A COMPARISON OF BRAIN TRAUMA PROFILES BETWEEN ELITE MEN'S RUGBY UNION 15s AND RUGBY UNION 7s GAME PLAY

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ABSTRACT

Head impact and subsequent brain trauma is a concern in contact sports including rugby. Head collisions present acute and long term injury risks to the brain ranging from non-symptomatic, concussion, and neurodegeneration. Rugby Union 15s and Rugby Union 7s are the two most played codes of the sport and the physical and tactical differences may affect how brain trauma is experienced (Cunniffe, Proctor, Baker, & Davies, 2009; Colin W Fuller, Taylor, & Molloy, 2010; L. J. Suarez-Arrones, J. Nunez, Portillo, & Mendez-Villanueva, 2012). It is important to consider all parameters contributing to acute and long term injury risk in order to appropriately capture brain trauma experienced in a contact/collision sport (Karton & Hoshizaki, 2018). Impact frequency, frequency-magnitude, and interval between impact have all been reported to affect brain trauma. Trauma profiling is a method used to describe brain trauma using the variables relating to brain injury risk. The purpose of this study was to compare head impacts experienced in rugby union 15s and 7s using frequency of impact events, frequency-magnitude of brain deformation, and time interval between impacts.

Thirty-six hundred (3600) player minutes of footage were analysed for each code, and all head impacts were categorised. Twenty (20) impact conditions were observed and reconstructed. Head to shoulder, hip and knee events were reconstructed using a pneumatic linear impactor, head to head events were reconstructed using a pendulum system, and head to ground events were reconstructed using a monorail drop rig. Results from both codes were compared using non-parametric Mann-Whitney U tests and demonstrated that Rugby 7s had a higher overall frequency of head impact, a greater number of head impacts causing higher trauma , and a shorter time interval between head impacts. These results suggest that rugby 7s presents a greater risk for sustaining brain trauma. These results will help expand the understanding of conditions leading to injury, and may lead to better interventions, such as equipment or rule changes, to mitigate risk.

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CHAPTER 1: INTRODUCTION

1.1 Overview/Problem Statement

Since its debut as a professional sport in 1995, rugby's increase in popularity is evident through its growing athletic participation numbers, currently totaling over 9.6 million participants worldwide (Duthie, Pyne, & Hooper, 2003; World Rugby, 2017). World rugby oversees 8 codes of rugby. The two most common codes of the sport, Rugby Union fifteens (rugby union; 15s) and Rugby Union sevens (rugby sevens; 7s) (Higham, Pyne, Anson, & Eddy, 2012), have tactical and physical differences (Cunniffe, Proctor, Baker, & Davies, 2009; Colin W Fuller, Taylor, & Molloy, 2010; L. J. Suarez-Arrones, J. Nunez, Portillo, & Mendez-Villanueva, 2012). Both forms of rugby are played on the same pitch dimensions, and with essentially the same regulations. Rugby 15s has a season lasting up to twenty-two weeks, with approximately one match per week, and all matches lasting eighty minutes. The fourteen-minute matches in rugby 7s are played in tournament style (i.e. playing upwards of six games in a one- to three-day period) with a varying number of weeks in-between. Fuller and colleagues (2010) reported that 7s rugby players are shorter and lighter than their rugby 15s counterparts. Distributions of running velocities demonstrated that 38% less time is spent standing and walking and 7.6% more time is spent sprinting in rugby 7s games compared to 15s (Cunniffe, Proctor, Baker, & Davies, 2009; L. J. Suarez-Arrones, J. Nunez, Portillo, & Mendez-Villanueva, 2012).

A surge in rugby 7s' participation is expected following the sport's reappearance in the Olympic Games in 2016 (Ross, Gill, & Cronin, 2014). This increase in participation is accompanied by a concern for the head contact experienced in the sport, and the associated health risks (Patricios & Kemp, 2013; Raftery, 2014; Stewart, McNamara, Lawlor, Hutchinson, & Farrell, 2016). Individual head impacts are referred to as impact events, and their effect on brain tissue is

influenced by their unique impacting characteristics: mass, velocity, location, vector, and compliance (Oeur et al., 2015). Event types create unique brain trauma due to the interaction of the above characteristics. Event types most commonly reported to cause concussion in rugby are head-to-head, shoulder-to-head, hip-to-head, knee-to-head, and head-to-ground (Ignacy, 2017). These event types occur in both rugby 15s and rugby 7s, but it is unknown how much the differences in mass and running velocities interact to affect the brain trauma experienced by athletes. Furthermore, it is unknown if differences in location and impact vector exist between the sports codes, or if the frequency distribution of event types differ.

Head impacts in contact sports such as rugby have been associated with a range of brain trauma: including sub-concussive head impacts, mild traumatic brain injuries (mTBI), traumatic brain injuries (TBI), and potentially long-term neurological conditions including Chronic Traumatic Encephalopathy (CTE) (Gavett, Stern, & McKee, 2011; Kiernan, Montenigro, Solomon, & McKee, 2015; McKee et al., 2009; Omalu et al., 2005). The 2007 Rugby World Cup (RWC) injury surveillance study reported a concussion rate of 2.6/1000 player hours (Fuller, Laborde, Leather, & Molloy, 2008). These surveillance reports, starting at the 2003 RWC, reveal a rise in concussion rates of 3% at the 2007 RWC, 9% at the 2011 RWC, and up to 14% at the 2015 RWC (Fuller, Laborde, Leather, & Molloy, 2008; Fuller, Sheerin, & Targett, 2013; Fuller, Taylor, Kemp, & Raftery, 2017). World Rugby implemented a new concussion management approach in 2015 that increased the identification resources dedicated to concussion including doctors and video review (Fuller et al., 2017). Rugby 7s studies have reported the proportion of total injuries sustained as concussions in the Sevens World Series (SWS) and 2016 Rio Olympics ranges from 7.1 to 17.0 percent (Fuller, Taylor, & Raftery, 2017). These injury rates are 16.8, 18.6, and 8.8 concussions/1000 player hours in the 2014/2015 SWS, 2015/2016 SWS, 2016 Rio

Olympics, respectively. Concussions are diffuse brain injuries brought on by shear forces applied to the brain tissue that lead to a neurometabolic cascade including neuronal depolarization, ionic shifts, altered cerebral blood flow, and impaired axonal function (Bandak, Ling, Bandak, & De lanerolle, 2015; Giza & Hovda, 2001).

Cognitive deficiencies and behavioural changes have been reported for youth, collegiate, and retired rugby players. At the schoolboy level, significant decreases were reported in rugby players' postseason attentional tasks with speeded visuomotor component when compared to non-contact controls (Shuttleworth-Edwards et al., 2008). These alterations in brain function could be associated with brain damage caused by the repetition of sub-concussive impacts, which may put players at risk for more severe brain deterioration later in life (Gavett, Stern, Cantu, Nowinski, & McKee, 2010; Gavett et al., 2011; McKee et al., 2009). Retired international rugby players reported lower scores on a verbal learning test ($p=0.022$) and on fine co-ordination of the dominant hand ($p=0.038$) when compared to controls (McMillan et al., 2017). Thornton and colleagues (2008) also reported a dose-dependent relationship between post-concussion symptoms and the number of concussions sustained by retired and older rugby players. CTE is a neurological disease associated with the repetitive exposure of head contact manifested later in life. Two cases of CTE have been diagnosed in former rugby players (Maroon et al., 2015; McKee et al., 2009). Common characteristics of this tauopathy are atrophy of the cerebrum, cerebellum, and cavum septi pellucidi (Mawdsley & Ferguson, 1963), as well as septal anomalies, loss of pigment in substantia nigra, and neurofibrillary tangle occurring in the absence of senile plaques (Corsellis, Bruton, & Freeman-Browne, 1973).

The risk of sustaining a brain injury from a singular event is determined by the magnitude of trauma (Meaney & Smith, 2011; Oeur t al., 2015; Post et al., 2015). The higher the magnitude

of brain deformation for a mTBI or TBI, the more severe the injury (Gurdjian, Roberts, & Thomas, 1966; Kleiven, 2007; Willinger & Baumgartner, 2003; Zhang, Yang, & King, 2004). Researchers have correlated a higher frequency of head impacts, shorter time intervals between impacts, and longer careers in contact sports to an increased likelihood of long-term neurodegeneration (Martland, 1928; McKee et al., 2009; Meehan, Zhang, Mannix, & Whalen, 2012; Ojo et al., 2016; Omalu et al., 2005). Risk curves for mTBIs and TBIs have been created using brain deformation values (Zhang et al., 2004), but no consensus has been reached on threshold values needed to significantly increase one's risk of developing long-term neurological conditions (Gavett et al., 2010, 2011; McKee et al., 2009). It is important to consider all parameters contributing to acute and long term injury risk in order to appropriately capture brain trauma experienced in a contact/collision sport (Karton & Hoshizaki, 2018). Trauma profiling is a method used to describe brain trauma using the variables relating to brain injury risk. The purpose of this study was to compare head impacts experienced in rugby union 15s and 7s using the frequency of impact events, magnitude of brain deformation, and time interval between impacts.

1.2 Research Question

Are there significant differences in brain trauma profiles occurring during game play between elite men's Rugby Union 15s and rugby 7s?

1.3 Objectives

1. Compare the overall frequency of head impacts occurring in elite men's rugby union 15s and 7s matches.

2. Compare the frequency of impact event type occurring in elite men's rugby union 15s and 7s matches.

3. Compare the frequency of brain tissue deformation created by head impacts in elite men's rugby union 15s and 7s matches.

4. Compare the time interval between head impacts within each MPS category between elite men's rugby union 15s and 7s.

1.4 Hypothesis

1. It was hypothesized that frequency of head impact events will be greater in rugby union 15s in comparison to rugby union 7s.

2. It was hypothesized that rugby union 15s will have a greater number of head-to-head and head-to-ground impact event types, while rugby union 7s will have a greater number of head-to-shoulder and head-to-hip impact event types.

3. It was hypothesized that the brain tissue deformation measures created by head impacts in rugby union 7s will be greater than those created in rugby union 15s.

4. It was hypothesized that the time interval between head impacts will be greater in rugby union 7s in comparison to rugby union 15s.

1.5 Null Hypothesis

1. There will be no difference in the overall frequency of head impact in game play between elite men's rugby 15s and 7s matches.

2. There will be no difference in the frequency of head-to-head impacts in game play between elite men's rugby 15s and 7s matches.

3. There will be no difference in the frequency of head-to-shoulder impacts in game play between elite men's rugby 15s and 7s matches.

4. There will be no difference in the frequency of head-to-hip impacts in game play between elite men's rugby 15s and 7s matches.

5. There will be no difference in the frequency of head-to-knee impacts in game play between elite men's rugby 15s and 7s matches.

6. There will be no difference in the frequency of head-to-ground impacts in game play between elite men's rugby 15s and 7s matches.

7. There will be no difference in the frequency of other impacts in game play between elite men's rugby 15s and 7s matches.

8. There will be no difference in the frequency of impacts creating “very low” peak maximal principle strains (<8%) in game play between elite men’s rugby 15s and 7s matches.

9. There will be no difference in the frequency of impacts creating “low” peak maximal principle strains (8-16.9%) in game play between elite men’s rugby 15s and 7s matches.

10. There will be no difference in the frequency of impacts creating “medium” peak maximal principle strains (17-25.9%) in game play between elite men’s rugby 15s and 7s matches.

11. There will be no difference in the frequency of impacts creating “high” peak maximal principle strains (26-34.9%) in game play between elite men’s rugby 15s and 7s matches.

