analysis showed that there is a significant difference between the three groups. Tukey's HSD results demonstrate that the Control group had participated in rugby for a significantly fewer number of years than did rugby players in both the Rugby Not Concussed ( $p = < .001$ ) and Rugby

Concussed groups ( $p = < .001$ ). There was no significant differences in years of playing rugby between the Rugby Not Concussed and Rugby Concussed group.

Regarding alcohol usage, again there was a significant between-group difference with Tukey's HSD showing that both the Rugby Not Concussed ( $p = .007$ ) and Rugby Concussed ( $p = .045$ ) groups had significantly higher levels of alcohol usage than the Control group.

Table 2

*Between-group Differences: Participant Characteristics per Group (Control vs. Rugby Not Concussed vs. Rugby Concussed)*

|                    | Control ( $n = 23$ ) |                 | Rugby Not Concussed ( $n = 26$ ) |              | Rugby Concussed ( $n = 31$ ) |                 | Test statistics |           |
|--------------------|----------------------|-----------------|----------------------------------|--------------|------------------------------|-----------------|-----------------|-----------|
|                    | Range                | $M (SD)$        | Range                            | $M (SD)$     | Range                        | $M (SD)$        | F               | $P$       |
| Age                | 18-25                | 21.48<br>(2.17) | 18-23                            | 19.85 (1.49) | 18-27                        | 20.55<br>(2.23) | 4.06            | .021*     |
| Years of Education | 12-17                | 14.35<br>(1.48) | 11-16                            | 13.12 (1.69) | 12-18                        | 14.06<br>(2.11) | 3.23            | .045*     |
| Shipley-Full IQ    | 34-57                | 47.48<br>(6.30) | 24-57                            | 42.15 (8.04) | 29-59                        | 44.81<br>(7.20) | 3.30            | .042*     |
| Rugby Years        | 0-5                  | 1.02<br>(1.67)  | 5-17                             | 10.77 (3.37) | 4-20                         | 11.32<br>(3.97) | 78.02           | < .001*** |
| BDI-II             | 0-14                 | 5.09<br>(3.69)  | 0-20                             | 6.92 (5.00)  | 0-19                         | 6.23<br>(4.85)  | .983            | .379      |
| AUDIT              | 0-15                 | 5.00<br>(4.01)  | 0-23                             | 9.81 (6.60)  | 0-24                         | 8.61<br>(5.14)  | 5.23            | .007**    |

*Note.* For each comparison here, degrees of freedom = (2, 77). AUDIT = Alcohol Use Disorder Identification Test; BDI-II = Beck Depression Inventory-II; IQ = intelligence quotient.

\* $p < 0.05$  \*\* $p < 0.01$  \*\*\* $p < 0.001$

Table 3 shows the concussion history for all three groups. The descriptive results show that among all of the rugby players in the sample ( $n = 57$ ), 31 participants (i.e., the Rugby Concussed group; 54%) reported previous formally diagnosed concussions, with an average time since last reported injury of 2.97 years ( $SD = 3.29$ ). Of these 31 participants, 10

also reported suspected undiagnosed concussions (32%). Furthermore, amongst those with formally diagnosed concussions making up the Rugby Concussed group, 22 participants (71%) reported having lost consciousness during at least one of their concussions. Of these 22 participants in the Rugby Concussed group reporting a loss of consciousness, duration of loss of consciousness ranged from a few seconds to 20 minutes with 54.55% reporting having lost consciousness at least once for less than a minute and 36.36% for between 1 and 5 minutes. Only one participant in the sample, from the Rugby Concussed group, reported a loss consciousness for longer than 5 minutes, (i.e., 20 minutes). One participant also from the Rugby Concussed group was unsure as to the duration of his loss of consciousness.

Table 3

*Participant Characteristics: Concussion History per Group (Control vs. Rugby Not Concussed vs. Rugby Concussed)*

|                                       | Control<br>( <i>n</i> = 23) |       | Rugby Not<br>Concussed<br>( <i>n</i> = 26) |       | Rugby Concussed<br>( <i>n</i> = 31) |       |
|---------------------------------------|-----------------------------|-------|--|-------|-------------------------------------|-------|
|                                       | No. of<br>players           | Range | No. of<br>players                          | Range | No. of<br>players                   | Range |
| Diagnosed concussions                 | 0 (0%)                      | 0     | 0 (0%)                                     | 0     | 31 (100%)                           | 1 – 4 |
| Suspected, undiagnosed<br>concussions | 6 (26%)                     | 0 – 2 | 8 (31%)                                    | 0 – 2 | 10 (32%)                            | 0 – 2 |
| Concussions with LOC                  | 0 (0%)                      | 0     | 0 (0%)                                     | 0     | 22 (71%)                            | 1-4   |
| LOC duration::                        |                             |       |  |       |                                     |       |
| < 1 min                               | 0 (0%)                      | 0     | 0 (0%)                                     | 0     | 12 (55%)                            | 1 – 3 |
| 1 – 5 min                             | 0 (0%)                      | 0     | 0 (0%)                                     | 0     | 8 (36%)                             | 1 – 3 |
| > 5 min                               | 0 (0%)                      | 0     | 0 (0%)                                     | 0     | 1 (5%)                              | 1     |
| Unreported                            | 0 (0%)                      | 0     | 0 (0%)                                     | 0     | 1 (5%)                              | 1     |

*Note.* LOC = loss of consciousness.

Percentage values are rounded to the nearest percentage.

The remainder of the rugby players that were recruited formed the Rugby Not Concussed group ( $n = 26$ ). As per the inclusion criteria for this group, none of these players had been previously formally diagnosed with a concussion. However, it was reported that eight players (31%) in the Rugby Not Concussed group had suspected undiagnosed concussions. None of the players in the Rugby Not Concussed group reported a LOC at any point, however.

Finally, while the Control group reported no formally diagnosed concussions, six participants in this group (26%) reported instances in which they suspected they may have experienced a concussion due to symptoms of memory loss or disorientation; despite their presentation, no diagnosis was made by a medical health professional. These symptoms are self-report and could not officially be verified.

**Between-groups analysis: Cognitive outcomes.** Table 4 shows descriptive statistics and between-groups outcomes for the cognitive test performances. There were no significant differences between the three groups on any of these variables, except Verbal Memory ( $p = .015$ ). The results of Tukey's HSD shows that the Control group performed significantly better than the Rugby Not Concussed group ( $p = .017$ ) on this variable. However, the control group also differed significantly from the Rugby Not Concussed group on a number of screening variables (i.e., age, and Shipley-Full IQ score and potentially years of education ( $p = 0.051$ )). Given those outcomes and the fact that the effect sizes are negligible for all results, this is likely a spurious result.

Table 4

*Cognitive Outcomes Scores per Group (Control vs. Rugby Not Concussed vs. Rugby Concussed)*

|               | Control                | Rugby Not Concussed    | Rugby Concussed        | Test statistics |          |          |
|---------------|------------------------|------------------------|------------------------|-----------------|----------|----------|
|               | ( <i>n</i> = 23)       | ( <i>n</i> = 26)       | ( <i>n</i> = 31)       | F               | <i>p</i> | $\eta^2$ |
|               | <i>M</i> ( <i>SD</i> ) | <i>M</i> ( <i>SD</i> ) | <i>M</i> ( <i>SD</i> ) |                 |          |          |
| Verbal Memory | 90.26 (7.43)           | 83.81 (9.09)           | 88.71 (7.50)           | 4.45            | .015*    | .104     |
| Visual Memory | 80.87 (9.51)           | 77.04 (16.88)          | 79.55 (12.60)          | .524            | .595     | .013     |
| VMS           | 38.54 (6.79)           | 37.00 (7.45)           | 39.40 (6.17)           | .896            | .412     | .023     |
| RT            | .57 (.07)              | .62 (.14)              | .58 (.08)              | 1.71            | .187     | .043     |

*Note.* For each comparison here, degrees of freedom = (2, 77). VMS = visual motor speed; RT = reaction time;  $\eta^2$  is the measure of effect size.

\**p* < 0.05

**Correlational analyses: Cognitive outcomes and indicators of a history of concussion severity.** These analyses were computed using participants in the two rugby groups (Rugby Not Concussed and Rugby Concussed) to determine whether specific indicators of a history of concussion severity were related to the cognitive outcomes discussed above (see Table 5). To do so, players' exposure to and severity of concussion was graded using 1) total concussions (number of previous diagnosed and suspected undiagnosed concussions), 2) the presence of a LOC, 3) a LOC composite score, and 4) a concussion composite score.

The results show no significant correlations between cognitive outcomes and indicators of prior concussion severity. However, a medium correlation existed between a measure of reaction time and total concussions ( $r = -.209$ ,  $p = .059$ ) and the concussion composite score ( $r = -.215$ ,  $p = .054$ ), both of which approached statistical significance. Nevertheless, these results suggest that having sustained a loss of consciousness from prior

head injuries (presence of LOC and LOC composite) and the overall severity of one's concussion history (concussion composite) has no significant long-term effect on cognitive outcome measures.

Table 5  
*Correlations Between Cognitive Outcomes and Indicators of a History of Concussion Severity for Rugby Players*

|                         |                     | Verbal<br>Memory | Visual<br>Memory | VMS  | RT    |
|-------------------------|---------------------|------------------|------------------|------|-------|
| Total<br>concussions    | Pearson correlation | .166             | .194             | .167 | -.209 |
|                         | <i>P</i>            | .109             | .074             | .107 | .059  |
| Presence of<br>LOC      | Pearson correlation | .194             | .025             | .075 | -.107 |
|                         | <i>P</i>            | .074             | .426             | .289 | .215  |
| LOC composite           | Pearson correlation | .048             | .107             | .132 | -.176 |
|                         | <i>P</i>            | .362             | .215             | .164 | .096  |
| Concussion<br>composite | Pearson correlation | .120             | .169             | .167 | -.215 |
|                         | <i>P</i>            | .188             | .105             | .107 | .054  |

*Note.* 'Total concussions' includes both suspected undiagnosed and diagnosed concussions; 'LOC composite' refers to the accumulated duration of loss of consciousness; 'Concussion composite' refers to overall concussion history severity; LOC = loss of consciousness; VMS = visual motor speed; RT = reaction time.

## Discussion

Despite some variation in the literature, the majority of research done retrospectively suggests that sports players presenting with single and multiple concussions do not demonstrate any detectable enduring symptoms or cognitive impairment (i.e., after three months). However, there is some, albeit limited evidence, to indicate prolonged neurological and neuropsychological changes, particularly in individuals presenting with multiple concussions with a slightly delayed recovery beyond two weeks post-concussion. Furthermore, research has debated the possibility that sports players exposed to these repeated injury conditions may potentially be vulnerable to long-term neuropsychological

impairment, even if only later on in life. Against this backdrop, Study 1 sought to investigate the relationship between having a history of concussion and its potential impact on current neuropsychological functioning. To this end, I investigated whether, at the beginning of a rugby season, rugby players presenting with a history of concussion differed on their cognitive performance to rugby players without a history of concussion and non-contact sportsmen.

In light of the literature and, therein, the well-known dose-response relationship between severity and neuropsychological outcomes post TBI, my hypothesis was that rugby players with a history of concussion would perform worse on neuropsychological outcomes than rugby players without a history of concussion. Furthermore, I expected to observe a correlation between the severity of a participant's concussion history (based on number of concussions, and the presence and duration of loss of consciousness) and performance on cognitive measures. I also used non-contact sport players as a control group, and I expected that these control subjects would show better scores on neuropsychological outcomes than rugby players.

**Summary of results.** Regarding the demographic variables, results showed that the Control group participants were significantly older and scored significantly higher on the intelligence scale than the rugby players in the Rugby Not Concussed group. These differences are likely due to recruitment limitations with there being a limited number of male psychology undergraduate students. As such we were not in a position to restrict the Control group to undergraduate students; athletes were mostly recruited from the non-contact sport clubs at UCT of which some were also completing post-graduate degrees. On the other hand, as Figure 1 shows, a large proportion (58%) of all rugby players recruited for Study 1 were playing in the under 20 age group (18-20 years). Although there were no significant differences between the two rugby groups on the demographic variables, descriptively, the

Rugby Not Concussed group was, on average, slightly younger, had a slightly lower number of years of education and IQ score, than the Rugby Concussed group, which could explain why the significant differences were limited to the Control and Rugby Not Concussed group.

In terms of the screening measures, results showed that the rugby players recruited in this study generally had significantly higher levels of alcohol usage than the Control group participants. The fact that there is no significant difference in alcohol usage between the two rugby groups (Rugby Concussed and Rugby Not Concussed) however, suggests that the high alcohol consumption is related to being part of the rugby sample generally, rather than exposure to concussion. Regarding participants' alcohol consumption, the developers of the AUDIT suggest that scores between 8 and 15 indicate hazardous drinking, scores between 16 and 19 indicate high level alcohol problems and scores of 20 or above indicate clinically significant alcohol dependence (Babor et al., 2001). To give an indication of the level of alcohol use amongst rugby players compared to the Control group, 58% of rugby players in this study had scores equal to or higher than 8 compared to 30% of the Control group. In addition, no participants in the Control group had a score of 16 or greater (high level alcohol problems), while 18% of rugby players recorded scores in that range, with 5 rugby players recording scores in the clinically significant alcohol dependence range.

These findings are not however atypical, with high alcohol consumption rates and binge drinking commonly associated with rugby (Lawson & Evans, 1992; Potgieter, et al., 2014; Quarrie, et al., 1996; Sekulic et al., 2014). Notably, these studies also refer to male participants of a similar age to the sample used for this study. One such study found that 78% of 348 rugby players that completed the AUDIT scored at or above 8 in a sample aged 14 to 39 years, with a mean age of 19.6 and 20.6 years for females and males, respectively (Quarrie, et al., 1996). In addition, a South African study, with a mean sample age of 21.9 years ( $SD = 1.2$ ), found that Varsity Cup rugby players at Stellenbosch University consumed

on average 5.4 drinks ( $SD = 6$ ), which are most often beer, after each rugby game (Potgieter, et al., 2014). Given the available evidence and the potential adverse effects of alcohol on physical and cognitive recovery and performance these findings indicate the need for further research and education with this population regarding the adverse effects of alcohol (Quarrie, et al., 1996; Potgieter, et al., 2014).

Regarding the cognitive outcomes assessed in Study 1, the results showed that having a history of concussion, had no association with cognitive performance. Although there is some previous literature to suggest that athletes with a history of concussion may perform worse on cognitive measures than athletes with no concussion history at baseline (Collins et al., 1999; Covassin, Elbin, Kontos, Larson, 2010; Iverson, Echemendia, La Marre, Brooks, & Gaetz, 2012; Moser, Schatz, & Jordan, 2005), the results in the current study were inconsistent with such findings. The results show that rugby players with previously diagnosed concussion(s), even when including players with up to 4 previous concussions, did not perform significantly different to the Rugby Not Concussed group, and both groups did not perform more poorly than the non-contact sport Control group on most measures. The only exception to the results was on the Verbal Memory task, with the Rugby Not Concussed group performing significantly worse than the Control group. However, given the significant differences in almost three demographic and screening variables between the same groups, and negligible effect sizes, this result is likely spurious.

Although no significant correlations existed between characteristics of concussive head injuries and cognitive outcomes measured in this study, it should be noted that for all measures used to quantify characteristics and severity of a player's concussion history (total concussions, presence of LOC, LOC composite score, and a concussion composite score) there was a trend indicating a moderate negative correlation with reaction time performance. Particularly, there existed medium correlations for 'total concussions' and the 'concussion

composite' score with reaction time. However, a lower reaction time performance is regarded as a good performance, indicating quicker reaction time speeds. These results are not however in line with research that has found a slower processing speed and reaction time with rugby players and other contact sportsmen, and with patients with mixed mechanism closed concussion injuries presenting with a history of one or more concussions (Collie et al., 2003; Eckner, Kutcher, Broglio, & Richardson, 2014; Gardner et al., 2010; Heitger et al., 2006; Lau et al., 2009; Mathias et al., 2004; McCauley et al., 2014; Rohling et al., 2011).

In sum, the hypothesis put forward for Study 1 was not confirmed, with results reflecting a seemingly absent relationship between the history of multiple concussions and poorer neuropsychological outcomes. These largely non-significant findings require consideration.

First, one must consider the dominant view to the relationship between concussion history and cognitive outcomes: that cognitive impairment post-concussion, even when presenting with a history of sports-related concussion(s), is not enduring for current sports players (Broglio & Puetz, 2008; Brooks et al., 2013; Cancelliere et al., 2014; Carroll et al., 2004; Guskiewicz et al., 2003; Henry et al., 2016b; Lau et al., 2011; Makdissi et al., 2010; Mannix et al., 2014; Marshall et al., 2015; McCrea et al., 2013; Nelson et al., 2016), despite some research investigating baseline neuropsychological performance using the ImPACT in samples of collegiate, elite, and adult sport players presenting with a history of multiple concussions (Broglio et al., 2006; Collie et al., 2006; Thornton et al., 2008) reporting otherwise.

The commonly reported dose-response relationship in TBI literature between injury severity and cognitive, behavioural, and psychosocial outcome (Anderson, Northam, Hendy, & Wrennall, 2001) would have one expect that a greater history of concussive injury, given some evidence for possible cumulative effects, would lead to poorer outcomes. Yet, research

findings largely report contrary findings. Some possible explanations for these findings are considered below.

First, it may be that having a history of concussion is associated with enduring or late onset concussive symptoms, but not cognitive difficulties. To support this, a recent study involving 5232 male adolescent football players, report that there was no differences in baseline cognitive scores using the ImPACT between athletes with a history of self-reported concussions (1 to 4 or more) and those with no prior concussion (Brooks et al., 2016). Nevertheless, athletes with more than three concussions reported more symptoms than athletes with no or one prior concussion. This evidence is further validated by research indicating that having a history of multiple concussions was associated with a higher symptom burden, with no differences found on cognitive outcomes using the ImPACT (Brooks et al., 2013; Mannix et al., 2014). Furthermore, Mannix et al. (2014), with a sample of 6075 student athletes, also report that other secondary factors, such as a mental health history, headache/migraine history, gender, developmental and/or learning problems could account for differences in reported concussive symptoms.

A second consideration could be in terms of time since injury and other characteristics associated with concussion severity not being accounted for. In the current study the average time since injury was 2.97 years with a range of 0 (not within three months of testing) to 13 years. Furthermore, in the studies cited above there is huge variation regarding the characteristics of the concussion group (e.g., time since the most recent concussion, but also time between concussions), with some studies not reporting on such characteristics. Perhaps carrying out individual investigations with the players with histories of multiple concussions with shorter and longer time since injury might yield varying results.

A third consideration is with regard to research design. What the predominant findings report is a general transient recovery of concussion symptoms and cognitive

difficulties found on more thorough neuropsychological assessments, with a minority of patients presenting beyond this typical phase of recovery. It is thus not surprising that, by using a group design, comparing concussed athletes to non-concussed athletes, and correlating increased exposure to concussion with neuropsychological outcomes, a typical favourable recovery is shown. Given the non-specific nature of concussive symptoms, delayed symptom report could be associated with a number of other secondary factors unrelated to participation in sport or a history of concussion. With group testing, while still relying on diagnostic issues with concussion, these secondary factors are negated, and research is consequently unable to adequately control for a variety of potential confounding factors.

A fourth consideration has to do with the sensitivity of the measures. Despite the fact that the ImPACT is a focused cognitive assessment for investigating cognitive domains and concussive symptoms mostly implicated with concussion, prior research using the ImPACT for baseline assessments has not found significant differences between participants with a history of concussion and those without (Brooks et al., 2013; Broglio et al., 2006; Collie et al., 2006; Mannix et al., 2014; Thornton, Cox, Whitfield, & Fouladi, 2008). Nevertheless, ImPACT has been shown to provide some evidence for delayed cognitive recovery, persisting beyond two weeks and up to a month post-concussion (Henry et al., 2016b; Lau et al., 2009; Lau et al., 2011).

In contrast, limited research (e.g., Heitger et al. (2006)), in which persisting, long-term neuropsychological difficulties have been reported, have used individualised neuropsychological and neurological tasks that are preferred in a clinical setting. Furthermore, given the relatively favourable prognosis of clinical and cognitive symptoms, more recent research has demonstrated the value of using objective markers of prolonged recovery that may be more sensitive to indications of 'mild' brain trauma (Baillargeon et al.,

2012; Ellis et al., 2015; Henry et al., 2011). Given the potential lack of sensitivity of computerised neuropsychological tests for assessing cognitive outcomes further out from injury, prospective research using a multifaceted assessment protocol (neuropsychological and neurological measures) to assess recovery in the acute and post-acute phase of concussion may prove more successful for identifying different recovery trajectories.

Finally, the lack of significant results could be a function of other study limitations.

**Limitations and future directions.** One limitation relates to the self-report measures used in the study, particularly for concussion history. A retrospective design lends itself to problems related to poor reliability associated with a self-reported concussion history and difficulty assessing concussion severity, accurately. In this regard, given the problematic nature of diagnosing sports-related concussion, the true incidence of concussion may have been underreported. Further, to add to the difficulty diagnosing and recognising concussion, particularly on the sideline of a sports match, the lack of awareness and knowledge about concussion and its diagnosis amongst players and potentially coaches as well as other secondary factors, such as motivation to continue playing and a lack of physician observation at games, could have further resulted in an underreporting of players' concussion history (Baker, Devitt, Green, & McCarthy, 2013; Belanger et al., 2010; Carroll et al., 2014; Cusimano et al., 2009; Echlin et al., 2012; Hollis et al., 2011; McCrory et al., 2013a; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards et al., 2008). A prospective and longitudinal investigation prior to an athlete's exposure to contact sport that involves a high risk of concussion may help to address this issue. However, with retrospective research, other than obtaining medical records, a reliance on self-report is inevitable.

Further, the study sample size was limited and uneven across groups, affecting power and homogeneity between groups in the analyses. In this regard, the control group was older,

and scored higher on the intelligence scale than the Rugby Not Concussed group. At the outset of the study, it was my intention to match Control group participants to the rugby playing participants on sex, age, general intellectual functioning, and years of formal education. However, this was made increasingly difficult given the limited number of younger male university students in the Department of Psychology (hence through the SRPP) who played a non-contact sport. In terms of the multiple non-contact playing sport clubs at UCT (i.e., squash club, rowing club, tennis club), there was a general lack of interest in participating in this study from prospective control participants associated with these clubs. This is likely to have been due to a lack of incentive for them; participants that were not studying psychology were not able to benefit from the SRPP point system by their participation (collecting SRPP points by participating in research during the semester is a requirement for all undergraduate psychology students, only).

Given these limitations and aforementioned considerations, future studies should aim to adopt a longitudinal and prospective design to enable researchers to better detect the overall impact of a history of concussion, as well as a player's repeated exposure to possible subconcussive head injuries over time, and in-season. I would further advocate for the incorporation of a repeated measures research design that aims to assess participants individually and on a continuous basis from their baseline level of performance; ideally, such research should target the assessment of baseline cognitive abilities amongst young sports players, prior to their accumulation of concussion and repeated head injuries.

