
# Sports-related concussions in New Zealand amateur rugby union and league: Identification, assessment and impact forces involved

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**School of Sport and Recreation**

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In conducting this research I was often asked why I wanted to speak out about these types of injuries and why can't I just leave this this topic alone. In response to those many people who questioned my intentions I offer up a quote that guides my decision making process as a health professional and, as a human being.

***"On some positions, cowardice asks the question, is it expedient? And then expedience comes along and asks the question, is it politic? Vanity asks the question, is it popular? Conscience asks the question, is it right? There comes a time when one must take the position that is neither safe nor politic nor popular, but he must do it because conscience tells him it is right."***

***Martin Luther King Jr. 1929-1968***

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## ATTESTATION OF AUTHORSHIP

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements.

Chapters 2 to 8 of this thesis represent separate papers that have either been published or have been submitted to peer-reviewed journals for consideration for publication. My contribution and the contributed by the various co-authors to each of these papers are outlined at the beginning of each chapter. All co-authors have approved the inclusion of the joint work in this doctoral thesis.

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Douglas Alistair King

5th May 2015

## CANDIDATE CONTRIBUTIONS TO CO-AUTHORED PAPERS

| Chapter publication reference | Author % |
|---|---|
| **Chapter 2.** King, D.A., Brughelli, M., Hume, P.A. & Gissane, C. Assessment, management and knowledge of sport-related concussion: Systematic Review. *Sports Medicine* 2014; **44**(4): 449-471 | DK: 80%<br>MB: 7%<br>PH: 7%<br>GC: 6% |
| **Chapter 3.** King, D.A., Gissane, C., Brughelli, M., Hume, P.A. & Harawira, J. Sport-related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs. *Journal of Science and Medicine in Sport* 2014; **17**(3): 250-255 | DK: 80%<br>CG: 5%<br>MB: 5%<br>PH: 5%<br>JH: 5% |
| **Chapter 4.** King, D.A., Gissane, C. & Clark, T. Concussion in amateur rugby league players in New Zealand: A review of player concussion history *New Zealand Journal of Sports Medicine* 2014, **40**(2): 64-69 | DK: 85%<br>CG: 10%<br>TC: 5% |
| **Chapter 5.** King, D.A., Brughelli, M., Hume, P.A. & Gissane C. Concussions in amateur rugby union identified with the use of a rapid visual screening tool. *Journal of Neurological Sciences.* 2013; **326**(1-2):59-63 | DK: 85%<br>MB: 5%<br>PH: 5%<br>CG: 5% |
| **Chapter 6.** King, D.A., Gissane, C., Hume, P.A. & Flaws, M. The King-Devick was useful in management of concussion in amateur rugby union and rugby league in New Zealand. *Journal of Neurological Sciences*. 2015, **351**(1-2): 58-64 | DK: 75%<br>CG: 10%<br>PH: 10%<br>MF: 5% |
| **Chapter 7.** King, D.A., Hume, P.A., Brughelli, M. and Gissane, C. Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches. *American Journal of Sports Medicine,* 2015, **43**(3); 614-624 | DK: 80%<br>PH: 10%<br>MB: 5%<br>CG: 5% |
| **Chapter 8.** King, DA., Hume, P.A, Gissane, C., Brughelli, M. and Clark, T. The influence of head impact threshold for reporting data in contact and collision sports: Consensus needed. *Sports Medicine* (submitted) | DK: 75%<br>PH: 10%<br>CG: 5%<br>MB: 5%<br>TC: 5% |

We, the undersigned, hereby agree to the percentages of participation to the chapters identified above

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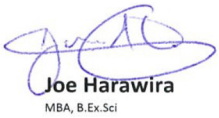


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# ACKNOWLEDGEMENTS

There are many people who have been involved in my progress towards this project and have spurred my interest into research into the game. Unfortunately due to many reasons my first aim was to conduct this research within rugby league but this did not initially eventuate and sadly my first sporting love was left behind in some of the steps undertaken in this thesis to advance player care and welfare. Despite the switching between rugby union and rugby league, many people have remained supportive and encouraging to me in my efforts for this research. These are just but a few.

To my ever loving and patient children Zara, Elena and Nathaniel who for years have only ever seen the back of me as I spent hours on the computer working or I was off gathering data for the many projects I get involved in. Your love, interest and time has allowed me to undertake this second PhD and, despite all that has occurred you have loved me unconditionally and stuck with me in both good and bad days. Forever I am indebted to the time, support, patience and tolerance they showed towards my research and time required for this to be completed.

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And lastly this thesis is dedicated to Mary and Ted Johnson. Sadly Ted, loving husband of Mary and a friend too many league people, left us behind. My lasting memory of Ted will always be the both of us standing on the sideline watching league and enjoying the short time we had together. Ted and Mary believed in me, supported me in my endeavours to achieve lots in the game and always had a positive view on all situations. Ted lost his battle with cancer aged 75 in 2009. The game has lost a true character,

avid supporter and life time league loyalist. Many thanks Ted for the time, opportunities, support and love. Resting in peace and pain free now, Ted will always be with me in my writings and endeavours in rugby league. To Mary, my second mother. A true friend, supporter and a very special lady in my life. Many thanks for your love, support and caring to my children and to me. Without you, I would not have undertaken this challenge.

***Ethical approval***

Ethical approval for this research was granted by the Auckland University of Technology Ethics Committee (AUTEC):

- AUTEC: 12/156 Impact forces associated with match participation in rugby league and rugby union and the identification of sport related concussion.

*“When truth is buried underground it grows, it chokes, it gathers such an explosive force that on the day it bursts out, it blows up everything with it. The truth is on the march, and nothing shall stop it.”*

Emile Zola, "J'Accuse!" L'Aurore, Jan. 13, 1898

# ABSTRACT

What are the costs and frequency of concussions in sports in New Zealand and how can we identify and measure them? The overarching aim of the thesis was to examine sports-related concussions in New Zealand amateur rugby union and rugby league via identification, assessment and impact forces involved in concussion incidents. Methodological approaches included epidemiological analysis, a survey, prospective cohort analysis utilising data obtained from the King-Devick (K-D) saccadic test, the Sports Concussion Assessment Tool 3rd edition (SCAT3), and impact analyses utilising instrumented mouthguards.

Identification of the costs, and epidemiology, of concussions in New Zealand was undertaken by an epidemiological analysis of the Accident Compensation Corporation database, then via a survey of 213 amateur rugby league players. Of 20,902 claims ($NZD 16,546,026) recorded between 1st July 2001 and 30th June 2011, 1,330 (6.4%) were Moderate to Severe Claims. In reviewing 213 amateur rugby league player concussion questionnaires, there were an average of 4.0 ± 2.6 concussive injuries per player in 2010 to 2012.

After-match assessment of players with the K-D test identified 22 concussive incidents over a single competition season for senior amateur rugby union. Although five concussive incidents were witnessed, 17 were unrecognised at the time of the event, but identified post-game with the K-D, and later confirmed via medical assessment by a physician. For every 1 point reduction in the post-injury Standard Assessment of Concussion (SAC) components of the SCAT3, there was a corresponding increase (worsening) of K-D test times post-match for changes in orientation (2.9 s), immediate memory (1.8 s), concentration (2.8 s), delayed recall (2.0 s) and SAC total score (1.7 s).

Identification of the magnitude, frequency and distribution of head impacts in real-time occurring during rugby union matches was evaluated using an instrumented mouthguard. A total of 20,687 impacts >10*g* were recorded during matches over the 2013 domestic club competition season. The mean number of impacts per player was 563 ±618 resulting in 95 ±133 impacts to the head per player, per match.

While trying to compare the results with other published studies, an issue in the way head impact data were reported was identified. A retrospective review was conducted to compare published data reporting impact thresholds obtained from accelerometer systems with data recorded at the 10*g* impact threshold obtained from 38 senior rugby players in New Zealand. The comparison was undertaken to identify the percentage, and number, of impacts that were removed with different reporting thresholds.

This PhD research has contributed knowledge regarding costs of concussions in seven sporting codes; the percentage of concussions medically managed in a cohort of amateur rugby league players; the benefit of a sideline remove-from-play screening tool for concussions; and a description of the magnitude, frequency and duration of impacts to the head in senior amateur rugby union players. This PhD research also identified the need for a consensus approach to the reporting of impact biomechanics

in future studies. As a result of this research a new cyclical model of concussion management has been developed.

# PERSONAL VIEW

Standing on the sideline in 1998, little did I know, or realise, how the events of that day would affect my life and change my direction of what I wanted to be, or where I would end up. Being dragged onto the field of a game I was watching, and holding a player in my arms from a head injury, started a series of events that have culminated in me sitting here today. I was unaware the player passed away when he arrived at the hospital and when I entered the club rooms a short time later I was shunned by those that knew this and the nickname of "Dr Death" stuck with me for the next 10 years.

This incident resulted in my removal from any patient contact when I was a Nursing Officer in the Army while I was investigated about my involvement in the player's death by the national sporting body, the Military Police, the Police and my club. I was eventually cleared of any medical malpractice by the Police, Military Police and my club and informally praised for helping the player but was never placed back into a clinical environment or employed as a Registered Nurse within the military again. To this day I have never been given an apology by the national sporting body for the accusation about my involvement in the death of this player.

Following this tragic event I became involved in trying to raise the awareness of head injuries in rugby league locally and nationally. I became informally involved with the only medical person in the New Zealand rugby league at the time (he was the vice-president) and we formulated the first unofficial 'medical panel' and tried to establish the guidelines for concussion, based on the Cantu guidelines, but I was often disregarded and left out of teams due to the nickname I had inherited and my stance on concussion.

Eventually I left the military and moved to the South Island to go back into Forensic Psychiatric Nursing and to get a fresh start within the game, but the nickname followed me and I had to continually battle against the accusations and the doubt remained with me about my involvement in the game, where I was heading in my future and what it would involve. It took two years for me to find out I was not being blamed for the death of the player through a newspaper article I saw on a clinic wall but the effects of this death, the subsequent nickname and the removal from teams and clubs left me wanting to know why this had occurred and started me on my academic pathway in Sports Medicine. In those days it was hard for a nurse to be seen as anything other than a 'hand-maiden' to a medical doctor and to try to do anything other than nursing courses often required knocking on closed doors or being limited in what I could undertake. Eventually Associate Professor David Gerrard offered me a placed on the Post Graduate Diploma in Sports Medicine course and I finally got a foot in the door.

I had also changed my role to an Emergency Nurse and became more involved in the sideline care of players medically, worked with the New Zealand Rugby League on their trainers' courses and pushed for a more formalised sports medic course to be incorporated into the game. Alongside this I eventually got to work with Daniel Anderson when he was the head coach of the New Zealand Warriors, then

development manager for the New Zealand Rugby League. Through Daniel, and with the help of Dr Chris Hanna, we established a more formal arrangement for a medical panel in rugby league. Unfortunately once this was established I was advised as I was not a medical doctor, nor a sports physiotherapist, I was not deemed appropriate to be included as a member on this panel. I did however get a concussion policy implemented through my association with some members of the panel and this was based on the first Concussion in Sports Group outcomes but the medical panel eventually folded and the personnel at NZRL changed.

During my first PhD I was approached by a new employee of the NZRL to be an adviser on a resurrected medical panel for NZRL, but I was not asked to be a member for the same previous reasons. However I was able to push for, and establish, a more updated concussion in sports guideline, based on the second Concussion in Sports Group guidelines and through my association with some members of the medical panel at the time. I remained as an adviser for several years but was never involved in a more active capacity due to the political environment at the time. I have subsequently made myself unavailable for the medical panel due to ongoing problems and conflicts that have occurred.

In 2010, just after I had completed my first PhD, I was standing on the sideline of a game working with a representative team and witnessed an opposition player hit the ground convulsing following a tackle. My first reaction was one of fear and a feeling of “here we go again”. The players own team management did not know what had occurred, nor how to manage the situation, so I stepped in for the player’s safety. Working with a good friend, and team coach, we managed to keep him alive and had him transferred to the nearest hospital. To this day I still question myself whether I did the right thing by keeping him alive as I am well aware of the consequences of, and potential outcomes related to, second impact syndrome. Following the game I went to the hospital where his mother (a clinical neuropsychologist) advised me this was his fourth head injury this year, the last one a week ago, but he was cleared by a medical doctor to return to play as “it was just a minor knock”. This was the event where I felt more had to be done and I eventually approached the university to see if I could undertake another PhD, but this time focusing on sports-related concussions.

Originally this was more of a medical knowledge focused project but with review of the outline with my supervisors a more formalised structure of the PhD started to take shape. I did complain about what had occurred with that player, and the coach, but when I applied for a national team position I was advised that because I had complained about what had occurred, and not just accepted it, I would not be positioned with any more representative teams again. To this day I have not worked with a representative team and despite volunteering, I have been overlooked being advised that it is best for the game that I not be involved. Although this was a blow to my aspirations to represent my district and my country, I was determined to conduct the research and started to look at ways in which to conduct this and still be involved with teams to see if I can start to make some changes. This involved me changing codes to rugby union, but again the controversy began and people questioned my reasons with comments such

as I was 'scaremongering' over a small injury, through to "he's a league supporter so wants to kill rugby union" being made to me and to those around me. Despite this I continued on with the research and just kept moving on whenever it became untenable to remain with the sport. There were many questions I wanted to research but eventually the research took the shape into the PhD in the thesis.

**Epidemiology of concussion in New Zealand**

There is no formalised collection of player numbers or the injuries that occur, especially concussions, within all national sporting bodies in New Zealand. Most reports on player participation rates, and the number of injuries have used estimated numbers but as many New Zealanders participate in multiple sporting activities the establishment of the numbers required would necessitate the use of inconsistent, and incomplete player data. As a result trying to establish the concussion injury incidence rate was not undertaken.

At the start of the thesis, there were no recent epidemiological studies reporting on sports-related concussion with only estimates being provided that 1.6 to 3.8 million sports-related concussions occur annually in the USA. Recently there has been a New Zealand based study of sports-related brain injury in the general population and that 98% of the sporting injuries were classified as mild traumatic brain injuries, however the costs of these injuries were not reported. In reporting these injuries it was identified that children were more commonly medically assessed than adults but 19% of the injuries identified were not reported in medical notes by health professionals as they focused on other injuries instead. This highlights that even at the health professional level these injuries, in my opinion, are not given the respect they deserve.

With the assistance of Joseph Harawira, access was obtained to New Zealand's national taxpayer funded no-fault injury compensation system administered by the Accident Compensation Corporation (ACC). Data pertaining to the sporting code, age, ethnicity, gender and year of competition and injury entitlement claims was downloaded and analysed. This analysis was utilised to formulate Chapter 3.

**Sport-related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs.**

In reviewing ten years of injury entitlement claims for sports-related concussions in New Zealand in Chapter 3 a snapshot was produced for 1,330 being recorded as moderate to serious. Of concern was that this accounted for 6% of the total claims but accounted for nearly 80% of the total costs. This study provided for the first time a longitudinal view of the costs associated with sports-related concussions in seven sports in New Zealand. This study also highlighted that although the majority of sport-related concussions may be minor in severity, the related economic costs associated with more serious sport-related concussions can be high. The finding that rugby union recorded the most moderate to severe claims in the current study was not unexpected as this is our national game. Of concern was that rugby league recorded a low number of moderate to severe claims but had the highest mean cost per claim

and that New Zealand Maori had the highest total and mean cost per sports related concussion moderate to severe claim.

Having been the team medic for different representative teams, I had collected, as part of the team management role, medical questionnaires on players to enable and identification of potential medical related issues before the players undertook representative activities. The questionnaires were reviewed for the concussion history of players for the previous two years, prior history of concussion and the Post-Concussion Symptom Scale that was used for the establishment of baseline data for players competing in various rugby league competitions. This analysis was utilised to formulate Chapter 4.

**Concussion in amateur rugby league players in New Zealand: A review of player concussion history**

In reviewing concussion history questionnaires from representative amateur rugby league players it was identified that there as an average of four concussions per player over two years. Of concern was that only 8% of these players were medically assessed, 5% completed a three week graduated return to play process and no under 15 yr. old player reported having seen any medical personnel for the concussions they reported. This study provided for the first time the number of players in a small cohort who did not report their concussions, the lack of knowledge of the risks and potential consequences of concussion of players in New Zealand.

**Identification and assessment of concussion**

Concussion is difficult to diagnose and is a challenging injury for the healthcare provider. The publication of the many guidelines, assessment tools and, more recently, the SCAT3 and ChildSCAT3 can cause some confusion for the healthcare provider. This is more so for the non-medical person as most of these assessment tools are reported to be for medical practitioners use only so often the assessment of the concussion is left to the experience, knowledge and awareness of both the person assessing the participant and the honesty of the sports participant. No simple effective tool has been identified, made publicly available, nor supported by any national sporting organisation worldwide. The identification of a simple to use assessment tool is seen as an essential step in assisting the many parents, partners and team management personnel who are not medically trained in enabling them to identify sports participants who may have a concussion and removing them from further participation.

One such tool that was identified was the King-Devick rapid number reading test. Having read an article I came upon when sitting on the computer one night, I found the associated website and rang the number listed and spoke with Steve Devick. Talking with Steve I identified I wanted to try the test out and he mailed a few through to me to play with. This all occurred at the end of the domestic season, and just as we were preparing for the representative season, so these players became my test subjects. Of no relationship to me (I am often asked if I am the King in the King-Devick) this test surprised me in what was identified when using it.

This occurred when we were trying the test out after a game as we were waiting in the airport, and to occupy some time, we tested all the players in an under 15 yr. old representative team. The team manager approached me and told me that three players had slower times when compared with their baseline scores. I discussed the findings with the other team management and no-one saw any indications these players were injured, let alone concussed. I got the three players to repeat the test again and then gave them the PCS component of the SCAT2 to complete and they were indicating that they had possible concussions. These players were subsequently referred for further evaluation by their own healthcare provider and all three were medically assessed as having a concussive injury.

Having seen what the tool could identify I approached both the New Zealand Rugby Union, and the New Zealand Rugby League, with the King-Devick but was shown the door nicely at both places as they had their own assessment procedures and did not want to look at utilising the King-Devick within any teams they managed. Despite this knockback I decided that I would continue to use this as an assessment tool for teams I was the medic for. I have recently learnt that the K-D tool is now being utilised at the professional level of participation for rugby union but only for witnessed concussions. International media coverage has also reported that the tool is now part of the assessment process for the Canadian Football league and the National Football League in America. Conversations with Steve have identified that as a direct result from my, and two other researchers recent published result, these organisations have started to utilise the K-D as their first line assessment process. The initial findings I saw helped develop the process, and formulated the groundwork, for Chapters 5 and 6.

**Concussions in amateur rugby union identified with the use of a rapid visual screening tool.**

Working with a well-established rugby club was an interesting place to be. Trying to raise concussion awareness within this environment was another interesting concept but the club president and premier team coach supported me in conducting some research with the King-Devick. Getting the players to buy into the concept was another interesting challenge in itself but by conducting a prospective observational study of amateur senior domestic rugby union players, the King-Devick test and the SCAT2 were utilised to establish baseline scores for every player before they participated in any competition match. A player self-recall concussion history was also collected as part of the baseline assessment process. These players were then tested after every match with the King-Devick test irrespective of whether they were suspected, or witnessed to have had a concussive injury from match participation. Although five concussions were identified on-field, a further 17 players were identified to have concussive events through the use of the King-Devick. All these players were medically assessed by their own healthcare provider and all players identified were confirmed to have had a concussion.

**The King-Devick test was useful in management of concussion in amateur rugby union and rugby league in New Zealand.**

In reviewing the literature it was identified that the King-Devick had only ever been reported for an event, team, or a club over one competition period. No study reporting on the King-Devick had been undertaken over a longitudinal period. Having completed two years with a rugby union I left the club following the consequences of the study reported in Chapter 7 and worked with a rugby league team at the same level of participation. By combining the data from Chapter 5 with subsequent prospective observational studies, this study was completed over a three year period. Interestingly in the two years of conducting this research with the rugby union side the same number of concussive injuries were recorded but there was a difference in the way in which they were identified. In the second year of the study only two concussions were witnessed and 20 were identified post-match with the King-Devick. The only difference between the first and second years were that the players incorporated more neck strengthening exercises pre-season and as part of the pre-game warm up process. This observation has made me question if neck strengthening really does decrease the risk of concussion or does it simply make the sport look safer by hiding the visible effects of concussive injuries. A surprising finding in this study was the correlation between changes in the Standardised Assessment of Concussion component of the SCAT and the increase or worsening of the King-Devick post-match scores. These changes highlighted the changes that occur with a concussive injury and how using a saccadic reading tool can detect these changes in the areas of attention, spatial and temporal orientation and working memory and how this tool can identify changes in players with no clinically-observed symptoms.

**Impact forces involved**

The definition of concussion identifies that a concussion occurs with "*any disturbance in brain function caused by a direct or indirect force to the head*" and a way to see what these are is to measure that force. With the advances in medical and scientific technology the availability of equipment such as the Head Impact Telemetry System in American football to measure these forces have become more readily available. Although the original concept was to be able to directly identify those players with a high force so they can be medically assessed, the research undertaken in this section produced results that have left me wondering if there is more to this than just medically monitoring players with impact monitors.

By having the technology available for the measuring of the players in non-helmeted sports such as rugby union with an instrumented mouthguard enabled a new world of information to open up in terms of concussion research. Undertaking this research on a team participating in the national sport of New Zealand was going to be controversial to say the least but the results, and the subsequent reactions to these results have left me somewhat shocked and dismayed. The use of an instrumented mouthguard in an amateur domestic rugby union team had never been done before and this created some media coverage and had people starting to ask a lot of questions about what was occurring and how this will shape the future of this, and other, contact sports. The XGuard was utilised for the collection of linear and rotational accelerations and the resultant analysis was utilised for Chapters 7 and 8.

**Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches**

I initially asked the question of a medical official of a sporting code "how many impacts are there in a game?" I was told, repeatedly, that there are "not many impacts to the head and it's a safe game to play". So I wanted to find out more about what was occurring. Through the identification of the instrumented mouthguard from the United States of America the research was undertaken. Having pre-fitted the mouthguards for the senior amateur domestic rugby union team, the season involved monitoring the players for 19 competition games. I had the support of the team, the management and the club and was constantly in contact with them over the findings each week. This same cohort were also involved in the first two years of the studies resulting in Chapters 5 and 6. The number of impacts per player position was higher than that reported in previous studies for American football at a similar level of age participation yet the average forces involved were similar to the American football results. So my question was answered with the results that there are a large number of impacts to the head in rugby union, and therefore the question remains, is the game really that much safer than American football?

Having seen these results I was left to wonder what had I done in my sporting career. I have played rugby league and rugby union for a better part of 40 yrs. and have vivid memories of the effects of the concussions I have had throughout these playing years. My memory of my daughter at age four sitting on me on the sideline grabbing me and telling me "No daddy" as I tried to get back in the game from being knocked out in the second half of a match, and I had no memory of the game starting, remains with me today. What's even worse was I was left to drive myself and both my kids home on a journey of over 100 kms. Looking back I'm horrified that I actually did this, but worse still, I was allowed to do this by my team mates and the team management. Had I known then, what I know now, then maybe I would have stopped the "harden up" attitude I played with, and encouraged other players to undertake, when we took a bell ringer, shook out the cobwebs from our heads and carried on playing.

**The influence of threshold limitations in the reporting of impact data for concussion risk: Consensus needed.**

In conducting the research in Chapter 7, I identified that there were a lot of inconsistencies in the published research reporting on head impact telemetry. These inconsistencies caused me to have to limit what, and how, the data were reported when I tried to do comparisons with the other published studies. In sports such as rugby union, soccer and cricket there are consensus statements identifying what, and how, the injury data should be reported on. Previously I had a paper published where we suggested guidelines for the reporting of rugby league injuries, although this was not by consensus, but at least there was a standardised format which enabled inter-study comparisons to be undertaken. This was not the case for the reporting on head impact telemetry.

The future ahead for this technology appears to be growing and within the research I have established links with other researchers worldwide. With some studies reporting average and standard deviations for the forces recorded and some reporting the median, with interquartile ranges, the comparisons were somewhat limited and, in some cases, difficult to complete. Add in different linear thresholds and the comparisons become more complex which, if the data in Chapter 7 was to be compared with all these studies, the tables would have been very complex. By completing this chapter it is hoped that a start point for a consensus is established in terms of reporting standards, whereby future studies reporting on head impact telemetry is standardised enabling cross sport inter-study comparisons can be undertaken.

**Summary**

Although the thesis has been conducted to the best of my abilities there were several limitations identified and reported. These limitations can be attributed to the limited support available for a researcher undertaking a controversial topic such as head injuries in the sporting environment. Although some people are very supportive of this type of research being undertaken, there are those that will endeavour to limit its effects and will try to stop such activities being undertaken. An example of some of these experiences are in the quotes I incorporated within the thesis.

I have been asked if all that I have been through has been worth conducting this research. When I reflect back on how I can help one person at a time then I can only answer that yes it had. The events I have had before undertaking this research steadied my resolve to conduct the research. The events I have encountered when the research results were made public have been a real learning curve and I feel I am better prepared for what lies ahead. I now wear with pride the fact that I have three life bans from clubs for my stance on concussion and despite the abuse I will not back off my stance on how I manage concussion.

A benefit of this research was the establishment of a policy, now being incorporated into three district health boards' emergency departments, looking at the management of concussion. The development of this policy has seen new information provided to patients who present with a concussion in the form of return to work, school and sport guidelines, the promotion of cognitive rest and the standardised use of a concussion assessment tool, the SCAT3 and ChildSCAT3. This is a first for New Zealand and is slowly being adopted further afield.

In summary this thesis has addressed the question of what is the epidemiology and associated costs of rugby code-related concussions in New Zealand, is there a sideline tool that can assist with the identification of concussion and what are the impact forces from match participation in amateur rugby union by describing the prevalence, costs associated, identification of unwitnessed concussions and the forces involved. In preparing for my PhD oral examination it helped me reflect back on why I undertook this research and why I am still wanting to persevere with future studies looking at impact forces and

concussion identification. The feedback from the reviewers of the manuscripts that have been published have helped to further enhance the quality of the thesis.

*"Stop looking for injuries that are not there – Players just have to harden up"*

Amateur Representative Coach

# CHAPTER 1: INTRODUCTION AND RATIONALISATION (PREFACE)

## Background

The most commonly cited framework in sports injury prevention is the sequence of injury prevention framework.[602] This framework (see Fig 1) outlined a four stage approach towards sports injury prevention representing a translation of the standard health prevention model to the sports injury context.[166] When this framework was first published, the aspects of the first (extent and severity of injury) and second (aetiology) stages of the four stage framework were the primary focus in the development towards injury prevention programs.

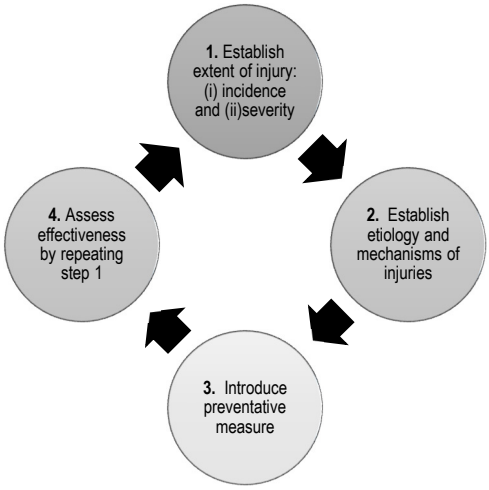


**Figure 1:** The four steps in the sequence of prevention of sports injuries.[602]

More recently there has been an increase in epidemiological studies contributing information to the first two steps of the sequence of prevention of the sports injury model: (1) Establishing the extent of the injury problem (injury incidence), and (2) Establishing the aetiology and mechanisms of injury (mechanisms involed and factors provoking the injury).[75] To understand the importance of the problem it is necessary to know the injury profile for the sporting activity, such as the injury frequency (i.e. the number of injuries per 1,000 hours of training or match activities), location of the injury site (i.e. anatomical body site), the severity of the injury (i.e. transient, mild, moderate or severe depending on injury definition) and the typology of the injuries that occur.[172, 173] Although widely published, the sequence of prevention of sports injuries model has been clearly demonstrated[60, 166, 373, 560] to be of limited use for sports, as well as causation of sports injuries with more recent models being developed.[445, 446]

Building on the sequence of prevention of sports injuries, a multifactorial linear model was identified for the investigation of sports injuries.[445] This model proposed an indefinite number of intrinsic risk factors (see Table 1) that may predispose an individual to injury. As a result of this predisposition, extrinsic factors (exposure to factors of risk) (see Table 1) could exert an influence on this predisposition resulting

in an injury occurring. Given this predisposition, the injury that occurs is a result of a further "initiating event". In terms of a sports-related concussion this would require a collision where the force is transferred to the head. The initiating event may be observed and the resulting injury witnessed, but little attention may be given to the factors that are more distant from the event, e.g. the susceptibility of the indivual to the injury.[229]

**Table 1:** Published intrinsic and extrinsic risk factors for sports injury

| Intrinsic Risk Factors | Extrinsic Risk Factors |
|---|---|
| Physical characteristics | Exposure[384] |
| Age[384, 612] | Type of sports[384] |
| Sex[384, 612] | Playing time[384] |
| Somatotype[384] | Position in the team[384] |
| Body size[612] | Level of competition[384] |
| previous injury[384, 612] | Warm-up[612] |
| Physical fitness[384] | Personal equipment[612] |
| Joint mobility[384, 612] | Training[384] |
| Muscle tightness[384, 612] | Coaching[612] |
| Ligamentous laxity[384] | Refereeing[612] |
| Malalignment of lower extremities[384, 612] | Control of game[612] |
| Dynamic strength[612] | Opponents |
| Static strength[612] | Foul play[612] |
| Skill level[612] | Opponent's physique[612] |
| Psychological characteristics[384] | Environment[384] |
| Psychosocial characteristics[384] | Type and condition of playing surface[384, 612] |
| Skill level[612] | Weather conditions[384, 612] |
| Willingness to take risks[612] | Time of day[384] |
| Interaction with other players[612] | Time of season[384] |
| Experience of sport[612] | Equipment |
| | Protective equipment[384] |
| | Footwear[384] |
| | Orthotics[612] |

Taking the multifactorial linear model further, a cyclical operational model for the investigation of contact sports events was identified.[229] This model[229] expanded the traditional approach of epidemiological investigation of sports injuries and took into consideration the multifactorial nature of sports injuries that may predispose the contact sports participant. Consisting of five linked stages, the model[229] allowed for the development of appropriate strategies for the prevention of injury at the primary, secondary and tertiary levels of care and to further develop descriptive and analytical epidemiological approaches to sports injury research.

Building on the cyclical operational model for the investigation of contact sports events,[229] the multifactorial model of athletic injury etiology[445] was revised and a dynamic, recursive model of etiology in sport injury was produced[446] (see Fig 2). This model allowed for the recursive nature of exposure and how these can alter the risk factors changing the individuals predisposition to injury. As a result, the

individual may be exposed to the same, or a varying set of extrinsic risk factors and have a different susceptibility as a result of this exposure.

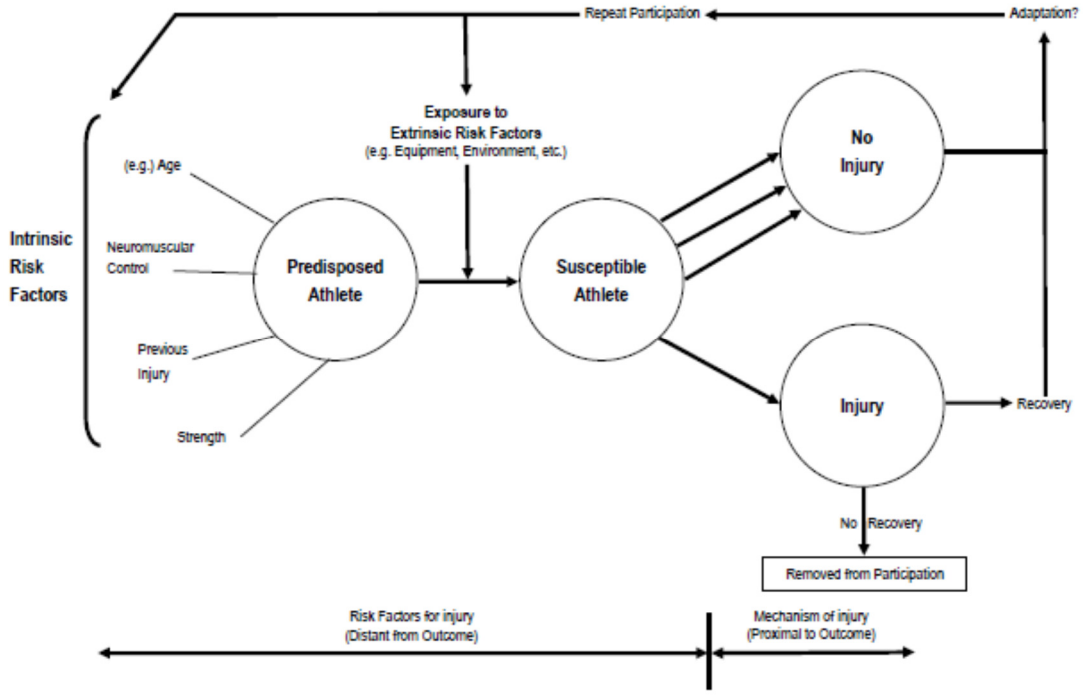


**Figure 2:** Dynamic, recursive model of etiology in sport injury[446] (reused with permission: Clin J Sports Med: 3566850397412).

The cyclical operational model for the investigation of contact sports events[229] and recursive model of etiology in sport injury[446] contain a loop-back system. However, what is not identified in these models, in terms of sports-related concussions, are those injuries that may go un-reported or un-witnessed. Sports participants who do become injured and, for many reasons, do not report these injuries,[280, 380, 409, 522, 620] may be unaware of the injury occurring,[96, 353, 402] or may not know what these injuries present like,[148, 298, 331, 477, 576, 577, 601] and therefore can be re-exposed to injury. In these cases the participants may suffer a more serious injury that may not be evident for many years such as depression,[250] mild cognitive impairment,[249] prolonged recovery from subsequent concussions,[105, 251] electrophysiological changes[123] and chronic traumatic encephalopathy (CTE)[435] but to date there have been no direct causal relationships identified to support these relationships.[427]

The under- and/or non-reporting of concussion makes identifying the true incidence of these injuries difficult. In addition the diagnosis and management of concussion is a challenge. The longitudinal effects of concussive, and sub-concussive, impacts to the head are now being reported,[105, 123, 249-251, 435] but there is still no direct causal link between these concussive events[427] and the long term effects. There is a building body of evidence[209, 435, 437, 572, 573] that concussions may well have a serious effect on the individual, their family, the sport and society in general.

As shown in the dynamic, recursive model of etiology of sports injury (see Fig 2), it is identified that if the player is injured they either undergo recovery towards adaption or they do not recover and are removed from participation. But in the case of sport-related concussion these injuries may not be identified or reported and the participants are re-exposed to the extrinsic factors. Although this model did establish the groundwork for the undertaking of this thesis, what is missing within the dynamic, recursive model of etiology of sports injury is where to position a capture point for these types of injuries, and how this would occur. In addition to identifying this capture point, and focusing on non-reported and non-witnessed sports-related concussive injuries, it was identified that there are other areas that there is a paucity of information: (1) Epidemiology of sports-related concussion in New Zealand; (2) Use of sideline assessment for the rapid identification and removal of a concussed player; and (3) Impacts that occur from participation in amateur rugby union and rugby league.

These gaps in the knowledge lead to the formulation of the thesis question: "What are the costs and frequency of concussions in sports in New Zealand and how can we identify and measure them?"

The thematic sections to address the thesis question include:

Section 1: Review published literature for concussion;

Section 2: Describe the epidemiology of concussion injuries in New Zealand;

Section 3: Examine the assessment of concussions; and

Section 4: Examine impacts that occur as a result of participation in amateur rugby union.

**Structure**

The thesis is structured as a series of related chapters (see Fig 3) that culminate in an overall discussion (Chapter 9). Most of these chapters have been submitted for publication in journals which has allowed the author to gain international peer reviewed feedback on the content. As a result of this international feedback the relevant chapters have been improved. Two technical report's (see Appendix 1 & 2) have also been provided to New Zealand Rugby League (Appendix 1 & 2) and the National Rugby League (see Appendix 2) providing them with feedback useful for their injury prevention programmes, and to gain feedback to aid in development of projects for later chapters.

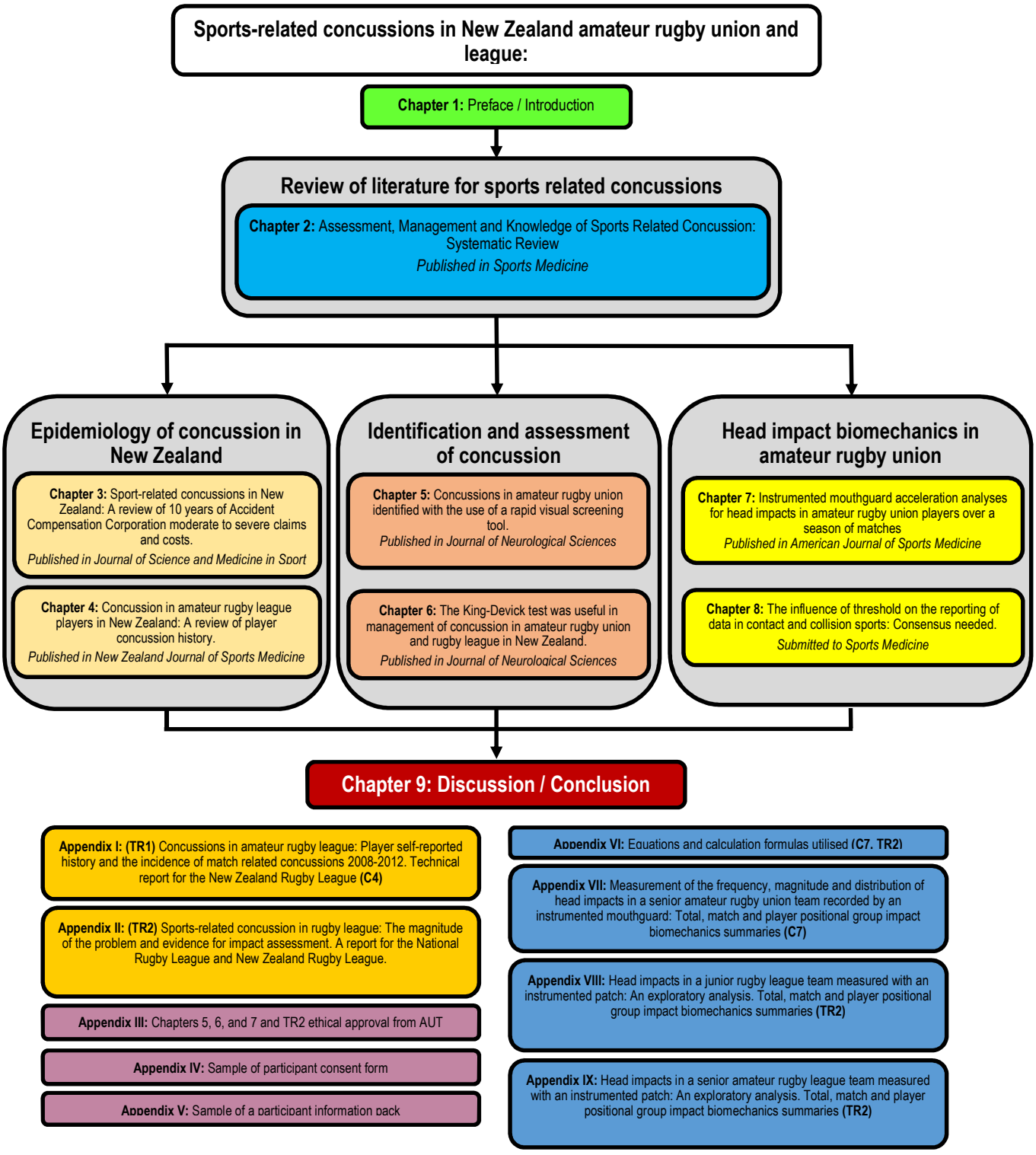


**Figure 3:** Overview of doctoral thesis chapter flow.

The first thematic section of the thesis (Chapter 2) is focused on reviews of literature for sports-related concussion. Chapter 2 (published in *Sports Medicine,* 2014) comprises a review of the literature on concussions by updating the descriptive data on the history, pathophysiology, recognition, assessment, management and knowledge of concussion. This chapter also discusses the issues surrounding the under-reporting of concussions, the confusion and lack of awareness of the many published guidelines available for assessment, management and return-to-play process for concussion. The publication resulting from Chapter 2 was:

King, D.A., Brughelli, M., Hume, P.A. & Gissane, C. Assessment, management and knowledge of sport-related concussion: Systematic Review. *Sports Medicine* 2014; **44**(4): 449-471.

The second thematic section of the thesis (Chapters 3 and 4) examined epidemiology of concussion in New Zealand. Chapter 3 (published in *Journal of Science and Medicine in Sport,* 2014) reports the total and mean costs of Accident Compensation Corporation (ACC) moderate-to-serious injury entitlement claims (MSC) for sports-related concussion over a 10 year period for the seven most frequently reported sports activities. Chapter 4 (published in *New Zealand Journal of Sports Medicine,* 2014) reports a review of club and representative amateur rugby league player concussion history over a two year period. Publications resulting from Chapters 3 and 4 were:

King, D.A., Gissane, C., Brughelli, M., Hume, P.A. & Harawira, J. Sport-related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs. *Journal of Science and Medicine in Sport* 2014, **17**(3); 250-255.

King, D.A., Gissane, C. & Clark, T. Concussion in amateur rugby league players in New Zealand: A review of player concussion history *New Zealand Journal of Sports Medicine* 2014, **40**(2): 64-69.

The third thematic section of the thesis (Chapters 5 and 6) examined the identification and assessment of concussion. Chapter 5 (published in *Journal of Neurological Sciences,* 2013) reports the use of the King-Devick test in association with the Mayo Clinic and Sports Concussion Assessment Tool 2 in amateur senior rugby union players for the identification and assessment of sports-related concussion. Chapter 6 (published in the *Journal of Neurological Sciences*, 2015) reports the use of the King Devick test in association with the Mayo Clinic in the identification and assessment of concussion in amateur rugby union and rugby league players over three years. Publications resulting from Chapters 5 to 6 were:

King, D.A., Brughelli, M., Hume, P.A., & Gissane C. Concussions in amateur rugby union identified with the use of a rapid visual screening tool. *Journal of Neurological Sciences* 2013; **236**(1-2): 59-63.

King, D.A., Gissane, C., Hume, P.A., & Flaws, M. The King-Devick test was useful in management of concussion in amateur rugby union and rugby league in New Zealand. *Journal of Neurological Sciences,* 2015. doi: 10.1016/j.jns.2015.02.035.

The fourth thematic section of the thesis (Chapters 7 and 8) examined impact forces involved. Chapter 7 (published in *American Journal of Sports Medicine,* 2015) reports the frequency, magnitude and distribution of impacts to the head in senior amateur rugby union players measured with an instrumented mouthguard. Chapter 8 (submitted to *Sports Medicine*) reports the differing methods utilised in reporting head impact biomechanics in sports-related concussions and how they limit inter-study comparisons. The publication resulting from Chapter 7 was:

King, D.A., Hume, P.A., Brughelli, M. and Gissane, C. Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches. *American Journal of Sports Medicine,* 2015 **43**(3); 614-624.

The manuscript submitted for publication resulting from Chapter 8 was:

King, D.A., Hume, P.A., Gissane, C., Brughelli, M. & Clark, T. The influence of threshold limitations in the reporting of impact data for concussion risk: Consensus needed. *Sports Medicine (submitted).*

Chapter 9 consists of a general discussion of findings from the presented research projects, comments on limitations of the research studies, provides suggestions for future research, and provides concluding statements on the key findings from the thesis. The new model for cyclic management of concussion, developed as a results of the thesis work, is presented.

The appendices contain material for the chapters that were presented as technical reports to New Zealand Rugby League (Appendices I & II) and the National Rugby League (Appendix II) or as supplemental data (Appendices VII, VIII & IX). A sample participant consent form and information pack are provided in Appendices III and IV. Appendix V contains notifications from the Auckland University of Technology Ethics Committee (AUTEC) regarding ethical approval where required.

The research key points and the links between each chapters in the four thematic sections of the research are outlined in Table 2.

**Table 2:** Research key points from each chapter and the links between each chapter and the four thematic sections of the research conducted.

| **SPORTS-RELATED CONCUSSIONS IN NEW ZEALAND AMATEUR RUGBY UNION AND LEAGUE: IDENTIFICATION, ASSESSMENT AND IMPACT FORCES INVOLVED** |
|---|

| **Outcome:** The thesis identified the epidemiology and costs associated with sport-related concussion in New Zealand. The use of a saccadic sideline reading tool was able to identify both witnessed and un-witnessed concussions in junior and senior contact sports players. Instrumented mouthguards were used to quantify the frequency, magnitude and distribution of head impact biomechanics in amateur senior contact sports participants. Suggested standards are provided for reporting head impacts in sports for future studies. A new model for cyclic management of concussion was developed. |
|---|

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| **1** | **Introduction.** | **MAIN QUESTIONS OF THE THESIS:**<br>1. What are the associated costs of sports related concussion in New Zealand through amateur sports participation?<br>2. What sideline tools are available for use by non-medical personnel for the assessment of sport-related concussion?<br>3. What impacts to the head occur from participation in amateur rugby league and union at junior and senior levels of participation?<br>**RATIONALE FOR THE QUESTIONS:**<br>The key outcomes of the thesis were to:<br>(a) Provide an overview of the costs of sport-related concussion in New Zealand;<br>(b) Identify a sideline assessment tool that can be used as part of the continuum of care for the assessment and management of sports related concussion; and<br>(c) Identify the impacts to the head associated with rugby league and union. |

**Section 1: Review of Literature**

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| **2** | **Review of Literature. Assessment, management and knowledge of sports-related concussion: A systematic review** | **QUESTION:**<br>What does the literature say about the assessment, management and knowledge of sports-related concussion?<br>**APPROACH:**<br>Systematic literature review.<br>**FINDINGS:**<br>• Under-reporting of concussion makes identifying the true incidence difficult and makes diagnosis and management a challenge;<br>• Pathophysiological effects of concussion can be seen several weeks post-injury;<br>• Due to the increased metabolic dysfunction of the brain as a result of concussion, the brain has an increased vulnerability to other consequences;<br>• Numerous definitions, methodologies and reporting modalities have been utilized in the reporting of concussion;<br>• A definition and assessment tool has been produced by the Concussion In Sports Group but is not universally utilized or accepted;<br>• Recent tools such as the Sports Concussion Assessment Tool 3rd edition (SCAT3) and ChildSCAT3 have been produced;<br>• New tools such as the King-Devick in association with the Mayo Clinic test have been used successfully to identify concussion; and<br>• The use of head impact accelerometry for recording the magnitude, duration and frequency of impacts to the head has been shown to be an effective measurement tool.<br>**NOVEL CONTRIBUTION:**<br>Statements regarding the current knowledge of the pathophysiology, assessment, management and education of sport-related concussion. |

*Link between Chapters 1 & 2:*

Having identified gaps in the literature regarding the magnitude of the problem, assessment, management and knowledge of sports-related concussion, the first step was to quantify the magnitude of sport-related concussion in New Zealand.

**Section 2: Epidemiology of concussion in New Zealand**

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| 3 | **Sports related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs** | **QUESTION:**<br>What is the epidemiological overview of sport-related concussion and associated costs over 10 years in New Zealand?<br>**RATIONALE FOR THE QUESTION:**<br>To provide information on the magnitude of the concussion problem in sport in New Zealand.<br>**APPROACH:**<br>Detailed descriptive epidemiological data analysis was provided including costs associated with treatment for sport-related concussion in seven sports with comparisons by gender, ethnicity and age.<br>**FINDINGS:**<br>• Over 2001-11 there were 20,902 sport-related concussion claims from seven sports codes costing $16,546,026.<br>• Rugby union ($6,252,870) and rugby league ($4,572,625) recorded the highest total costs over 2001-11.<br>• Males participating in rugby league recorded the highest mean costs per MSC claim ($26,182).<br>• Females participating in netball recorded the highest total ($833,530) and mean costs per female MSC claim ($12,080).<br>• New Zealand Māori recorded the highest mean costs per MSC claim for rugby league ($43,604) and rugby union ($14,186).<br>**NOVEL CONTRIBUTION:**<br>Identification of the costs of sport-related concussions in New Zealand for seven sports codes. Previous studies had reported an estimated cost in total but had not established the costs per sport, per person and by gender. |

*Link between Chapters 3 & 4:*

Having identified the costs of sports-related concussion it was important to identify the player perspective of what occurs with the identification, assessment and management of sports-related concussion.

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| 4 | **Concussion in amateur rugby league players in new Zealand: A review of player concussion history** | **QUESTION:**<br>To review self-reported concussive history of amateur rugby league players to identify concussions within a two year period, concussive events prior to this time frame and medical review and clearances for return-to-play.<br>**RATIONALE FOR THE QUESTION:**<br>Players with a concussive injury returning to their sport are at a greater risk of complications. These complications are related to subsequent concussive events and may result in prolonged concussive symptoms and cumulative cognitive deterioration. Often the identification of a sport-related concussion is left to the team coach as there are seldom any medical personnel available at the games and training sessions. As a result concussive signs may be missed, especially if the player does not know they have had a concussion or they do not want to report they have suffered a concussion.<br>**APPROACH:**<br>Survey of players.<br>**FINDINGS:**<br>• There was an average of 4.0 ±2.6 concussive injuries per participant in the previous two years.<br>• A total of 7.5% of all participants reported seeing a medical doctor for their reported concussion.<br>• Only 5.2% completed the required three week return-to-play process.<br>• Few players (2.8%) reported seeing a medical doctor for a clearance for return-to-play.<br>• Despite 39.0% of players reporting they did not have a concussion in the previous two years they did report they had loss of consciousness and "bell ringer" / "dings" from match participation.<br>**NOVEL CONTRIBUTION:**<br>Identified the number of players who did not report their concussions, the lack of knowledge of the risks and potential consequences of concussion in players in New Zealand for the first time. |

*Link between Section 1 and 2:*

Having established the costs associated with sport-related concussion, and the number of unreported concussions, it was beneficial to identify a tool that would to be able to assist with the identification of concussive injuries for players at all levels.