12. There will be no difference in the frequency of impacts creating “very high” peak maximal principle strains (>35%) in game play between elite men’s rugby 15s and 7s matches.

13. There will be no difference in the overall time interval of confirmed impacts in game play between elite men’s rugby 15s and 7s matches.

14. There will be no difference in the interval between impacts creating “very low” peak maximal principle strains (<8%) in game play between elite men’s rugby 15s and 7s matches.

15. There will be no difference in the interval between impacts creating “low” peak maximal principle strains (8-16.9%) in game play between elite men’s rugby 15s and 7s matches.

16. There will be no difference in the interval between impacts creating “medium” peak maximal principle strains (17-25.9%) in game play between elite men’s rugby 15s and 7s matches.

17. There will be no difference in the interval between impacts creating “high” peak maximal principle strains (26-34.9%) in game play between elite men’s rugby 15s and 7s matches.

18. There will be no difference in the interval between impacts creating "very high" peak maximal principle strains (>35%) in game play between elite men's rugby 15s and 7s matches.

1.6 Limitations

1. Inherent limitations of video analysis include hidden players and contact moments, zooming lenses, a moving camera screen, or the lack of in frame field markings to apply a grid for distance calculations. An inclusion criteria was created for labeling impacts (Table 1, appendix A).
2. The 50th percentile Hybrid III headform is commonly used in event reconstructions as a representation of the human head. It is made of metal and rubber, and is not fully biofidelic for living humans. Its dynamic responses may not be true to real-life events (Deng, 1989; Samaka & Tarlochan, 2013).
3. Finite element models, including the UCDBTM, are created from CT scans, MRI information, and other imaging devices of human cadavers (Horgan & Gilchrist, 2003). They are tested against cadaveric data and may therefore not create responses true to the living human brain.

1.7 Delimitations

1. This study used footage from men's rugby in the 2015 Rugby World Cup (RWC) and 2016 Olympic Games and the results are only applicable to male rugby players at elite levels of competition.
2. The results of this study only describe brain trauma profiles for game situations and do not consider head impacts occurring in practice situations. Consequently, the overall brain trauma profile of participating in either codes of rugby may be greater than the in-game profile presented in this study.

1.8 Significance

This is the first study to complete a brain trauma profile for elite men's Rugby Union 15s and Rugby Union 7s. Identifying and quantifying the characteristics of brain trauma will help expand the understanding of conditions leading to injury. Once there is an understanding of which conditions lead to trauma, interventions may be considered to mitigate risk. Based on the findings, equipment can be developed to protect against brain trauma sustained through specific impacting characteristics. More importantly, regulations can be put in place to help control the frequency of, or time interval between, head impacts with the intention of making the game safer. The comparison of the two profiles may also lead individuals and institutions to choose one code over the other.

CHAPTER 2: LITERATURE REVIEW

2.1 Rugby Union 15s v 7s Physical and Tactical Characteristics

Rugby 15s is played in two 40 minute halves (Duthie et al., 2003), with 15 players per side, seven substitutes, and seven interchanges (L. J. Suarez-Arrones et al., 2012); World Rugby, 2017). Sevens rugby is played in two 7 minute halves, with 7 players per side, five substitutes, and five interchanges (Higham, Pyne, Anson, & Eddy, 2013; L. Suarez-Arrones et al., 2014; L. J. Suarez-Arrones et al., 2012). Both codes are played on the same pitch dimensions with similar playing laws (Higham et al., 2012; L. Suarez-Arrones et al., 2014).

Nicholas (1997) reported that forwards (numbered 1 through 8) are statistically heavier and taller than backs (numbered 9 through 15) for a rugby 15s team. Fuller and colleagues (2010) reported that 7s backs (numbered 4 through 7) are 2 cm shorter and 6 kg lighter than their 15s counterparts, and that 7s forwards (numbered 1 through 3) are 1 cm shorter and 13 kg lighter than their rugby 15s counterparts.

Distributions of running velocities in rugby shown in Table 2, revealed that 38% less time is spent standing and walking and 7.6% more time is spent sprinting during games in rugby 7s compared to 15s (Cunniffe et al., 2009; L. J. Suarez-Arrones et al., 2012). Hendrick and colleagues (2012) found that the mean inbound velocities prior to tackling in rugby 15s ranged from 3.9 m/s to 6.4 m/s. McIntosh and colleagues (2000) reported the mean inbound velocity of concussive head impacts in rugby 15s to be 7.0 m/s, reaching a maximum value of 14 m/s. No tackling inbound velocity measures have been reported for rugby 7s.

Table 2

Percentage of average time spent in each velocity category for 7s rugby players (Suarez-Arrones et al., 2012) and 15s rugby players (Cunniffe et al., 2009) in a match.

Speed	Rugby 15s	Rugby 7s	Difference
Standing and walking (0-6 km/hr)	72.5%	34%	38.5
Jogging (6.1-12.0 km/hr)	18.7%	26%	7.3
Cruising (12.1-14.0 km/hr)	3.25%	9.8%	6.55
Striding (14.1-18.0 km/hr)	3.75%	15.5%	11.75
High Intensity Running (18.1-20.0 km/hr)	1%	5%	4
Sprinting (>20.0 km/hr)	1.1%	8.7%	7.6

2.2 Presence of Brain Injuries in Rugby

2.2.1 Incidence of mTBI and TBI

The 4th International Conference on Concussion stated that a concussion, often interchanged with mTBI, is "a brain injury… defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces" (McCrory et al., 2013). A systematic review by Gardner and colleagues (2014), including 37 articles, reported the overall concussion rate in rugby union 15s to be 4.75 concussions per 1000 player match hours, with a lower reported rate of 1.19 at the elite level. The meta-analysis also reported an overall concussion rate of 3.01 concussions per 1000 player match hours in rugby 7s (Gardner et al., 2014). For an individual player, 1000 player match hours is equivalent to 750 matches of rugby 15s, and to 4285 matches of rugby 7s. When considering a whole team, 1000 player match hours is equivalent to 50 games

of rugby 15s, and 612 games of rugby 7s. Concussive rates from the last four Rugby World Cups, the Sevens World Series, and the Rio Olympics can be found in Table 3. In terms of risk of concussion, rugby 7s athletes proves to be at a higher risk of injury.

Table 3

Incidence rates for concussion in RWC, SWS, and Olympics.

Author	Event	Incident rate (concussions/1000 player match hours)	Percentage of Overall injuries sustained (%)
Rugby Union 15s			
Best, McIntosh, & Savage (2005)	2003 RWC		2.1
Fuller, Laborde, Leather & Molloy (2008)	2007 RWC	2.6	
Fuller, Sheerin, & Targett (2012)	2011 RWC	7.8	
Fuller, Taylor, Kemp, & Raftery (2016)	2015 RWC	12.6	13.9
Rugby Union 7s			
Fuller, Taylor & Raftery (2015)	2008-2013 SWS	8.3	
Fuller, Taylor, & Raftery (2017)	2014-2015 SWS	16.8	15.6
	2015-2016 SWS	18.6	17.0
	2016 Rio Olympic Games	8.8	7.1

TBIs fall under the category of catastrophic injuries in rugby. Catastrophic injuries in rugby, defined by Fuller (2008) as "fatalities and brain/spinal cord injuries resulting in a significant permanent neurological deficit … which [are] a direct consequence of playing rugby union", have similar risk levels as other sports according to the UK Health and Safety Executive (HSE) standards (Fuller, 2008). The UKHSE's 'acceptable' and 'tolerable' risk categories are 0-1.9 and 2-100 cases/100 000 population at risk per year, respectively (Fuller, 2008). Rugby union 15s in England, Ireland, and Argentina all show risks between 0.8 and 1.9/100 000 per year, falling in the 'acceptable' region. Rugby 15s in New Zealand, Australia, and Fiji fell into the 'tolerable' region of risk, with a highest risk score of 13 cases (Fuller, 2008). This data has not been collected for Rugby 7s.

2.2.2 Signs of Cognitive Impairment and Long-Term Neurodegeneration in Rugby

Cognitive deficiencies and behavioural changes have been reported for current and retired rugby players at various playing levels. A study by McMillan and colleagues (2017) reported that retired international rugby players had lower scores on a verbal learning test ($p=0.022$) and on fine co-ordination of the dominant hand ($p=0.038$) compared to controls. Another study examining the frequency of common mental disorders (CMD) in retired rugby union players found that 25% reported distress, 28% reported anxiety or depression, 29% reported sleeping disturbance, and 24% reported adverse alcohol behaviour (Gouttebarge, Kerkhoffs, & Lambert, 2015). These findings are higher than matched control anxiety/depression rates (ranging from 12-25%) and distress rates (ranging from 5-18%) (Gouttebarge et al., 2015). Furthermore, Thornton and colleagues (2008) found a dose-dependent relationship between post-concussion symptoms and the number of concussions sustained by retired and older rugby players.

Studies involving university and school boy levels have examined the accumulated cognitive effects of a rugby season on its players. Shuttleworth-Edwards and colleagues (2014) found that non-contact sport control students ($n=106$) at the university and high school levels improved their pre- and post-season visual motor speed (VMS) through a practice effect. This same practice effect was not found in rugby players ($n=145$) as their scores showed no improvement from pre- to post-season (Shuttleworth-Edwards et al., 2014). In an earlier study significant decreases in rugby players' postseason attentional tasks with speed visuomotor component in comparison to non-contact controls was reported (Shuttleworth-Edwards et al., 2008). Using pre- and post-game resting-state functional magnetic resonance imaging (rs-fMRI) of collegiate level rugby players Johnson and colleagues (2014) reported short-term bursts of sub-concussive head trauma lead to altered default mode network (DMN) connectivity patterns. These

alterations in brain function could be associated with brain damage caused by the repetition of sub-concussive impacts, which may put players at risk for neurological conditions later in life.

CTE is a neurological diseases associated with the repetitive exposure of head contact manifested later in life. It has been associated with playing contact/collision sports. Two cases of CTE have been diagnosed in former rugby players (Maroon et al., 2015; McKee et al., 2009). Martland (1928) observed physical and mental deficiencies in retired boxers – concluding that a special brain injury coined "punch-drunk" could derive from a single or multiple impacts to the head. This tauopathy, only diagnosable through post-mortem biopsy, is now recognized as *chronic traumatic encephalopathy* (CTE) (Corsellis et al., 1973; McKee et al., 2009). Common characteristics of CTE affected brains are atrophy of the cerebrum, cerebellum, and cavum septi pellucidi (Mawdsley & Ferguson, 1963), as well as septal anomalies, loss of pigment in substantia nigra, and neurofibrillary tangle occurring in the absence of senile plaques (Corsellis et al., 1973). This brain deterioration leads to staggered gait, poor balance, tremors, speech abnormalities (Gavett et al., 2011; Martland, 1928; McKee et al., 2016), as well as memory loss, dementia, anxiety, and aggression (McKee et al., 2009; Montenigro et al., 2014). A history of concussion is common, but not necessary, for the development of CTE (Stein, Alvarez, & McKee, 2015). To date a total of 153 cases of CTE have been found in former boxers, American football players, hockey players, rugby players, and wrestlers (Maroon et al., 2015; McKee et al., 2009). A concern has emerged in the rugby community as knowledge on the risks of CTE and how it may affect players increases (Patricios & Kemp, 2013; Raftery, 2014; Stewart et al., 2016).