**Conclusion.** There is a huge disparity in research investigating the effect of concussion on cognitive outcomes, particularly in sport, whereby diagnosing and recognising concussion and the appropriate clinical management thereof is especially problematic. Part of this problem has been the difficulty collaborating research outcomes, given the variable nature of methodologies used. In this regard, differences in participant characteristics and

operational definitions of concussion and cognitive domains, the severity of each concussion, the time since the last and each concussion, the nature of the comparison group, and the type of assessments used are just some of the moderating variables that can potentially impact on a lack of consistency in being able to assess the neuropsychological impact of concussion injuries. Given these difficulties, it is not surprising although a large body of literature suggests no real association between history of concussion and neuropsychological outcomes, there remains, however limited, evidence suggesting the contrary. Continued well designed studies investigating this relationship are needed.

Further, continued research of this nature would benefit from 1) there being a standard definition for sports-related concussion, 2) relying less on a concussion diagnosis, and rather focusing on monitoring continuously all contact sportsmen and women prospectively, using subtle and focused neuropsychological testing and neuroimaging, and an objective measure of a player's exposure to high-impact head collisions.

**Study 2: A novel method investigating the use of the xPatch (X2Biosystems Inc.), a means of head impact telemetry (HIT), to determine the number of subconcussive head injuries in a sample of university rugby players**

There are inherent difficulties associated with diagnosing and recognising concussion, with these being especially problematic in a contact sports environment where symptoms may not always be evident from the sideline, and/or may be underreported. Even though retrospective collection of data using self-report methods in particular may be limited in terms of reliability, prospective research could still be limited in terms of accuracy in diagnoses. In addition to the limitations with being able to accurately diagnose concussion on the sports field, research suggests that not all high-impact head collisions manifest in clinical

symptoms that would allow for a concussion diagnosis and for the appropriate guidelines to be put in place. Furthermore, should these symptoms become evident or recognisable, making a diagnosis may be delayed, potentially leaving contact sports players susceptible to repeat head injuries while in a state of vulnerability.

Further, some high-impact head injuries may give rise to what are referred to as subconcussive head injuries, that is, injuries that occur below the concussion threshold. In the absence of clinical symptoms, sports players may subsequently return to contact sport earlier than would be deemed fit if an actual concussion was diagnosed, leaving them potentially vulnerable to further injury. Players are thus likely to expose themselves to a risk of repeated head injuries on a potentially vulnerable brain in the absence of further medical management or rest from play. In light of these issues, many players could be at risk of a high exposure to repeated instances of concussion and/or subconcussive head injuries, which on the face of it, often warrants no further investigation.

Despite ongoing efforts for the development and introduction of precautionary measures for diagnosing and monitoring concussion in sport, these measures are taken after the fact, that is, after a player presents with concerning symptoms. By relying on subjective and post-hoc data, the potential impact and contribution of repeated subconcussive head injuries leaving a player vulnerable to more severe outcomes in the future, such as a subsequent concussion or the development of enduring and long-term neuropsychological or neurological decline, is left unexplored. In recognition of these flaws, recent research has set out to identify objective measures of high-impact head collisions (i.e., HIT) during a contact sports game, and how these can be used to recognise and predict player vulnerability (King et al., 2014; Talavage et al., 2014).

## **Aims and Hypothesis**

Study 2 aims to pilot the use of the xPatch, a means of HIT, to demonstrate the utility of this technology in better describing and understanding the frequency, magnitude, and direction of all forces sustained to the head in a university rugby sample. Given that this is a pilot study and exploratory in nature, no specific hypotheses are proposed.

## **Methods**

**Design and setting.** Study 2 is a cross-sectional and descriptive, pilot study. The nature of this research is exploratory. I collected data for Study 2 across the duration of four contact rugby matches that formed part of the UCT Internal Rugby League, a social contact rugby league for male rugby players. All matches were officiated at UCT's rugby fields, on the Upper Campus, UCT.

**Participants.** I used a sub-sample of 8 rugby players who had been playing for one of the rugby teams participating in the UCT Internal Rugby League – this sub-sample of players will hereon be referred to as the UCT IRL team. The UCT IRL team play in a social league, and thus the level of competition and intensity of play is not as would be expected at an amateur men's rugby club, or at a semi-professional or professional level of play.

The sample size is limited to 8 players, and thus not a full team (15 players). Not all rugby players who were approached in the UCT IRL team participating in this league could, at the beginning of the season, commit to playing each match that was specified for data collection; these players had prior commitments. Furthermore, I could not recruit members of other teams because rugby games in this league are played simultaneously, on different fields, and thus being in more than one place at the same time to administer, monitor, and collect the xPatches was not possible. Further, I only had xPatches for one full team; given that this was a pilot study and due to financial constraints we were only able to purchase a limited number of xPatches.

The UCT IRL team for this study comprised of 5 Forwards and 3 Backline players (see Appendix H for more explanation on these positional categories). The participants we recruited were 21 to 23 years of age. All of the players comprising the UCT IRL team previously participated in Study 1 of this research project, and are thus all male, English-speaking, and did not meet any of the exclusion criteria outlined in Study 1.

### **Materials.**

*Head impact telemetry (HIT).* The xPatch, provided by X2Biosystems Inc., is a device containing a low-power, high-g accelerometer chip that fits securely onto an adhesive plaster. The device and plaster are small enough to fit behind the ear. We used this device to measure continuously, acceleration and other linear and rotational forces to the head during rugby games. The resolution by which these forces can be measured has a range of up to 1000 Hz. The accelerometer can capture an acceleration and rotational time history for 3-axes of acceleration and 3-axes of velocity.

Peer-reviewed publications using an American football sample of players, including college level players, have previously trialled the use of the xPatch; this research has been conducted with and without the use of helmets (Reynolds et al., 2016; Swartz et al., 2015). Similar technology, also produced by X2Biosystems Inc., has been used for head injury research with a rugby (King et al., 2014) and an American football sample (Camarillo, Shull, Mattson, Shultz, & Garza, 2013). For these studies, rather than the xPatch, a mouth guard instrumented with an accelerometer were fitted for each player. More recently though, the xPatch has been pioneered with rugby players with Saracens Rugby Club based in the United Kingdom (BBC Sport, 2015) and with Randwick Rugby Club in Sydney, Australia (Guinness, 2015), and with rugby league players with the Central Butcher Boys based in Newcastle, Australia (Callinan, 2016). The use of this technology has come with the increasing concern, especially amongst professional sport clubs, for the potential deleterious long-term

neuropsychological and neurological effect of high-impact head collisions in the absence of obvious clinical symptoms (BBC Sport, 2015; Rossingh & Kitamura, 2015).

**Procedure.**

**Recruitment.** I recruited the UCT IRL team ( $n = 8$ ) using convenience sampling techniques. The recruitment of these players formed a part of the recruitment phase for Study 1. I approached one of the rugby teams participating in the UCT Internal Rugby League to participate in Study 1, but I also extended these players the opportunity to pilot the xPatch during four contact rugby games; an additional presentation of the Study 2 protocol was given to this team. Members of the entire UCT IRL team thus comprised a proportion of the two Rugby groups of Study 1.

**Protocol for data collection.** I randomly assigned an xPatch to each of the 8 rugby players, with each xPatch being labelled with a number from one to eight. Players wore their same xPatch for each of the four rugby matches. I collected the xPatches between rugby matches so that the data from each device could be immediately downloaded after each match, so that the devices could be charged, and also to avoid any potential damage and/or loss and any accidental and/or spurious data being captured by the device. One hour prior to the match starting, I personally attached the assigned xPatch to each participant. The device was placed, using an adhesive plaster (specifically made and supplied by X2Biosystems Inc.) directly on the skin, behind either the right or left ear the specific ear was noted for the analyses.

**Data analysis.** The xPatch recorded all data regarding the location, and acceleration associated with each collision event in real time and from a 360 degree perspective, during the rugby matches. I only analysed head impacts recorded above the minimal data acquisition

limit of 10g, used for this research<sup>4</sup>. A 10g acquisition limit has previously been used in a research study with American football and rugby players using HIT designed and manufactured by X2Biosystems Inc. (King et al., 2014; Reynolds et al., 2016). Following each match, I uploaded this data to an online ‘cloud’ data base, the X2Biosystems Inc. Injury Monitoring System (IMS), from which the raw data could be retrieved and analysed.

In terms of the analysis of such data, basic descriptive statistics including the number of impacts sustained, the resultant linear and rotational acceleration forces per impact, and the location of each of these impacts to the head were obtained (see Figure 2 for how the data are represented on the IMS). I also calculated each player’s total linear acceleration and rotational acceleration force which they had sustained to the head for the duration of the study; this is known as the total impact frequency burden (IFB). These descriptive statistics were evaluated per player and position. I conducted t-tests to assess differences between player positions (e.g. Forwards and Backline players) regarding their total exposure to head impacts. I also used a one-way ANOVA to assess differences in the severity of impacts based on impact location. Data analysis was done using IBM SPSS version 22 with a threshold significance level set at  $\alpha = .05$ .

In addition to this, all recorded impacts were assessed in terms of an injury tolerance level for a concussion, defined by previously published injury risk tolerance levels (Broglia et al., 2010; Broglia et al., 2011; Guskiewicz et al., 2007) for linear acceleration ( $>95g$ ) and rotational acceleration ( $>5500 \text{ rad/s}^2$ ) forces. Impacts were also evaluated by injury severity

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<sup>4</sup> A data acquisition limit is what investigators use as a minimal limit for which recorded data using head impact telemetry is analysed, and is a necessary limit so as to exclude ‘noise’ that is not related to head collisions. As recommended by X2 Biosystems, the majority of research reports using a data acquisition limit of 10g (Crisco et al., 2012; Daniel et al., 2012; Hanlon & Bir, 2012; King et al., 2014; Mihalik et al., 2012; Reynolds et al., 2016).

levels for linear acceleration (mild, <66g; moderate, 66-106g; severe, >106g) and rotational acceleration (mild, <4600 rad/s<sup>2</sup>; moderate, 4600-7900 rad/s<sup>2</sup>; severe, >7900 rad/s<sup>2</sup>) forces as recommended by previous literature (Harpham et al., 2014; Ocwieja et al., 2012; Zhang et al., 2004).



*Figure 2:* Example of the data display provided by the IMS, with data including, but not limited to, xPatch player assignment and charge levels, and number, location, and severity of impacts recorded per xPatch (X2Biosystems Inc., 2013).

**Ethical considerations.** The ethical approval by the UCT Faculty of Health Sciences' Human Research Ethics Committee (HREC REF 010/2015) and the Department of Psychology Research Ethics Committee referred to in Study 1, incorporates Study 2. The participants in Study 2, by their inclusion in Study 1, underwent the same informed consent

and debriefing procedures referred to in the Ethical Considerations section of Study 1. However, the UCT IRL team were given an additional presentation regarding the nature of Study 2. In this presentation, I explained that the xPatch may be slightly uncomfortable for some of the players. This is in response to the players involved in the scrum (notably the Forwards players) from Saracens Rugby Club in the United Kingdom reporting that they were not entirely comfortable with the patch and were concerned of it being rubbed off during play (Cleary, 2015). However, I gave each participant the opportunity to trial the xPatch for a non-contact rugby practice session prior to agreeing to participate in Study 2.

## Results

**Linear and rotational acceleration forces.** Table 6 and 7 shows the number of impacts sustained, the resultant linear and rotational acceleration forces per impact, and the total IFB for rotational and linear acceleration forces for Forwards and Backline players.

A total of 546 impacts to the head were recorded for all players at a force of acceleration equal to or greater than 10g for linear acceleration (range: 10.00g – 141.16g) over the duration of this study. An average of 21.44 impacts to the head were sustained per player, per match. The data revealed that the majority of impacts recorded above 10g were at the ‘mild’ end of the injury severity level, with a mean linear acceleration of 20.87g ( $SD = 14.18$ ) and a mean rotational acceleration of 3158.23 rad/s<sup>2</sup> ( $SD = 2729.76$ ). However, it should be noted that for Forwards and Backline players there were a greater number of impacts involving ‘moderate’ and ‘severe’ rotational accelerations (113 ‘moderate’ and ‘severe’ impacts; 90 impacts for Forwards and 13 impacts for Backline players) than for resultant linear accelerations (9 ‘moderate’ and ‘severe’ impacts; 7 impacts for Forwards and 2 impacts for Backline players).

The number and force of linear and rotational acceleration impacts to the head varied according to different player positions (see Tables 6 and 7). It was evident that Forwards

players had sustained a significantly greater number of impacts than the Backline players per game,  $t(7) = 2.76, p = .033$ . However, although the Backline players sustained fewer impacts than the Forwards, there was no significant difference in the mean linear ( $p = .277$ ) and rotational acceleration forces ( $p = .897$ ) per impact experienced by Forwards and Backline players. Nevertheless, it is thus not surprising that the Backline players recorded, on average, lower total frequency impact burden for linear ( $p = .055$ ) and rotational acceleration forces ( $p = .060$ ) than the Forwards, with these differences approaching a significant level.

Of the Forwards players that were recruited, the Hooker had sustained the greatest number of impacts per match, and had recorded the highest total linear acceleration IFB, as well as the highest total rotational acceleration IFB. Despite the Hooker having incurred more impacts and a greater total IFB, the Eighth Man had sustained the highest mean linear and rotational accelerations per impact. Furthermore, only one player (Eighth Man) recorded a mean acceleration force per impact that registers above a 'mild' injury severity – this was for a mean rotational acceleration force in the 'moderate' injury severity level. The Eighth Man also recorded the greatest number of impacts in the 'moderate' and 'severe' injury level for rotational acceleration.

Of the 3 Backline players that were recruited, the Outside Center sustained the most impacts per game, with the Wing having sustained the least impacts per game. The highest mean linear acceleration sustained among the Backline players was 21.29g by the inside centre, with the outside centre sustaining the highest mean rotational acceleration of 3485.17 rad/s<sup>2</sup>. No Backline player recorded a mean linear or rotational acceleration force above a 'mild' injury severity level.

Table 6  
*Resultant Linear and Rotational Acceleration Forces, and the Total Impact Frequency Burden (IFB) for Forwards and Backline Players*

| Player | Position       | Games played | Impacts/<br>game | Resultant PLA          |              |                 |               | Resultant PRA |  |                                  |                                      |                                  |                                 |
|--------|----------------|--------------|------------------|------------------------|--------------|-----------------|---------------|---------------|--|----------------------------------|--------------------------------------|----------------------------------|---------------------------------|
|        |                |              |                  | Mean force (g)<br>(SD) | Impacts <66g | Impacts 66-106g | Impacts >106g | Total IFB (g) | Mean force (rad/s <sup>2</sup> )<br>(SD) | Impacts <4600 rad/s <sup>2</sup> | Impacts 4600-7900 rad/s <sup>2</sup> | Impacts >7900 rad/s <sup>2</sup> | Total IFB (rad/s <sup>2</sup> ) |
| 1209   | T-H Prop       | 3            | 20.67            | 22.23<br>(13.66)       | 61           | 1               | 0             | 1378.27       | 3878.77<br>(3209.41)                     | 42                               | 13                                   | 7                                | 240483.57                       |
| 1401   | Hooker         | 4            | 40.75            | 19.17<br>(9.82)        | 162          | 2               | 0             | 3123.97       | 2621.96<br>(2006.25)                     | 144                              | 13                                   | 6                                | 427379.10                       |
| 1317   | Lock           | 2            | 32               | 19.81<br>(17.42)       | 63           | 0               | 1             | 1268.16       | 2397.33<br>(2121.83)                     | 58                               | 5                                    | 1                                | 153428.90                       |
| 1318   | Lock           | 3            | 31.33            | 21.53<br>(17.07)       | 92           | 1               | 1             | 2024.09       | 2811.54<br>(2257.02)                     | 77                               | 13                                   | 4                                | 264284.30                       |
| 1315   | Eighth Man     | 4            | 16               | 25.01<br>(15.74)       | 63           | 1               | 0             | 1600.82       | 4767.52<br>(3861.26)                     | 36                               | 15                                   | 13                               | 305121.50                       |
| 1319   | Inside Center  | 2            | 12               | 21.29<br>(20.82)       | 22           | 2               | 0             | 510.81        | 3305.60<br>(2757.40)                     | 18                               | 3                                    | 3                                | 79334.43                        |
| 1210   | Outside Center | 4            | 15.75            | 20.53<br>(11.55)       | 63           | 0               | 0             | 1293.56       | 3485.17<br>(2820.62)                     | 48                               | 8                                    | 7                                | 219565.40                       |
| 1207   | Wing           | 4            | 3                | 16.65<br>(7.14)        | 12           | 0               | 0             | 199.81        | 2899.73<br>(3228.91)                     | 10                               | 1                                    | 1                                | 34796.79                        |

*Note.* IFB = impact frequency burden; T-H = tight-head; PLA = Peak Linear Acceleration at the centre of gravity of the head; PRA = Peak Rotational Acceleration at the centre of gravity of the head.

Mean resultant linear acceleration and injury severity levels measured in g's; mean resultant rotational acceleration and injury severity levels measured in rad/s<sup>2</sup>.

Injury severity levels: <66 (mild); 66-106 (moderate); >106 (severe).

<4600 (mild); 4600-7900 (moderate); >7900 (severe).

Table 7

*Average Resultant Linear and Rotational Acceleration Forces, and the Total Impact Frequency Burden (IFB) for Forwards and Backline Players*

|          | Games played | Impacts/game | Resultant PLA       |              |                 |               | Resultant PRA |                                       |                                  |                                      |                                  |                                 |
|----------|--------------|--------------|---------------------|--------------|-----------------|---------------|---------------|---------------------------------------|----------------------------------|--------------------------------------|----------------------------------|---------------------------------|
|          |              |              | Mean force (g) (SD) | Impacts <66g | Impacts 66-106g | Impacts >106g | Total IFB (g) | Mean force (rad/s <sup>2</sup> ) (SD) | Impacts <4600 rad/s <sup>2</sup> | Impacts 4600-7900 rad/s <sup>2</sup> | Impacts >7900 rad/s <sup>2</sup> | Total IFB (rad/s <sup>2</sup> ) |
| Forwards | 3.20         | 28.15        | 21.55               | 88.2         | 1               | 0.4           | 1879.06       | 3295.42                               | 71.4                             | 11.8                                 | 6.2                              | 278139.50                       |
| Backline | 3.33         | 10.25        | 19.49               | 32.33        | .67             | 0             | 668.06        | 3230.17                               | 25.33                            | 4                                    | 3.67                             | 111232.20                       |

*Note.* IFB = impact frequency burden; T-H = tight-head; PLA = Peak Linear Acceleration at the centre of gravity of the head; PRA = Peak Rotational Acceleration at the centre of gravity of the head.

Mean resultant linear acceleration and injury severity levels measured in g's; mean resultant rotational acceleration and injury severity levels measured in rad/s<sup>2</sup>.

Injury severity levels: <66 (mild); 66-106 (moderate); >106 (severe).

<4600 (mild); 4600-7900 (moderate); >7900 (severe).

**Location of impacts to the cranium.** Tables 8 and 9 show the number of impacts to different areas of the head for Forwards and Backline players. On average, the highest number of impacts were recorded to the side of the head ( $M = 22.75$ ), with the least number of impacts being recorded to the top of the head ( $M = 3.00$ ). Also, a comparison of linear acceleration impact forces to different areas of the cranium (impact location) showed significant differences  $F(3, 542) = 3.083$ ,  $p = .027$ . More specifically, the impacts to the back of the head resulted in a significantly lower force of linear acceleration than impacts to the front of the head ( $p = .013$ ). Regarding rotational accelerations, there was also a significant difference in the force of impact based on the impact location,  $F(3, 542) = 3.498$ ,  $p = .015$ , again with impacts to the back of the head resulting in a significantly lower force of rotational acceleration than for impacts to the front of the head ( $p = .014$ ).

Table 8  
*Head Impacts (> 10g) by Location of Impact*

| Player | Position   | Games played | Front    |               | Side     |               | Back     |               | Top      |               |
|--------|------------|--------------|----------|---------------|----------|---------------|----------|---------------|----------|---------------|
|        |            |              | <i>n</i> | <i>n/game</i> | <i>n</i> | <i>n/game</i> | <i>n</i> | <i>n/game</i> | <i>n</i> | <i>n/game</i> |
| 1209   | T-H Prop   | 3            | 26       | 8.67          | 20       | 6.67          | 15       | 5             | 1        | 0.33          |
| 1401   | Hooker     | 4            | 45       | 11.25         | 62       | 15.5          | 50       | 12.5          | 6        | 1.5           |
| 1317   | Lock       | 2            | 4        | 2             | 27       | 13.5          | 30       | 15            | 3        | 1.5           |
| 1318   | Lock       | 3            | 33       | 11            | 24       | 8             | 28       | 9.33          | 9        | 3             |
| 1315   | Eighth Man | 4            | 30       | 7.5           | 18       | 4.5           | 15       | 3.75          | 1        | 0.25          |
| 1319   | Centre     | 2            | 12       | 6             | 9        | 4.5           | 2        | 1             | 1        | 0.5           |
| 1210   | Centre     | 4            | 25       | 6.25          | 18       | 4.5           | 17       | 4.25          | 3        | 0.75          |
| 1207   | Wing       | 4            | 6        | 1.5           | 4        | 1             | 2        | 0.5           | 0        | 0             |

*Note.* T-H = tight-head

Table 9  
*Average Head Impacts (> 10g) by Location of Impact for Forwards and Backline Players*

|          | Games played | Front    |               | Side     |               | Back     |               | Top      |               |
|----------|--------------|----------|---------------|----------|---------------|----------|---------------|----------|---------------|
|          |              | <i>n</i> | <i>n/game</i> | <i>N</i> | <i>n/game</i> | <i>n</i> | <i>n/game</i> | <i>n</i> | <i>n/game</i> |
| Forwards | 3.20         | 27.60    | 8.63          | 30.20    | 9.44          | 27.60    | 8.63          | 4        | 1.25          |
| Backline | 3.33         | 14.33    | 4.30          | 10.33    | 3.10          | 7        | 2.10          | 1.33     | 0.40          |

Table 10 shows the spread of impacts according to head injury tolerance and impact severity limits. Of the 546 impacts that were recorded for Study 2, there were only 2 impacts that were recorded above the linear injury risk limit and 84 impacts above the rotational injury risk limit for a concussion occurring. Also, the majority of impacts that were recorded for the UCT IRL team players registered in the ‘mild’ impact severity limit for linear (98.53%) and rotational (79.30%) acceleration forces.

Table 10  
*Head Impacts (> 10g) and Resultant Linear and Rotational Acceleration Forces by Injury Tolerance Level and Impact Severity Limits*

|  | <i>n</i> | %     | Mean per player |
|--|----------|-------|-----------------|
| <b>Injury tolerance level</b>            |          |       |                 |
| >95g                                     | 2        | 0.37  | 0.25            |
| >5500 rad/s <sup>2</sup>                 | 84       | 15.38 | 10.50           |
| <b>Resultant PLA (g)</b>                 |          |       |                 |
| <66 (mild)                               | 538      | 98.53 | 67.25           |
| 66-106 (moderate)                        | 7        | 1.28  | 0.88            |
| >106 (severe)                            | 2        | 0.37  | 0.25            |
| <b>Resultant PRA (rad/s<sup>2</sup>)</b> |          |       |                 |
| <4600 (mild)                             | 433      | 79.30 | 54.13           |
| 4600-7900 (moderate)                     | 71       | 13.00 | 8.88            |
| >7900 (severe)                           | 42       | 7.69  | 5.25            |

*Note.* PLA = Peak Linear Acceleration at the centre of gravity of the head; PRA = Peak Rotational Acceleration at the centre of gravity of the head. An injury tolerance level for linear and rotational acceleration impacts has been defined by previous research which have attempted to identify the force at which concussion injuries are more likely.