**Section 3: Identification and assessment of concussion**

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| 5 | **Concussions in amateur rugby union identified with the use of a rapid visual screening tool** | **QUESTION:**<br>To utilise the King-Devick test (K-D) with the SCAT2 in amateur rugby union players over a domestic competition season to determine if it could identify witnessed and incidentally identified episodes of concussion that occurred from match participation.<br>**RATIONALE FOR THE QUESTION:**<br>Originally developed as a reading tool to assess the relationship between poor oculomotor functions and learning disabilities, the K-D uses a series of charts of numbers that progressively become more difficult to read in a flowing manner. Poor oculomotor function has been reported as one of the most robust discriminators for the identification of a mild-traumatic brain injury.<br>**APPROACH:**<br>Prospective field based data collection over one season for one rugby union team.<br>**FINDINGS:**<br>• There were 22 concussive incidents recorded over the duration of the competition. Five were witnessed, 17 were unwitnessed (later confirmed by medical diagnosis) yet were identified post-game with the K-D. This resulted in a 1:3 ratio of witnessed to unwitnessed concussion being recorded.<br>• Unwitnessed concussions identified with the K-D recorded on average fewer symptoms, lower symptom severity, better balance examination and better immediate and delayed memory scores than witnessed concussions.<br>• Witnessed concussions recorded, on average, a significantly longer K-D on the day of injury than unrecognised concussions when compared with their baseline K-D.<br>• Most, but not all, players with an unrecognised concussive injury had their K-D equal to their baseline K-D by day 14 of the required stand-down period.<br>**NOVEL CONTRIBUTION:**<br>The first published study on sideline assessment of concussion in amateur rugby union. Identification of the 1:3 ratio of witnessed to un-witnessed concussions that occurred in match play for amateur senior rugby union. |

*Link between Chapters 5 & 6:*

Having identified that the K-D test can assess senior amateur rugby union players, it was necessary to identify whether the K-D sideline concussion tool would be useful for other contact sporting activities.

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| 6 | **The King-Devick test was useful in management of concussion in amateur rugby union and rugby league in New Zealand** | **QUESTION:**<br>To examine the worth of the K-D test as part of a sideline assessment process of players participating in contact sport over a three year period.<br>**RATIONALE FOR THE QUESTION:**<br>It has been reported that a larger scale research over a longer period of time may provide increased validity of the K-D test as part of a continuum of concussion assessment tools.[395, 603] The inclusion of a visual dimension tool such as the K-D test may assist in increasing the capacity for the identification of concussed players.<br>**APPROACH:**<br>Prospective field based data collection using the K-D over several seasons for senior amateur rugby union and rugby league teams.<br>**FINDINGS:**<br>• A total of 52 (8 witnessed; 44 unwitnessed) concussive events were identified over the duration of the study.<br>• There was a six-fold difference between witnessed and unwitnessed concussions recorded.<br>• For every 1 point reduction in each of the post-injury SAC components there was a corresponding increase (worsening) of K-D test times post-match for changes in orientation (2.9 s), immediate memory (1.8 s) concentration (2.8 s), delayed recall (2.0 s) and SAC total score (1.7 s).<br>• The ICC between the first and second baseline tests were 0.89 (2012), 0.89 (2013), 0.94 (2014) and 0.92 (combined).<br>• Over the duration of the study the K-D test had sensitivity of 0.92 (95% CI 0.79 to 0.98); specificity of 1.00 (0.94 to 1.00), and kappa of 0.98 (95% CI: 0.94 to 1.00).<br>**NOVEL CONTRIBUTION:**<br>This is the first longitudinal study on the use of the K-D. Using the K-D test, as part of a continuum for the assessment and monitoring of players with a concussion, can assist healthcare providers to evaluate adequate cognitive rest and inform clinical decisions regarding return-to-play and return-to-academic activities. |

*Link between Section 3 & 4*

Having identified the worth of the K-D test as part of sideline assessment, and the incidence of sport-related concussion in junior rugby league and amateur senior rugby league and rugby union, it was important to identify what impacts to the head actually occurred during match participation in amateur rugby union and rugby league.

**Section 4: Examine impacts that occur as a result of participation in amateur rugby union and rugby league**

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| 7 | **Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches** | **QUESTION:**<br>What is the frequency, magnitude, distribution of head impacts in senior amateur rugby union players from match participation over a season of matches?<br>**RATIONALE FOR THE QUESTION:**<br>Concussion is now reportedly the most common injury in rugby union and, due to the nature of the game, there are a lot of contacts. But what is the frequency and magnitude of head contacts?<br>**APPROACH:**<br>Prospective field based data collection using an instrumented mouthguard over a season for a rugby union team.<br>**FINDINGS:**<br>• A total of 20,687 impacts to the head over 10*g* (range 10.0-164.9*g*) were recorded over the duration of the study.<br>• The mean number of impacts per player over the duration of the season of matches was 564 ±618 resulting in a mean of 95 ±133 impacts to the head per player, per match over the duration of the season of matches.<br>• The mean impacts per match were 1,379 ±578 resulting in a mean of 77 ±42 impacts to the head per player position, per match.<br>• The hooker recorded a mean linear acceleration of 27 ±19*g* per match.<br>• The open-side flanker recorded a mean of 4,472 ±4,085 rad/s$^2$.<br>• There were 181 impacts recorded above the linear impact threshold and 4,452 impacts above the rotational impact threshold.<br>**NOVEL CONTRIBUTION:**<br>The first publication of impacts to the head in an amateur senior rugby union team. Identification of the frequency and magnitude of head impacts. Amateur senior rugby union players recorded more impacts than for American football collegiate and high school players. |
| | *Link between Chapter 7 & 8:* | The reporting of head impacts varies depending on the study methodology making inter-study comparisons difficult. Similar to studies reporting injuries in rugby union, football and rugby league, there is a need for a standardization of recording and reporting of the findings from these studies. To date, no consensus has been established for reporting of head impacts in sport. |

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| 8 | **The influence of threshold on the reporting of impact data in contact and collision sports: Consensus needed** | **QUESTION:**<br>The differences observed in studies reporting head impacts in sports make inter-study comparisons limited. What would be a suggested guide for the reporting of head impacts in future studies?<br>**RATIONALE FOR THE QUESTION:**<br>Reporting of head impact data is important to help understand the biomechanics of head injury and help develop potential injury prevention strategies. However, there is currently no standardized format for reporting head impacts in sports to enable comparison between studies.<br>**APPROACH:**<br>Literature review critique and application of thresholds identified from the literature to NZ data.<br>**FINDINGS:**<br>• A total of 47 publications were identified that reported head impacts in sports.<br>• A third (39%) of the studies reported impacts using the 10*g* data threshold.<br>• The majority of studies (91%) reported resultant linear accelerations while slightly less (76%) reported resultant rotational accelerations.<br>• A quarter (26%) of studies reported the Head Impact Telemetry severity profile (HITsp) and 10 (22%) studies reported the Head Impact Criterion (HIC) for 15ms ($HIC_{15}$).<br>• More than half (52%) of the studies reported the head impact data as mean ± standard deviation (±SD).<br>• Some studies also reported the head impacts as median but not all included the interquartile ranges (IQR) for these data.<br>• All of the studies reviewed identified the number of impacts that occurred. However, this varied for match only, match and training and combined but did not identify what the number of match and training impacts were.<br>• By reporting both linear and rotational accelerations there is an improved correlation between impact biomechanics and concussion, than when linear accelerations are reported alone.<br>• Studies reporting at the data threshold above 10*g* may have removed 2,100 to 206,573 impacts when applied to NZ data.<br>**NOVEL CONTRIBUTION:**<br>Suggested reporting standards for studies of head impacts in sports. |

| Chapter No. | Chapter Title | Chapter Content - Question/Rationale/Findings |
|---|---|---|
| 9 | **Conclusions / Recommendations .** | **CONCLUSION:**<br>The key outcome of this thesis was to identify the costs associated with sport-related concussions, to identify a sideline screening tool for the identification of sport-related concussion, and to identify the frequency, magnitude and distribution of head impacts in rugby union and rugby league in New Zealand. A result of undertaking this thesis was the identification of the need for consensus for the reporting of head impacts in sports for future studies. The K-D test was useful in the identification of witnessed and unwitnessed concussions<br>**RECOMMENDATIONS:**<br>• Educational training should be implemented at all levels of sports participation for the identification, assessment, management and return-to-activity process following a sport-related concussion;<br>o Future research is warranted for the evaluation of the effects of educational packages on sport-related concussion.<br>• The K-D test should be utilised at all levels of sports participation to assist in the continuum of assessment for sport-related concussions;<br>o Future research is warranted to enable identification of normative values by age groups.<br>• Further biomechanical evaluations should be undertaken for impacts to the head in junior rugby league and amateur senior rugby union and rugby league;<br>o Future research is warranted at all levels of rugby union and rugby league to assist in the identification of head impact biomechanics of these sports and to help in the identification of the biomechanics resulting in a sport-related concussion.<br>• A consensus should be established for reporting head impact biomechanics across all sporting activities to assist with inter-study comparisons. |

Based on the recursive, dynamic model of etiology of sports injury,[446] and incorporating the neurobiopsychosocial model for concussion,[407] the cyclic model for concussion management was developed to standardise the identification, assessment and management of concussion.

**Research publications resulting from this doctoral thesis**

**Section 1: Review of literature for sport-related concussions**

**Chapter 2: Assessment, management and knowledge of sport-related concussion: Systematic review.**

King, DA., Brughelli, M., Hume, P., Gissane, C. Assessment, management and knowledge of sport-related concussion: Systematic Review. *Sports Medicine* 2014; **44**(4): 449-471
(Author contribution percentages DK: 80%, MB: 7%, PH: 7%, GC: 6%)

**Section 2: Epidemiology of concussion in New Zealand**

**Chapter 3: Sport-related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs.**

King, D.A., Gissane, C., Brughelli, M., Hume, P. & Harawira, J. Sport-related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs. *Journal of Science and Medicine in Sport* 2014, **17**(3): 250-255
(Author contribution percentages DK: 80%, GC: 5%; MB: 5%; PH: 5%, JH: 5%)

**Chapter 4: Concussion in amateur rugby league players in New Zealand: A review of player concussion history**

King, DA., Gissane, C. & Clark, T. Concussion in amateur rugby league players in New Zealand: A review of player concussion history *New Zealand Journal of Sports Medicine* 2014, **40**(2): 64-69
(Author contribution percentages DK: 85%, GC: 10%, TC: 5%)

**Section 3: Identification and assessment of concussion**

**Chapter 5: Concussions in amateur rugby union identified with the use of a rapid visual screening tool.**

King D, Brughelli M, Hume P, Gissane, C. Concussions in amateur rugby union identified with the use of a rapid visual screening tool. *Journal of Neurological Sciences* 2013; **326**(1-2):59-63
(Author contribution percentages DK: 85%, MB: 5%, PH: 5%, GC: 5%)

**Chapter 6: The King-Devick test was useful in management of concussion in amateur rugby union and rugby league in New Zealand.**

King, DA., Gissane, C., Hume, PA and Flaws, M. The King-Devick was useful in management of concussion in amateur rugby union and rugby league in New Zealand. *Journal of Neurological Sciences* 2015, **351**(1-2): 58-64

(Author contribution percentages: DK: 75%, PH: 10%, CG 10%, MF 5%)

**Section 4: Head impact biomechanics in amateur rugby union and league.**

**Chapter 7: Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches.**

King, DA., Hume, P., Brughelli, M. and Gissane, C. Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches. *Am J Sports Medicine,* 2015, **43**(3): 614-624

(Author contribution percentages DK: 80%; PH: 10%; MB 5%; CG 5%)

**Chapter 8: The influence of head impact threshold for reporting data in contact and collision sports: Consensus needed.**

King, DA., Hume, P., Gissane, C., Brughelli, M. and Clark, T. The influence of head impact threshold for reporting data in contact and collision sports: Systematic review and original data analysis. *Sports Medicine* (submitted)

(Author contribution percentages: DK: 75%, PH: 10%, CG: 5%, MB: 5%, TC: 5%)

**Technical Reports**

**King, DA.** Concussions in amateur rugby league: Player self-reported history and the incidence of match related concussions 2008 – 2012: A technical report to the New Zealand Rugby League. 2014: 18 pages

**King, DA.,** Hume, P., Gissane, C. and Clark, T. Sports-related concussions in rugby league: The magnitude of the problem and evidence for impact assessment. A report for the National Rugby League and New Zealand Rugby League. 2015: 54 pages

**Other publications since starting the Doctor of Philosophy**

**King, DA.,** Clark, T., & Gissane, C. Use of a rapid visual screening tool for the assessment of concussion in amateur rugby league: a pilot study *Journal of Neurological Sciences* 2012, **320**(1-2); 16-21

**King, DA.**, Gissane, C., Clark, T. and Marshal, S.W. The incidence of match and training injuries in rugby league: A pooled data analysis of published studies. *International Journal of Sports Science and Coaching* 2014 **9**(2): 417-432

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**King, DA.** Identification and management of concussion in the Emergency Department. Advanced MI study day. HVDHB Emergency Department. October 2014.

**King, DA.** Assessment of concussion for Enhanced Care Paramedics. ECP Advanced Training. Porirua Station, Wellington Free Ambulance. September 2014.

**King, DA.** Assessment and management of sports related concussions. Vibe Medical Centre, August 2014

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**King, DA.** Assessment and management of sports-related concussion. Minor Injury Study Day. HVDHB Emergency Department. April 2014.

**King, DA.** Paediatric Concussions. HVDHB ED staff presentation March 2014.

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**King, DA.** It's just a knock so don't worry about it. Minor Injury Study Day. HVDHB Emergency Department. March 2012

# Section 1:

# Review of Literature

***“There are not many concussions in the game so stop trying to make this a problem”***

Sports code board member

# CHAPTER 2:

# ASSESSMENT, MANAGEMENT AND KNOWLEDGE OF SPORT-RELATED CONCUSSION: SYSTEMATIC REVIEW

This chapter comprises the following paper published in *Sports Medicine:*

**Reference**

King, DA., Brughelli, M., Hume, P., Gissane, C. Assessment, management and knowledge of sport-related concussion: Systematic Review. *Sports Med* 2014; **44**(4): 449-471.

**Author contribution**

King, D. 80%, Hume, P. 7%, Brughelli, M. 7%, Gissane, C. 6%.

## Overview

**Background:** Sport-related concussions are a subset of mild traumatic brain injuries and are a concern for many sporting activities worldwide.

**Objective**: To review and update literature in regard to the history, pathophysiology, recognition, assessment, management and knowledge of concussion.

**Methods:** Searches of electronic literature databases were performed to identify studies published up until April 2013.

**Results:** 292 publications focussing on concussion met the inclusion criteria, so were quality rated and reviewed.

**Conclusion:** Concussion is hard to recognize and diagnose. Initial sideline assessment via the Sports Concussion Assessment Tool 3(SCAT3), Child-SCAT3 or King-Devick test should be undertaken to identify athletes with concussion as part of a continuum of assessment modalities and athlete management. Sports medicine practitioners should be cognisant of the definition, extent and nature of concussion, and should work with coaches, athletes and trainers to identify and manage concussions. The most common reason for variations in management of concussion is lack of awareness of - and confusion about - the many available published guidelines for concussion. Future research should focus on better systems and tools for recognition, assessment and management of concussion. Sport participants' knowledge of concussion should be evaluated more rigorously, with interventions for sports where there is little knowledge of recognition, assessment and appropriate management of concussion.

## Introduction

Known as the 'silent injury'[340] and often trivialized by the media and sporting circles as a 'knock to the head',[305] sport-related concussions (hereafter called 'concussion') are a subset of mild traumatic brain injuries (mTBIs)[423] and have become an increasingly serious concern for all sporting activities worldwide.[100, 123, 416] The term 'concussion' is a historical term, as it represents low-velocity injuries that cause 'brain

shaking' resulting in clinical symptoms, and it is often used interchangeably with 'mTBI' in the sporting context and published literature.[423] In the USA it is estimated that 1.6-3.8 million sport-related concussions occur annually[358] accounting for 5-9% of all sport-related injuries.[212, 506] Amongst 15- to 24-years-olds, concussions are second to road trauma as the most common causes of traumatic brain injury (TBI).[564] Guskiewicz[256] reported that of the 5.1% of 17,549 collegiate and high school football players who sustained at least one concussion during matches over a single season, 14.7% sustained a subsequent concussion, with 30 % of players returning to the same match.[285] Despite the frequency of concussions, they are often underreported.[285] In the past 30 years, clinicians have gone from anecdotal strategies to an international consensus-based approach for (1) identification and management of concussions; (2) evidenced-based practice; and (3) a new focus on education and injury prevention.[425]

Concussions are known to affect reaction time,[105] memory,[105, 291, 400] balance[408] and planning skills.[400] Previous concussions may place the athlete at higher risk (1.4-11.1) of sustaining a subsequent concussion.[128, 251, 256] Additionally, 1-29% of concussions occurring in a single season are reported as subsequent concussions.[256, 303] Repeat concussions may result in long-term outcomes, which include depression,[250] mild cognitive impairment,[249] prolonged recovery from subsequent concussions,[105, 251] electrophysiological changes[123] and chronic traumatic encephalopathy (CTE),[435] but to date there have been no direct causal relationships identified to support these relationships.[427] Despite these findings, the range and extent of long-term effects from repeat identified concussions remain unclear.[266]

Although there is increased understanding about the consequences of returning the concussed athlete too soon and the effects of repeated concussions over time, it is important to remember that every concussion is unique and should be managed individually.[443, 491] With this in mind, the aim of this article is to review and update the literature on concussion in relation to the history, pathophysiology, recognition, assessment, management and knowledge of concussion.

## Methods

Guidelines for the reporting of observational studies (MOOSE: Meta-analysis Of Observational Studies in Epidemiology),[575] systematic reviews (PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses),[369] and observational studies (STROBE: STrengthening the Reporting of OBservational studies in Epidemiology),[609] and for appraising research (AGREEII: Appraisal of Guidelines for REsearch & Evaluation v.II)[58] were followed for the different studies included in the review. These checklists contain specifications for conduct and review of the various studies that were included.

### *Search strategy for identification of publications*

A total of 38,333 studies published, in press or pre-publication from 1948 to April 2013 were identified through databases were screened for eligibility (see Fig. 4). The keywords that were utilized for the search of relevant research studies included combinations of 'sport*-related', 'sport*', 'concussion', 'mild Traumatic Brain Injury', 'mTBI', 'epidemiology', 'history', 'pathophysiology', 'return-to-play' (RTP), 'RTP', 'management',

'gender', 'academic', 'history', 'post-concussion', 'assessment', 'management' and 'knowledge'. Additional relevant studies were identified using the bibliographies of those articles found in the literature searches.

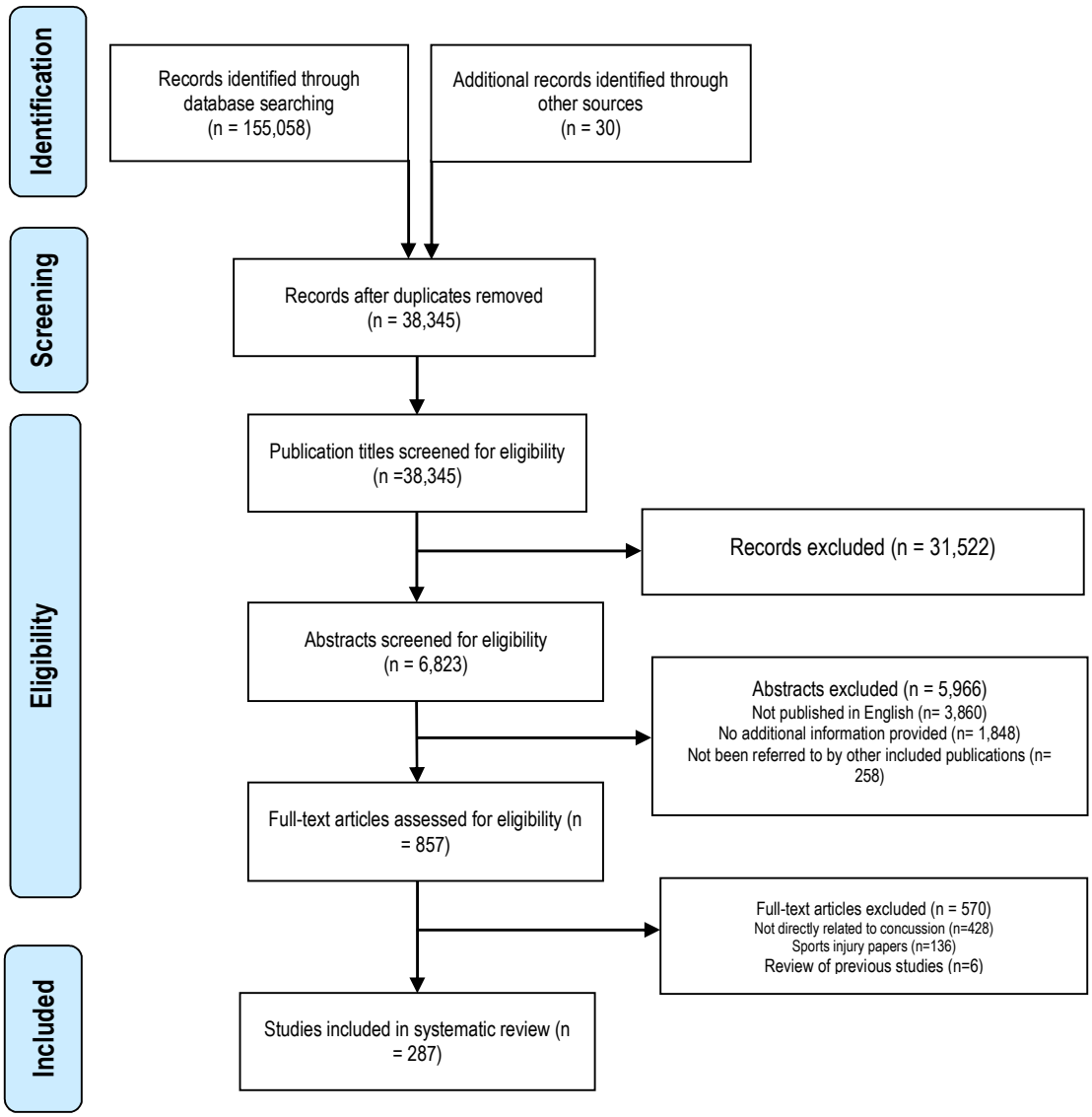


**Figure 4**: Flow of identification, screening, eligibility and inclusion for the literature review of sports-related concussions.

To establish some control over the heterogeneity of the different studies,[575] inclusion criteria were established. Any published study or book that did not meet the inclusion criteria was excluded from the study. A total of 287 publications were identified that reported on concussion and met the following inclusion criteria:

(i) The study was published in a peer reviewed journal or book; and

(ii) The study specifically addressed areas relating to concussion review (i.e. history, pathophysiology, definitions, symptom assessment and management, risk factors for and modifiers of linear and/or rotational acceleration-deceleration forces, and knowledge and understanding of player, team management, medical personnel, return to play and other activities).

Reviewed studies were excluded from this review if it was identified that the publication:

(i) Was unavailable in English; or

(ii) Did not provide additional information for any of the identified sections and subsections of this review; or

(iii) Had not been referred to by other included publications.

***Assessment of publication quality***

All studies that met the inclusion criteria were assessed for quality on the basis of previously published checklists.[58, 369, 575, 609] Heterogeneity of the studies included in the literature review was expected as there might be differences in the study design, population and outcomes.[575] For this review, quality was described as confidence that the study design, conduct and analysis minimized bias in estimation of the effect of the risk factor on the outcome measures.[377]

The quality scores by checklist were:

(i) MOOSE:[575] median score 5/6; range 2-6;[1, 16, 23-25, 28, 29, 31, 34, 39, 40, 49, 52, 53, 57, 80, 86, 90, 92, 100, 101, 103-107, 123, 127, 128, 141, 147, 148, 155, 158, 159, 165, 170, 202, 203, 206, 210, 212, 220, 237, 244, 246, 249-251, 254, 256, 261, 266, 279-281, 287, 289-291, 297, 298, 303, 308, 314, 317, 325, 331, 349, 350, 360, 362, 366, 372, 376, 380, 382, 387, 391, 394, 398, 400, 403, 406, 408, 409, 435, 437, 438, 444, 454, 459, 460, 466, 476, 477, 488, 489, 496-498, 501, 503, 506, 510, 511, 521, 536, 541, 542, 552, 553, 557, 561-564, 566, 576, 577, 580, 582, 584, 586, 587, 597, 598, 601, 605, 606, 608, 620, 634]

(ii) PRISMA:[369] median score 23/27; range 17-24;[4, 32, 48, 64, 122, 143, 145, 146, 149, 153, 208, 234, 238, 294, 363, 392, 396, 417, 421, 424, 468, 500, 512, 513, 520, 527, 532, 583, 589]

(iii) STROBE:[609] median score 10/14; range 4-12;[59, 89, 270, 310, 405, 599, 633] and

(iv) AGREEII:[58] median score 124/161; range 51-136.[8, 9, 11, 64, 91, 112, 215, 255, 260, 263, 355, 390, 392, 422-427, 514, 540, 558, 559, 568, 610]

The remaining included studies[15, 18, 21, 43, 46, 67, 70, 71, 93, 94, 98, 102, 118, 121, 124, 126, 132, 135, 138, 149, 152, 156, 231, 232, 240, 243, 252, 253, 258, 259, 267, 274, 277, 285, 288, 292, 296, 301, 340, 341, 348, 354, 356, 358, 361, 365, 374, 378, 379, 386, 389, 393, 405, 412-414, 416, 418, 419, 429, 439, 441, 443, 461, 482, 490, 491, 494, 505, 527, 535, 548, 551, 555, 589, 590, 595, 614, 624] recorded a median score of 5/27 (range 2-6) on PRISMA but provided additional information for the review and were included despite their low PRISMA ratings.

## Results and Discussion

***Historical perspectives***

Descriptions and recordings on head injuries or 'commotio cerebri' date back more than 3,000 years.[258, 418, 590] Writing from Greek medicine as early as 1700 BC through to Roman, Byzantine, Arabic and French medical writings include descriptions of the understanding and management of head injuries.[418, 419] The term concussion was not used until the seventeenth century, when Venetian physician Petri de Marchetti (1665) described the condition as being transient, with a short duration of "*alienation of the mind, with privation of*

*sense and motion*."[418, 419] In the nineteenth century, Bell introduced a new concept to the entity of concussion, describing the use of clinical signs to distinguish between different types of brain injury (concussion, compression and inflammation).[418]

As the understanding of the pathology of concussion developed, new physiological theories[418, 419, 548,] and models[361] were formulated and advocated, emphasizing a functional rather than a structural process of concussion.[418, 419, 548, 590] Despite these theories, concussion remains a mystifying subject in sports medicine.[590] Theories have provided valuable knowledge towards modern-day understanding of concussion, providing a glimpse into the pathophysiology that the brain undergoes when a concussion occurs.[418] Although some of these theories have been rejected, others continue to be used in developing an understanding of concussion.[418] Future research on the understanding of concussion is warranted.

***Definitions***

A concussion is hard to recognize and diagnose.[263, 386] Use of terms associated with injuries to the head, such as 'dings' or having one's 'bell rung' are commonplace and serve only to diminish the perception of injury severity and to perpetuate the notion that concussion is something people can play through.[386] By definition, concussion and mTBI overlap, as both terms represent the less severe end of the TBI spectrum.[234, 263, 292, 412, 423] Both terms identify that there is acute neurological dysfunction in the absence of significant microstructural damage.[234, 253] This generally recovers over time, with most people typically having resolution of symptoms within 7-10 days.[423] The full spectrum of TBIs (mild, moderate and severe) would see concussion below the mild classification in a 'minimal' range as TBI reflecting no neurosurgical significance of a pathological injury.[424] In Europe, the term 'commotio cerebri' is often used in place of the term 'concussion' to represent a low-velocity injury that results in 'brain shaking' resulting in clinical symptoms not necessary related to pathological injury.[424] The term 'concussion' is more frequently utilized in sports and clinical settings.[292] It is the preferred terminology, as it is more easily understood by most patients, easier to communicate regarding the prognosis and is less likely to have an adverse psychological effect on the person when they learn about their injury.[292]

Ever since concussion was described in the medical literature, there have been numerous attempts to establish a working definition.[9, 11, 93, 112, 301, 341, 422, 425, 494, 506, 568, 610] Consequently, the definition of concussion has changed over time,[418] as universal agreement on a single definition has been difficult to reach.[94, 356] In 2001 at the First International Concussion In Sport (CIS) Conference, the Concussion In Sport Group (CISG) considered a more inclusive and elaborative definition of concussion.[11] This definition incorporated common concepts from different definitions of concussion that had been previously published, and did not consider loss of consciousness as an essential defining characteristic.[491] Despite this, loss of consciousness may, or may not, be present as a feature of concussion, as symptoms are transient and global in nature.[491] The subtle nature of the symptoms and pathology makes the diagnosis of concussion a challenge.[491]

Since the publication of the first CIS consensus statement and definition of concussion, variations of the definition have been produced;[35, 64, 256, 260, 263, 502, 527, 558, 559, 589] until a universal definition is used for

identification and reporting of concussion, the true epidemiological incidence of concussion will not be identified.

***Concussion pathophysiology***

When direct or indirect linear and/or rotational forces are applied to the brain,[11, 118, 422, 443] the underlying neural elements are exposed to a shearing strain.[232, 365, 366, 482] Following this shearing strain, there is an alteration to normal brain functioning, termed 'neurometabolic cascade'. This places the brain cells in a vulnerable state as a result of ionic, metabolic and pathophysiological events accompanied by microscopic axonal injury.[15, 231, 232, 288, 297, 348, 443, 511, 551, 590, 598] These disruptions require energy to re-establish homeostasis, but this occurs in the presence of ongoing mitochondrial dysfunction and decreased cerebral blood flow, resulting in an imbalance of energy supply and demand.[15, 511] This may occur anywhere from minutes to days following the event.[118, 590]

The pathophysiological effects of concussion can be seen within 24 h of the event, lasting up to several weeks post-injury.[232, 348, 443] Experimental evidence indicates that the concussed brain may be less responsive to physiological neural activation.[15, 511] A second injury to the brain in this vulnerable period may result in a worsening of cellular metabolic changes associated with more significant cognitive deficits.[511, 551] These perturbations are more pronounced in youth, raising concerns that the immature brain may be more susceptible to repeat concussions before complete recovery occurs.[551] Excessive cognitive or physical activity before complete recovery may result in prolonged dysfunction.[15, 551] As a result of the increased metabolic dysfunction, the brain may have increased vulnerability to other consequences, such as second-impact and post-concussion syndromes if a subsequent insult (even minor) were to occur,[348, 551] although this has not been well established.[417]

***Subconcussive pathophysiology***

A subconcussive injury is a theoretically very mild, biomechanically induced brain injury. These may occur in the absence of overt clinical symptoms of concussion,[234] loss of match or training time, or concussion-related symptoms that linger for a prolonged period of time.[31, 40, 118] Non-concussive impacts that occur during sport participation may result in sub-clinical decline.[25, 40, 43, 118] Some athletes without a clinically diagnosed concussion or clinically observed symptoms of concussion have had neurocognitive and neurophysiological impairments that may be accumulative.[580]

Similar to the pathophysiology of concussion, subconcussive incidents have also been shown to induce a neuro-inflammatory response in rat studies.[553] This neuro-inflammatory response can occur in the absence of any significant axonal injury or of emotional, cognitive or sensorimotor disturbances.[118] The neuro-inflammatory response seen in repeated subconcussive head traumas may have cumulative effects.[118, 498, 552] This response has been linked to neurodegenerative disorders such as CTE,[21, 437, 553, 566] post-concussion syndrome, post-traumatic stress disorder, mild cognitive impairment and dementia pugilistica[118] but no direct causation between concussions and these disorders has been demonstrated.[423]

***Symptom assessment***

Difficult to diagnose,[424] the symptoms of a concussion can vary in nature, and the measurement of these depends upon the self-reporting of the athlete. Traditionally, loss-of-consciousness (LOC) was considered to be the hallmark of concussion[208] but this has been revised, with only 8-9% of all concussions[251, 256] resulting in LOC. More common symptoms of concussive events are amnesia (13-24%)[158, 251, 496] and confusion/disorientation which may present in 45-90% of all athletes with a concussion.[158, 387] Other symptoms now being recognised as resulting from a concussive event are changes to the athlete's physical, cognitive affective (or emotional) and sleep domains.[488]

Physical symptoms that may manifest are headache, dizziness, nausea, vomiting, sensitivity to light and/or noise, and drowsiness.[488] Reported cognitive changes are that athletes feel they are in a 'fog', have difficulty remembering and feel slowed down.[488] Several cognitive domains are negatively affected following a concussive incident. This is evidenced by the deficits identified through neuropsychological testing, with changes recorded in attention/concentration,[147, 244] speed of information processing,[387] verbal learning,[147] visuo-spatial memory,[380] working memory,[147] verbal memory,[147] and reaction time.[608] Affective symptoms that may present are irritability, sadness and anxiety, while changes in the athlete's sleep domain may manifest as sleeping less or more than usual, trouble falling asleep and drowsiness.[488] To complicate the assessment of concussion further, some of these deficits may not be present for every athlete, thus making this an individualized injury.

**Table 3:** Summary of the concussion symptom scales/checklist published from 1995.[4]

| Year | Scale Name | Grading scale | No items |
|---|---|---|---|
| 1980's; 1990's | Pittsburgh Steelers Post-Concussion Scale[a] | 7-point Likert Scale | 17 |
| 1998 | Post-Concussion Scale (PCS)[b] | 7-point Likert Scale | 20, 21, 18 |
| 1999 | Post-Concussion Symptom Assessment Questionnaire (PCSQ)[a] | Yes/No with 10cm VAS | 10 |
| 2000 | Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) Post-Concussion Symptom Scale (ImPACT-PCSS)[b, c] | 7-point Likert Scale | 22, 21, 19 |
| 2001 | Concussion Resolution Index - Post Concussion Questionnaire (CRI)[a, c] | 4-point Likert Scale | 15 |
| 2001 | Vienna Post-Concussion Symptom Scale[b] | 7-point Likert Scale | 20 |
| 2001 | McGill Abbreviated Concussion Evaluation - Post Concussion Symptom Scale[b] | 7-point Likert Scale | 20 |
| 2003 | Graded Symptom Checklist/Scale (GSC/GSS)[b] | 7-point Likert Scale | 20, 27, 18, 17 |
| 2003 | Head Injury Scale (HIS)[b] | 7-point Likert Scale | 16, 9 |
| 2003 | CogState-Sport Symptom Checklist[a, b] | 7-point Likert Scale | 25, 21, 14 |
| 2004 | Signs and symptoms Checklist (SSC)[a] | Yes/No | 34 |
| 2004 | Sport Concussion Assessment Tool - Post Concussion Symptom Scale (SCAT-PCSS)[a] | 7-point Likert Scale | 25 |
| 2009 | Concussion Symptom Inventory (CSI)[a] | 7-point Likert Scale | 12 |

(a)= core scales; (b)= variants of Pittsburgh Steelers Post-Concussion Scale; (c)= computer based scale; VAS= Visual Analog Scale

Concussion symptoms typically resolve in 80-90% of all sport participants by 7-10 days post-injury.[48, 94, 170, 372, 391, 394, 406, 444] Concussion symptoms, by their very nature, are subjective and depend upon awareness,[409] honesty and willingness of the athlete to accurately provide the information.[4, 409] Resolution of concussion symptoms may not always indicate complete cognitive recovery, as persistent deficits may still be present,[379, 406] and the clinical importance of these changes in the absence of symptoms is unknown.[263] Throughout the development of knowledge of concussion, there have been a number of self-reported

symptom scales[425] and checklists[57] published in an endeavour to assist the clinician to objectively document the symptoms of concussion and any changes that occur[4] (see Table 3).

Following the 2004 Second CIS Conference, the Sports Concussion Assessment Tool (SCAT) was published as part of the summary and agreement statement of those who attended the conference.[422] The SCAT was based on expert consensus of the best measures to assess concussion that were currently available.[422] The Third International CIS Conference in Zurich[425] resulted in the SCAT being amended, and the SCAT2 and the PocketSCAT2 were produced. Embedding the Sideline Assessment of Concussion (SAC) and a modified Balance Error Scoring System (BESS), the SCAT2 uses a score range of 0-100 points with lower scores indicating poorer performance. The SCAT2 was designed for serial use after a concussion and included a score card designed to enable tracking of the concussed athlete's performance. The SCAT2 is a longer sideline concussion tool designed for medical practitioners to enable more detailed assessment of concussion, while the PocketSCAT2 was designed for sideline recognition of screening for concussion for the non-medically trained.[423] Although the SCAT2 is an improvement over the original the SCAT, it requires additional time to complete on the sideline, making this more of a training room assessment tool.[149]

More recently, the Fourth CIS Conference reviewed the SCAT2, and the SCAT3 was produced, along with the Child-SCAT3 and the Concussion Recognition Tool (CRT).[423] The components of the SCAT3 have been shown to be reliable and valid through several studies and have resulted in psychometric properties for reliability (0.54-0.94), sensitivity (0.34-0.94) and specificity (0.76-1.0).[255] As a result of the review of the SCAT2, the SCAT3 is designed for participants over 12 years of age, while the Child-SCAT3 is for sport participants between 5 and 12 years, with modification of the Maddocks questions.[423] The CRT is an updated version of the PocketSCAT2 but does not have a child version for the modified Maddocks questions asked in the Child-SCAT3.

***Neuropsychological assessment (NP)***

Introduced in the 1980s, neuropsychological assessment has become widespread with the availability of computers enabling computer-based testing, and it is seen as a 'cornerstone' in the management of concussion.[145, 263, 294, 300, 422, 423] Designed to identify occult cognitive impairment post-injury, neuropsychological testing has been able to identify neurocognitive deficits within 2-48 h post-injury[143] and can show cognitive deficits despite athletes reporting they are asymptomatic.[153, 294, 422, 425, 620] Neuropsychology is also a useful tool for documenting athletes' recovery from a concussion by measuring several domains of cognitive function.[153, 294, 422, 423, 425] The areas measured vary by test type and focus on cognitive processing speed, reaction time and memory.

Neuropsychological tests can be divided into two types on the basis of their method of administration, either pencil-and-paper or computer based. Both forms of neuropsychological testing have some variability in regard to the measurement of domains and performance in areas such as the Reliable Change Index, sensitivity and validity.[89, 263, 520] Pencil-and-paper tests are administered and interpreted by a neuropsychologist,[145, 422] are often more comprehensive and may test additional domains, enabling assessment for other conditions that may masquerade as a concussion or post-concussion syndrome.[520]

However, these tests often require more time to administer, can be labour intensive and are more expensive than computerized neuropsychological testing.[89, 146, 153]

Computerized neuropsychological tests have become more common in the athletic setting, as they can be administered concurrently to groups of athletes, have more precise measurements of reaction time, provide instant information to the provider, take less time to administer, are less expensive and can store large amounts of data.[143, 146, 153, 263, 294, 422] These tests have been adopted as a core component of many concussion management programmes.[525] Computerized neuropsychological tests have been validated against paper-and-pencil tests[5, 88] and post-concussion symptoms scores[77, 359] by different groups,[5, 77, 88, 153, 359] with overall specificity and sensitivity of between 80 and 90%.[359] The advantages of using computerized neuropsychological tests include ease of administration; ease of data retrieval; automated data collection, storage, analysis and interpretation; high sensitivity to subtle cognitive effects; measurement of multiple domains of performance and variability; measurement of the extent of cognitive malfunction during the recovery process; and ability to control stimuli and stimuli characteristics.[491, 554] However, there are several drawbacks to computerized neuropsychological testing when compared with pencil-and-paper neuropsychological tests. Computerized neuropsychological tests (1) do not fully assess memory functioning - they only examine recognition memory; (2) minimize the interaction between the neuropsychologist and the athlete, reducing observation of performance; (3) limit the assessment of the effort and motivation of the athlete, because of group administration; (4) limit the ability to examine the injured athlete's problem-solving and information-learning processes;[143] and (5) have varying test-retest reliability.[45, 519, 525] Other possible limitations to the use of computerized neuropsychological testing are that computer programs can introduce complex instrumentation errors due to timing accuracy across different computer platforms, the computer's processor speed, the type of mouse used and if the test is administered across the internet.[143] Despite the drawbacks, computerized neuropsychological tests are utilized through a variety of computer-based batteries such as Immediate Postconcussion Assessment and Cognitive Testing (ImPACT Inc., Pittsburgh, PA), CogSport (CogState Ltd, Melbourne, Australia), Automated Neuropsychological Assessment Metrics (ANAM developed by the US Department of Defense) and Headminder (ImPACT Applications, Inc; Axon Sports, LLC).[143, 146]

Neuropsychological tests for pre-adolescent sport participants have been reported.[146, 245, 420] Although the key assessment domains in this group of sport participants are generally similar to those in adolescents and adults, important differences must be accounted for, such as pre-adolescent cognitive, physical and emotional differences, as well as the capacity of the pre-adolescent to be aware of and to report their symptoms.[146, 222] The computerized neuropsychological tests utilized in pre-adolescent sport participants have shown promising results, but there is a lack of evidence to draw any firm conclusion in relation to the clinical utility of neuropsychology in this group.[146, 234] There is a paucity of evidence of neuropsychological tests having been applied to pre-adolescents who have sustained a sport-related concussion.[146]

Although neuropsychological testing has moderate sensitivity for the detection of post-concussive cognitive deficiencies,[153, 263] it has not been validated as a diagnostic tool.[263] As such, neuropsychological testing has

been promoted as a monitoring tool for the recovery of the concussed athlete.[425] It has been recommended that neuropsychology should be utilized not as the sole basis of management of concussions but as an aid to the clinical-decision making process in conjunction with a range of assessments of different clinical domains and investigations.[143, 146, 263, 423] Healthcare professionals may often administer neuropsychological tests, but the interpretation of these tests can be complex. It is therefore recommended that interpretation of neuropsychological test results is best undertaken by a trained neuropsychologist.[146, 423, 554] The use of baseline, or pre-competition, neuropsychological assessments may be of benefit to assist in post-concussion evaluations, and it is thought that these may increase the diagnostic accuracy when compared with post-injury scores.[146] Mandatory baseline neuropsychological testing is not considered a requirement for the assessment process, nor is there sufficient evidence to recommend this, but baseline neuropsychological tests can add an educative opportunity to discuss the significance of the injury.[423] For a more in-depth review of the advances in neuropsychological assessment of sport-related concussion, readers are directed to Echemendia et al.[146]

***On-field or sideline assessment of concussion***

Typically, sideline assessment of concussion involves brief tests that can be conducted on the sideline of a field or court.[599] The purpose of these tests is to rule out a more serious injury and to commence an individualized concussion management process based on the results of the assessment of cognitive and balance deficits or impairments.[149] These sideline tests are designed to evaluate attention and memory (Maddocks test),[389] cognition (SAC)[405] and postural control (BESS).[103] The use of sideline tools for assessment of concussion is seen as an abbreviated sideline assessment and not designed to take the place of more comprehensive evaluation or neuropsychological testing.[512]

Undertaking a sideline or on-field assessment of concussion has been a challenging responsibility of the healthcare provider.[46, 514] This is often a rapid assessment process in the midst of competition with time constraints. Without qualified healthcare providers available to make the assessment, often players are returned to the field of participation.[215] Even with qualified health care professionals available at the sideline, they may not be aware of the latest guidelines or assessment tools to assist in the evaluation of a concussed sport participant.[121] The assessment can be difficult to undertake if classic indicators (e.g. loss of consciousness, amnesia, focal neurological abnormalities) are not manifested and only subtle signs and symptoms are present, raising the suspicion that 'something's off' and that a concussion may have occurred.[405] Some sports are now introducing a 'concussion bin' where players can be removed from match participation by substitution. The suspected concussed player can be assessed, in a specified time frame, enabling testing to be completed to identify, or rule out, the occurrence of a concussive injury.[514] It has been recommended that in conducting a sideline assessment for concussion, a minimum rest period of 15 minutes should occur before the full test is completed, to avoid the influence of exertion or fatigue on the assessment.[255] Further research is required in order to enable validation that these tests are able to identify concussions even with subtle changes occurring.

There have been robust public education outreach efforts undertaken to assist non-healthcare-trained individuals to recognize and manage individuals with signs of concussion while they are participating in sporting activities.[514] These efforts have included freely available online information and encouragement for referral of the concussed athlete to a qualified healthcare provider for confirmation and management of the concussion.[514] This has been undertaken to the extent that legislative efforts have been implemented to improve safety measures for younger athletes and to standardize the roles and responsibilities of the athletes, parents/guardians, coaches and healthcare providers.[514] As the CISG update[423] has only recently been published, there are currently no published studies to see if this information has aided in the promotion of concussion awareness. Further research is warranted to identify a wide range of sports team administration knowledge on the recognition and management of athletes with concussion.

Until the recent publication of the Child-SCAT3,[423] there were no specifically developed sideline concussion assessment tools for children.[122] Symptom scales need to be available that include the language that is understood by this age group and symptoms that are familiar to them.[122] The use of the SAC has been evaluated for use in children but no symptom scales have been evaluated for use on the sideline with children.[122]

Originally developed as a reading tool to assess the relationship between poor oculomotor functions and learning disabilities, the King-Devick test uses a series of charts of numbers that progressively become more difficult to read in a flowing manner.[202, 203] The use of oculomotor function requires integration of multiple sensory inputs, motor efforts[374] and cognitive processes such as target selection, sustenance of attention, spatio-temporal memory and expectation.[18] More recently, poor oculomotor function has been determined as one of the most robust discriminators for identification of an mTBI.[270]

Recently, the King-Devick test has been used successfully to identify concussion in mixed martial arts; boxing;[202] university sports such as American football, soccer and basketball;[203] ice hockey;[206] rugby league;[317] and rugby union.[314] Designed as a saccadic rapid number reading tool, the King-Devick test[202, 203, 206, 314, 317] is able to be completed on the sideline in less than a minute and has been correlated with the Military Acute Concussion Evaluation (MACE) ($r_s$= -0.54; $p$=0.07)[86] and the Standardized Assessment of Concussion (SAC) ($r_s$= 0.25; $p$=0.01).[206] Although the King-Devick test has been able to identify unwitnessed concussive events on-field[314, 317] and when there have been no changes on the SCAT2 SAC components,[206] there have been no longitudinal studies to assess the reliability, validity or generalizability of the King-Devick test, nor has it been tested across various age groups.[203]

***Grading scales***

Historically, the decision on the management of concussion has been based upon 'grading scales' of the severity of concussion.[363] These were used to differentiate the strategies to enable return of the athlete to the sporting environment. Three commonly utilized scales were the American Academy of Neurology (AAN),[8] the Cantu,[67] and the Colorado Medical Society[91] scales (see Table 4).

The grade (or severity) of the concussion was based on the symptoms that were present and whether they abated in a specified time frame, and guided the RTP of the athlete. For example, in the AAN scale[8] a grade 1, or mild, first concussion resulted in a player being removed from the activity but being able to return on the same day if they reported no symptoms 15 min after they were removed. Should they incur a second concussion on the same day, then they were required to be removed from the activity but could return a week later. When compared with the Cantu scale,[67] there was no time limit for a first-time grade 1 being returned to play, but they were required to sit out 2 weeks if they incurred another concussion. The Colorado scale differed slightly, with a grade 1 concussion to be returned in 20 min if symptom free, and if they recorded another concussive injury, they could go back to the activity a week later. The variability and multitude of the grading scales highlighted the lack of consensus on the grading of concussion, which resulted from the absence of evidence-based data.[363]

**Table 4:** Return-to-play guidelines.

| RTP guideline | Severity | 1st Concussion | 2nd Concussion | 3rd concussion |
|---|---|---|---|---|
| Colorado Medical Society[91] | | | | |
| | Grade 1 (Mild) | RTP if no sx 20 mins | Terminate game, RTP if no sx 1 week | Terminate season, RTP 3 months if no sx |
| | Grade 2 (Moderate) | Terminate game, RTP if asx 1 week | Consider terminate season, but consider RTP if no sx 1 month | Terminate season, RTP next season if no sx |
| | Grade 3 (Severe) | Terminate game, transport to ER, RTP 1 month after 2 weeks with no sx. Ok to condition after 1 asx week | Terminate season, RTP next season if no sx | Terminate season, highly recommend avoidance of contact/collision sports |
| Cantu[67] | | | | |
| | Grade 1 (Mild) | RTP if no sx | RTP in 2 weeks if no sx 1 week | Terminate season, RTP next season if no sx |
| | Grade 2 (Moderate) | RTP if no sx 1 week | 1 month restriction, RTP if no sx 1 week, consider terminating season | Terminate season, may RTP next year if no sx |
| | Grade 3 (Severe) | 1 month restriction, RTP if no sx 1 week | Terminate season, RTP next year if no sx | |
| American Academy of Neurology[8] | | | | |
| | Grade 1 (Mild) | Removal from competition, examination q5 min, RTP same day if post-concussive sx resolve within 15 min | Incurring a 2nd concussion on the same day, RTP when no sx 1 week | |
| | Grade 2 (Moderate) | Removal from competition, if sx worsen or persist > 1 week, then extensive diagnostic testing. RTP when no sx 1 week | Incurring a Grade 2 concussion subsequent to a Grade 1 concussion on the same day, then restrict from competition until no sx 2 weeks | |
| | Grade 3 (Severe) | Brief: RTP when asx 1 week<br>Prolonged: RTP when asx 2 weeks<br>Prolonged LOC or abnormal neurologic signs on initial examination: Transport to ER. No sports activity until asx 1 month. Any athlete with abnormality on CT or MRI (swelling, contusion or other intracranial pathology) no RTP that season and strongly discouraged from future participation in contact sports | | |

RTP= return-to-play; asx= asymptomatic; sx= symptoms; LOC= loss of consciousness; ER= Emergency Room; CT= Computed Tomography; MRI= *Magnetic Resonance Imaging*. Asymptomatic in all return-to-play guidelines means no post-concussion symptoms, including retrograde or anterograde amnesia, at rest or with exertion.[71]

At least 16 different head injury grading scales have been published.[363] These were based on clinical experience and anecdotal evidence rather than research-based evidence,[69, 296, 363] with most scales being biased towards identification of the most severe injury.[363] As no perfect scale exists that is both scientifically validated and practical,[296, 363, 412] no scale was endorsed by the CISG at the First CIS Conference.[11] It was recommended that a clinical construct should be utilized on the basis of assessment of injury recovery.[11] Following the Second CIS Conference,[422] it was recommended that concussions be categorized as either simple (resolution of symptoms without complication over 7-10 days) or complex (persistent symptoms, specific sequelae or prolonged cognitive impairment following the injury). Although this was seen as an advancement in the classification of concussion injury severity, the use of this terminology was limited, as it was not able to predict injury severity at the time of the concussive injury occurring, and it did not fully define the entities of concussion.[425] Consequently, at the Third CISG Conference[425] the terminology of 'simple' and 'complex' was rejected. Consequently, there are no recently published scientifically validated grading scales available for use with concussion. Despite this, some studies[492] and guidelines[456] still utilize the grading scales for concussion assessment, further highlighting that the CIS statement is not universally accepted.