2.3 Biomechanical Considerations of Head Impact

Brain injuries are sustained from head impacts and the resulting head motion. Measuring the dynamic response of the head, including peak resultant linear acceleration (g) and peak

resultant rotation acceleration (rad/s^2), has been used to characterize different kinds of brain injury (Fréchède & McIntosh, 2009; Gurdjian et al., 1966; Gurdjian & Webster, 1945; Holbourn, 1943; King et al., 2014; King et al., 2003; Margulies et al., 1990). More recently, finite element (FE) models have been used to analyze reconstructed injury events and calculate relative stresses and strains within the brain tissues (Deck & Willinger, 2008; Horgan & Gilchrist, 2003; Patton, McIntosh, Kleiven, & Frechede, 2012; Ward, 1982; Zhang et al., 2004). These models generate values, such as maximum principle strain (MPS), which have been shown to have a better correlation with brain injuries and depict a clearer picture of the trauma occurring at the tissue level.

2.3.1 Linear Acceleration

Gurdjian and colleagues (1966) demonstrated an association between direct impact to the head and intracranial pressure changes through elastic skull deformation and relative brain skull motion. The correlation between intracranial pressure and linear acceleration has led to research using the latter as it is the more practical measure (Hoshizaki, Post, Oeur, & Brien, 2014). It was initially thought that linear acceleration was the main cause of head injuries (Gurdjian et al., 1966; Gurdjian & Webster, 1945), but later established that although it produces focal injuries it does not lead to diffuse injuries (Gennarelli et al., 1979; Post & Hoshizaki, 2012; King et al., 2003). Zhang and colleagues' (2004) mTBI injury curves calculated a 25%, 50%, and 80% probability of sustaining concussion when 66, 82, and 106 g were reported for professional football players.

2.3.2 Rotational Acceleration

The theory that rotation, and not translation, cause concussions was first introduced by Holbourn (1943). Gennarelli and colleagues (1979) demonstrated that both diffuse axonal injury (DAI) and acute subdural hematoma can be created with pure rotational acceleration. Using animal

experiments and numerical reconstructions it was proposed that rotational acceleration causes shear strain in the brain tissue and thus is an important contributor to the development of concussive injury, DAI, and subdural hematomas than linear acceleration (Gennarelli et al., 1979; Gurdjian et al., 1966; King et al., 2003; Ommaya & Gennarelli, 1974; Post & Hoshizaki, 2012; Post, Rousseau, Kendall, Walsh, & Hoshizaki, 2015; Yoganandan, Li, Zhang, Pintar, & Gennarelli, 2008). Zhang and colleagues' (2004) mTBI injury curves calculated a 25%, 50%, and 80% probability of sustaining concussion when 4600, 5900, and 7900 rad/s^2 are attained for impacts experienced in professional football.

2.3.3 Brain Tissue Deformation

Finite element modeling is defined as "a computational technique that is used to obtain approximate solutions to the sets of partial differential equations that predict the response of physical systems that are subjected to external influences" (Horgan & Gilchrist, 2003). Maximum principal strain (MPS) is a commonly used measure which quantifies brain tissue strain sustained during impact through the analysis of acceleration-time curves (Horgan & Gilchrist, 2003; Ignacy et al., 2017; King et al., 2003; Patton et al., 2012; Post, Hoshizaki, & Gilchrist, 2012). Although dynamic response provides a reliable measure of head kinematics, it is widely agreed that FE models provide further representation of intra-cranial response after head impact (Horgan & Gilchrist, 2003; Post & Hoshizaki, 2012; Ward, 1979; Zhang et al., 2004). Kleiven (2007) reported a 50% probability of sustaining concussion when MPS values of 0.21 and 0.26 were reached in the corpus callosum and gray matter, respectively.

2.4 Impacting Characteristics

The tissue response resulting from an impact event is influenced by the impacting characteristics: mass, velocity, location, vector, and compliance (Oeur et al., 2015). Event types

create unique brain trauma due to their interaction of the above characteristics – by predicting the dynamic response of the head, and in-turn the resulting brain tissue deformation (Gennarelli et al., 1982; Kleiven, 2003; Pellman et al., 2003; Post et al., 2014, Willinger & Baumgarthner, 2003; Zhang et al., 2001, 2004) Event types most commonly reported to cause concussion in rugby are head-to-head, shoulder-to-head, hip-to-head, knee-to-head, and head-to-ground (Ignacy, 2017). Each type of event creates unique loading curves of head acceleration over time (Kendall, 2016; Post & Hoshizaki, 2012).

As one of these characteristics is altered, the dynamic response and potential resulting brain injury will be effected. For example, helmets in contact/collision sports such as hockey and American football have a high compliance (soft) inner lining which is built to absorb energy coming into the system and prolong the impact duration (Hoshizaki et al., 2014). The increase in duration leads to a decrease in magnitude, and shifts the risk of injury from a TBI or focal injury, to an mTBI or diffuse injury (Dawson, 2016; Karton & Hoshizaki, 2018). Due to the viscoelastic nature of brain tissue however, it is possible for different distributions of peak acceleration and duration to lead to the same risk for brain injury, such as concussions in ice hockey and American football cause by head-to-shoulder impact and helmet-to-helmet impacts, respectively (Dawson, 2016; Karton & Hoshizaki, 2018). This reinforces the importance of considering both the shape and duration of the acceleration time curve when predicting brain tissue deformation (Post & Hoshizaki, 2012).

Known differences in running velocity and mass between rugby 15s and 7s may create difference in brain injury risk. It is unknown if differences in impact location or vector exist between the sport codes. It has been reported that impacts to the side of the head, as well as non-

centric impacts, create higher rotational acceleration resulting in higher risk of concussive injury (Karton, 2012; Oeur, 2018)

2.5 Measuring Brain Trauma: Defining the Trauma Profile

Trauma profiling is a method used to describe brain trauma using the variables relating to brain injury risk. There is a large spectrum of potential brain injury in contact/collision sports such as rugby: acute, long-term, symptomatic, non-symptomatic, etc., and relying solely on signs of injury or magnitude of head impact does not fully represent this spectrum. Impact magnitude has been used as a measure to examine the risk brain injury and create protective standards, however, it does not measure lower-energy impacts that do not present with immediate signs of injury. The importance of frequency and time interval between head impacts in relation to long-term brain injury risks is reported in the literature. Duration of participation has also been reported to impact once risk of sustaining long term neurodegenerative diseases. It is important to consider multiple parameters contributing to acute and long term brain injury risk in order to appropriately capture the load of brain trauma (Karton & Hoshizaki, 2018). This study will focus on the frequency, frequency magnitude, and time interval factors creating trauma profiles in rugby 15s and 7s as participation data was not available.

2.5.1. Frequency of Head Impact

The presence of structural changes in brain tissue and changes in blood biomarkers following sub-concussive head impacts over a season of play in contact/collision sport supports the argument that higher frequencies of lower-energy impacts have an effect on brain health (Bahrami et al., 2016; Bazarian et al., 2012; Breedlove et al., 2012; Karton & Hoshizaki, 2018; Koerte et al., 2012; Kuzminski et al., 2017; Talavage et al., 2014; Slobounov et al., 2017). These studies reported a positive correlation between the frequency of impacts and the extent of measured

changes (Bahrami et al., 2016; Bazarian et al., 2012; Breedlove et al., 2012; Karton & Hoshizaki, 2018; Koerte et al., 2012; Kuzminski et al., 2017; Talavage et al., 2014; Slobounov et al., 2017). Corsellis and colleagues (1973) and Roberts (1969) both observed that boxers involved in less fights sustained less neurological abnormalities later in life. Increased levels of neurofilament light polypeptide (NF-L) and total tau (T-tau) has been found in boxers after bouts and American football players after a season of play (Karton & Hoshizaki, 2018; Neselius et al., 2012; Oliver et al., 2016; Shahim et al., 2017; Zetterberg et al., 2006). Similarities between these blood biomarkers over the duration of a season and after a single severe traumatic event have been reported by Kondo and colleagues (2015). Similar pathologic outcomes comparing repetitive lower magnitude brain trauma to one severe event suggest similar neurological risks (Karton & Hoshizaki, 2018).

A study by King and colleagues (2014) evaluated the frequency and dynamic response of head impacts greater than 10g via instrumented mouth guards in rugby union 15s. A total number of 20,687 impacts were recorded in 19 matches (379 player match hours), resulting in a mean average of 564 ± 618 impacts per athlete per season (King et al., 2014). No study has evaluated the frequency of head contacts in rugby 7s.

2.5.2. Magnitude of Brain Trauma

It is well understood that brain tissue stress and strain measures derived from using finite element models provide an effective measure of risk for injury (Zhang et al., 2004; Doorly & Gilchrist, 2006; Kleiven, 2007; Sahoo et al., 2016). A positive correlation exists between magnitude of MPS and risk of sustaining a TBI or mTBI. Kleiven (2007) reported a 50% probability of sustaining concussion when 21% MPS and 26% MPS were reached in the corpus callosum and gray matter, respectively.

Recent research has provided evidence that injury to the brain can occur from an impact despite the lack of concussive symptoms (Bailes, Petraglia, Omalu, Nauman, & Talavage, 2013; Johnson et al., 2014; Robinson et al., 2015; Tarnutzer, Straumann, Brugger, & Feddermann-Demont, 2016; Tong, Winter, Jin, Bennett, & Waddell, 2015). Sub-concussive impacts do not necessarily result in a clinically diagnosed concussion, nor do they initiate the neurometabolic cascade of a concussion, but have been reported to have increasingly negative health effects with increased exposure (Bailes et al., 2013, Giza & Hovda, 2001; Shultz, MacFabe, Foley, Taylor, & Cain, 2012; Stern et al., 2011). In the absence of observable structural damage, studies have reported levels of strain as low as 5-15% to be associated with functional impairment of signal transmission (Margulies & Thibault, 1992; Bain & Meaney, 2000; Singh et al., 2006; Elkin & Morrison, 2007). Cellular cultures were used by Yuen and colleagues (2009) in reporting that the minimum level of injury required to induce calcium influx was 5% strain. Reconstructions corresponding to roughly 20-30g head accelerations in American football linemen during game play resulted in 9-12% MPS with no reported symptoms (Karton & Hoshizaki, 2018). Reconstructions of Kendo sword strikes, a sport with no history of long-term brain injury, resulted in 5-7% MPS (Karton, Hoshizaki, & Gilchrist, 2016).

Fréchède and McIntosh (2009) reconstructed concussive impacts from rugby union 15s using computer simulating software and found an average linear acceleration of 103g and an average rotational acceleration of 8022 rad/s^2. Reconstructions of concussive impacts from Rugby League using a dummy head resulted in linear accelerations ranging from 24.2g to 205g, rotational acceleration ranging from 2650 rad/s^2 to 15890 rad/s^2, and MPS values ranging from 27% to 57% (Ignacy, 2017). King and colleagues' (2014) data on overall head impacts experienced in rugby

union 15s resulted in a mean linear accelerations of 22.2g ± 16.2g, and a rotational acceleration of 3902.9 rad/s^2 ± 2948.8 rad/s^2 over a season.

2.5.3. Time Interval between Impacts

Researchers have proposed that a greater time interval between head impacts decreases an individual's likelihood of developing neurological diseases later in life (Gavett et al., 2010; McKee et al., 2009; R. A. Stern et al., 2011). NF-L biomarker levels have been analyzed post-injury, and it was reported that a singular concussive event in hockey took 7-8 days to return to baseline levels, whereas a boxer who suffered a knockout took approximately 36 weeks to return to baseline level (Neselius et al., 2012; Shahim et al., 2016). When a lack of recovery time between brain traumas occurs repeatedly, the development of chronic glymphatic system flow impairment may occur, leading to increased levels of interstitial tau and the potential promotion of tau aggregate formation leading to cell death (Jessen et al., 2015; Peng et al., 2016) Meehan and colleagues (2012) impacted animals with concussive blows to the head at varying frequencies per day, week, and month, finding that increased time between injuries improved cognitive outcomes.