## Discussion

The aim of this exploratory, pilot research study was to investigate a novel, accurate and efficient means for monitoring player exposure to high-impact head collisions. Study 2 involved the use of the xPatch to objectively monitor, in real time, head collisions above a linear acceleration data acquisition limit of 10g of force in a rugby contact match. Further, with the xPatch, I was able to record a number of head collisions and to provide data regarding the linear and rotational acceleration force of impacts above a 10g threshold, and the location of these impacts to the head during a contact rugby match.

**Summary of results.** Overall, 546 impacts to the head above 10g were recorded among a sub-sample of 8 rugby players, with a mean of 21.44 impacts per player, per match. The majority of recorded impacts involved rotational, rather than linear, acceleration forces to the head. Particularly, compared to linear accelerations, rotational acceleration forces were especially more frequent in the moderate and severe injury severity range. Regarding position, Forwards players when compared to Backline players, sustained the most impacts on average, per game. Thus, Forwards players inevitably sustained a greater cumulative impact frequency burden. However, the mean linear and rotational acceleration force per impact between Forwards and Backline players, was relatively similar. Furthermore, the most number of impacts were recorded to the side, and the least number of impacts to the top of the head, with the evidence showing that linear and rotational acceleration impact forces to the front of the head were significantly greater than these forces when sustained to the back of the head. I discuss each finding below.

**Mean number of head impacts.** Regarding the mean number of impacts per player, per match to the head, the mean of 21.44 impacts per player, per match, is somewhat lower than the mean of 77 impacts to the head per game recorded by King et al. (2014) with a sample of premier amateur adult rugby players. However, the sample utilised for Study 2

participated in the UCT internal rugby league, a social contact rugby league, which is considered to be of a lesser intensity when compared to rugby players participating in a premier amateur league in New Zealand. The equivalent of the team reported on by King et al. (2014) in South Africa is likely to be the UCT 1<sup>st</sup> Team that compete in the premier rugby league in the Western Cape (Western Province Super League A). Nevertheless, the impacts recorded in Study 2 are more frequent than impacts recorded per session (practice and game) with American collegiate football players, with a mean of 9 to 13 impacts (Crisco et al., 2010; Schnebel, Gwin, Anderson, & Gatlin, 2007). Given that the different positional categories in American football complete very different roles and have a consequent varying exposure to head collisions to players in rugby positions, this could account for lower impact rates, on average. In light of this information, Crisco et al. (2010) reported that the number of head impacts per game ranged from 7.3 for wide receivers to 29.8 for defensive liners. This research also utilised a different HIT system (devised by Simbex and marketed by Riddell Inc.) that was fitted into helmets worn by each player.

*Rotational vs. linear acceleration forces.* The higher number of rotational as compared to linear impacts above the injury threshold recorded in this study is consistent with results reported for a study with rugby players from New Zealand (King et al., 2014). Furthermore, these results are also in line with relative higher rotational compared to linear acceleration forces reported with collegiate and professional American football players (Pellman et al., 2003; Rowson et al., 2012). It is expected that possible tackle techniques and nature of different head collisions in different types of contact scenarios may explain the variations on these outcomes. Nevertheless, research investigating a vulnerability of contact sports players to high-impact rotational acceleration forces should be further investigated. This is especially pertinent given the diffuse nature of these injuries, and the implication of diffuse brain injury with neurological changes likely associated with concussion symptoms

and neuropsychological impairment (Henry et al., 2011; McKee et al., 2014; Meaney et al., 1995; Meehan & Bachur, 2009; Meier et al., 2016; Orr et al., 2016).

Regarding the impact of these forces applied, in the current study, the mean linear acceleration (20.87g) and mean rotational acceleration value per impact (3158.23 rad/s<sup>2</sup>) were similar, albeit slightly lower to the mean values reported for amateur adult rugby players (22g and 3990 rad/s<sup>2</sup>, respectively; King et al., 2014). In this regard, the spread of the force of registered impacts was negatively skewed, indicating that the majority of impacts were of a 'mild' severity (<66g and <4600 rad/s<sup>2</sup>), and thus unlikely to present with concussion-like symptoms (McCrea & Powell, 2012; Patton, McIntosh, Kleiven, & Fréchède, 2012; Pellman et al., 2003; Rowson et al., 2012). McCrea and Powell (2012) report that in an ongoing study using HIT with American college football players, an average magnitude of 95g for concussion impacts was observed, with a range of 60 – 120g. Furthermore, regarding linear acceleration impact forces, subconcussive head injuries have been reported to average between 26g to 57g, with reported concussions averaging 98g to 104g. Rotational acceleration forces implicated with subconcussive and concussive head injuries are recorded between 1230 and 4028 rad/s<sup>2</sup>, and 4726 and 6432 rad/s<sup>2</sup>, respectively (Pellman et al., 2003; Rowson et al., 2012).

Regarding linear acceleration forces recorded for Study 2, there were only 2 impacts above the linear injury risk limit for concussion (>95g). Furthermore, the mean linear acceleration force recorded in the current study (20.87g) is similar to mean values for high school (21 to 26g; Broglio et al., 2010; Eckner et al., 2011; Urban et al., 2013), and some collegiate (18 to 27g; Beckwith et al., 2013; Rowson et al., 2009) American football players, but somewhat lower than the mean linear acceleration reported with professional American football players (60g; Pellman et al., 2003). Consequently, a lower mean acceleration impact force can potentially be ascribed to increased professionalism, particularly given the expected

low intensity of contact experienced by the UCT IRL team. Nevertheless, given the nature of these different sporting codes, particularly given comparisons between non-helmeted and helmeted sports, these comparisons should be interpreted cautiously. In addition, more research is required to investigate head injury thresholds that are appropriate for non-helmeted contact sports.

In the current study 84 impacts were recorded above the rotational acceleration injury risk limit, with a far greater number of head impacts resulting in ‘moderate’ and ‘severe’ rotational acceleration forces, when compared to linear acceleration forces. Notably, 42 impacts were recorded in the ‘severe’ category of a rotational acceleration force, that being above  $7900 \text{ rad/s}^2$ . This is particularly concerning given impacts above this level have been known to produce concussion-like symptoms (McCrea & Powell, 2012; Patton et al., 2012; Rowson et al., 2012). Compared to a rotational acceleration mean of  $3158.23 \text{ rad/s}^2$  in the current study, higher mean rotational acceleration forces for real-life reconstructions of head collisions during matches in non-concussed ( $4043\text{-}4446 \text{ rad/s}^2$ ) and concussed ( $4870\text{-}7173 \text{ rad/s}^2$ ) players were also reported in professional American football (Pellman et al., 2003). However, the mean rotational acceleration forces reported in my study and by King et al. (2014) are greater than those reported with American high school and collegiate football players (Beckwith et al., 2013; Broglio et al., 2010; Rowson et al., 2009; Urban et al., 2013). In light of this information, rugby players may be more vulnerable to sustaining high-impact rotational acceleration injuries that are responsible for focal and diffuse neuronal injury, rather than linear impacts.

Following from the reported head impact data from the current study and the reported literature, it is evident that players do sustain multiple high-impact head collisions above what would be expected to result in a concussion. These have been shown to be especially prevalent for high-impact rotational acceleration forces, that are most implicated in focal and

diffuse neuronal injury, rather than linear impacts. However, McCrea and Powell (2012) reported that while 6 of 9 players who sustained a concussion recorded a concussive impact greater than 95g, the concussive impacts for these 6 players account for less than 1% of the 27000 non-concussive impacts that were recorded above 95g. This evidence is in keeping with the known concern for the potential cumulative neuropsychological and neurological impact of multiple subconcussive head injuries, whereby players, despite sustaining high-impact head injuries capable of producing concussion-like symptoms, do not present clinically as such.

Nevertheless, although injury tolerance levels used in this study (95g and 5500 rad/s<sup>2</sup>) have previously been investigated by other sports and concussion related research, these have predominantly been limited to helmeted sports. Higher injury threshold limits have in fact been reported for concussion using video analysis in AFL and rugby players (103g and 8022 rad/s<sup>2</sup>; Baugh et al., 2012). Also using video analysis with AFL, rugby league and rugby players, a tolerance level of 4500 rad/s<sup>2</sup> was proposed for the limit at which a loss of consciousness may occur (Patton et al., 2012). Given this variation, owing much to differences in protective gear used for different sports, differences in demographic and medical history variables, and differences in prior exposure to contact sport, future research should target more specific samples to publish accurate and relevant injury risk tolerance levels.

***Forward vs. Backline players.*** As per the current study, King et al. (2014) also reported differences in the number of head impacts between Forwards and Backline players, but similarly noted no significant differences in the mean linear and rotational acceleration force experienced between these two positional categories. These results are not surprising.

In rugby, contact situations involving scrums and mauls are mostly exclusively for Forwards players. In addition, one of the primary roles of Forwards are to involve themselves

in rucks so as to protect their own ball and compete for the opposition ball; a ruck is a phase of play in rugby whereby more than two players from opposing sides are competing for the ball. These phases of play involve the competition for ball on the ground, without using ones hands, thus rendering players more vulnerable to head collisions under these circumstances. Using video analysis to verify head collisions, King et al. (2014) reported multiple impacts at a ruck with multiple players involved in that ruck, not just the ball carrier. Forwards players are also often repeatedly utilised as a runner to carry the ball into contact when there is a lack of space on the field and would thus be expected to be involved in more tackle scenarios as a result of this. On the other hand, Backline players are often involved in open, running play, and are thus less vulnerable to sustaining head collisions.

Despite differences in the frequency of head collisions, it is not surprising that the mean linear and rotational acceleration forces are similar; all players may be vulnerable to experiencing high and low head impacts during contact.

***Impact locations.*** Regarding impact locations, there was also some interesting and relevant differences for the participants in the current study, compared to other contact sports. Consistent with data reported by King et al. (2014), participants in the current study had impacts more common to the front (33%) and side of the head (33%), with impacts to the top being the least common (4%). Similarly, 3% of impacts reported by King et al. (2014) were to the top of the head. Research has also shown that side impacts with a higher rotational acceleration have been reported to be the most likely to result in concussion with American football and soccer players (Delaney, Puni, & Rouah, 2006). As noted, rotational forces have been implicated with diffuse brain injuries, of which are more likely to be associated with the clinical and neuropsychological impairments observed with concussion, compared to focal brain injury. Given the reported increased exposure to side impacts and to high-impact rotational acceleration forces in contact sports generally, further research is warranted to

investigate delayed recovery and neuropsychological and neurological impairment with these types of injuries, as well as methods to minimising exposure to such injuries.

Impacts to the back of the head were also relatively significant in the current study, with 29% of head impacts recorded to the back of the head. This outcome is not a long way off of the 24% of impacts to the back of the head recorded by King et al. (2014), and is also not surprising with participation in rugby. These results possibly reflect the nature of play in rugby, whereby multiple opposing players are competing for the ball in ruck and maul situations. For example, a ruck occurs after a tackle is made. Thereafter, the tackler will ideally try to get back onto his feet as quickly as possible to attempt to wrestle with the opposing player to 'steal' the ball. Simultaneously, the tackler's teammates and opposition players will join the ruck to protect or compete for the ball, thus rendering players involved in this type of a situation vulnerable to multiple head impacts to all sides of the head, including potential impacts to the back of the head.

**Limitations and future directions.** This exploratory research was conducted as a pilot study and with an internal residence team that participated in a social contact rugby league. Given this, the sample size was small and the intensity of play and contact between players is considered to be below the level that would ordinarily be expected for an amateur men's rugby club, semi-professional, or professional rugby match. Also, none of the games were videotaped, and thus impacts could not be verified and could not be broken down into the various aspects of play, such as the tackle (tackler and non-tackler), ruck, scrum, maul, and lineout, for example. Furthermore, because there is a rolling substitutions concept that is employed in this league, whereby a team is given an unlimited number of substitutions, not every player had been exposed to the full playing time of each match, and not every player had participated in each match; thus, these results should not be interpreted to reflect the true incidence of head collisions in a full contact rugby match.

However, the aim of the current study was to model the use of HIT to obtain a snapshot of its utility in recording impacts to the head in a rugby match. The study itself and the results obtained provide a framework and motivation for future studies of this nature, in order to explore these findings further with more intense and longer rugby matches in professional leagues. These results, collected with a small sample of amateur and social rugby players, could potentially indicate a greater number and increased severity of subconcussive impacts with semi-professional or professional rugby players.

Furthermore, the use of the xPatch was a novel addition to this research. Despite the important results provided, the xPatch proved to be an annoyance for some of the players as it would sometimes lose its adhesion due to sweat, constant rubbing during scrummaging, rucking, or mauling, or was vulnerable to being ripped off by either an opposing player or teammate during collisions. Given this, more effort should be taken to improve the adhesive nature of the xPatches or redesign HIT specifically for non-helmeted sports whereby there is greater exposure. Recently, X2Biosystems Inc. has launched the X-Patch Pro (X2Biosystems Inc.) which has a better encasement of the device, however, the utility of this to rugby may be still be limited given its adhesion using only a plaster. Future studies will aim to include better measures to promote greater comfort and adhesion.

**Conclusion.** Given the potential deleterious neuropsychological and neurological implications of potential subconcussive head injuries in the absence of any obvious clinical signs of concussion, monitoring head impacts in contact sports (considered a possible measure of such potential injuries) can assist with the identification of those of particularly high-impact. In doing so, such data can alert medical personnel to evaluate and monitor players at risk for associated effects of high-impact head collisions even when these are not formally diagnosed as concussions. This format of clinical care provides an addition to the medical diagnosis and monitoring of concussion, rather than as a separate entity. In doing so,

monitoring repeated head injuries, both concussive and possible subconcussive ones, may help to reduce the incidence and severity of the resultant sequelae. By being able to apply precautionary measures immediately, without relying on the potential manifestation of clinical symptoms and being able to recognise these immediately post-injury, could aid this process. In doing so, these procedures are likely to help negate the development of more serious impairment, such as long-term neurodegeneration. Furthermore, head collision data related to potential subconcussive and concussive head injuries can be used to develop more accurate and relevant head injury tolerance levels, and may help to inform interventions that could reduce the risk of concussion. Such interventions could include tackle technique, the use of head protection gear, and regulations and laws of the game surrounding phases of play whereby players are vulnerable to a high frequency and severity of head collisions.

The successful use of this technology may in turn support, and feature in, the emergence of a larger, multi-faceted, and long-term prospective study that aims to investigate the neuropsychological effects of repeated concussions and subconcussive head injuries.

**Study 3: Investigating the relationship between the xPatch (X2Biosystems Inc.) data, and potential subconcussive head injuries, and cognitive outcomes, in a sample of university rugby players**

As demonstrated in Study 2, rugby players, even for the duration of only four contact rugby matches in a social and amateur rugby league, sustained multiple impacts to the head, many of which included acceleration forces of a severity that has previously been implicated with concussion. Nevertheless, as a concussion diagnosis is still predominantly reliant on the recognition of clinical symptoms to indicate a player's vulnerability, players may repeatedly be exposed to such high impact head collisions without appropriate and timely preventative

measures during their involvement in a contact sport. As such, a continued exposure to these head injury conditions, that can potentially be as, or more severe, than impacts associated with the development of concussion-like symptoms, may result in the gradual increase of a player's vulnerability – this involves, amongst other things, subsequent concussions, or more severe outcomes over time, such as second-impact syndrome and/or, as a small body of literature appears to suggest, potential neurodegeneration. However, because previous research has offered a limited means of prospectively monitoring a player's neuropsychological performance in conjunction with head injury exposure, not defined as concussions, there is limited evidence for the contribution of subconcussive head injuries, (which could potentially be associated with such high impact head injuries), compared to other demographic, medical, and head injury factors, to these more severe outcomes. Repeated high-impact subconcussive head injuries have also been implicated with subtle neuropsychological and neurological deficits, in terms of both short- and long-term impairments (Baugh et al., 2012; Gavett et al., 2011; McKee et al., 2009; Stern et al., 2011; Talavage et al., 2014). This raises some concern for potential cumulative brain trauma.

Recent studies have therefore begun looking at objective measures of high impact head collisions, as measured by HIT, in conjunction with neuropsychological testing and neuroimaging (Breedlove et al., 2012; Talavage et al., 2014). For example, Talavage et al. (2014) demonstrated that while peak acceleration forces were not predictive of neurological trauma (i.e., structural changes), there is a significant correlation between the number and severity of head collisions, as measured by a Riddell Revolution helmet (Riddell, Elyria, OH) fitted with a sensor array (HIT System), and performance on neurocognitive testing. These results showed that players who were not diagnosed with a concussion, but who demonstrated significantly lower scores on cognitive testing, had a significantly greater number of head collision events. Despite such emerging evidence on the association between objective

measures of high impact head collisions as measured by HIT, and neuropsychological outcomes, the evidence base is limited.

### **Aims and Hypothesis**

In Study 3, which serves as a continuation of Study 2, I aim to investigate the association between high impact head injuries and neuropsychological outcomes, with the supposition that such head injuries may in fact represent potential subconcussive head injuries. Although it is also exploratory, a hypothesis can be drawn based on the findings of Study 2.

In light of this aim, I will test the following hypothesis:

Rugby players that have been exposed to a greater number and higher severity of head collisions as measured by the xPatch, are likely to score more poorly on neuropsychological outcome measures than rugby players with a lower number and severity of these head collisions.

### **Methods**

**Design and setting.** Study 3 is also a pilot study. It is prospective and quantitative, and I used a repeated measures design. Participants in this study first completed a pre-test cognitive assessment phase (Time 1, which formed part of Study 1), after which they participated in a maximum of four contact rugby matches wearing the xPatch devices (described in Study 2), and then completed a post-test assessment (Time 2), using the same cognitive measures as with time 1. Thus, the within-subjects variables (symptom and cognitive testing measures) were assessed at two time points for each participant, namely at time 1 and time 2. I used the same xPatch data, recorded prospectively, as detailed in Study 2, for the evaluation in Study 3. I conducted each testing session in a private computer lab at UCT.

**Participants.** I utilised the same sample of participants as were recruited for Study 2 (the UCT IRL,  $n = 8$ ), for Study 3.

**Materials.**

**Head impact telemetry (HIT).** Refer to Study 2 for details regarding the xPatch. As is described in Study 2, I used the xPatch in Study 3 to measure continuously, acceleration and other linear and rotational forces to the head during rugby games.

**Screening measures.** I used the same screening measures that have been described in Study 1 for the pre-test cognitive assessment phase (time 1) in Study 3. These include the demographic and medical history questionnaire, AUDIT (Sanders et al., 1993), BDI-II (Beck et al., 1996), and the Shipley-2 (Shipley et al., 2009). Refer to Study 1 for details regarding these screening measures.

**Symptom and cognitive testing measures.** I used the same symptom and cognitive measures that have been described in Study 1 for the pre- and post-test cognitive assessment phases (time 1 and time 2) in Study 3. These include the ImPACT PCS and the ImPACT neuropsychological test battery (Lovell et al., 2000). Refer to Study 1 for details regarding these symptom and cognitive measures.

**Procedure.** At time 1, participants completed the screening, and symptom and cognitive testing measures. As described in Study 2, I randomly assigned an xPatch to each participant. The UCT IRL team wore the xPatch during a maximum of four contact rugby matches that formed a part of the UCT Internal Rugby League. More specifically, four players took part in all four matches, with two players participating in two and two players participating in three matches. After the completion of the last designated match, I conducted the post-test assessment at time 2, with alternate forms of the same pre-test measures. I then fully debriefed the participants as to the nature of the pilot study, and how the testing session at time 2 served as an additional assessment to investigate the presence of any

neuropsychological vulnerabilities that may be present following an exposure to potential cumulative head injuries without concussion. I again explained the use of the xPatch as an objective measure of a player's exposure to head collisions during (in this case) a rugby match. Along with the data from the xPatch, responses from the assessment at time 1 and time 2 were collected and analysed.

### **Data analysis.**

*Correlational analyses: Cognitive outcomes and head impact exposure.* I used Pearson's correlation to investigate the relationship between neuropsychological outcomes at time 2, and: 1) the number of head impacts (recorded impacts are above the 10g data acquisition threshold), 2) the total linear and rotational acceleration impact frequency burden (IFB: the total force incurred from of all recorded impacts), and 3) the number of impacts recorded at different injury severity levels for linear acceleration (mild, <66g; moderate, 66-106g; severe, >106g) and rotational acceleration (mild, <4600 rad/s<sup>2</sup>; moderate, 4600-7900 rad/s<sup>2</sup>; severe, >7900 rad/s<sup>2</sup>) forces.

*Reliable Change Index (RCI).* I used the RCI (Jacobson & Truax, 1991) to ascertain whether each player's change in performance on the neuropsychological outcome measures from time 1 to time 2 was clinically meaningful. Devilly (2004) developed a reliable change generator showing the degree of change at three different confidence intervals (68.26%, 95%, and 99%), and I used this clinical tool to calculate individual RCI scores for each participant. To generate a RCI, I entered pre- and post-test scores, the test-retest reliability for each of the sub-tests comprising the ImPACT neuropsychological test battery, and the standard deviation of the normative sample for each sub-test. An RCI of greater than 1.96 (corresponding to a 95% confidence interval) is considered to indicate a significant difference between the pre- and post-test scores.

The RCI is based on this formula,

$$SEd = \sqrt{2}(Se)^2, \text{ where } Se = s(\sqrt{1-rxx}),$$

where  $s$  is the standard deviation and  $rxx$  is the test-retest reliability coefficient. The standard error of difference ( $SEd$ ) calculates the change between the pre- and post-test scores, accounting for the test-retest reliability coefficient. This measure thus provides an estimate of the probability that a given difference between two scores would not be obtained as a result of measurement error, which includes an adjustment for practice effects (Barr, 2002; Iverson, Sawyer, McCracken, & Kozora, 2001).

**Ethical considerations.** The ethical approval by the UCT Faculty of Health Sciences' Human Research Ethics Committee (HREC REF 010/2015) and the Department of Psychology Research Ethics Committee referred to in Study 1, incorporates Study 3. The participants in Study 3, by their inclusion in Study 1 and Study 2, underwent the same informed consent and debriefing procedures referred to in the Ethical Considerations section of Study 1. However, the UCT IRL team were given an additional presentation regarding the nature of Study 2 and Study 3. In addition to the comments made about wearing the xPatch (as referred to in Study 2), in this presentation the participants were also informed that they would be required to complete another cognitive assessment following the four games that were allocated for the data collection phase of Study 3.

## Results

### **Correlational analyses: Cognitive outcomes and head impact exposure.**

**Total head impacts.** Table 11 shows that there was a strong and significant relationship between the number of recorded head impacts and the VMS composite score at time 2 ( $r = .826, p = .006$ ). The direction of this correlation is unexpectedly positive: player's performance on a task of visual motor speed improves with a greater number of recorded head collisions. The remaining correlations were not significant. However, there was a

medium negative correlation for the Verbal Memory composite score with the total number of head impacts ( $r = -.438, p = .139$ ). Despite the non-significance of the results, the strength of the relationship suggests that players with a greater number of head impacts may have performed poorly on verbal memory components of the subtests.