***Risk factors / modifiers of concussion***

Several risk factors and modifiers, including age and gender, may influence the risk of a sustaining a concussion or of having a protracted period of concussion resolution, although the Fourth CIS Conference found no universal agreement that female gender was a modifying factor.[423] Females sustain more concussions than males, have a greater number and severity of concussion symptoms and require a longer duration to recover when compared with males on an exposure adjusted basis.[16, 57, 102, 104, 106, 107, 135, 212, 281, 354, 372, 394, 541, 587, 595, 634] Although younger sport participants may have a more prolonged recovery period and are more susceptible to concussions accompanied by a catastrophic injury,[165, 232, 460, 551, 557, 633] there is a paucity of studies reporting on recovery patterns for participants under the age of 15 years. The risk for younger sports participants is hypothesized to be related to physiological differences between younger and more developed brains.[341, 562]

Genetics, mood disorders, migraines, learning disabilities and attention disorders are associated with modifiers for concussions and prolonged recovery.[392] Although some studies have suggested that there may be an association between concussion and genetic polymorphisms (i.e. apolipoprotein e4 [APOE e4] and APOE G-219T)[350, 584, 586] these studies have been limited by methodological weaknesses and did not support definitive conclusions.[584, 586] A mood disorder history, either pre-existing or resulting from a concussive injury, may compound the diagnosis and management of concussion.[349, 536] However, there is no evidence that pre-existence of a mood disorder predisposes the sport participant to a concussion.[263] It is difficult to evaluate an individual for symptoms of a concussion without being able to differentiate between symptoms that preceded the concussion from those caused by the concussion and to ascertain what symptoms are exacerbated by the concussion.[156, 250] This is similar for sport participants with learning disabilities such as attention deficit disorder/attention deficit hyperactivity disorder and migraines.[90, 237, 308, 454, 563]

***Effects of multiple concussions***

Following a concussion, the sport participant's risk of a subsequent concussion is increased, especially in the 7- to 10-day period following the initial acute injury.[118, 127, 251] There is a six-fold increase in the risk of sustaining a subsequent concussion for athletes who experience a loss of consciousness, compared with those who have no loss of consciousness.[127] The reasons for this have not been fully elucidated but may be related to (a) the style of play predisposing them to another injury; (b) susceptibility of the athlete; (c) age and level of participation; (d) the possibility that the athlete may receive more exposure time; or (e) the fact that once a concussion occurs, the brain is more susceptible to a concussive injury.[127] Athletes with prior concussions may take longer to recover from an acute concussive injury.[251]

The effects of repeated concussions are reportedly cumulative and permanent.[118, 124, 152, 251, 290, 291, 460, 490, 510] These effects range from an increased risk of a repeat concussion (5.8 times), on-field loss of consciousness (6.7 times), confusion (4.1 times), and anterograde amnesia (3.8 times) with each subsequent concussion[251, 291] through to decreased performance in attention, concentration, immediate memory recall and visual motor coordination.[98] These effects can be more pronounced if the period between concussions is shorter and the effects are additive.[490] Some,[290, 291, 460] but not all,[123] researchers have identified that athletes with multiple concussions report more symptoms and have a worse neuropsychological test performance than athletes with no history of concussions. This might be reflective of the cumulative and additive effects of multiple concussions[555] with resolution of the symptoms occurring over a period from several days to months to years, or they may be permanent.[251, 291, 624]

A reported but extremely rare and controversial condition that can occur as a result of a subsequent concussion is second-impact syndrome.[413, 535] Second-impact syndrome is reportedly more common in children and adolescents younger than 21 years old.[413, 614] Re-injury need not occur on the same day and has been reported to have occurred within up to 2 weeks following the first concussive injury.[535] The second injury may initially appear to be of the mildest degree but can quickly evolve to collapse, loss of consciousness and respiratory failure, and death may occur.[70] These events have been attributed to cerebrovascular dysregulation, vascular engorgement, herniation of brain tissue, worsening cellular metabolic changes and more significant cognitive deficits[159, 376, 413, 582, 597, 598, 614] although the exact pathophysiological pathway remains unknown.[238, 421]

More recently, it has been reported that multiple concussions may be linked with CTE,[435, 437] but there is a paucity of published studies, and it is not possible to determine if there is a cause-and-effect relationship between concussions or exposures to contact sports.[423, 427] CTE has been found in athletes as young as 18 years who have died with a history of concussions.[574] Athletes with multiple concussions can have neurobehavioural manifestations of CTE, such as changes in memory, behaviour, personality, gait and speech, similar to Parkinsonism-type symptoms.[435, 437] Although concussion has been described as a risk factor for CTE,[579] what is not known is whether this may be caused by concussive and/or subconcussive events, the frequency of these types of events to occur for CTE to develop, and if there is a predisposition for only a small number of athletes to be at risk of having CTE.[416] More studies are required to determine if

there is a direct causal link between concussion and CTE,[423, 427] at what age the nervous system is most susceptible to the effects of concussion, and whether proper management of concussion can reducing late-life neurodegenerative dementias.[416]

Not all impacts to the head will result in concussions. In situations where the brain is exposed to forces (linear and/or rotational), it is difficult to reliably identify non-concussive episodes.[566] High school football players have recorded significant neurophysiological changes despite having no clinical symptoms of concussion.[580] This was further identified in rats that underwent a subconcussive lateral fluid percussion.[552] The percussion resulted in a neurophysiological change but did not affect behaviour or have any other injury severity measures such as loss of consciousness.[552] Further research is therefore warranted for the monitoring of subconcussive injuries in several areas, to identify (1) tools that are available to assist in the identification of subconcussive injuries; (2) how often subconcussive incidents occur in contact sports such as rugby league and rugby union; and (3) what changes occur over a period of repeated subconcussive incidents.

***Return to Play (RTP)***

The use of RTP guidelines has been based around the biomechanical concepts of concussion.[590] The first reported RTP rule was the 'three strike rule' originally proposed by Quigley[414] in 1945 and then adapted by Thorndike in 1952.[414] This rule recommends that if an athlete has three concussions, then they should terminate future sport participation.[267] Subsequent RTP guidelines (see Table 4) extended and expanded Quigley's rule and were established with the intention of preventing catastrophic injuries and cumulative effects that may occur as a result of concussion.[243]

All of the established guidelines focused on ensuring that the sport participants must be free of any post-traumatic amnesia symptoms and post-concussion symptoms both at rest and during exercise.[71] The original RTP guidelines were based on either clinical experience[67] or the result of a catastrophic incident.[8, 91] In the mid-1990's these guidelines were critiqued regarding their scientific basis, resulting in a workshop to re-evaluate the guidelines and establish practical alternatives.[624] The guideline published as a result of this workshop did not differ substantially from previous guidelines but started the move from numerical grading to individualized management of concussions.[378]

More recently, the CISG identified that none of the published RTP guidelines were adequate to assure proper management of every concussion.[11] The CISG[423, 425] also published a graduated RTP protocol (see Table 5) for a stepwise process of rest to exertion for returning the athlete to the sporting environment, separated by at least 24 h between the different stages. The RTP should be individualized to the concussed player, utilized in conjunction with symptom assessment and cognitive examinations, and used for tracking the recovery of the concussed player.[159, 251, 263, 354, 391, 426, 513] The RTP protocol is appropriate for children and adolescents as young as 10 years.[425] Athletes under the age of 18 years would require a more conservative RTP protocol, while athletes under the age of 10 years would require age-appropriate symptoms checks.[425]

On the basis of the SCAT2, the International Rugby Board (IRB) has developed online resources for both the healthcare provider and non-healthcare provider (http://www.irbplayerwelfare.com/?documentid=module&module=1) in the assessment and management of a concussed athlete, utilizing the consensus statement and its associated assessment tools. The IRB also introduced a 'Pitch-Side Concussion Assessment' (PSCA) for use in the World U20 IRB competition (2012) and in professional competitions (2013) but, again, there have been no published studies reporting on this as a sideline protocol for assessment of concussion. PSCA is undertaken in a 5 min 'concussion bin' period but is well short of the CIS guidelines of 15 min for sideline concussion assessment.[255] To date, there are no published studies reporting the effectiveness of these implementations in the identification and management of rugby-related concussive events.

**Table 5:** Fourth CISG graduated return-to-play protocol.[423]

| | Rehabilitation stage | Functional exercise at each stage of rehabilitation | Objective of each stage |
|---|---|---|---|
| 1. | No activity | Physical and cognitive rest | Recovery |
| 2. | Light aerobic exercise | Walking, swimming or stationary cycling keeping intensity, <70% maximum predicted heart rate; no resistance training. | Increase in heart rate |
| 3. | Sport-specific exercise | Skating drills in ice hockey, running drills in soccer; no head impact activities. | Add movement |
| 4. | Non-contact training drills | Progression to more complex training drills, e.g., passing drills in football and ice hockey; may start progressive resistance training. | Exercise, coordination, and cognitive loading |
| 5. | Full contact practice | Following medical clearance, participate in normal training activities | Restore confidence and assess functional skills by coaching staff |
| 6. | Return-to-play | Normal game play | |

CSIG= Concussion in Sport Group

Compliance with RTP regulations has been reported to be poor. Players with a reported concussion undertaking the required stand-down regulations have varied from 33%[1] to 100%[280] non-compliance, despite receiving RTP advice. Following the 2012 CISG Conference, the newly produced SCAT3[423] has altered the RTP requirement for medical clearance. SCAT2[425] identified that with RTP, there should be 24 h (or longer) between stages and that medical clearance should be given before RTP. SCAT3[423] differs from RTP, as athletes should now "be medically cleared and then follow a stepwise supervised program, with stages of progression", where there should be 24 h (or longer) between stages, and medical clearance should be given before RTP.

***Linear and rotational head acceleration-deceleration***

A concussion typically occurs as a result of a direct impact to the head or from an indirect impact applied to the body that is transmitted to the head.[11, 422, 425, 441] This results when the torso is either decelerated or accelerated rapidly.[310] Consequently, the head sustains a combination of linear and rotational acceleration. Direct impacts with the head (linear acceleration-deceleration) and inertial loading of the head (rotational acceleration-deceleration) have been postulated as the two major mechanisms of head-related injuries such as concussion.[310] Linear acceleration produces focal injuries, while rotational acceleration produces both focal and diffuse injuries.[310]

Denny-Brown and Russell[132] first described concussion as a result of sudden velocity changes, terming this 'linear acceleration-deceleration'. Considered as the most important mechanism of head injuries, linear

impact forces were attributed to intracranial damage resulting in pressure gradients and deformation of the skull, which were the key factors for a concussion occurring.[52, 253, 310] Although pressure within the brain varied during an impact, there was a strong correlation ($r$=0.42) between linear acceleration and brain deformation.[505] Concussive events that occurred during a linear acceleration-deceleration were the result of the brain's relatively low inertia being unable to keep up with the movement of the skull.[252] Also, acceleration per se was not the primary cause of injury, as rapid motion causes displacement of the hard bony structures of the skull against the soft tissues of the brain.[606] Primarily because of limitations in techniques and equipment being available to measure rotational acceleration,[439] the linear acceleration-deceleration hypothesis has been the most frequently measured.

In 1945, Holburn[277] first stated that concussions were a result of rotational movements with or without direct impact, and termed this 'rotational acceleration'. It was later proposed that rotational acceleration was the cause of gliding contusion resulting from excessive strain of the cerebral blood vessels.[382] Rotational acceleration was reported to contribute more than linear acceleration to concussive injuries, diffuse axonal injuries and subdural haematomas.[310] Rotational, or shearing forces, applied to the head deform brain tissue more readily than any other biological tissues, and this is the predominant mechanism of injury in concussion.[441, 482] Rotational acceleration-deceleration may contribute to, and is linked with, cytoskeleton damage in animal models of concussion.[52, 361]

**Forces associated with concussion**

There is limited published research on the rotational and linear accelerational forces associated with concussion (see Table 6). The majority of the data have come from American football matches at various levels of participation by on-field monitoring, and using impact reconstruction of concussive impacts.[398, 497, 498, 605, 606] In laboratory reconstructions, it was reported that concussive events have occurred with face mask linear accelerations of 78 ±18 *g*[497] to 94 ±27 *g*[310] but greater accelerations were recorded on other areas of the head, with an average of 107-117*g*.[497] Using a helmet telemetry system, real-time head acceleration measurements were recorded for high school football players, with peak linear accelerations varying from 55.7 to 136.7*g* for concussive events.[53] This was extended further with the helmet telemetry system monitoring impacts in collegiate football players, with peak linear accelerations ranging from 60 to 120*g* for concussive events, with the majority of peak accelerations being above 95*g*.[28, 29, 253, 254]

Laboratory reconstructions have also provided rotational accelerations.[310, 497, 498, 606] In duplicating National Football League (NFL) concussive events, rotational accelerations of 6,398 ±1,798 radians per second per second (rad/s$^2$) occurred for concussive events.[310] Studies using the helmet telemetry systems on high school football players measured rotational accelerations of 163.4-8,994.4 rad/s$^2$ over 7-16 ms[28, 29, 49, 398] when concussive events occurred, while collegiate football players recorded higher rotational accelerations of 5,582-9,515 rad/s$^2$.[28, 254] Findings were similar in comparisons of collegiate football (7,092 ±1214 rad/s$^2$ over 9.6 ±2.8 ms)[49] with NFL (6,569 ±1,866 rad/s$^2$ over 9.7 ±1.7 ms).[605] Utilizing a logistic regression analysis on previously published data[498] there was a 75% chance of a concussion occurring[310] when an event occurred that resulted in linear acceleration of 98*g* combined with rotational acceleration of 7,130

rad/s$^2$.[141] Unfortunately, not all studies reporting impact forces associated with concussive events have provided linear, rotational and impact duration data. From the studies that have reported all of these components, it can be seen that the force duration associated with concussive events varies by participation level.

Helmets that are used in these sports have some protective effect in regard to concussive events.[310] When conducting hybrid head tests with helmets for impact reconstruction, there was a 21-29% reduction in linear accelerations but an increase in rotational accelerations when compared with the non-helmeted tests.[310] To date, there are no published studies reporting the impact that results in concussive events from participation in non-helmeted sports such as rugby union and rugby league. Further research is warranted to investigate the impacts that occur in concussive events in non-helmeted sports to enable broader understanding of the forces associated with concussion.

**Table 6:** Linear and rotational forces, impact duration and standard deviations associated with non-concussive and concussive events.

| Author | Sports code | No. of concussions | Session type (no. of impacts[a]) | Linear acceleration, (*g*) [mean ± SD] | Rotational acceleration (rad/s$^2$), [mean ± SD] | Impact duration (ms) [mean ± SD] |
|---|---|---|---|---|---|---|
| **Non-concussive impact forces** | | | | | | |
| Broglio et al.[49] | High school football | N/A | Practice (29,287) | 24.8 ± 14.8 | 1,569.8 ± 1124.8 | 10.2 ± 3.8 |
| | | N/A | Game (25,312) | 26.7 ± 17.1 | 1,728.9 ± 1319.3 | 10.2 ± 3.6 |
| Guskiewicz et al.[254]; Guskiewicz and Mihalik[253] | Collegiate football | N/A | Game (104,714) | - | - | - |
| Duma et al.[141] | Collegiate football | N/A | Practice (2,114) | 32 ± 25 | 905 ± 1,075 (x-axis) | - |
| | | N/A | Games (1,198) | 32 ± 25 | 2,020 ± 2,042 (y-axis) | - |
| Reed et al.[521] | Bantam ice hockey | N/A | Games (2,989) | 22.1 ± 0.4 | 1,557.4 ± 26.9 | - |
| Viano and Pellman[605b] | NFL | N/A | Game | 67.8 ± 14.7 | 4,847.6 ± 929.8 | 7.9 ± 1.9 |
| Pellman et al.[497b] | NFL | N/A | Laboratory[b] | 59.7 ± 23.9 | 4,234.7 ± 1,716.3 | 7.1 ± 2.6 |
| | | N/A | Laboratory[c] | 56.2 ± 22.2 | 3,982 ± 1,402 | 9.3 ± 1.9 |
| Brolinson et al.[53] | Collegiate football | N/A | Game (11,604) | 20.9 ± 18.7 | - | - |
| Naunheim et al.[466] | High school football | N/A | Game | 29.2 ± 1.0 | - | - |
| | High school hockey | N/A | Game | 35.0 ± 1.7 | - | - |
| | High school soccer | N/A | Game | 54.7 ± 4.1 | - | - |
| **Concussive impact forces** | | | | | | |
| Broglio et al.[49] | High school football | 2 | Practice (3.5 ± 2.1[d]) | 105.9 ± 19.5 | 7,982.2 ± 21.3 | 10.0 ± 1.4 |
| | | 11 | Game (19.7 ± 21.3 [d]) | 100.0 ± 1.3 | 7,092.6 ± 1,214.2 | 9.6 ± 2.8 |
| Guskiewicz et al.[254]; Guskiewicz and Mihalik[253] | Collegiate football | 13 | - | 102.8 ± 32.0 | 5,311.6 ± 4,111.0 | - |
| Duma et al.[141] | Collegiate football | 1 | - | 81 | 7,912 | - |
| Viano and Pellman[605b] | NFL | 22 | Game | 94.3 ± 27.5 | 6,569.0 ± 1,866.4 | 9.7 ±1.7 |
| Pellman et al.[497b] | NFL | 25 | Game | 98.2 ± 28.1 | 6,552.4 ± 1,770.5 | 9.3 ± 1.9 |
| Brolinson et al.[53] | Collegiate football | 3 | Game | 89.1 ± 43.0 | - | - |
| Viano et al.[606] | NFL | 25 | Game (struck) | 98 ± 28 | 6,432 ± 1,813 | 9.3 ±1.9 |
| | | | Game (striking) | 58.5 ± 21.4 | 4,225 ± 1,405 | - |
| Beckwith et al.[28] | Collegiate and high school | 105 | Competition[e] | 112.1 ± 35.4 | 4,253 ± 2,287 | 4.3 ± 1.7 |
| Beckwith et al.[29] | Collegiate and high school | 105 | Competition[f] | 102.5 ± 33.8 | 3,977 ± 2,272 | 3.7 ± 1.6 |

a: Except where stated otherwise. b: Laboratory reconstruction of struck player in NFL collisions. c: Laboratory reconstruction of the striking player in NFL collisions. d: No. of impacts (mean ± SD) before concussion. e: Mean severity of impacts prior to immediately diagnosed concussions. f: Average peak linear and angular acceleration for all impacts associated with diagnosed concussion. *g*= gravitational acceleration; rad/s$^2$ = radians per second per second; ms = millisecond; N/A = not applicable; NFL= National Football League.

### *Knowledge and understanding*

Several studies have reported on the knowledge and understanding of concussion for team management[148, 298, 331, 477, 577, 601] and parents.[148, 576] Studies reporting coaches' knowledge and management of concussion

identified that 16%[477] to 51%[601] of coaches were unable to correctly identify factors relating to concussion recognition, management and prevention techniques. Between 40%[331] and 42%[601] of coaches thought that a player needed to lose consciousness for a concussion to occur. Over a quarter, 26%[331] to 32%[601] would not remove a concussed player from the field of play. Similarly, 20%[331] to 26%[601] would let a symptomatic player RTP, and 30%[331] to 50%[501] of coaches believed that head-gear could minimize the risk of concussion. In assessing parents' understanding of concussion[576] it was reported that 83% of parents believed they could recognize a concussion in their teenager, 5% reported that a player needed to lose consciousness for a concussion to occur, 19% of parents would not have their teenager see a medical practitioner if there was a suspected concussion, and 4% would let a symptomatic teenager RTP.

Studies on the effectiveness of concussion education resources, such as the Centre for Disease Control concussion education website, found that 80%[246] of football coaches identified these as 'moderately useful', 63% of coaches realised concussions were more serious than previously thought, 50% of coaches made future changes in regard to dealing with concussion, and 72% of coaches used the resources to educate other coaches, parents and athletes.[100] Comparison is difficult, as no studies have reported changes in coach and team management awareness and knowledge through the use of concussion education in sporting activities. Future studies reporting the knowledge and awareness of team management are warranted.

***Health practitioner knowledge and management of concussion***

Concussion clinical practice guidelines (CPGs) are available,[112, 468] but their utilization by health professionals has not been well documented.[503] The quality and consistency of the concussion CPGs have been reported to be lower than those of other medical CPGs.[32] They have considerable variability in the methodological quality, guidance[32, 500] and recommendations.[500] Stakeholder involvement[532, 583] and consideration is limited for the applicability of the recommendations (i.e. cost implications, monitoring procedures, etc.).[532] In some cases, the guidelines conflicted in the management, which may affect the decision-making process for follow-up and further management.[583]

Studies[24, 210, 503] have shown that 27-32% of the discharge instructions provided to patients agreed with available published guidelines for RTP following a concussion. This was similar for primary care providers, with only 20% indicating that they utilized guidelines in the management of concussion.[503] Emergency department practitioners' use of concussion guidelines was slightly greater with 44% of those surveyed indicating that they utilized a concussion guideline in the management, but there was little consistency in the guideline being utilized.[220]

In regard to RTP decisions, there were differences reported for both primary care providers and emergency department practitioners.[23, 24, 220, 393, 503] Of concern was that 7% of emergency department practitioners would return the patient to the same match, 31% in 1 day and 27% in 1 week if asymptomatic. A third (33%) recommended RTP only after clearance by a physician for an initial concussion, but 67% recommended this for a subsequent concussion. Twelve percent[476] of primary care providers have been reported to use RTP

guidelines, identifying that clinical examination (89-93%)[101] was the most commonly used method for RTP decisions.

The most common reason for the variations in the management of concussion is lack of awareness of, and confusion with, the many published guidelines that are available.[24, 362, 503] The use of differing guidelines for the assessment and management of concussion may be the primary reason for variations reported in health professional's RTP decisions.[220, 503] The most frequently cited guidelines utilized in RTP decisions by health professionals were the American Academy of Neurology, Colorado Medical Society Guidelines and Cantu guidelines.[220, 503] The new guidelines indicate no RTP when a concussion is diagnosed or suspected. We need adherence to these guidelines in the sporting context.

To date there is a paucity of research on emergency department and primary care practitioners' management of concussion. An advice card may be given out with information on when to RTP if symptoms return or persist - but once a structural injury is ruled out, little consideration is provided to the functional brain injury.

Additionally, a lot of the published research reporting on primary care providers and emergency department management of concussion was prior to the publication of the third CISG guidelines and the recent increase in concussion awareness. Further research is warranted to explore these areas to assist in the development of evidenced-based information for this group of health practitioners. Until this evidence is available, the decision will remain a clinical one, utilizing the guidelines from the new consensus statement in April 2013 that advocate the use of the King-Devick visual test, clinical reaction tests with the new systems of SCAT3, Child-SCAT, a concussion recognition tool and other neuropsychological testing systems.

***Academic considerations***

There is a paucity of studies reporting on academic-level sport participants such as students at primary, secondary and tertiary level, and the management of the individual with a concussion on reintegration into the academic environment. To date, there are no published standardized guidelines for the return of a sport participant to academic studies.[263] From the studies reporting on the academic consequences of concussion, it has been reported that students may struggle to concentrate, may fail to perform academically, and may have verbal and memory learning difficulties.[360, 429, 460] Students with two or more concussions and a learning disability had significantly worse executive functioning and speed of mental processing test results, when compared with students with a similar concussion history and no learning disability.[90] Persistent neurocognitive difficulties were reported in high school athletes with two or more concussions.[460] These students had lower cumulative academic grade averages, and it was uncertain whether this was as a result of the concussion history, a characteristic of predisposition for concussion or a combination of both of these factors.[460]

It has been recommended that a period of cognitive rest is needed to prevent exacerbation of the symptoms of concussion.[259, 429, 459, 461] This may include a leave of absence from the school environment,[288, 429, 459, 461] shortening of the academic day, reductions in workloads in school and allowance of more time to complete

assignments or tests.[341, 429, 459, 461] Although there are some studies reporting on the effects of concussion at high school-level,[460] collegiate-level[34] and secondary-level[360] academia, there are no published studies to date on lower levels of academia nor on academic institutions outside the USA. Additional resources to help explain student academic issues as a result of concussion should be developed for both clinicians and educators.[263] Future research into the return-to-academia procedures is warranted.

***Implications for the assessment and management of concussion***

The prevention of concussions should be a priority, as athletes will always be exposed to head impacts.[240] The incidence can be mitigated to some degree by education of athletes,[39] coaches and team management,[331, 477, 601] family members[438] and health professionals.[24, 59, 80] Development of up-to-date educational resources is a priority to enable dissemination of appropriate up-to-date information for all people involved in the identification and management of a concussion. Although there are resources available through various international sources,[80, 100] individual sporting codes should also be responsible for the dissemination of this information to participants, volunteers, medical personal and family members.

Often players do not recognize that a concussion may have occurred, with as few as 23% reporting symptoms that may represent a concussion.[128] McCrea et al.[409] identified that only 47% of high school football players reported having sustained a concussion during match or training activities. Often the assessment and management of concussions relies upon visual cues to a concussion occurring, such as head clashes, player stumbling, loss of consciousness or players reporting they have had a concussive event.[521] Consequently, a number of concussions remain unnoticed.[141, 620]

Head impact systems are effective at recording head accelerations during various sporting activities[53, 141] and have been used to assist in the development of injury tolerance curves, severity indices and identification of individual players requiring further medical assessment. Despite this technological development, head impact telemetry systems are not a diagnostic tool for the assessment of concussion,[53] but can assist in identification of potentially concussed players needing further medical assessment. By monitoring players through a head impact telemetry system, medical personnel can monitor the impacts that players receive and have a visually recorded cue, should an unreported or unwitnessed concussive event occur. This technology is in its infancy and may well be out of reach of amateur-level sport participation, but research at higher levels of sport participation may be beneficial to identify the impacts that do occur. As the published literature is limited to a few sports (e.g. ice hockey,[521] football,[53, 141] soccer[261]), further longitudinal studies are warranted in a wider spectrum of sports to assist in broadening knowledge of the effects of impacts in sports and concussion.

Management of concussion varies depending on the knowledge of the practitioner and the information and resources that are available to assist with decision making.[24, 503] Information provided to the individuals can vary[24, 503] and this may unwittingly place the person with a concussion at risk of a more serious injury should they receive another concussion.[23, 24, 210, 220, 393, 503] Although research has been increasing in recent years and the information available changes, medical facilities and sporting organizations need to provide the most appropriate up-to-date identification, management and return-to-sport protocols to sport participants. This

may require regular reviews of the policies and standards available to ensure these are current. For example, the TBI guideline[468] available to health practitioners in New Zealand was produced in 2007 and, despite the publication of the CISG consensus statement, this has not been revised nor have there been any updates to this guideline.

Diagnosing a concussion represents a significant clinical challenge.[240] Often concussion severity and significance cannot be definitively determined until symptom resolution.[240] Sideline injury screenings employ symptom checklists, cognitive tests and balance evaluations that are reasonably sensitive, and specific, for the initial identification of a concussion.[202, 203] This is dependent upon two critical factors: (1) the player must be identified as having a suspected concussion; and (2) someone must be available to conduct the screening process.[240] If either of these factors is not present, then there is a risk of a suspected concussion being missed.[240] At the amateur level, this is often the problem, and more than 50% of concussions remain unreported.[409] To assist in reducing the number of missed concussions, sports codes could include specific concussion assessment educational sessions, make sideline assessment tools readily available and encourage use of appropriately trained qualified personnel in head injury management at all levels of participation.

The effects of multiple or repetitive concussions are not well understood but are currently being researched.[240] There is animal study evidence of the brain's increased vulnerability to structural and/or functional damage following a concussive injury.[240, 361] There is anecdotal evidence that recent concussive impacts increase the likelihood and severity of functional impairment to the brain.[249, 250, 435] There is a strong connection[126] between the effects of concussion, especially repeat concussion, and brain function decline[98, 250] such as the progressive neurodegenerative syndrome and CTE.[574] This may be caused by either a single, an episodic or repetitive blunt force impacts to the head.

Although research into CTE, and its connection to concussion, is in its infancy,[21] it is proposed that widespread monitoring of concussion should occur at all levels of participation.[240] This should be done for concussive as well as subconcussive incidents. This has the potential to enhance the identification of a concussive event and may assist in the reduction of underreporting.[240]

## Conclusions

Concussion still remains a mystifying subject in the sports medicine arena. The underreporting of concussion makes identifying the true incidence difficult, and makes diagnosis and management of concussion a challenge. The pathophysiological effects of a concussion can be seen from within 24 h of the event, occurring up to several weeks post-injury. As a result of increased metabolic dysfunction, the brain has increased vulnerability to other consequences. A concern with the reporting of concussion is that studies have utilized different definitions, methodologies (retrospective and prospective) and reporting modalities. The CISG has made advances in the identification and management of concussion; however, until the CISG definition of concussion is universally used by all researchers, the true incidence and cost of concussion will not be known.

Symptoms of concussion are variable, and measurement of these symptoms depends on self-reporting. The CISG has produced SCAT3 representing the newest sideline concussion assessment tool to date. More recently, the King-Devick test has been used successfully to identify concussion. Initial sideline assessment should be undertaken to identify the athlete with a concussion, but this should only be part of a continuum of assessment modalities. More advanced assessments such as SCAT3 and Child-SCAT3 can then be undertaken to further evaluate the player with a suspected concussion. The use of head impact telemetry for head impacts has been shown to be an effective measurement tool. By monitoring players through an impact telemetry system, medical personnel can monitor the impacts that players receive and have a visually recorded cue when unreported or unwitnessed potential concussive events occur, but this will be beyond the financial capacities of many amateur sporting activities. Medical practitioners should be cognisant of the extent and nature of concussion and should work with parents, teachers, coaches, athletes and trainers to identify and manage concussions.

# Section 2:

# Epidemiology of concussion in New Zealand

***“He’s just trying to be a scaremonger and stop people from playing the game. The fact is that concussions are not a problem and medical people not involved in the game just need to harden up a bit more”***

Sports Code Medical director - 2014

# CHAPTER 3:

# SPORT-RELATED CONCUSSIONS IN NEW ZEALAND: A REVIEW OF 10 YEARS OF ACCIDENT COMPENSATION CORPORATION MODERATE TO SEVERE CLAIMS AND COSTS

This chapter comprises the following paper published in the *Journal of Science and Medicine in Sports*

**Reference**

King, D.A., Gissane, C., Brughelli, M., Hume, P.A. & Harawira, J. Sport-related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs. *J Sci Med Sport* 2014; **17**(3): 250-255.

**Author contribution:**

D King 80%, C. Gissane 5%, M Brughelli: 5%, P Hume 5%, J Harawira 5%.

## Overview

**Objective:** This paper provides an overview of the epidemiology of sport-related concussion and associated costs in New Zealand requiring medical treatment from 2001 to 2011 in seven sports codes.

**Methods:** Data were analysed by sporting code, age, ethnicity, gender and year of competition for total and moderate-to-severe (MSC) Accident Compensation Corporation (ACC) claims and costs.

**Results:** A total of 20,902 claims costing $NZD 16,546,026 were recorded over the study period of which 1,330 (6.4%) were MSC claims. The mean yearly number and costs of MSC claims was 133 ±36 and $1,303,942 ±$378,949. Rugby union had the highest number of MSC claims per year (38; 95% CI 36 to 41 per 1,000 MSC claims). New Zealand Māori recorded the highest total ($6,000,759) and mean cost ($21,120) per MSC claim.

**Discussion:** Although MSC injury claims were only 6.4% of total claims, they accounted for 79.1% of total costs indicating that although the majority of sport-related concussions may be minor in severity, the related economic costs associated with more serious sport-related concussion can be high. The finding that rugby union recorded the most MSC claims in the current study was not unexpected. Of concern is that rugby league recorded a low number of MSC claims but the highest mean cost per claim. Due to the high mean cost per concussion, and the high total and mean cost for New Zealand Māori, further investigation is warranted.

## Introduction

Sport-related concussions are topical in the sports medicine domain. In the United States of America 1.6 to 3.8 million sports related concussions occur annually[358] with a cumulative (direct and indirect) cost estimated

to be US$56 billion.[358] Most sport-related concussions occur in sports that involve physical contact between participants.[34] However rates are also high for non-contact sports such as baseball and volleyball.[506] Despite the increasing number of international studies on sport-related concussion, there is a paucity of studies describing sport-related concussion in New Zealand sports-participants. To date no published studies have undertaken an epidemiological review of the costs to a nation from sport-related concussion in different sporting activities. To enable appropriate targeting of injury prevention initiatives, we were interested in differences for sport-related concussion incidence and characteristics by age, sports code and gender. Given the ethnic differences in the New Zealand population, it was identified that there may be variance in the ethnic representation of sport-related concussions. With a population of 4.1 million[569] the four largest ethnic groups were New Zealand Europeans (61.2%), New Zealand Māori (13.2%), Pacific Peoples (6.2%), and Asian (8.4%). With this in mind the aim of this study was to provide an epidemiological overview of sport-related concussion and associated costs over 10 years. New Zealand's national taxpayer funded no-fault injury compensation system administered by the Accident Compensation Corporation (ACC) means that New Zealand is uniquely positioned to provide detailed descriptive epidemiological data including costs associated with treatment. Using these data, comparisons of the incidence and cost of concussions for players in seven sports with comparisons by gender, ethnicity and age over the 2001/2002 to 2010/2011 reporting periods were conducted.

## Methods

As there is no reliable data capturing system for concussive injuries through national sports organizations, the ACC database was utilised to provide detailed descriptive epidemiological data including costs associated with treatment for injuries that occur in sporting activities. The database records the number of injury claims but is unable to report missed match and training time, hospitalization duration and level of participation. ACC records, and reports, on two types of acute personal injury claims.[215] These are Minor and Moderate-to-Serious claims (MSC). Both are defined under the Injury Prevention, Rehabilitation and Compensation (IPRC) Act, 2001 with ACC responsible for meeting the costs of the injuries.[215] People qualify for cover when they present with a personal acute injury as a result of an accident to any of the ACC recognised 30,000 registered medical practitioner's throughout New Zealand.[215] A claim is classified as 'minor' when ACC only pays for the registered medical practitioner (e.g., Physiotherapist, General Practitioner) for the medical treatment provided.[215] Typically this involves a few treatments with ACC meeting most of the costs.[215] To be classified as MSC, these injuries usually require assistance beyond medical treatment alone.[215] MSC's may involve a combination of medical care, rehabilitation costs and income replacement for employment time lost as a result of the injury.[215] Each ACC claim can only be lodged once. Payments for a claim may be incurred in subsequent years, but were be included in the total for the year of lodgment of the claim.

For the purpose of the study, we focused on MSC claims that were recorded from 1st July 2001 to 30th June 2011 as a result of participating in top seven sporting activities that recorded the highest ACC MSC claims. The definition utilised for this study was *"any injury that had been assessed and reported by a registered*

*health practitioner as a result of sports participation. The injury had to have been classified and recorded as a concussion utilising the ACC read code (S60..). The injury had to have been accepted as being an ACC claim during the study period to be recorded in the study dataset.*"

Epidemiological studies are dependent on data quality for any analysis to be undertaken.[216] Data provided for our analyses was from the ACC database and this is dependent on several factors.[337] This database was utilised as there were no other available databases for collection of player specific data such as numbers participating in the different sports code activities, age of players participating, identification of the ethnicity of players, and number of matches/events/activities completed enabling calculation of match and training exposure hours. A potential identified limitation related to the use of this database is the way the data is retrieved to protect client confidentiality by limiting the access to low level results. As a result any data less than, or equal to, three injury claims was rounded to represent three claims only.

As there were no reliable participation data collected by the different sports-codes, New Zealand population data was obtained from official government data. This data provide estimates of resident populations between each five year census.[569] The population of New Zealand over the study period was ~4.1 million people based on the 2006 census.[569]

Ethical consent was sought from the AUT Ethics Committee but was not required. Informed consent from the injured participants was not obtained as de-identified data were collected from the ACC data base without individual participant identification or follow-up.

All data collected were entered into a Microsoft Excel spreadsheet and analysed with MedCalc for Windows, version 12.0 (MedCalc Software, Mariakerke, Belgium). Injury incidences were calculated as number of injuries per 1,000 MSC claims.[337] Data are reported as means and standard deviations (±SD) with 95% confidence intervals (CI) where appropriate.[591] Injury incidences were compared for two selected periods chosen as the start (2001-2002) and end (2010-2011) of the study period (2001-11). In addition comparisons were undertaken for successive years over the study period (2001-11) using each yearly data point for analysis. Comparisons between reporting years were calculated using a one sample chi-squared ($\chi^2$) test. Costs are reported in NZ Dollars ($).

## Results

Over 2001-11 there were 20,902 sport-related concussion claims from seven sports codes costing $16,546,026. The mean ±SD number and cost per year of sport-related concussion MSC claims for the seven sports codes was 299 ±6 and $2,363,719 ±$62,311. MSC-concussions claims represented 6.4% (1,330) of the total seven sports-codes claims but 78.8% ($13,039,416) of the costs. The MSC-concussion claims and costs increased from 2001-2002 to 2002-2003 (claims: 8.2%; costs: 2.8%) (see Table 7). The MSC-concussion claims varied from 85 (2001-2002; $1,101,537) to 188 (2009-2010; $1,824,667) but this was not significant ($\chi^2$=7; df=9; $p$=0.631). The mean number ±SD and costs of MSC-concussion claims over 2001-11 was 133 ±36 and $1,303,942 ±$378,949. The mean cost per MSC-concussion claim was $9,804 ±$8,859 while the mean cost per non-MSC-concussion claim was $182 ±$27 over 2001-11.

**Table 7:** Total number, rate and percentage of seven sports codes' for total number and rate per 1,000 MSC claims with 95% confidence intervals and percentage of total claims for total concussion MSC claims, total and mean MSC costs and differences over reporting years for MSC claims.

| . | Total Concussion MSC Claims | | | Total Concussion MSC Costs | | Difference from previous year for total MSC Injury claims | | Difference from 2001-02 for Total MSC injury claims | |
|---|---|---|---|---|---|---|---|---|---|
| **Years** | **No.** | **Rate *(95% CI)*** | **%[a]** | **NZD** | **Mean NZD** | **$\chi^2$ (df=1), *p* value** | | **$\chi^2$ (df=1), *p* value** | |
| 2001-2002 | 85 | 41.2 *(33.3 to 50.9)* | 4.1 | $1,101,537 | $12,959 | - | | - | |
| 2002-2003 | 92 | 44.9 *(36.6 to 55.1)* | 4.5 | $1,131,918 | $12,303 | 0.28 | 0.599 | 0.28 | 0.599 |
| 2003-2004 | 118 | 54.6 *(45.6 to 65.4)* | 5.5 | $ 944,658 | $8,006 | 3.22 | 0.073 | 5.36 | 0.021 |
| 2004-2005 | 110 | 52.7 *(43.7 to 63.6)* | 5.3 | $ 930,160 | $8,456 | 0.28 | 0.596 | 3.21 | 0.073 |
| 2005-2006 | 125 | 59.6 *(50.0 to 71.0)* | 6.0 | $1,005,168 | $8,041 | 0.96 | 0.328 | 7.62 | 0.006 |
| 2006-2007 | 120 | 56.8 *(47.5 to 67.9)* | 5.7 | $1,125,872 | $9,382 | 0.10 | 0.749 | 5.98 | 0.015 |
| 2007-2008 | 158 | 74.7 *(63.9 to 87.3)* | 7.5 | $1,325,756 | $8,391 | 5.19 | 0.023 | 21.93 | <0.001 |
| 2008-2009 | 182 | 86.1 *(74.5 to 99.6)* | 8.6 | $1,919,143 | $10,545 | 1.69 | 0.193 | 35.24 | <0.001 |
| 2009-2010 | 188 | 90.4 *(78.3 to 104.3)* | 9.0 | $1,824,667 | $ 9,706 | 0.10 | 0.755 | 38.86 | <0.001 |
| 2010-2011 | 152 | 75.4 *(64.3 to 88.3)* | 7.5 | $1,730,537 | $11,385 | 3.81 | 0.051 | 18.94 | <0.001 |
| **Total** | **1330** | **63.6 *(60.3 to 67.1)*** | **6.4** | **$13,039,416** | **$ 9,804** | **-** | | **-** | |

Rate per 1,000 total injury claims; CI: Confidence Interval. (a)=Percentage of total claims for the sports codes recorded for 2001-11.

**Table 8:** Sports code and gender concussion injury summary for total number and rate per 1,000 MSC claims with 95% Confidence intervals for total concussion MSC claims, total and mean MSC costs and differences over reporting years for MSC claims.

| | Total Concussion MSC Claims | | Total Concussion MSC Costs | | Difference over 2001-11 for total MSC injury claims | | Difference 2001-02 and 2010-11 for total MSC injury claims | |
|---|---|---|---|---|---|---|---|---|
| **Sport** | **No.** | **Rate *(95% CI)*** | **NZD** | **Mean NZD** | **$\chi^2$ (df=9), *p* value** | | **$\chi^2$ (df=1), *p* value** | |
| Rugby Union | 802 | 38.4 *(35.8 to 41.1)* | $6,252,870 | $7,797 | 39.3 | <0.001 | 7.7 | 0.006 |
| Soccer | 183 | 8.8 *(7.6 to 10.1)* | $1,143,408 | $6,248 | 36.6 | <0.001 | 2.6 | 0.108 |
| Rugby League | 179 | 8.6 *(7.4 to 9.9)* | $4,572,625 | $25,545 | 16.3 | 0.06 | 5.1 | 0.023 |
| Netball | 74 | 3.5 *(2.8 to 4.4)* | $841,426 | $11,371 | 19.8 | 0.019 | 6.3 | 0.012 |
| Hockey | 35 | 1.7 *(1.2 to 2.3)* | $164,661 | $4,705 | 15.6 | 0.076 | 6.0 | 0.014 |
| Touch Rugby | 34 | 1.6 *(1.2 to 2.3)* | $80,215 | $2,359 | 12.7 | 0.178 | 0.0 | 1.000 |
| Softball/Baseball | 23 | 1.1 *(0.7 to 1.7)* | $34,211 | $1,487 | 11.0 | 0.276 | 3.0 | 0.083 |
| **Female** | | | | | | | | |
| Netball | 69 | 3.3 *(2.6 to 4.2)* | $833,530 | $12,080 | 18.9 | 0.026 | 6.3 | 0.012 |
| Rugby Union | 67 | 3.2 *(2.5 to 4.1)* | $608,215 | $9,078 | 10.3 | 0.328 | 1.3 | 0.248 |
| Soccer | 51 | 2.4 *(1.9 to 3.2)* | $289,080 | $5,668 | 14.8 | 0.097 | 0.5 | 0.48 |
| Touch Rugby | 27 | 1.3 *(0.9 to 1.9)* | $54,099 | $2,004 | 3.0 | 0.964 | 0.0 | 1.000 |
| Hockey | 23 | 1.1 *(0.7 to 1.7)* | $30,801 | $1,339 | 8.0 | 0.534 | 3.0 | 0.083 |
| Rugby League | 12 | 0.6 *(0.3 to 1.0)* | $121,732 | $10,144 | 16.0 | 0.067 | 0.0 | 1.000 |
| Softball/Baseball | 9 | 0.4 *(0.2 to 0.8)* | $6,220 | $691 | 24.0 | <0.001 | 0.0 | 1.000 |
| **Total Female** | **258** | **12.3 *(10.9 to 13.9)*** | **$1,943,675** | **$7,554** | **41.8** | **<0.001** | **8**.0 | **0.005** |
| **Male** | | | | | | | | |
| Rugby Union | 735 | 35.2 *(32.7 to 37.8)* | $5,644,655 | $7,680 | 32.8 | <0.001 | 6.5 | 0.011 |
| Rugby League | 170 | 8.1 *(7.0 to 9.5)* | $4,450,893 | $26,182 | 13.3 | 0.15 | 5.1 | 0.023 |
| Soccer | 135 | 6.5 *(5.5 to 7.6)* | $854,328 | $6,328 | 24.2 | 0.004 | 1.3 | 0.248 |
| Touch Rugby | 25 | 1.2 *(0.8 to 1.8)* | $26,116 | $1,045 | 13.8 | 0.13 | 0.0 | 1.000 |
| Hockey | 21 | 1.0 *(0.7 to 1.5)* | $133,860 | $6,374 | 9.0 | 0.437 | 3.0 | 0.083 |
| Softball/Baseball | 18 | 0.9 *(0.5 to 1.4)* | $27,991 | $1,555 | 11.0 | 0.276 | 3.0 | 0.083 |
| Netball | 12 | 0.6 *(0.3 to 1.0)* | $7,896 | $658 | 16.0 | 0.067 | 0.0 | 1.000 |
| **Total Male** | **1116** | **53.4 *(50.3 to 56.6)*** | **$11,145,747** | **$9,987** | **50.3** | **<0.001** | **11.5** | **<0.001** |

CI: Confidence Interval

Rugby union (38.4; 95% CI 35.8 to 41.1 per 1,000 MSC-concussion claims) and soccer (8.8; 7.6 to 10.1 per 1,000 MSC-concussion claims) had the highest number of claims (see Table 8). Rugby union ($6,252,870) and rugby league ($4,572,625) recorded the highest total costs over 2001-11. Rugby league (92.2%;

$25,545) and netball (86.6%; $11,371) recorded the highest percentage of sport code total costs and mean costs per ACC claim. Males recorded significantly more MSC-concussion claims than females ($\chi^2$=535.8; df=1; $p$<0.001) (see Table 8). Males participating in rugby union recorded the highest total costs for MSC-concussion claims ($5,644,655). Males participating in rugby league recorded the highest mean costs per MSC-concussion claim ($26,182). Females participating in netball recorded the highest total ($833,530) and mean costs per female MSC-concussion claim ($12,080).

**Table 9:** Ethnicity and sports code concussion injury summary for total number and rate per 1,000 MSC claims with 95% Confidence intervals for total concussion MSC claims, total and mean MSC costs and differences over reporting years for MSC claims.

| | Total Concussion MSC Claims | | Total Concussion MSC Costs | | Difference over 2001-11 for total MSC injury claims | | Difference between 2001-02 and 2010-11 for total MSC injury claims | |
|---|---|---|---|---|---|---|---|---|
| | No. | Rate *(95% CI)* | NZD | Mean NZD | $\chi^2$ (df=9), $p$ value | | $\chi^2$ (df=1), $p$ value | |
| **NZ European's** | **786** | **37.6 *(35.1to 40.3)*** | **$3,885,454** | **$4,943** | **77.2** | **<0.001** | **14.9** | **<0.001** |
| Rugby Union | 469 | 22.4 *(20.5 to 24.6)* | $2,076,589 | $4,428 | 36 | <0.001 | 6.5 | 0.011 |
| Soccer | 143 | 6.8 *(5.8 to 8.1)* | $906,236 | $6,337 | 25.6 | 0.002 | 2.9 | 0.09 |
| Netball | 55 | 2.6 *(2.0 to 3.4)* | $434,500 | $7,900 | 19.7 | 0.02 | 3.8 | 0.052 |
| Rugby League | 41 | 2.0 *(1.4 to 2.7)* | $331,312 | $8,081 | 10.5 | 0.312 | 5.0 | 0.025 |
| Hockey | 31 | 1.5 *(1.0 to 2.1)* | $72,779 | $2,348 | 13.3 | 0.148 | 5.0 | 0.025 |
| Touch Rugby | 24 | 1.1 *(0.8 to 1.7)* | $29,827 | $1,243 | 13.0 | 0.163 | 0.0 | 1.000 |
| Softball/Baseball | 23 | 1.1 *(0.7 to 1.7)* | $34,211 | $1,487 | 11.0 | 0.276 | 3.0 | 0.083 |
| **NZ Māori** | **322** | **15.4 *(13.8 to 17.2)*** | **$6,800,759** | **$21,120** | **11.6** | **0.239** | **0.5** | **0.46** |
| Rugby Union | 173 | 8.3 *(7.1 to 9.6)* | $2,454,246 | $14,186 | 14.9 | 0.093 | 9.8 | 0.002 |
| Rugby League | 89 | 4.3 *(3.5 to 5.2)* | $3,880,800 | $43,604 | 7.1 | 0.626 | 0.3 | 0.617 |
| Netball | 30 | 1.4 *(1.0 to 2.1)* | $379,549 | $12,652 | 0.0 | 1.000 | 0.0 | 1.000 |
| Touch Rugby | 18 | 0.9 *(0.5 to 1.4)* | $48,204 | $2,678 | 8.0 | 0.534 | 3.0 | 0.083 |
| Soccer | 12 | 0.6 *(0.3 to 1.0)* | $37,960 | $3,163 | 16.0 | 0.067 | 0.0 | 1.000 |
| **Pacific Peoples** | **144** | **6.9 *(5.9 to 8.1)*** | **$1,566,595** | **$10,879** | **14.3** | **0.113** | **4.2** | **0.041** |
| Rugby Union | 108 | 5.2 *(4.3 to 6.2)* | $1,458,222 | $13,502 | 8.7 | 0.463 | 1.5 | 0.221 |
| Rugby League | 36 | 1.7 *(1.2 to 2.4)* | $108,373 | $3,010 | 14.0 | 0.122 | 5.0 | 0.025 |
| **Asian** | **33** | **1.6 *(1.1 to 2.2)*** | **$105,891** | **$3,209** | **22**.0 | **0.009** | **1**.0 | **0.317** |
| Rugby Union | 12 | 0.6 *(0.3 to 1.0)* | $11,291 | $941 | 16.0 | 0.067 | 0.0 | 1.000 |
| Hockey | 9 | 0.4 *(0.2 to 0.8)* | $86,017 | $9,557 | 18.0 | 0.035 | 0.0 | 1.000 |
| Soccer | 9 | 0.4 *(0.2 to 0.8)* | $7,861 | $873 | 18.0 | 0.035 | 3.0 | 0.083 |
| Touch Rugby | 3 | 0.1 *(0.0 to 0.4)* | $722 | $241 | 6.0 | 0.74 | 0.0 | 1.000 |
| **Unknown/Other** | **182** | **8.7 *(7.5 to 10.1)*** | **$730,723** | **$4,015** | **8.4** | **0.49** | **0.3** | **0.564** |
| Rugby Union | 65 | 3.1 *(2.4 to 4.0)* | $252,524 | $3,885 | 1.0 | 0.999 | 0.0 | 1.000 |
| Soccer | 42 | 2.0 *(1.5 to 2.7)* | $191,354 | $4,556 | 10.5 | 0.312 | 3.0 | 0.083 |
| Rugby League | 36 | 1.7 *(1.2 to 2.4)* | $252,136 | $7,004 | 4.7.0 | 0.859 | 0.0 | 1.000 |
| Netball | 21 | 1.0 *(0.7 to 1.5)* | $27,380 | $1,304 | 16.0 | 0.067 | 0.0 | 1.000 |
| Hockey | 15 | 0.7 *(0.4 to 1.2)* | $5,866 | $391 | 15.0 | 0.091 | 3.0 | 0.083 |
| Touch Rugby | 3 | 0.1 *(0.0 to 0.4)* | $2,185 | $728 | 6.0 | 0.74 | 0.0 | 1.000 |

CI: Confidence Interval.