Time interval between head impacts of rugby 15s and rugby 7s will be affected by the differences in match length (i.e. 80 minutes versus 14 minutes) and season organization (i.e. weekly matches versus tournaments). Time intervals between head impacts during play have not been calculated for neither rugby 15s nor 7s.

CHAPTER 3: METHODOLOGY

3.1 Variables

3.1.1 Independent Variables

1. Type of Rugby Being Played
    a. Rugby Union 15s
    b. Rugby Union 7s

2. Velocity
    a. Very Low (0-2m/s)
    b. Low (2-3.5 m/s)
    c. Medium (3.5-6 m/s)
    d. High (>6m/s)

3. Impact Event
    a. Head to Head
    b. Head to Knee
    c. Head to Shoulder
    d. Head to Hip
    e. Head to Ground

3.1.2 Dependent Variables

1. Frequency of Head Impacts

2. Brain Deformation
    a. Peak Maximum Principle Strain (MPS)

3. Time Interval between Head Impacts

3.2 Procedures

3.2.1 Study Population: Inclusion/Exclusion Criteria

Thirty-six hundred player minutes of video were collected through media broadcasting for both the 2015 Rugby World Cup (RWC) and for the 2016 Rio Summer Olympics. This resulted in the analysis of 3 rugby 15s halves and 18 rugby 7s matches, each resulting in the analysis of 60 player hours. The average player age, weight, and height can be found in Table 4. The game selection included teams with different levels of success (initial rankings and final placement in the tournaments) and different coaching styles.

Table 4
The average player age, weight, and height for the 2015 RWC (Fuller et al., 2016) and 2016 Rio Olympic Games (Fuller, Taylor & Raftery, 2017).

	Age (yrs)	Weight (kg)	Height (cm)
Rugby Union 15s			
2015 RWC	27.4 ± 3.8	104.1 ± 13.2	185.9 ± 7.2
Rugby Union 7s			
2016 Rio Olympic Games	25.9 ± 3.5	90.5 ± 9.4 kg	182.6 ± 7.5

3.2.2 Impact Conditions

Twenty possible reconstruction conditions for each code of rugby were made by using 5 common impact events (head-to-head, shoulder-to-head, hip-to-head, knee-to-head, and head-to-ground) at 4 velocity ranges (very low, low, medium, and high). Table 5 describes these conditions. No reconstructions were completed for conditions not observed during the video analysis. Any impacts caused by a different event were labeled as "other" impacts, and were only used for frequency and interval calculations.

Table 5
Possible reconstructive conditions based on common event and velocity categories seen in rugby15s and 7s.

	A1	**A2**	**A3**	**A4**
B1	A1 B1	A2 B1	A3 B1	A4 B1
B2	A1 B2	A2 B2	A3 B2	A4 B2
B3	A1 B3	A2 B3	A3 B3	A4 B3
B4	A1 B4	A2 B4	A3 B4	A4 B4
B5	A1 B5	A2 B5	A3 B5	A4 B5

Legend:

Closing Velocity:
A1: very low (<2.0 m/s)
A2: low (2.0 – 3.49 m/s)
A3: medium (3.5 m/s – 6.0 m/s)
A4: high (> 6.0 m/s)

Event Type:
B1: head-to-head
B2: shoulder-to-head
B3: hip-to-head
B4: knee-to-head
B5: head-to-ground

3.2.3 Video Analysis

All recorded footage was re-played and recorded at a known frame speed of 25 fps using WM Capture 8 (open source, Recorder.com). Each head impact was labeled as 'confirmed' or 'suspected' (Table 1, Appendix A). In order to be considered as an exemplar video, a confirmed impact needed to include clear reference markings on the playing surface. Closing distances of these impacts was calculated using Kinovea software 0.8.20 (open source, Kinovea.org) by converting image pixels into meters (Figure 1). The known time and distance of impact was then used to calculate closing velocity (v=d/t). Suspected impacts were omitted from statistical analysis and only considered in the discussion.

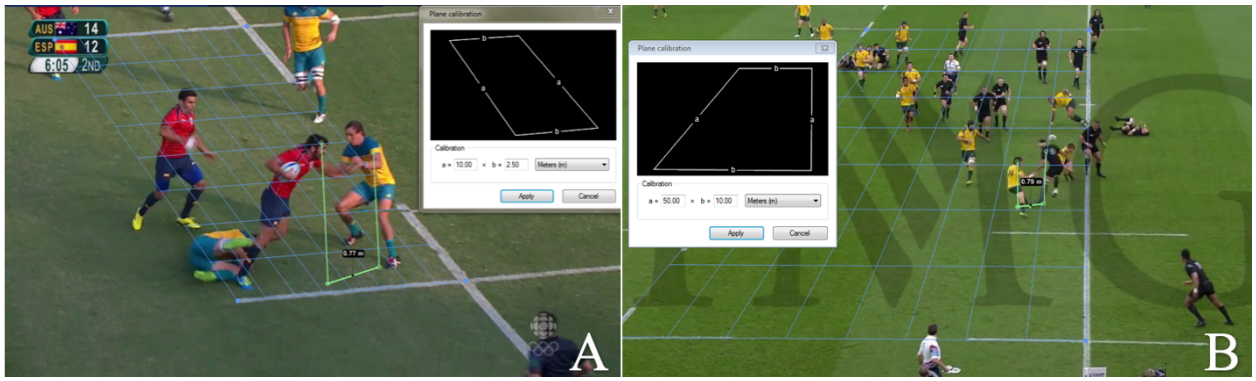

Figure 1. Example of Kinovea system determining distance between two points: A) Medium head to shoulder impact from Rugby 7s; B) Medium head to shoulder impact from Rugby 15s.

Video analysis is the preferred method of examination in rugby research (Reardon, Tobin, Tierney, & Delahunt, 2016), and a procedure commonly used in the field of head biomechanics (Hendricks et al., 2012; Rock & Graham, 2016; Rousseau & Hoshizaki, 2015). The head was divided into 4 locations in order to determine the most common impact location (front, rear, side, top) (King et al., 2014). Impacts considered for reconstruction also had location logged by a 48-option grid overlaying the head (Figure 2). Player position and scenario of play were documented in order to provide a better population description.

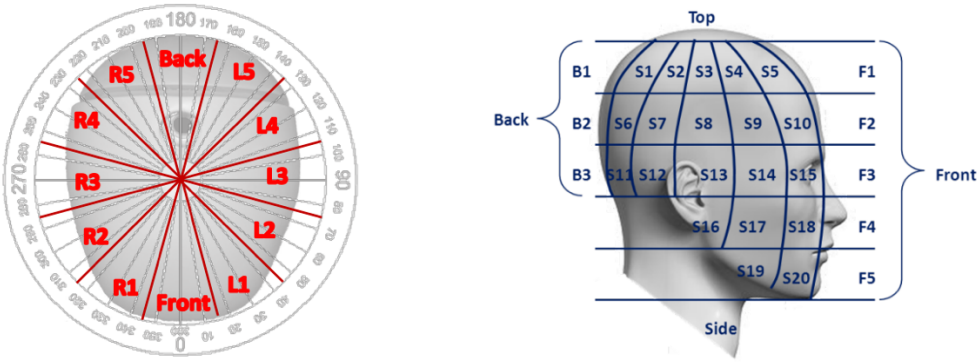

Figure 2. Location grids for reconstruction.

3.2.4 Reconstruction Protocol

One exemplar per condition, per rugby code, was selected for reconstruction. Conditions that were not observed during video analysis include head-to-head at the medium and high velocities, head-to-ground at the high velocity, and knee-to-head at the medium and high velocities. No exemplar was reconstructed to represent these conditions. Final selection of the exemplar impacts was made by re-analyzing the videos meeting the initial criteria, and selecting the impact matching the most common impact location (front, rear, side, top), with the clearest reference markings, and with the reference markings closest to the camera.

Three trials were conducted per exemplar, resulting in a total of 90 reconstructions. Collision events (head-to-head, head-to-shoulder, head-to-hip, and head-to-knee) were reconstructed using the linear impactor and pendulum system, and fall events (head-to-ground) were reconstructed using the monorail drop rig. The compliance of the impacting surface was made to represent the real life compliance by matching acceleration-time curves in order to increase the accuracy of the reconstruction. Three foam pads were used on the impacting arm of the pneumonic impactor for shoulder and hip collisions. A secondary headform was used on the pendulum system for head-to-head collisions, and turf was used on the anvil of the monorail drop rig for falls. Dynamic response data from both instruments was collected using the TDAS ProLab

Module rer(DTS, Seal Beach, CA) software. The acceleration-time history curves for each reconstruction was then input into the University College Dublin Brain Trauma Model (UCDBTM) FE model to calculate brain tissue deformation.

3.2.5 Brain Trauma Categories

The Neurotrauma Science Laboratory (NISL) identified five brain trauma categories to represent different levels of brain tissue trauma (Table 6) and represent the magnitude of head damage caused by head impact.. Once the maximal principle strain values were calculated using the UCDBTM, each condition along with its associated frequency, was classified into a brain trauma category. The values associated with the brain trauma categories were used in the statistical analysis (Karton & Hoshizaki, 2018).

Table 6

Brain trauma categories based on maximal principle strain.

Category	Maximal principle strain range
Very Low	< 0.08
Low	0.08 – 0.169
Medium	0.17 – 0.259
High	0.26 – 0.345
Very High	≥ 0.35

3.3 Equipment

3.5.1 Hybrid III Headform and Unbiased Neckform

A male 50th percentile Hybrid III headform (Figure 3) was used for all reconstruction conditions. It has a mass of 4.54 +/- 0.01 kg and is equipped with nine single-axes Endevco 7264C-2KTZ-2-300 accelerometers arranged in an orthogonal "3-2-2-2" array (FTSS, Plymouth MI). This accelerometer arrangement was developed by Padgaonkar and colleagues (1975) to measure three-dimensional motion. The system of accelerometers triggers data collection passed the threshold of 3g linear acceleration. The head was attached to an unbiased neck form, meaning the individual

disks are of uniform thickness. This headform is commonly used in the literature and is considered to be representative of the human head (Post et al., 2015).

Figure 3. 50th percentile Hybrid III headform and unbiased neck

3.3.2 Linear Impactor

The pneumonic linear impactor (Figure 4) was comprised of a steel frame, an impacting arm, a piston, and a pressurized tank. The steel frame supports an impacting arm which is propelled horizontally by the piston. The rugby 7s collisions were reconstructed using a 13.1 +/- 0.01 kg arm, whereas the rugby 15s collisions using a 15.3 +/- 0.01 kg arm to better represent the higher overall mass of the athletes. The velocity at which the impacting arm travels is controlled by the air pressure of the tank and the distance of the piston, and is calculated using an electric time gate attached to the steel frame. At the end of the arm is a striker used to represent the compliance of the event. Compliance of the hip and shoulder were previously determined and represented by using three foam pads attached to the top of a nylon base (Figure 5A) (Ignacy, 2017; Rock, 2016). The knee impactor used an EPP base, a 2 part metal cylinder, and a metal cap attached at the top (Figure 5B). The middle shaft of the knee impactor was removed in order to reduce the noise created by the interaction of different materials.