***Total impact frequency burden (IFB).***

*Total linear acceleration IFB.* The results in Table 10 show a strong and significant relationship between the total linear acceleration IFB and the VMS composite score ( $r = .825, p = .006$ ). The direction of the correlation is again unexpectedly positive: players with a greater total linear acceleration performed better on the VMS task. The remaining correlations were not significant. Among these though, there was a negative moderate relationship between total linear acceleration IFB and the Verbal Memory composite score ( $r = -.442, p = .136$ ), suggesting that greater total linear acceleration may correlate with poorer performance on task of verbal memory.

*Total rotational acceleration IFB.* Table 10 shows that there was a strong and significant relationship between the total rotational acceleration IFB and the VMS composite score ( $r = .812, p = .007$ ). The direction of this correlation is again unexpectedly positive: player's performance on a task of visual motor speed improves with a greater total rotational acceleration. The remaining correlations were not significant. Among these, there was a negative moderate correlation between total rotational acceleration IFB and the Verbal Memory composite score ( $r = -.448, p = .133$ ), again suggesting that verbal memory performance on the subtests may also decline, however slightly, with greater total rotational acceleration.

Table 11.  
*Correlations Between Head Impact Exposure and Cognitive Outcomes.*

|                                |                     | Verbal<br>Memory | Visual<br>Memory | VMS    | RT    |
|--------------------------------|---------------------|------------------|------------------|--------|-------|
| Total head impacts             | Pearson Correlation | -.438            | -.169            | .826   | -.095 |
|                                | <i>P</i>            | .139             | .345             | .006** | .411  |
| Linear acceleration<br>IFB     | Pearson Correlation | -.442            | -.139            | .825   | -.009 |
|                                | <i>P</i>            | .136             | .371             | .006** | .492  |
| Rotational<br>acceleration IFB | Pearson Correlation | -.448            | .086             | .812   | -.036 |
|                                | <i>P</i>            | .133             | .420             | .007** | .466  |

*Note.* IFB = impact frequency burden; VMS = visual motor speed; RT = reaction time.

\* $p < 0.05$  \*\* $p < 0.01$

**Individual comparisons: RCI analyses.** Table 12 provides a summary of the results from the RCI analysis, indicating the level of reliable change for the four primary composite scores of the ImPACT concussion battery assessed at time 1 and time 2 for the UCT IRL team. In this table, I have included head impact exposure per player, which is especially relevant to investigating comparative changes in cognitive outcomes.

The results show that following the four rugby matches, there was a significant decline for at least one player, defined as a change with 95% confidence, for the Verbal Memory, Visual Memory, and the Reaction Time task. However, there were also some players across these domains who seemed to show positive significant changes, that is, improved significantly in their performances from time 1 to time 2. For the VMS measure, although one player scored more poorly at time 2, this reliable change was at a low confidence level of 68.26% confidence. The majority of the players, in fact, seem to score better on the tests from time 1 to time 2.

Notably, as Table 12 shows, three of the five Forwards players performed significantly worse on at least one of the subtests, with these changes at the 95% confidence

level. On the other hand, other than one change at the 68.26% confidence level, none of the Backline players experienced a significant decrease in performance at time 2. In this regard, two Forwards players performed significantly worse at time 2 for Visual Memory and the Reaction Time composite, with one Forward player scoring significantly worse at time 2 for Verbal Memory.

Table 12.

*RCI Analyses for Cognitive Outcome Changes from time 1 to time 2 with the Internal Rugby group.*

| Player          | Total head impacts | Cognitive Outcomes |                    |               |        |      |               |        |      |        |        |      |        |        |      |
|-----------------|--------------------|--------------------|--------------------|---------------|--------|------|---------------|--------|------|--------|--------|------|--------|--------|------|
|                 |                    | RLA IFB            | RRA IFB            | Verbal Memory |        |      | Visual Memory |        |      | VMS    |        |      | RT     |        |      |
|                 |                    | g                  | rad/s <sup>2</sup> | time 1        | time 2 | RCI  | time 1        | time 2 | RCI  | time 1 | time 2 | RCI  | time 1 | time 2 | RCI  |
| <b>Forwards</b> |                    |                    |                    |               |        |      |               |        |      |        |        |      |        |        |      |
| 1209            | 62                 | 1378.27            | 240483.57          | 83            | 71     | None | 76            | 83     | ▲    | 34.90  | 43.20  | ▲▲   | 0.60   | 0.60   | None |
| 1401            | 164                | 3123.97            | 427379.10          | 92            | 84     | ▲    | 90            | 85     | None | 42.75  | 50.63  | ▲▲   | 0.46   | 0.51   | None |
| 1317            | 64                 | 1268.16            | 153428.90          | 96            | 85     | ▲▲   | 89            | 76     | ▲▲▲  | 44.47  | 44.95  | None | 0.56   | 0.57   | None |
| 1318            | 94                 | 2024.09            | 264284.30          | 89            | 96     | ▲    | 86            | 69     | ▲▲▲  | 42.90  | 45.72  | None | 0.56   | 0.69   | ▲▲   |
| 1315            | 64                 | 1600.82            | 305121.50          | 96            | 90     | ▲    | 97            | 99     | None | 51.68  | 45.97  | ▲    | 0.49   | 0.59   | ▲▲   |
| <b>Backline</b> |                    |                    |                    |               |        |      |               |        |      |        |        |      |        |        |      |
| 1319            | 24                 | 510.81             | 79334.43           | 100           | 94     | ▲    | 81            | 79     | None | 31.17  | 37.33  | ▲    | 0.67   | 0.55   | ▲▲   |
| 1210            | 63                 | 1293.56            | 219565.40          | 89            | 99     | ▲▲   | 85            | 94     | ▲    | 43     | 46.63  | ▲    | 0.58   | 0.52   | ▲    |
| 1207            | 12                 | 199.81             | 34796.79           | 83            | 97     | ▲▲▲  | 83            | 91     | ▲    | 36.58  | 42.75  | ▲    | 0.61   | 0.57   | None |

*Note.* RLA = Resultant linear acceleration; RRA = Resultant rotational acceleration; IFB = impact frequency burden; RCI = reliable change index; VMS = visual motor speed; RT = reaction time.

Players 1401, 1315, 1210, and 1207 played 4 games; 1209, and 1308 played 3 games; 1317, and 1319 played 2 games.

The triangles indicate whether a participant's performance has improved (green triangle) or worsened (red triangle) at the post-test assessment (time 2).

The degree of reliable change is indicated at the following confidence levels:

▲ : 68.26%; ▲▲ : 95%; ▲▲▲ : 99%.

## Discussion

The aim of Study 3 was to investigate the relationship between neuropsychological outcomes and high impact head injuries as measured by the xPatch, in four internal rugby league matches. Given evidence for a potential decrease in performance on cognitive measures in players exposed to repeated subconcussive head injuries, which high impact head injuries may be suggestive of, and as demonstrated by Talavage et al. (2014) and Breedlove et al. (2012), it was my hypothesis that rugby players exposed to a greater number and a higher severity of high impact head injuries would likely perform more poorly than rugby players with a lower number and severity of these head injury conditions. This hypothesis was only partially confirmed.

**Summary of results.** The first part of the results from Study 3 focussed on the correlation between the cognitive outcome measures (at time 2) and objective data about head collisions from the xPatch, specifically for the number of impacts and the total linear and rotational impact force sustained by each player. Specifically, the results showed positive correlations for VMS with total head impacts, total linear acceleration IFB, and total rotational acceleration IFB, all suggesting, surprisingly, a relationship between an increased and more severe exposure to subconcussive head injuries and faster visual motor speed performance. Results also showed a medium negative correlation for the Verbal Memory composite score with the total number of head impacts, total linear acceleration IFB, and total rotational acceleration IFB, indicating that an increased and more severe exposure to high impact head injuries may correlate with poorer performance on task of verbal memory. Overall, however, there was a lack of sufficient evidence to indicate any significant and meaningful correlations to suggest that player's increased exposure to repeated high-impact head collisions results in a generally poorer neuropsychological performance.

With regards to individual changes in cognitive performance from time 1 to time 2, only some players experienced a decline in performance at time 2, while others did not. In

fact, the majority of players did not experience a significant decline in cognitive performance at time 2, with only three of the eight players demonstrating a reliable (negative) change at or above a confidence of 95%. For these three players, only one player demonstrated a decrease on the Verbal Memory composite score, with two players demonstrating a significant decrease for Visual Memory and Reaction Time. Predominantly Forwards rather than Backline players showed significant decreases in performance. Further, a number of players actually showed positive increases in test performance from time 1 to time 2. I discuss each set of results below.

*Correlations.* Notably, significant and strong correlations were found between a measure of visual motor processing speed (measured at time 2) and total number of head collisions, and total linear and rotational acceleration IFB. The direction of this correlation however indicates that a player's performance of visual motor speed (VMS composite score) improves with more frequent and severe head collisions. This result is thus unexpected, and poses some concerning questions regarding the possible impact of practice effects, given that the participants had previously completed the ImPACT at time 1. Evidence of improved performance on the VMS task with repeated testing of the ImPACT was also demonstrated with undergraduate college students with no history of concussion by Schatz and Ferris (2013) over a four-week interval between testing sessions; no other scores improved significantly over this interval in that study. Given that the ImPACT test battery accommodates for repeat testing, evidenced by its multiple and randomised test-forms with alternating stimuli, Schatz and Ferris (2013) propose that the improved performance on the VMS composite score may be due to a familiarity with the physical mechanics of working the computerised task. It happens that the VMS task may be especially vulnerable in this regard.

However, using a South African sample of rugby players, Shuttleworth-Edwards, Radloff, Whitefield-Alexander, Smith, and Horsman (2014) reported a lack of practice effect with rugby players presenting with a history of concussion compared to non-contact sport controls. In this regard, it is proposed that a control group is absolutely necessary to provide a more accurate estimation of visual motor speed vulnerabilities following concussion and/or subconcussive head injuries.

Although the remaining correlations reported in Study 3 were not significant, a negative moderate relationship was found between verbal memory performance and total number of head collisions, and total linear and rotational acceleration IFB. These results thus show a potential vulnerability of verbal memory abilities following a more frequent and severe exposure to head collisions; this trend may be more obvious and significant with a larger study. Nevertheless, there is an abundance of research implicating vulnerable memory abilities, and specifically verbal memory abilities, with concussion, and a repeated high exposure to subconcussive head injuries (Belanger et al., 2005; Belanger & Vanderploeg, 2005; Clark et al., 2016; Comerford et al., 2002; Karr et al., 2014; Mathias, Beall, & Bigler, 2004; Rohling et al., 2011; Talavage et al., 2014). Further research has also shown the vulnerability of neural structures crucial for the optimal functioning of memory following concussion and repeated subconcussive head injuries (Aoki & Inokuchi 2016; Blumbergs et al., 1994; Henry et al., 2016; Talavage et al., 2014).

***RCI results.*** The results from the RCI analysis show that only three of the eight players demonstrated a significant decline in their performance on any of the cognitive measures from time 1 to time 2, with these three players being Forwards. In light of this information, the three Forwards players demonstrating a decline in performance at time 2 also presented with the second, third and fourth most impacts and highest total rotational acceleration IFB, and the second, third and fifth highest total linear acceleration IFB. In

addition, the player who sustained the most number of severe rotational acceleration forces (Player 1315) did not show any improvement on any of the cognitive measures, although he only performed significantly worse on the Reaction Time measure. Also, interestingly, these results seem consistent with those reported in Study 2 in that the Backline players, for whom, there was no significant decline in performance on any of the measures, each sustained a lower linear and rotational acceleration IFB's compared to all of the Forwards players, bar one. These results show the potential negative impact on neuropsychological performance of a repeated exposure to high impact head collisions in the absence of any concussion-like symptoms.

Even though the RCI takes into account test retest reliability coefficients, we did not include a control group and therefore cannot rule out the possibility that these results may also be subject to practice effects using the ImPACT. Only one player demonstrated a decrease in performance on the VMS composite score, with this being a reliable change at the 68% confidence interval. Further, an improved performance from time 1 to time 2, with at least a reliable change at 68%, was in fact indicated for each composite score tested in the RCI analysis.

These results thus should be interpreted with caution, and further research is needed to assess the reliability of the ImPACT and other neuropsychological measures across short and long intervals of testing. This is pertinent given that players optimally need to be assessed repeatedly in the acute post-concussion phase and repeatedly over the course of a season to assess recovery trajectories related to an exposure to subconcussive and concussion injuries.

In sum, despite a few significant differences, the RCI analysis did not provide convincing evidence across all eight players for a significant decline in cognitive performance after sustaining repeated subconcussive head injuries. However the pattern of

change within the data are suggestive of possible practice effects, which would undermine the results.

**Limitations and future directions.** Given the nature of Study 3 as a pilot study, the results are limited in terms of being able to draw significant comparisons between neuropsychological outcomes and an exposure to high impact and potential subconcussive head injuries. This may be a function of (among other limitations already highlighted in Study 2 and which also apply here) a small sample size and the limited number of contact rugby matches used for data collection, thus also shortening the test-retest interval. Also, these games formed part of a social contact rugby league of which the intensity of contact is not as would be expected at an amateur club level of play. Future studies should therefore be conducted with rugby teams at varying professional levels and over longer periods of play.

Furthermore, as I have noted, tasks of visual motor speed specifically, but also other measures potentially, may have been subject to practice effects. It is reasonable to suggest that at time 2 players may have been more comfortable with the working and mechanics of the tasks, and this is likely to have improved their performance. Future studies should therefore include control groups in order to manage such effects.

Nevertheless, some, albeit limited, significant changes in players' neuropsychological performance between pre- and post-testing were observed, and this provides some encouragement for future research to explore further, in a larger longitudinal study, the possibility of multiple subconcussive and concussive head injuries to such changes. Furthermore, in a larger prospective study, further research should focus neuropsychological and neurological investigations with players who have sustained a large number of severe acceleration forces, particularly regarding rotational acceleration.

**Conclusion.** Objective data such as that collected from the xPatch and other varieties of HIT have been shown to be valuable for the immediate identification of on-field

associations between the extent and nature of high impact head collisions and head injury implications. The data obtained can be used in a variety of ways, from being able to assess head injury tolerance levels to looking at tackle techniques and ruck laws to reduce the frequency of high impact head collisions, particularly for impacts resulting in concussion. However, despite this information and potential for informing better laws and precautions to ensuring the safety of contact sportsmen and women, there is still limited research to show that a repeated exposure to subconcussive head injuries may in fact contribute in short- and/or long-term neuropsychological and neurological impairment.

However, this should not be considered the end of the road. There is still some evidence for the delayed presentation of concussion symptoms, neuropsychological impairment, and neurological changes in a minority of sports-players post-concussion. Moreover, compared to non-contact sport controls, there is evidence for decreased neuropsychological performance and neurological impairment for athletes with a high exposure to head collisions, but who present with no clinical signs of concussion and thus no concussion diagnosis. In some cases, players presenting with these repeated and high-impact subconcussive head injuries have been shown to be as impaired on these measures as players with a recent concussion diagnosis. Although this evidence points to a minority of athletes, and not demonstrated clearly here, Study 3 has shown the utility of HIT devices for being able to more accurately identify players who may be vulnerable to these effects, suggesting the need for a multifaceted framework for assessing players relevant to their current exposure to impact collisions in sport.

### **General Summary and Conclusion**

There is an increased concern for accurately diagnosing and appropriately managing concussion in the acute post-concussion phase given the increased risk of long-term neuropsychological and neurological consequences that have been implicated with a history

of concussion and/or a lengthy participation in contact sport. Athletes in a contact sports environment in particular are especially vulnerable to a high rate of exposure to repeated head injuries, including concussion. However, in light of this information, as well as the potential deleterious neuropsychological and neurological impact of multiple concussions and/or subconcussive head injuries in the acute post-injury phase, there has been limited research to draw accurate causal associations between these injurious conditions and long-term, enduring neuropsychological and neurological impairments.

The majority of research has shown that concussion symptoms typically resolve within the first two weeks post-concussion, with a complete resolution of any residual neuropsychological difficulties by three months (i.e., within the acute post-concussion phase). There are however numerous moderating factors that could potentially result in a slightly delayed recovery even if only in a minority of sports players. These include, but are not limited to, for example, having a history of multiple concussions, the severity of a concussion history, a younger age, psychological difficulties, and some situational factors such as litigation or motivation. Nevertheless, players presenting with a delayed recovery still show a full recovery of concussive symptoms within the acute post-concussion phase, and are thus likely to return to play within this time frame.

However, despite a relatively favourable resolution of concussive symptoms, neuropathological deficits post-concussion can be enduring, and may persist beyond the acute-post concussion phase. Again, evidence for this is however limited, occurring with a small minority of players presenting with a slightly delayed symptom recovery. What this evidence thus points to is a potential enduring vulnerability in the absence of clinical symptoms and consequently also a lack of rest from play, or of clinical management or treatment. In addition to this, upon returning to a contact sports environment players are exposed to repeated potential subconcussive head injuries, which may also be associated with

short-term vulnerability to unfavourable neuropsychological and neurological outcomes, which could potentially be cumulative over time.

However, despite this vulnerability for ongoing neuropsychological difficulties and subtle neurological changes, retrospective research has found little, if any significant differences in neuropsychological performance between contact sport athletes with a history of concussion and those without. However, such research often fails to account for the possible contribution of subconcussive head injuries to these results, and thus a lack of differences between two rugby groups could potentially be due to both groups having a recent exposure to cumulative head injuries. Furthermore, the issues previously discussed with regards to retrospective research limit this research.

Against this backdrop, the overarching aim of the three studies presented in this dissertation was to investigate concussive and potential subconcussive head injuries, as measured by HIT, in relation to neuropsychological outcomes.

In Study 1, Rugby players with and without a history of concussion were compared to non-contact sports players without a recent exposure to repeated head collisions.

Similar to past literature, results showed heightened alcohol use among the rugby players compared to non-contact controls. Although this was not an intended aim of the study, but an incidental and nonetheless important finding as a function of the screening measures included in the study, these results speak to the need for intervention around drinking among rugby players, particularly in terms of potential increased vulnerability to injury if such behaviour perhaps overlaps with game and between game, rest time.

Also similar to the majority of retrospective research looking at neuropsychological outcomes at baseline, Study 1 largely provided no evidence for distinct between groups differences on neuropsychological outcomes. Concussion history was however based on retrospective accounts through self-report, for which the limitations are well known. Further,

questions around sensitivity of the measures employed and the motivation of players regarding test completion are further limitations to the study, which may have impacted on results. On the other hand, it may very well be that there are no enduring neuropsychological effects of concussion. To reliably address these questions, well-designed prospective, longitudinal studies of contact sports players are needed which include regular assessments using several measures.

Although the majority of evidence point to a quick and favourable recovery from concussion, insight into individual differences in recovery from concussion may shed some insight as to why or how some individuals develop deficits later on in life. Investigating individual differences in recovery however requires a means for continuously monitoring and assessing these players for their exposure to head collisions and concussion and their resulting neuropsychological and neurological performance. Furthermore, assessing recovery in the acute post-concussion phase typically and ideally needs to be assessed from an individual's baseline performance, prior to that person having had sustained any previous concussions and without a recent exposure to multiple head injuries. Hence investigating a younger sample earlier in their rugby history for example may be ideal.

Studies 2 and 3 involved the exploratory use and investigation of a HIT device, the xPatch. While study 2 was largely a descriptive study on the forces applied to the head, as captured by the xPatch, the data show that there are a large number of subconcussive head collisions that are recorded above a level of force that has previously been implicated with concussion. The results regarding greater vulnerability of players, and Forwards players in particular, to rotational rather than linear forces, are also important. Both findings present potential windows for intervention. Although Study 3 showed limited association and reliable changes in terms of decreases in cognitive performances as a function of high-impact head injuries, there were some data showing that those with higher impact head collisions more

often showed negative reliable change. Once again, Forwards players were implicated as more vulnerable to Backline players, even with limited results. Overall, Studies 2 and 3 demonstrate utility in using HIT such as the xPatch and highlight particular positional categories than may require more attention and intervention. Future, larger scaled studies of this nature that are also prospective in nature and include video-recorded games, against which the HIT data can be verified, are needed.

While objectively monitoring a player's exposure to cumulative head injuries, researchers may at least in part, relieve themselves of having to rely solely on the accurate diagnoses of concussion, given the previously highlighted difficulties herewith. Furthermore, in doing so, medical and management personnel can evaluate the neuropsychological and neurological effects of an increased exposure to repeated head injuries, not just concussion. HIT enables researchers to monitor all forces sustained to the head in a contact sports environment.

Importantly, this increased exposure to repeated high-impact head collisions, without the diagnosis of concussion and the appropriate intervention thereafter, may also be playing a substantial role in the increasing vulnerability of players to a subsequent concussion, delayed recovery, and potentially to cumulative brain trauma. As a consequence of these factors and premature return to play, players may be increasing their vulnerability to potential long-term neuropsychological and neurological impairment. Associations between head impact exposure and individual variations in neuropsychological and neurological recovery over time could provide an informative view for identifying players at risk of long-term impairment.

The data presented in this dissertation, although it was recorded with a limited sample size and in a low-intensity contact sports environment, highlights the importance of investigating and monitoring players not only by relying on a concussion diagnosis, but also

looking at their overall exposure to high-impact head collisions, for which athletes are especially vulnerable to in contact sport. The data presented here are for an internal, social rugby league team; one might extrapolate how much more severe and intense the data for more professional teams in longer and more intense games, may be.

Despite the use of HIT in the current research, a key emphasis is to use a multi-faceted approach to monitoring players prospectively and to investigate the impact of subconcussive and concussive head injuries on short-term recovery and the relationship of this to potential enduring neuropsychological and neurological impairment. Further, it is recommended that research of this nature take a thorough demographic and medical history and an in-depth neuropsychological investigation of baseline performance. It is also advised that investigators take great precaution when selecting a cognitive test battery so that it may be resistant to the effects of repeated testing and sensitive to detecting subtle changes in functioning, and also that a thorough assessment tool be used specifically for on-field concussion assessment.

Monitoring athletes on an individual basis and prospectively is imperative to investigating player vulnerability to the gradual development of long-term neuropsychological or neurological impairment. While the majority of research shows the quick resolution of concussion symptoms in the acute post-concussion phase, monitoring player recovery using a multi-faceted approach may help to identify subtle indicators of a player's potential increased vulnerability to more severe outcomes should they return to play. In doing so, this type of research is crucial for identifying the potential contribution of such injuries to long-term term impairment and for dismissing a reliance on only diagnosed concussions. Moreover though, the potential of this type of research to better inform precautionary and safety measures regarding head impact exposure and players' neuropsychological and neurological vulnerability, toward identifying injury threshold limits,

and to inform coaching strategies and laws to lower the risk of high-impact head collisions, should be recognized.