New Zealand European's recorded the highest number of MSC claims (37.6; 35.1 to 40.3 per 1,000 MSC claims; $\chi^2$=77.2; df=9; $p$<0.001) over the duration of the study and this was significant (see Table 9). New Zealand Māori (15.4; 13.8 to 17.2 per 1,000 MSC-concussion claims) recorded the highest total ($6,000,759) and mean costs ($21,120) per MSC-concussion claims over 2001-11. Over 2001-11 period, New Zealand Māori recorded the highest mean costs per MSC-concussion claim for rugby league ($43,604) and rugby union ($14,186) (see Table 9). There were significant differences over the duration of the study for the number of MSC-concussion claims for New Zealand Europeans participating in rugby union ($\chi^2$=36.0; df=9; $p$<0.001).

The 20-29 age group recorded the highest MSC-concussion costs ($4,820,083) (see Table 10). The 30-39 age group recorded the highest mean costs per MSC-concussion claim ($19,910). The number of concussion injury entitlement claims increased over the duration of the study for the 0-9 ($\chi^2$=36.3; df=9; $p$<0.001), 10-19 ($\chi^2$=49.0; df=9; $p$<0.001) and 20-29 ($\chi^2$=46.7; df=9; $p$<0.001) age groups. People participating in rugby league activities in the 30-39 age group recorded the highest total ($2,268,398) and mean cost ($48,211) per MSC-concussion claim (see Table 4). Although people participating in rugby union activities in the 20-29 age group recorded the highest total MSC-concussion costs ($2,150,341) the 30-39 age group recorded the highest mean cost ($17,376) per MSC-concussion claim.

**Table 10:** Sports code and age concussion injury summary for total number and rate per 1,000 MSC claims with 95% Confidence intervals for total concussion MSC claims, total and mean MSC costs and differences over reporting years for MSC claims.

| | Total Concussion MSC Claims | | Total Concussion MSC Costs | | Difference over 2001-2011 for total MSC injury claims | | Difference between 2001-02 and 2010-11 for total MSC injury claims | |
|---|---|---|---|---|---|---|---|---|
| **Age** | **No.** | **Rate *(95% CI)*** | **Total NZD** | **Mean NZD** | **$\chi^2$ (df=9), *p* value** | | **$\chi^2$ (df=1), *p* value** | |
| **Hockey** | | | | | | | | |
| 10-19 | 23 | 1.1 *(0.7 to 1.7)* | $46,824 | $2,036 | 23.0 | 0.006 | 4.0 | 0.046 |
| 20-29 | 18 | 0.9 *(0.5 to 1.4)* | $15,432 | $857 | 14.0 | 0.122 | 3.0 | 0.083 |
| 30-39 | 3 | 0.1 *(0.0 to 0.4)* | $3,246 | $1,082 | 6.0 | 0.740 | 0.0 | 1.000 |
| 40-49 | 6 | 0.3 *(0.1 to 0.6)* | $13,145 | $2,191 | 21.0 | 0.013 | 3.0 | 0.083 |
| 50-59 | 9 | 0.4 *(0.2 to 0.8)* | $86,017 | $9,557 | 24.0 | 0.004 | 0.0 | 1.000 |
| **Netball** | | | | | | | | |
| 0-9 | 3 | 0.1 *(0.0 to 0.4)* | $328 | $109 | 6.0 | 0.740 | 3.0 | 0.083 |
| 10-19 | 23 | 1.1 *(0.7 to 1.7)* | $36,921 | $1,605 | 11.0 | 0.276 | 3.0 | 0.083 |
| 20-29 | 43 | 2.1 *(1.5 to 2.8)* | $612,670 | $14,248 | 8.0 | 0.534 | 1.0 | 0.317 |
| 30-39 | 24 | 1.1 *(0.8 to 1.7)* | $171,045 | $7,127 | 6.6 | 0.679 | 0.0 | 1.000 |
| 40-49 | 3 | 0.1 *(0.0 to 0.4)* | $431 | $144 | 6.0 | 0.740 | 3.0 | 0.083 |
| 50-59 | 3 | 0.1 *(0.0 to 0.4)* | $13,191 | $4,397 | 6.0 | 0.740 | 0.0 | 1.000 |
| 60+ | 12 | 0.6 *(0.3 to 1.0)* | $6,845 | $570 | 16.0 | 0.067 | 0.0 | 1.000 |
| **Rugby Union** | | | | | | | | |
| 0-9 | 3 | 0.1 *(0.0 to 0.4)* | $1,838 | $613 | 6.0 | 0.740 | 0.0 | 1.000 |
| 10-19 | 350 | 16.7 *(15.1 to 18.6)* | $1,792,876 | $5,123 | 21.5 | 0.011 | 4.4 | 0.036 |
| 20-29 | 317 | 15.2 *(13.6 to 16.9)* | $2,150,341 | $6,783 | 25.3 | 0.003 | 7.2 | 0.007 |
| 30-39 | 113 | 5.4 *(4.5 to 6.5)* | $1,963,506 | $17,376 | 4.4 | 0.884 | 0.4 | 0.513 |
| 40-49 | 22 | 1.1 *(0.7 to 1.6)* | $227,415 | $10,337 | 10.0 | 0.351 | 0.0 | 1.000 |
| 50-59 | 12 | 0.6 *(0.3 to 1.0)* | $91,754 | $7,646 | 16.0 | 0.067 | 0.0 | 1.000 |
| 60+ | 9 | 0.4 *(0.2 to 0.8)* | $25,142 | $2,794 | 24.0 | 0.004 | 0.0 | 1.000 |
| **Rugby League** | | | | | | | | |
| 10-19 | 48 | 2.3 *(1.7 to 3.0)* | $130,352 | $2,716 | 21 | 0.013 | 4.0 | 0.046 |
| 20-29 | 64 | 3.1 *(2.4 to 3.9)* | $1,694,239 | $26,472 | 6.2 | 0.717 | 1.3 | 0.248 |
| 30-39 | 47 | 2.2 *(1.7 to 3.0)* | $2,265,938 | $48,211 | 12.1 | 0.207 | 0.5 | 0.48 |
| 40-49 | 32 | 1.5 *(1.1 to 2.2)* | $482,094 | $15,065 | 1.3 | 0.998 | 0.0 | 1 |
| **Soccer** | | | | | | | | |
| 0-9 | 9 | 0.4 *(0.2 to 0.8)* | $2,409 | $268 | 24.0 | 0.004 | 0.0 | 1.000 |
| 10-19 | 59 | 2.8 *(2.2 to 3.6)* | $245,364 | $4,159 | 10.3 | 0.324 | 0.1 | 0.706 |
| 20-29 | 63 | 3.0 *(2.4 to 3.9)* | $340,362 | $5,403 | 31.7 | <0.001 | 0.1 | 0.706 |
| 30-39 | 37 | 1.8 *(1.3 to 2.4)* | $281,209 | $7,600 | 5.3 | 0.809 | 0.0 | 1.000 |
| 40-49 | 33 | 1.6 *(1.1 to 2.2)* | $189,323 | $5,737 | 4.0 | 0.911 | 0.0 | 1.000 |
| 50-59 | 3 | 0.1 *(0.0 to 0.4)* | $10,145 | $3,382 | 6.0 | 0.740 | 3.0 | 0.083 |
| 60+ | 15 | 0.7 *(0.4 to 1.2)* | $74,595 | $4,973 | 15.0 | 0.091 | 3.0 | 0.083 |
| **Softball/Baseball** | | | | | | | | |
| 0-9 | 3 | 0.1 *(0.0 to 0.4)* | $812 | $271 | 6.0 | 0.740 | 0.0 | 1.000 |
| 10-19 | 9 | 0.4 *(0.2 to 0.8)* | $6,511 | $723 | 24.0 | 0.004 | 0.0 | 1.000 |
| 20-29 | 6 | 0.3 *(0.1 to 0.6)* | $2,160 | $360 | 21.0 | 0.013 | 0.0 | 1.000 |
| 30-39 | 3 | 0.1 *(0.0 to 0.4)* | $4,227 | $1,409 | 6.0 | 0.740 | 0.0 | 1.000 |
| 40-49 | 9 | 0.4 *(0.2 to 0.8)* | $20,501 | $2,278 | 24.0 | 0.004 | 3.0 | 0.083 |
| **Touch Rugby** | | | | | | | | |
| 10-19 | 16 | 0.8 *(0.5 to 1.2)* | $25,950 | $1,622 | 16.3 | 0.060 | 3.0 | 0.083 |
| 20-29 | 18 | 0.9 *(0.5 to 1.4)* | $4,879 | $271 | 11.0 | 0.276 | 0.0 | 1.000 |
| 30-39 | 9 | 0.4 *(0.2 to 0.8)* | $9,507 | $1,056 | 24.0 | 0.004 | 0.0 | 1.000 |
| 40-49 | 12 | 0.6 *(0.3 to 1.0)* | $39,879 | $3,323 | 16.0 | 0.067 | 3.0 | 0.083 |
| **All sport** | | | | | | | | |
| 0-9 | 18[bcdeg] | 0.9 *(0.5 to 1.4)* | $5,387 | $299 | 36.3 | <0.001 | 3.0 | 0.083 |
| 10-19 | 528[adefg] | 25.3 *(23.2 to 27.5)* | $2,284,798 | $4,327 | 49.0 | <0.001 | 11.5 | 0.001 |
| 20-29 | 529[adefg] | 25.3 *(23.2 to 27.6)* | $4,820,083 | $9,112 | 46.7 | <0.001 | 10.5 | 0.001 |
| 30-39 | 236[abcefg] | 11.3 *(9.9 to 12.8)* | $4,698,678 | $19,910 | 12.7 | 0.177 | 0.0 | 0.876 |
| 40-49 | 117[abcdfg] | 5.6 *(4.7 to 6.7)* | $972,788 | $8,314 | 4.2 | 0.900 | 0.0 | 1.000 |
| 50-59 | 27[abcde] | 1.3 *(0.9 to 1.9)* | $201,107 | $7,448 | 17.8 | 0.038 | 3.0 | 0.083 |
| 60+ | 36[abcde] | 1.7 *(1.2 to 2.4)* | $106,582 | $2,961 | 15.0 | 0.091 | 3.0 | 0.083 |

Rate per 1,000 total injury claims; CI: Confidence Interval. (a)=Percentage of total claims by age for the sports code recorded for 2001-11. Non-MSC age groups not included. Significant difference (p<0.05) than (**a**)=0-9; (**b**)=10-19; (**c**)=20-29; (**d**)=30-39; (**e**)=40-49; (**f**)=50-59; (**g**)=60+ age groups.

## Discussion

This study identified the number of ACC claims lodged, and the associated costs of sport-related concussions that occurred from participation in seven sports in New Zealand over a 10 year period (2001-11). As shown there were 20,902 injury entitlement claims reporting concussions as a result of sports participation but only 6.4% (1,330) of these were classified as MSC injury entitlement claims. The majority of the ACC claims (19,572; 93.6%) reflect of the number of sport-related concussions that resolved without further additional assistance. The numbers presented in this study are not an accurate reflection of how many concussions are occurring in the individual sporting codes but how many concussive injuries were recorded by the injured individual sporting code participants as a result of participating in that sport. As well the term MSC is for the accounting purposes by ACC and does not reflect the severity classification of the head injury itself. All the concussive injuries reported in this study were classified as concussions (S60..) and recorded on the ACC system but required additional assistance beyond mere medical treatment only.[215]

MSC claims accounted for 6.4% of the total sport-related concussions for the seven sports surveyed but accounted for 79.1% of total costs. This indicates that although sport-related concussions may be minor in severity, the related economic costs attached to a sport-related concussion with ongoing symptoms can be high. The finding that rugby union recorded the most MSC claims was not unexpected as this is the national game in New Zealand.[211] Previous international studies have identified that ice hockey[235] and the national football league (NFL)[371] have some of the highest incidences of concussion of all sports but these sports have low participation levels in countries such as New Zealand. Denominator data in terms of the number of participants exposed to concussion through play, and the hours of exposure to play in these sports is needed to enable an accurate comparison of the risk of concussion in different sports but this data is not available for this study.

A reason for high concussion claim numbers in sport may be related to the focus on concussion identification and management education resulting in increased reporting. Rugby league had the third highest number of claims (n=179) for reported concussions over the ten years. However, the overall knowledge level of concussion identification and management was only 42% for rugby league team coaches and trainers in New Zealand which was lower than other studies reporting concussion knowledge of team coaches (62%[601] to 84%[477]). Of 95 rugby league coaches and team managers, 26% reported they would not remove a player with symptoms of concussion, 20% would return a symptomatic player to participation and 39% reported that concussion only occurred when a player lost consciousness.[331] With the number of reported concussions in this study, and given the lack of knowledge surrounding concussion identification and management previously reported there is possibly an under-reporting of concussion in rugby league. Recently it has been reported that un-witnessed concussive events were identified with the use of a saccadic reading test in rugby league[317] and rugby union.[314] The number of un-witnessed to witnessed concussions occurred at a 3.4 to 1 ratio.[314] If this is indicative of the true incidence then the reported numbers, and costs in this study may only be a third of the actual incidence and economic costs associated with sport-related concussions.

Of concern in the current study is that rugby league recorded a low number of MSC claims but the highest mean cost per claim. In particular, males and Māori participating in rugby league activities recorded the highest mean costs of all sports. This may be reflective of the nature of rugby league when compared with rugby union or the management of these types of injuries. For example, the differences in the types and numbers of tackles between the two sports may expose players in rugby league to more head impacts resulting in more severe head trauma and therefore more cost per claim. Studies reporting on rugby union[516] and rugby league[332] tackles have reported that contact with the head during the tackle occurred at a rate of 0.6 per 1,000 tackles in rugby union[516] but at a rate of 89.9 per 1,000 tackles in rugby league.[332] This higher rate of contact with the head in rugby league may be reflected in the higher costs recorded in the current study. Further studies are warranted to explore the incidence of impacts that occur and the management of these injuries in rugby union and rugby league.

A limitation to the current study is the finding that 14% of claims and 6% of associated costs were reported as ethnicity unknown or other. This does limit the analysis by ethnicity but may be related to people not wanting to list their ethnicity or the available ethnic groups on the ACC forms do not include their ethnicity. Of the MSC claims reporting ethnicity Asians had the lowest and New Zealand Māori had the highest mean cost per MSC concussion claim. This finding is similar to a previous study reporting ethnicity variations in rugby league[337]. Interestingly the previous costs per Māori rugby league concussion claim has increased ($38,118[337] versus $43,604) but this is probably related to increasing costs of medical care due to the different reporting periods between these studies. The explanation to this finding is beyond the scope of this study but raises similar questions to the previous epidemiological study[337] reporting injury claims and costs: (1) Do New Zealand Māori have an increased risk of complications from concussions when compared to other ethnic groups? (2) Is there a difference for the costs associated with different ethnic groups in the medical management of concussion? (3) Are the sport-related concussions MSC claims from a single concussive event, a concussive event where the injured player has returned to participation before the concussion has been fully resolved (a repeat concussion)? and (4) Where the original injury was a structural mild Traumatic Brain Injury (sTBI) but has been misdiagnosed as a concussion from sports participation in these sporting codes? The reasons for the mean high cost per concussion, and in particular for New Zealand Māori, are areas that warrant further investigation.

The finding that MSC claims was higher for females (8.5%) than males (6.2%) was similar to previous studies.[106, 506] This may be reflective of the identified increased risk factors for female sports participants. These include being of a smaller stature, weaker neck muscles, less head/neck mass than males, increased head mass, greater acceleration of the head and neck and a lower ability to protect their heads compared with male sports-participants.[17, 135] In addition, females are more likely to report their injuries, utilise health systems and report symptoms than males.[34] Consequently there would be an increase in claim numbers when compared with males. Further studies on the anatomical, mechanical and physiological differences between male and female sports participants recording a sports related concussion are warranted.

The finding that some sporting codes do not have MSCs for players under the age of 9 yr. or for older age groups does not accurate estimate of the players by age in that sport. Similarly the recording of MSCs for younger players in other sporting codes does not provide an accurate estimate the risk of a concussion occurring in these sporting codes. The differences recorded between the age groups may be related to several factors. The low number of MSC claims may be reflective of the low sports-participation in this age group in the different sports codes. During Adolescence (10-19 yr) sports-participants may have increased risk of concussion due to reduced ability to dissipate forces applied to the head as a result of weaker neck muscles, decreased nerve myelination, greater head-to-body ratio and growth spurts resulting in increases in the body's weight and mass.[61] The physiological changes that occur to this age group can result in increased forces and momentum during a collision when participating in sports activities.[61] Adolescents and children may also have a protracted recovery after concussion when compared with adults as there may be a more diffuse and prolonged cerebral swelling can occur after an injury to the brain.[165] This suggests that adolescents and children may be more at risk for secondary intracranial hypertension and ischemia.

The reporting of MSCs for sports participants over the age of 39 years is not unexpected. Players over this age often participate in modified versions of the sports-code activity in annual events (e.g. Masters games) or in President or Veterans grades. These games are often designed to encourage continued sports activity, team participation and club membership. The 30-39 age group had the highest mean cost per MSC claim. This may be reflective of this age group likely being less compliant with current recommendations for return-to-play procedures. Or more likely the cost of work income replacement (costs were from earnings-related compensations claims) being greater in this age group compared with younger players. Future studies need to explore why this age group has an increased cost per concussion claim than the other age groups.

Given the findings of increased incidence in rugby union, increased cost per claim in male Māori rugby and rugby league players, and increased incidence in female players, injury prevention initiatives should be targeted at these types of players in New Zealand once greater understanding of the risk factors and mechanisms of injury are ascertained. Additional injury prevention initiatives may also include a wider understanding of concussion, the assessment, diagnosis and management education for team medical personnel and coaching staff; more support for the removal from play of players with suspected concussions to enable a complete sideline assessment and the identification of a removal-from-play tool that is usable at all levels of sports participation.

## Conclusions

The average cost per claim for a moderate to severe concussive injury varied by sports code, ethnicity, gender and age over a 10 year period for seven sporting codes in New Zealand. Māori rugby league males aged between 30 and 39 years, female players and rugby union players need to be targeted for injury prevention initiatives. A wider understanding of concussion identification for team coaches and first aiders and removal from play for assessment at all levels of participation may assist in the identification of concussive injuries. Further longitudinal studies with specific details on injury mechanisms and participation data are warranted to further explore the incidence of sport-related concussion that occurs in New Zealand.

***Practical implications***

- The costs of sport-related concussions can vary by ethnicity, gender and sporting activity completed;
- The majority of reported sport-related concussions resolve without further ongoing medical assistance;
- More education is required for all sports-team management on identification and management of sport-related concussions; and
- Māori sports participants may require a more personalised concussion rehabilitation program for returning to activities.

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***“It’s just a knock to the head, all you have to do is harden up and get back out there – give him a hit in the head so you’re even”***

Father of a 10 yr old player who had been knocked out - 2014

# CHAPTER 4:

# CONCUSSION IN AMATEUR RUGBY LEAGUE PLAYERS IN NEW ZEALAND: A REVIEW OF PLAYER CONCUSSION HISTORY

This chapter comprises the following paper published in *New Zealand Journal of Sports Medicine*

**Reference**

King, DA., Gissane, C. & Clark, T. Concussion in amateur rugby league players in New Zealand: A review of player concussion history *NZ J Sports Med* 2014, **40**(2): 64-69.

**Author contribution:**

D. King 85%, C. Gissane 10%, T. Clark 5%.


## Overview

**Aim:** To review self-reported concussive history of amateur rugby league players to identify concussions within a two year period, concussive events outside of this time frame and medical review and clearances for return-to-play.

**Method:** A retrospective analysis was undertaken on all medical pre-competition questionnaires completed during the 2010 to 2012 competition periods by amateur rugby league players.

**Results:** A total of 213 (mean ±SD: age, 19.2 ±4.4) amateur rugby league player concussion questionnaires were reviewed. There was an average of 4.0 ±2.6 concussive injuries per participant in the previous two years and an average of 5.0 ±4.6 concussive injuries per participant the period preceding this. A total of 7.5% participants saw a medical doctor for their concussion; 5.2% completed the required three week return-to-play; and 2.8% reported seeing a medical doctor for medical clearance. No Under 15 yr. old player reported seeing a medical doctor or having a stand down period for return to play.

**Discussion:** Players across all age groups were administered the same questionnaire in different settings where they were gathered for the respective age group competition meetings. An unexpected finding was that 7% of all players had active concussive symptoms. This was a cause for concern. A lack of knowledge of the risks and potential consequences of concussion was reported to be the most common reason for the non-reporting of a possible concussion.


## Introduction

Traditionally viewed as a transient injury void of long term consequences,[43] sport-related concussions were once trivialised by coaches and "playing through the pain" was regarded as a sign of the individual's toughness and commitment to the team.[43] Even clashes to the head that were often thought of as "just a ding" are now receiving more attention as a potentially serious injury.[595] More recently there has been an increased awareness of the long-term effects of sport-related concussion and the possible link with the

pathology termed chronic traumatic encephalopathy (CTE).[43] This link with CTE as well as other neurological conditions such as depression and mild cognitive impairment have been reportedly associated with players who have a history of more than one concussive event.[43, 425]

Players undertaking any sporting activity are at risk of receiving a concussive event.[434] The risk of receiving a concussive event is reported to be increased in players with a history of a previous or multiple concussive events.[291] A way to assist the team medical personnel to manage this risk is to record the players individual concussion history. Often this requires self-disclosure by the player or the management of a concussion history by the team medical person through sideline and post-match examinations. Recently there has been an increase in published research on sport-related concussion relating to the identification, assessment and management in all sporting environments.[123, 391] Concussion has become one of the most troublesome injuries facing the sports medicine professional,[347] especially with regard to the early identification of concussive signs and symptoms and appropriate concussive management facilitation.[273, 409] Amateur sports such as rugby league in New Zealand do not have the resources available for qualified medical personnel to be at every match.[331] As such it is often the coach or team manager, if the team has one who needs to make the decisions with regard to player welfare, and this includes concussion identification and management. The awareness of team management regarding concussion has been previously reported[331, 477, 601] and highlights the need for concussion education for all people involved in the management level of sports participation.

Players with a concussive injury returning to their sport are at a greater risk of complications.[291, 388] These complications are related to subsequent concussive events and may result in prolonged concussive symptoms and cumulative cognitive deterioration (Chronic Traumatic Encephalopathy (CTE)).[391] Often the identification of a sport-related concussion is left to the team coach as there are seldom any doctors available at the games and training sessions.[347] As a result, concussive signs may be missed, as the coach may be unable to take the required time to fully assess the player and may miss signs of a player with a concussion. The aim of this study was therefore to review self-reported concussive history of amateur rugby league players to identify concussions within a two year period, concussive events prior to this time frame and medical review and clearances for return-to-play.

## Methods

A retrospective analysis was undertaken on all medical pre-competition questionnaires completed during the 2010 to 2012 competition periods by amateur rugby league players in Wellington, New Zealand. The pre-competition questionnaires were requested as part of the assessment process for the provision of medical coverage to the players by one of the authors (DK) prior to the player commencing any match activities. In the case of representative players this occurred at the end of the domestic competition season and just prior to the representative competition season in the same calendar year. The questionnaires were reviewed for the concussion history of players for the previous two years, prior history of concussion and the Post-Concussion Symptom Scale that was used for the establishment of baseline data for players competing in various rugby league competitions.

### *Subjects*

Over the study period a total of 213 male participants (mean ±SD: age, 19.2 ±4.4 yr.) from domestic and representative amateur rugby league teams agreed to take part in recording their concussion history. All participants were provided with an information sheet and gave either personal informed consent to participate or had parental informed consent before completing the history questionnaire.

### *Concussion history questionnaire*

All players were asked about their concussion history (see Appendix VI). This included history of concussions in the current and previous playing seasons, number of concussions, residual symptoms from any concussions reported and medical clearance to return-to-play. Participants were also asked to list the total number of years they had played rugby league. Participants were also asked to identify whether they considered a mild Traumatic Brain Injury or a Concussion to be more serious, or whether they were the same.

### *Post-Concussion Symptom Scale (PCSS)*

The PCSS is a 22-item neuropsychological assessment scale that is a part of the larger Sport Concussion Assessment Tool (SCAT). Developed in the late 1980's,[381] the PCSS was designed to measure the severity of symptoms in the acute phase of recovery from a concussion. Using a 7-point Likert-type scale anchored at 0 (complete absence) to 6 (most severe) players rank each symptom according to the severity that they have experienced. The symptoms are not specific to concussion and even non-injured participants have recorded symptoms on the PCSS[381]. A combined total of 7 symptoms was set as the threshold for the duration of the study.[359] Any player reporting more than 7 symptoms on any assessment were referred for further medical evaluation. This threshold was adopted as an indicator of a head injury having occurred based on prior studies.[359] The internal consistency reliability (Cronbach's α) for the PCSS has been reported to range from 0.88 to 0.94.[381]

### Statistical Analysis

All the data collected were entered into a Microsoft Excel spreadsheet and analysed with Statistical Package for Social Sciences (SPSS v.19.0; IBM SPSS Inc., Chicago, IL, USA for windows). Data are reported as means and standard deviations (SD) where appropriate. Correlations between concussion history and symptoms reported were analysed using a Spearman's Correlation Coefficient ($r_s$). To ascertain reliability of the questionnaire and player recall of their concussion history, players who were selected in more than one group (n=20) had their concussion history computed with a weighted Cohen $\kappa$ coefficient.[85, 607] A one-way analysis of variance (ANOVA) was used to investigate differences between the reported concussions, previous history of concussions and the symptoms identified. Statistical significance was set at $p<0.05$. All data are reported as mean ±SD.

## Results

Participants reported an average of 4.3 ±2.7 yr. playing experience in rugby league. There was an average of 4.0 ±2.6 concussive injuries per participant in the previous two years (see Table 11). A total of 7.5% of all participants reported seeing a doctor for their reported concussion while only 5.2% completed the required three week return-to-play process. Even fewer players (2.8%) reported seeing a medical doctor for a clearance for return-to-play. No Under 15 yr. old player reported seeing a medical doctor or having a stand down period for return-to-play. It was identified that 7.0% (n=27) of the participants had high PCSS scores and were assessed to have an active concussion at the time the assessment was being conducted.

**Table 11:** Demographics of amateur rugby league participants for age, playing position group, playing experience, concussive history and associated symptoms for previous two years, concussive history for prior years, return to play requirements and current post-concussion symptoms scores and severity by means, standard deviations and ranges reported.

| | Total | U15 | U17 | Premier |
|---|---|---|---|---|
| **Demographics** | | | | |
| Age, average SD [range] | 19.2 ±4.4 [13-34] | 14.5 ±0.6 [13-15] | 16.5 ±0.5 [16-17] | 21.9 ±3.9 [18-34] |
| Forwards | 119 | 24 | 26 | 69 |
| Backs | 94 | 18 | 21 | 55 |
| Experience, average SD [range] | 4.3 ±2.7 [1-12] | 4.7 ±2.8 [1-9] | 3.4 ±2.5 [1-10] | 4.4 ±2.7 [1-12] |
| **Concussive history in previous two years** | | | | |
| Concussion, average SD [range] | 1.9 ±1.2 [0-5] | 1.8 ±1.2 [0-5] | 2.0 ±1.3 [0-5] | 1.9 ±1.3 [0-5] |
| Loss of Consciousness, average SD [range] | 1.2 ±0.2 [0-2] | 1.3 ±0.5 [0-2] | 1.1 ±0.3 [0-2] | 1.2 ±0.4 [0-2] |
| "Bell Ringer" / "Ding", average SD [range] | 2.2 ±2.0 [0-10] | 2.3 ±2.4 [0-10] | 2.3 ±1.9 [0-10] | 2.2 ±1.9 [0-10] |
| Total, average SD [range] | 4.0 ±2.6 [0-11] | 4.1 ±2.8 [0-11] | 3.8 ±2.5 [0-11] | 4.0 ±2.5 [0-11] |
| **Symptoms previous two years** | | | | |
| Headaches, average SD [range] | 1.9 ±1.4 [0-6] | 2.4 ±1.8 [0-6] | 1.6 ±0.6 [0-3] | 1.9 ±1.4 [0-6] |
| Dizziness, average SD [range] | 2.0 ±1.5 [0-6] | 1.5 ±0.7 [0-3] | 2.2 ±1.6 [1-6] | 2.0 ±1.5 [0-6] |
| Memory Difficulties, average SD [range] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] |
| Visual Disturbances, average SD [range] | 1.3 ±0.4 [0-2] | 1.0 ±0.0 [0-1] | 1.3 ±0.5 [0-3] | 1.3 ±0.4 [0-2] |
| Hearing Problems, average SD [range] | 1.4 ±0.8 [0-3] | 1.7 ±1.0 [0-3] | 1.0 ±0.0 [0-1] | 1.4 ±0.8 [0-3] |
| Other Symptoms, average SD [range] | 1.8 ±1.2 [0-4] | 1.8 ±1.2 [0-4] | 1.8 ±1.5 [0-4] | 1.8 ±1.2 [0-4] |
| **Concussive history in previous years** | | | | |
| Concussion, average SD [range] | 2.8 ±2.6 [0-10] | 3.0 ±2.8 [0-10] | 2.5 ±2.6 [0-10] | 2.9 ±2.6 [0-10] |
| Loss of Consciousness | 1.8 ±1.5 [0-6] | 1.8 ±1.4 [0-6] | 1.8 ±1.7 [0-6] | 1.7 ±1.5 [0-6] |
| "Bell Ringer" / "Ding", average SD [range] | 2.4 ±2.3 [0-10] | 1.9 ±1.1 [0-4] | 2.2 ±2.1 [0-10] | 2.5 ±2.4 [0-10] |
| Total, average SD [range] | 5.0 ±4.6 [0-19] | 4.5 ±4.3 [0-19] | 4.5 ±4.7 [0-19] | 5.2 ±4.6 [0-19] |
| **Return to play** | | | | |
| Seen by Dr, n (% of total) | 16 (7.5) | 0 - | 3 (1.4) | 12 (5.6) |
| Stand down (3 weeks) completed, n (% of total) | 11 (5.2) | 0 - | 2 (0.9) | 8 (3.8) |
| Medical clearance to return to play, n (% of total) | 6 (2.8) | 0 - | 2 (0.9) | 4 (1.9) |
| **Post-Concussion Symptoms Scale** | | | | |
| Score, average SD [range] | 7.7 ±6.1 [0-22] | 6.5 ±4.8 [0-22] | 8.4 ±6.9 [0-22] | 7.7 ±6.1 [0-22] |
| Severity, average SD [range] | 10.9 ±17.1 [0-61] | 8.6 ±12.1 [0-57] | 12.0 ±19.5 [0-58] | 10.9 ±17.1 [0-61] |

Despite 39.0% of players reporting that they had not had a concussion in the previous two years they did report that they had loss of consciousness and "bell ringer" / "dings" from match participation (see Table 12). Players reporting three or more concussions (n=31) also reported the highest mean number of headaches (2.0 ±1.0), dizziness (2.9 ±1.9) and previous concussions (6.5 ±4.1) than players reporting fewer concussions in the previous two years.

**Table 12:** Demographics of amateur rugby league participants reporting none, one, two, 3 or more concussions in the previous two years by age, playing experience, concussive history and associated symptoms, concussive history for prior years, return to play requirements and current post-concussion symptoms scores and severity by means, standard deviations and ranges reported.

| | Previous 2 yr history | | | |
|---|---|---|---|---|
| | No Concussions (n=83) | 1 concussion (n=66) | 2 concussions (n=33) | 3+ concussions (n=31) |
| **Demographics** | | | | |
| Age, average SD [range] | 18.7 ±4.5 [13-34] | 19.7 ±4.6 [14-32] | 19.2 ±4.5 [14-32] | 19.5 ±3.8 [14-28] |
| Experience, average SD [range] | 4.6 ±2.8 [1-10] | 4.2 ±2.8 [1-12] | 4.0 ±2.9 [1-10] | 3.7 ±2.2 [1-8] |
| **History in previous two years** | | | | |
| Concussion, average SD [range] | 0.0 - | 1.0 ±0.0 | 2.0 ±0.0 | 3.8 ±1.5 [3-5] |
| Loss of Consciousness, average SD [range] | 1.0 ±0.0 [0-1] | 1.1 ±0.4 [0-2] | 1.0 ±0.0 [0-1] | 1.5 ±0.5 [0-2] |
| "Bell Ringer" / "Ding", average SD [range] | 3.6 ±2.9 [0-10] | 1.2 ±0.4 [0-2] | 1.5 ±0.5 [0-2] | 2.6 ±1.4 [0-5] |
| Total, average SD [range] | 3.7 ±3.2 [0-11] | 2.8 ±0.9 [1-4] | 3.3 ±0.9 [2-4] | 7.1 ±2.5 [5-10] |
| **Symptoms previous two years** | | | | |
| Headaches, average SD [range] | 0.0 - | 2.1 ±1.7 [0-6] | 1.3 ±0.5 [0-3] | 2.0 ±1.0 [0-3] |
| Dizziness, average SD [range] | 3.3 ±2.1 [0-6] | 1.3 ±0.4 [0-2] | 1.5 ±0.5 [0-2] | 2.9 ±1.7 [0-5] |
| Memory Difficulties, average SD [range] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] |
| Visual Disturbances, average SD [range] | 1.5 ±0.5 [0-2] | 1.0 ±0.0 [0-1] | 2.0 ±0.0 [0-2] | 1.0 ±0.0 [0-1] |
| Hearing Problems, average SD [range] | 0.0 - | 1.4 ±0.8 [0-3] | 1.0 ±0.0 [0-1] | 0.0 - |
| Other Symptoms, average SD [range] | 0.0 - | 1.6 ±0.4 [0-2] | 4.0 ±0.0 [0-4] | 0.0 - |
| **Previous history of concussive events** | | | | |
| Concussion, average SD [range] | 1.4 ±0.5 [0-2] | 2.1 ±1.2 [0-5] | 2.5 ±1.4 [0-4] | 6.5 ±4.1 [0-10] |
| Loss of Consciousness | 1.0 ±0.0 [0-1] | 1.4 ±1.0 [0-4] | 2.0 ±0.0 [0-2] | 4.0 ±2.1 [0-6] |
| Bell Ringer / Ding, average SD [range] | 4.0 ±3.8 [0-10] | 1.3 ±0.4 [0-2] | 2.6 ±1.9 [0-6] | 2.5 ±0.5 [0-3] |
| Total, average SD [range] | 3.2 ±3.8 [0-13] | 4.0 ±2.0 [0-9] | 5.5 ±2.9 [2-10] | 10.1 ±7.6 [2-19] |
| **Return to play** | | | | |
| Seen by Dr, n (% of total) | 5 (2.3) | 7 (3.3) | 1 (0.5) | 3 (1.4) |
| Stand down (3 weeks) completed, n (% of total) | 3 (1.4) | 5 (2.3) | 1 (0.5) | 2 (2.9) |
| Medical clearance to return to play, n (% of total) | 3 (1.4) | 2 (0.9) | 0 - | 1 (0.5) |
| **Post-Concussion Symptoms Scale** | | | | |
| Score, average SD [range] | 6.5 ±5.5 [0-20] | 6.1 ±3.0 [0-12] | 7.7 ±7.1 [2-20] | 18.5 ±3.6 [0-22] |
| Severity, average SD [range] | 14.4 ±16.7 [0-57] | 7.4 ±5.4 [0-18] | 14.2 ±22.6 [0-61] | 21.4 ±24.7 [0-57] |

There was a correlation observed between concussions reported in the last two years and previous concussions ($r_s$=0.597; $p$<0.001); loss of consciousness and previous loss of consciousness ($r_s$=0.681; $p$<0.001) and "bell-ringer" / "ding" and dizziness ($r_s$=0.622; $p$<0.001) and these were significant (see Table 13). This was similar for previous loss of consciousness and headaches ($r_s$=0.671; $p$<0.001); previous concussions and headaches ($r_s$=0.627; $p$<0.001) and PCSS score and PCSS severity ($r_s$=0.916; $p$<0.001).

The reliability of the self-reported concussion questionnaire yielded a weighted $\kappa$ coefficient with substantial reliability for concussions ($\kappa$=0.797; $p$<0.001), loss of consciousness ($\kappa$=0.816; $p$<0.001) and bell ringer / ding ($\kappa$=0.777; $p$<0.001) in the previous two years. When compared with the players history of previous concussions ($\kappa$=0.864; $p$<0.001) and loss of consciousness ($\kappa$=0.914; $p$<0.001), the results were similar.

**Table 13:** Spearman's correlations between reported history of concussion, loss of consciousness and bell ringers; symptoms experienced post event in the past two seasons; previous history of concussion, loss of consciousness and bell ringers; post-concussion score and severity of amateur rugby league players in New Zealand.

| | Concussion | LOC | "Bell Ringer" | Headaches | Dizziness | Memory | Visual | Hearing | Other | Previous Concussion | Previous LOC | Previous "Bell Ringer" | PCSS Score | PCSS Severity |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Concussion | - | | | | | | | | | | | | | |
| LOC | *0.30*[a] | - | | | | | | | | | | | | |
| "Bell Ringer" | *0.18*[a] | 0.17[b] | - | | | | | | | | | | | |
| Headaches | *0.45*[a] | *0.51*[a] | 0.06 | - | | | | | | | | | | |
| Dizziness | *0.30*[a] | *0.32*[a] | *0.62*[a] | *0.42*[a] | - | | | | | | | | | |
| Memory | *0.28*[a] | *0.34*[a] | *0.20*[a] | *0.26*[a] | *0.18*[a] | - | | | | | | | | |
| Visual | 0.07 | 0.16[b] | *0.20*[a] | *0.35*[a] | *0.22*[a] | *0.36*[a] | - | | | | | | | |
| Hearing | 0.13 | *0.20*[a] | 0.11 | *0.29*[a] | *0.35*[a] | 0.12 | *0.30*[a] | - | | | | | | |
| Other | 0.14[b] | *0.32*[a] | -0.11 | *0.48*[a] | 0.16[b] | 0.14[b] | 0.10 | *0.47*[a] | - | | | | | |
| Previous Concussion | *0.60*[a] | *0.45*[a] | -0.05 | *0.63*[a] | *0.20*[a] | *0.23*[a] | *0.40*[a] | *0.29*[a] | *0.33*[a] | - | | | | |
| Previous LOC | *0.23*[a] | *0.68*[a] | *0.18*[a] | *0.67*[a] | *0.40*[a] | *0.48*[a] | *0.42*[a] | 0.12 | *0.51*[a] | *0.50*[a] | - | | | |
| Previous "Bell Ringer" | *0.31*[a] | *0.25*[a] | 0.04 | *0.31*[a] | *0.31*[a] | 0.13 | *0.41*[a] | *0.24*[a] | 0.15[b] | *0.49*[a] | *0.22*[a] | - | | |
| PCS | 0.14[b] | *0.34*[a] | -0.02 | *0.21*[a] | *0.24*[a] | *0.30*[a] | 0.14[b] | 0.08 | -0.07 | 0.16[b] | *0.21*[a] | *0.30*[a] | - | |
| PCS Severity | 0.07 | *0.22*[a] | -0.03 | 0.06 | 0.12 | *0.31*[a] | 0.15[b] | -0.12 | -0.26 | 0.01 | 0.07 | *0.29*[a] | *0.92*[a] | - |

LOC: Loss of Consciousness; PCS: Post-Concussion Symptoms Scale; Correlation is significant at (a) $p < 0.01$; (b) $p < 0.05$

## Discussion

An average of four concussive events per player for all age groups is concerning. Even more concerning is the finding that despite players reporting having had no concussions in the previous two year period they reported an average of nearly four concussive events. Although the history of concussions reported in this study was undertaken by the use of a self-reporting questionnaire, there is some support that the concussion history reported may be reliable.[304] The questionnaire utilised in this study was identical for all groups as part of their medical assessment in the pre-competition period and the use of a self-reported history of concussion measure was of moderate reliability.[304]

Players across all age groups were administered the same questionnaire in different settings where they were gathered for the respective age group competition meetings. Players who were involved in more than one assessment process (n=20) were removed from the subsequent assessment analysis to ensure there were no duplication of the results. The first assessment process these players recorded were kept as part of the study. These players subsequent assessments did provide the source for the reliability analysis of the self-reported concussion history indicating the weighted $\kappa$ statistics were in the "substantial" agreement range according to the guidelines of Landis and Loch.[357]

By utilising the PCSS for a baseline assessment it was planned to compare any PCSS assessment for any player that was suspected of having a concussion. An unexpected finding upon completing the baseline assessment was that 7% of players were identified as having active concussive symptoms. None of the players identified had seen a medical professional for their symptoms nor reported these symptoms to anyone as they felt this was all part of "playing the game". All of these players were advised to seek further medical assistance and required to provide a full medical clearance before they would be allowed to return to play. One player was subsequently medically cleared two days later by his own medical practitioner as he "had no signs of a concussion". This was despite the player initially reporting he had received a head clash in a match 10 days previously and had symptoms when the baseline assessment was conducted. Unfortunately this player recorded a concussive incident in the following match.[317] Consequently he was advised not to return to any match activities for the rest of the competition, to seek a full medical review and to refrain from training until medically cleared to do so.

A previous study[409] identified that players will not report concussive symptoms because they do not think it is sufficiently serious enough to necessitate reporting. A lack of knowledge of the risks and potential consequences of concussion was reported to be the most common reason for the non-reporting of a possible concussion.[409] It was also identified that more than a third of players failed to report a concussive injury as they did not recognize that they had sustained a concussive injury based on their symptoms.[409] This may have been the case in the current study with 7% of players showing current signs of a concussive event. In a recent study[331] involving amateur rugby league team management's knowledge, recognition and management of concussion it was reported that there was an average of 42% of concussion specific questions being answered correctly. Furthermore, 39% of team management reported that concussion occurred only when there was loss of consciousness, 26% of team management would not remove a

concussed player from the field of play and 20% would let a symptomatic player continue on playing. These findings may be reflective of the concussive history reported with some players reported loss of consciousness but not as concussion. Further research is warranted to explore the concussion awareness of amateur rugby league players.

It is a major concern that only 7.5% of players reported their concussive history identified that they sought medical assistance after having a concussive event. Similarly the finding that only 5.2% of players with concussive events completed a 3 week stand down and even less (2.8%) sought a medical clearance is also concerning. The New Zealand Rugby League has a concussion policy, based on an international consensus agreement,[425] in place for the management of concussive events. This policy provides an annual update for the identification and management of concussion. However, it appears this information is not reaching all members of this sporting community. Additionally, there are no readily available tools for the assessment of concussion on the sideline for the non-medical support person with amateur rugby league teams.

A possible tool that may assist the team medic / trainer is the King-Devick (K-D) test. Originally designed as a saccadic reading test,[486] the K-D test has been utilised with amateur rugby league,[317] martial arts fighters, boxing, basketball and American collegiate football players[202, 203]and has been shown to readily identify concussive events.[202, 203, 317] Additionally the K-D test does not require the operator to be a medical professional and requires less than a minute to administer,[202] unlike the Sports Concussion Assessment Tool which can take approximately 20 minutes to complete away from the sideline environment.[149]

Non-compliance with return-to-play protocols is not unique to amateur rugby league players and has been reported in studies on rugby union,[280, 578] ice hockey[1] and US high school athletes.[394] In a study[280] reporting on compliance with return-to-play regulations in schoolboy and community rugby union it was reported that schoolboy rugby had 100% non-compliance with player return-to-play after a concussive event. The current study is similar to suburban rugby non-compliance (95% vs. 97%) but greater than other studies reporting on high school rugby[578] (77.1%), ice hockey (33%) and US high school sports (16.7%) non-compliance indicates that there are differences in how return-to-play regulations are completed in sporting environments despite international agreement with management of sport-related concussions. Further research is warranted to explore the return to play practices of amateur sports such as rugby union and rugby league.

Players reporting three or more concussions in the previous two years also reported more prior concussions than those players reporting none, one or two concussions. This finding was not unexpected as a prior history of concussion places the brain at risk to repeat injury and symptom exacerbation.[434] As well sports participants with two or more concussions have also been reported to have more neurobehavioral symptoms and impairment on selected neuropsychological tests than players with a history of a single concussion.[90] Recently it has been identified that in some individuals with a history of concussion, there were electroencephalographic and motor changes observed in otherwise healthy individuals with no clinically apparent deficits after their concussion injuries.[43] Unfortunately there are no longitudinal studies completed on the effects of these changes but it was hypothesised that as the individuals age, the changes identified

may manifest into clinically significant functional impairments.[43] Of concern is if the players recorded in this study will have these long term changes and, if those with the higher number of reported concussions have a greater electroencephalographic and motor decline than other players with fewer reported concussions. Further longitudinal research is warranted to monitor players with a history of sport-related concussion.

This study does have limitations. Primarily the study is retrospective in nature by getting the players to document their concussion history. As such, the data obtained is purely reliant upon: (a) The player being honest in recording their individual concussion history; and (b) The player being able to remember the total number of concussive events, associated symptoms and when these occurred. By incorporating the terminology “Ding” and “Bell ringer” into the questionnaire it was hoped that players would be more honest in their replies and this would give a more accurate indication of the actual number of concussive events that occur from participation in rugby league injuries. Despite this, the study was viewed as a beginning in the path towards identification of the number of concussive incidents that occur in amateur rugby league in New Zealand.

## Conclusion

In getting players to record their concussion history as part of a pre-competition medical history it was noted that although some players reported as never had a concussion; they did report that they had previous concussive events. As well only 7.5% of players sought medical assistance after their concussive event and even less reported completing a stand down period or a medical clearance for the concussion. What this may indicate is that there is a lack of knowledge pertaining to the risks and potential consequences of concussions by players and team management. These findings suggest that further educational efforts are warranted targeting both players and team management on the risks associated with, and management of, sport-related concussion.

# Section 3:
# Identification and assessment of concussion

***"If you can find a concussion in any player today tell me, so I can send him back to the other team to learn to harden up"***

Coach of a premier team after losing a game with no witnessed concussions - 2013

# CHAPTER 5:
# CONCUSSIONS IN AMATEUR RUGBY UNION IDENTIFIED WITH THE USE OF A RAPID VISUAL SCREENING TOOL

This chapter comprises the following paper published in the *Journal of Neurological Sciences*

**Reference**

King, D., Brughelli, M., Hume, P.A. & Gissane C. Concussions in amateur rugby union identified with the use of a rapid visual screening tool. *J Neurol Sci* 2013; **236**(1-2): 59-63.

**Author contributions**

D. King 85%, M. Brughelli 5%, P.A. Hume 5%, C. Gissane 5%.

## Overview

**Aim:** To use the King-Devick (K-D) test and Sports Concussion Assessment Tool 2 (SCAT2) in amateur rugby union players to identify witnessed and unrecognised episodes of concussion that occurred from match participation.

**Methods:** A prospective observational cohort study was conducted on a premier club level amateur rugby union team during the 2012 competition in New Zealand. Every player completed a pre-competition questionnaire on concussion history, a baseline Post-Concussion Symptom Scale (PCSS) and two trials of the K-D before they participated in any match activities.

**Results:** For players reporting a concussion in the previous three years there was an average of 4.0 ±2.8 concussions per player. There were 22 concussive incidents recorded over the duration of the competition (46 per 1,000 match hours). Five concussive incidents were witnessed (11 per 1,000 match hours) and 17 unwitnessed concussive incidents were identified with the K-D (37 per 1,000 match hours). Witnessed concussions recorded, on average, a longer K-D on the day of injury (5.5 ±2.4 s) than unwitnessed concussions (4.4 ±0.9 s) when compared with their baseline K-D.

**Discussion:** The K-D was able to identify players that had not shown, or reported, any signs or symptoms of a concussion but who had meaningful head injury. The current rate of concussion identified with the K-D was a ten-fold increase in previously reported concussion injury rates. This makes the K-D suitable for rapid assessment in a limited time frame on the sideline such as a five-minute window to assess and review suspected concussed players in rugby union.

## Introduction

Rugby union is one of the most popular contact team sports in the world with nearly 200 countries affiliated to the International Rugby Board.[55, 303] Rugby is played at the professional, amateur and junior levels by both males and females. [56] Rugby also has a modification of the match rules for the different age groups

and competition types (i.e. international, junior, sevens and masters' competitions) that are competed in.[56] As with any contact or collision sport there is a risk of an injury occurring when playing due to the number of collisions and contacts that are integral to the game.[56] One injury that has received attention in rugby union is concussion or mild traumatic brain injury. Studies reporting on concussion injuries in rugby union have identified that the incidence was relatively low with reports varying from 0.2[119] to 4.3[54] per 1,000 playing hours, and 3.8[397] to 5.7[430] per 1,000 athlete exposures.