Placed at the end of the pneumatic linear impactor is an adjustable sliding table (m=12.782 +/- 0.001kg; Cadex, St-jean-sur-Richelieu, QC) to which the head and neck forms are attached.

The headform was oriented to match the locations and impact vector of the real life exemplar impact.

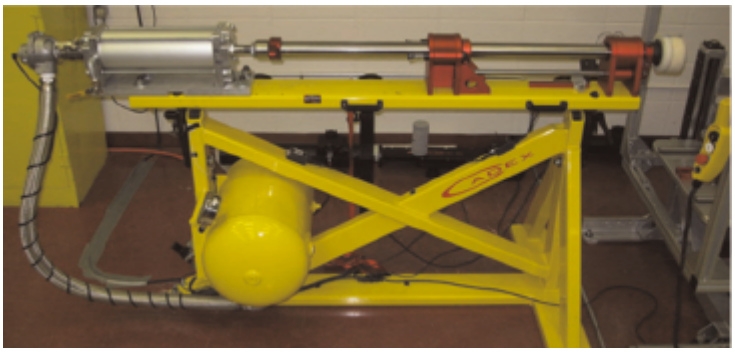

Figure 4. Pneumonic Linear Impactor

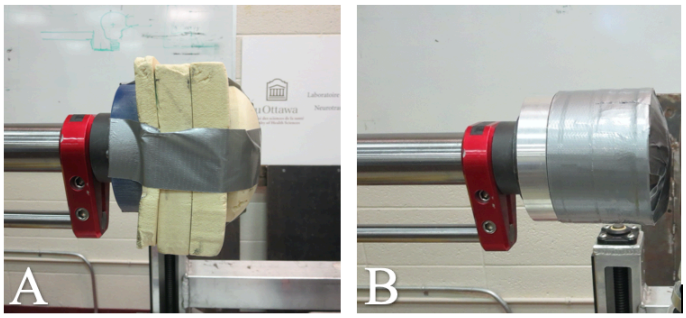

Figure 5. A) The striker used to represent the shoulder and hip compliance; B) The striker used to represent the knee compliance.

3.3.3 Pendulum

A pendulum system was used to represent the head-to-head collisions (Figure 6). A NOCSAE (National Operating Committee on Standards for Athletic Equipment) headform weighing 4.90 kg was suspended by 4 stainless steel 3/32 cables attached to the ceiling directly above the sliding table in order to represent the compliance of a head upon impact (Pellman et al., 2003; Zhang, Yang, & King, 2001). The alignment of the wires ensured that the suspended headform was parallel to the table upon impact in order to reduce the vertical component of velocity. A latch that released with the use of a magnet was attached to the chin of the NOCSAE headform (Karton, 2012). Pendulum height was adjusted to reflect velocity of the impact established from video footage. A High Speed Imaging PCI-512 Fastcam camera (Photron USA

Inc., San Diego, CA, USA) was positioned perpendicular to the impact site and recorded the impact at 250 frames per second to monitor the velocity at time of impact. Velocity was calculated 5 frames prior to impact, each frame representing 0.004 seconds. Table position and orientation of the head and neck were adjusted to represent location and direction of impact.

Figure 6. Pendulum System

3.3.4 Monorail Drop Rig

The Cadex monorail drop rig (Figure 7A) is a machine consisting of a 4.7m guided rail designed to drop a headform attached to a carriage. The carriage is released by a pneumatic piston, where it travels down the rail to impact. To reduce friction, the carriage is attached by bushings. A photoelectric time gate situated 0.02m above the impact site calculates the impacting velocity. The anvil (impacting surface) was covered by a patch of artificial turf in order to duplicate the real life impacting surface (Figure 7B).

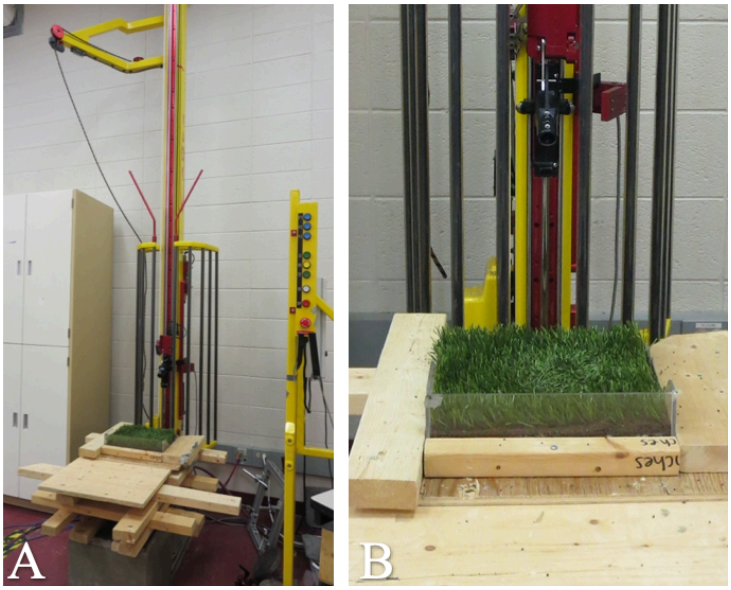

Figure 7. A) Monorail Drop Rig; B) anvil covered in artificial turf

3.3.5 UCDBTM

The three-dimensional dynamic response of the Hybrid III head form, the x, y, and z-axes acceleration loading curves for both linear and angular acceleration, were input into the UCDBTM finite element brain model (Horgan & Gilchrist, 2003). The UCDBTM was created through fall reconstructions in aim to calculate maximum principle strains (MPS) incurred through head impact (Horgan & Gilchrist, 2003). The model geometry was created through the use of CT scans and MRI imaging of adult male cadavers and validated against both cadaveric and real world TIB reconstructions (Horgan & Gilchrist, 2003; Nahum et al., 1977). The model is comprised of 26, 000 hexahedral elements divided into ten sections including the scalp, a 3-layered skull, dura, CSF, pia, falx, tentorium, cerebral hemispheres, cerebellum, and brainstem. The validation process compared the predicted pressure-time histories obtained from previous data collected by Nahum and colleagues (1977). Tables 7 and 8 summarize the mechanical properties of the anatomical model components.

Table 7

Material Properties of UCDBTM

Material	Young's Modulus (PA)	Poisson's Ratio	Density (kg/m^2)
Scalp	16,7	0.42	1000
Cortical Bone	15000	0.22	2000
Trabecular Bone	1000	0.24	1300
Dura	31.5	0.45	1130
Pia	11.5	0.45	1130
Falx and Tentorium	31.5	0.45	1130
Brain	Hyperelastic	0.49	1040
CSF	Water	0.50	1000
Facial Bone	5000	0.23	2100

Table 8

Material characteristics of brain tissue components for UCDBTM

Material	G_0	G_∞	Decay Constant (Gpa)	Bulk Modulus (s^{-1})
Cerebellum Brain	10	2	80	2.19
Stem	22.5	4.5	80	2.19
White Matter	12.5	2.5	80	2.19
Grey Matter	10	2	80	2.19

3.4 Statistical Analysis

When evaluating the brain deformation, all statistical analysis were conducted using the SPSS software for Windows (IBM INC., Armonk, NY, USA). A Shapiro-Wilk test showed the data was not normally distributed. Non-parametric Mann-Whitney U tests (α=0.05) were then chosen to compare the brain trauma categories, the overall frequency as well as the frequency per event type between codes, and the interval between head impacts.

CHAPTER 4: RESULTS

To determine differences in brain trauma profiles between the two codes of rugby, seventeen Mann-Whitney U tests were conducted. The following section presents the results for overall frequency, frequency of event types, frequency magnitude of brain trauma, and time interval between impacts

4.1 Frequency Results

All visible head impacts were logged during the video analysis, but only head-to-head, head-to-shoulder, head-to-hip, head-to-ground, and head-to-knee events were chosen to create the impact conditions established for reconstruction. Table 9 reports the number of all confirmed head impacts that occurred on the field categorized by MPS. A total of 312 confirmed "other" impacts were also recorded in rugby 15s. There were 203, 83, 24, and 2, recorded impacts at very low, low, medium, and high closing velocities respectively. A total of 349 confirmed "other" impacts were also recorded in rugby 7s. There were 178, 100, 59, and 12, recorded impacts at very low, low, medium, and high closing velocities respectively. All suspected impacts are reported in Table 10 (appendix B). Figure 10 displays the distribution of event types for rugby 7s and rugby 15s.

Table 9

Absolute number of confirmed impacts recorded through video analysis.

		MPS Category				
		Very Low	**Low**	**Medium**	**High**	**Very High**
15s						
	Head-to-Head		38		8	
	Head-to-Shoulder	119	80	50		
	Head-to-Hip	188	33			9
	Head-to-Ground		87	22		2
	Head-to-Knee		46			11
Total		**307**	**284**	**72**	**8**	**22**
7s						
	Head-to-Head		24		10	
	Head-to-Shoulder		244			13
	Head-to-Hip		102	82	102	19
	Head-to-Ground		68	55		21
	Head-to-Knee		38		21	
Total		**0**	**476**	**137**	**133**	**53**

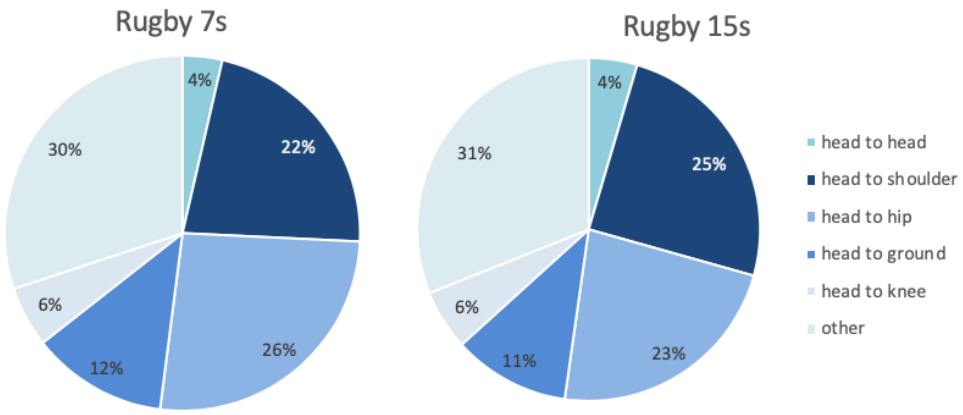

Figure 8. Distribution of event type during open play by both rugby 15s and rugby 7s

4.1.1 Comparison of Each Event Frequency for the Two Codes of Rugby

The frequencies calculated where normalized over 60 minutes and divided by the number of players on the field. A non-parametric Mann-Whitney U was performed for each event type across the two codes of rugby to determine if differences in frequency exist. No significant difference in the frequency of impacts were found for any event type between rugby 7s and rugby 15s. Therefore, the six null hypothesis concerning the frequency of event types were accepted. Figure 11 displays the frequency of impact for each event type.