### References

- Abbas, K., Shenk, T. E., Poole, V. N., Breedlove, E. L., Leverenz, L. J., Nauman, E. A., ... & Robinson, M. E. (2015). Alteration of default mode network in high school football athletes due to repetitive subconcussive mild traumatic brain injury: A resting-state functional magnetic resonance imaging study. *Brain Connectivity, 5*, 91-101.
- Adams, S., Savahl, S., Isaacs, S., & Zeta Carels, C. (2013). Alcohol consumption amongst young adults living in a low socio economic status community in Cape Town. *Social Behaviour and Responsibility, 41*, 971-980.
- Alla, S., Sullivan, S. J., & McCrory, P. (2012). Defining asymptomatic status following sports concussion: Fact or fallacy? *British Journal of Sports Medicine, 46*, 562-569.
- American Academy of Neurology (AAN). (1997). Practice parameter: The management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology, 48*, 581-585.
- American Psychiatric Association (APA). 2000. DSM-IV: Diagnostic and Statistical Manual of Mental Disorders. Washington D.C.
- Anderson, V., Northam, E., Hendy, J., & Wrennall, J. (2001). Developmental Neuropsychology. Hove: Psychology Press.
- Aoki, Y., & Inokuchi, R. (2016). A voxel-based meta-analysis of diffusion tensor imaging in mild traumatic brain injury. *Neuroscience & Biobehavioral Reviews, 66*, 119-126.
- Aoki, Y., Inokuchi, R., Gunshin, M., Yahagi, N., & Suwa, H. (2012). Diffusion tensor imaging studies of mild traumatic brain injury: A meta-analysis. *Journal of Neurology, Neurosurgery & Psychiatry, 83*, 870-876.

- Aubry, M., Cantu, R., Dvorak, J., Graf-Baumann, T., Johnston, K., Kelly, J., ... & Schamasch, P. (2002). Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001. *British Journal of Sports Medicine*, *36*, 6-10.
- Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). *The Alcohol Use Disorders Identification Test: Guidelines for use in primary care*. Geneva: World Health Organisation.
- Bailes, J. E., Petraglia, A. L., Omalu, B. I., Nauman, E., & Talavage, T. (2013). Role of subconcussion in repetitive mild traumatic brain injury: A review. *Journal of Neurosurgery*, *119*, 1235-1245.
- Baillargeon, A., Lassonde, M., Leclerc, S., & Elleberg, D. (2012). Neuropsychological and neurophysiological assessment of sport concussion in children, adolescents and adults. *Brain Injury*, *26*, 211-220.
- Baker, J. F., Devitt, B. M., Green, J., & McCarthy, C. (2013). Concussion among under 20 rugby union players in Ireland: Incidence, attitudes and knowledge. *Irish Journal of Medical Science*, *182*, 121-125.
- Baker, J. G., Leddy, J. J., Darling, S. R., Shucard, J., Makdissi, M., & Willer, B. S. (2016). Gender differences in recovery from sports-related concussion in adolescents. *Clinical Pediatrics*, *55*, 771-775.
- Barr, W. B. (2002). Neuropsychological testing for assessment of treatment effects: Methodologic issues. *CNS Spectrums*, *7*, 300-306.
- Baugh, C. M., Stamm, J. M., Riley, D. O., Gavett, B. E., Shenton, M. E., Lin, A., ... & Stern, R. A. (2012). Chronic traumatic encephalopathy: Neurodegeneration following

repetitive concussive and subconcussive brain trauma. *Brain Imaging and Behavior*, 6, 244-254.

Bazarian, J. J., Zhu, T., Blyth, B., Borrino, A., & Zhong, J. (2012). Subject-specific changes in brain white matter on diffusion tensor imaging after sports-related concussion. *Magnetic Resonance Imaging*, 30, 171-180.

BBC Sport, Rugby Union. (2015, January 04). Concussion: Saracens trial new impact-measuring device. *BBC*. Retrieved from <http://www.bbc.com/sport/rugby-union/30671380>.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory manual (2nd Ed.)*. San Antonio, TX: Psychological Corporation.

Beckwith, J. G., Greenwald, R. M., Chu, J. J., Crisco, J. J., Rowson, S., Duma, S. M., ... & Anderson, S. (2013). Head impact exposure sustained by football players on days of diagnosed concussion. *Medicine and Science in Sports and Exercise*, 45, 737-746.

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., Spiegel, E., & Vanderploeg, R. D. (2010). Neuropsychological performance following a history of multiple self-reported concussions: A meta-analysis. *Journal of the International Neuropsychological Society*, 16, 262-267.

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.

- Belson, K. (2015, December 13). How a boy's concussion death changed British sports. *The New York Times*. Retrieved from [http://www.nytimes.com/2015/12/14/sports/rugby/tragedy-forges-alliance-for-change-in-concussion-protocol.html?\\_r=0](http://www.nytimes.com/2015/12/14/sports/rugby/tragedy-forges-alliance-for-change-in-concussion-protocol.html?_r=0).
- Benson, B. W., Meeuwisse, W. H., Rizos, J., Kang, J., & Burke, C. J. (2011). A prospective study of concussions among National Hockey League players during regular season games: The NHL-NHLPA Concussion Program. *Canadian Medical Association Journal*, *183*, 905-911.
- Bigler, E. D., Farrer, T. J., Pertab, J. L., James, K., Petrie, J. A., & Hedges, D. W. (2013). Reaffirmed limitations of meta-analytic methods in the study of mild traumatic brain injury: A response to Rohling et al. *The Clinical Neuropsychologist*, *27*, 176-214.
- Bigler, E. D., & Maxwell, W. L. (2012). Neuropathology of mild traumatic brain injury: Relationship to neuroimaging findings. *Brain Imaging and Behavior*, *6*, 108-136.
- Blank, M. L., Connor, J., Gray, A., & Tustin, K. (2015). Screening for hazardous alcohol use among university students using individual questions from the Alcohol Use Disorders Identification Test-Consumption. *Drug and Alcohol Review*, *34*, 540-548.
- Blumbers, P. C., Scott, G., Manavis, J., Wainwright, H., Simpson, D. A., & McLean, A. J. (1994). Staining of amyloid precursor protein to study axonal damage in mild head injury. *The Lancet*, *344*, 1055-1056.
- Bohn, M. J., Babor, T. F., & Kranzler, H. R. (1995). The Alcohol Use Disorders Identification Test (AUDIT): Validation of a screening instrument for use in medical settings. *Journal of Studies on Alcohol*, *56*, 423-432.

- Boyle, E., Cancelliere, C., Hartvigsen, J., Carroll, L. J., Holm, L. W., & Cassidy, J. D. (2014). Systematic review of prognosis after mild traumatic brain injury in the military: Results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of Physical Medicine and Rehabilitation, 95*, S230-S237.
- Breedlove, E. L., Robinson, M., Talavage, T. M., Morigaki, K. E., Yoruk, U., O'Keefe, K., ... & Nauman, E. A. (2012). Biomechanical correlates of symptomatic and asymptomatic neurophysiological impairment in high school football. *Journal of Biomechanics, 45*, 1265-1272.
- Brenner, L. A., Terrio, H., Homaifar, B. Y., Gutierrez, P. M., Staves, P. J., Harwood, J. E., ... & Warden, D. (2010). Neuropsychological test performance in soldiers with blast-related mild TBI. *Neuropsychology, 24*, 160-167.
- Broglio, S. P., Cantu, R. C., Gioia, G. A., Guskiewicz, K. M., Kutcher, J., Palm, M., & Valovich McLeod, T. C. (2014). National Athletic Trainers' Association position statement: Management of sport concussion. *Journal of Athletic Training, 49*, 245-265.
- Broglio, S. P., Eckner, J. T., Martini, D., Sosnoff, J. J., Kutcher, J. S., & Randolph, C. (2011). Cumulative head impact burden in high school football. *Journal of Neurotrauma, 28*, 2069-2078.
- Broglio, S. P., Ferrara, M. S., Piland, S. G., & Anderson, R. B. (2006). Concussion history is not a predictor of computerised neurocognitive performance. *British Journal of Sports Medicine, 40*, 802-805.
- Broglio, S. P., Macciocchi, S. N., & Ferrara, M. S. (2007a). Neurocognitive performance of concussed athletes when symptom free. *Journal of Athletic Training, 42*, 504-508.

- Broglio, S. P., Macciocchi, S. N., & Ferrara, M. S. (2007b). Sensitivity of the concussion assessment battery. *Neurosurgery, 60*, 1050-1058.
- Broglio, S. P., & Puetz, T. W. (2008). The effect of sport concussion on neurocognitive function, self-report symptoms and postural control: A meta-analysis. *Sports Medicine, 38*, 53-67.
- Broglio, S. P., Schnebel, B., Sosnoff, J. J., Shin, S., Feng, X., He, X., & Zimmerman, J. (2010). The biomechanical properties of concussions in high school football. *Medicine and Science in Sports and Exercise, 42*, 2064-2071.
- Brooks, B. L., Mannix, R., Maxwell, B., Zafonte, R., Berkner, P. D., & Iverson, G. L. (2016). Multiple Past concussions in high school football players are there differences in cognitive functioning and symptom reporting? *The American Journal of Sports Medicine, Vol. XX, No. X*. DOI: 10.1177/0363546516655095.
- Brooks, B. L., McKay, C. D., Mrazik, M., Barlow, K. M., Meeuwisse, W. H., & Emery, C. A. (2013). Subjective, but not objective, lingering effects of multiple past concussions in adolescents. *Journal of Neurotrauma, 30*, 1469-1475.
- Brown, N. J., Mannix, R. C., O'Brien, M. J., Gostine, D., Collins, M. W., & Meehan, W. P. (2014). Effect of cognitive activity level on duration of post-concussion symptoms. *Pediatrics, 133*, e299-e304.
- Callinan, J. (2016, April 22). Butcher Boys part of ground-breaking concussion research. Newcastle Herald. Retrieved from <http://www.theherald.com.au/story/3864222/impact-study/>.

- Camarillo, D. B., Shull, P. B., Mattson, J., Shultz, R., & Garza, D. (2013). An instrumented mouthguard for measuring linear and angular head impact kinematics in American football. *Annals of Biomedical Engineering*, *41*, 1939-1949.
- Cancelliere, C., Donovan, J., & Cassidy, J. D. (2016). Is sex an indicator of prognosis after mild traumatic brain injury: A systematic analysis of the findings of the World Health Organization Collaborating Centre Task Force on Mild Traumatic Brain Injury and the International Collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of Physical Medicine and Rehabilitation*, *97*, S5-S18.
- Cancelliere, C., Hincapié, C. A., Keightley, M., Godbolt, A. K., Côté, P., Kristman, V. L., ... & Cassidy, J. D. (2014). Systematic review of prognosis and return to play after sport concussion: Results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of Physical Medicine and Rehabilitation*, *95*, S210-S229.
- Cantu, R. C., Guskiewicz, K., & Register-Mihalik, J. K. (2010). A retrospective clinical analysis of moderate to severe athletic concussions. *American Academy of Physical Medicine and Rehabilitation*, *2*, 1088-1093.
- Carroll, L. J., Cassidy, J. D., Cancelliere, C., Côté, P., Hincapié, C. A., Kristman, V. L., ... & Hartvigsen, J. (2014). Systematic review of the prognosis after mild traumatic brain injury in adults: Cognitive, psychiatric, and mortality outcomes: Results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of Physical Medicine and Rehabilitation*, *95*, S152-S173.
- Carroll, L., Cassidy, J. D., Peloso, P., Borg, J., Von Holst, H., Holm, L., ... & Pépin, M. (2004). Prognosis for mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, *43*, 84-105.

Cassidy, J. D., Carroll, L., Peloso, P., Borg, J., Von Holst, H., Holm, L., ... & Coronado, V. (2004). Incidence, risk factors and prevention of mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine, 43*, 28-60.

Castile, L., Collins, C. L., McIlvain, N. M., & Comstock, R. D. (2011). The epidemiology of new versus recurrent sports concussions among high school athletes, 2005–2010. *British Journal of Sports Medicine, 46*, 603-610.

Catale, C., Marique, P., Closset, A., & Meulemans, T. (2009). Attentional and executive functioning following mild traumatic brain injury in children using the Test for Attentional Performance (TAP) battery. *Journal of Clinical and Experimental Neuropsychology, 31*, 331-338.

The Centers for Disease Control and Prevention (CDC). (February, 2016). Basic information about traumatic brain injury and concussion. <http://www.cdc.gov/traumaticbraininjury/basics.html>.

Clark, A. L., Sorg, S. F., Schiehser, D. M., Luc, N., Bondi, M. W., Sanderson, M., ... & Delano-Wood, L. (2016). Deep white matter hyperintensities affect verbal memory independent of PTSD symptoms in veterans with mild traumatic brain injury. *Brain Injury, 30*, 1-8.

Clay, M. B., Glover, K. L., & Lowe, D. T. (2013). Epidemiology of concussion in sport: A literature review. *Journal of Chiropractic Medicine, 12*, 230-251.

Cleary, M. (2015, January 07). Saracens defend xPatch use: 'It is not a gimmick'. *The Telegraph*. Retrieved from

<http://www.telegraph.co.uk/sport/rugbyunion/club/11331756/Saracens-defend-xPatch-use-It-is-not-a-gimmick.html>.

- Collie, A., Maruff, P., Makdissi, M., McCrory, P., McStephen, M., & Darby, D. (2003). CogSport: Reliability and correlation with conventional cognitive tests used in postconcussion medical evaluations. *Clinical Journal of Sport Medicine, 13*, 28-32.
- Collie, A., McCrory, P., & Makdissi, M. (2006). Does history of concussion affect current cognitive status? *British Journal of Sports Medicine, 40*, 550-551.
- Collins, M. W., Grindel, S. H., Lovell, M. R., Dede, D. E., Moser, D. J., Phalin, B. R., ... & McKead, D. B. (1999). Relationship between concussion and neuropsychological performance in college football players. *The Journal of the American Medical Association, 282*, 964-970.
- Colvin, A. C., Mullen, J., Lovell, M. R., West, R. V., Collins, M. W., & Groh, M. (2009). The role of concussion history and gender in recovery from soccer-related concussion. *The American Journal of Sports Medicine, 37*, 1699-1704.
- Comerford, V. E., Geffen, G. M., May, C., Medland, S. E., & Geffen, L. B. (2002). A rapid screen of the severity of mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 24*, 409-419.
- UP TO HERE. Conigrave, K. M., Hall, W. D., & Saunders, J. B. (1995). The AUDIT questionnaire: Choosing a cut-off score. *Addiction, 90*, 1349-1356.
- Coronado, V. G., Haileyesus, T., Cheng, T. A., Bell, J. M., Haarbauer-Krupa, J., Lionbarger, M. R., ... & Gilchrist, J. (2015). Trends in sports-and recreation-related traumatic brain injuries treated in US emergency departments: The National Electronic Injury

- Surveillance System-All Injury Program (NEISS-AIP) 2001-2012. *The Journal of Head Trauma Rehabilitation*, 30, 185-197.
- Coronado, V. G., McGuire, L. C., Faul, M., Sugerman, D. E., & Pearson, W. S. (2012). Traumatic brain injury epidemiology and public health issues. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain Injury Medicine, 2<sup>nd</sup> Edition: Principles and Practice* (pp. 84-100). New York, NY: Demos Medical Publishing.
- Corwin, D. J., Zonfrillo, M. R., Master, C. L., Arbogast, K. B., Grady, M. F., Robinson, R. L., ... & Wiebe, D. J. (2014). Characteristics of prolonged concussion recovery in a pediatric subspecialty referral population. *The Journal of Pediatrics*, 165, 1207-1215.
- Covassin, T., Elbin, R. J., & Nakayama, Y. (2010). Tracking neurocognitive performance following concussion in high school athletes. *Physician and Sportsmedicine*, 38, 87-93.
- Covassin, T., Elbin, R., Kontos, A., & Larson, E. (2010). Investigating baseline neurocognitive performance between male and female athletes with a history of multiple concussion. *Journal of Neurology, Neurosurgery & Psychiatry*, 81, 597-601.
- Covassin, T., Moran, R., & Wilhelm, K. (2013). Concussion symptoms and neurocognitive performance of high school and college athletes who incur multiple concussions. *The American journal of sports medicine*, 41, 2885-2889.
- Covassin, T., Stearne, D., & Elbin III, R. (2008). Concussion history and postconcussion neurocognitive performance and symptoms in collegiate athletes. *Journal of Athletic Training*, 43, 119-124.

- Covassin, T., Swanik, C. B., Sachs, M., Kendrick, Z., Schatz, P., Zillmer, E., & Kaminaris, C. (2006). Sex differences in baseline neuropsychological function and concussion symptoms of collegiate athletes. *British Journal of Sports Medicine*, *40*(11), 923-927.
- Crisco, J. J., Fiore, R., Beckwith, J. G., Chu, J. J., Brolinson, P. G., Duma, S., ... & Greenwald, R. M. (2010). Frequency and location of head impact exposures in individual collegiate football players. *Journal of Athletic Training*, *45*, 549-559.
- Crisco, J. J., Wilcox, B. J., Beckwith, J. G., Chu, J. J., Duhaime, A. C., Rowson, S., ... & Greenwald, R. M. (2011). Head impact exposure in collegiate football players. *Journal of Biomechanics*, *44*, 2673-2678.
- Crisco, J. J., Wilcox, B. J., Machan, J. T., McAllister, T. W., Duhaime, A. C., Duma, S. M., ... & Greenwald, R. M. (2012). Magnitude of head impact exposures in individual collegiate football players. *Journal of Applied Biomechanics*, *28*, 174-183.
- Croatto, P. (2014, October 25). *A year later, trying to comprehend a young hockey player's suicide*. Retrieved from The New York Times: <http://www.nytimes.com/2014/10/26/sports/hockey/a-year-later-trying-to-comprehend-a-young-hockey-players-suicide.html>.
- Cusimano, M. D., Chipman, M. L., Volpe, R., & Donnelly, P. (2009). Canadian minor hockey participants' knowledge about concussion. *Canadian Journal of Neurological Sciences/Journal Canadien des Sciences Neurologiques*, *36*, 315-320.
- Cywes, S. (1990). The neglected disease of modern society and the Child Accident Prevention Foundation of Southern Africa. *South African Medical Journal*, *78*, 381.
- Daniel, R. W., Rowson, S., & Duma, S. M. (2012). Head impact exposure in youth football. *Annals of Biomedical Engineering*, *40*, 976-981.

- Dashnaw, M. L., Petraglia, A. L., & Bailes, J. E. (2012). An overview of the basic science of concussion and subconcussion: Where we are and where we are going. *Neurosurgical Focus*, 33, E5: 1-9.
- Datalys Center for Sports Injury Research and Prevention. (2013). *NATA NATION preliminary concussion rates, 2010–2012*. Indianapolis, IN: Institute of Medicine-National Research Council.
- Davis, D. P., Serrano, J. A., Vilke, G. M., Sise, M. J., Kennedy, F., Eastman, A. B., ... & Hoyt, D. B. (2006). The predictive value of field versus arrival Glasgow Coma Scale score and TRISS calculations in moderate-to-severe traumatic brain injury. *Journal of Trauma and Acute Care Surgery*, 60, 985-990.
- Dean, P. J. A., & Sterr, A. (2013). Long-term effects of mild traumatic brain injury on cognitive performance. *Frontiers in Human Neuroscience*, 7, 1-11.
- Delaney, J. S., Puni, V., & Rouah, F. (2006). Mechanisms of injury for concussions in university football, ice hockey, and soccer: a pilot study. *Clinical Journal of Sport Medicine*, 16, 162-165.
- Devilly, G. J. (2004). *The Reliable Change Generator for Window version 2.0 [computer programme]*. The Centre for Neuropsychology, Swinburne University, Australia.
- Dimou, S., & Lagopoulos, J. (2014). Toward objective markers of concussion in sport: a review of white matter and neurometabolic changes in the brain after sports-related concussion. *Journal of Neurotrauma*, 31, 413-424.
- Dodrill, C. B., & Warner, M. H. (1988). Further studies of the Wonderlic Personnel Test as a brief measure of intelligence. *Journal of Consulting and Clinical Psychology*, 56, 145-147.

- Dozois, D. J., Dobson, K. S., & Ahnberg, J. L. (1998). A psychometric evaluation of the Beck Depression Inventory-II. *Psychological Assessment, 10*, 83-89.
- Echemendia, R. J., Putukian, M., Mackin, R. S., Julian, L., & Shoss, N. (2001). Neuropsychological test performance prior to and following sports-related mild traumatic brain injury. *Clinical Journal of Sport Medicine, 11*, 23-31.
- Echlin, P. S., Skopelja, E. N., Worsley, R., Dadachanji, S. B., Lloyd-Smith, D. R., Taunton, J. A., ... & Johnson, A. M. (2012). A prospective study of physician-observed concussion during a varsity university ice hockey season: Incidence and neuropsychological changes. Part 2 of 4. *Neurosurgical Focus, 33*, E2: 1-11.
- Eckner, J. T., Kutcher, J. S., Broglio, S. P., & Richardson, J. K. (2014). Effect of sport-related concussion on clinically measured simple reaction time. *British Journal of Sports Medicine, 48*, 112-118.
- Eckner, J. T., Sabin, M., Kutcher, J. S., & Broglio, S. P. (2011). No evidence for a cumulative impact effect on concussion injury threshold. *Journal of Neurotrauma, 28*, 2079-2090.
- Ellis, M. J., Cordingley, D., Vis, S., Reimer, K., Leiter, J., & Russell, K. (2015). Vestibulo-ocular dysfunction in pediatric sports-related concussion. *Journal of Neurosurgery: Pediatrics, 16*, 248-255.
- Erez, A. B. H., Rothschild, E., Katz, N., Tuchner, M., & Hartman-Maeir, A. (2009). Executive functioning, awareness, and participation in daily life after mild traumatic brain injury: A preliminary study. *The American Journal of Occupational Therapy, 63*, 634-640.

- Fazio, V. C., Lovell, M. R., Pardini, J. E., & Collins, M. W. (2007). The relation between post concussion symptoms and neurocognitive performance in concussed athletes. *NeuroRehabilitation, 22*, 207-216.
- Feigin, V. L., Theadom, A., Barker-Collo, S., Starkey, N. J., McPherson, K., Kahan, M., ... & Jones, K. (2013). Incidence of traumatic brain injury in New Zealand: A population-based study. *The Lancet Neurology, 12*, 53-64.
- Fleming, M. F., Barry, K. L., & Macdonald, R. (1991). The alcohol use disorders identification test (AUDIT) in a college sample. *International Journal of the Addictions, 26*, 1173-1185.
- Ford, J. H., Giovanello, K. S., & Guskiewicz, K. M. (2013). Episodic memory in former professional football players with a history of concussion: An event-related functional neuroimaging study. *Journal of Neurotrauma, 30*, 1683-1701.
- Fréchède, B., & McIntosh, A. S. (2009). Numerical reconstruction of real-life concussive football impacts. *Medicine and Science in Sports and Exercise, 41*, 390-398.
- Frenchman K. A., 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 Experimental Neuropsychology, 27*, 334-351.
- Gandy, S., & DeKosky, S. T. (2012). APOE ε4 status and traumatic brain injury on the gridiron or the battlefield. *Science Translational Medicine, 4*, 134ed4.
- Gardner, A. J., Iverson, G. L., Huw Williams, W., Baker, S., & Stanwell, P. (2014). A systematic review and meta-analysis of concussion in rugby union. *Sports Medicine, 44*, 1717-1731.