A tool that may assist sideline assessment of sport-related concussion, the Sports Concussion Assessment Tool (SCAT), was produced as part of the summary and agreement statement of those attending the Concussion in Sport (CIS) Consensus Group in 2004.[422] The third international conference on CIS in Zurich[425] resulted in the SCAT being amended to the SCAT2. The SCAT2 represents the only new sideline assessment tool published since 2009.[425] The SCAT2 is a longer sideline concussion tool and, although is an improvement over the original SCAT, it requires approximately 20 minutes to complete on the sideline making this more of a training room assessment tool as opposed to a readily available sideline assessment tool.[149]

Another sideline tool, the King-Devick (K-D), has been reported as a rapid assessment tool that can assist in the identification of concussion[203, 317] and is reportedly not affected by post activity fatigue.[203] Originally developed as a reading tool to assess the relationship between poor oculomotor functions and learning disabilities, the K-D uses a series of charts of numbers that progressively become more difficult to read in a flowing manner.[486] Poor oculomotor function has been reported as one of the most robust discriminators for the identification of a mild-traumatic brain injury.[270]

Recently the K-D has been utilised in representative rugby league[317] and identified non-witnessed concussions. Players identified with changes in their baseline K-D were further assessed with the SCAT2 and the identification of a sport-related concussion was confirmed. Although completed over a short duration, the study[317] highlighted the potential to detect subconcussive impacts that may accumulate over a period and can lead to neurological changes.[317] With this in mind the current study undertook to use the K-D sideline test with the SCAT2 in amateur rugby union players over a domestic competition season to see if it could identify witnessed and incidentally identified episodes of concussion that occurred from match participation.

## Methods

A prospective observational cohort study was conducted on a premier club level amateur rugby union team during the 2012 competition in New Zealand. All players were considered amateur as they received no remuneration for participating in rugby union activities and derived their main source of income from other employment activities. The matches were played under the rules and regulations of the New Zealand Rugby Football Union. The Auckland University of Technology Ethics Committee approved all procedures used in this study (AUTEC 12/159) and all players participating in the study gave informed consent prior to participating.

***Previous concussion history***

Prior to the competition commencing all players provided a written record of their concussion history. This included history of concussions in the current and previous playing seasons, number of concussions, residual symptoms from any concussions reported and medical clearance to return-to-play.

***King-Devick (K-D) tool***

The King-Devick (K-D) tool is a saccadic test measuring the speed of rapid-number naming.[486] The K-D utilises three test cards with a series of single-digit numbers that are read aloud from left to right. The test includes one practice (demonstration) card and three test cards that vary in appearance. Players were asked to read the numbers on each card aloud from left to right as quickly as possible without making any mistakes. The time taken for each card was recorded as was the number of reading errors made and this was combined to provide a summary score for the entire test, the K-D score. The entire baseline assessment test required less than two minutes to administer per player. The K-D has been reported to have an inter-class correlation for test-retest reliability of 0.97 (95% CI: 0.90 to 1.0).[202] The K-D tests utilised were v2.2.0 (http://www.kingdevicktest.com) on an IPad2.

***Sport Concussion Assessment Tool (SCAT)***

The SCAT2[425] is a tool developed by combining existing concussion assessment tools.[422] Established as having face validity, the SCAT2 reliability and change scores have not been reported to date.[4] The SCAT2 consists of both subjective and evaluative components consisting of the Post-Concussion Symptom Scale (PCSS), modified Maddock's questions, cognitive assessment and neurological screening. The cognitive assessment consists of a five word immediate (upon hearing the words) and delayed (following concentration tasks) recall assessment, reciting the months of the year in reverse order and repeating single digits in reverse order. The SCAT2 (v1.3; http://www.scat2.org/) assessments were completed on an IPad2.

***Match exposure, concussion and subconcussive definition***

Injury rates, expressed as the number of injuries sustained per 1,000 playing hours, were determined using previously described methods.[323] The calculation of the injury rates was undertaken by the team sports medic who recorded, and reported all injuries that had occurred for every match participated in. Over the duration of the competition, all concussive match injuries were recorded by the team sports medic who was a registered comprehensive nurse with tertiary sports medicine qualifications and accredited in injury prevention, assessment, and management. The definition of a concussion utilised for this study was "*any disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific symptoms and often does not involve loss of consciousness. Concussion should be suspected in the presence of any one or more of the following: (a) Symptoms (such as headache), or (b) Physical signs (such as unsteadiness), or (c) Impaired brain function (e.g. confusion) or (d) Abnormal behaviour.*[425]

***Testing procedure***

Every player named for the premier team completed a pre-competition questionnaire on concussion history, a baseline PCSS and two trials of the K-D before they participated in any match activities. The fastest time of the K-D with no errors was recorded as the baseline score. During matches, players observed to have received a direct blow to the head, were slow to rise from a tackle or collision, or appeared unsteady on their feet following a collision, were assessed on-field. Any signs of delayed answering, incorrect answers to questions, or if the player appeared to be impaired in any way, resulted in the player being removed from the match activity and rested on the sideline. Players with a loss of consciousness were treated for a cervical spine injury and managed accordingly.

Players who reported any sign(s) of a concussion or who were suspected to have incurred a concussion as a result of match participation were removed from the match and assessed with the K-D on the sideline and the SCAT2 post-match. The assessment and management of players identified with a concussion were identical to a previous study.[317]

No player identified as having a suspected concussion was allowed to return to match or training activities until medically cleared. In addition, all players identified by the team medic were required to undertake further assessments and complete the stipulated return to play protocols as required by the International Rugby Board (IRB) (http://www.irbplayerwelfare.com/pdfs/IRB_Concussion_Guidelines_EN.pdf). To ensure that the procedures undertaken in this study was conducive to the IRB guidelines, the team medic studied the IRB concussion guidelines with an NZRU official and completed the IRB online concussion management programmes for both the medical practitioner and healthcare provider present (09_23_36), along with the medical practitioner and healthcare provider not present (09_23_36-1) modules. These were completed online at the IRB player welfare website (http://www.irbplayerwelfare.com/index.php?documentid=module&module=1).

***Modified Repeat High Intensity Endurance test (RHIET)***

The RHIET was completed by all members of the premier rugby team to test whether fatigue had any effect on the results of the K-D test. Players' were required to complete a series of six 70-m sprints in a 20-m grid with each sprint departing on a 30-s cycle. Testing was conducted on an artificial grass turf floor in an inside area and players wore their own running shoes. Players' were instructed to stand on the 0-m cone and on "go" sprint to the 5-m cone, turn, sprint to the 0-m cone, turn, sprint to the 10-m cone, turn, sprint to the 0-m, turn, sprint to the 20-m cone, turn, sprint to the 0-m cone. The timing for each sprint was standardised. A 5-s warning was given; the player assumed the ready position and waited for the countdown. On the command "go" the player commenced the sprint. On completion of the sprint the players were allowed to facilitate passive recovery. Players were provided with verbal feedback to ensure that they were ready for the commencement of the subsequent sprint. The RHIET test scores for time, percentage change and fatigue were calculated as previously identified.[463, 565] Two minutes after completing the exercise, players were asked to rate their perceived exertion using the Borg RPE scale[36] and to complete the K-D on the IPad2.

### *Statistical analyses*

All data collected were entered into a Microsoft Excel spread sheet and analysed with SPSS v19.0.0. Differences in K-D scores from pre-competition (baseline establishment) were calculated, baseline and post-match K-D scores were compared using the Wilcoxon signed-rank test. The relationships of pre-competition K-D scores to the PCSS scores were determined using Spearman rank-correlations. The differences between the RHIET run tests were assessed using the Wilcoxon signed-rank test. Internal consistency reliability for the three test cards vs. total time scores at baseline were measured using Cronbach's alpha (α). Statistical significance was set at $p<0.05$.

## Results

Over the duration of the competition 37 players (mean ±SD age; 22.0 ±4.0 yr.) participated in the premier team. The team competed in 24 games (5 pre-season and 19 competition games) over the period of the study resulting in a match exposure of 478.8 hours.

### *Previous concussion history and baseline assessments*

Significantly more players (81.1%) reported a sport-related concussion in the previous three years than players who did not report a previous concussion ($\chi^2=14.3$; $p<0.001$) (see Table 14). This was similar for lifetime concussions (94.6%) reported ($\chi^2=33.1$; $p<0.001$). There was an average of 4.0 ±2.8 concussions per player for players reporting a concussion in the previous three years. Some players reported they had loss of consciousness and "bell-ringers" while participating in rugby union matches but had not had a concussion. When asked about this, the players reported they did not think that loss of consciousness and "bell-ringer's" were concussions.

**Table 14:** Age, reported concussion history for previous three years, King-Devick scores and post-concussion symptom scores with percentages, median scores and ranges for amateur rugby union players competing in a domestic competition in New Zealand.

| | No previous Concussion (n=7) | Previous Concussion (n=30) | All players (n=37) |
|---|---|---|---|
| Age at baseline, yrs. ±SD | 23.0 ±5.7 | 21.0 ±3.4 | 22.0 ±4.0 |
| **Self-reported concussion history** | | | |
| Number of concussions, mean (range) | 0 | 4.0 (1 to 11) | 4.0 (1 to 11) |
| Stand down period completed (%) | - | 12 (32.4) | 12 (32.4) |
| Stand down period, median (range) days | - | 14 (2 to 21) | 14 (2 to 21) |
| Medical clearances to RTP (%) | - | 10 (27.0) | 10 (27.0) |
| **King-Devick baseline test** | | | |
| Pre-competition test 1, s, median (range) | 50.0 (29.0 to 83.0) | 50.4 (39.8 to 86.7) | 50.0 (29.0 to 86.7) |
| Pre-competition test 2, s, median (range) | 47.3[a] (28.0 to 80.3) | 48.9[a] (39.2 to 73.0) | 47.3[a] (28.0 to 80.3) |
| difference, s, median (range) | -2.6 (-15.7 to 0.0) | -2.8 (-13.7 to -0.6) | -2.6 (-15.7 to 0.0) |
| **Baseline post-concussion symptom scale** | | | |
| Physical, median (range) | 0 (0 to 6) | 0 (0 to 6) | 0 (0 to 6) |
| Cognitive, median (range) | 0 (0 to 3) | 0 (0 to 6) | 0 (0 to 6) |
| Sleep, median (range) | 0 (0 to 2) | 0 (0 to 2) | 0 (0 to 2) |
| Emotional, median (range) | 0 (0 to 2) | 0 (0 to 5) | 0 (0 to 5) |

RTP=return to play; s=seconds; Significant difference (p<0.05) than (**a**)=Pre-competition test 1.

Players reporting a previous concussion also reported an average of 4.4 ±1.8 symptoms that occurred in the preceding three years. There were observable learning effects between the first and second K-D

baseline testing for all players (z=3.82; *p*<0.001); players reporting a previous concussion (z=1.96; *p*=0.025) and players with no previous concussion (z=2.197; *p*=0.014) in the preceding three years (see Table 14). The internal consistency reliability of the three K-D cards was acceptable (α=0.72 for card 1; α=0.78 for card 2; α=0.76 for card 3) between card scores and total time score at baseline. The internal consistency reliability of the PCSS was excellent (α=0.98).

### *Concussions*

There were 22 concussive incidents recorded over the duration of the competition (45.9; 95% CI: 30.3 to 69.8 per 1,000 match hours) (see Table 15). Five concussive incidents were witnessed (10.9; 4.5 to 26.2 per 1,000 match hours) and 17 unwitnessed concussive incidents were identified with the K-D (37.0; 23.0 to 56.9 per 1,000 match hours). There were significantly more unwitnessed than witnessed concussive incidents recorded (RR: 3.4; 1.3 to 9.1; *p*=0.039) over the duration of the study.

**Table 15:** Total, witnessed and identified concussions recorded by player positional group by number of concussions and per 1,000 match hours, by SCAT2 results and King-Devick times for baseline, day on injury, 3, 7, 14 and 21 days post injury.

| | Concussions | | | | | |
|---|---|---|---|---|---|---|
| | Total | | Witnessed | | Unwitnessed | |
| | n=22 | rate *(95% CI)* | n=5 | rate *(95% CI)* | n=17 | rate *(95% CI)* |
| **Player position** | | | | | | |
| Front row | 3 | 32.7 *(10.5 to 101.4)* | 1 | 10.9 *(1.5 to 77.4)* | 2 | 21.8 *(5.5 to 87.1)* |
| Second row | 7 | 114 *(54.6 to 240.0)* | 2 | 32.7 *(8.2 to 130.7)* | 5 | 81.7 *(34.0 to 196.4)* |
| Loose forwards | 2 | 21.8 *(5.5 to 87.1)* | 1 | 10.9 *(1.5 to 77.4)* | 1 | 10.9 *(1.5 to 77.4)* |
| Inside backs | 3 | 49.0 *(15.8 to 152.0)* | 0 | 0.0 - | 3 | 49 *(15.8 to 152.0)* |
| Centre backs | 4 | 65.4 *(24.5 to 174.2)* | 0 | 0.0 - | 4 | 65.4 *(24.5 to 174.2)* |
| Outside backs | 3 | 32.7 *(10.5 to 101.4)* | 1 | 10.9 *(1.5 to 77.4)* | 2 | 21.8 *(5.5 to 87.1)* |
| **SCAT2 domain scores** | | | | | | |
| Symptom (22) | | 14.0 ±6.0 | | 6.8 ±2.6 | | 16.1 ±5.5 |
| Symptom severity (132) | | 16.5 ±16.6 | | 42.4 ±28.7 | | 8.8 ±8.6 |
| Physical signs (2) | | 1.0 ±0.8 | | 0.8 ±0.4 | | 1.0 ±0.9 |
| Glasgow Coma (15) | | 14.9 ±0.2 | | 14.8 ±0.4 | | 15.0 ± 0.0 |
| Balance examination (30) | | 7.0 ±6.63 | | 12.8 ±5.6 | | 5.2 ±4.8 |
| Coordination (1) | | 0.8 ±0.2 | | 0.6 ±0.5 | | 0.8 ±1.3 |
| **Subtotal of 70 score** | | **55.5 ±11.3** | | **40.2 ±4.0** | | **58.9 ±9.1** |
| Orientation (5) | | 4.3 ±1.0 | | 3.2 ±1.1 | | 4.6 ±0.5 |
| Immediate memory (15) | | 13.5 ±1.8 | | 13.0 ±2.5 | | 13.7 ±1.4 |
| Concentration (5) | | 2.4 ±1.4 | | 1.6 ±1.5 | | 2.6 ±1.2 |
| Delayed recall (5) | | 3.8 ±3.8 | | 2.6 ±1.5 | | 4.2 ±1.3 |
| **SAC subtotal of 30** | | **24.1 ±3.8** | | **20.4 ±3.5** | | **25.3 ±2.7** |
| **SCAT2 total of 100** | | **79.6 ±13.5** | | **60.6 ±4.2** | | **84.2 ±9.6** |
| Maddocks score (5) | | 4.5 ±1.3 | | 3.4 ±1.9 | | 4.9 ±0.5 |
| **King-Devick scores; median s (range)** | | | | | | |
| Baseline | | 43.5[bcdf](25.3-69.2) | | 50.4 (41.3-61.5) | | 43.0[bcef](25.3-69.2) |
| Injury | | 48.4[adef](29.0-74.8) | | 58.6(45.7-70.5) | | 47.7[acdef](29.0-74.8) |
| Difference from baseline (range) | | 4.6(3.0-9.0) | | 4.4 (3.0-9.0) | | 4.6 (3.1-6.6) |
| 3 days post | | 50.2[adef](30.2-71.3) | | 56.8(47.8-68.5) | | 47.1[abdef](30.2-71.3) |
| Difference from baseline (range) | | 3.3(0.0-14.2) | | 6.5 (4.3-7.0) | | 2.8 (0.0-14.2) |
| 7 days post | | 48.2[abcef] (26.3-71.2) | | 54.5(44.1-67.8) | | 46.5[bcef](26.3-71.2) |
| Difference from baseline (range) | | 1.2(-1.0-6.3) | | 4.0 (0.3-6.3) | | 1.0 (-1.0-5.7) |
| 14 days post | | 45.5[bcdf] (25.2-68.5 | | 53.6(42.5-62.8) | | 43.7[abcd](25.2-68.5) |
| Difference from baseline (range) | | -0.2(-2.7-3.2) | | 2.7 (1.2-3.2) | | -0.3(-2.7-3.0) |
| 21 days post | | 43.5[abcde](25.0-68.0) | | 51.6 (41.2-61.9) | | 42.8[abcd](25.0-68.0) |
| Difference from baseline (range) | | -0.5(-2.6-1.9) | | 0.4(0.0-1.9) | | -0.6(-2.6-0.0) |

CI = Confidence Interval; s=seconds; Significant difference (*p*<0.05) than (a) = Baseline; (b) = Injury; (c) = 3 days post; (d) = 7 days post; (e) = 14 days post; (f) = 21 days post

### *Post-match SCAT2 and King-Devick scores*

The mean (±SD) SCAT2 score for all concussions recorded was 79.6 ±13.5 (see Table 15). There were no significant differences in witnessed (60.6 ±4.2) and unwitnessed (84.2 ±9.6; z=-0.565; *p*=0.286) concussions. There was no significant correlation between the SCAT2 and the K-D scores ($r_s$=0.190; *p*=0.197). Unwitnessed concussions identified with the K-D recorded on average fewer symptoms (16.1 vs. 6.8; z=-0.560; *p*=0.288), lower symptom severity (8.8 vs. 42.4; z=-1.025; *p*=0.153) better balance examination (5.2 vs. 12.8; z=1.450; *p*=0.074) and better immediate (13.7 vs. 13.0; z=0.309; *p*=0.379) and delayed (4.2 vs. 2.6; z=-0.207; *p*=0.418) memory scores than witnessed concussions, but none of these differences were significant.

Witnessed concussions recorded, on average, a significantly longer K-D on the day of injury (5.5 ±2.4s; z=-2.03; *p*=0.063) than unwitnessed concussions (4.4 ±0.9s; z=-3.73; *p*<0.001) when compared with their baseline K-D (see Table 15). Witnessed concussions recorded a non-significant increase in K-D on the day 3 test (6.2 ±1.1s; z=-2.02; *p*=0.063) when compared with their baseline K-D before the K-D began to return to baseline. Unwitnessed concussions recorded a significant decline in their K-D on the day 3 test (2.7 ±1.5s; z=-3.962; *p*<0.001) when compared with their baseline K-D. Most, but not all, players with an unwitnessed concussive injury had their K-D equal to, but not significantly faster than their baseline K-D by day 14 of the required stand-down period (-0.8 ±1.4s; z=-1.73; *p*=0.084).

### *RHIET and post exercise K-D results*

All non-injured members of the premier team completed the RHIET. No player was tested if they had any signs or symptoms suggestive of a concussion or had a recorded difference in their K-D baseline. The total time run was significantly longer than the predicted time for the RHIET (z=1.95; *p*=0.026) (see Table 16). Differences between baseline (44.9 s; 25.4 s to 69.2 s) and post exercise (43.6 s; 25.3 s to 68.4 s; z=3.39; *p*<0.001) times for the K-D were significant. Players reported a RPE of 16.6 ±3.3 units post exercise. The effects of exercise on the K-D were a lowering of the baseline K-D by a mean of 1.2 s (0.1 s to 3.9 s) post exercise.

**Table 16:** Results of the repeat high intensity endurance test (RHIET) for the effects of exercise on the results of the King-Devick for premier club level amateur rugby union players.

| | **Testing Results** |
|---|---|
| **RHIET** | |
| Total Time, s (range) | 105.9 (93.8-131.7) |
| Predicted time, s (range) | 91.5 (84.4-99.2) |
| Change, % (range) | 4.5 (2.2-6.9) |
| Drop Off, s (range) | 15.5 (8.1-33.5) |
| Fatigue Index, % (range) | 28.9 (13.6-60.3) |
| Sprint Decrement, % (range) | 15.5 (8.1-33.6) |
| **King-Devick** | |
| Baseline, s (range) | 44.9 (25.4-69.2) |
| Post Test 2 min, s (range) | 43.6[a] (25.3-68.4) |
| Difference, s (range) | 1.2 (0.1-3.9) |

Data reported as s = seconds; % = percentage; Significant difference (p<0.05) than (**a**) = Baseline

## Discussion

This study was conducted to assess the K-D in an amateur rugby union environment for the sideline screening of concussion. The K-D has been reported[203] to be a practical sideline screening tool that is quicker than other concussion screening tools such as the Immediate Post-Concussion and Cognitive Testing (ImPACT), Cognitive Status Sport (Cog Sport),[88] the Standardized Assessment of Concussion (SAC)[410] and the Sports Concussion Assessment Tool 2 (SCAT2).[425] Although these screening tools are useful in assessing for suspected concussion they do not assess eye movements or brain stem function well.[271] The K-D does however, test for impairment of eye movement, attention, language and other areas that correlate with sub-optimal brain function that may occur following a concussive episode.[271]

The current findings provide further evidence in support of previous studies[202, 203, 317] on the K-D as a sideline tool designed to complement the many other diagnostic sport-related concussion assessment tools available. The K-D was useful in rapidly confirming a concussion diagnosis in players suspected of having sustained a concussion during a witnessed event by being able to provide instant feedback to the player and to team management. The K-D was able to identify players that had not shown, or reported, any signs or symptoms of a concussion but who had meaningful head injury. Similar to previous studies[202, 203, 317] reporting on the K-D, this study found that players with a clinical diagnosis of concussion had a median increase on the players baseline. The data support previous studies[202, 203, 317] that the K-D is sensitive to neurological changes such as those seen with sports-related concussion.

The finding of five witnessed concussions was not unexpected but the number of unwitnessed concussions is of concern. Previous studies reporting injuries in rugby union have reported a concussion rate of 0.2[119] to 4.3[54] per 1,000 match hours. The current rate (46 per 1,000 match hours) is a ten-fold increase in the previously reported concussion injury rate.[54, 432] If the present finding is any indication that there is a ratio of 3.4 identified concussions for every witnessed concussion occurring then studies will be under-estimating the incidence of concussions. The use of assessment tools such as ImPACT, CogState and AxonSports are primarily used for the establishment of baselines and the identification and management of sport-related concussion.[458] These computer based tools typically take 10 – 20 minutes to complete, must be used within 72 hours of the injury and generally are utilised to manage players with a witnessed concussion,[458] missing the opportunity to capture players with un-observed concussive injury.

An interesting finding was that players who recorded a slower K-D, and were treated as an unwitnessed concussion, had changes in SCAT2 but returned to their baseline result in a shorter period than players with a witnessed concussion. This finding may be reflective of the severity of the neurometabolic changes that are occurring as a result of a concussion occurring.[232] Lengthening of time for a witnessed concussion may also be reflective of the severity of the impact that may have occurred when the concussion occurred. Unfortunately what is not known is whether there was a history of subconcussive impacts that may have been accumulated resulting in the witnessed concussions and whether the players selected had received some subconcussive impacts not severe enough nor numerous enough to have resulted in a witnessed concussion.[566] Further research is warranted to explore the impacts that occur from participation in rugby

union. This research will be beneficial to identify the correlation between witnessed and incidentally identified concussions such as those reported here and the impacts that occur from match participation.

Similar to a previous study,[203] the K-D in this study showed no worsening of scores by exercise with players recording a median improvement of 1.2 s on their baseline post intense exercise session. This finding suggests that the effect of exercise enhances the capacity of these players in regards to reading speed and if there were worsening of the K-D scores then it would most likely be neurological in nature. This makes the K-D suitable for rapid assessment of suspected concussed players in rugby union in a limited time frame on the sideline such as a five-minute window. The K-D has the potential to be utilised by trainers/managers with all contact sports irrespective of the availability of sideline medical personnel.

A limitation to this study was that not every player was tested with the K-D post-match. There were two to three players who would try to avoid being tested post-match but these players were seen at the next training session and completed the K-D. Having a team medic on the sideline as the researcher also meant that other injuries were required to be tended to and some players did leave the changing rooms prior to being tested. To assist in reducing the possibility of a concussive injury remaining unnoticed the coach, manager and team medic identified players who were required to be assessed and these players were told to remain in the changing rooms until tested. Future studies involving the K-D as a sideline assessment tool for concussion may need to have more than one K-D available so that all players can be tested post-match. This study highlighted the relevance of the K-D as a quick and effective tool for sideline assessment of concussive incidence in rugby union at premier club level. The K-D was able to identify players with a suspected concussion and players with a concussion that was not reported or witnessed. Although the K-D was able to identify people at risk of concussive injuries, the current 'gold standard' for the diagnosis of concussion is by a medical physician.[514] The ease-of-use of the K-D made it more acceptable to team management and players, as it provided immediate feedback to the player and coaching staff.

***“I am the coach of this team and I will decide who is concussed and who is not. Just you worry about not upsetting me so you can stay with this team and only assess those people I tell you to assess”***

Senior club coach and club captain - 2007

[Club imposed life ban for removing a concussed player]

Junior (U9) coach - 2015

# CHAPTER 6:

# THE KING-DEVICK TEST WAS USEFUL IN IDENTIFICATION OF CONCUSSION IN AMATEUR RUGBY UNION AND RUGBY LEAGUE IN NEW ZEALAND

This chapter comprises the following paper published by the *Journal of Neurological Sciences*

**Reference**

King, D.A., Gissane, C., Hume, P.A. & Flaws, M. The King-Devick test was useful in management in amateur rugby union and rugby league in New Zealand. *J Neurol Sci* (2015) **351**(1-2): 58-64.

**Author contributions**

D. King 75%, C. Gissane, 10%, P.A. Hume 10%, M Flaws 5%.

## Overview

**Aim:** To use the King-Devick (K-D) test in senior amateur rugby union and rugby league players over a domestic competition season to see if it could identify witnessed and unwitnessed episodes of concussion that occurred from participation in competition matches over three years.

**Methods:** A prospective observational cohort study was conducted on a club level senior amateur rugby union team (n=36 players in 2012 and 35 players in 2013) and a rugby league team (n=33 players in 2014) during competition seasons in New Zealand. All 104 players completed two trials 10 minutes apart of the K-D at the beginning of their competition season. Concussions (witnessed or unwitnessed) were only recorded if they were formally diagnosed by a health practitioner.

**Results:** A total of 52 (8 witnessed; 44 unwitnessed) concussive events were identified over the duration of the study resulting in a concussion injury incidence of 44 (95% CI: 32 to 56) per 1,000 match participation hours. There was a six-fold difference between witnessed and unwitnessed concussions recorded. There were observable learning effects observed between the first and second K-D test baseline testing (50 vs. 45 s; z=-8.81; $p$<0.001). For every 1 point reduction in each of the post-injury SAC component of the SCAT3, there was a corresponding increase (worsening) of K-D test times post-match for changes in orientation (2.9 s), immediate memory (1.8 s) concentration (2.8 s), delayed recall (2.0 s) and Standardized Assessment of Concussion (SAC) total score (1.7 s).

**Discussion:** The rate of undetected concussion was higher when the K-D test was routinely applied to all players post-match than solely relying on the witnessing of potentially concussive episodes during a game. Worsening of the K-D test post-match was associated with reduction in components of the SAC. The appeal of the K-D test is in the rapid, easy manner of its administration and the reliable, objective results it provides to the administrator. The K-D test helped identify cognitive impairment in players without clinically observable symptoms.

## Introduction

The number of sport-related concussions has raised concern in the public, media and clinical arenas in recent years.[588] The incidence of sports-related concussion has increased over the past decade but the actual incidence is likely higher than documented as there is a tendency for sports participants to under-report their symptoms.[263] Concussion has become one of the most troublesome injuries facing the sports medicine professional,[347] especially in regards to early identification of concussive signs and symptoms, and appropriate concussive management facilitation.[409] A sports-related concussion is a unique and individualized injury that can present with a myriad of physical, emotional, somatic, cognitive and sleep-related symptoms and impairments.[423] Due to the nature and variability of concussions, these injuries should have a multifaceted approach in the assessment and management of these injuries.

In the upper levels of sport on-site health professionals are available to assess players on the sideline for the signs and symptoms of concussion. Yet symptoms may not manifest for several hours post event, so many participants may not produce symptoms that meet the clinical criteria for concussion.[581] More recently interest has increased in the impacts to the head that do not result in clinically-observed symptoms associated with concussion.[398] Termed 'subconcussive', these impacts are often not recognised as a concussion, but may result in a rapid acceleration-deceleration of the body or head, moving the brain within the cranium creating a "slosh" phenomenon.[13] The number of impacts that can occur vary, but over time there are repetitive occurrences of these impacts and the cumulative exposure of these may become deleterious.[13] Players not reporting or showing any signs or symptoms of concussion can still have neurophysiological changes.[581]

Following any brain trauma eye function movements may become impaired.[81, 236] In acute traumatic brain injuries there are reported latency and inaccuracy of saccades following the injury.[269] This can remain in people with post-concussion syndrome, where there are a higher number of saccades and poor motor movement timings with longer durations and slower velocities of movement.[270] Poor oculomotor function is one of the most robust discriminators for the identification of,[270] and one of the most widely reported visual problems in,[81, 236] a mild-traumatic brain injury.

Originally developed as a reading tool to assess the relationship between poor oculomotor functions and learning disabilities, the King-Devick (K-D) test utilises a series of charts of numbers that progressively become more difficult to read in a flowing manner.[352] The K-D test requires eye movements, language function and attention in order to perform tasks reflective of suboptimal brain function in hypoxia, [570] extreme sleep deprivation,[120] Parkinson's[370] and concussion.[134, 202, 203, 206, 314, 317, 367, 395] Several sports, such as boxing and mixed martial arts[202, 367], professional ice hockey,[206] representative rugby league[317] and domestic rugby union, have utilised the K-D and identified unwitnessed concussive events. These studies highlight the potential of the K-D test to detect subconcussive impacts that may accumulate over a period and can lead to neurological changes.[317] The K-D test has been recommended as a sideline test to enhance the detection of players with a concussive injury in conjunction with other concussion tests.[142, 395] Recently it was identified that larger scale research over a longer period of time may provide increased validity of the K-D test as part

of a continuum of concussion assessment tools.[395, 603] As such, the purpose of this study was to examine the worth of the K-D test as part of a sideline assessment process of players participating in contact sport over a three year period.

## Methods

### *Experimental approach to the problem*

A prospective observational cohort study was conducted on a club level senior amateur rugby union team (n=36 players in 2012 and 35 players in 2013) and a rugby league team (n=33 players in 2014) during competition seasons in New Zealand. A total of 104 male players participated in the study with a mean age of 23.7 ±5.0 yr. Two players were enrolled for all three years of the study, 17 players were enrolled for two years (2012-2013) while 85 players completed one year of the study. The first year (2012) of data (22 concussive events; 5 witnessed concussions and 17 un-witnessed concussive events) for rugby union players has been previously reported.[314] All players were amateurs receiving no remuneration for participating in match activities. The matches were played under the laws of the respective codes in New Zealand. The Auckland University of Technology Ethics Committee approved all procedures involved in this study (AUTEC 12/156) and all players participating in the study gave informed consent prior to participating.

### *King-Devick (K-D) test in association with Mayo Clinic*

Based on the time to perform rapid number naming, the K-D test takes less than two minutes to administer.[202, 203] The K-D test involved the players reading aloud a series of random single-digit numbers from left to right. The K-D test included one practice (demonstration) card and three test cards varied in format on either a moisture-proof 6x8 inch spiral bound physical test or as an application on a IPad platform. Players were asked to read the numbers from left to right across the card as quickly as they could without making any errors using standardized instructions. The time was kept for each test card, and the K-D summary score for the entire test was based on the cumulative time taken to read all three test cards. The number of errors made in reading the test cards was recorded. Baseline K-D times for all participants were established at either the preseason, or when participants joined the team after the season had commenced. The best time (fastest) of two trials 10 minutes apart without errors became the established baseline K-D test time [202]. When a potentially concussive episode was witnessed, the K-D test was used as a screening tool. The test was administered once using the same instructions, and the time and errors are recorded then compared to the subject's baseline. Worsening of time and/or errors identified on the sideline or post-match K-D test have been associated with concussive injury.[134, 202, 203, 206, 314, 317, 395] K-D test performance has been previously shown to be unaffected in various noise levels and testing environments.[567] The K-D test has been reported to have significant correlations ($p$<0.0001) with the visual motor speed (VMS), reaction time (RT), verbal memory (VEM) and visual memory (VIS) of the Immediate Post-concussion Assessment Cognitive Test (ImPACT®)[588] computerised concussion evaluation system. The K-D test has been reported to have an inter-class correlation for test-retest reliability of 0.96[367] and 0.97.[202] The K-D test utilised was v2.2.0 (http://www.kingdevicktest.com) on an IPad2. The IPad2 version enables the use of the K-D test with two different number sets and these were varied over the duration of the study.

***Sport Concussion Assessment Tool v.3 (SCAT3)***

The SCAT3 is a concussion assessment tool developed by combining existing concussion assessment tools.[255] A modification of the SCAT2, the SCAT3 has removed the use of the composite score. The SCAT3 consists of both subjective, and evaluative components comprising of the Post-Concussion Symptom Scale (PCSS), modified Maddocks[389] questions, cognitive assessment (Standardized Assessment of Concussion (SAC))[405] and neurological screening. The cognitive assessment consists of a five word immediate (upon hearing the words) and delayed (following concentration tasks) recall assessment, reciting the months of the year in reverse order and repeating single digits in reverse order. Balance testing is assessed through the modified Balance Error Scoring System (BESS).[255] Only the BESS double stance and tandem stance tests[286] were completed as baseline and post-incident tests. All players completed the SCAT3 baseline assessment prior to participating in any match activity. All players identified with a witnessed concussive incident, or a low K-D-test score (unwitnessed concussion – later confirmed by a physicians' clinical assessment), were required to complete a post-match SCAT3 assessment. There were no players with a worsening of the post-match K-D test, and no changes in the SCAT3, who were not diagnosed as having a concussion.

***Concussion definition***

Concussions were classified as witnessed (a concussive injury that met the definition of a concussion,[423] that was identified during match activities resulting in removal from match activities and had >3 s for pre to post-match K-D with associated changes pre- to post-match SCAT3, and later confirmed by a physician's clinical assessment) or unwitnessed (changes >3 s for pre to post-match K-D with associated changes pre- to post-match SCAT3, and later confirmed by a physician's clinical assessment). The 3 s threshold for changes in the post-match K-D is identical to previous studies reporting the use of the K-D test.[314, 317] The definition of a concussion utilised for this study was "*any disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific symptoms and often does not involve loss of consciousness. Concussion should be suspected in the presence of any one or more of the following: (a) Symptoms (such as headache), or (b) Physical signs (such as unsteadiness), or (c) Impaired brain function (e.g. confusion) or (d) Abnormal behaviour.*"[423] An 'unwitnessed' concussion was defined for the purpose of this study as "*any disturbance in brain function caused by a direct, or indirect force, to the head that does not result in any immediate observable symptoms, physical signs, impaired brain function or abnormal behaviour but had a delay in the post-match K-D score of >3 s and associated changes in the post-match SCAT3"*.

***Testing procedures***

At baseline (prior to the player participating in any match activity), every player named for the premier teams completed a baseline SCAT3 and two trials 15 mins apart of the K-D test. All players were asked to read aloud the practice (demonstration) card before reading aloud all three test cards. Only the three test cards times were recorded. The fastest time of the K-D with no errors was recorded as the baseline score. Players were asked to additionally complete the K-D test after each match they participated in (post-match) and

again at the end of the competition season (post-season). Players with post-match changes in the K-D test >3 s underwent a further SCAT3 assessment to test for signs of concussion. Players with changes in the SCAT3 from their baseline were referred for further medical evaluation. All witnessed concussions were assessed with the K-D and SCAT3 tests and were referred for further medical evaluation. All concussions (witnessed or unwitnessed) were only recorded if they were formally diagnosed by a health practitioner.

During matches, the team medic (and lead researcher), observed players for any signs of a direct blow to the head, for being slow to rise from a tackle or collision, or for being unsteady on their feet following a collision. If this occurred the players were assessed on-field. If there were any signs of delayed answering, incorrect answers to questions, or if the player appeared to be impaired in any way, the player would be removed from the match activity and rested on the sideline. Players who reported any sign(s) of a concussion, who were suspected to have received a concussion, or who were removed from match participation were assessed with the K-D test on the sideline after a 15 minute rest period; not allowed to return to play on the same day; and, referred for further medical assessment. No player identified with delayed (worsening) post-match K-D times was allowed to return to training or match activities without a full medical clearance. Players with a loss of consciousness were treated for a cervical spine injury and managed accordingly. The identification, assessment and management of players identified with a suspected concussive injury were identical to a previous study.[317] All suspected concussive injuries were evaluated by the players own health professional. All players who were identified with a delay (worsening) of the K-D test from their baseline were assessed by their health professional and were diagnosed as having a concussion underwent the return-to-play K-D test monitoring process. No player that was identified with a delay (worsening) of the K-D test and referred to their health professional were immediately cleared for return to play. All players identified as having a concussion were further evaluated with the K-D test on days 3, 7, 14, and 21 following the injury. No player was allowed to return to full match activities until they were medically cleared and, had returned to their baseline K-D score.

***Statistical analysis***

All data collected were entered into a Microsoft Excel spread sheet and analysed with SPSS v22.0.0. Data are presented as mean (±SD) for player data, concussive injury per 1,000 match hours with 95% confidence interval (95% CI) and median [25$^{th}$ to 75$^{th}$ inter-quartile range] for K-D scores. Differences in K-D scores from pre-competition (baseline establishment) were calculated, baseline and post-match K-D scores were compared using the Wilcoxon signed-rank test by the sporting code and as a combined composite score. Risk-ratio (RR) was calculated[125] for the number of witnessed vs. unwitnessed concussions with 95% CI. The sensitivity and specificity of the K-D test was calculated using a 2-by-2 contingency table with 95% CI by year and for the total study with a Cohen kappa ($\kappa$) with 95% CI to assess for intra-rater concordance.[7, 357] Test-retest reliability was also estimated utilising the intra-class correlation coefficient (ICC), with 95% CI, to examine agreement between first and second baseline test scores and the post-season scores. The post concussive injury return to play test-retest reliability of the K-D test was also estimated utilising the ICC and Pearson correlation coefficient ($r$). Linear regression models ($R^2$) were utilised to examine the

relationship of components of the SCAT3 SAC to post match changes of the K-D test. Regression coefficients were reported as measures of the magnitude of association between post-match K-D times and SAC scores of the SCAT3; coefficients represent the number of seconds associated with a 1 point difference in the SAC score. Normality was assessed with the Shapiro-Wilk test.[213] Statistical significance was set at $p$=0.05.

**Results**

Over the duration of the study there were 43 rugby union (2012-2013) and 19 rugby league (2014) matches resulting in a combined match exposure of 1,187 h (see Table 17). A total of 52 concussive events were identified over the duration of the study resulting in a concussion injury incidence of 44 (95% CI: 32 to 56) per 1,000 match participation hours. There was a six-fold difference (RR: 5.5 [95% CI: 2.6 to 11.6]; $p$<0.0001) between the number of witnessed (n=8) and unwitnessed (n=44) concussions recorded.

There were observable learning effects observed between the first and second K-D test baseline testing (50 vs. 45 s; z=-8.81; $p$<0.001) (see Table 17). The ICC between the first and second baseline tests were 0.89 (2012), 0.89 (2013), 0.94 (2014) and 0.92 (combined). Over the duration of the study the K-D test had sensitivity of 1.00 (95% CI 0.93 to 1.00); specificity of 0.94 (0.84 to 0.99), and kappa of 0.98 (95% CI: 0.94 to 1.00). The learning effects were also obvious with the post-season test when compared with the baseline score for all players (45 vs. 38 s; z=-8.80; $p$<0.001).

**Table 17:** Characteristics of participants by mean (±Standard Deviation) for age and King-Devick test baseline and post- season scores in seconds by median score and 25th to 75th interquartile range.

| | 2012 | 2013 | 2014 | Combined |
|---|---|---|---|---|
| Number of players enrolled | 36 | 35 | 33 | 104 |
| Age, y ±SD | 22.8 ±3.4 | 23.3 ±6.1 | 24.9 ±5.0 | 23.7 ±5.0 |
| Matches played [pre-season; match] (match h.) | 24 [5; 19] (479) | 19 [1; 18] (379) | 19 [2; 17] (329) | 62 [8; 54] (1,187) |
| Concussive incidents (total) [witnessed; unwitnessed] | 22 [5; 17] | 22 [2; 20] | 8 [1; 7] | 52 [8; 44] |
| Concussion incidence per 1,000 match h. (95% CI) | 45.9 (26.7 to 65.1) | 58.1 (33.8 to 82.3) | 24.3 (7.8 to 41.2) | 43.8 (31.9 to 55.7) |
| No with a concussive injury, n=; [re-injury in current season] | 17 [5] | 16 [6] | 6 [2] | 39 [13] |
| Pre-season K-D test 1, s, median [IQR] | 50.3 [45.8 to 58.7] | 48.1 [43.8 to 53.5] | 53.7 [47.5 to 59.0] | 50.1 [44.3 to 55.5] |
| Pre-season K-D test 2, s, median [IQR] | 47.3 [41.0 to 54.2][a] | 43.6 [39.7 to 49.3][a] | 49.7 [44.4 to 54.6][a] | 45.1 [41.1 to 50.6] |
| Difference test 1 vs. test 2, s, median [IQR] | -2.8 [-5.7 to -1.6] | -3.5 [-5.4 to -1.3] | -3.3 [-5.5 to -1.9] | -3.3 [-53.4 to -1.7] |
| Post season K-D test, s, median [IQR] | 36.4 [31.9 to 43.0][b] | 31.2 [26.5 to 38.2][b] | 41.8 [37.7 to 49.3][b] | 37.5 [30.7 to 43.4] |
| Difference baseline vs. post season, s, median [IQR] | -6.0 [-10.0 to -4.0] | -11.3 [-15.2 to -8.6] | -7.0 [-11.7 to -3.3] | -8.5 [-12.0 to -4.9] |
| ICC (95% CI), K-D Baseline 1 vs. baseline 2 | 0.89 (0.78 to 0.94) | 0.89 (0.78 to 0.94) | 0.94 (0.88 to 0.97) | 0.92 (0.88 to 0.94) |
| ICC (95% CI), K-D Baseline vs. post season | 0.93 (0.87 to 0.97) | 0.88 (0.76 to 0.94) | 0.94 (0.84 to 0.96) | 0.91 (0.82 to 0.94) |
| Sensitivity (95% CI) K-D test | 1.00 (0.80.0 to 1.00) | 1.00 (0.84 to 1.00) | 1.00 (0.62 to 1.00) | 1.00 (0.93 to 1.00) |
| Specificity (95% CI) K-D test | 0.94 (0.73 to 0.99) | 0.92 (0.63 to 0.99) | 0.97 (0.80 to 0.99) | 0.94 (0.84 to 1.00) |

K-D = King-Devick test; s = seconds; IQR = [Inter Quartile Range]; ICC: = Intraclass Correlation Coefficient; Significant difference ($p$<0.05) than (a) = test 1 of baseline; (b) = established baseline

Witnessed concussive incidents recorded, on average, a longer K-D post-match test score (6 s; z=-2.52; $p$=0.012) than unwitnessed concussive events (5 s; z=-5.84; $p$<0.001) when compared with their baseline K-D test scores (see Table 18). The mean (±SD) SCAT3 symptom scores for all concussions recorded were 9 ±5 symptoms recorded and SAC total score of 24 ±3 (see Table 18). Unwitnessed concussive incidents had a lower symptom severity (23 vs. 31; z=-1.60; $p$=0.109), lower immediate (13 vs. 14; z=-0.43; $p$=0.671) recall and better delayed (4 v 3; z=-2.06; $p$=0.040) memory scores, had a higher SAC total score (24 vs 23;

z=-0.77; *p*=0.440) and a lower BESS score (13 vs. 18; z=-0.53; p=0.598) and a better coordination score (0.5 vs. 0.3' z=-2.45; *p*=0.014) when compared with witnessed concussive incidents.

Lower scores on the SAC of the SCAT3 were associated with increased (worse) times required to complete the K-D test post-match for players with a concussive injury. The scores were normally distributed with a skewness of 0.58 (SE=0.42), a kurtosis of 1.24 (SE=0.82) and normality (SW=0.95; *p*=0.2025). For every 1 point reduction in each of the post-injury SAC components there was a corresponding increase (worsening) of K-D test times post-match for changes in orientation (2.9 s [95% CI: 2.7 to 3.2]; $R^2$=0.85; p<0.001), immediate memory (1.8 s [1.7 to 2.0]; $R^2$=0.94; *p*<0.001) concentration (2.8 s [2.6 to 3.0]; $R^2$=0.87; *p*<0.001), delayed recall (2.0 s [1.8 to 2.2]; $R^2$=0.93; *p*<0.001) and SAC total score (1.7 s [1.6 to 1.8]; $R^2$=0.95; *p*<0.001) (see Fig 5).

**Table 18:** Changes and differences in K-D scores in seconds by median [25th to 75th inter quartile range] and SCAT3 scores in means (±standard deviation) for witnessed and unwitnessed concussive incidents of over the 2012 to 2014 amateur senior match competition seasons for rugby union (2012-2013) and rugby league (2014).

| | Witnessed concussion | Unwitnessed concussions | Total concussions |
|---|---|---|---|
| Age, y, mean (±SD) | 23 ±3.1 | 22.8 ±4.6 | 22.9 ±4.4 |
| Baseline K-D score, s, median [IQR] | 43.6 [31.1 to 54.3] | 40.6 [34.2 to 48.6] | 41.4 [34.2 to 48.6] |
| Post-match K-D test score, s, median [IQR] | 48.0 [38.8 to 58.6][c] | 45.9 [38.1 to 53.3][c] | 46.5 [38.3 to 53.4][c] |
| K-D change baseline vs. post-match, s, median [IQR] | *6.2 [4.0 to 8.8]*‡ | *4.6 [3.6 to 6.0]*‡ | *4.6 [3.8 to 6.7]*‡ |
| ***SCAT3 domain scores*** | | | |
| **Symptom Evaluation** | | | |
| Score (22), mean (±SD) | 8.6 ±3.7 | 8.6 ±4.8 | 8.6 ±4.6 |
| Severity (132) mean (±SD) | 31.0 ±22.8 | 23.1 ±17.2 | 24.3 ±18.1 |
| **Cognitive Assessment** | | | |
| Orientation (5) mean (±SD) | 4.0 ±0.5 | 4.4 ±0.8 | 4.3 ±0.8 |
| Immediate memory (15) mean (±SD) | 13.5 ±1.2 | 12.8 ±1.8 | 12.9 ±1.7 |
| Concentration (5) mean (±SD) | 2.5 ±1.1 | 2.7 ±1.2 | 2.7 ±1.2 |
| Delayed recall (5) mean (±SD) | 3.0 ±1.3[b] | 3.6 ±1.2[a] | 3.5 ±1.2 |
| **SAC Total (30), mean (±SD)** | **23.0 ±2.1** | **23.5 ±3.1** | **23.5 ±3.0** |
| BESS (20), mean (±SD) | 17.5 ±3.4 | 12.5 ±6.2 | 13.3 ±6.1 |
| Coordination (1), mean (±SD) | 0.3 ±0.5[b] | 0.8 ±0.4[a] | 0.7 ±0.5 |
| Maddocks Score | 4.5 ±0.8 | 4.5 ±1.1 | 4.5 ±1.1 |

K-D = King-Devick test; SD = Standard Deviation; s = seconds; IQR = interquartile range; ‡ = positive numbers for change in K-D score indicates longer (worsening) than baseline score; Significant difference (*p*<0.05) than (a) = witnessed concussion; (b) = unwitnessed concussion' (c) = baseline score

Post-match K-D test scores were longer than the baseline score (4.6 s; z=-6.40; *p*<0.001) for all concussive injuries identified (see Table 19). Post-injury evaluations of the players with a concussive injury were longer than the baseline score on post day 3 (6.3 s; z=-6.33; *p*<0.001) before they began to return to the baseline score. By day 14, most, but not all, unwitnessed concussive injury players had improved their K-D test (-0.1 s; z=-7.14; *p*=0.475) when compared with their pre-injury baseline score. Most, but not all, players with a witnessed concussive injury had returned to, or improved on, their K-D test score (0.8 s; z=-1.99; *p*=0.046) when compared to their pre-injury baseline score by day 21 post injury.

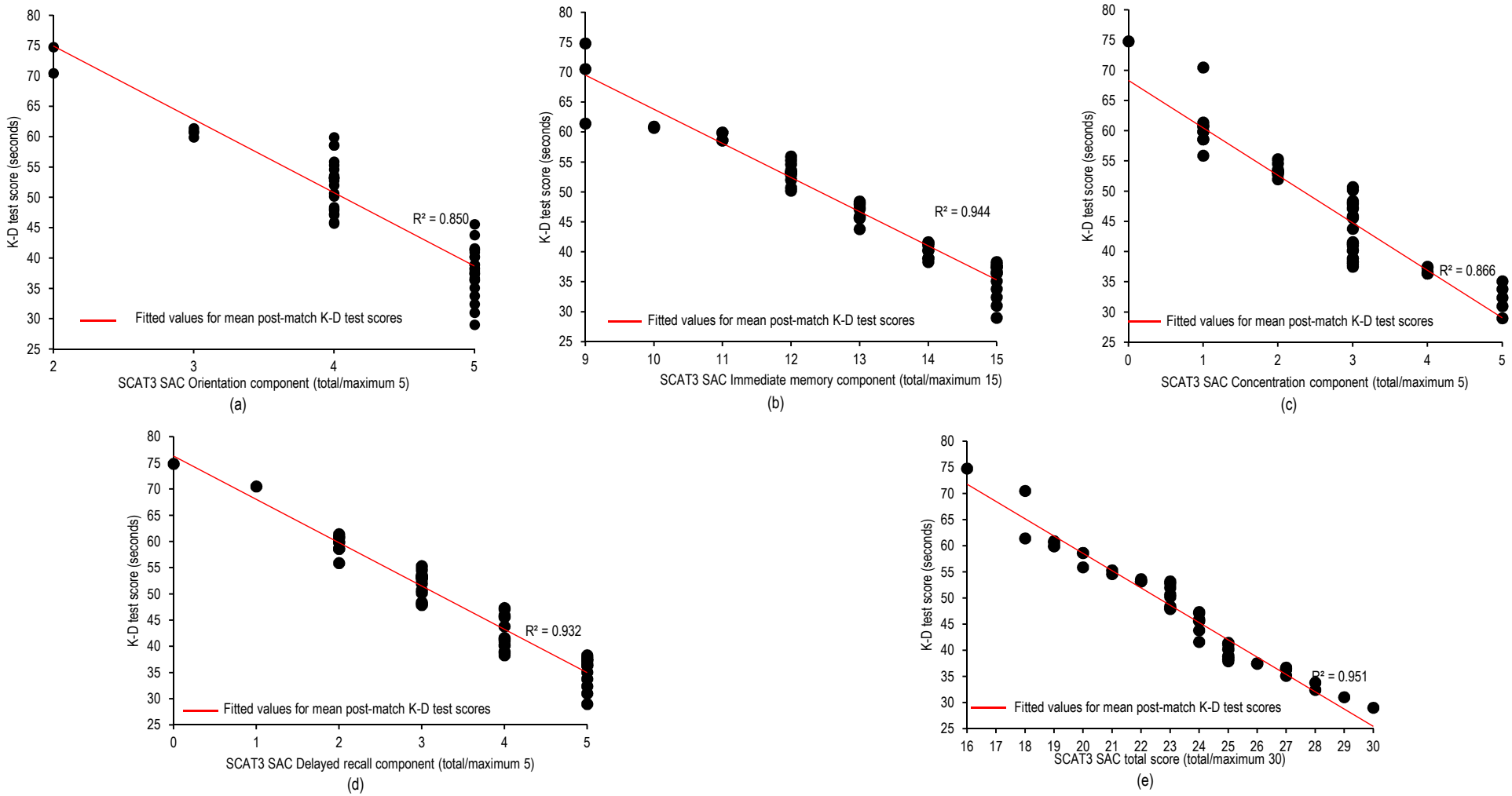


**Figure 5:** Scatter plot and linear regression line showing the relation of post-match King–Devick (K–D) test time scores to Sport Concussion Assessment Tool 3 (SCAT3) Standardized Assessment of Concussion (SAC) components for orientation (a), immediate memory (b), concentration (c), delayed recall (d) and total SAC score (e). The regression line represents fitted values for the post-match K–D time score for each value of SCAT2 SAC total score. Lower (worse) total SAC scores were associated with higher (worse) K–D time scores at baseline; on average, for every 1-point reduction in component score, we found a corresponding increase (worsening) of K–D time.