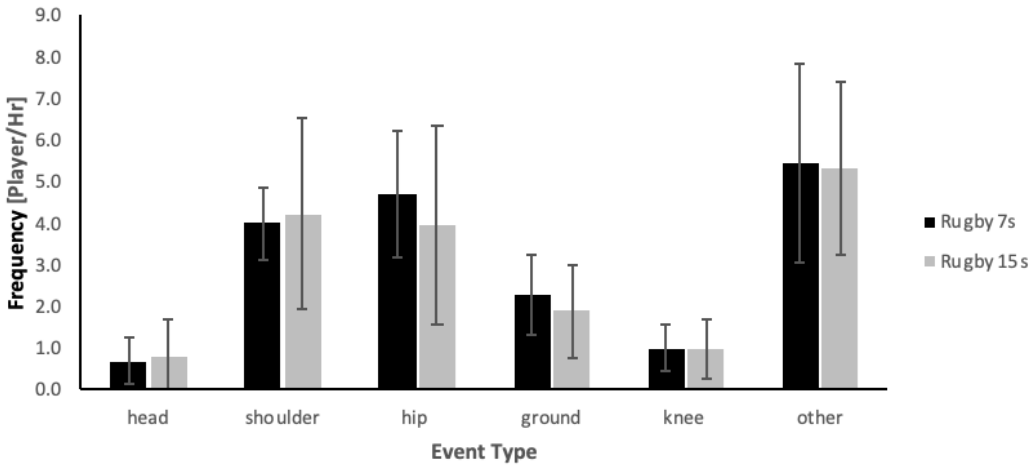

Figure 9. Mean rate of impact in each event type measured in impacts per player per hour of open play in competitive matches.

4.1.2 Rates of Impact

Including all confirmed impacts, the rates of head impact standardized over 60 minutes of play is 18.09 impacts/hour in rugby 7s, and 16.75 impacts/hour in rugby 15s. When only analysing reconstructed impacts (removing impacts from the "other" event type), rugby 7s athletes will receive an average of 12.45 impacts/hour and rugby 15s athletes will receive an average of 11.55 impacts/hour. Separated by MPS categories, a rugby 7s athlete will receive an average of 0, 7.42, 2.14, 2.07, and 0.83 impacts/hour in the very low, low, medium, high, and very high MPS categories respectively. A rugby 15s athlete will receive an average of 5.12, 4.73, 1.20, 0.13, and 0.37 impacts/hour in the very low, low, medium, high, and very high MPS categories respectively.

4.2 Frequency Magnitude Results

Figure 8 provides the distribution of brain trauma for rugby 7s and rugby 15s. Out of the 40 impact conditions established for reconstruction between the codes, 30 were observed in the video analysis and matched with an exemplar. Closing velocity (ranging from 0.5m/s to 9m/s), location, and striking angle were represented in each real-life impact reconstruction. Table 11 (Appendix B) reports the maxima, minima, and average closing velocities calculated in video analysis for each impacting condition, as well as the average velocity recorded during reconstructions of each exemplar. Upon finite element modeling, the frequency associated with each condition was classified in a brain trauma category based on the resulting Maximal Principle Strain. Table 12 summarizes the classification of impact conditions into brain trauma categories. Results for each impact trial can be found in Table 13, Appendix B.

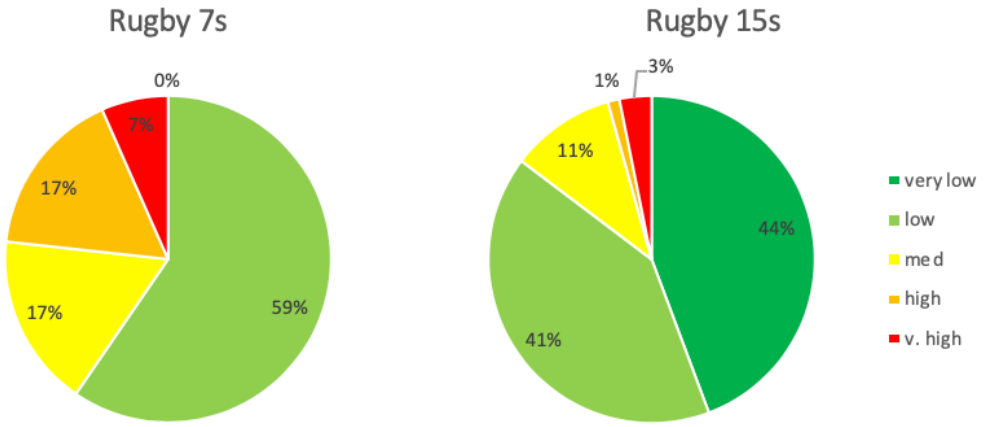

Figure 10. Distribution of brain trauma created by a total of 30 exemplar reconstructions and their associated frequency.

Table 12

Mean maximal principle strain values for each observed condition in both rugby codes, and their associated brain trauma category.

	15s		7s	
Impact Conditions	**maximal principle strain**	**brain trauma category**	**maximal principle strain**	**brain trauma category**
Head-to-Head				
Very Low velocity	0.1019	Low	0.1019	Low
Low velocity	0.2837	High	0.2837	High
Head-to-Shoulder				
Very Low velocity	0.0506	Very Low	0.1015	Low
Low velocity	0.0898	Low	0.1107	Low
Medium velocity	0.1965	Medium	0.1327	Low
High velocity	0.2430	Medium	0.4642	Very High
Head-to-Hip				
Very Low velocity	0.0597	Very Low	0.1256	Low
Low velocity	0.0723	Very Low	0.1909	Medium
Medium velocity	0.1587	Low	0.2675	High
High velocity	0.4022	Very High	0.5608	Very High
Head-to-Ground				
Very Low velocity	0.1380	Low	0.1380	Low
Low velocity	0.2259	Medium	0.2259	Medium
Medium velocity	0.3508	Very High	0.3508	Very High
Head-to-Knee				
Very Low velocity	0.1112	Low	0.1687	Low
Low velocity	0.3912	Very High	0.3323	High

Head-to-Shoulder

The impacts occurring at "very low" closing velocities (1.82m/s for rugby 7s; 1.27m/s for rugby 15s) resulted in different levels of trauma. The higher closing velocity, coupled with an impact location and vector which elicited more rotation of the head, resulted in more trauma for rugby 7s. The rugby 7s impact occurred behind the ear, whereas the rugby 15s impact was struck directly to the temple, with no head tilt, eliciting a low rotational response. The impacts occurring at "low" closing velocities (2.23m/s for rugby 7s; 2.79m/s for rugby 15s) both resulted in low level trauma. Although the rugby 7s impact had a lower velocity, it's directionality elicited more rotation, leading to both impacts creating trauma in the same category. The impacts occurring at "medium" closing velocity (4.03m/s for rugby 7s; 3.95 m/s for rugby 15s) resulted in different levels of trauma, with the rugby 15s impact location elicited greater rotational response. The impacts occurring at "high" closing velocities (8.24 m/s for rugby 7s; 6.52 m/s for rugby 15s) resulted in different levels of trauma. Both locations and vectors elicited rotation, and although the impacting arm was heavier for rugby 15s, the closing velocity had a larger contribution.

Head-to-Hip

The impacts occurring at "very low" closing velocities (1.96m/s for rugby 7s; 1.43m/s for rugby 15s) resulted in the different levels of trauma. The rugby 7s impact was to the temple and elicited low levels of rotational acceleration, whereas the rugby 15s impact was behind the ear and elicited a greater rotational response. Impacts occurring at "low" closing velocity (3.64 m/s for rugby 7s; 2.47m/s for rugby 15s) also resulted in different levels of trauma. The combination of higher velocity, along with an impact location to the temple of a forward tilted head eliciting high rotational acceleration, resulted in the rugby 7s impact producing more trauma. The impacts at "medium" closing velocity (4.89 m/s for rugby 7s; 3.67 m/s for rugby 15s) resulted in different

levels of brain trauma. The rugby 15s impact location was perpendicular to the temple producing greater compressional forces to the brain tissue rather than shear forces, and resulted in only low levels of brain trauma. The rugby 7s reconstruction had a forward tilt, impacting the front boss and eliciting rotation through multiple axes to produce high levels of brain trauma. The impacts occurring at "high" closing velocity (7.14 m/s for rugby 7s; 6.21 m/s for rugby 15s) resulted in the same levels of brain trauma. Both reconstructions were struck at similar locations and vectors.

Head-to-Knee

The impacts occurring at "very low" closing velocities (1.9 m/s for rugby 7s; 0.7 m/s for rugby 15s) resulted in the same level of brain trauma. With locations and vectors being similar, impacting the head in the temple area, the greater mass of the impacting arm for rugby 15s reconstructions drove the resulting level of brain trauma. Impacts occurring at "low" closing velocities (2.99 m/s for rugby 7s; 3.43 m/s for rugby 15s) resulted in the different levels of brain trauma. Higher velocity and mass resulted in rugby 15s sustaining more trauma.

Head-to-Head & Head-to-Ground

The same mechanical forces were used in head-to-head or head-to-ground impacts for both rugby 7s and 15s because there were no differences in compliance or mass. As the most common locations were the same, the same exemplars were used to represent both codes. Differences in these impact conditions will be measured through frequency and interval rather than magnitude of brain trauma.

4.2.1 Comparison of Magnitude Distribution for the Two Codes of Rugby

A non-parametric Mann-Whitney U test was performed for each brain trauma category across the two codes of rugby to determine if a difference in frequency exists. Significant differences were found at each level of brain trauma between rugby 7s and rugby 15s. Therefore,

the five null hypothesis concerning the frequency of impacts creating brain trauma within each category were rejected.

The population pyramid curve shapes for the very low, low, high, and very high brain trauma categories were different between rugby 7s and rugby 15s, allowing for the comparison of mean rank scores. At the very low brain trauma category, rugby 15s had a higher frequency distribution (mean rank = 27.50) compared to rugby 7s (mean rank score = 9.50). At the low, high, and very high brain trauma categories, rugby 7s (mean rank = 23.83, 27.39, and 21.56) has a higher frequency distribution compared to rugby 15s (mean ranks = 13.17, 9.61, and 15.44). The curve shape for the medium brain trauma category was the same between the two codes, allowing for the comparison of medians. At the medium brain trauma category rugby 7s had a higher frequency when compared to rugby 15s. Figure 9 displays the mean rate of head impacts per player for each brain trauma category.

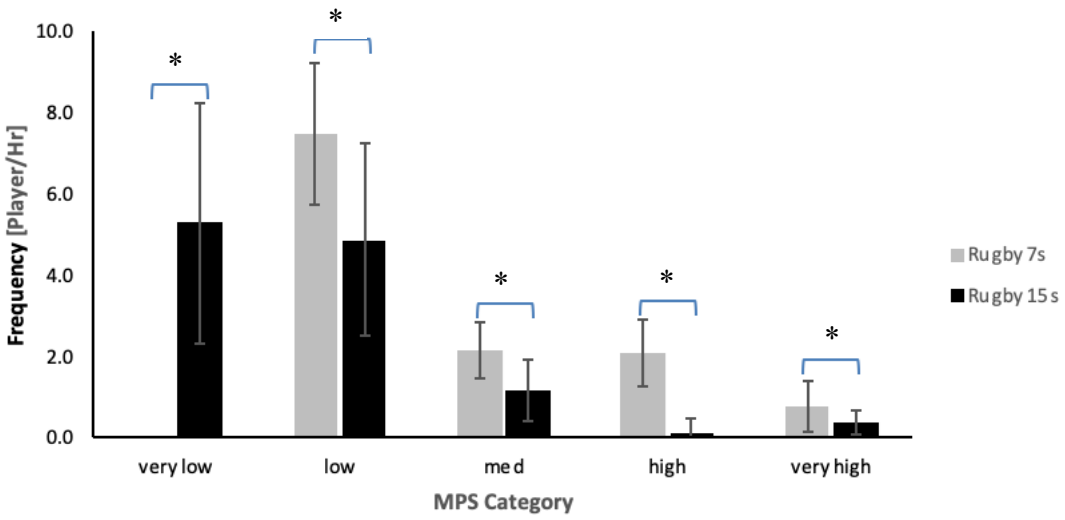

Figure 11. Mean rate of impact in each brain trauma categories measured in impacts per player per hour of open play in competitive matches.