- Gardner, A., Shores, E. A., & Batchelor, J. (2010). Reduced processing speed in rugby union players reporting three or more previous concussions. *Archives of Clinical Neuropsychology*, *25*, 174-181.
- Gavett, B. E., Stern, R. A., & McKee, A. C. (2011). Chronic Traumatic Encephalopathy: A potential late effect of sport-related concussive and subconcussive head trauma. *Clinics in Sports Medicine*, *30*, 179-188.
- Geddes, J. F., Vowles, G. H., Nicoll, J. A. R., & Révész, T. (1999). Neuronal cytoskeletal changes are an early consequence of repetitive head injury. *Acta Neuropathologica*, *98*, 171-178.
- Gessel, L. M., Fields, S. K., Collins, C. L., Dick, R. W., & Comstock, R. D. (2007). Concussions among United States high school and collegiate athletes. *Journal of Athletic Training*, *42*, 495-503.
- Giza, C. C., & Hovda, D. A. (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training*, *36*, 228-235.
- Giza, C. C., Kutcher, J. S., Ashwal, S., Barth, J., Getchius, T. S., Gioia, G. A., . . . Zafonte, R. (2013). Summary of evidence-based guideline update: Evaluation and management of concussion in sports. *Neurology*, *80*, 2250 –2257.
- Gregory, S. (2010, January 28). The problem with Football: How to make it safer. Time. <http://content.time.com/time/magazine/article/0,9171,1957459,00.html>.
- Grossman, E. J., Jensen, J. H., Babb, J. S., Chen, Q., Tabesh, A., Fieremans, E., ... & Grossman, R. I. (2013). Cognitive impairment in mild traumatic brain injury: a longitudinal diffusional kurtosis and perfusion imaging study. *American Journal of Neuroradiology*, *34*, 951-957.

- Guinness, R. (2015, March 19). Randwick rugby get the gallop on pioneering concussion study. *The Sydney Morning Herald*. Retrieved from <http://www.smh.com.au/rugby-union/union-news/randwick-rugby-get-the-gallop-on-pioneering-concussion-study-20150319-1m376p.html>.
- Guskiewicz, K. M., McCrea, M., Marshall, S. W., Cantu, R. C., Randolph, C., Barr, W., ... & Kelly, J. P. (2003). Cumulative effects associated with recurrent concussion in collegiate football players: The NCAA Concussion Study. *The Journal of the American Medical Association*, *290*, 2549-2555.
- Guskiewicz, K. M., Mihalik, J. P., Shankar, V., Marshall, S. W., Crowell, D. H., Oliaro, S. M., ... & Hooker, D. N. (2007). Measurement of head impacts in collegiate football players: Relationship between head impact biomechanics and acute clinical outcome after concussion. *Neurosurgery*, *61*, 1244-1253.
- Gysland, S. M., Mihalik, J. P., Register-Mihalik, J. K., Trulock, S. C., Shields, E. W., & Guskiewicz, K. M. (2012). The relationship between subconcussive impacts and concussion history on clinical measures of neurologic function in collegiate football players. *Annals of Biomedical Engineering*, *40*, 14-22.
- Haines, D. E. (2013). *Fundamental neuroscience for basic and clinical applications, Fourth Edition*. Philadelphia, PA: Elsevier.
- Haneef, Z., Levin, H. S., Frost Jr, J. D., & Mizrahi, E. M. (2013). Electroencephalography and quantitative electroencephalography in mild traumatic brain injury. *Journal of Neurotrauma*, *30*, 653-656.
- Hanlon, E. M., & Bir, C. A. (2012). Real-time head acceleration measurement in girls' youth soccer. *Medicine and Science in Sports and Exercise*, *44*, 1102-1108.

- Harpham, J. A., Mihalik, J. P., Littleton, A. C., Frank, B. S., & Guskiewicz, K. M. (2014). The effect of visual and sensory performance on head impact biomechanics in college football players. *Annals of Biomedical Engineering*, *42*, 1-10.
- Harris, A. W., Voaklander, D. C., Jones, C. A., & Rowe, B. H. (2012). Time-to-subsequent head injury from sports and recreation activities. *Clinical Journal of Sport Medicine*, *22*, 91-97.
- Hart, J., Kraut, M. A., Womack, K. B., Strain, J., Didehbani, N., Bartz, E., ... & Cullum, C. M. (2013). Neuroimaging of cognitive dysfunction and depression in aging retired National Football League players: A cross-sectional study. *The Journal of the American Medical Association, Neurology*, *70*, 326-335.
- Heitger, M. H., Jones, R. D., Dalrymple-Alford, J. C., Frampton, C. M., Ardagh, M. W., & Anderson, T. J. (2006). Motor deficits and recovery during the first year following mild closed head injury. *Brain Injury*, *20*, 807-824.
- Henry, L. C., Elbin, R. J., Collins, M. W., Marchetti, G., & Kontos, A. P. (2016b). Examining recovery trajectories after sport-related concussion with a multimodal clinical assessment approach. *Neurosurgery*, *78*, 232-241.
- Henry, L. C., Tremblay, J., Tremblay, S., Lee, A., Brun, C., Lepore, N., ... & Lassonde, M. (2011). Acute and chronic changes in diffusivity measures after sports concussion. *Journal of Neurotrauma*, *28*, 2049-2059.
- Henry, L. C., Tremblay, S., & De Beaumont, L. (2016a). Long-term effects of sports concussions bridging the neurocognitive repercussions of the injury with the newest neuroimaging data. *The Neuroscientist*. DOI: 10.1177/1073858416651034.

- Hill, B. D., Womble, M. N., & Rohling, M. L. (2015). Logistic regression function for detection of suspicious performance during baseline evaluations using concussion vital signs. *Applied Neuropsychology: Adult, 22*, 233-240.
- Hollis, S. J., Stevenson, M. R., McIntosh, A. S., Li, L., Heritier, S., Shores, E. A., ... & Finch, C. F. (2011). Mild traumatic brain injury among a cohort of rugby union players: Predictors of time to injury. *British Journal of Sports Medicine, 45*, 997-999.
- Holm, L., Cassidy, J. D., Carroll, L., & Borg, J. (2005). Summary of the WHO collaborating centre for neurotrauma task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, 37*, 137-141.
- Hootman, J. M., Dick, R., & Agel, J. (2007). Epidemiology of collegiate injuries for 15 sports: Summary and recommendations for injury prevention initiatives. *Journal of Athletic Training, 42*, 311-319.
- Houck, Z., Asken, B., Bauer, R., Pothast, J., Michaudet, C., & Clugston, J. (2016). Epidemiology of sport-related concussion in an NCAA Division I Football Bowl Subdivision sample. *The American Journal of Sports Medicine*, DOI: 10.1177/0363546516645070.
- Iverson, G. L., Brooks, B. L., Lovell, M. R., & Collins, M. W. (2006). No cumulative effects for one or two previous concussions. *British Journal of Sports Medicine, 40*, 72-75.
- Iverson, G. L., Echemendia, R. J., LaMarre, A. K., Brooks, B. L., & Gaetz, M. B. (2012). Possible lingering effects of multiple past concussions. *Rehabilitation Research and Practice, 2012*, 1-12.
- Iverson, G. L., Gaetz, M., Lovell, M. R., & Collins, M. W. (2004). Cumulative effects of concussion in amateur athletes. *Brain Injury, 18*, 433-443.

- Iverson, G. L., Sawyer, D. C., McCracken, L. M., & Kozora, E. (2001). Assessing depression in systemic lupus erythematosus: Determining reliable change. *Lupus, 10*, 266-271.
- Jacobson, N. S., & Truaz, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59*, 12-19.
- Jennett, B., & Teasdale, G. (1981). *Management of Head Injuries*. Philadelphia, PA: FA Davis.
- Johnson, J. A., Lee, A., Vinson, D., & Seale, J. P. (2013). Use of AUDIT-based measures to identify unhealthy alcohol use and alcohol dependence in primary Care: A validation study. *Alcoholism: Clinical and Experimental Research, 37*(s1), E253-E259.
- Jordan, B. D. (1987). Neurologic aspects of boxing. *Archives of neurology, 44*, 453-459.
- Jordan, B. D. (2013). The clinical spectrum of sport-related traumatic brain injury. *Nature Reviews Neurology, 9*, 222-230.
- Karr, J. E., Areshenkoff, C. N., & Garcia-Barrera, M. A. (2014). The neuropsychological outcomes of concussion: A systematic review of meta-analyses on the cognitive sequelae of mild traumatic brain injury. *Neuropsychology, 28*, 321-336.
- Keightley, M. L., Singh Saluja, R., Chen, J. K., Gagnon, I., Leonard, G., Petrides, M., & Ptito, A. (2014). A functional magnetic resonance imaging study of working memory in youth after sports-related concussion: Is it still working? *Journal of Neurotrauma, 31*, 437-451.
- King, D., Hume, P. A., Brughelli, M., & Gissane, C. (2014). Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches. *The American Journal of Sports Medicine, 43*, 614-624.

- Kinnunen, K. M., Greenwood, R., Powell, J. H., Leech, R., Hawkins, P. C., Bonnelle, V., ... & Sharp, D. J. (2011). White matter damage and cognitive impairment after traumatic brain injury. *Brain*, 347, 1-15.
- Kontos, A. P., Elbin, R. J., Lau, B., Simensky, S., Freund, B., French, J., & Collins, M. W. (2013). Posttraumatic migraine as a predictor of recovery and cognitive impairment after sport-related concussion. *The American Journal of Sports Medicine*, Vol. XX, No. X DOI: 10.1177/0363546513488751.
- Kristman, V. L., Borg, J., Godbolt, A. K., Salmi, L. R., Cancelliere, C., Carroll, L. J., ... & Donovan, J. (2014). Methodological issues and research recommendations for prognosis after mild traumatic brain injury: Results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of Physical Medicine and Rehabilitation*, 95, S265-S277.
- Landre, N., Poppe, C. J., Davis, N., Schmaus, B., & Hobbs, S. E. (2006). Cognitive functioning and postconcussive symptoms in trauma patients with and without mild TBI. *Archives of Clinical Neuropsychology*, 21, 255-273.
- Langlois, J. A., & Sattin, R. W. (2005). Traumatic brain injury in the United States. *Journal of Head Trauma Rehabilitation*, 20, 187-188.
- Lau, B. C., Collins, M. W., & Lovell, M. R. (2011). Sensitivity and specificity of subacute computerized neurocognitive testing and symptom evaluation in predicting outcomes after sports-related concussion. *The American Journal of Sports Medicine*, 39, 1209-1216.
- Lau, B. C., Kontos, A. P., Collins, M. W., Mucha, A., & Lovell, M. R. (2011). Which on-field signs/symptoms predict protracted recovery from sport-related concussion

- among high school football players? *The American Journal of Sports Medicine*, *39*, 2311-2318.
- Lau, B., Lovell, M. R., Collins, M. W., & Pardini, J. (2009). Neurocognitive and symptom predictors of recovery in high school athletes. *Clinical Journal of Sport Medicine*, *19*, 216-221.
- Lawson, J. S., & Evans, A. R. (1992). Prodigious alcohol consumption by Australian rugby league footballers. *Drug and Alcohol Review*, *11*, 193-195.
- Lehman, E. J., Hein, M. J., Baron, S. L., & Gersic, C. M. (2012). Neurodegenerative causes of death among retired National Football League players. *Neurology*, *79*, 1970-1974.
- Levin, H. S., & Diaz-Arrastia, R. R. (2015). Diagnosis, prognosis, and clinical management of mild traumatic brain injury. *The Lancet Neurology*, *14*, 506-517.
- Levin, K. (2004). Paediatric traumatic brain injury in South Africa: Some thoughts and considerations. *Disability and Rehabilitation*, *26*, 306-314.
- Lovell, M. R., Collins, M. W., Iverson, G. L., Field, M., Maroon, J. C., Cantu, R., ... & Fu, F. H. (2003). Recovery from mild concussion in high school athletes. *Journal of Neurosurgery*, *98*, 296-301.
- Lovell, M. R., Collins, M. W., Podell, K., Powell, J., & Maroon, J. (2000). *ImPACT: Immediate post-concussion assessment and cognitive testing*. Pittsburgh, PA: NeuroHealth Systems, LLC.
- Lovell, M. R., Iverson, G. L., Collins, M. W., Podell, K., Johnston, K. M., Pardini, D., ... & Maroon, J. C. (2006). Measurement of symptoms following sports-related concussion: Reliability and normative data for the post-concussion scale. *Applied Neuropsychology*, *13*, 166-174.

- Lovell, M. R., Pardini, J. E., Welling, J., Collins, M. W., Bakal, J., Lazar, N., ... & Becker, J. T. (2007). Functional brain abnormalities are related to clinical recovery and time to return-to-play in athletes. *Neurosurgery*, *61*, 352-360.
- Lundin, A., Hallgren, M., Balliu, N., & Forsell, Y. (2015). The use of alcohol use disorders identification test (AUDIT) in detecting alcohol use disorder and risk drinking in the general population: Validation of AUDIT using schedules for clinical assessment in neuropsychiatry. *Alcoholism: Clinical and Experimental Research*, *39*, 158-165.
- Maas, A. I., Stocchetti, N., & Bullock, R. (2008). Moderate and severe traumatic brain injury in adults. *The Lancet Neurology*, *7*, 728-741.
- Maerlender, A., Flashman, L., Kessler, A., Kumbhani, S., Greenwald, R., Tosteson, T., & McAllister, T. (2013). Discriminant construct validity of ImPACT™: A companion study. *The Clinical Neuropsychologist*, *27*, 290-299.
- Majerske, C. W., Mihalik, J. P., Ren, D., Collins, M. W., Reddy, C. C., Lovell, M. R., & Wagner, A. K. (2008). Concussion in sports: Postconcussive activity levels, symptoms, and neurocognitive performance. *Journal of Athletic Training*, *43*, 265–274.
- Makdissi, M., Cantu, R. C., Johnston, K. M., McCrory, P., & Meeuwisse, W. H. (2013a). The difficult concussion patient: What is the best approach to investigation and management of persistent (> 10 days) postconcussive symptoms? *British Journal of Sports Medicine*, *47*, 308-313.
- Makdissi, M., Darby, D., Maruff, P., Ugoni, A., Brukner, P., & McCrory, P. R. (2010). Natural history of concussion in sport markers of severity and implications for management. *The American Journal of Sports Medicine*, *38*, 464-471.

- Makdissi, M., Davis, G., Jordan, B., Patricios, J., Purcell, L., & Putukian, M. (2013b). Revisiting the modifiers: How should the evaluation and management of acute concussions differ in specific groups? *British Journal of Sports Medicine*, *47*, 314-320.
- Malec, J. F., Brown, A. W., Leibson, C. L., Flaada, J. T., Mandrekar, J. N., Diehl, N. N., & Perkins, P. K. (2007). The Mayo classification system for traumatic brain injury severity. *Journal of Neurotrauma*, *24*, 1417-1424.
- Mannix, R., Iverson, G. L., Maxwell, B., Atkins, J. E., Zafonte, R., & Berkner, P. D. (2014). Multiple prior concussions are associated with symptoms in high school athletes. *Annals of Clinical and Translational Neurology*, *1*, 433-438.
- Mannix, R., Meehan, W. P., Mandeville, J., Grant, P. E., Gray, T., Berglass, J., ... & Peters, N. V. (2013). Clinical correlates in an experimental model of repetitive mild brain injury. *Annals of Neurology*, *74*, 65-75.
- Marshall, S. W., Guskiewicz, K. M., Shankar, V., McCrea, M., & Cantu, R. C. (2015). Epidemiology of sports-related concussion in seven US high school and collegiate sports. *Injury Epidemiology*, *2*, 1-10.
- Martini, D., Eckner, J., Kutcher, J., & Broglio, S. (2013). Subconcussive head impact biomechanics: Comparing differing offensive schemes. *Medicine and Science in Sports and Exercise*, *45*, 755-761.
- Maruta, J., Suh, M., Niogi, S. N., Mukherjee, P., & Ghajar, J. (2010). Visual tracking synchronization as a metric for concussion screening. *The Journal of Head Trauma Rehabilitation*, *25*, 293-305.

- Mathias, J. L., Beall, J. A., & Bigler, E. D. (2004). Neuropsychological and information processing deficits following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 10*, 286-297.
- McAllister, T. W., Flashman, L. A., Maerlender, A., Greenwald, R. M., Beckwith, J. G., Tosteson, T. D., ... & Grove, M. R. (2012). Cognitive effects of one season of head impacts in a cohort of collegiate contact sport athletes. *Neurology, 78*, 1777-1784.
- McAllister, T. W., Flashman, L. A., Sparling, M. B., & Saykin, A. J. (2004). Working memory deficits after traumatic brain injury: Catecholaminergic mechanisms and prospects for treatment—a review. *Brain Injury, 18*, 331-350.
- McCauley, S. R., Wilde, E. A., Barnes, A., Hanten, G., Hunter, J. V., Levin, H. S., & Smith, D. H. (2014). Patterns of early emotional and neuropsychological sequelae after mild traumatic brain injury. *Journal of Neurotrauma, 31*, 914-925.
- McClinchy, M. P., Lovell, M. R., Pardini, J., Collins, M. W., & Spore, M. K. (2006). Recovery from sports concussion in high school and collegiate athletes. *Brain Injury, 20*, 33-39.
- McCrea, M. (2008). *Mild traumatic brain injury and postconcussion syndrome*. New York, NY: Oxford University Press.
- McCrea, M., & Powell, M. R. (2012). Biomechanics and pathophysiology of concussion. In J. N Apps & K. D. Walter (Eds.), *Pediatric and Adolescent Concussion: Diagnosis, Management and Outcomes* (pp. 29-53). New York, NY: Springer. ..
- McCrea, M., Barr, W. B., Guskiewicz, K., Randolph, C., Marshall, S. W., Cantu, R., ... & Kelly, J. P. (2005). Standard regression-based methods for measuring recovery after sport-related concussion. *Journal of the International Neuropsychological Society, 11*, 58-69.

- McCrea, M., Guskiewicz, K. M., Marshall, S. W., Barr, W., Randolph, C., Cantu, R. C., ... & Kelly, J. P. (2003). Acute effects and recovery time following concussion in collegiate football players: The NCAA Concussion Study. *The Journal of the American Medical Association, 290*, 2556-2563.
- McCrea, M., Guskiewicz, K., Randolph, C., Barr, W. B., Hammeke, T. A., Marshall, S. W., ... & Kelly, J. P. (2013). Incidence, clinical course, and predictors of prolonged recovery time following sport-related concussion in high school and college athletes. *Journal of the International Neuropsychological Society, 19*, 22-33.
- McCrea, M., Hammeke, T., Olsen, G., Leo, P., & Guskiewicz, K. (2004). Unreported concussion in high school football players: Implications for prevention. *Clinical Journal of Sport Medicine, 14*, 13-17.
- McCrea, M., Randolph, C., & Kelly, J. P. (2000). *Standardized Assessment of Concussion (SAC): Manual for administration, scoring and interpretation*. Waukesha, WI: CNS.
- McCrory, P., Meeuwisse, W. H., Aubry, M., Cantu, B., Dvorak, J., Echemendia, R. J., ... & Sills, A. (2013b). SCAT3. *British Journal of Sports Medicine, 47*, 259-262.
- McCrory, P., Meeuwisse, W. H., Aubry, M., Cantu, B., Dvorak, J., Echemendia, R. J., ... & Turner, M. (2013a). Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012. *British Journal of Sports Medicine, 47*, 250-258.
- McCrory, P., Meeuwisse, W., Johnston, K., Dvorak, J., Aubry, M., Molloy, M., & Cantu, R. (2009). Consensus statement on concussion in sport – the 3rd International Conference on Concussion in Sport held in Zurich, November 2008. *South African Journal of Sports Medicine, 21*, 36-46.

- McKee, A. C., Cantu, R. C., Nowinski, C. J., Hedley-Whyte, E. T., Gavett, B. E., Budson, A. E., ... & Stern, R. A. (2009). Chronic traumatic encephalopathy in athletes: Progressive tauopathy after repetitive head injury. *Journal of Neuropathology & Experimental Neurology*, *68*, 709-735.
- McKee, A. C., Daneshvar, D. H., Alvarez, V. E., & Stein, T. D. (2014). The neuropathology of sport. *Acta Neuropathologica*, *127*, 29-51.
- McKee, A. C., Stein, T. D., Nowinski, C. J., Stern, R. A., Daneshvar, D. H., Alvarez, V. E., ... & Riley, D. O. (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain*, *136*, 43-64.
- Meaney D. F., Smith D. H., Shreiber D. I., Bain A. C., Miller R. T., Ross D. T., & Gennarelli T. A. (1995). Biomechanical analysis of experimental diffuse axonal injury. *Journal of Neurotrauma* *12*, 689–694.
- Meares, S., Shores, E. A., Taylor, A. J., Batchelor, J., Bryant, R. A., Baguley, I. J., ... & Marosszeky, J. E. (2011). The prospective course of postconcussion syndrome: The role of mild traumatic brain injury. *Neuropsychology*, *25*, 454-465.
- Meehan III, W. P., Zhang, J., Mannix, R., & Whalen, M. J. (2012). Increasing recovery time between injuries improves cognitive outcome after repetitive mild concussive brain injuries in mice. *Neurosurgery*, *71*, 885-892.
- Meehan, W. P., & Bachur, R. G. (2009). Sport-related concussion. *Pediatrics*, *123*, 114-123.
- Meehan, W. P., Mannix, R. C., Stracciolini, A., Elbin, R. J., & Collins, M. W. (2013). Symptom severity predicts prolonged recovery after sport-related concussion, but age and amnesia do not. *The Journal of Pediatrics*, *163*, 721-725.

- Meier, T. B., Savitz, J., Singh, R., Teague, T. K., & Bellgowan, P. S. (2016). Smaller dentate gyrus and CA2 and CA3 volumes are associated with kynurenine metabolites in collegiate football athletes. *Journal of Neurotrauma*, *22*, 1349-1357.
- Menon, D. K., Schwab, K., Wright, D. W., & Maas, A. I. (2010). Position statement: Definition of traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *91*, 1637-1640.
- Messé, A., Caplain, S., Paradot, G., Garrigue, D., Mineo, J. F., Soto Ares, G., ... & Péligrini-Issac, M. (2011). Diffusion tensor imaging and white matter lesions at the subacute stage in mild traumatic brain injury with persistent neurobehavioral impairment. *Human Brain Mapping*, *32*, 999-1011.
- Mihalik, J. P., Guskiewicz, K. M., Marshall, S. W., Blackburn, J. T., Cantu, R. C., & Greenwald, R. M. (2012). Head impact biomechanics in youth hockey: Comparisons across playing position, event types, and impact locations. *Annals of Biomedical Engineering*, *40*, 141-149.
- Miller, J. H., Gill, C., Kuhn, E. N., Rocque, B. G., Menendez, J. Y., O'Neill, J. A., ... & Ferguson, D. (2016). Predictors of delayed recovery following pediatric sports-related concussion: A case-control study. *Journal of Neurosurgery: Pediatrics*, *17*, 491-496.
- Miller, J. R., Adamson, G. J., Pink, M. M., & Sweet, J. C. (2007). Comparison of preseason, midseason, and postseason neurocognitive scores in uninjured collegiate football players. *The American Journal of Sports Medicine*, *35*, 1284-1288.
- Moser, R. S., Schatz, P., & Jordan, B. D. (2005). Prolonged effects of concussion in high school athletes. *Neurosurgery*, *57*, 300-306.