Table 19: **Post-match K-D test scores in seconds by median [25th to 75th inter quartile range] for witnessed, unwitnessed and total concussions compared with baseline K-D test scores for post-match, Days 3, 7, 14, 21 and 28 post concussive injury and the association between these tests.**

| Testing phase | Witnessed concussions | Association | | Unwitnessed concussions | Association | | Total concussions | Association | |
|---|---|---|---|---|---|---|---|---|---|
| | s, median [IQR] | ICC | *r* | s, median [IQR] | ICC | *r* | s, median [IQR] | ICC | *r* |
| Baseline K-D | 43.6 [31.1 to 54.3][bcdef] | | | 40.6 [34.2 to 48.6][bcdf] | | | 41.4 [34.2 to 48.6][bcdef] | | |
| Post-Match K-D | 48.0 [38.8 to 58.6][aef] | 0.984 | 0.969 | 45.9 [38.1 to 53.3][acef] | 0.986 | 0.974 | 46.5 [38.3 to 53.4][acef] | 0.985 | 0.972 |
| *Difference from baseline* | *6.2 [4.0 to 8.8]‡* | | | *4.6 [3.6 to 6.0]‡* | | | *4.6 [3.8 to 6.7]‡* | | |
| Post day 3 K-D | 51.9 [44.7 to 59.1][aef] | 0.923 | 0.891 | 47.8 [38.6 to 57.6][abdef] | 0.947 | 0.902 | 48.2 [40.0 to 57.5][abdef] | 0.942 | 0.891 |
| *Difference from baseline* | *6.8 [6.4 to 10.1]‡* | | | *6.1 [3.3 to 10.0]‡* | | | *6.3 [4.2 to 10.0]‡* | | |
| Post day 7 K-D | 49.8 [44.7 to 55.6][af] | 0.894 | 0.829 | 46.8 [37.8 to 53.4][acef] | 0.954 | 0.914 | 46.8 [38.6 to 53.8][acef] | 0.944 | 0.893 |
| *Difference from baseline* | *4.3 [3.1 to 11.4]‡* | | | *4.3 [1.7 to 7.3]‡* | | | *4.2 [2.0 to 7.3]‡* | | |
| Post day 14 K-D | 45.5 [36.1 to 57.5][abcf] | 0.994 | 0.992 | 41.0 [34.1 to 48.1][bcdf] | 0.989 | 0.979 | 42.1 [34.7 to 48.4][abcdf] | 0.988 | 0.976 |
| *Difference from baseline* | *3.0 [1.6 to 10.1]‡* | | | *-0.1 [-0.3 to 1.2]‡* | | | *0.0 [-0.2 to 2.6]‡* | | |
| Post day 21 K-D | 43.6 [34.5 to 56.0][abcde] | 0.994 | 0.989 | 40.9 [33.9 to 47.3][abcde] | 0.994 | 0.988 | 41.2 [34.0 to 48.0][abcde] | 0.993 | 0.987 |
| *Difference from baseline* | *0.8 [0.0 to 2.7]‡* | | | *-0.3 [-0.9 to -0.1]* | | | *-0.3 [-0.7 to 0.0]* | | |

K-D = King-Devick test; CI = Confidence Interval; ICC = Intraclass Correlation Coefficient; *r*= Pearson correlation coefficient; s = seconds; IQR = inter-quartile range; Significant difference ($p$<0.05) than (a) = Baseline; (b) = Post-match; (c) = Day 3; (d) = Day 7; (e) = Day 14; (f) = Day 21; ‡ = positive numbers for change in K-D score indicates longer (worsening) than baseline score

## Discussion

This study was conducted to assess the worth of the K-D test in amateur senior rugby union and rugby league in New Zealand. The two sporting codes enrolled in the study (rugby union and rugby league) are similar in terms of the physicality required to play the games, and the nature of the tackling and running required in the sports. However, there are differences in the number of players (rugby union 15 vs. rugby league 13) on the field during the match, and rugby league does not have rucks, mauls and lineouts, and players do not push in the scrum. Despite these differences, often players involved in rugby union have participated in rugby league, or vice versa. Two players enrolled in the study did participate in both sporting codes over the duration of the study.

Rugby union has contestable scrums, lineouts, rucks and mauls whereas rugby league does not undertake any of these activities. Rugby league allows for a set of six tackles to be undertaken for the team in possession of the ball and when this is completed the ball is handed over to the opposition. Rugby union allows for contesting for the ball when the tackle is made through the use of rucks and mauls whereas rugby league requires the tackled player be allowed to get off the ground and play the ball back to the attacking team. Likely as a result of these differences there were observable differences recorded in the K-D tests between rugby union and rugby league. Only two of the players involved in the rugby league cohort had been previously exposed to the K-D test through their rugby union participation.

There was a drop in the number of concussive events identified in the final year of the study and an increase in the median K-D baseline score (see Table 17). The 2014 season data was from a different cohort of players (rugby league) whereas the first two years of data was from a similar cohort (rugby union). The differences between years may be related to the number of players involved in the different sporting codes combined with the different match activities undertaken between rugby union and rugby league.[12, 56]

In this study of amateur senior rugby union and rugby league players, worsening of the K-D test post-match were associated with a reduction in components of the SAC. The changes are likely to be reflective of the anatomical aspects that the K-D and SAC tests capture.[206] Pathways for saccades, or fast eye movements, in the brain are widely distributed and involve several areas of the brain such as the frontal eye fields, supplementary eye field, dorsolateral prefrontal cortex, parietal lobes and deeper structures including the brainstem, necessitating the involvement of several cortical areas in the production, and regulation, of saccades.[206] As saccades can be used to assess cognitive domains such as attention, spatial and temporal orientation and working memory, injuries involving the disruption of the areas involved in saccades production and regulation can result in changes in these cognitive domains.[206] Undertaking the K-D test enables the analysis of numerous circuits throughout the brain involving attention, motivation, visual-spatial integration, motor planning, and spatial organization.[133] The K-D test requires saccades to enable completion of the test and may also reflect concentration and language function.[206] Injuries to any of the areas involved in saccade production and regulation may explain the eye movement and memory related problems that can occur following a concussive injury.[206]

Many concussion assessment tools are still undergoing research and development and these all have the potential for widespread clinical application.[149] Although no one concussion assessment tool can be definitive, the use of a continuum of assessment tools may provide a broader screening process available to the clinician assessing concussions.[255] The K-D test is available in either a moisture-proof 6x8 inch spiral bound physical test or as an application on an IPad platform and this makes the K-D test a readily portable, and easily adaptable, sideline administration tool for the assessment of concussive injuries.[203] The appeal of the K-D test is in the rapid, easy manner of its administration, and the reliable, objective results it provides to the administrator.[588] The K-D test has been shown to be sensitive to subtle changes that can occur with concussive injuries in high school football players without clinically-diagnosed concussion but with functionally-detected cognitive impairment.[581]

Impacts to the head from participation in high school American football have resulted in some players exhibiting no clinically-observed symptoms associated with concussion but demonstrated measurable neurocognitive and neurophysiological impairments.[581] These players had decreased functional magnetic resonance imaging (fMRI) activation levels in areas strongly associated with working memory and significant reductions (score outside 99% confidence interval and flagged by ImPACT® as significantly decreased) in the VMS and/or VIS composite scores of the ImPACT® computerised concussion evaluation system.[581] The VMS component of ImPACT® has been shown to have the most reliable composite scores and is commonly utilised for determining visual-motor deficits.[588] The VMS, and other composite scores (RT and VIS), provide unique information incorporating visual processing, acuity and oculomotor speed and any deficits in these areas may reflect axonal damage to oculomotor neurons.[588] The finding[588] that the K-D test was significantly correlated with changes in the VMS ($r$=-0.696; $p$<0.0001), RT ($r$=0.633; $p$<0.0001), and VIS ($r$=-0.482; $p$<0.0001) components of ImPACT® indicates that the K-D test appears sensitive to revealing visual performance-related effects of concussion in players with no clinically-observed symptoms. The use of the K-D test as part of a continuum for the assessment and monitoring of players with a concussion can assist healthcare providers to evaluate adequate cognitive rest and informed clinical decisions regarding return-to-play and return-to-academic activities.[588]

In the absence of concussion, the K-D test has been shown to have learning effects associated with repeat testing.[203] This is commonly associated with repeated performance measures, or timed testing [203] and can be seen by the median improvement of 3.4 s between the two tests undertaken to establish the individual players baseline score. This is similar to previous studies reporting on the K-D in sports activities with improvements of 1.9 s to 3.1 s[202, 203, 206, 314, 317, 367] for establishing the baseline score of participants. The use of dual baseline assessments allows familiarization of the test and can help alleviate any possible confounding factors[600] such as previous exposure to similar tests. Although employing serial K-D assessments has a learning effect, which is not uncommon in timed test,[202] there should be an equalling of, or an improvement in, the score upon each subsequent test administration.[600] The IPad version of the K-D test (v2.2.0) utilised in our study had two sets of demonstration and test cards with different numbers along the same pattern. The post-match K-D tests were varied weekly to eliminate any possibility of players learning the numbers. Although these were varied, players showed an improvement in the scores from their

baseline. Any worsening of the scores is likely to be a reliable indicator that player should be evaluated further for a possible concussive event.[203]

The procedures utilised for the assessment and management of players identified to have a worsening of their K-D test score are identical to a previous study.[317] The post-match testing was undertaken at least 15 minutes after the game had finished to allow for any of the complex cascade of ionic, neurometabolic, neurochemical and physiologic events to occur.[233] This is reported to occur in the first 10 minutes from a concussive event and then a neuronal depression period occurs where cognitive dysfunction manifests.[233] Previous studies[202, 203, 314, 317, 367] have reported K-D test times of participants with a concussive injury 5 s slower (range 3.2 s to 18.0 s) than their baseline. Our study showed that players with a concussive injury had a similar slower than baseline K-D test score with a median of 5 s for players with a decrease in their post-match assessment, and a median of 6 s for players with a witnessed concussive injury identifying that these players needed further medical evaluation. All players with a worsening of their K-D test score were subsequently diagnosed with a concussion by their medical practitioner and underwent the required return-to-sport evaluations and clearances before they were allowed to participate in training and match activities.

Over the duration of the study there were three false positive cases of players who had slower post-match K-D test scores. None of these players had changes on the post-match SCAT3 and were cleared by their own health practitioner. Upon further investigation it was identified that these players were night-shift workers who had not had a sleep prior to commencing the match activities. It has been previously reported[120] that the K-D test is sensitive to the impact of sleep deprivation on cognitive functioning and this may have been the result of the false-positives detected post-match. Despite the three false-positive cases identified, sensitivity (1.00), specificity (0.94) and kappa (0.98) of the K-D test shows a high accuracy and near perfect agreement[7, 357] for the detection of disruption of the pathways involved in saccades following an injury to these areas.[206]

By using a return-to play monitoring process with the K-D test, the team medic was able to identify players that could commence graduated return-to-play activities while still monitoring the players' recovery. Improvements in the K-D test performance have been shown to be paralleled with improvements of the components of ImPACT® and appear effective in monitoring concussion recovery and symptom resolution. Although no player was allowed to return to full match activities until they equalled or improved their baseline score, several players were medically cleared by their own health practitioner before this occurred. All the witnessed concussed players were medically cleared by day 14 post injury but were withheld from match activities until their K-D scores equalled or bettered their baseline score. For witnessed concussions one player (14%) returned to their baseline score at the 21 days post-match test while the other seven (86%) returned to their baseline at the day 28 post-match test which was similar to a previous study.[314] Most, but not all (n=2; 4%), players with an unwitnessed concussion returned to their baseline K-D scores 14 days after the incident and, when medically cleared, were allowed to return to full training and match activities. This finding supports the need for the management of concussion on an individualized basis.[423]

The use of multiple sideline tools such as the SCAT3 and the BESS examine a wide range of neurologic dysfunction and, while one tool may not identify a concussive injury, other assessment tools may show abnormalities[395] but the 'gold standard for the diagnosis of concussion is by a medical physician.[514] A composite of rapid brief tests such as the K-D test, the SAC and the BESS are likely to provide a series of effective clinical tools to assess players on the sideline with a suspected concussive injury. Further longitudinal studies over different player cohorts will assist in the identification of a continuum approach to sports-related concussion.

### *Limitations*

A limitation to this study was that not every player underwent the K-D test after every match they participated in. There were a few players who would try to avoid being tested post-match but these were assessed at the next training session (2 days later) and were required to complete the K-D test before undertaking any training activities or being named in the team for the ensuing weekend. Having a team medic on the sideline as the researcher also meant that other injuries were required to be tended to and some players did leave the changing rooms prior to being tested. To assist in reducing the possibility of a concussive injury remaining unnoticed the coach, manager and team medic identified players who were required to be assessed and these players were told to remain in the changing rooms until tested. Future studies involving the K-D as a sideline assessment tool for concussion may need to have more than one K-D administrator available so that all players can be tested post-match.

## Conclusion

The K-D and SCAT3 tests helped identify cognitive impairment in players without clinically observable symptoms post-match whether or not they were witnessed as being involved in a potentially concussive incident. The rate of undetected concussion was higher when the K-D test was routinely applied to all players post-match than solely relying on the witnessing of potentially concussive episodes during a game. By undertaking a return-to play monitoring process with the K-D test, the team medic was able to identify players that could commence graduated return-to-play activities while still monitoring the players' recovery. The inclusion of a visual dimension tool such as the K-D test may assist in increasing the capacity for the identification of concussed players and decrease the likelihood of players with a concussive injury not reporting the symptoms and exposing themselves to further concussive events. Using the K-D test as part of a continuum for the assessment and monitoring of players with a concussion can assist healthcare providers to evaluate adequate cognitive rest and informed clinical decisions regarding return-to-play and return-to-academic activities. A composite of rapid brief tests such as the K-D test, the SAC and the BESS are likely to provide a series of effective clinical tools to assess players on the sideline with a suspected concussive injury.

# Section 4:

# Head impact biomechanics in amateur rugby union and rugby league

***"There's not many impacts in the game and of the impacts that do occur these are typically of no real significance"***

International sporting code medical representative - 2011

# CHAPTER 7:

# INSTRUMENTED MOUTHGUARD ACCELERATION ANALYSES FOR HEAD IMPACTS IN AMATEUR RUGBY UNION PLAYERS OVER A SEASON OF MATCHES

This chapter comprises the following paper published in the *American Journal of Sports Medicine*

**Reference**

King, DA., Hume, P., Brughelli, M. and Gissane, C. Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches. *Am J Sports Med,* 2015 **43**(3): 614-624.

**Author contributions**

D. King 80%, P.A. Hume 10%, M. Brughelli 5%, C. Gissane 5%.

## Overview

**Background:** Direct impacts with the head (linear acceleration or pressure) and inertial loading of the head (rotational acceleration or strain) have been postulated as the two major mechanisms of head related injuries such as concussion. Although data are accumulating for soccer and American football there are no published real-time data for non-helmeted collision sports such as rugby union.

**Purpose:** To quantify head impacts via instrumented mouthguard acceleration analyses for rugby union players over a season of matches.

**Study Design:** Cross-sectional study.

**Methods:** Data on impact magnitude and frequency were collected with moulded instrumented mouthguards worn by thirty eight premier amateur senior rugby players participating in the 2013 domestic season of matches.

**Results:** A total of 20,687 impacts >10*g* (range 10.0-164.9*g*) were recorded over the duration of the study. The mean number of impacts per player over the duration of the season of matches was 563 ±618 resulting in a mean of 95 ±133 impacts to the head per player, per match over the duration of the season of matches. The impact magnitudes for linear accelerations were skewed to the lower values ($S_p$=3.7 ±0.02; $p$<0.001) with a mean linear acceleration of 22.2 ±16.2*g*. Rotational accelerations were also skewed to the lower values ($S_p$= 2.0 ±0.02; $p$<0.001) with a mean rotational acceleration of 3,902.9 ±3,948.8 rad/s$^2$.

**Conclusion:** The acceleration magnitudes and number of head impacts in amateur rugby union players over a season of matches measured via instrumented mouthguard accelerations, were higher than for most sports previously reported. Mean linear acceleration measured over the a season of matches was similar to the mean linear accelerations previously reported for youth American football players, high school American football players, and collegiate American football players, but lower than female soccer youths. Mean

rotational acceleration for measured over the a season of matches was similar to for youth American football players, high school American football players, and collegiate American football players, but less than female soccer youths, concussed collegiate American football players and professional American football players.

**Key terms:** injury, linear, rotational, impact, instrumented mouthguard.

**What is already known:**

- High cumulative impacts with a low linear acceleration magnitude of <10*g* may not have any cumulative or long term effects.
- Impacts that do not show any overt clinical symptoms of concussion, loss of match or training time, or concussion related symptoms, are termed subconcussive.
- Subconcussive impacts to the head that occur during football participation may lead to subclinical cognitive decline

**What this study adds:**

- The number of head impacts per player per game for rugby union is higher than that reported for American high school football, American collegiate football and youth ice hockey.
- The majority of the linear and rotational acceleration impacts recorded in senior amateur rugby union fall into the mild category of impact severity.

## Introduction

The potentially severe consequences associated with repetitive sport related concussion have provoked intense discussion in both the academic[28, 423] and public sectors. A concussion typically occurs as a result of a direct impact to the head, or from an indirect impact to the body that is transmitted to the head.[441] When the torso is either decelerated or accelerated rapidly[310] the head sustains a combination of linear and rotational accelerations. Direct impacts with the head (linear acceleration-deceleration) and inertial loading of the head (rotational acceleration-deceleration) have been postulated as the two major mechanisms of head related injuries such as concussion.[310] Linear acceleration produces focal injuries while rotational acceleration produces both focal and diffuse injuries.[310] Repetitive impacts that do not result in an observable, diagnosed or reported concussion i.e. “subconcussive impacts”, may potentially lead to longitudinal deleterious effects such as chronic traumatic encephalopathy.[28, 40, 398] Exposure to repetitive subconcussive impacts show anisotropic and diffusional white matter changes,[25, 398] however, there has been no clear causality identified between repeated concussions or subconcussive impacts and these anatomical changes.[427]

The incidence of concussion in sports provides a unique opportunity to collect biomechanical data on head accelerations involved in mild traumatic brain injury (mTBI).[528] By instrumenting athletes with accelerometer instrumented headbands[457] and helmets,[524] head acceleration data have provided proof of concept and laid the groundwork for future research.[141, 528] By quantifying the head impacts experienced by professional football players, the National Football league (NFL) reconstructed injurious game impacts based on video

analysis.[498] By recreating 31 impacts, 25 of which were concussive, injury risk curves were developed for mTBI's.[528] Nominal values for an injury were a peak linear acceleration of 98*g* and peak rotational acceleration of 6,432 rad/s$^2$.[498] Although this study did characterize concussive biomechanics, the injury risk curves likely over-estimate the risk as head impacts that did not result in concussions were not accounted for.[530] Undertaking reconstructions of real-life concussive events in Australian football and rugby players,[168] the mean peak values recorded for linear acceleration was 103*g* and 8,022 rad/s$^2$ for head angular acceleration. Further, more recent studies[28, 49, 50, 52, 141, 150, 449, 528, 530, 531, 538, 596] have contributed to the ongoing development of biomechanical data related to head impacts but these have utilised different impact measuring data acquisition limits and reporting formats (see Table 20).

Despite the increasing number of studies reporting impacts to the head and concussions, no study, to date, has identified a data acquisition limit that constitutes a subconcussive impact. Impacts under 10*g* have been reported[473] to be a result of activities such as walking, jumping, running and sitting and are considered to be non-contact events.[110, 449] Impacts greater than 10*g* that do not result in a participant presenting with acute signs or symptoms of concussion, are identified as subconcussive impacts[13] and repetitive subconcussive impacts may have negative long-term effects.[566] As shown in Table 20, several studies[28, 29, 49, 50, 52, 531, 596, 626] have utilised data acquisition limits greater than 10*g* and these studies may have removed subconcussive impacts from their dataset. The data obtained from these types of impacts are non-normally distributed. Utilising a data acquisition limit greater than 10*g* may remove a large amount of impacts that could be subconcussive. The data acquisition limit of 10*g* was therefore selected by us to identify impacts that were considered to have occurred from impact accelerations enabling elimination of activities undertaken in daily living.[110]

Non-helmeted sports have also utilised accelerometers in measuring impacts from sports participation. Heading the ball by female soccer youths resulted in peak accelerations of 63*g* and 8,869 rad/s$^2$.[261] No concussions were reported,[261] as no injury nor injury risk was assessed, even though some of the rotational accelerations were within the nominal values for an injury to the head when compared with NFL data[498] and injury risk tolerance levels.[632] Although data are accumulating for soccer and NFL there are no published real-time data for non-helmeted collision sports such as rugby union. The aim of this study was to quantify head impacts via instrumented mouthguard acceleration analyses for amateur rugby union players over a premier season of matches in New Zealand.

**Table 20:** Previous studies reporting head impacts by data acquisition level utilised, linear and rotational values and athlete population studied.

| Author | Data acquisition level | Linear Acceleration (*g*) | | Rotational Acceleration (rad/s²) | | Athletes population (no of participants) |
|---|---|---|---|---|---|---|
| | | Mean (±SD) | 95% CI / SD | Mean (±SD) | 95% CI | |
| Mihalik et al.[451] | 10*g* | 21[a] | 20-223 | 1,418[a] | 1,335-1,506 | Youth (13-14 yr.) Ice Hockey (n=16) |
| Mihalik et al.[452] | 10*g* | 18.4 | 18-19 | 1,466 | 1,449-1,480 | Youth (13-14 yr.) Ice Hockey (n=52) |
| Mihalik et al.[449] | 10*g* | 22 ±2 | - | - | - | Collegiate Football (n=72) |
| Crisco et al.[110] | 10*g* | 21[b] | - | 1,400[b] | - | Collegiate Football (n=314) |
| Crisco et al.[111] | 10*g* | 20[be] | 20-20 | 1,187[be] | 1,166-1,210 | Collegiate Football (n=254) |
| | | 20[bd] | 20-21 | 1,197[bd] | 1,170-1,225 | |
| Hanlon et al.[261] | 10*g* | 19 ±11 | - | 1,717 ±1,300 | - | Girls Youth (u14) Soccer (n=24) |
| Daniel et al.[115] | 10*g* | 18 | - | 901 | - | Youth Football (n=7) |
| Broglio et al.[49] | 15*g* | 25 ±15 | - | 1,627 ±1,183 | - | High School Football (n=78) |
| | | 105 ±18[c] | - | 7,230 ±1,158[c] | - | |
| Broglio et al.[50] | 15*g* | 25 ±16[d] | - | 1,670 ±1,249[d] | - | High School Football (n=35) |
| Broglio et al.[52] | 15*g* | 24 ±15 to 27 ±18[e] | - | 1,506 ±1,262 to 1,633 ±1,150[e] | - | High School Football (n=95) |
| | | 25 ±15 to 29 ±20[d] | - | 1,659 ±1,191 to 1,789 ±1,354[d] | - | |
| Urban et al.[596] | 14.4*g* | 22 ±2[df] | - | 953 ±132[df] | - | High School Football (n=40) |
| Schnebel et al.[538] | 10*g* | 84 ±28[gs] | - | N/S | - | Collegiate & High School Football (n=56) |
| | | 78 ±24[hs] | - | N/S | - | |
| Rowson et al.[531] | 14.4*g* | N/S | - | 1,158 ±972 (6DOF)[q] | - | Collegiate Football (n=335) |
| | | N/S | - | 1,230 ±915 (HIT System)[q] | - | |
| | | N/S | - | 5,022 ±1,791 (HIT System)[c] | - | |
| Rowson et al.[528] | 10*g* | 18 | - | 1,017 | - | Collegiate Football (n=10) |
| Pellman et al.[498] | N/S | 98 ±28[dij] | - | 6,432 ±1,813[dij] | - | American Professional Football (n=31) |
| | | 56 ±22[dgj] | - | 3,983 ±1,402[djk] | - | |
| Duhaime et al.[140] | N/S | 86 ±43[c] | - | 3,620 ±2,166[c] | - | Collegiate Football & Ice Hockey (Male & Female) (n=44) |
| Beckwith et al.[28] | 14.4*g* | 21[l] | 20-55[m] | 848[k] | 799-922[l] | Collegiate & High School Football (n=95) |
| | | 23[c] | 19-27[m] | 874[c] | 723-1,040[l] | |
| Beckwith et al.[29] | 14.4*g* | 103 ±34[c] | - | 3,977 ±2,272[c] | - | Collegiate & High School Football (n=95) |
| Fréchède et al.[168] | N/S | 103[jkn] | - | 8,022[ko] | - | Australian Football and Rugby Union (n=27) |
| Zhang et al.[632] | N/S | 103 ±30[kp] | - | 7,354 ±2,897[kp] | - | American Professional Football (n=24) |
| | | 55 ±21[kq] | - | 4,204 ±1,411[kq] | - | |
| Duma et al.[141] | 10*g* | 32 ±25 | - | 905 ±1,075 (x-axis); 2,020 ±2,042 (y-axis) | - | Collegiate Football (n=38) |
| Brolinson et al.[53] | 10*g* | 21 ±19 | - | N/S | - | Collegiate Football (n=52) |
| Wong et al.[626] | 30*g* | 47 ±14 | - | N/S | - | Pop Warner (12-13 yr.) Football (n=22) |
| Reed et al.[521] | 10*g* | 21 ±0 | - | 1,557 ±27 | - | Youth Ice Hockey (n=13) |

a = non-infraction impacts; b = 50th percentile; c = concussion impact data; d = game impacts; e = practice impacts f = median values reported; g = Collegiate impact data; h = High School impact data; i = struck player; j = striking player; k = impact reconstruction; l = non concussion day impacts; m = 25% - 75% interquartile range; n = peak linear accelerations; o = peak angular accelerations; p = injury impact data; q = non-injury impact data; r = subconcussive impact values; s = top 5% of impacts; N/S = not stated; 6DOF = six degree of freedom measurement; HIT System = Head Impact Telemetry System

## Materials and Methods

A prospective observational cohort study was conducted on a premier club level amateur rugby union team during the 2013 season of matches in New Zealand. All thirty eight male players (mean ±SD age; 22 ±4 y) were amateur receiving no remuneration for participating in rugby union activities. The matches were played under the laws of the New Zealand Rugby Football Union. The researchers' university ethics committee approved all procedures in the study (AUTEC 12/156) and all players gave informed consent prior to participating.

Players were fitted with a moulded instrumented mouthguard (X2Biosystems, Inc., Seattle, WA, USA) sampling at 1,000 Hz, prior to the start of the season. Mouthguards contained a low-power, high *g* tri-axial accelerometer (H3LIS331DL) with 200*g* maximum per axis, and a tri-axial angular rate gyroscope (L3G4200D; ST Microelectronics, Geneva, Switzerland; www.st.com).[63] The mouthguards utilised were similar to those utilised in a previous study.[63] The accelerometer and gyroscope calculated an acceleration and rotational time history of the head's estimated centre of gravity for all impacts that occurred during match participation. The time history incorporated three axes (x, y, z) of acceleration and three axes of velocity. With the player standing upright, these planes described sideways (medio-lateral), forward–back (anterior–posterior) and vertical acceleration and deceleration. The mouthguards[63] have strong correlations for peak linear acceleration (PLA) ($r^2$=0.937), peak rotational velocity (PRV) ($r^2$=0.966), and peak rotational acceleration (PRA) ($r^2$=0.882) when compared with the head's centre of gravity. The moulded instrumented mouthguards were reported[63, 401] to have normalized root-mean-square errors for impact time traces of 9.9 ± 4.4% for linear acceleration, 9.7 ± 7.0% for angular acceleration, and 10.4 ± 9.9% for angular velocity but miss or misclassify ~4% of impacts.[556] The average error offset for impact location was 1.63 ±3.74° azimuth and -1.57 ±0.48° elevation.[401] The mouthguards recorded head linear and rotational acceleration, impact location and duration. All data was recorded on the X2Biosystem Injury Management Software (IMS) and transferred to an Excel spreadsheet for further analysis.

Impacts were identified as any linear acceleration above 10*g* measured at the mouthguard. This data acquisition limit was chosen based on a review of data acquisition limits used in previous studies (see Table 20). These impacts could be a result of a direct blow to the head, face, neck or elsewhere on the body with an 'impulsive' acceleration transmitted to the head. Each recorded impact was categorised into four general locations (front, side, back and top).[109] The direction of impact (azimuth θ) was defined from -180° to 180° with 0° at the x-axis with positive θ on the right side of the players head. The height of impact (elevation α) was defined from 0° (horizontal plane that passes through the head's centre of gravity) to 90° (crown of the head at the 'z' axis). The 'xz' plane represented the midsagittal plane with positive x corresponding to the caudal direction. The 'xy' plane represented the coronal plane with positive 'y' corresponding to the right side of the head. Impacts with α > 65° were defined as top; while impacts with θ of -45° to 45° were defined as back, ±45° to ±135° side and -135° to 135° front direction.

Over the course of the 2013 domestic rugby union season of matches, a total of 20,687 impacts exceeded our study data acquisition limit of 10*g* for a head impact and were retained for data analyses. Impacts <10*g*

of linear acceleration were considered negligible in regards to impact biomechanical features and to eliminate head accelerations from non-impact events such as jumping and running.[473] Their relationships to head trauma make it difficult to distinguish between head impacts and voluntary head movement.[450]

A subconcussive impact was defined as any "*impact that does not result in a concussion diagnosis, does not result in time-loss of participation in practice or games and does not result in concussion related symptoms that linger for a prolonged period of time.*"[31] Over the study two concussion diagnoses were confirmed by medical personnel including the witnessing of the event resulting in the concussion. Impact data obtained from these two confirmed concussions (94.8*g* and 5,319.8 rad/s$^2$ player A and 54.9*g* and 9,935.2 rad/s$^2$ player B) were not included in the dataset utilised for analyses.

The biomechanical measures of head impact severity consisted of impact duration in milliseconds (ms), linear acceleration (*g*), and rotational head acceleration (rad/s$^2$) Resultant linear acceleration is the rate of change in velocity of the estimated centre of gravity of the head attributable to an impact and the associated direction of motion of the head.[450] Resultant rotational acceleration is the rate of change in rotational velocity of the head attributable to an impact, and its direction in a coordinate system with the origin at the estimated centre of gravity of the head.[450] The rotational acceleration was calculated through the IMS utilising a five-point stencil from the rotational velocity measured by the tri-axial angular rate gyroscope (L3G4200D; ST Microelectronics, Geneva, Switzerland; www.st.com).

Head impact exposure including frequency, magnitude and location of impacts was quantified using previously established methods.[109-111] Data were not collected at team trainings as the researchers were most interested in the characteristics of head impacts sustained during rugby union matches in the season of matches. Four measures of impact frequency were computed for each player: *player impacts*, the total and average number of head impacts recorded for a player during all matches; *player position impacts*, the total and average number of head impacts recorded for the playing position for all matches; *player group impacts*, the total and average number of recorded head impacts for the playing group (forwards and backs) for all matches and *impacts per match*, the total and average number of impacts per match for all matches.

For the impact to be recorded, a total of 100 ms of data were stored, including 25 ms prior to, and 75 ms following, the impact. Software provided by X2Biosystems calculated the peak linear acceleration, rotational acceleration (x-axis and y-axis angular accelerations), impact location, HIC,[604] GSI,[201] and date and time stamp for later download and analyses. An example of the impacts recorded for a single player in a single amateur rugby union match can be seen in Figure 6. All matches were videotaped (Sony HDR-PJ540 Camcorder) to enable verification of the impacts recorded.

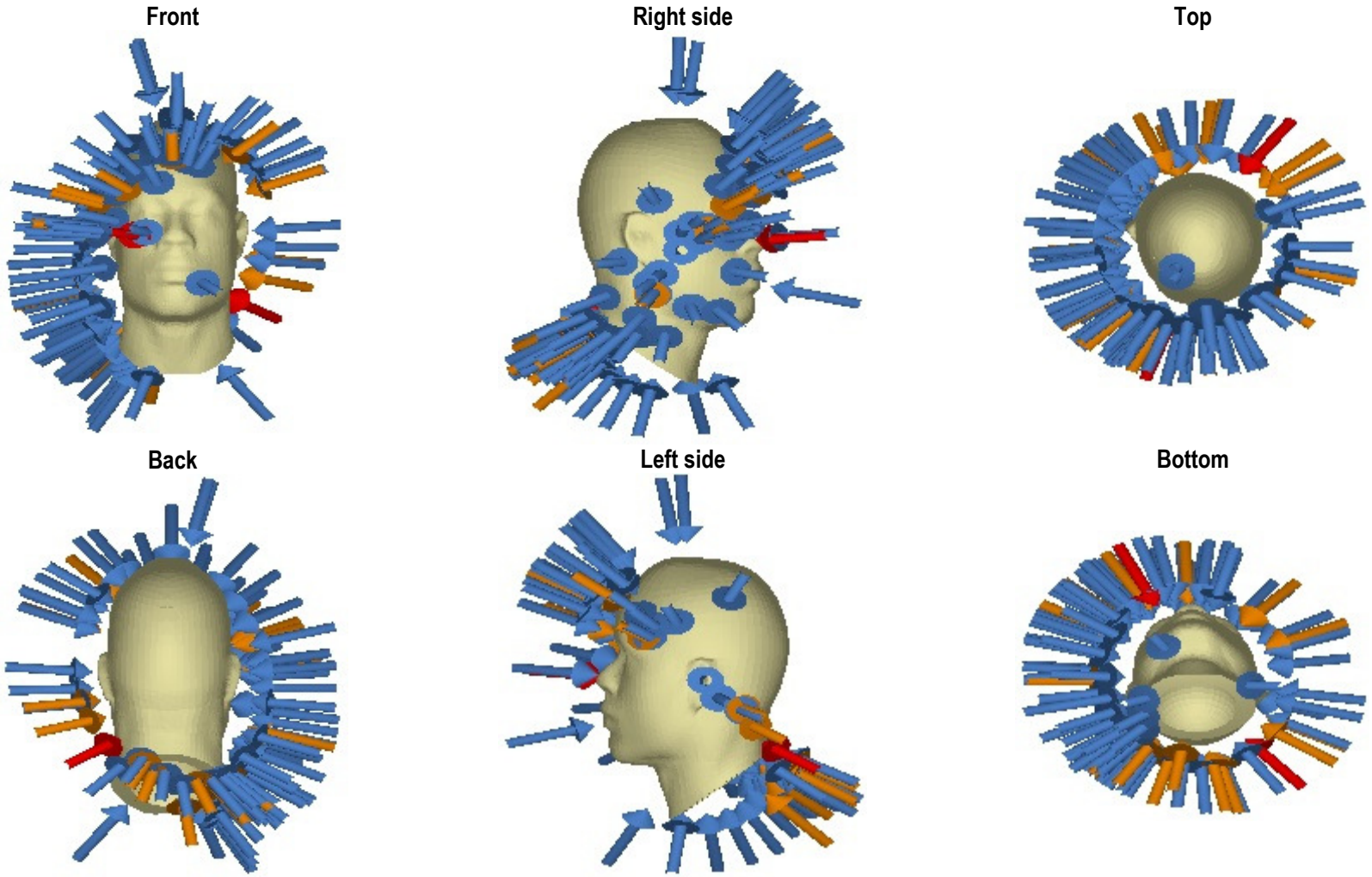


**Figure 6:** Example of the locations of impacts for one monitored player with the X2mouthguard from a single player in a single amateur rugby union match on the Impact Management System (X2Biosystems, Seattle, WA, USA). Blue = 10-30g, orange = 40-60g and red = 70g+.

All impacts recorded were assessed for head movement or biting by the players when the mouthguard was worn. Impacts that were identified as having occurred through these activities were termed 'clacks'. All impacts were assessed through the IMS utilising a "de-clacking algorithm" that involved two methods. The first method utilised various parameters (time above 10*g* data acquisition limit, ratio of PLA to area under curve, filtered/unfiltered PLA ratio, PLA vs. number of points above data acquisition limit) to assess waveform characteristics of the aggregate of the various features of the acceleration waveform to determine an impact vs a 'clack'. The second method utilized a cross-correlation pattern matching (with configurable cross-correlation coefficient) by comparing the impact form to a Gaussian-like reference waveform, looking for a cross-correlation coefficient above a configurable data acquisition limit (0.90 is the default i.e. 90% match). This method assumed a "good" shape for a head impact and matches recorded impacts against this reference waveform (J.Thibado, personnel correspondence, May 1, 2014). All impacts identified as 'clacks' were removed from the dataset prior to downloading for further analysis. All data collected were entered into a Microsoft Excel spreadsheet and analysed with SPSS V.22.0.0. The impact variables were not normally distributed (Sharpo-Wilk test; $p<0.001$). Therefore data were expressed as mean ±standard deviation (SD), as severity measures (95$^{th}$ percentile linear acceleration, 95$^{th}$ percentile rotational acceleration), risk ratios (RR) between player positional groups (forwards vs. backs), and 95% confidence intervals (95% CI).[282, 283] Additionally the cumulative impact burden per match competition, per playing group, per player position per game, per player per match competition and per player per match were analysed using a Krusal-Wallis one-way ANOVA with a Dunn's post-hoc test for all pairwise comparisons. Although there is no accepted method to quantify cumulative impact burden,[52] the sum of linear and rotational accelerations associated with each individual head impact over the course of the study were calculated for all of these parameters.

The impact location variables were computed as azimuth and elevation angles relative to the centre of gravity (CG) of the head centred on the mid-sagittal plane.[108] These were categorized as front (Left: θ = 180° to -135°; Right: θ = 180° to 135°), side (Left: θ =–135° to -45°; Right: θ =135° to 45°), back (Left: θ =-45° to 0°: Right: θ =45° to 0°) and top (Left: θ =180° through negative θ to 0°; Right: θ = 180° through positive θ to 0°) . Impacts to the top of the head were defined as all impacts above an α of 65° from a horizontal plane through the CG of the head.[241] Impact locations were analysed by left and right side impacts using a Friedman repeated measures ANOVA on ranks.

Head impacts were assessed for injury tolerance level for a concussion occurring using previously published injury tolerance levels[49, 52, 254] for linear (>95*g*) and rotational acceleration (>5,500 rad/s$^2$). Head impacts were assessed for impact severity using previously published levels for linear acceleration (mild <66*g*, moderate 66-106*g*, severe >106*g*) and rotational acceleration (mild <4,600 rad/s$^2$, moderate 4,600-7,900 rad/s$^2$, severe >7,900 rad/s$^2$).[264, 478, 632] Both injury tolerance and impact severity levels were analysed using a Friedman repeated measures ANOVA on ranks. Post hoc analysis with Wilcoxon signed-rank tests was conducted with a Bonferroni correction applied. Statistical significance was set at $p<0.05$.

## Results

Thirty eight players participated in 19 matches giving a match exposure of 379 player match hours for the season. A total of 20,687 impacts to the head over 10*g* (range 10.0-164.9*g*) were recorded over the duration of the study (see Table 21). The impact magnitudes for linear accelerations were skewed to the lower values ($S_p$=3.7 ±0.02; $p$<0.001) with a mean linear acceleration of 22.2 ±16.2*g*. Rotational accelerations were also skewed to the lower values ($S_p$= 2.0 ±0.02; $p$<0.001) with a mean rotational acceleration of 3,902.9 ±3,948.8 rad/s$^2$. The mean number of impacts per player over the duration of the season of matches was 563 ±618 resulting in a mean of 95 ±133 impacts to the head per player, per match over the duration of the season of matches. The mean impacts per match were 1,379 ±578 resulting in a mean of 77 ±42 impacts to the head per player position, per match. Forwards recorded more impacts than backs (RR: 10.89; [95% CI: 1.85 to 1.93]; $p$<0.001) over the duration of the season of matches.

The number of head impacts, linear and rotational accelerations and the cumulative impact burden per player varied by player position (see Table 22). In the forwards, the hooker recorded a mean of 112 ±73 impacts per match, the hooker recorded a mean linear acceleration of 27 ±19*g* per match. For rotational accelerations, the open-side flanker recorded a mean of 4,472 ±4,085 rad/s$^2$. The hooker recorded the highest cumulative linear acceleration burden of 56,523*g* and the left hand prop recorded the highest rotational acceleration impact burden of 9,036,995 rad/s$^2$. In the backs, the first five-eight recorded a mean of 117 ±141 impacts per match and the centre recorded a mean linear acceleration of 27 ±19*g* per match. For rotational accelerations, the fullback recorded and mean of 7,106 ±5,807 rad/s$^2$. The first five-eight recorded a cumulative linear acceleration impact burden of 56,713*g* and a cumulative rotational impact burden of 8,763,716 rad/s$^2$.

The number of impacts to area of the head varied over the season of matches (see Table 23). The right side of the head recorded the most impacts. The back left side of the head recorded the highest linear acceleration (27 ±4*g*) for player position, for the forwards and per player. The top right side of the head recorded the highest linear acceleration for the backs (28 ±11*g*). Linear accelerations were higher on the left than the right side of the head for impacts recorded to the front ($\chi^2$=43.68; $p$<0.001) and the back ($\chi^2$=17.83; $p$<0.001) per player. The top right side of the head recorded the highest rotational accelerations (6,160 ±2,908 rad/s$^2$) for player positions and this was similar for backs (7,273 ±3,379 rad/s$^2$) and per players (5,333 ±3,673 rad/s$^2$). Backs recorded higher rotational accelerations than forwards for impacts to the left ($\chi^2$=31.84; $p$<0.0001) and the back left ($\chi^2$=7.53; $p$=0.008) of the head. The top 5%, 2%, 1% and 0.05% of impacts magnitudes recorded for total, player positions groups and by player position can be seen in tables 24 and 25. Although both backs and forwards recorded similar linear accelerations in the top 5% of impacts, backs recorded a higher resultant rotational acceleration than forwards (14,083 rad/s$^2$ vs. 11,023 rad/s$^2$).

**Table 21:** Impacts to the head greater than 10*g* by total and player group in amateur rugby union for total, forwards, backs and per player over a season of matches for linear and rotational acceleration. Data are presented as mean, standard deviation and cumulative scores per player.

| | Impacts | | | | Resultant Linear Acceleration (*g*) | | Resultant Rotational Acceleration (rad/s²) | |
|---|---|---|---|---|---|---|---|---|
| | Number impacts recorded | Average per match competition season Mean ±SD | Average per player Mean ±SD | duration (*ms*) Mean ±SD | Mean ±SD | Total Frequency Impact burden | Mean ±SD | Total Frequency Impact burden |
| Player Position[1] | 20,687 | 1,379 ±578 | 77 ±42 | 12 ±9 | 22 ±16 | 18,145 ±15,037 | 3,990 ±3,949 | 2,724,788 ±2,142,682 |
| Forwards[2] | 13,340 | 1,668 ±385 | 84 ±19 | 11 ±9 | 22 ±16 | 36,741 ±9,470 | 3,620 ±3,605 | 6,038,798 ±2,082,258 |
| Backs[3] | 7,347[a] | 1,050 ±609 | 54 ±30 | 12 ±9 | 23 ±16 | 23,647 ±15,348 | 4,417 ±4,461 | 4,365,606 ±3,005,570 |
| Per player[4] | 20,687 | 564 ±618 | 95 ±133 | 11 ±20 | 22 ±6 | 12,029 ±13,822 | 3,847 ±1,408 | 2,101,028 ±2,702,572 |

Significantly ($p$<0.05) different than (a) = Forwards; 1 = 15 positions; 2 = 8 player positions; 3 = 7 player positions; 4 = 38 players

**Table 22:** Impacts to the head greater than 10*g* by player position in amateur rugby union over a season of matches for linear and rotational acceleration. Data are presented as mean, standard deviation and cumulative impact burden per player.

| | Impacts | | | Resultant Linear Acceleration (g) | | Resultant Rotational Acceleration (rad/s²) | |
|---|---|---|---|---|---|---|---|
| | Total | Average per player per match Mean ±SD | Duration *(ms)* Mean ±SD | Mean ±SD | Total Impact Frequency Burden | Mean ±SD | Total Impact Frequency Burden |
| L-H Prop | 2028 | 101 ±149 | 9 ±7 | 18 ±13 | 35,685 | 4,456 ±4,256 | 9,036,995 |
| Hooker | 2128 | 112 ±73 | 14 ±10 | 27 ±19 | 56,523 | 3,441 ±3,012 | 7,321,483 |
| R-H Prop | 2098 | 100 ±142 | 10 ±8 | 19 ±13 | 39,099 | 3,538 ±3,881 | 7,422,848 |
| Left Lock | 1190 | 63 ±115 | 15 ±10 | 24 ±15 | 28,675 | 2,486 ±1,980 | 2,957,883 |
| Right Lock | 1581 | 79 ±179 | 15 ±10 | 25 ±18 | 39,885 | 2,799 ±2,635 | 4,424,478 |
| B-S Flanker | 1224 | 61 ±54 | 12 ±10 | 24 ±19 | 29,923 | 3,654 ±3,847 | 4,472,473 |
| O-S Flanker | 1688 | 84 ±102 | 10 ±8 | 22 ±17 | 37,757 | 4,472 ±4,085 | 7,576,284 |
| No. Eight | 1403 | 74 ±60 | 9 ±7 | 19 ±13 | 26,381 | 3,634 ±3,629 | 5,097,938 |
| Halfback | 1160 | 61 ±63 | 11 ±8 | 23 ±17 | 26,624 | 7,041 ±5,657 | 8,167,675 |
| First 5/8 | 2330 | 117 ±141 | 13 ±9 | 24 ±16 | 56,713 | 3,761 ±3,547 | 8,763,716 |
| Left Wing | 735 | 39 ±51 | 10 ±7 | 15 ±11 | 10,909 | 2,472 ±3,602 | 1,816,600 |
| Second 5/8 | 602 | 32 ±42 | 15 ±10 | 26 ±17 | 15,732 | 2,919 ±2,480 | 1,757,180 |
| Centre | 599 | 32 ±45 | 16 ±10 | 27 ±19 | 16,320 | 3,188 ±2,785 | 1,909,485 |
| Right Wing | 1130 | 60 ±58 | 8 ±7 | 18 ±12 | 19,742 | 3,906 ±3,676 | 4,413,690 |
| Fullback | 791 | 40 ±53 | 13 ±8 | 25 ±18 | 19,489 | 7,106 ±5,807 | 5,620,898 |

L-H Prop = Loose-head Prop; T-H Pop = Tight-head prop; B-S Flanker = Blind-side Flanker; O-S Flanker = Open-side Flanker

**Table 23:** Impacts to the head for the season of matches by location of total impacts recorded, average linear (*g*) and rotational (rad/s$^2$) acceleration of impacts by player position, forwards, backs and per players in amateur senior rugby union.

| | | | Player Position | | | | Forwards | | | | Backs | | | | Per Player | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Location | Total Impacts | Side | Number | | PLA (*g*) | PRA (rad/s$^2$) | Number | | PLA (*g*) | PRA (rad/s$^2$) | Number | | PLA (*g*) | PRA (rad/s$^2$) | Number | | PLA (*g*) | PRA (rad/s$^2$) |
| | | | n= | Mean ±SD | Mean ±SD | Mean ±SD | n= | Mean ±SD | Mean ±SD | Mean ±SD | n= | Mean ±SD | Mean ±SD | Mean ±SD | n= | Mean ±SD | Mean ±SD | Mean ±SD |
| Front | 5,989 | Right | 3,431 | 229 ±147 | 20 ±7 | 2,781 ±709 | 2,086 | 261 ±122 | 21 ±6 | 2,710 ±419 | 1,345 | 192 ±173 | 19.9 ±6.9 | 2,862 ±1,107 | 3,431 | 107 ±161 | 22 ±8 | 3,092 ±1,026 |
| | | Left | 2,558 | 171 ±82 | 21 ±5 | 3,569 ±1,048 | 1,757 | 220 ±80 | 21[b] ±5 | 3,359[b] ±962 | 801 | 114 ±37 | 21.6 ±5.9 | 3,810 ±1,292 | 2,558 | 85 ±104 | 25[a] ±10 | 4,119[a] ±1,430 |
| Side | 9,232 | Right | 4,791 | 319 ±245 | 21 ±4 | 3,887 ±1,195 | 2,883 | 360 ±154 | 23 ±5 | 3,971 ±1,272 | 1,908 | 273 ±328 | 19.3 ±3.8 | 3,790 ±1,257 | 4,791 | 150 ±177 | 21 ±7 | 3,9947 ±1,617 |
| | | Left | 4,441 | 296 ±116 | 24 ±4 | 4,848 ±2,061 | 2,841 | 355 ±64 | 23 ±4 | 4,001 ±2,043 | 1,600 | 229 ±129 | 24.8[b] ±3.7 | 5,816[a] ±2,539 | 4,441 | 148 ±136 | 22 ±6 | 4,389 ±1,955 |
| Back | 4,927 | Right | 2,027 | 135 ±64 | 23 ±6 | 4,024 ±1,256 | 1,375 | 172 ±59 | 22 ±5 | 3,475 ±962 | 652 | 93 ±40 | 23.4 ±6.7 | 4,650 ±1,478 | 2,027 | 68 ±78 | 23 ±6 | 4,045 ±1699 |
| | | Left | 2,900 | 193 ±132 | 27 ±4 | 4,392 ±1,283 | 2,101 | 263 ±136 | 27 ±4 | 4,026 ±1,176 | 799 | 114 ±74 | 26.4 ±4.1 | 4,810[ab] ±1,544 | 2,900 | 94 ±127 | 27[a] ±6 | 4,686 ±1,899 |
| Top | 539 | Right | 290 | 19 ±18 | 27 ±9 | 6,160 ±2,908 | 150 | 19 ±9 | 26 ±7 | 5,181 ±2,648 | 140 | 20 ±26 | 28.4 ±11.3 | 7,272 ±3,379 | 290 | 13 ±13 | 25 ±18 | 5,333 ±3,673 |
| | | Left | 249 | 17 ±9 | 26 ±6 | 5,864 ±2,627 | 147 | 18 ±7 | 26 ±7 | 5,568 ±2,384 | 102 | 15 ±11 | 25.0 ±3.9 | 6,202 ±3,391 | 249 | 10 ±11 | 22 ±12 | 4,857 ±3,397 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); Significant difference ($p$<0.05) than: (a) = forwards; (b) = left side

**Table 24:** Percentile rankings of post-impacts for linear and rotational accelerations intensity by total impacts recorded and impacts recorded by forwards and backs player positional group for the top 5%, 2%, 1% and 0.05% of impacts in amateur senior rugby union.

| | Total | | Forwards | | Backs | |
|---|---|---|---|---|---|---|
| Percentile | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) |
| 5% | 53 | 12,204 | 53 | 11,023 | 53 | 14,083 |
| 2% | 72 | 16,456 | 73 | 14,715 | 72 | 18,317 |
| 1% | 91 | 18,923 | 92 | 17,454 | 91 | 20,879 |
| 0.5% | 108 | 21,528 | 110 | 19,595 | 104 | 23,266 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$)

**Table 25:** Percentile rankings of post-impacts for linear and rotational accelerations intensity by player position for the top 5%, 2%, 1% and 0.05% of impacts in amateur senior rugby union.

| | L-H Prop | | Hooker | | R-H Prop | | Left Lock | | Right Lock | | B-S Flanker | | O-S Flanker | | No. Eight | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Percentile | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) |
| 5% | 39 | 13,382 | 63 | 9,222 | 41 | 11,123 | 53 | 6,679 | 61 | 7,993 | 62 | 11,416 | 51 | 13,372 | 44 | 11,463 |
| 2% | 53 | 16,265 | 82 | 11,808 | 59 | 15,936 | 75 | 8,749 | 88 | 11,193 | 82 | 15,263 | 80 | 17,474 | 61 | 14,964 |
| 1% | 75 | 18,393 | 101 | 14,301 | 76 | 19,309 | 87 | 10,331 | 99 | 13,952 | 104 | 18,433 | 111 | 18,678 | 70 | 17,739 |
| 0.5% | 96 | 20,173 | 115 | 17,773 | 97 | 23,315 | 107 | 12,717 | 108 | 15,643 | 125 | 22,633 | 124 | 19,794 | 95 | 20,747 |

| | Halfback | | First 5/8 | | Left Wing | | Second 5/8 | | Centre | | Right Wing | | Fullback | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Percentile | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) | PLA (*g*) | PRA (rad/s²) |
| 5% | 56 | 18,725 | 51 | 10,706 | 33 | 9,602 | 59 | 8,129 | 65 | 8,825 | 40 | 11,844 | 58 | 18,273 |
| 2% | 85 | 21,581 | 68 | 15,535 | 48 | 14,409 | 77 | 11,385 | 90 | 11,483 | 56 | 14,960 | 81 | 22,953 |
| 1% | 94 | 24,086 | 90 | 18,766 | 72 | 17,504 | 92 | 12,001 | 102 | 13,014 | 71 | 17,948 | 101 | 24,008 |
| 0.5% | 109 | 25,833 | 110 | 21,588 | 90 | 21,306 | 104 | 14,119 | 110 | 16,252 | 78 | 20,595 | 109 | 26,586 |

L-H Prop = Loose-head Prop; T-H Pop = Tight-head prop; B-S Flanker = Blind-side Flanker; O-S Flanker = Open-side Flanker; PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$)

There were 181 impacts recorded above the linear injury risk limit and 4,452 impacts above the rotational injury risk limit (see Table 26). Forwards recorded more impacts in the moderate rotational acceleration (4,600 to 7,900 rad/s$^2$) range than backs ($p$=0.008). The majority of impacts were in the low impact severity limit for linear (97%) and rotational (73%) data acquisition limits.