4.3 Interval Results

Table 14 reports the interval, normalized per minute, between all confirmed head impacts (including "other" head impacts that were not reconstructed), and between impacts associated to a brain trauma category. Interval measures were calculated for athletes as a team, and divided by the number of players on the field. Interval measures for each game where then averaged. Table 15 (Appendix B) reports the maxima and minima intervals for all confirmed impacts, and for each MPS category. Each category needed to have at least 2 impacts in order to calculate an interval.

Table 14

Mean interval between impacts collapsed, and within each brain trauma category.

	15s	**7s**
Brain Trauma Category	**Interval** min/player (SD)	**Interval** min/player (SD)
Confirmed Impacts	3.35 (1.32)	3.54 (0.97)
Very Low	10.99 (6.91)	
Low	13.33 (7.63)	8.47 (2.87)
Medium	25.41 (17.91)	29.26 (11.55)
High	0.06 (0.09)	28.05 (14.02)
Very High	47.75 (17.61)	59.01 (46.68)

4.3.1 Comparison of Time Between Impact for the Two Codes of Rugby

A non-parametric Mann-Whitney U was performed for confirmed impacts collapsed, and within each brain trauma category between the two rugby codes. Rugby 7s did not have any impacts in the very low trauma, and therefore no interval measures, and rugby 15s only had three interval measure for its high trauma impacts. This made a comparison at the very low and high brain trauma categories unreasonable.

Significance was found for the low brain trauma category between the rugby codes and the null hypothesis was rejected. Mean rank scores were compared as the curve shapes between

rugby codes were different, and rugby 15s (mean rank = 22.39) had a higher interval between impacts in comparison to rugby 7s (mean rank = 14.61). No significance was found at the medium and very high brain trauma categories, therefore the null hypothesis was accepted.

CHAPTER 5: DISCUSSION

The term brain injury encompasses a large group of specific injuries with unique injury mechanisms and clinical presentations (Blennow, Hardy, & Zetterberg, 2012; Mizobuchi & Nagahiro, 2016; Oeur et al., 2015). TBIs lead to visible injuries, while mTBIs are not visible but diagnosed from an array of possible signs and symptoms depending on the location and severity of the head impact, as well as the unique traits of the individual. It is now recognized that not all head impacts causing injury manifest in signs or symptoms. All head impact impose forces on the brain tissue and may lead to brain tissue deformation (Mainwaring, Ferdinand, Mylabathula, & Alavie, 2018), regardless of clinical presentation. Creating brain trauma profiles can provide more information than measuring clinically presented injuries alone.

The rugby 7s and rugby 15s brain trauma profiles are different in rate of impact, frequency magnitude, and overall interval between impacts. The rugby 7s brain trauma profile demonstrates a higher rate of impact, more dangerous playing conditions due to the higher number of impacts creating medium, high, and very high maximal principle strain values, and shorter overall time interval between head impacts.

Frequency

This study showed almost identical results in the distribution of event types. The number of impacts occurring to the shoulder, hip, knee, head, and ground are the same in both codes (Figure 10). Both codes of rugby abide by similar playing laws (World Rugby, 2017) and it therefore makes sense that impacts would occur in the same manner.

When using the frequencies of reconstructed impacts standardized over 60 minutes of play, an average rugby 7s athlete will receive 12.45 impacts/hour and an average rugby 15s athlete will receive 11.55 impacts/hour. Including the confirmed impacts labeled as "other" event types, which were not reconstructed, the overall rates of impact increase to 18.09 impacts/hour in rugby 7s, and 16.75 impacts/hour in rugby 15s. When the "suspected" impacts for the 5 impact conditions and the "other" category were included, the overall rates of impact increase to 24.03 impacts/hour in rugby 7s, and 20.40 impacts/hour in rugby 15s. Including suspected impacts increases rugby 7s frequency by roughly 30%, and rugby 15s by roughly 20%. Suspected impacts could not be confirmed because either the location, mechanism, or timing was unclear in the video analysis. The confirmed impacts used for the trauma profile provided a conservative measure.

Although the above data demonstrated that rugby 7s have a higher impact frequency, it is believed that a high number of impacts in rugby 15s could not be recorded. The open field tackles for both codes were clear and generally simple to analyze using video. Close contact however is harder to record. A typical ruck in rugby 7s has 3-5 people involved, whereas a typical ruck in rugby 15s can have up to 8-10 people (Quarrie & Hopkins, 2008). The greater number of bodies in the same space makes it harder to clearly see all head impacts. It is possible that rugby 15s has a higher frequency of head impacts occurring in rucks and mauls that are not well accounted for during video analysis due to the nature of the event. Figure 12 demonstrates the scenarios of play, which lead to brain trauma. Rucks accounted for a larger number of impacts leading to low and very low levels of brain trauma. At the medium, high, and very high brain trauma levels, tacking and being tackle are the more prevalent scenario.

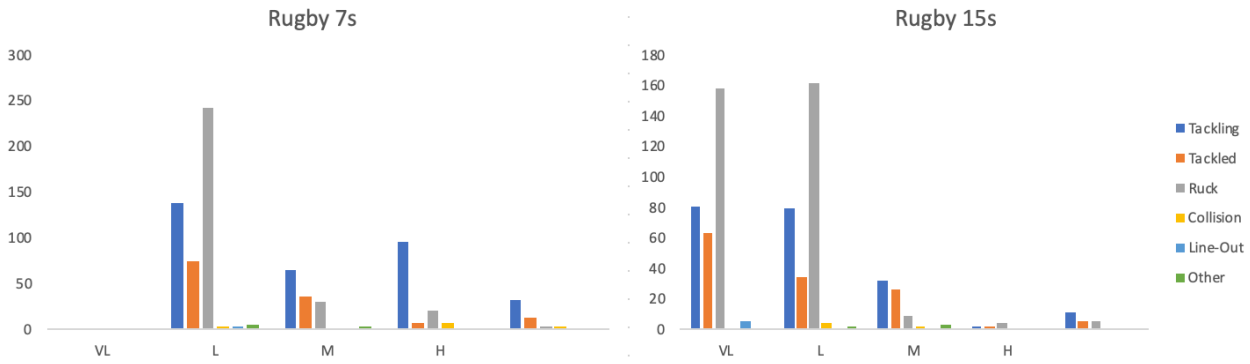

Figure 12. Frequency divided by trauma categories from scenario of play.

Magnitude

The magnitude of brain tissue deformation created by an impact is defined by its impacting characteristics: velocity, mass, compliance, location, and vector. Unique combinations of these five characteristics produce unique brain tissue deformation, which in this study is represented by MPS. Closing velocity and mass are two characteristics that differ between rugby 7s and rugby 15s, which may contribute to the differences in brain trauma profiles. The compliance of each impact event remain the same across sports and therefore it was not a contributing factor in the differences in trauma profiles. The most common locations and associated vectors, were used as criteria for choosing each exemplar. These locations and vectors were different between rugby codes and may be a contributing factor for the differences in trauma profiles.

It was hypothesized that the greater average running speeds of rugby 7s athletes would lead to higher maximal principal strain values when compared to rugby 15s. Athletes in rugby 7s spend 23% more time sprinting (> 5.5 m/s), high intensity running (5 – 5.5 m/s) and striding (4 – 5 m/s) than rugby 15s athletes (Cunniffe et al., 2009; L. J. Suarez-Arrones et al., 2012). In comparison, rugby 15s athletes spend 38.5% more time walking or standing during a match (Cunniffe et al., 2009; L. J. Suarez-Arrones et al., 2012). The higher running speeds in rugby 7s

led to higher closing velocity impacts, which in term led to a higher percentage of impacts causing medium, high, and very high brain trauma.

It was predicted that the greater average mass of rugby 15s athletes would lead to higher maximal principal strain values when compared to rugby 7s. The head to knee impacts, where both location and vector were similar, the 15s impact caused higher strain values (39% at a low velocity impact compared to 33%). This example demonstrated that when location, vector, and compliance are similar, and closing velocities are low, that mass will be more influential on the resulting brain trauma than the velocity (Karton, 2012). Differing mass between rugby 7s players and rugby 15s player does affect their brain trauma profiles, but only in specific situations. Due to the interaction of impacting characteristics the rugby 15s profile does not show an overall trend of greater brain trauma.

Interval

The interval between all confirmed head impacts was 3.54 minutes in rugby 7s, 3.36 minutes in rugby 15s. The interval was also calculated for each trauma category. According to this game-based interval measure, there is no significant difference between the codes of rugby. Due to the high variance of when impact conditions occur, without any real pattern, a game-basis interval measure was not the most distinguishing measure.

With the in-match intervals between rugby codes being similar, the differences lie in the playing format from a day- week- and season-basis. Rugby 15s are in game-play for 80 minutes, with a full week to recover until their following match. Rugby 7s are in game-play for 14 minutes, with a few hours to recover before their following match of the day. The tournament style of rugby 7s means they play up to 4 games in one day, and tournaments lasting 1-3 days (Higham et al., 2012). During the Rio Olympics the final teams played 6 games over 3 days. Research has

demonstrated that longer breaks between head impacts leads to better cognitive outcomes through less symptoms and a shorter recovery period (Meehan et al., 2012). It is unknown in a longer burst with a week rest, or multiple shorter burst with the same or longer rest period is better in terms of brain trauma and injury risk.

CHAPTER 6: CONCLUSIONS

Creating brain trauma profiles for various sports help with understanding the trauma experienced by players, and the injury risk associated with those sports. There were no difference in the distributions of events types occurring between rugby 7s and rugby 15s. When comparing the overall rate of impacts per game, rugby 7s had a higher rate of impacts (18.09 impacts/hour in rugby 7s, and 16.75 impacts/hour in rugby 15s). Therefore, the six null hypothesis related to the frequency of event types causing brain trauma were accepted. The five null hypothesis related to the frequency magnitude of brain trauma were rejected. Rugby 7s experienced more impacts causing higher levels of brain trauma. Three null hypothesis related to interval could not be measured. The null hypothesis for interval at the high MPS category was accepted, and the null hypothesis for interval at the low MPS category was rejected, as rugby 7s had shorter intervals between impacts. Overall, rugby 7s showed a higher rate of head impact, a higher distribution of impacts causing medium, high, and very high maximal principle strain values, and a shorter time interval between impacts causing low MPS values. This information revealed that rugby 7s present more dangerous playing conditions in regards to risk of brain injury when compared to rugby 15s.

6.1 Future Work

To better understand the trauma profile of each sport, future research should add a layer of detail to the profiling, such as creating conditions with each location rather than using the most common location to represent them all. In order to see separation in event type, future

research should look at forwards versus backs within each code. Forwards are known to crash the ball more often and play in close courters such as rucks, scrums, and mauls, whereas backs tend to make the big runs and make the open field tackles. The open field tackles lead to higher brain trauma in this study. Finally, to understand the implications of these results, additional research should include interval, and understanding the difference between a repeated short burst with moderate rest or longer bursts with longer rest intervals.

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Appendix A: Methodology – Video Analysis

Table 1

Definitions and inclusion criteria for the labeling of head impact.