- Myer, L., Smit, J., Le Roux, L., Parker, S., Stein, D. J., & Seedat, S. (2008). Common mental disorders among HIV-infected individuals in South Africa: Prevalence, predictors, and validation of brief psychiatric rating scales. *AIDS Patient Care and STDs*, *22*, 147-158.
- Nakayama, Y., Covassin, T., Schatz, P., Nogle, S., & Kovan, J. (2014). Examination of the test-retest reliability of a computerized neurocognitive test battery. *The American Journal of Sports Medicine*, *42*, 2000-2005
- Namjoshi, D. R., Good, C., Cheng, W. H., Panenka, W., Richards, D., Cripton, P. A., & Wellington, C. L. (2013). Towards clinical management of traumatic brain injury: A review of models and mechanisms from a biomechanical perspective. *Disease Models and Mechanisms*, *6*, 1325-1338.
- National Center for Injury Prevention and Control. (2003). *Report to Congress on Mild Traumatic Brain Injury in the United States: Steps to Prevent a Serious Public Health Problem*. Atlanta, GA: Centers for Disease Control and Prevention.
- Nelson, L. D., Guskiewicz, K. M., Barr, W. B., Hammeke, T. A., Randolph, C., Ahn, K. W., ... & McCrea, M. A. (2016). Age differences in recovery after sport-related concussion: a comparison of high school and collegiate athletes. *Journal of Athletic Training*, *51*, 142-152.
- Ng, T. P., Bussone, W. R., & Duma, S. M. (2006). The effect of gender and body size on linear accelerations of the head observed during daily activities. *Biomedical Sciences Instrumentation*, *42*, 25-30.
- Niendam, T. A., Laird, A. R., Ray, K. L., Dean, Y. M., Glahn, D. C., & Carter, C. S. (2012). Meta-analytic evidence for a superordinate cognitive control network subserving

- diverse executive functions. *Cognitive, Affective, & Behavioral Neuroscience*, 12, 241-268.
- Ocwieja, K. E., Mihalik, J. P., Marshall, S. W., Schmidt, J. D., Trulock, S. C., & Guskiewicz, K. M. (2012). The effect of play type and collision closing distance on head impact biomechanics. *Annals of Biomedical Engineering*, 40, 90-96.
- Oppenheimer, D. R. (1968). Microscopic lesions in the brain following head injury. *Journal of Neurology, Neurosurgery & Psychiatry*, 31, 299-306.
- Orr, C. A., Albaugh, M. D., Watts, R., Garavan, H., Andrews, T., Nickerson, J. P., ... & Hudziak, J. J. (2016). Neuroimaging biomarkers of a history of concussion observed in asymptomatic young athletes. *Journal of Neurotrauma*, 33, 803-810.
- Patel, A. V., Mihalik, J. P., Notebaert, A. J., Guskiewicz, K. M., & Prentice, W. E. (2007). Neuropsychological performance, postural stability, and symptoms after dehydration. *Journal of Athletic Training*, 42, 66-75.
- Patton, D. A., McIntosh, A. S., Kleiven, S., & Fréchède, B. (2012). Injury data from unhelmeted football head impacts evaluated against critical strain tolerance curves. *Proceedings of the Institution of Mechanical Engineers, Part P: Journal of Sports Engineering and Technology*, 226, 177-184.
- Pearce, A. J., Hoy, K., Rogers, M. A., Corp, D. T., Davies, C. B., Maller, J. J., & Fitzgerald, P. B. (2015). Acute motor, neurocognitive and neurophysiological change following concussion injury in Australian amateur football. A prospective multimodal investigation. *Journal of Science and Medicine in Sport*, 18, 500-506.
- Pearce, A. J., Hoy, K., Rogers, M. A., Corp, D. T., Maller, J. J., Drury, H. G., & Fitzgerald, P. B. (2014). The long-term effects of sports concussion on retired Australian football

- players: A study using transcranial magnetic stimulation. *Journal of Neurotrauma*, 31, 1139-1145.
- Pellman, E. J., Viano, D. C., Tucker, A. M., Casson, I. R., & Waeckerle, J. F. (2003). Concussion in professional football: Reconstruction of game impacts and injuries. *Neurosurgery*, 53, 799-814.
- Petraglia, A. L., Maroon, J. C., & Bailes, J. E. (2012). From the field of play to the field of combat: A review of the pharmacological management of concussion. *Neurosurgery*, 70, 1520-1533.
- Potgieter, S., Visser, J., Croukamp, I., Markides, M., Nascimento, J., & Scott, K. (2014). Body composition and habitual and match-day dietary intake of the FNB Maties Varsity Cup rugby players. *South African Journal of Sports Medicine*, 26, 35-43.
- Povlishock, J. T. (1992). Traumatically induced axonal injury: Pathogenesis and pathobiological implications. *Brain Pathology (Zurich, Switzerland)*, 2, 1-12.
- Prichep, L. S., McCrea, M., Barr, W., Powell, M., & Chabot, R. J. (2013). Time course of clinical and electrophysiological recovery after sport-related concussion. *The Journal of Head Trauma Rehabilitation*, 28, 266-273.
- Quarrie, K. L., Feehan, M., Waller, A. E., Cooke, K. R., Williams, S., & McGee, R. (1996). The land rugby injury and performance project: Alcohol use patterns within a cohort of rugby players. *Addiction*, 91, 1865-1868.
- Randolph, C., Karantzoulis, S., & Guskiewicz, K. (2013). Prevalence and characterization of mild cognitive impairment in retired national football league players. *Journal of the International Neuropsychological Society*, 19, 873-880.

- Randolph, C., Millis, S., Barr, W. B., McCrea, M., Guskiewicz, K. M., Hammeke, T. A., & Kelly, J. P. (2009). Concussion symptom inventory: An empirically derived scale for monitoring resolution of symptoms following sport-related concussion. *Archives of Clinical Neuropsychology, 24*, 219-229.
- Reynolds, B. B., Patrie, J., Henry, E. J., Goodkin, H. P., Broshek, D. K., Wintermark, M., & Druzgal, T. J. (2016). Practice type effects on head impact in collegiate football. *Journal of Neurosurgery, 124*, 501-510.
- Rohling, M. L., Binder, L. M., Demakis, G. J., Larrabee, G. J., Ploetz, D. M., & Langhinrichsen-Rohling, J. (2011). A meta-analysis of neuropsychological outcome after mild traumatic brain injury: Re-analyses and considerations of Binder et al. (1997), Frenchman et al. (2005), and Pertab et al. (2009). *The Clinical Neuropsychologist, 25*, 608-623.
- Roozenbeek, B., Maas, A. I., & Menon, D. K. (2013). Changing patterns in the epidemiology of traumatic brain injury. *Nature Reviews Neurology, 9*, 231-236.
- Rossingh, D., & Kitamura, M. (2015, March 20). Rugby is having an NFL moment as concussions bring legal scrutiny. *Bloomberg*. Retrieved from <http://www.bloomberg.com/news/articles/2015-03-20/rugby-is-having-an-nfl-moment-as-concussions-bring-legal-scrutiny>.
- Rowson, S., Brolinson, G., Goforth, M., Dietter, D., & Duma, S. (2009). Linear and angular head acceleration measurements in collegiate football. *Journal of Biomechanical Engineering, 131*, 10-16.

- Rowson, S., Duma, S. M., Beckwith, J. G., Chu, J. J., Greenwald, R. M., Crisco, J. J., ... & Maerlender, A. C. (2012). Rotational head kinematics in football impacts: an injury risk function for concussion. *Annals of Biomedical Engineering*, *40*, 1-13.
- Saunders, J. B., Aasland, O. G., Babor, T. F., De la Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption-II. *Addiction*, *88*, 791-804.
- Schatz, P. (2010). Long-term test-retest reliability of baseline cognitive assessments using ImPACT. *The American Journal of Sports Medicine*, *38*, 47-53.
- Schatz, P., & Ferris, C. S. (2013). One-month test-retest reliability of the ImPACT test battery. *Archives of Clinical Neuropsychology*, *28*, 499-504.
- Schatz, P., & Sandel, N. (2013). Sensitivity and specificity of the online version of ImPACT in high school and collegiate athletes. *The American Journal of Sports Medicine*, *41*, 321-326.
- Schatz, P., Pardini, J. E., Lovell, M. R., Collins, M. W., & Podell, K. (2006). Sensitivity and specificity of the ImPACT Test Battery for concussion in athletes. *Archives of Clinical Neuropsychology*, *21*(1), 91-99.
- Schnebel, B., Gwin, J. T., Anderson, S., & Gatlin, R. (2007). In vivo study of head impacts in football: A comparison of National Collegiate Athletic Association Division I versus high school impacts. *Neurosurgery*, *60*, 490-496.
- Schretlen, D. J., & Shapiro, A. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry*, *15*, 341-349.

- Schwarz, A. (2010, January 04). Congress examines N.F.L. concussions. *The New York Times*. Retrieved from <http://www.nytimes.com/2010/01/05/sports/football/05concussions.html>.
- Scopaz, K. A., & Hatzenbuehler, J. R. (2013). Risk modifiers for concussion and prolonged recovery. *Sports Health: A Multidisciplinary Approach*, DOI: 10.1177/1941738112473059.
- Sekulic, D., Bjelanovic, L., Pehar, M., Pelivan, K., & Zenic, N. (2014). Substance use and misuse and potential doping behaviour in rugby union players. *Research in Sports Medicine*, 22, 226-239.
- Seppä, K., Mäkelä, R., & Sillanaukee, P. (1995). Effectiveness of the Alcohol Use Disorders Identification Test in occupational health screenings. *Alcoholism: Clinical and Experimental Research*, 19, 999-1003.
- Seth, P., Glenshaw, M., Sabatier, J. H., Adams, R., Du Preez, V., DeLuca, N., & Bock, N. (2015). AUDIT, AUDIT-C, and AUDIT-3: Drinking patterns and screening for harmful, hazardous and dependent drinking in Katutura, Namibia. *PloS one*, 10, 1-16.
- Shibley, W. C., Gruber, C. P., Martin, T. A., & Klein, A. M. (2009). *Shibley-2 Manual*. Los Angeles: Western Psychological Services.
- Shum, D., Levin, H., & Chan, R. C. (2011). Prospective memory in patients with closed head injury: A review. *Neuropsychologia*, 49, 2156-2165.
- Shuttleworth-Edwards, A. B., & Radloff, S. E. (2008). Compromised visuomotor processing speed in players of Rugby Union from school through to the national adult level. *Archives of Clinical Neuropsychology*, 23, 511-520.

- Shuttleworth-Edwards, A. B., Border, M., Reid, I., & Radloff, S. (2004). South African Rugby Union. In M. R. Lovell, R. E. Echernendia, J. T. Barth, & M. W. Collins, *Traumatic brain injury in sports: An international neuropsychological perspective* (pp. 149-168). Lisse: Swets and Zeitlinger.
- Shuttleworth-Edwards, A. B., Noakes, T. D., Radloff, S. E., Whitefield, V. J., Clark, S. B., Roberts, C. O., ... & Mitchell, J. L. (2008). The comparative incidence of reported concussions presenting for follow-up management in South African Rugby Union. *Clinical Journal of Sport Medicine, 18*, 403-409.
- Shuttleworth-Edwards, A. B., Radloff, S. E., Whitefield-Alexander, V. J., Smith, I. P., & Horsman, M. (2014). Practice effects reveal visuomotor vulnerability in school and university rugby players. *Archives of Clinical Neuropsychology, 29*, 86-99.
- Shuttleworth-Edwards, A. B., Whitefield-Alexander, V. J., Radloff, S. E., Taylor, A. M., & Lovell, M. R. (2009). Computerized neuropsychological profiles of South African versus US athletes: A basis for commentary on cross-cultural norming issues in the sports concussion arena. *Physician and Sportsmedicine, 37*(4), 45-52.
- Skipsey, K., Burleson, J. A., & Kranzler, H. R. (1997). Utility of the AUDIT for identification of hazardous or harmful drinking in drug-dependent patients. *Drug and Alcohol Dependence, 45*, 157-163.
- Spiotta, A. M., Shin, J. H., Bartsch, A. J., & Benzel, E. C. (2011). Subconcussive impact in sports: A new era of awareness. *World Neurosurgery, 75*, 175-178.
- Stein, M. B., Ursano, R. J., Campbell-Sills, L., Colpe, L. J., Fullerton, C. S., Heeringa, S. G., ... & Jain, S. (2016). Prognostic indicators of persistent post-concussive symptoms

- after deployment-related mild traumatic brain injury: A prospective longitudinal study in US Army soldiers. *Journal of Neurotrauma*, 33, 1-8.
- Stein, S. C. (1996). Classification of head injury. In R. K. Narayan, J. T. Povlishock, & J. R. Wilberger (Eds.), *Neurotrauma* (pp. 31-42). New York: McGraw-Hill.
- Stein, T. D., Alvarez, V. E., & McKee, A. C. (2014). Chronic traumatic encephalopathy: A spectrum of neuropathological changes following repetitive brain trauma in athletes and military personnel. *Alzheimer's Research & Therapy*, 6, 1-11.
- Stephens, R., Rutherford, A., Potter, D., & Fernie, G. (2010). Neuropsychological consequence of soccer play in adolescent UK school team soccer players. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 22, 295-303.
- Stern, R. A., Riley, D. O., Daneshvar, D. H., Nowinski, C. J., Cantu, R. C., & McKee, A. C. (2011). Long-term consequences of repetitive brain trauma: Chronic traumatic encephalopathy. *American Academy of Physical Medicine and Rehabilitation*, 3, S460-S467.
- Stewart, W., McNamara, P. H., Lawlor, B., Hutchinson, S., & Farrell, M. (2015). Chronic traumatic encephalopathy: A potential late and under recognized consequence of rugby union? *QJM: An International Journal of Medicine*, 109, 11-15.
- Straume-Naesheim, T. M., Andersen, T. E., Dvorak, J., & Bahr, R. (2005). Effects of heading exposure and previous concussions on neuropsychological performance among Norwegian elite footballers. *British Journal of Sports Medicine*, 39, i70-i77.
- Swartz, E. E., Broglio, S. P., Cook, S. B., Cantu, R. C., Ferrara, M. S., Guskiewicz, K. M., & Myers, J. L. (2015). Early results of a helmetless-tackling intervention to decrease head impacts in football players. *Journal of Athletic Training*, 50, 1219-1222

- Tagliaferri, F., Compagnone, C., Korsic, M., Servadei, F., & Kraus, J. (2006). A systematic review of brain injury epidemiology in Europe. *Acta neurochirurgica*, *148*, 255-268.
- Talavage, T. M., Nauman, E. A., Breedlove, E. L., Yoruk, U., Dye, A. E., Morigaki, K. E., ... Leverenz, L. J. (2014). Functionally-detected cognitive impairment in High School football players without clinically-diagnosed concussion. *Journal of Neurotrauma*, *31*, 327-338.
- Tellier, A., Marshall, S. C., Wilson, K. G., Smith, A., Perugini, M., & Stiell, I. G. (2009). The heterogeneity of mild traumatic brain injury: Where do we stand? *Brain Injury*, *23*, 879-887.
- Terry, D. P., Faraco, C. C., Smith, D., Diddams, M. J., Puente, A. N., & Miller, L. S. (2012). Lack of long-term fMRI differences after multiple sports-related concussions. *Brain Injury*, *26*, 1684-1696.
- The Centers for Disease Control and Prevention. Basic information about traumatic brain injury and concussion. (2016). Retrieved from <http://www.cdc.gov/traumaticbraininjury/basics.html>.
- Thornton, A. E., Cox, D. N., Whitfield, K., & Fouladi, R. T. (2008). Cumulative concussion exposure in rugby players: neurocognitive and symptomatic outcomes. *Journal of Clinical and Experimental Neuropsychology*, *30*, 398-409.
- Urban, J. E., Davenport, E. M., Golman, A. J., Maldjian, J. A., Whitlow, C. T., Powers, A. K., & Stitzel, J. D. (2013). Head impact exposure in youth football: High school ages 14 to 18 years and cumulative impact analysis. *Annals of Biomedical Engineering*, *41*, 2474-2487.

- Vanderploeg, R. D., Curtiss, G., & Belanger, H. G. (2005). Long-term neuropsychological outcomes following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 11*, 228-236.
- Vasterling, J. J., Brailey, K., Proctor, S. P., Kane, R., Heeren, T., & Franz, M. (2012). Neuropsychological outcomes of mild traumatic brain injury, post-traumatic stress disorder and depression in Iraq-deployed US Army soldiers. *The British Journal of Psychiatry, 201*, 186-192.
- Von Holst, H., & Cassidy, J. D. (2004). Mandate of the WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, 36*, 8-10.
- Voss, J. D., Connolly, J., Schwab, K. A., & Scher, A. I. (2015). Update on the epidemiology of concussion/mild traumatic brain injury. *Current Pain and Headache Reports, 19*, 1-8.
- Wasserman, E. B., Kerr, Z. Y., Zuckerman, S. L., & Covassin, T. (2015). Epidemiology of sports-related concussions in National Collegiate Athletic Association athletes from 2009-2010 to 2013-2014 symptom prevalence, symptom resolution time, and return-to-play time. *The American Journal of Sports Medicine, 44*, 225-233.
- Wechsler, D. (2003). *Wechsler intelligence scale for children—Fourth Edition (WISC-IV)*. San Antonio, TX: The Psychological Corporation.
- Wills, S. M., & Leathem, J. M. (2001). Sports-related brain injury research: Methodological difficulties associated with ambiguous terminology. *Brain Injury, 15*, 645-648.
- World Rugby. Player Numbers. (2016). Retrieved from <http://www.worldrugby.org/development/player-numbers>.

- Yoshino, A., Hovda, D. A., Kawamata, T., Katayama, Y., & Becker, D. P. (1991). Dynamic changes in local cerebral glucose utilization following cerebral concussion in rats: Evidence of a hyper- and subsequent hypometabolic state. *Brain Research*, *561*, 106-119.
- Zhang, L., Yang, K. H., & King, A. I. (2004). A proposed injury threshold for mild traumatic brain injury. *Journal of Biomechanical Engineering*, *126*, 226-236.

### **Appendix A: Concussion definition and symptoms**

The following definition for concussion was obtained from McCrory et al., (2013):

*Concussion is a brain injury and is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces. Several common features that incorporate clinical, pathologic and biomechanical injury constructs that may be utilised in defining the nature of a concussive head injury include:*

- 1. Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head.*
- 2. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, symptoms and signs may evolve over a number of minutes to hours.*
- 3. Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies.*
- 4. Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course. However, it is important to note that in some cases symptoms may be prolonged.(p. 250)*

*The suspected diagnosis of concussion can include one or more of the following clinical domains:*

- 1. Symptoms – somatic (e.g., headache), cognitive (e.g., feeling like in a fog) and/or emotional symptoms (e.g., lability);*
- 2. Physical signs (e.g., loss of consciousness (LOC), amnesia);*
- 3. Behavioural changes (e.g., irritability);*
- 4. Cognitive impairment (e.g., slowed reaction times);*
- 5. Sleep disturbance (e.g., insomnia).*

*If one or more of these components are present, a concussion should be suspected and the appropriate management strategy instituted.*

**Appendix B: Demographic and medical history questionnaire (Control group)**

Name:

Age:

Race:

Occupation:

Telephone Number:

Email address:

Student Number (if applicable):

Height (cm):

Weight (kg):

Are you fluent in English?

YES

NO

Please specify any current learning disabilities:

Have you been previously diagnosed with a psychiatric illness?

YES

NO

*If yes, please specify what you were diagnosed with:*

Have you been previously formally diagnosed with a concussion by a medical health practitioner? *(please specify dates, and details regarding whether or not there was a loss of consciousness and for how long. Please also state how long you were booked off playing sport for.*

| Date of diagnosis | Loss of consciousness | Duration of loss of consciousness | Duration booked off sport participation |
|-------------------|-----------------------|-----------------------------------|---|
|                   | YES NO                |                                   |   |
|                   | YES NO                |                                   |   |
|                   | YES NO                |                                   |   |

If you answered yes to the question above, indicate in a few words what it was like being concussed. What was your experience in terms of the symptoms you experienced, ability to return to exercise, ability to do school or office work, your relationships with your peers etcetera?

Is there any instance(s) where you feel you may have been concussed despite no formal diagnosis? (*headache, nausea, dizziness, ringing in the ears etc.*) Please provide details of this regarding when it occurred, what symptoms you felt, and for how long you were booked off sport after this injury.

YES            NO

WHEN?

SYMPTOMS

DURATION BOOKED OFF SPORT FOR

Please list what types of sporting/exercise activities you have been or are currently involved in e.g., running, swimming, squash, hiking, gym.

*Please also state how often you are involved in each one and at what level you play (social, school team, university team etc.)*

| Sport | Duration of participation | Level of play |
|-------|---------------------------|---------------|
|       |                           |               |
|       |                           |               |
|       |                           |               |
|       |                           |               |

Have you ever been or are currently involved in contact sport that may result in head collisions?

e.g., rugby, American football, soccer.

*If yes, please specify which sport, the duration of your participation and at what level you play(ed) (social, school team, university team etc.)*

| Sport | Duration of participation | Level of play |
|-------|---------------------------|---------------|
|       |                           |               |
|       |                           |               |
|       |                           |               |

**Appendix C: Demographic and medical history questionnaire (Rugby group)**

Name:

Age:

Race:

Occupation:

Rugby team currently playing for:

Student Number (if applicable):

Rugby Playing Position(s):

Cell Number:

Email address:

Height (cm):

Weight (kg):

Are you fluent in English?

YES

NO

Please specify any current/past learning disabilities:

Have you been previously diagnosed with a psychiatric illness?

YES

NO

If YES, please specify what you were diagnosed with, when you were diagnosed and list your medication under the relevant headings below.

| Diagnosis | Age at diagnosis/<br>Year diagnosed | Medication | Still on<br>Medication? |
|-----------|-------------------------------------|------------|-------------------------|
|           |                                     |            |                         |
|           |                                     |            |                         |
|           |                                     |            |                         |

Have you been previously formally diagnosed with a concussion by a medical health practitioner? *(please specify dates, and details regarding whether or not there was a loss of consciousness and for how long. Please also state how long you were subsequently booked off playing sport.*

| Date of diagnosis | Loss of<br>consciousness | Duration of loss of<br>consciousness | Duration booked off<br>sport participation |
|-------------------|--------------------------|--------------------------------------|--|
|                   | YES NO                   |                                      |  |
|                   | YES NO                   |                                      |  |
|                   | YES NO                   |                                      |  |

If you answered yes to the question above, indicate in a few words what it was like being concussed. What was your experience in terms of symptoms you experienced, ability to

return to exercise, ability to do school or office work, your relationships with your peers etcetera?

Is there any instance(s) where you feel you may have been concussed despite no formal diagnosis? (*headache, nausea, dizziness, ringing in the ears etc.*) Please provide details of this regarding when it occurred, what symptoms you felt, and for how long you were booked off sport after this injury.

YES            NO

WHEN?

SYMPTOMS

**DURATION BOOKED OFF SPORT FOR**

How many years have you been playing contact rugby for? \_\_\_\_\_

Please list what types of sporting/exercise activities you have been or are currently involved in e.g., soccer, American football, running, swimming, squash, hiking, gym etc.