**Table 26:** Number of impacts to the head over a season of matches for injury tolerance level,[7, 9, 23, 38] and impact severity limits[264, 478, 632] by resultant linear and rotational accelerations for impacts per player positions, impacts per player group and impacts for all players in amateur senior rugby union by total impacts recorded, percentage of impacts recorded (%), means and standard deviation.

| | Total n= *(%)* | Player Position Mean ±SD | Forwards Mean ±SD | Backs Mean ±SD | Players Mean ±SD |
|---|---|---|---|---|---|
| **Injury Tolerance level** | | | | | |
| **>95*g*** | 181 *(0.9)* | 12 ±8 | 15 ±8 | 8 ±6 | 5 ±8 |
| **>5,500 rad/s$^2$** | 4,452 *(21.5)* | 296 ±164 | 323 ±164 | 265 ±197 | 118 ±183 |
| **Resultant PLA(*g*)** | | | | | |
| **<66*g*** | 20,140 *(97.4)* | 1,343 ±567 | 1,624 ±379 | 1,023 ±598 | 5480 ±602 |
| **66-106*g*** | 436 *(2.1)* | 297 ±17 | 35 ±19 | 22 ±12 | 12 ±18 |
| **>106*g*** | 111 *(0.5)* | 8 ±5 | 10 ±5 | 5 ±4 | 3 ±5 |
| **Resultant PRA (rad/s$^2$)** | | | | | |
| **<4,600 rad/s$^2$** | 15,034 *(72.7)* | 1,003 ±455 | 1,249 ±257 | 721 ±483 | 413 ±474 |
| **4,600-7,900 rad/s$^2$** | 3,020 *(14.6)* | 201 ±101 | 237[a] ±90 | 160 ±104 | 82 ±89 |
| **>7,900 rad/s$^2$** | 2,633 *(12.7)* | 176 ±124 | 183 ±114 | 168 ±144 | 69 ±127 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); Significant difference ($p$<0.05) than (a) = Backs

## Discussion

This study described the magnitude and frequency of head impacts sustained by amateur premier club rugby union players over the season of matches. The players sustained a mean of 95 ±133 impacts per player over the season of matches, with a wide range of impacts recorded depending upon the position on-field. With no other published studies reporting impacts in rugby union utilising similar technology, comparisons are limited to studies utilising headbands and helmet fitted accelerometers reporting impacts in youth ice hockey,[451] girls youth soccer,[261] American youth,[115] high school,[49, 50, 150, 531, 596] collegiate,[28, 52, 141, 449, 528, 531, 538] and professional football.[497, 498]

Gysland[257] identified that players experiencing high cumulative impacts with a low linear acceleration magnitude of 10*g* may not have any cumulative or short term effects. Several authors have reported subconcussive impacts as those that do not result in a diagnosis of concussion.[257, 398, 531, 596] The impact acceleration that is adequate to produce a non-structural brain injury with the neuronal changes of concussion is yet to be established.[21] These neuronal changes may not result in any apparent clinical symptoms of concussion being recognised.[21] Yet, these accelerations may be sufficient to initiate the neurodegenerative cascade that may result in long term neurocognitive complications.[21, 209] The rotational accelerations in our study were calculated through the gyroscope embedded in the mouthguard whereas, in the previous study[531] rotational accelerations were calculated from a linear acceleration vector, the inertial properties of the head, and an mean direction of acceleration.

Players in our study recorded a mean of 77 impacts to the head per game or 1,379 impacts per player, per season of matches which was higher than any other published study to date. American high school football studies had a mean impact per player of 16 to 29 per game or 520 to 652 per season.[52, 538] American collegiate football impacts studies varied from 9 to 13 impacts per session (practice and games combined) or 414 to 1,400 impacts per season (practice and game combined).[109, 141, 538] There are no published studies of impacts at the senior level of sports participation for any sporting code so comparisons are limited given different sports with different age groups.

The mean linear acceleration value of impacts (22*g*) measured over the course of the season of matches was higher than the mean value reported for studies in youth (15*g*),[115] but similar to high school (21-26*g*),[49, 50, 150, 596] some collegiate football (18-27*g*),[28, 528] but lower than female youth soccer (25-63*g*),[261] some American collegiate (32*g*)[141] and professional American football (60*g*)[497] mean values. The mean rotational acceleration for impacts (3,990 rad/s$^2$) measured over the course of the season of matches was higher than American youth (671 rad/s$^2$),[115] and American high school football (973-1,711 rad/s$^2$),[49, 50, 596] American collegiate football (848-1,107 rad/s$^2$),[28, 528] subconcussive American collegiate impacts (1,230 rad/s$^2$),[531] but less than female youth soccer (8,889 rad/s$^2$),[261] concussed American collegiate players (5,022 rad/s$^2$),[531] non-concussed (4,043-4,446 rad/s$^2$)[497] and concussed professional American football reconstructions (4,870-7,173 rad/s$^2$).[497] The differences in the linear and rotational accelerations reported may suggest that higher severity impacts in some, but not all, of the studies[596] when compared with our cohort. It must be noted that although the resultant linear and rotational accelerations reported are lower than female youth soccer,[261] the impacts recorded were from a heading drill and are not reflective of the impacts that may occur during match participation. Although this study identifies the mean linear and rotational accelerations that occur during participation in amateur senior rugby union, further studies are warranted to explore these factors at the junior, female and professional levels of participation.

The distribution of impacts varied by location, player position, player group and by players. Impacts were more commonly recorded on the side of the head (n=9,232; 45%) with the right side (23%) more commonly impacted. The distribution of the impacts recorded is similar to male collegiate ice hockey (30%)[616] but different from American high school and American collegiate football where up to 45% of impacts occurred in the front.[49, 50, 449, 596] Impacts to the top of the head (n=539; 3%) were less commonly recorded. Backs recorded the highest mean linear (28*g*) and rotational (7,273 rad/s$^2$) accelerations on the top-right side. This was similar to high school and collegiate football where impacts to the top of the head varied from 19 to 38*g*.[49, 50, 449, 596] Differences between the impact locations could be related to the different sporting codes and the protective equipment utilised. Rugby union does not employ the padding and helmets utilised in American football, and is played under different rules, such as not leaving ones feet during a tackle (i.e. not taken to ground). An interesting finding was the frequency of impacts identified from below the head around the neck (see Fig. 7). This may be related to the nature of rugby union where players are tackled to the ground then try to retain possession of the ball while other players form a ruck to try to push the player off the ball. This may be similar in the maul situation where the ball carrier is held up and other players join the tackle to try to either retain possession of the ball stopping the opposition from turning the possession over

while the opposition try to turn the player around to regain possession. Similarly, under the rules of rugby union, players engage in the scrum by impacting against each other. As there are eight players per side in the scrum, these impacts are transmitted through the body and can be directly onto the neck. Further research is warranted to better understand the impact pattern that occurs in rugby union. Although the mean linear acceleration was less on the side than the top of the head, side impacts with a higher rotational acceleration component are the most likely impact scenario to result in a concussion.[129] Further research is warranted to compare the different tackling techniques with linear and rotational accelerations to the head.

The mean impact duration over the duration of the study was 12 ms. This is slightly longer than youth[84] and high school[49, 50] American football (8.8 to 10.1 ms) but similar to collegiate[528] American football (14.0 ms). When viewed by player position the mean impact durations varied from 8.0 ms to 16.0 ms which is close to the range of impact durations reported on concussion reconstructions in professional[499] American football (14 to 18 ms). Players in the roles of forwards had shorter mean impact duration than backs (11 ms vs. 12 ms) and lower mean rotational accelerations (3,620 rad/s$^2$ vs. 4,417 rad/s$^2$). The impacts involved in these contacts may be related to the tackle techniques employed, and the subsequent duration and torque, in in rugby union when compared with American football. Further research is recommended to identify the impacts (linear and rotational) involved in the different tackles types (i.e. arm, jersey, shoulder, lift, collision and smother tackles) reported[171] in rugby union.

As previously reported, impacts that do not show any overt clinical symptoms of concussion,[234] loss of match or training time or concussion related symptoms[31, 40, 118] are termed subconcussive. Subconcussive impacts may have a cumulative effect, however little is known about the effects of these impacts.[257] Subconcussive impacts that occur during sports participation may result in some form of sub-clinical decline[25, 40, 118] even when signs and symptoms are absent.[140] It has been identified that football players diagnosed with a concussive injury on the day of participation are closely associated with high kinematic measures.[28] Football players with a delayed diagnosis were moderately associated with high kinematic measures but had an increased number of low kinematic measures.[29] Some concussive events may not present on the day of the impact but may be reported several days after the event with some form of transient alteration in cognition and headache.[140, 387] Possible subconcussive data acquisition limits in terms of rotational acceleration have been reported to average between 1,230[531] and 4,028[498] rad/s$^2$ with concussive events between 4,726[531] and 6,432[498] rad/s$^2$. In relationship to linear acceleration, subconcussive events averaged 26*g*[531] to 57*g*[498] with concussive events averaging 98[531] to 104*g*.[498] Our players recorded a mean of 22*g* and 3,847 rad/s$^2$ and for impacts over the season of matches. The mean rad/s$^2$ recorded was lower than American collegiate[531] and professional American football.[498] Other factors may be involved in the body's response to subconcussive impacts such as match intensity, impact frequency, activity undertaken, ethnicity, age, mass, height, fitness level, hydration, nutrition, concussive history and previous exposure to subconcussive impacts for any clear data acquisition limit to be established. This needs to be investigated further.

The relationships between linear and rotational acceleration and injury tolerance level for impacts have been previously described.[49, 52, 254, 498] The injury tolerance level for impacts are impacts greater than 95*g* and

5,500 rad/s$^2$ and impacts to the front, top or back of helmets,[49, 52, 254, 498] but this is primarily limited to helmeted sports studies. In a numerical reconstruction using a rigid-body model of a video analysis of concussive impacts[168] in Australian football and rugby union from video analysis of concussions, there were mean peak linear and rotational accelerations of 103*g* and 8,022 rad/s$^2$ when a concussion occurred. More recently, in a video analysis of Australian football, rugby league and rugby union, a review of 100 medically-verified concussion cases was undertaken.[492] Utilising a human head finite model reconstruction of 27 concussions and 13 no-injury head impacts it was suggested that rotational kinematics above 4,500 rad/s$^2$ was a tentative tolerance level for loss of conscious to occur.[492] The use of different data acquisition limits in studies makes comparison difficult. We decided to utilise data acquisition limits of 95*g* and 5,500 rad/s$^2$. There were 181 impacts greater than 95*g* and 4,452 impacts greater than 5,500 rad/s$^2$ recorded in our study indicating that there are some impacts above the injury tolerance level for impacts of concussion occurring in rugby union. The number of impacts above these levels varied by player group, player position and players enrolled in the study. This was similar for impacts in the low, moderate and severe injury risk data acquisition limits for both linear and rotational accelerations with the majority of these being recorded in the low injury severity limits previously identified.[264, 478, 632] As there are no previously published studies reporting impacts to the head in rugby union, these levels may not be relevant to non-helmeted sporting codes. The reporting of un-weighted summated impact measures for linear (*g*'s) and rotational (rad/s$^2$) accelerations is an informative way to record the total impact measures over a period, giving some useful information about a possible cumulative impact to the head.[596] However, Urban et al.[596] has identified that this format ignores the nonlinear relationship between peak acceleration level and injury tolerance for concussion and the results presented in this study may provide a misleading picture of the exposure[596] for the participants enrolled. Although the use of reporting of un-weighted summated impact measures is beneficial for undertaking monitoring of these players, and can assist with comparisons between competition formats and training activities, when utilised for inter-study comparisons, it may provide a misleading picture of what is occurring. A format not utilised in conducting our study that may provide a more appropriate comparison is the risk weighted cumulative exposure (RWE) measure.[596] By adjusting the impacts contribution to cumulative exposure according to its associated impact tolerance injury, a RWE for linear, rotational and a combined measure can be established. This enables the identification of the RWE from impacts to the head for individual players, player group and for the sport. The identification of this impact RWE may assist in the identification of players with a potential cumulative exposure concussion. Further studies reporting impacts to the head in rugby union should consider incorporating these measures into the analysis.

Monitoring head impacts in contact sports has been suggested to assist in the identification of high risk events and to alert medical personnel of the need to perform a concussion evaluation of the identified player.[42, 240] Monitoring may reduce the incidence and severity of concussions by reducing subsequent impacts that may cause impact brain injury.[240] The ability to directly measure and record impacts to players in contact sports is also valuable to develop further the body of knowledge of the relationship between head impacts, concussive injuries and the potential cumulative effects of subconcussive impacts.[109] Knowledge can be achieved through monitoring contact sports participants with the use of equipment such as

accelerometer and gyroscope fitted mouthguards. This knowledge will assist in formulating a detailed understanding of the exposure and mechanism of injury for any head related trauma.[42, 109] This may assist in the evaluation of the injury tolerance of concussive type injuries, the future development of interventions to reduce the likelihood of any concussive type injuries and, to establish a broader understanding of the potential role of subconcussive events and long term health.[109] Research with this type of technology is warranted in all contact sporting activities to assist in the development of this knowledge.

***Limitations***

The use of the X2 mouthguard was novel in that they are an All-In-Mouth (AIM) mouthguard incorporating the accelerometer and gyroscope into the structure. The mouthguards were bulky to fit and required some individual customizing but all players were required by the laws of the game to wear these during match participation. The effects of saliva resulted in some of the activation contacts in the mouthguard becoming inactive and the impact data were not able to be downloaded. As well it has been reported that the XGuard can miss or misclassify ~4% of impacts.[556] As a result of this missing or misclassifying impacts, not all impacts that may have occurred have been recorded. Although we recorded the impact data, there are no consistent reliability studies for the XGuard so we don't know how accurate these measure may or may not be and the data reported maybe incomplete and the number of impacts could be more than have been reported.

The AIM mouthguard has a 10% error for linear and rotation acceleration and for angular velocity with an average offset of 2° for azimuth and elevation impact location.[63, 401] The correlation of the AIM mouthguard with laboratory head-forms is good but the impact measurements should be assumed to have some form of error that is dependent on impact conditions and the measure of interest and the variability tested.[27, 140] It is unlikely that the mouthguard was tested under all of the activities seen in rugby union matches such as the rucks, mauls, lineouts and scrum situations and how these correlate to the laboratory conditions results. The large percentage errors reported for the reliability of the impact variables may partially explain why no strong relationships were found between impact variables and concussions. As such, the results presented in this study should be interpreted with some caution.

All games were videotaped to enable verification of the impacts recorded. Unfortunately only those impacts that occurred in the tackle with the player standing were able to be verified as identification of the impacts occurring to the players in rucks and mauls were not able to be completed. The percentage of impacts identified at the 10*g* inclusion limit that were able to be visualised by video review and analysis was 65% to 85% of the total impacts recorded per match. The ball carrier with possession of the ball in a ruck situation is on the ground and other players are attempting to push them off the ball to regain possession. This may involve multiples of players all attempting to move the opposition off the ball, while the team in possession is attempting to retain possession. These collisions resulted in multiples of impacts being recorded by the players involved in the ruck, not just the ball carrier. As a result, multiples of impacts were recorded at the same times. The impacts were not broken down into tackles and non-tackles and this may be of use in future studies on impacts in rugby union. Another limitation was the nature of the scrum during the time of the

study. All forwards were involved in the scrum and they would go through a series of positions before they engaged. If the referee was not happy with the scrum formation they would stop the scrum and get the teams to reform and commence the scrum again. When the teams engage in the scrum, the players all push against each other from a distance enabling a collision situation to occur. Multiples of impacts were recorded.

## Conclusions

Our study was conducted to explore and describe the magnitude and frequency of head impacts sustained by a single amateur senior premier rugby union team. By utilising accelerometer fitted mouthguards in a single premier team throughout the 2013 domestic season of matches there were 20,687 impacts recorded above 10*g*. Although the inter-study comparisons showed that the frequency of impacts recorded were in the low injury impact severity limit (<66g and <4,600 rad/s$^2$), the comparison studies utilised different formats of accelerometer fitted equipment. Mean linear acceleration measured over the season of matches was similar to mean linear accelerations reported in studies for American high school football, some American collegiate football, but lower than female soccer youths Mean rotational accelerations measured were higher than American youth, high school and collegiate football, but lower than female youth soccer. The majority of the linear and rotational acceleration impacts recorded in senior amateur rugby union fall into the mild category of impact severity.

***"It's like this …. you complained about the player who was knocked out and played the next week so we feel that we can't carry your application any further. You won't be getting an appointment to a team ever again"***

Sporting code representative - 2010

# CHAPTER 8:

# THE INFLUENCE OF HEAD IMPACT THRESHOLD FOR REPORTING DATA IN CONTACT AND COLLISION SPORTS: SYSTEMATIC REVIEW AND ORIGINAL DATA ANALYSIS

This chapter comprises the following paper submitted to *Sports Medicine*

**Reference**

King, DA., Hume, P., Gissane, C., Brughelli, M. & Clark T. The influence of head impact threshold for reporting data in contact and collision sports: Systematic review and original data analysis. *Submitted to Sports Medicine.*

**Author contribution:**

D. King 75%, P.A. Hume 10%, C. Gissane 5%, M. Brughelli 5%, T. Clark 5%

## Overview

**Background:** Head impacts and resulting head accelerations cause concussive injuries. There is no standard for reporting head impact data in sports to enable comparison between studies.

**Objective:** To outline methods for reporting head impact acceleration data in sport and the effect of the acceleration thresholds on the number of impacts reported.

**Methods:** A systematic review of accelerometer systems utilised to report head impact data in sport. Calculation of the effect of using different thresholds on a set of impact data from 38 amateur senior rugby players in New Zealand (NZ) over a competition season.

**Results:** Of 52 studies identified, 42% reported impacts using >10*g* threshold. Studies reported descriptive statistics as mean ±standard deviation, median, 25$^{th}$ to 75$^{th}$ interquartile range, and 95$^{th}$ percentile. Application of the different impact thresholds to the NZ data set resulted in 20,687 impacts >10*g*; 11,459 (45% less) impacts >15*g*; and 4,024 (81% less) impacts >30g.

**Discussion:** Linear and angular raw data were most frequently reported. Metrics combining raw data may be more useful, however validity of the metrics has not been adequately addressed for sport. Differing data collection methods and descriptive statistics for reporting head impacts in sports limits inter-study comparisons. Consensus on data analysis methods for sports impact assessment is needed, including impact reporting thresholds. Based on the available data, the 10*g* threshold is the most commonly reported impact threshold and should be reported as the median with 25th and 75$^{th}$ interquartile ranges as the data is non-normal distributed. Validation studies are required to determine the best threshold and metrics for impact acceleration data collection in sport.

**Conclusion:** Until in-field validation studies are completed, it is recommended that head impact data should be reported as median and interquartile ranges using the 10*g* impact threshold.

## Introduction

### *Head impacts cause injury – evidence*

Known as the 'silent injury',[340] and often reported by the media and sporting circles as a 'knock to the head',[305] sport-related concussions (hereafter called 'concussion') are a subset of mild traumatic brain injuries (mTBIs)[423] and have become an increasingly serious concern for all sporting activities worldwide.[100, 123, 416] Research into concussions[315] has increased over the years leading to greater insight into the causes and the effects of these injuries. Research[22, 84, 110, 168, 221, 261, 330, 431, 471, 497, 498, 521, 528, 549, 605, 606, 616, 622, 631, 632] has sought to better determine the head linear and rotational accelerations involved in concussion injuries through the use of telemetry. By adapting radio-telemetry that was utilised for astronauts,[523] the telemetry system has been in use since 1961 for the recording of impacts for football players and concussions[524] that have occurred.

### *A cumulative head impact threshold may be related to concussion*

The immediate and long term effects of multiple and repeated blows to the head that athletes receive in contact sporting environments are a growing concern in clinical practice.[21, 209] Concern has grown about the effects of subconcussive impacts to the head and how these impacts may adversely affect cerebral functions.[21, 209, 257] Subconcussive events are impacts that occur where there is an apparent brain insult with insufficient force to result in the hallmark signs and symptoms of a concussion.[209, 613, 623] Although subconcussive events do not result in observable signs and apparent behavioural alterations,[20, 118] they can cause damage to the central nervous system and have the potential to transfer a high degree of linear and rotational acceleration forces to the brain.[52] Proposed decades previously,[592, 593] exposure to repetitive subconcussive blows to the head may result in similar, if not greater damage to the central nervous system than a single concussive event[613] and may have cumulative effects.[553]

Participants can be exposed to a high number of impacts per season.[257] It has been suggested[566, 581] that brain injuries come from concussive events and also from the accumulation of subconcussive impacts that result in pathophysiological changes in the brain. As subconcussive impacts do not result in observable concussion related signs and symptoms, these are often not medically diagnosed. The accumulation of subconcussive blows can result in neuropsychological changes.[13, 21, 156, 209, 435, 504, 581] However, similar to the literature focused on concussion and mild traumatic brain injury (mTBI), the literature on subconcussive head trauma is limited.[533] What is not known is the number of head impacts, and their intensity, that are required that might result in a concussive injury occurring (i.e. a concussion cumulative threshold). The injury threshold is likely to be different for each person given the multifactorial nature of injuries, similar to other thresholds for injuries to tendons, ligaments, muscle and bone.[268, 547] If a threshold could be determined, then players could be monitored to reduce their potential risk for a subsequent concussive injury – akin to cricket monitoring 'player loading' to the body during bowling events via the number of overs in an attempt to reduce the risk of back stress fractures.[295]

***Impacts can be measured with a number of technologies***

Head impact dynamics have been analysed through the use of video analysis,[431] in game measurements,[84, 110, 141, 330, 466, 521, 528, 531, 538, 616, 631] numerical methods[22, 168, 549, 632] and reconstructions using anthropometric test devices[221, 471, 497, 498, 605, 606, 622] in helmeted sports such as American football[84, 110, 528, 631] and ice hockey[521, 616] and in un-helmeted sports such as soccer[261] and rugby union.[330]

The on-field assessment of head impacts has been captured with a head impact telemetry system (HITS) (Simbex, LLC, Lebanon, NH) using helmet mounted accelerometers enabling determination of the head linear and rotational accelerations in American football,[109, 110, 115, 141, 473, 631] ice hockey[521, 616] and in a headband in youth soccer.[261] The data collected through the HITS has enabled analytical risk functions,[498, 529-531] concussion risk curves,[531] and risk weighted exposure metrics[596] to be developed further assisting in the identification of sports participants at risk of concussive injuries. More recently, instrumented mouthguards known as XGuard (X2biosystems, Inc., Seattle, WA, USA) have documented head impacts in rugby union.[330]

***Thresholds have differed for reporting impact data in contact and collision sports***

Although there is an increasing amount of published literature reporting impact accelerations to the head in the sporting environment, there is less attention focussed on identifying what is a subconcussive impact and where this occurs. Studies[6, 299, 473] have been conducted reporting the impacts absorbed by the head during activities undertaken daily. Although impacts to the head and body under 10*g* have been reported[473], these activities such as walking, jumping, running and sitting are considered to be non-contact events.[110, 449] However, impacts greater than 10*g* occurring from events that do not result in acute signs or symptoms of concussion, are identified as subconcussive impacts.[13]

***To enable comparison of studies, a consistent threshold for reporting is needed***

Head impact data are essential to understand the biomechanics of head injury and to develop potential injury prevention strategies. Researchers have utilised different thresholds with the most common being 9.6*g* and 14.4*g* depending on the accelerometer. The collection of the impact data is based on one accelerometer and the unfiltered / unprocessed data and the value obtained only loosely relates to the final measure being sought. The impact data is processed with a hard exclusion cut-off of 10*g* enabling data collection to become manageable as acceleration lower than 10*g* without impacts occurring becomes more common (personal correspondence S. Broglio 22nd September 2015). There is currently no standard for reporting head impact data to enable comparison between studies. Currently the use of accelerometers may not necessarily provide the meaningful inter-study comparisons that are sought due to data collection, processing and methodologies not being standardized.[495] Studies utilising different impact thresholds have proposed varying conclusions based on the methodological and reporting approaches undertaken.

***Aim of the study***

The rationale for this study is based on questions around the magnitude of a single impact that may result in concussion, the number of impacts needed to result in signs and symptoms of concussion, and individual

player differences that might affect injury tolerance levels for concussion. Given head impacts are likely to cause concussive injury, and the number of head impacts may be related to a potential concussion threshold (i.e. a cumulative threshold), the number of head impacts should be monitored in players. However, given impacts can be measured with a number of technologies (e.g. instrumented behind the ear patches, mouthguards, head gear), and thresholds have differed for reporting impact data in contact and collision sports, a threshold for reporting impact data in sport is needed to enable comparison of studies.

Therefore the aims of this study were to: a) summarise the methods for reporting head impact data in sport to date; and b) assess the impact of different acceleration thresholds on the likely identification of concussive injuries.

***Methods***

To outline methods for reporting head impact data, a systematic review of the literature was conducted. The guideline for reporting observational studies (MOOSE: Meta-analysis Of Observational Studies in Epidemiology)[575] was followed for the empirical literature evidence included in this study. The MOOSE checklist contains specifications and guidelines for the conduct and review of the studies. To evaluate the effects of acceleration thresholds on the number of impacts reported, variable thresholds were applied to head impact data obtained from 38 senior amateur rugby union players during 19 matches in New Zealand.[330]

***Literature review to identify thresholds for reporting head impact data in contact and collision sport***

***Search strategy for identification of publications***

A total of 53,183 studies available online from Jan 1990 to June 2015 identified through the SCOPUS (n=10,090), SportDiscus (n= 1,187), OVID (n= 9,729), Science Direct (n= 27,803) and Health Sciences (n= 4,376) databases were screened for eligibility (see Fig. 7). The keywords utilized for the search of relevant research studies included combinations of 'head impact telemetry system*', 'HITS', 'concussion', 'impact*', 'traumatic brain injury', 'chronic traumatic encephalopathy', 'angular', 'linear', 'rotational', 'acceleration', 'biomechanics', 'head acceleration' and 'risk'. An example of the Health Sciences search strategy is provided in the Table 27. Searches were limited to 'English language' and 'humans' only. The references of all relevant articles were searched for further articles. All publications identified were initially screened by publication title and abstract to identify eligibility. In cases of discrepancies of eligibility another author assessed the publication to screen for eligibility.

To establish some control over heterogeneity of the studies,[575] inclusion criteria were established. Any published study or book that did not meet the inclusion criteria was excluded from the study. Publications were included if they reported head impact biomechanics and met the following inclusion criteria:

(i) The study was published in a peer reviewed journal or book; and

(ii) The study reported the biomechanics of impacts to the head in a sporting environment; and

(iii) The study addressed one or more of the keywords relating to this study.

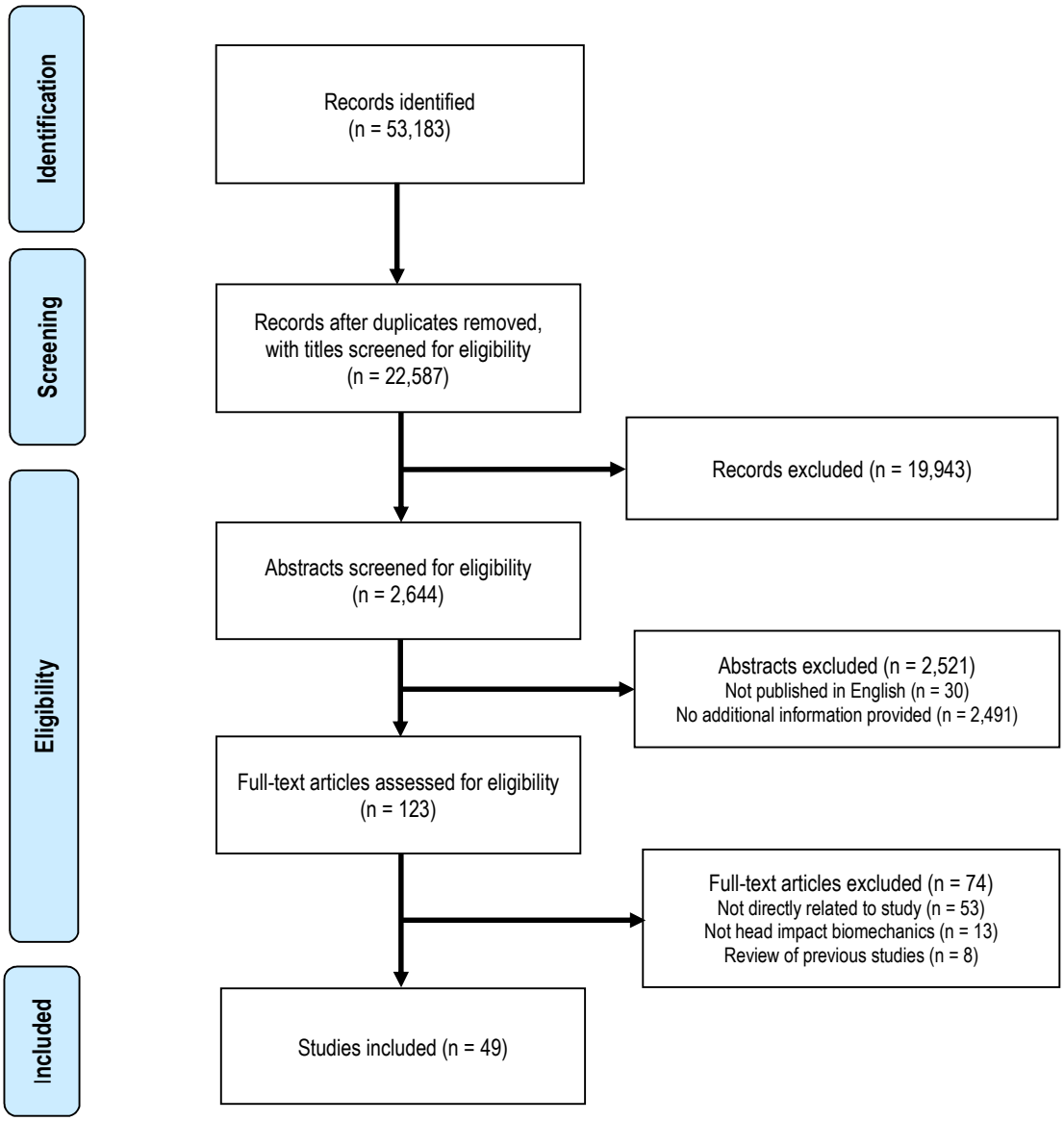


**Figure 7:** Flow of identification, screening, eligibility and study inclusion of published studies.

**Table 27:** An example of the Health Sciences search strategy for head impact telemetry

The use of the '*' is a truncation

| | |
|---|---|
| **1.** | Traumatic brain inj* |
| **2.** | Concussion* |
| **3.** | Head impact telemetry system |
| **4.** | Linear |
| **5.** | Rotational |
| **6.** | 1 and 3 |
| **7.** | 2 and 3 |
| **8.** | 3 and 4 |
| **9.** | 3 and 5 |
| **10.** | 6 or 7 or 8 or 9 |
| **11.** | Limit 10 to (English language and humans) |

**Table 28:** MOOSE scores, data acquisition impact thresholds, study groups, sporting codes and duration, instrumented equipment, participant numbers, impacts recorded for total and per player and metrics for reporting data

| Study | MOOSE[575] Score | Data acquisition Limit (*g*) | Study group | Sport, No. seasons | No Participants | Impacts total | Impacts per player | Raw data | | Derived variables | | | | Reporting statistics | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | PLA(*g*) | PRA(rad/s²) | $HIC_{15}$ | $HIC_{36}$ | GSI | HITsp | Mean (SD) | Median | IQR | 95% | Other |
| Hernandez et al.[272] | 4/6 (67%) | 7; 10 | Coll; P | Am F, MM, B | | | 2 concussions; T: 513 | Y | Y | | | | | | Y | Y | | |
| Brolinson et al.[53] | 5/6 (83%) | 10 | Coll | Am F, 2 | 52 | 11,604 | T; 223[a] | Y | | | | | | Y | | | | |
| Bazarian et al.[26] | 5/6 (83%) | 10 | Coll | Am F, 1 | 10 | 9,769 | T; 977[a] | Y | Y | | | | | Y | | | | |
| Crisco et al.[110] | 5/6 (83%) | 10 | Coll | Am F, 3 | 314 | 286,636 | T: 420;[b] P: 250;[b] M: 128[b] | Y | Y | | | | Y | | Y | | Y | Y |
| Crisco et al.[111] | 5/6 (83%) | 10 | Coll | Am F, 2 | 254 | 184,358 | T: 726[a] | Y | Y | | | | Y | | | | y | |
| Daniel et al.[115] | 5/6 (83%) | 10 | Youth | Am F, 1 | 7 | 748 | T: 107; P: 63; M: 44 | Y | Y | | | | | | Y | | Y | Y |
| Duma et al.[141] | 5/6 (83%) | 10 | Coll | Am F, 1 | 38 | 3,312 | T: 87[a] | Y | | Y | | Y | | Y | | | | |
| Funk et al.[174] | 5/6 (83%) | 10 | Coll | Am F, 4 | 98 | 37,128 | T: 379[a] | Y | | Y | | | | | | | | |
| Hanlon et al.[261] | 5/6 (83%) | 10[H] | Youth | Soccer, P | 24 | 47 H 20 NH | N/S | Y | Y | Y | | Y | | | | | | |
| Harpham et al.[265] | 5/6 (83%) | 10 | Coll | Am F, 1 | 38 | N/S | N/S | Y | Y | | | | Y | Y | | | | |
| King et al.[330] | 5/6 (83%) | 10[M] | Snr Amat | RU, 1 | 38 | 20,687 | T: 564; M: 77 | Y | Y | | | | | Y | | | | |
| Mihalik et al.[449] | 5/6 (83%) | 10 | Coll | Am F, 2 | 72 | 57,024 | T: 9,504[a] | Y | | | | | | Y | | | | Y |
| Mihalik et al.[453] | 5/6 (83%) | 10 | Youth | IH, 1 | 37 | 7,770 | T: 1,945[a] | Y | Y | | | | | Y | | | | Y |
| Mihalik et al.[452] | 5/6 (83%) | 10 | Youth | IH, 2 | 52 | 12,253 | T: 223;[b] P: 83;[b] M: 24[b] | Y | Y | | | | Y | Y | | | Y | Y |
| Mihalik et al. [451] | 5/6 (83%) | 10 | Youth | IH, 1 | 16 | 4,608 | T: 288[a] | Y | Y | | | | Y | Y | | | | Y |
| Munce et al.[464] | 5/6 (83%) | 10 | Youth | Am F, 1 | 22 | 6,183 | T: 281[a] | Y | Y | | | | Y | Y | Y | | Y | |
| Ocwieja et al.[478] | 5/6 (83%) | 10 | Coll | Am F, 1 | 46 | 7,992 | T: 174[a] | Y | Y | | | | Y | Y | | | | Y |
| Reed et al.[521] | 5/6 (83%) | 10 | Youth | IH, 1 | 13 | 1,821 | T: 140; M: 5 | Y | Y | Y | Y | | | | | | | Y |
| Rowson et al.[528] | 5/6 (83%) | 10 | Coll | Am F, 1 | 10 | 1,712 | T: 171[a] | Y | Y | | | | | Y | | | | Y |
| Schnebel et al.[538] | 5/6 (83%) | 10 | Coll | Am F, 1 | 40 | 54,154 | T: 1,354[a] | Y | | | | | | | | | | Y |
| | | 10 | HS | Am F, 1 | 16 | 8,326 | T: 520[a] | Y | | | | | | | | | | Y |
| Beckwith et al.[28, 29] | 5/6 (83%) | 14.4 | Coll / HS | Am F, 6 | 95 | 161,732 | T: 1,702[a] | Y | Y | Y | Y | | | | | | | Y |
| Broglio et al.[47] | 5/6 (83%) | 14.4 | HS | Am F, 1 | 42 | 32,510 | T: 744; P: 11;[c] M: 24 | Y | Y | | | | Y | | | | | |
| Cobb et al.[84] | 5/6 (83%) | 14.4 | Youth | Am F, 1 | 50 | 11,978 | T: 240; P: 10; M: 11 | Y | Y | | | | | | Y | | Y | Y |
| Crisco et al.[109] | 5/6 (83%) | 14.4 | Coll | Am F, 1 | 188 | 3,878 | T: 21;[a] P: 6; M: 14 | | | | | | | | | | | Y |
| Daniel et al.[116] | 5/6 (83%) | 14.4 | Youth | Am F, 1 | 17 | 4,678 | T: 275;[a] P: 163; M: 112 | Y | Y | | | | | | Y | | Y | Y |
| Rowson et al.[531] | 5/6 (83%) | 14.4 | Coll | Am F, 2 | 314 | 300,977 | T: 959[a] | | Y | | | | | | | | | |
| Talavage et al.[581] | 5/6 (83%) | 14.4 | HS | Am F, 1 | 21 | 15,264 | T: 727[a] | | | | | | | | | | | |
| Urban et al.[596] | 5/6 (83%) | 14.4 | HS | Am F, 1 | 40 | 16,502 | T: 413[a] | Y | Y | | | | | Y | Y | Y | Y | |
| Young et al.[631] | 5/6 (83%) | 14.4 | Youth | Am F, 1 | 19 | 3,059 | T: 161; P: 95; M: 65 | Y | Y | | | | | Y | Y | | Y | Y |
| Broglio et al.[49] | 5/6 (83%) | 15 | HS | Am F, 3 | 78 | 54,247 | T: 695[a] | Y | Y | | | | | Y | | | | Y |
| Broglio et al.[50] | 5/6 (83%) | 15 | HS | Am F, 1 | 35 | 19,224 | T: 549;[a] P: 9; M: 25 | Y | Y | | | | | | | | | Y |
| Broglio et al.[44, 52] | 5/6 (83%) | 15 | HS | Am F, 4 | 95 | 101,994 | T: 652 | Y | Y | | | | Y | Y | | | | Y |
| Eckner et al.[150] | 5/6 (83%) | 15 | HS | Am F, 2 | 20 | 30,298 | T: 1,515[a] | Y | Y | | | | Y | Y | Y | Y | Y | Y |
| Martini et al.[398] | 5/6 (83%) | 15 | HS | Am F, 2 | 83 | 35,620 | T: 429[a] | Y | Y | | | | Y | Y | | | | |
| Wilcox et al.[616] | 5/6 (83%) | 20 | Coll | IH M/F,3 | 91 | 37,411 | T: 19,980[d] / 17,531[e] | Y | Y | | | | Y | | Y | Y | Y | Y |
| Wilcox et al.[618] | 5/6 (83%) | 20 | Coll | IH M/F,1/3 | 54 | 616 | T: 270[d] / 242 | Y | Y | | | | Y | Y | | | | |
| Wong et al.[626] | 5/6 (83%) | 30 | Youth | Am F, 1 | 22 | 480 | T: 22;[a] P: 4; M: 2 | Y | | | | | | Y | | | | |
| Gysland et al.[257] | 5/6 (83%) | <60 >90 | Coll | Am F, 1 | 46 | N/S | T: 1,177; 12 >90*g* | Y | | | | | | | | | | Y |
| McCaffrey et al.[403] | 5/6 (83%) | <60 >90 | Coll | Am F, 1 | 43 | N/S | N/S | Y | | | | | | Y | | | | Y |
| Fréchède et al.[168 A] | 4/6 (67%) | Recon | Prof | AFL / RU, 3 | - | - | N/S | | Y | Y | | | | Y | | | | Y |
| McIntosh et al.[433 A] | 4/6 (67%) | Recon | Prof | AFL, 3 | - | - | N/S | Y | Y | | | | | | | | | Y |
| Pellman et al.[498 A] | 4/6 (67%) | Recon | Prof | Am F, 5 | - | - | N/S | Y | Y | Y | | | | Y | | | | |
| Zhang et al.[632 A] | 4/6 (67%) | Recon | Lab | - | - | - | - | Y | Y | Y | | | | Y | | | | Y |
| Breedlove et al.[40 A] | 4/6 (67%) | N/S | HS | Am F, 2 | 24 | N/S | N/S | Y | Y | | | | | | Y | | | Y |
| Duhaime et al.[140 A] | 4/6 (67%) | N/S | Coll | Am F, IH, 4 | 450 | 486,594 | T: 1,081[a] | Y | Y | | | | | | | | | |
| Greenwald et al.[241 A] | 4/6 (67%) | N/S | Coll / HS | Am F, 3 | 449 | | 17 concussions only | Y | Y | Y | | | Y | | | | Y | |
| Guskiewicz et al.[254 A] | 4/6 (67%) | N/S | Coll | Am F, 2 | 88 | 104,714 | T: 1,190[a] | Y | | | | | | Y | | | | |
| Rowson et al.[530 A] | 4/6 (67%) | N/S | Coll | Am F | N/S | 63,011 | Combined data | Y | Y | | | | | | | | | Y |
| Wilcox et al.[617] | 4/6 (67%) | N/S | Coll | IH F, 3 | 58 | | 9 concussions | Y | Y | | | | Y | Y | | | | |
| **Mean study quality** | 4.8 ±0.4 (79.6% ±7.0) | | | | | | Percentage of studies | 91.5 | 92.0 | 76.6 | 76.0 | 21.3 | 18.0 | 4.3 | 4.0 | 6.4 | 4.0 | |

Instrumented equipment used is helmet unless the data acquisition limit is reconstructed = Recon, or superscript M = Mouthguard or H = Headband. Coll = Collegiate; HS = High School; Snr Amat = Senior Amateur; Prof = Professional; Am F = American Football; IH = Ice Hockey; RU = Rugby Union; AFL = Australian Football League; MM = Mixed martial Arts; B = Boxing; T = Total impacts; P = Practice Impacts; M = Match impacts; a = calculated number of impacts; b = Median results; c = contact practice; d = male; e = female; H = Header; NH = non-header; N/S = Not Stated; PLA(g) = Peak Linear Acceleration; PRA(rad/s2) = Peak Rotational Accelerations in radians/second/second (rad/s2); HIC15 = Head Impact Criterion 15 milliseconds; HIC36 = Head Impact Criterion 36 milliseconds; GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; SD = Standard Deviation; IQR = Inter-Quartile Range; 95% = 95th Percentile.

Reviewed studies were excluded from this review if it was identified that the publication:

(i) Was unavailable in English; or

(ii) Did not provide additional information specifically addressing areas relating to this study;

(iii) Was a case study; or

(iv) Reviewed head impact studies.

***Assessment of publication quality***

The 52 studies[26, 28, 29, 40, 44, 47, 49, 50, 52, 53, 84, 109-111, 115, 116, 140, 141, 150, 168, 174, 241, 254, 257, 261, 265, 272, 330, 398, 403, 433, 449, 451-453, 464, 478, 498, 521, 528, 530, 531, 538, 581, 596, 616-618, 626, 631, 632] meeting the inclusion criteria (see Table 28) were assessed for quality by two of the authors on the basis of the MOOSE[575] published checklist. Heterogeneity of the studies included in the literature review was expected as there might be differences in the study design, population and outcomes.[575] As a result of the MOOSE[575] checklist, the studies included had a median score of 4.8/6.0 with a range of 4.0-5.0.

***Application of head impact thresholds identified from the literature to the rugby head impact data set***

The data set, used for the application of the head impact thresholds identified from the literature review, was from 38 amateur rugby union players who wore instrumented mouthguards over a season of matches.[330] The raw data set was filtered by linear acceleration thresholds at increments of 1*g* to establish the percentage of impacts removed at each threshold from 10.0*g* to 30.0*g*. This percentage was then used to calculate the possible number of impacts removed for the impact thresholds used in the different studies reviewed.

All data estimations were calculated on an Excel spreadsheet. The data were analysed using SPSS v22.0.0 (SPSS Inc.) and, as the data were non-normally distributed (Shapiro-Wilk test $p<0.001$), data were analysed using a Friedman repeated measures ANOVA on ranks. Post hoc analysis with Wilcoxon signed-rank tests was conducted with a Bonferroni correction applied. Statistical significance was set at $p<0.05$. The estimated number of impacts were calculated by dividing the number of reported impacts by the estimated percentage of impacts removed at the different thresholds. The estimated total number of reported impacts were subtracted from the reported number of impacts to identify the possible number of impacts removed from the data set e.g. Number of impacts reported = 161,732;[28, 29] Impact threshold = 14.4*g*; Based on New Zealand rugby union dataset for 20,687 impacts recorded at 10.0*g* when reassessed at 14.4*g* there were 12,091 impacts. A total of 8,569 impacts were removed or 42% of the data set (see Fig 8). Therefore 161,732 (number of impacts reported) ÷ 42% (percentage of impacts removed at 14.4*g*) gave a possible total number of impacts at the 10*g* threshold of 385,076. The possible total number of impacts removed from the dataset was 223,344 (i.e. 385,076 – 161,732 impacts).

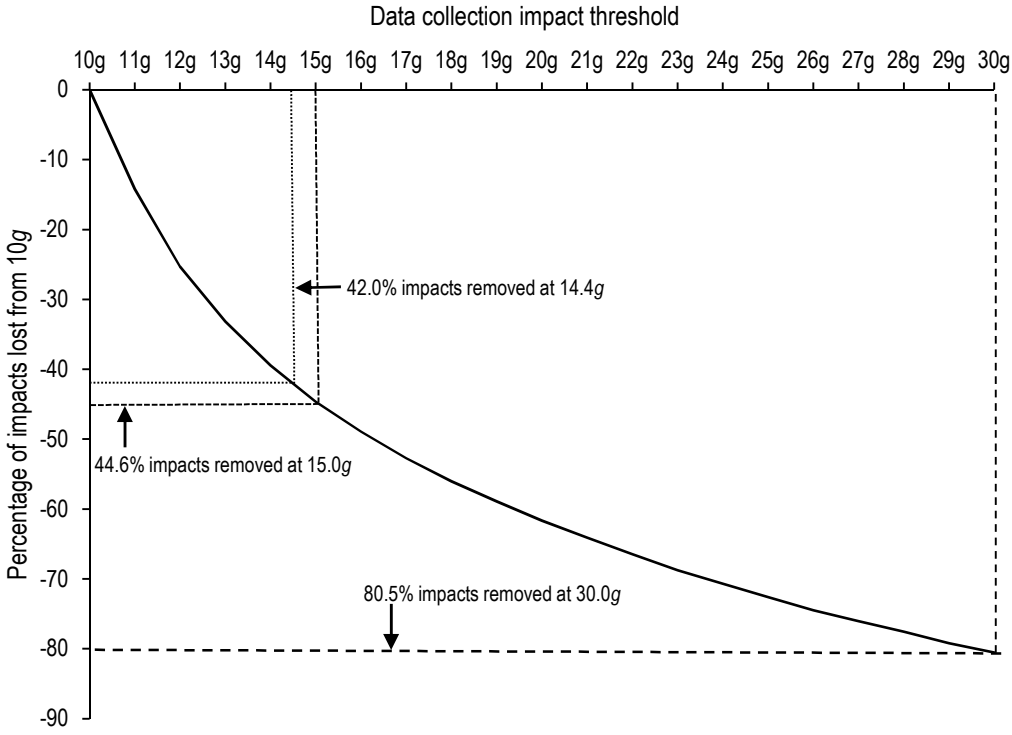


**Figure 8:** Percentage of impacts removed when applying different data impact threshold limits compared with original 10g threshold limit for the New Zealand data set of head impacts to senior amateur rugby union players for one season.