Confirmed	A confirmed head impact is in agreeance with the following 3 conditions: i) an impact is certain and results in visible motion of the head, ii) the impact type is clear, **and** iii) the exact moment of impact can be identified.
Suspected	A suspected head impact occurs when an impact is seen on screen but there is: 1) no resulting head motion of the head visible from the impact, ii) location is unclear, **or** iii) event type is unclear.

Appendix B: Results

Table 10

Number of suspected impacts recorded through video analysis.

		Very Low	Low	Medium	High	Very High
15s						
	Head-to-Head		8		2	
	Head-to-Shoulder	37	18	10		
	Head-to-Hip	49	4			
	Head-to-Ground		8	1		
	Head-to-Knee		6			2
Total		**86**	**44**	**12**	**2**	**2**
7s						
	Head-to-Head		12		2	
	Head-to-Shoulder		104			
	Head-to-Hip		29	23	20	1
	Head-to-Ground		13	10		2
	Head-to-Knee		12		3	
Total		**0**	**170**	**33**	**25**	**3**

*****A total of 74 suspected "other" impacts were also recorded in rugby 15s. There were 44, 23, 6, and 1, recorded impacts at very low, low, medium, and high closing velocities respectively. A total of 145 suspected "other" impacts were also recorded in rugby 7s. There were 102, 25, 16, and 2, recorded impacts at very low, low, medium, and high closing velocities respectively.

Table 11

Description of closing velocities by maxima, minima, mean, and chosen exemplar, per rugby code by MPS category.

	15s				7s			
	very low	low	med	high	very low	low	med	high
Head to Head								
Max	1.1				0.9	2.7		
Min	1.1				0.9	2.7		
Average	1.1				0.9	2.7		
Exemplar	0.92				0.92	2.65		
Head to Shoulder								
Max	1.9	3.4	5.7	8.3	1.95	3.5	5.95	8.35
Min	1	2.05	3.55	6.4	0.4	2.05	3.7	6.25
Average	1.675	2.56	4.76	7.5125	1.35	2.862	4.629	7.128
Exemplar	1.27	2.79	3.95	6.52	1.8	2.23	4.03	8.24
Head to Hip								
Max	1.8	3.25	4.95	8.65	1.85	3.39	5.89	11.8
Min	1.5	2.45	3.8	6.2	0.55	2.15	3.6	6.1
Average	1.68	2.813	4.495	6.86	1.37	2.77	4.553	7.56
Exemplar	1.43	2.47	3.71	6.21	1.96	2.45	4.89	7.14
Head to Ground								
Max	1.75	3.4	5.625		1.917	3.5	4.42	
Min	1.75	2.25	4.76		0.5	2	4.42	
Average	1.75	2.825	5.1925		1.14	2.66	4.42	
Exemplar	1.03	2.98	4.79		1.03	2.98	4.79	
Head to Knee								
Max	0.7	3.35			1.84	3.15		
Min	0.7	3.35			0.8125	2.18		
Average	0.7	3.35			1.45	2.78		
Exemplar	0.7	3.42			1.93	2.99		
Other								
Max	1.75	3.35	5.6	8.5	1.9	3.45	6.35	14.17
Min	0.55	2.4	3.95	8.5	0.25	2	3.55	6.5
Average	1.23	2.9	4.592	8.5	1.17	2.76	4.515	8.85

Table 13a

Dynamic and brain tissue response for each exemplar trial using the Pneumonic Linear Impactor, where a 13.1 Kg arm was used for the Rugby 7s life event reconstructions, and a 16.1 Kg arm was used for the Rugby 15s life event reconstructions.

Event Condition	Trial #	Velocity (m/s)	Linear Acc. (g)	Rotational Acc. (rads/sec^2)	Maximal Principal Strain
Rugby 7s					
Head to Shoulder					
Very Low	1	1.80	6.5	924	0.1006
(S17-L2)	2	1.79	6.2	924	0.1020
	3	1.86	6.4	965	0.1019
Avg		**1.82**	**6.4**	**938**	**0.1015**
SD		0.03	0.12	19	0.0006
Low	1	2.40	9.0	1102	0.124
(S14-R2)	2	2.12	8.0	925	0.1041
	3	2.16	8.0	925	0.1041
Avg		**2.23**	**8.3**	**984**	**0.1107**
SD		0.12	0.47	84	0.0094
Med	1	4.07	14.5	1120	0.1348
(S12-L4)	2	4.07	14.8	1163	0.1339
	3	3.95	14.6	1154	0.1295
Avg		**4.03**	**14.6**	**1146**	**0.1327**
SD		0.06	0.13	18	0.0023
High	1	8.42	49.2	4345	0.4559
(S13-R3)	2	8.15	46.8	4282	0.4481
	3	8.15	53.2	4712	0.4886
Avg		**8.24**	**49.7**	**4446**	**0.4642**
SD		0.13	2.6	189	0.0175
Head to Hip					
Very Low	1	2.10	7.5	1077	0.1598
(S17-R2)	2	1.94	6.7	1078	0.1074
	3	1.84	6.5	1172	0.1096
Avg		**1.96**	**6.9**	**1109**	**0.1256**
SD		0.11	0.43	44	0.0242
Low	1	3.83	16.1	1812	0.1851
(S12-R4)	2	3.60	14.4	1653	0.2153
	3	3.51	13.8	1592	0.1724
Avg		**2.45**	**14.8**	**1685**	**0.1909**
SD		1.73	0.97	93	0.0179
Med	1	5.05	25.6	2133	0.3006
(S14-R2)	2	4.86	24.4	1946	0.2202
	3	4.76	23.3	1971	0.2817
Avg		**4.89**	**24.4**	**2017**	**0.2675**
SD		0.12	0.94	83	0.0343
High	1	7.21	33.7	5925	0.5479
(S14-R2)	2	7.01	34.0	6050	0.5452
	3	7.21	37.1	6546	0.5893
Avg		**7.14**	**34.9**	**6174**	**0.5608**
SD		0.09	1.54	268	0.0202

Head to Knee					
Very Low	1	1.88	51.9	2175	0.1612
(S12-R4)	2	1.97	55.4	2125	0.1700
	3	1.96	53.2	2579	0.1750
Avg		**1.94**	**53.5**	**2293**	**0.1687**
SD		0.04	1.4	204	0.0057
Low	1	2.94	79.0	7260	0.3291
(S13-L3)	2	3.04	81.4	7378	0.3344
	3	3.01	80.4	7175	0.3333
Avg		**2.99**	**80.3**	**7271**	**0.3323**
SD		0.04	0.98	83	0.0023
Rugby 15s					
Head to Shoulder					
Very Low	1	1.28	5.7	384	0.0516
(S8-L3)	2	1.36	6.1	357	0.0489
	3	1.17	5.1	347	0.0514
Avg		**1.27**	**5.6**	**363**	**0.0506**
SD		0.08	0.41	16	0.0012
Low	1	2.81	9.2	888	0.0932
(S7-R4)	2	2.77	9.1	889	0.0927
	3	2.81	9.3	847	0.0837
Avg		**2.79**	**9.2**	**875**	**0.0899**
SD		0.02	0.08	19	0.0044
Med	1	3.95	16.4	2002	0.1954
(S13-L3)	2	3.95	16.0	1995	0.1978
	3	3.95	16.3	1994	0.1963
Avg		**3.95**	**16.2**	**1997**	**0.1965**
SD		0.0	0.17	3.5	0.0009
High	1	6.64	32.2	2589	0.2575
(S9-L2)	2	6.47	33.8	2309	0.2734
	3	6.47	33.0	2111	0.1982
Avg		**6.53**	**33.0**	**2336**	**0.2430**
SD		0.08	0.65	196	0.0323
Head to Hip					
Very Low	1	1.40	3.9	463	0.0604
(S13-L3)	2	1.43	4.2	469	0.0650
	3	1.46	4.4	476	0.0537
Avg		**1.43**	**4.2**	**469**	**0.0597**
SD		0.02	0.2	5.1	0.0046
Low	1	2.48	8.1	704	0.0737
(S4-R2)	2	2.55	8.3	743	0.0781
	3	2.38	7.7	525	0.0662
Avg		**2.47**	**8.0**	**657**	**0.0727**
SD		0.07	0.25	95	0.0049
Med	1	3.66	16.3	1554	0.1533
(S13-L3)	2	3.77	16.8	1634	0.1602
	3	3.71	16.7	1632	0.1626
Avg		**3.71**	**16.6**	**1607**	**0.1587**
SD		0.04	0.22	37	0.0039
High	1	6.16	34.4	3921	0.3963
(S13-R3)	2	6.31	35.8	4041	0.4155

	3	6.16	33.8	3749	0.3948
Avg		**6.21**	**34.7**	**3904**	**0.4022**
SD		0.07	0.84	119	0.0094
Head to Knee					
Very Low	1	0.58	11.0	663	0.0908
(S8-R3)	2	0.72	15.4	997	0.1174
	3	0.80	18.2	1193	0.1273
Avg		**0.70**	**14.9**	**951**	**0.1118**
SD		0.09	2.9	219	0.0154
Low	1	3.46	78.2	3928	0.3825
(S14-R2)	2	3.41	79.0	3990	0.3965
	3	3.41	78.3	4050	0.3947
Avg		**3.43**	**78.5**	**3989**	**0.3912**
SD		0.02	0.35	50	0.0062

Table 13b

Dynamic and brain tissue response for each head-to-head and head-to-ground exemplar trial used to represent both Rugby 7s and Rugby 15s.

Event Condition	Trial #	Velocity (m/s)	Linear Acc. (g)	Rotational Acc. (rads/sec^2)	Duration (ms)	Maximal Principal Strain
Head to Ground						
Very Low	1	0.97	17.4	1233		0.1126
(S13-L3)	2	1.05	22.4	1465		0.1508
	3	1.08	24.4	1521		0.1507
Avg		**1.03**	**21.4**	**1406**		**0.1380**
SD		0.05	2.9	124		0.0179
Low	1	2.93	43.8	2656		0.2562
(S4-L2)	2	2.99	48.2	2698		0.1996
	3	3.04	50.5	2908		0.2221
Avg		**2.99**	**47.5**	**2754**		**0.2259**
SD		0.05	2.8	110		0.0233
Med	1	4.80	94.4	4767		0.3596
(S4-R2)	2	4.80	94.1	4675		0.3545
	3	4.78	93.0	4568		0.3382
Avg		**4.79**	**93.8**	**4670**		**0.3508**
SD		0.01	0.6	81		0.0091
Head to Head						
Very Low	1	0.92	16.2	1951		0.1010
(S19-R2)	2	0.92	17.0	2072		0.1028
	3	0.92	16.8	2055		0.1019
Avg		**0.92**	**16.7**	**2026**		**0.1019**
SD		0	0.3	53		0.0007
Low	1	2.65	107.6	9250		0.3002
(S7-L4)	2	2.65	93.5	9004		0.2712
	3	2.65	97.1	8719		0.2796
Avg		**2.65**	**99.4**	**8991**		**0.2837**
SD		0	5.9	217		0.0122

Table 15

Maxima and minima interval between impacts collapsed, and within each brain trauma category.

Brain Trauma Category	**15s Interval** min/player (SD)		**7s Interval** min/player (SD)	
	Maxima	Minima	Maxima	Minima
Confirmed Impacts	6.45	1.64	6.31	2.21
Very Low	26.17	4.50		
Low	31.70	5.27	17.48	4.59
Medium	62.67	1.00	54.02	14.57
High	0.17	0.0	69.30	0.93
Very High	78.0	24.0	199.73	13.94