*Please also state how often you are involved in each one and at what level you play(ed) (social, school team, university team etc.)*

| Sport | Duration of participation | Level of play |
|-------|---------------------------|---------------|
|       |                           |               |
|       |                           |               |
|       |                           |               |
|       |                           |               |
|       |                           |               |

**Appendix D: The ImPACT neuropsychological test battery**

The ImPACT neuropsychological test battery computerised tasks and composite scores.

| Test name                     | Cognitive domain measured   |
|-------------------------------|---|
| Word Memory                   | Verbal recognition memory (learning and retention)  |
| Design Memory                 | Spatial recognition memory (learning and retention)   |
| X's and O's                   | Visual working memory and cognitive speed   |
| Symbol Match                  | Memory and visual-motor speed   |
| Colour Match                  | Impulse inhibition and visual-motor speed   |
| Three Letter Memory           | Verbal working memory and cognitive speed   |
| Symptom Scale                 | Rating of individual self-reported symptoms   |
|                               |   |
| Composite Scores              | Contributing tasks  |
| Verbal Memory                 | Word Memory (learning and delayed), Symbol Match memory score, Three Letters Memory Score   |
| Visual Memory                 | Design Memory (learning and delayed), X's and O's percent correct   |
| Reaction Time                 | X's and O's (average correct distracters), Symbol Match (average weighted reaction time for correct responses), Colour Match (average reaction time for correct response) |
| Visual Motor Processing Speed | X's and O's (average correct distracters), Symbol Match (average correct responses), Three letters (number of correct numbers correctly counted)                          |
| Impulse Control               | X's and O's (number of incorrect distracters), Colour Match (number of errors)  |

## Appendix E: Informed consent document (Control group)



University of Cape Town  
Psychology Department  
Telephone: +27 21 650-3430  
Fax: +27 21 650-4104

### Investigating history of concussion and data from head impact telemetry (xPatch) in relation to neuropsychological outcomes in a sample of adult rugby players in Cape Town

#### *Informed Consent to Participate in Research and Authorisation for Collection, Use, and Disclosure of Protected Health Information*

This form provides you with information about the study and seeks your authorization for the collection, use and disclosure of your protected health information necessary for the study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Your participation is entirely voluntary. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand. By participating in this study you will not be penalized or lose any benefits to which you would otherwise be entitled.

This study will be conducted in a manner that adheres to the ethical guidelines and principles of the International Declaration of Helsinki (Fortaleza, Brazil, 2013).

#### 1. Name of Participant

---

#### 2. Title of Research Study

Investigating history of concussion and data from head impact telemetry (xPatch) in relation to neuropsychological outcomes in a sample of adult rugby players in Cape Town.

#### 3. What is the purpose of this research study?

The purpose of this research study is to better understand whether or not, and how repeated instances of concussions and/or other head injuries contribute to altered brain functioning. More specifically the research intends to find out how these injuries manifest how the individual thinks, feels and behaves, and in any microstructural brain abnormalities. Also, the purpose is to observe how individuals with head injuries and concussions compare to people who have had no such injuries.

#### 4. Principle Investigator(s) and Telephone Number(s)

Leigh Schrieff-Elson, Ph.D. (PI and supervisor)

Dale Stephen (Masters student)

Psychology Department

Psychology Department

University of Cape Town

University of Cape Town

0216503708

0722352009

Lydia Wepener (Honours student)

Psychology Department

University of Cape Town

0716771182

### **5. What will be done if you take part in this research study?**

During this study, you will be required to complete a number of questionnaires and scales to obtain individual demographic information, personal characteristics, an approximation of your ability to think as well as the different ways in which you act and how you feel. Following initial testing, you may be contacted for repeated testing later in the year; this comprises part of a larger research study that is attached to this one. These testing procedures will be conducted in a private room at the Cape Universities Brain Imaging Centre (CUBIC), Groote Schuur Hospital. By signing the consent form, you are consenting to participation in the possible follow-up assessments as well.

### **6. What are the possible discomforts and risks?**

There is minimal risk associated with this study. You may be required to return for a repeated assessment later in the year. You will be contacted by the Principle Investigator if this is the case. The testing procedures take approximately 1 ½ hours per person. Due to it being a more lengthy process, participants may feel fatigued or irritable during testing as the tasks require concentration. However, each participant will be given breaks where necessary as well as refreshments. The follow-up session is however not as time consuming.

### **7. What are the possible benefits of this study?**

Significantly, this research aims to contribute to practical information regarding return-to-play decisions, thresholds of concussion injuries, and diagnostic indicators of concussion that are important for player safety. However, in order to do so it is necessary to compare the results of our rugby sample to those of individuals who have not sustained a concussion.

Also, as an undergraduate Psychology student you will be awarded 3 SRPP points for your participation in the initial testing session. If you are contacted for the repeated testing session you will be awarded a further 3 SRPP points.

### **8. Can you withdraw from this research study and if you withdraw, can information about you still be used and/or collected?**

You may withdraw your consent and stop participation in this study at any time. Information already collected may still be used.

If you have a complaint or complaints about your rights and welfare as research participants, please contact the Human Research Ethics Committee.

Tel: 021 406 6492

E-mail: [sumaya.ariefdien@uct.ac.za](mailto:sumaya.ariefdien@uct.ac.za)

**9. Once personal information is collected, how will it be kept confidential in order to protect your privacy and what protected health information about you may be collected, used and shared with others?**

If you agree to be in this research study, it is possible that some of the information collected might be copied into a "limited data set" to be used for other research purposes. If so, the limited data set will only include information that does not directly identify you. So, your identity will remain anonymous. Data will be labelled using participant numbers rather than names, so that they cannot be used to directly identify any particular individual. A separate and private log will be used simply to relate participant names to numbers in the event that a participant needs to be contacted or contacts the Principle Investigator. This contact will only be with the Principle Investigator or Dale Stephen.

All information collected will be stored in locked filing cabinets and on computers with security passwords, in a secure computer lab at the University of Cape Town. Only certain people - the researchers for this study - have the legal right to review these research records. Your research records will not be released without your permission unless required by law or a court order. This data may be used to compliment further research in the field of concussion and head injuries, and provides researchers at UCT with a very specific and unique data set. However, the researchers involved in this study will only keep the data for a maximum of 5 years following the final hand-in of the Masters thesis pertaining to Dale Stephen for which this project was intended. Once this time has elapsed, all data pertaining to individual participants stored on the computers will be permanently deleted, and all hard copies of this data will be shredded.

Do you agree to have your data stored for future use? Please circle.

AGREE        /        DISAGREE

**10. Potential Risks**

As discussed, some participant may be recalled for a brain scan, and this forms part of a larger research study that is attached to this one. While undergoing the brain scan some participants may feel anxious or claustrophobic. Before the scan, an assistant will explain the scanning procedure to you. The research assistant will also allow you to have a "mock scan" where you will experience what it is like to have a scan, before undergoing the actual scan. The scan will not hurt you and it will not be dangerous in any way.

During the MRI neuroimaging assessment, certain metal objects, such as watches, credit cards, hairpins, and writing pens, may be damaged by the MRI scanner or pulled away from the body by the magnet. For these reasons, the participant will be asked to remove these objects before entering the scanner. When the scanner takes the images, the bed may vibrate, and the participant will hear loud banging noises. The participant will be given earplugs or earphones to protect the ears. Also, some people feel nervous in a small enclosed space such as that of the scanner. The participant will be able to see out of the scanner at all times, and the radiographer will not start the procedure until he/she tell us that you are comfortable. The participant will be able to stop the procedure at any time by squeezing a ball and can talk to the radiographers using an intercom that is built into the scanner. There are no known

harmful long-term effects of the magnetic fields used in this study. Scans will be no longer than 1 hour.

In the event that this research-related activity results in an injury, treatment will be available including first aid, emergency treatment and follow-up care, as needed. If you have suffered a research related injury, let the investigator know right away.

If you wish to discuss the information above or any discomforts you may experience, you may ask questions now or call the Principal Investigators listed on this form.

Please note that the University of Cape Town carries a No Fault Clinical Liability policy for participants who suffer a research-related injury in researcher-initiated clinical research:

[http://www.health.uct.ac.za/usr/health/research/hrec/forms/No Fault Insurance 2013.pdf](http://www.health.uct.ac.za/usr/health/research/hrec/forms/No_Fault_Insurance_2013.pdf)

### 11. What if something goes wrong?

*The University of Cape Town (UCT) has insurance cover for the event that research-related injury or harm results from your participation in the trial. The insurer will pay all reasonable medical expenses in accordance with the South African Good Clinical Practice Guidelines (DoH 2006), based on the Association of the British Pharmaceutical Industry Guidelines (ABPI) in the event of an injury or side effect resulting directly from your participation in the trial. You will not be required to prove fault on the part of the University.*

*The University will not be liable for any loss, injuries and/or harm that you may sustain where the loss is caused by*

- *The use of unauthorised medicine or substances during the study*
- *Any injury that results from you not following the protocol requirements or the instructions that the study doctor may give you*
- *Any injury that arises from inadequate action or lack of action to deal adequately with a side effect or reaction to the study medication*
- *An injury that results from negligence on your part*

*“By agreeing to participate in this study, you do not give up your right to claim compensation for injury where you can prove negligence, in separate litigation. In particular, your right to pursue such a claim in a South African court in terms of South African law must be ensured. Note, however, that you will usually be requested to accept that payment made by the University under the SA GCP guideline 4.11 is in full settlement of the claim relating to the medical expenses”.*

*An injury is considered trial-related if, and to the extent that, it is caused by study activities. You must notify the study doctor immediately of any side effects and/or injuries during the trial, whether they are research-related or other related complications.*

*UCT reserves the right not to provide compensation if, and to the extent that, your injury came about because you chose not to follow the instructions that you were given while you were taking part in the study. Your right in law to claim compensation for injury where you prove negligence is not affected. Copies of these guidelines are available on request.*

## **12. Management of incidental findings on MRI scans**

A radiologist on CUBIC staff and linked to this study, is going to review all the structural MRI scans for incidental findings. In an unfortunate case of an incidental finding a participant will be referred for further evaluation. Professor Figaji is a neurosurgeon who is regularly referred incidental lesions on MRI scan. He will undertake to consult, examine and counsel the participant where necessary as well as determine any further course of management that may be needed.

## **13. Signatures**

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; the alternatives to being in the study; and how the participant's protected health information will be collected, used, and shared with others:

You have been informed about this study's purpose, procedures, and risks; how your protected health information will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You voluntarily agree to participate in this study. You hereby authorize the collection, use and sharing of your protected health information. By signing this form, you are not waiving any of your legal rights.

\_\_\_\_\_  
Signature of Person Obtaining Consent and Authorization      Date

\_\_\_\_\_  
Signature of Person Consenting and Authorizing      Date

**Appendix F: Informed consent document (Rugby group)**

University of Cape Town  
 Psychology Department  
 Telephone: +27 21 650-3430  
 Fax: +27 21 650-4104

**Investigating history of concussion and data from head impact telemetry (xPatch) in relation to neuropsychological outcomes in a sample of adult rugby players in Cape Town**

*Informed Consent to Participate in Research and Authorisation for Collection, Use, and Disclosure of Protected Health Information*

This form provides you with information about the study and seeks your authorization for the collection, use and disclosure of your protected health information necessary for the study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Your participation is entirely voluntary. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand. By participating in this study you will not be penalized or lose any benefits to which you would otherwise be entitled.

This study will be conducted in a manner that adheres to the ethical guidelines and principles of the International Declaration of Helsinki (Fortaleza, Brazil, 2013).

**1. Name of Participant**

---

**2. Title of Research Study**

Investigating history of concussion and data from head impact telemetry (xPatch) in relation to neuropsychological outcomes in a sample of adult rugby players in Cape Town

**3. What is the purpose of this research study?**

The purpose of this research study is to better understand whether or not, and how repeated instances of concussions and/or other head injuries contribute to altered brain functioning. More specifically the research intends to find out how these injuries manifest in how the individual thinks, feels and behaves, and in any microstructural brain abnormalities. Also, the purpose is to observe how individuals with head injuries and concussions compare to people who have had no such injuries.

**4. Principle Investigator(s) and Telephone Number(s)**

Leigh Schrieff, Ph.D. (PI and supervisor)

Dale Stephen (Masters student)

Psychology Department

Psychology Department

University of Cape Town

University of Cape Town

0216503708

0722352009

Lydia Wepener (Honours student)

Psychology Department

University of Cape Town

0716771182

### **5. What will be done if you take part in this research study?**

During this study, you will be required to complete a number of questionnaires and scales to obtain individual demographic information, personal characteristics, and an approximation of your ability to think. Following initial testing, you may be required to participate in a pilot study to trial the use of head impact telemetry (xPatch) which is a device that can be used to measure the force to the head of all head impacts during a contact rugby match. For the purposes of this study and for gathering important quantitative data, you may be required to wear a plaster (xPatch) behind either your left or right ear – this contains an accelerometer. This is a head collision monitoring device and will enable us to track head movements and forces involved in all head collisions during rugby games. The xPatch will not cause any danger to you or others while playing a rugby match, and should not cause any discomfort. You will have the opportunity to view this device before signing consent.

Additionally, you may also be contacted by one of the researchers to complete a brain scan and post-concussion assessment within 48 hours of a concussion, and this forms part of a larger research study attached to this study. The brain scan and post-concussion assessment will be conducted in a private room at the Cape Universities Brain Imaging Centre (CUBIC), Groote Schuur Hospital. By signing the consent form, you are consenting to participation in these possible follow-up assessments as well.

### **6. What are the possible discomforts and risks?**

There is minimal risk associated with this study. You may be required to wear a plaster (xPatch) which you will be able to view before signing consent to participation. You may also be required to return for repeated testing at the Cape Universities Brain Imaging Centre. You will be contacted by the Principle Investigator if this is the case. Each assessment will last approximately 1½ hours per person to complete. Due to it being a more lengthy process, participants may feel fatigued or irritable during testing as the tasks require concentration. However, each participant will be given breaks where necessary as well as refreshments.

### **7. What are the possible benefits of this study?**

Rugby coaches and players will have access to the overall results upon completion of the study. Each rugby player will have the option of contacting the Principle Investigator for their individual brain scan and test results, if these are administered, and upon completion of the study. Significantly, this research aims to contribute to practical information regarding return-to-play decisions, thresholds of concussion injuries, and diagnostic indicators of concussion that are important for player safety. It thus provides all those involved with contact sport, including the health services, evidence for the neuropsychological effects of repeated head trauma.

**8. Can you withdraw from this research study and if you withdraw, can information about you still be used and/or collected?**

You may withdraw your consent and stop participation in this study at any time. Information already collected may still be used.

If you have a complaint or complaints about your rights and welfare as research participants, please contact the Human Research Ethics Committee.

Tel: 021 406 6492

E-mail: sumaya.ariefdien@uct.ac.za

**9. Once personal information is collected, how will it be kept confidential in order to protect your privacy and what protected health information about you may be collected, used and shared with others?**

If you agree to be in this research study, it is possible that some of the information collected might be copied into a "limited data set" to be used for other research purposes. If so, the limited data set will only include information that does not directly identify you. So, your identity will remain anonymous. Data will be labelled using participant numbers rather than names, so that they cannot be used to directly identify any particular individual. A separate and private log will be used simply to relate participant names to numbers in the event that a participant needs to be contacted or contacts the Principle Investigator. This contact will only be with the Principle Investigator or Dale Stephen.

All information collected will be stored in locked filing cabinets and on computers with security passwords, in a secure computer lab at the University of Cape Town. Only certain people - the researchers for this study and certain University of Cape Town officials - have the legal right to review these research records. Your research records will not be released without your permission unless required by law or a court order. This data may be used to compliment further research in the field of concussion and head injuries, and provides researchers at UCT with a very specific and unique data set. However, the researchers involved in this study will only keep the data for a maximum of 5 years following the final hand-in of the Masters thesis pertaining to Dale Stephen for which this project was intended. Once this time has elapsed, all data pertaining to individual participants stored on the computers will be permanently deleted, and all hard copies of this data will be shredded.

Do you agree to have your data stored for future use? Please circle.

AGREE / DISAGREE

**10. Potential Risks**

Some participants in the research study may feel anxious or claustrophobic with regards to the brain scan. Before the scan, an assistant will explain the scanning procedure to you. The research assistant will also allow you to have a "mock scan" where you will experience what it is like to have a scan, before undergoing the actual scan. The scan will not hurt you and it will not be dangerous in any way.

During the MRI neuroimaging assessment, certain metal objects, such as watches, credit cards, hairpins, and writing pens, may be damaged by the MRI scanner or pulled away from the body by the magnet. For these reasons, the participant will be asked to remove these objects before entering the scanner. When the scanner takes the images, the bed may vibrate, and the participant will hear loud banging noises. The participant will be given earplugs or earphones to protect the ears. Also, some people feel nervous in a small enclosed space such as that of the scanner. The participant will be able to see out of the scanner at all times, and the radiographer will not start the procedure until he/she tell us that you are comfortable. The participant will be able to stop the procedure at any time by squeezing a ball and can talk to the radiographers using an intercom that is built into the scanner. There are no known harmful long-term effects of the magnetic fields used in this study. Scans will be no longer than 1 hour.

In the event that this research-related activity results in an injury, treatment will be available including first aid, emergency treatment and follow-up care, as needed. If you have suffered a research related injury, let the investigator know right away.

If you wish to discuss the information above or any discomforts you may experience, you may ask questions now or call the Principal Investigators listed on this form.

Please note that the University of Cape Town carries a No Fault Clinical Liability policy for participants who suffer a research-related injury in researcher-initiated clinical research:

[http://www.health.uct.ac.za/usr/health/research/hrec/forms/No\\_Fault\\_Insurance\\_2013.pdf](http://www.health.uct.ac.za/usr/health/research/hrec/forms/No_Fault_Insurance_2013.pdf)

### ***11. What if something goes wrong?***

***The University of Cape Town (UCT) has insurance cover for the event that research-related injury or harm results from your participation in the trial. The insurer will pay all reasonable medical expenses in accordance with the South African Good Clinical Practice Guidelines (DoH 2006), based on the Association of the British Pharmaceutical Industry Guidelines (ABPI) in the event of an injury or side effect resulting directly from your participation in the trial. You will not be required to prove fault on the part of the University.***

***The University will not be liable for any loss, injuries and/or harm that you may sustain where the loss is caused by***

- The use of unauthorised medicine or substances during the study***
- Any injury that results from you not following the protocol requirements or the instructions that the study doctor may give you***
- Any injury that arises from inadequate action or lack of action to deal adequately with a side effect or reaction to the study medication***
- An injury that results from negligence on your part***

***“By agreeing to participate in this study, you do not give up your right to claim compensation for injury where you can prove negligence, in separate litigation. In***

*particular, your right to pursue such a claim in a South African court in terms of South African law must be ensured. Note, however, that you will usually be requested to accept that payment made by the University under the SA GCP guideline 4.11 is in full settlement of the claim relating to the medical expenses”.*

*An injury is considered trial-related if, and to the extent that, it is caused by study activities. You must notify the study doctor immediately of any side effects and/or injuries during the trial, whether they are research-related or other related complications.*

*UCT reserves the right not to provide compensation if, and to the extent that, your injury came about because you chose not to follow the instructions that you were given while you were taking part in the study. Your right in law to claim compensation for injury where you prove negligence is not affected. Copies of these guidelines are available on request.*

## **12. Management of incidental findings on MRI scans**

A radiologist on CUBIC staff and linked to this study, is going to review all the structural MRI scans for incidental findings. In an unfortunate case of an incidental finding a participant will be referred for further evaluation. Professor Figaji is a neurosurgeon who is regularly referred incidental lesions on MRI scan. He will undertake to consult, examine and counsel the participant where necessary as well as determine any further course of management that may be needed.

## **13. Signatures**

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; the alternatives to being in the study; and how the participant’s protected health information will be collected, used, and shared with others:

---

Signature of Person Obtaining Consent and Authorization      Date

You have been informed about this study’s purpose, procedures, and risks; how your protected health information will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You voluntarily agree to participate in this study. You hereby authorize the collection, use and sharing of your protected health information. By signing this form, you are not waiving any of your legal rights.

---

Signature of Person Consenting and Authorizing      Date

## Appendix G: Debriefing form



University of Cape Town  
Psychology Department  
Telephone: +27 21 650-3430  
Fax: +27 21 650-4104

### Formal Study Debriefing Form

Thank you for participating in the research study.

This form provides you with information about the study in which you have just participated, and explains in full the methods of collection of data for this research study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also explain this study to you in full and answer all of your questions.

#### 1. Name of Participant ("Study Subject")

---

#### 2. Title of Research Study

Investigating history of concussion and data from head impact telemetry (xPatch) in relation to neuropsychological outcomes in a sample of adult rugby players in Cape Town

#### 3. What is the purpose of this research study?

The purpose of this research study was to better understand the nature of repeated concussive and/or subconcussive head injuries in rugby and how they affect brain functioning compared with those not involved in a contact sport and are not exposed to such injuries. Furthermore, the study was designed to collect data on the forces involved with head injuries sustained by rugby players during the season and, and whether these impacted on brain functioning.

#### 4. What was done during this research study?

During this study, you were required to complete a number of questionnaires and scales to obtain individual demographic information, personal characteristics, and an approximation of cognitive functioning. If you were a rugby player in the study you may have been required to wear a small patch that could monitor head collisions in real time to determine the amount and intensity of head collisions.

#### 5. Was any deception used in this research study?

No.

#### 6. Is anything further required of you?

There is nothing further required of you. If you do however have any questions or concerns regarding my research you may contact the Principle Investigator involved: either Dale Stephen ([dalste12@gmail.com](mailto:dalste12@gmail.com)) or Dr. Leigh Schrieff-Elson ([leigh.e.elson@gmail.com](mailto:leigh.e.elson@gmail.com)).

## 7. Confidentiality

All data collected for the study will be kept confidential – this is not to be confused with the results of the study which will be made available. Data will be labelled using participant numbers rather than names, so that they cannot be used to directly identify any particular individual. Furthermore, all data will be stored in a locked filing cabinets in the department. Data will also be stored on a password-protected computer. Only certain people – the researchers for this study – are afforded the legal right to review these research records.

## 8. Signatures

As a representative of this study, I have explained to the participant, in detail, the purpose, the procedures, and any deception used in this research study.

---

Signature of Person Obtaining Consent and Authorization      Date

I have been informed, in detail, about this study's purpose, procedures, and deceptions. I have been given the opportunity to ask questions before I sign. By signing this form, I am not waiving any of my legal rights.

---

Signature of Person Consenting and Authorizing      Date

## Appendix H: Rugby playing positions

In the game of rugby union, there are fifteen players that start on the field for each team, with eight substitutes allowed per team. In the modern game, eight Forwards and seven Backline (backs) players comprise the starting team.

### Forwards players

The Forwards, make up what is called a scrum, which is a phase of play. The Forwards must consist of eight players: the ‘front row’ (two props, a loose-head and tight-head, and a hooker), the ‘second row’ (two locks), and a ‘back row’ (two flankers, and a number eight). Forwards compete for the ball in scrums and line-outs and are generally bigger and stronger than Backline players, as they are often involved in the more physical part of the game and often take more contact. It is usually expected that the ‘back row’ are involved in the majority of tackles and play an important role in securing possession of the ball for their team.

### Backline players

The players outside the scrum are referred to as the backs: scrum-half/half-back, fly-half, two centres (inside and outside centre), two wings (left and right wing), and a fullback. The backs are generally of a lighter build and faster than Forwards and are more involved in the attacking part of the game, usually taking less contact. The scrum-half has the role of providing the Backline with ball to attack with, the fly-half’s role is generally to direct the Backline, the centres’ key attacking roles are to try and break through the defensive line and to then link with wingers. With this in mind, it would usually be expected that centres’ would take the most contact in the Backline on defence and attack.



Figure 3. Rugby union positions and set-up from a scrum