***Literature review***

A total of 52 publications were identified that reported head impacts and met the inclusion criteria. Studies reported impacts to the head via technology in American football,[28, 29, 49, 50, 52, 53, 110, 111, 115, 141, 261, 265, 272, 449, 464, 528, 531, 538, 596, 626] ice hockey,[451, 452, 521, 616-618] soccer,[261] rugby union[330] and mixed martial arts and boxing.[272]

***Impact threshold***

Studies utilised different data impact acceleration thresholds (see Table 26): 42% of studies[26, 53, 110, 111, 115, 141, 174, 261, 265, 272, 330, 449, 451-453, 464, 478, 521, 528, 538] used 10*g*; 18% of studies[28, 29, 47, 84, 109, 116, 531, 581, 596, 631] used 14.4*g*;10% of studies[44, 49, 50, 52, 150, 398] used 15*g*; 4% of studies[616, 618] used 20*g*; 2% of studies[626] used 30*g*; 4% of studies[257, 403] reported impact data within 10*g* to 60*g* and greater than 90*g*. Four studies[168, 433, 498, 632] (8%) were reconstruction studies from video analysis but were included as they reported impact biomechanics. Six studies[40, 140, 241, 254, 530, 617] (12%) did not report the impact threshold but did report head impact biomechanics. One study[272] (2%) used a 7*g* and 10*g* threshold with different sporting activities.

***Acceleration raw data and metrics***

Apart from raw resultant linear accelerations[53, 141, 174, 254, 257, 403, 449, 538, 626] (reported in 91% of studies) and rotational acceleration data[168, 531] (reported in 76% of studies),[26, 28, 29, 40, 44, 47, 49, 50, 52, 84, 110, 115, 116, 140, 150, 241, 261, 265, 330, 398, 433, 451-453, 464, 478, 498, 521, 528, 530, 596, 631, 632] several head impact derived variables were reported such as the Gadd Severity Index (GSI),[201] the Head Impact Criterion (HIC),[604] Head Impact Telemetry Severity Profile ($HIT_{SP}$)[241] and the Risk Weighted Cumulative Exposure (RWE)[596] metrics.

Three (4%) of the studies[28, 141, 261] reported the Gadd Severity Index (GSI). In 1966, Gadd[201] proposed the GSI head injury severity index based on the Wayne State Tolerance Curve (WSTC) (see Eq. 1). Developed from animal and cadaver impact data, the GSI simplified the WSTC by taking into consideration the shape of the linear acceleration time history, providing a weighting factor of 2.5 enabling the whole body acceleration data to be plotted on log-log coordinates along a straight line. The critical value of the GSI is 1,000. If the GSI is less than 1,000 then the head impact is considered probabilistically safe. The GSI is used to quantify severe skull fractures and brain injury risk but is not recommended for use to quantify a risk of concussion.[19] A concern of the GSI is that it can give unrealistically high values for impacts that have a much longer pulse duration.[368] The mathematical expression for the GSI is:

$$GSI = \int_0^T a(t)^{2.5} dt \quad (1)$$

where *a* is the 'effective' acceleration (thought to have been the average linear acceleration) of the head measured in terms of *g*, the acceleration of gravity, and *t* is the time in milliseconds from the start of the impact.[439]

In 1971 a modification of the Gadd Severity Index, the Head Injury Criterion (HIC), was proposed[604] to focus the severity index on that part of the impact that was likely to be relevant to the risk of injury to the brain. This was done by averaging the integration of the resultant acceleration/time curve over whatever time interval yielded the maximum value of HIC. Because this varies from one impact to another, the expression for the modified index simply refers to times *t*1 and *t*2. The HIC is computed based on the following expression:

$$HIC = \left[\frac{1}{t_2 - t_1} \int_{t_1}^{t_2} a(t)dt\right]^{5/2} (t_2 - t_1) \quad (2)$$

where $t_2$ and $t_1$ are any two arbitrary time points during the acceleration pulse (see Eq. 2). Acceleration is measured in multiples of the acceleration of gravity [g] and time is measured in seconds. The resultant acceleration is used for the calculation. The US National Highway Traffic Safety Administration (NHTSA) requires $t_2$ and $t_1$ not to be more than 36 ms apart (thus called $HIC_{36}$) and the maximum $HIC_{36}$ not to exceed 1,000. In 1998[342] the NHTSA introduced the $HIC_{15}$ where $t_2$ and $t_1$ was not to be more than 15 ms apart and the maximum $HIC_{15}$ was not to exceed 700. In a numerical study[310] it was estimated that a mild Traumatic Brain Injury (mTBI) tolerance for the $HIC_{15}$, where there is a 25%, 50% and 75% likelihood of an mTBI occurring, had $HIC_{15}$ values of 136, 235 and 333 respectively. Only two studies[29, 521] (4%) reported $HIC_{36}$ with ten studies (18%) reporting the $HIC_{15}$.[28, 29, 141, 168, 174, 241, 261, 498, 521, 632]

In 2008,[241] the principal component score (PCS), a weighted sum of linear acceleration, rotational acceleration, HIC and GSI, with objectively defined weights, was published (see Eq.3). Now more commonly termed the Head Impact Telemetry Severity Profile ($HIT_{SP}$), the $HIT_{SP}$ is a weighted composite score including linear and rotational accelerations, impact duration, as well as impact location. The resulting formula is:

$$HIT_{SP} = 10x([0.4718\ x\ sGSI + 0.4742\ x\ sHIC + 0.4336\ x\ sLIN + 0.2164\ x\ sROT] + 2 \quad (3)$$

where sX = (X-mean[X])/(SD[X]), LIN = linear acceleration, ROT = rotational acceleration, HIC = head injury criterion, and GSI = Gadd Severity Index. The offset by 2 and scaling by 10 generates $HIT_{SP}$ values greater than 0 and in the numerical range of the other classic measures studied. A $HIT_{SP}$ score of 63 or greater is reported to be an indication there is a 75% risk of a concussive injury occurring.[241] More than a quarter (30%) of the studies[44, 47, 52, 110, 150, 241, 265, 398, 451, 452, 464, 478] reported the $HIT_{SP}$.

In 2013, a novel cumulative exposure metric, the Risk Weighted Cumulative Exposure (RWE) equation was developed[596] with four previously published analytical risk functions. The four different analytical risk functions (see Table 29) were the linear resultant acceleration,[498, 529] rotational resultant acceleration[531] and combined probability (linear and rotational) resultant accelerations.[530] These risk functions were utilised to elucidate individual player and team-based exposure to head impacts. The RWE equations comprise of $a_L$ as the measured peak linear acceleration, $a_R$ as the measured peak rotational acceleration, and $n_{hits}$ as the number of head impacts in a season for a given player.

**Table 29:** Risk Weighted Cumulative Exposure (RWE) equations, where $a_L$ is the measured peak linear acceleration, $a_R$ is the measured peak rotational acceleration, and $n_{hits}$ is the number of head impacts in a season for a player.

| Risk function(s) | Equation |
|---|---|
| Linear[497, 528] | $RWE_{Linear} = \sum_{i=1}^{n_{hits}} \boldsymbol{R}(\boldsymbol{a}_L)i$ |
| Rotational[531] | $RWE_{Rotational} = \sum_{i=1}^{n_{hits}} \boldsymbol{R}(\boldsymbol{a}_R)i$ |
| Combined Probability[530] | $RWE_{CP} = \sum_{i=1}^{n_{hits}} \boldsymbol{CP}(\boldsymbol{a}_L, \boldsymbol{a}_R)i$ |

Logistic regression equations and regression coefficients of the injury risk functions utilised in the prediction of injury, where $\alpha$ and $\beta$ are the regression coefficients and $x$ is the measured acceleration for the linear and rotational risk functions (see Table 30).[596]

**Table 30:** Logistic regression equations and regression coefficients of the four injury risk functions utilized in the prediction of injury, where $\alpha$ and $\beta$ are the regression coefficients and $x$ is the measured acceleration for the linear,[12, 13] and rotational[531] risk functions.

| Logistic Regression equation | Risk Function | Regression coefficients |
|---|---|---|
| $R[a] = \frac{1}{1+e^{-\alpha+\beta x}}$ | Linear[497, 528] | $\alpha$ = -9.805, $\beta$ = 0.0510 |
| | Rotational[531] | $\alpha$ = -12.531, $\beta$ = 0.0020 |
| $\mathrm{CP} = \frac{1}{1+e^{-(\beta_0+\beta_1\alpha+\beta_2\alpha+\beta_3\,a\alpha}}$ | Combined Probability (CP)[530] | $\beta_0$ = -10.2, $\beta_1$ = 0.0433, $\beta_2$ = 0.000873, $\beta_3$ = -9.2E-07 |

$\beta_0$, $\beta_1$, $\beta_2$ and $\beta_3$ are regression coefficients, $a$ is the measured linear acceleration, and $\alpha$ is the measured rotational acceleration for the combined probability risk function. The three metrics provided as a result of these equations are for linear ($RWE_{Linear}$), rotational ($RWE_{Rotational}$) and combined (linear and rotational) probability ($RWE_{CP}$). Only one study[596] has reported on the RWE as an injury risk function.

In an attempt to delineate injury causation and to establish a meaningful injury criterion through the use of actual field data, Zhang et al.[632] proposed tolerance levels for human head injury based on input kinematics scaled from animal data and non-injurious volunteer test results. Injury predictors and injury levels were analysed based on resulting brain tissue responses and these were correlated with the site and occurrence of a concussion occurring. The calculated shear stress around the brainstem region could be an injury

predictor and statistical analyses were performed to establish a brain injury tolerance level. As a result of the analyses undertaken, and based on linear logistic regression analyses, it was reported[632] that the maximum resultant translational acceleration at the center of gravity (CG) of the head was estimated to be 66*g*, 82*g* and 106*g* for a 25%, 50% and 80% probability of sustaining an mTBI respectively.

For resultant rotational acceleration at the CG of the head this was estimated to be 4,600 rad/s$^2$, 5,900 rad/s$^2$ and 7,900 rad/s$^2$ for a 25%, 50% and 80% probability of sustaining an mTBI respectively.[632] The estimated $HIC_{15}$ thresholds were 151, 240 and 369 for a 25%, 50% and 80% probability of sustaining an mTBI.[632] These thresholds are considerably less than the $HIC_{15}$ limit of 1,000 for sustaining a serious brain injury.[632] If the head was exposed to a combined translational and rotational acceleration with an impact duration between 10 to 30 ms, the suggested tolerable reversible brain injury was 85*g* (translational acceleration), 6,000 rad/s$^2$ (rotational acceleration) and $HIC_{15}$ value of 240.[632] It was reported[632] that these values may change as more human data become available but to date no published updates of these values have been available.

Although other variables have been proposed (Generalised Acceleration Model for Brain Injury Threshold (GAMBIT),[272, 470, 471] and Head Impact Power (HIP);[472] these were not utilised in any studies reporting head impacts in contact sport.

Nearly all of the studies reviewed identified the number of impacts that were recorded, however 4% studies reported impacts in matches only, 23% recorded impacts separately for both match and practice activities, and 55% combined both match and practice activity impacts reporting them as a single dataset. The remaining 15% of studies reviewed reported on impacts above 90*g* or were reconstruction of impacts from video analysis The number of impacts ranged from 480 impacts from 22 players in Pop Warner American football[626] to 486,594 impacts from 450 players in collegiate American football and ice hockey[140] (see Table 26).

Over half (52%) of the studies[26, 28, 49, 52, 53, 141, 150, 168, 254, 265, 330, 398, 403, 449, 451-453, 464, 478, 498, 528, 596, 617, 618, 626, 631, 632] reported the impact biomechanics data as mean ± standard deviation (±SD). Some studies[28, 150, 272, 464, 596, 616, 631] (22%) also reported the head impacts as median, but not all[464, 631] (4%) included the interquartile ranges (IQR) for the data. Of the studies that reported the impact biomechanics by the median,[28, 138, 254, 441, 568, 587, 601] only 7% reported the IQR. Most of the studies reporting the median also reported the 95$^{th}$ percentile of the impacts. Other data reporting methodologies utilised within the data sets reviewed were the median of the 95$^{th}$ percentile,[110] the 98$^{th}$,[150, 241] 99$^{th}$,[150, 241] and 99.5$^{th}$[150] percentiles. Fourteen percent of studies also included lower and upper limits[449, 451, 452, 478] for the range of impacts,[140, 521] and the mean range[626] of the impacts. Less than a quarter of studies (23%) reported their impacts as x, y, z axis data,[528] +1SD,[538] Cumulative Distribution Functions (CDF),[115, 596] percentage of impacts,[109, 110] and the impact duration (ms).[28, 29, 44, 50, 498] In addition to the impact biomechanics being presented by various methodologies, 14% of studies[49, 52, 254, 265, 330, 478, 632] also incorporated impact tolerances and impact severity levels.

***Application of head impact thresholds to the rugby head impact data set***

By utilising data from a previously published study[330] that used the 10*g* impact threshold, data were re-extracted at differing impact thresholds from 10*g* to 30*g*. By adjusting the impact threshold (see Fig. 8) the number of impacts decreased as the impact threshold increased (see Table 31). There were significant differences observed ($p<0.05$) for each of the different acceleration thresholds for the number of impacts reported, the mean, median and the 95$^{th}$ percentile when compared with the impacts at the 10*g* linear acceleration threshold (see Table 29).

Based on the differences observed in the study reporting on impacts in amateur senior rugby union,[330] at the 14.4*g* threshold there could have been as many as 42% of the impacts recorded not being reported. As a result, studies[28, 29, 44, 47, 49, 50, 84, 116, 150, 531, 596, 626, 631] using impact thresholds above 10*g* may have removed 2,100 to 206,573 impacts. At the 30*g* impact threshold it can be estimated that 80 to 85% of impacts were not reported.[626] Again, based on the differences observed in this study through the analysis of different thresholds, it is possible that each player in the Pop Warner study[626] may have experienced a cumulative total of 1,885 impacts above 10*g*. Although the impacts may not have been recorded, the players may well have been exposed to this number of impacts between 10*g* and 30*g*. The differences between impacts reported and the possible number of impacts (480 vs. 2,365) may result in an underestimation of the exposure risk to these players to subconcussive impacts.

**Table 31:** Differences in the resultant linear (PLA(*g*)) and rotational (PRA(rad/s$^2$)) accelerations, head impact criterion (15ms) ($HIC_{15}$) and Gadd severity index (GSI) at different impact threshold limits by the mean and standard deviation (±SD), median [25$^{th}$ to 75$^{th}$ percentile] and 95$^{th}$ percentile for the New Zealand senior amateur rugby union total player dataset.

| Data acquisition impact threshold (*g*) | No of impacts | Resultant Linear Accelerations (PLA(*g*)) | | | Resultant Rotational Accelerations (PRA(rad/s$^2$)) | | | Head Impact Criterion 15ms ($HIC_{15}$) | | | Gadd Severity Index (GSI) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% |
| 10 | 20,687 | 22 ±16 | 16 [12-26] | 53 | 3,903 ±3,949 | 2,625 [1,324-4,934] | 12,204 | 32 ±99 | 9 [5-25] | 128 | 48 ±118 | 15 [8-398] | 192 |
| 11 | 17,747 | 24 ±17 | 18 [13-29] | 56 | 4,255 ±4,096 | 2,898 [1,549-5,389] | 12,945 | 37 ±106 | 11 [6-30] | 145 | 55 ±126 | 19 [10-47] | 218 |
| 12 | 15,454 | 26 ±17 | 20 [15-31] | 59 | 4,603 ±4,214 | 3,181 [1,781-5,860] | 13,581 | 42 ±112 | 14 [7-35] | 160 | 62 ±134 | 23 [12-55] | 241 |
| 13 | 13,825 | 28 ±17 | 22 [16-32] | 62 | 4,858 ±4,293 | 3,423 [1,967-6,263] | 13,948 | 46 ±118 | 17 [9-40] | 176 | 69 ±140 | 27 [14-62] | 262 |
| 14 | 12,531 | 29 ±18 | 24 [18-34] | 64 | 5,079 ±4,368 | 3,589 [2,123-6,596] | 14,325 | 51 ±123 | 19 [10-44] | 188 | 75 ±146 | 31 [17-69] | 278 |
| 15 | 11,459 | 31 ±18 | 25 [19-35] | 65 | 5,286 ±4,438 | 3,774 [2,263-6,908] | 14,647 | 55 ±128 | 22 [12-49] | 205 | 80 ±151 | 34 [19-76] | 297 |
| 16 | 10,570 | 32 ±18 | 26 [20-36] | 67 | 5,478 ±4,510 | 3,936 [2,400-7,180] | 14,994 | 59 ±133 | 24 [14-53] | 215 | 86 ±156 | 38 [21-82] | 318 |
| 17 | 9,784 | 33 ±18 | 27 [21-38] | 68 | 5,655 ±4,565 | 4,082 [2,538-7,394] | 15,235 | 63 ±137 | 27 [15-57] | 228 | 92 ±161 | 41 [24-88] | 331 |
| 18 | 9,095 | 34 ±18 | 28 [22-39] | 70 | 5,799 ±4,610 | 4,173 [2,644-7,567] | 15,486 | 67 ±141 | 29 [17-62] | 241 | 97 ±165 | 45 [27-95] | 348 |
| 19 | 8,500 | 35 ±19 | 29 [23-40] | 71 | 5,939 ±4,662 | 4,265 [2,731-7,744] | 15,823 | 70 ±145 | 32 [18-66] | 253 | 103 ±169 | 49 [29-102] | 364 |
| 20 | 7,934 | 36 ±19 | 30 [24-41] | 74 | 6,072 ±4,716 | 4,357 [2,810-7,931] | 16,256 | 74 ±150 | 34 [20-70] | 263 | 109 ±174 | 53 [31-109] | 374 |
| 21 | 7,430 | 37 ±19 | 31 [25-42] | 76 | 6,206 ±4,757 | 4,483 [2,896-8,158] | 16,470 | 79 ±154 | 37 [22-75] | 275 | 115 ±178 | 57 [34-114] | 391 |
| 22 | 6,938 | 39 ±19 | 32 [26-44] | 77 | 6,363 ±4,801 | 4,595 [2,992-8,426] | 16,806 | 83 ±158 | 40 [24-80] | 291 | 121 ±183 | 62 [37-121] | 415 |
| 23 | 6,463 | 40 ±19 | 33 [27-45] | 80 | 6,519 ±4,859 | 4,722 [3,096-8,628] | 17,073 | 88 ±163 | 43 [26-85] | 302 | 127 ±188 | 67 [40-129] | 444 |
| 24 | 6,060 | 41 ±19 | 34 [28-46] | 82 | 6,656 ±4,906 | 4,835 [3,201-8,798] | 17,282 | 92 ±167 | 46 [28-90] | 318 | 134±192 | 71 [43-135] | 466 |
| 25 | 5,666 | 42 ±20 | 35 [29-47] | 83 | 6,819 ±4,952 | 4,965 [3,305-9,012] | 17,435 | 97 ±172 | 49 [31-95] | 337 | 141 ±197 | 76 [47-144] | 485 |
| 26 | 5,275 | 43 ±20 | 36 [30-48] | 84 | 6,977 ±4,986 | 5,101 [3,428-9,297] | 17,622 | 102 ±177 | 53 [33-101] | 357 | 148 ±202 | 81 [50-152] | 512 |
| 27 | 4,955 | 44 ±20 | 37 [31-49] | 87 | 7,107 ±5,036 | 5,210 [3,495-9,459] | 17,844 | 107 ±181 | 57 [35-107] | 389 | 155 ±206 | 86 [54-162] | 536 |
| 28 | 4,642 | 45 ±20 | 39 [32-51] | 88 | 7,261 ±5,079 | 5,339 [3,607-9,704] | 18,131 | 113 ±186 | 60 [38-114] | 396 | 163 ±211 | 93 [58-173] | 557 |
| 29 | 4,305 | 47 ±20 | 40 [33-52] | 91 | 7,448 ±5,130 | 5,492 [3,778-9,917] | 18,221 | 119 ±192 | 65 [41-123] | 407 | 172 ±217 | 99 [64-186] | 583 |
| 30 | 4,024 | 48 ±20 | 41 [34-54] | 92 | 7,597 ±5,187 | 5,624 [3,875-10,129] | 18,436 | 125 ±197 | 69 [44-131] | 420 | 180±221 | 106 [68-196] | 606 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); Significant difference ($p$<0.05) than: (a) = 10*g*.

## Discussion

This study undertook to review the methods for reporting head impact data in sport and to outline the effect of various acceleration thresholds on the number of impacts reported. A consensus on a threshold for reporting data is important given the variation in conclusions that may be drawn if the same dataset is used with different thresholds, as identified by our application of the range of thresholds from prior literature applied to a New Zealand rugby union head impact data set. A standard threshold for head impact data is important given possible monitoring of player head impact acceleration data in the hope of identifying a cumulative threshold for concussion from subconcussive impacts.

The equipment utilised to record and report head impacts vary in the sensitivity and the types of algorithms they employ for the identification of impacts.[629] These differences may invariably influence the results of the published studies as, although some studies report the linear threshold as 14.4*g*, they may actually be recording from 10*g* and, if the researcher is unaware that this threshold is the default then the data may be included (personal correspondence S. Broglio; Sept 2015). In the recording of data for the HITS, the data is based on the triggering of one accelerometer, and the unfiltered / unprocessed data only loosely relates to the final measurement of interest at the heads centre of gravity.

The discussion surrounding subconcussive impacts has become popular.[13, 43, 132, 257, 398, 566] Initially the term subconcussive impact described an impact that did not result in severe, noticeable symptoms, especially loss of consciousness[132] However, recently, subconcussive is a term used to describe an asymptomatic non-concussive impact to the head.[13, 43, 257, 398, 566] The issue relating to the effects of subconcussive impacts is controversial as researchers and clinicians are divided on the true effects.[21, 209, 257, 435, 455, 581] Some research[257, 455] has reported that these impacts have minimal effect on cognitive functions, while others[21, 209, 435, 504, 581] have reported these impacts to be detrimental to cerebral and cognitive functions. To date, there is a paucity of evidence to identify the impact acceleration that is adequate to produce a non-structural brain injury associated with the neuronal changes of concussion.[21]

Animal models display metabolic changes associated with concussion, which may be similar in subconcussive impacts.[157] To research subconcussive impacts in isolation is challenging and there are, to date, no reports on animal models or other reliable methodologies that have been successful at identifying these impacts[157] Brain injury may occur from concussive events as well as from an accumulation of subconcussive impacts.[566] The effects of concussive events and multiple subconcussive impacts have been associated with long term progressive neuropathologies and cognitive deficits.[13, 437, 487, 572] Longitudinal impact monitoring at the level where these subconcussive events are beginning to occur is important, and a standard threshold needs to be established.

### *What threshold should be used to monitor head impacts?*

Impacts <10*g* of linear acceleration have been considered negligible in regards to impact biomechanical features. The <10*g* impact threshold has been used in research to eliminate head accelerations from non-impact events such as jumping and running.[110, 449, 473] The inclusion of these non-impact events to head

trauma make it difficult to distinguish between head impacts and voluntary head movement[450] and eliminating these will help identify the true extent of the number of impacts that do occur from sports participation. A suggestion for this may be to report the distribution of the impacts by the various resultant linear accelerations using a frequency analysis and reporting quartile ranges i.e. 25$^{th}$ and 75$^{th}$ interquartile range. It is usual statistical practice to report heavily skewed data through the use of percentiles.[136, 404] This may assist in identifying where the most frequent resultant linear accelerations occur in the different sports. Consensus for the impact threshold will need to be established, and should be based on validation studies to determine the best impact threshold for various sports and injury outcomes. Biomechanical modelling of impact forces and brain movement would first be needed to identify likely impact thresholds for injury, as well as in-field validation studies using prospective monitoring of players during tackles and impacts with the ground. As there is no established criterion for reporting head impact biomechanics, and the majority of studies (42%)[26, 53, 110, 111, 115, 141, 174, 261, 265, 272, 330, 449, 451-453, 464, 478, 521, 528, 538] reported the resultant linear acceleration threshold at 10*g*, then future studies should report all impacts above the 10*g* resultant linear acceleration threshold.

***What descriptive statistics should be used to report head impact biomechanics?***

There were a variety of descriptive statistics used in the reporting of head impact biomechanics in the reviewed studies which limits inter-study comparisons. Although more than half (52%) of the studies reviewed[26, 28, 49, 52, 53, 141, 150, 168, 254, 265, 272, 330, 398, 403, 449, 451-453, 464, 478, 498, 528, 596, 617, 618, 626, 631, 632] reported their results by means and standard deviations, the use of these statistics may not accurately represent the true centre of the data. By reporting the mean value of the data set, this method is subject to extreme values (i.e. outliers) such as those in skewed datasets. The use of the mean is suitable if the dataset has a symmetrical distributed. In non-normal distributed data, the median is the most useful for describing the center of the data.[127, 387] Of the studies[28, 150, 464, 596, 616, 631] reviewed (22%) that reported the results by the median would more accurately have identified the center of the dataset. The New Zealand senior amateur head impact data were non-normally distributed (i.e. not symmetrical) therefore the use of descriptive statistics that can better represent this skewness needed to be considered. To enable inter-study comparisons, and until a consensus is established for the reporting of head impact biomechanics, future studies should report the median [25$^{th}$ and 75$^{th}$ interquartile ranges] for all head impact biometrics.

***What acceleration metrics should be used to monitor head impacts?***

It has been suggested that both resultant linear and rotational accelerations should be reported with head impact metrics.[481] As there is an improved correlation between impact biomechanics and the occurrence of a concussion, than when linear accelerations are reported alone.[632] Research[68, 344, 346, 550, 606] suggests that the brain is more sensitive to rotational than linear accelerations. Rotational accelerations are reported[594, 632] to be correlated to the strain response of the brain and the primary mechanism for diffuse brain injury including concussion, contusion, axonal injuries and loss of consciousness.[311, 343, 344, 346] Linear accelerations are reported to result in the intracranial pressure response of the brain and be the primary mechanism for

skull fractures and epidural haematomas.[345, 594] Reporting both linear and rotational accelerations should assist with identification of possible brain injury.

More recently[530, 596] resultant linear and rotational acceleration results have been combined into a risk weighted exposure (RWE) metric. This metric can be beneficial for fully capturing the linear ($RWE_{Linear}$), rotational ($RWE_{Rotational}$) and combined probability (from linear and rotational) ($RWE_{CP}$) of the risk of a concussion as it accounts for the frequency and severity of each player's impacts. The HIC and GSI are the most frequently utilised head injury assessment functions in helmet and traffic restraint safety standards,[442, 632] however this was not reflected in the sport head impact studies reviewed. Based on the Wayne State University tolerance curve,[604] the HIC and GSI criteria are considered plausible ways of determining relative risk of severe head injury[375] but they do not account for the complex motion of the brain, or the contribution of resultant rotational acceleration to the head.[471, 472, 632] In particular the HIC only deals with frontal impacts and was not designed to be used for lateral impacts that can be found in head impact biomechanics[442] and arbitrarily defines an 'unsafe pulse" within a 'safe pulse" by discounting any data outside the two time points chosen for the calculation of the HIC value.[164] The GSI and HIC may be beneficial for evaluating acute head trauma due to single impacts but they are reportedly not beneficial for repeated impacts at lower acceleration magnitudes[442] such as those found in contact sports such as American football, rugby union and soccer. The inclusion of the HIC and GSI by studies reporting on head impact biomechanics may be more historical thus providing the ability for inter-study comparisons with previous studies. However, as they are used to calculate multiple impacts and provide a nonsensical number, the value of these metrics are limited. The use of HIC and GSI in future studies, and the value that these metrics provide, needs to be standardised. Consensus is required on the incorporation of these and other biomechanical metrics into future research.

***Limitations in the use of accelerometery***

The use of accelerometers to record and assess movement is not new to the scientific community.[87, 628] There have been some inter-study and international comparability limitations reported for use of accelerometers to report physical activity.[495] The identified limitations for physical activity accelerometers may be identical to areas now being faced by studies reporting the biomechanics of impacts to the head. The majority of studies reporting head impact biomechanics have utilised HITS,[26, 28, 29, 40, 44, 47, 49, 50, 52, 53, 84, 109-111, 115, 116, 140, 141, 150, 174, 241, 254, 257, 265, 398, 403, 449, 451-453, 464, 478, 521, 528, 530, 531, 538, 581, 596, 626, 631] or a variant.[261] More recently, an electronic mouthguard has been used to assess head impacts in rugby union.[330]

The issues identified with the use of accelerometers for physical activity[495] include affordability of the accelerometers,[495] and the administration burden[495] to the participants and researcher(s) given post data collection analysis. The choice of accelerator brand,[493] generation[62] and firmware version,[293] wearing position[630] based on the sports code requirements (i.e. helmet mounted vs. headband mounted vs. mouthguard embedded vs. patch), specifics of the research being undertaken such as the epoch length[151, 479] (match vs. training vs. combined), data imputation methods,[364] dealing with spurious data[161] and the reintegration of smaller epochs into larger epochs[307] are all considerations for use of accelerometers. In addition to the issues identified, there are technological developments, emerging methodological questions

and a lack of academic consensus that may also hinder the development of uniformity in the utilisation of accelerometers[495] for recording head impact biomechanics.

In comparing the New Zealand rugby union data with data collected with the use of the HITS, it must be noted that these are different impact telemetry systems. The mouthguard is reported to have a 10% error for linear and rotation acceleration and for angular velocity with an average offset of 2° for azimuth and elevation impact location.[63, 401] Although the correlation of the AIM mouthguard with laboratory head-forms is good, the impact measurements should be assumed to have some form of error that is dependent on impact conditions and the measure of interest and the variability tested.[27, 140] It is unlikely that the mouthguard was tested under all of the activities seen in rugby union matches such as the rucks, mauls, lineouts and scrum situations. How these rugby activities correlate to the laboratory conditions is unknown. .Although the majority of the impact biomechanics studies reported in this review are helmet based telemetry systems, there is a paucity of studies reporting on head impact biomechanics with other systems such as the mouthguard and headband. In addition there are no published studies comparing the HITS with other forms of impact telemetry systems such as the X2Biosystems All-In-Mouth (AIM) mouthguard.

A final consideration to the use of accelerometers in recording impacts is the need for concurrent video-analysis to enable comparison and verification of the impacts. This would enable the identification of non-impact activities where an impact has been recorded such as post-try celebrations, dropping equipment onto the ground, or other activities where the equipment may record an impact. In the case of the New Zealand rugby union data, only impacts that occurred in the tackle with the player standing were able to be verified.[330] The percentage of impacts that were identified at the 10*g* inclusion limit, that were able to be visualised by video review and analysis, varied from 65% to 85% of the total impacts recorded per match.[330]

***What are the long term implications of repeated head impacts?***

The use of impact tolerance and impact severity level data may be important if a risk assessment is undertaken for possible long term implications from repetitive head impacts (RHI). Recently in a small sample[26] of collegiate players with no reported concussions after a season of American football, there were white matter changes that correlated with multiple head impact measures. Participants with more than 30-40 RHI's with peak rotational accelerations >4,500 radians per second per second (rad/s$^2$) per season ($r$=0.91; $p$<0.001), and more than 10-15 RHI's >6,000 rad/s$^2$ ($r$=0.81; $p$<0.001), were significantly correlated with post-season white matter changes.[26] These changes post season imply a relationship between the number of RHIs that occur over a season of American football and white matter injury, despite no clinically evident concussion being recorded.[26]

The inclusion of impact tolerances and impact severity levels may assist with the identification of players at risk of possible long term injuries. Impact tolerance may also act as an indicator of when to rest players if they are exposed to RHIs above >4,500 rad/s$^2$ and >6,000 rad/s$^2$. This type of information will assist in formulating a detailed understanding of the exposure and mechanism of injury of concussion.[42, 109] Further research is required to evaluate the injury tolerance of concussive type injuries, to develop interventions to reduce the likelihood of any concussive type injuries, and to develop exposure durations and stand down

periods to establish a broader understanding of the potential role of subconcussive events and long term health.[109]

**Conclusion**

This study identified the methodological differences in the threshold limits of impacts to the head as a result of participation in contact sports. Of the 52 studies, 42% reported impacts at the 10*g* impact threshold[26, 53, 110, 111, 115, 141, 174, 261, 265, 272, 330, 449, 451-453, 464, 478, 521, 528, 538] while 18% of studies[28, 29, 47, 84, 109, 116, 531, 581, 596, 631] used the 14.4*g* impact threshold. Resultant linear accelerations were most frequently reported[53, 141, 174, 254, 257, 403, 449, 538, 626] (91%) while 76% reported resultant rotational accelerations. [26, 28, 29, 40, 44, 47, 49, 50, 52, 84, 110, 115, 116, 140, 150, 241, 261, 265, 330, 398, 433, 451-453, 464, 478, 498, 521, 528, 530, 596, 631, 632] Nearly three-quarters (74%) of studies reported both resultant linear and rotational accelerations. Impact data were most frequently (52%) reported [26, 28, 49, 52, 53, 141, 150, 168, 254, 265, 330, 398, 403, 449, 451-453, 464, 478, 498, 528, 596, 617, 618, 626, 631, 632] as mean ± standard deviation (±SD). Some (10%) studies[28, 150, 272, 464, 596, 616, 631] reported the head impact data as median, but not all[464, 631] (4%) included the interquartile ranges (IQR) for these data.

The influence of head impact thresholds was shown using head impact data obtained from 38 senior amateur rugby union players during 19 matches in New Zealand. Application of the varied head impact thresholds resulted in 20,687 impacts >10*g*; 11,459 (44.6% less), impacts >15*g*; and 4,024 (80.5% less) impacts >30g.

Given head impacts are likely to cause concussive injury, and the number of head impacts may be related to a potential concussion threshold (i.e. a cumulative threshold), the number and severity of head impacts should be monitored in players. However, impacts can be measured with several technologies (e.g. instrumented behind the ear patches, mouthguards, head gear), and thresholds have differed for reporting impact data in contact and collision sports. Consensus is therefore required to identify the reporting modalities (e.g. linear threshold, descriptive calculations), utilised in future impact studies to enable between study comparisons. Until in-field validation studies are completed, it is recommended that data should be reported as mean ± standard deviation, median and interquartile ranges using the 10g impact threshold to enable inter-study comparisons.

***“Thank you for explaining to me what a concussion is. Now I feel empowered and can make the decisions for the kids in my team and stop them playing when they may be injured.”***

Team Manager and mother of a concussed player - 2015

# CHAPTER 9: SUMMARY/CONCLUSIONS

The topic of sport-related concussions has drawn the attention of the sports medicine world, the media and the public in general. The issue of the longitudinal effects of concussion is an area that is increasing in the published research in sports such as American Football and boxing. What is not known is the incidence, costs, and the magnitude of these injuries in sports such as rugby league and rugby union. In addition, the effectiveness of concussion assessment tools for sideline use is not yet known for rugby codes. The research in this thesis aimed to assist in broadening the knowledge base of sport-related concussion by conducting a series of studies focused on: (1) The costs (Chapter 3), and frequency (Chapter 4) of sport-related concussions via epidemiological analyses of longitudinal data; (2) The identification of sport-related concussions with the use of a saccadic reading tool (Chapters 5 and 6) via prospective field based studies; (3) The frequency, distribution and magnitude of impacts to the head in senior amateur rugby union players (Chapter 7) via a prospective field based study; and (4) The need for a consensus for the reporting of impact biomechanics (Chapter 8) via a retrospective review of literature and analysis of head impact data using different thresholds.

**Epidemiology of concussion in New Zealand**

The epidemiology of sport-related concussive injuries and the associated costs in New Zealand requiring medical treatment were examined (Chapter 3) using the New Zealand national Accident Compensation Corporation (ACC) injury data for the period 2001 to 2011. The concussion history of a group of amateur rugby league players was examined through a retrospective review in a zonal region of New Zealand (Chapter 4).

***Sport-related concussions in New Zealand: A review of 10 years of Accident Compensation Corporation moderate to severe claims and costs***

In Chapter 3, data were analysed by seven sporting codes, gender, ethnicity and age. In the United States of America 1.6 to 3.8 million sports related concussions occur annually[358] with a cumulative (direct and indirect) cost estimated to be US$56 billion.[358] Most sport-related concussions occur in sports that involve physical contact between participants.[34] However, rates are also high for non-contact sports such as baseball and volleyball.[506] Despite the increasing number of international studies on sport-related concussion, there is a paucity of studies describing sport-related concussion in New Zealand sports-participants.

It conducting this study over the 2001-2011 reporting period, a total of 20,902 claims costing $NZD 16,546,026 were recorded of which 1,330 (6.4%) were moderate-to-severe claims (MSC). The mean yearly number and costs of MSC's were 133 ±36 and $1,303,942 ±$378,949. Rugby union ($6,252,870) and rugby league ($4,572,625) recorded the highest total costs over 2001-11. Rugby league (92.2%; $25,545) and netball (86.6%; $11,371) recorded the highest percentage of sport code total costs and mean costs per ACC claim.

Males participating in rugby union recorded the highest total costs for MSC claims ($5,644,655). Males participating in rugby league recorded the highest mean costs per MSC claim ($26,182). Females participating in netball recorded the highest total ($833,530) and mean costs per female MSC claim ($12,080). The finding that rugby union recorded the most MSC claims was not unexpected as this is the national game in New Zealand.[211] Previous international studies have identified that ice hockey[235] and the national football league (NFL)[371] have some of the highest incidences of concussion of all sports but these sports have low participation levels in countries such as New Zealand. MSC claims accounted for 6.4% of the total sport-related concussions for the seven sports surveyed but accounted for 79.1% of total costs. This indicates that although sport-related concussions may be minor in severity, the related economic costs attached to a sport-related concussion with ongoing symptoms can be high.

Rugby league had the third highest number of claims (n=179) for reported concussions over the ten years. However, the overall knowledge level of concussion identification and management was only 42% for rugby league team coaches and trainers in New Zealand which was lower than other studies reporting concussion knowledge of team coaches (62%[601] to 84%[477]). Of 95 rugby league coaches and team managers, 26% reported they would not remove a player with symptoms of concussion, 20% would return a symptomatic player to participation and 39% reported that concussion only occurred when a player lost consciousness.[331]

Over 2001-11 period, New Zealand Māori recorded the highest mean costs per MSC claim for rugby league ($43,604) and rugby union ($14,186). People participating in rugby league activities in the 30-39 age group recorded the highest total ($2,268,398) and mean cost ($48,211) per MSC claim. This may be reflective of the nature of rugby league when compared with other sporting activities. For example, the differences in the types and numbers of tackles between the two sports may expose players in rugby league to more head impacts resulting in more severe head trauma and therefore more cost per claim. Studies reporting on rugby union[516] and rugby league[332] tackles have reported that contact with the head during the tackle occurred at a rate of 0.6 per 1,000 tackles in rugby union[516] but at a rate of 89.9 per 1,000 tackles in rugby league.[332] This higher rate of contact with the head in rugby league may be reflected in the higher costs recorded in the current study.

The average cost per claim for a moderate to severe concussive injury varied by sports code, ethnicity, gender and age over a 10 year period for seven sporting codes in New Zealand. Māori rugby league males aged between 30 and 39 years, female players and rugby union players need to be targeted for injury prevention initiatives. A wider understanding of concussion identification for team coaches and first aiders and removal from play for assessment at all levels of participation may assist in the identification of concussive injuries.

***Concussion in amateur rugby league players in New Zealand: A review of player concussion history***

In Chapter 4 a retrospective analysis was undertaken on all medical pre-competition questionnaires completed during the 2010 to 2012 competition periods by amateur rugby league players. Players undertaking any sporting activity are at risk of receiving a concussive event.[434] The risk of receiving a concussive event is reported to be increased in players with a history of a previous or multiple concussive events.[291] A way to assist the team medical personnel to manage this risk is to record the players individual concussion history. Often this requires self-disclosure by the player or the management of a concussion history by the team medical person through sideline and post-match examinations. Recently there has been an increase in published research on sport-related concussion relating to the identification, assessment and management in all sporting environments.[123, 391]

Concussion has become one of the most troublesome injuries facing the sports medicine professional,[347] especially with regard to the early identification of concussive signs and symptoms and appropriate concussive management facilitation.[273, 409] Amateur sports such as rugby league in New Zealand do not have the resources available for qualified medical personnel to attend every match.[331] As such it is often the coach or team manager, if the team has one, to make decisions with regard to player welfare including concussion identification and management. The low level of concussion awareness amongst team management has been previously reported[331, 351, 477, 601, 615] and highlights the need for concussion education for all people involved in the management level of sports participation

A total of 213 (mean ± *SD*: age, 19.2 ±4.4) amateur rugby league player concussion questionnaires were reviewed. There was an average of 4.0 ±2.6 concussive injuries per participant in the previous two years and an average of 5.0 ±4.6 concussive injuries per participant the period preceding this. A total of 7.5% participants saw a medical doctor for their concussion; 5.2% completed the required three week return-to-play; and 2.8% reported seeing a medical doctor for medical clearance. No under 15 yr. old player reported seeing a medical doctor or having a stand down period for return to play.

A previous study[409] identified that players will not report concussive symptoms because they do not think it is sufficiently serious enough to necessitate reporting. A lack of knowledge of the risks and potential consequences of concussion was reported to be the most common reason for the non-reporting of a possible concussion.[409] It was also identified that more than a third of players failed to report a concussive injury as they did not recognize that they had sustained a concussive injury based on their symptoms.[409] This may have been the case in the current study with 7% of players showing signs of a concussive event.

Players across all age groups were administered the same questionnaire in different settings where they were gathered for the respective age group competition meetings. An unexpected finding was that 7% of all players had active concussive symptoms and was a cause for concern. None of the players identified had seen a medical professional for their symptoms nor reported these symptoms to anyone as they felt this was all part of “playing the game”. All of these players were advised to seek further medical assistance and required to provide a full medical clearance before they would be allowed to return to match activities. One player was subsequently medically cleared two days later by his own medical practitioner as he “had no

signs of a concussion". A lack of knowledge of the risks and potential consequences of concussion was reported to be the most common reason for the non-reporting of a possible concussion.

**Identification and assessment of concussion**

The identification and assessment of concussion was examined with the use of the King-Devick (K-D) saccadic reading test and the Sports Concussion Assessment Tool version 3 (SCAT3) in amateur senior rugby union (Chapters 5 and 6) and amateur senior rugby league (Chapter 6) domestic players.

***Concussions in amateur rugby union identified with the use of a rapid visual screening tool***

In Chapter 5, a prospective observational cohort (field based) study was conducted on a premier club level amateur rugby union team during the 2012 competition in New Zealand. Rugby union is one of the most popular contact team sports in the world with nearly 200 countries affiliated to the International Rugby Board.[55, 303] Rugby is played at the professional, amateur and junior levels by both males and females.[56] Rugby also has a modification of the match rules for the different age groups and competition types (i.e. international, junior, sevens and masters' competitions) that are competed in.[56] As with any contact or collision sport there is a risk of an injury occurring due to the number of collisions and contacts that are integral to the game.[56] Studies reporting on concussion injuries in rugby union have identified that the incidence was relatively low with reports varying from 0.2[119] to 4.3[54] per 1,000 playing hours, and 3.8[397] to 5.7 [430] per 1,000 athlete exposures.

Every player in this study completed a pre-competition questionnaire on concussion history, a baseline Post-Concussion Symptom Scale (PCSS) and two trials of the K-D before they participated in any match activities. For players reporting a concussion in the previous three years there was an average of 4.0 ±2.8 concussions per player. The KD has been reported[203] to be a practical sideline screening tool that is quicker than other concussion screening tools such as the Immediate Post-Concussion and Cognitive Testing (ImPACT), Cognitive Status Sport (Cog Sport),[88] the Standardized Assessment of Concussion (SAC)[410] and the Sports Concussion Assessment Tool 2 (SCAT2).[425] Although these screening tools are useful in assessing for suspected concussion they do not assess eye movements or brain stem function well.[271] The KD does however, test for impairment of eye movement, attention, language and other areas that correlate with sub-optimal brain function that may occur following a concussive episode.[271]

There were 22 concussive incidents recorded over the duration of the competition (46 per 1,000 match hours). Five concussive incidents were witnessed (11 per 1,000 match hours) and 17 unrecognised concussive incidents were identified with the K-D (37 per 1,000 match hours). Witnessed concussions recorded, on average, a longer K-D on the day of injury (5.5 ±2.4 s) than unrecognised concussions (4.4 ±0.9 s) when compared with their baseline K-D. The internal consistency reliability of the three K-D cards was acceptable (α=0.72 for card 1; α=0.78 for card 2; α=0.76 for card 3) between card scores and total time score at baseline. The internal consistency reliability of the PCSS was excellent (α=0.98).

Unwitnessed concussions identified with the K-D recorded on average fewer symptoms (6.8 vs. 16.1), lower symptom severity (8.8 vs. 42.4) better balance examination (5.2 vs. 12.8) and better immediate (13.7 vs. 13.0) and delayed (4.2 vs. 2.6) memory scores than witnessed concussions. Most, but not all, players with an unwitnessed concussive injury had their K-D equal to, but not significantly faster than their baseline K-D by day 14 of the required stand-down period (-0.8 ± 1.4s). The current rate (46 per 1,000 match hours) is a ten-fold increase in the previously reported concussion injury rate.[54, 432] If the present finding is any indication that there is a ratio of 3.4 identified concussions for every witnessed concussion occurring then studies will be under-estimating the incidence of concussions.

The K-D was useful in rapidly identifying players that had a witnessed concussive incident by being able to provide instant feedback to the player and to team management. The K-D was able to identify players that had not shown, or reported, any signs or symptoms of a concussion but who had meaningful head injury. An interesting finding was that players who recorded a slower K-D, and were treated as an unwitnessed concussion, had changes in SCAT2 but returned to their baseline result in a shorter period than players with a witnessed concussion. The ease-of-use of the K-D made it more acceptable to team management and players, as it provided immediate feedback to the player and coaching staff.

***The King-Devick test was useful in management of concussion in amateur rugby union and rugby league in New Zealand***

In Chapter 6 a prospective observational cohort (field based) study was conducted on a club level senior amateur rugby union team (n=36 players in 2012 and 35 players in 2013) and a rugby league team (n=33 players in 2014) during competition seasons in New Zealand. Originally developed as a reading tool to assess the relationship between poor oculomotor functions and learning disabilities, the K-D test utilises a series of charts of numbers that progressively become more difficult to read in a flowing manner [352]. The K-D test requires eye movements, language function and attention in order to perform tasks reflective of suboptimal brain function in hypoxia, [570] extreme sleep deprivation, [120] Parkinson's [370] and concussion [134, 202, 203, 206, 314, 317, 367, 395]. Previous studies[202, 203, 314, 317, 367] have reported K-D test times of participants with a concussive injury to be on average 5 s slower (range 3.2 s to 18.0 s) than their baseline.

Several sports, such as boxing and mixed martial arts [202, 367], professional ice hockey [206], representative rugby league [317] and domestic rugby union, have utilised the K-D and identified unwitnessed concussive events. These studies highlight the potential of the K-D test to detect sub-concussive impacts that may accumulate over a period and can lead to neurological changes [317]. The K-D test has been recommended as a sideline test to enhance the detection of players with a concussive injury in conjunction with other concussion tests [142, 395]. Recently it was identified that larger scale research over a longer period of time may provide increased validity of the K-D test as part of a continuum of concussion assessment tools [395, 603]. As such, the purpose of this study was to examine the worth of the K-D test as part of a sideline assessment process of players participating in contact sport over a three year period.

All 104 players enrolled in this study completed two trials 10 minutes apart of the K-D at the beginning of their competition season. Concussions (witnessed or unwitnessed) were only recorded if they were formally

diagnosed by a health practitioner. A total of 52 (8 witnessed; 44 unwitnessed) concussive events were identified over the duration of the study resulting in a concussion injury incidence of 44 (95% CI: 32 to 56) per 1,000 match participation hours. There was a six-fold difference between witnessed and unwitnessed concussions recorded. There were observable learning effects observed between the first and second K-D test baseline testing (50 vs. 45 s; z=-8.81; $p$<0.001). For every 1 point reduction in each of the post-injury SAC components there was a corresponding increase (worsening) of K-D test times post-match for changes in orientation (2.9 s), immediate memory (1.8 s) concentration (2.8 s), delayed recall (2.0 s) and SAC total score (1.7 s).

The changes observed are likely to be reflective of the anatomical aspects that the K-D and SAC tests capture.[206] Pathways for saccades, or fast eye movements, in the brain are widely distributed and involve several areas of the brain such as the frontal eye fields, supplementary eye field, dorsolateral prefrontal cortex, parietal lobes and deeper structures including the brainstem, necessitating the involvement of several cortical areas in the production, and regulation, of saccades.[206] As saccades can be used to assess cognitive domains such as attention, spatial and temporal orientation and working memory, injuries involving the disruption of the areas involved in saccades production and regulation can result in changes in these cognitive domains.[206]

The rate of undetected concussion was higher than detected concussions by using the K-D test routinely following matches. Worsening of the K-D test post-match was associated with reduction in components of the SAC. The appeal of the K-D test is in the rapid, easy manner of its administration and the reliable, objective results it provides to the administrator. The K-D test helped identify cognitive impairment in players without clinically observable symptoms.

**Head impacts in rugby union**

The magnitude and frequency of impacts were measured with an instrumented mouthguard (Chapter 7) in amateur senior rugby union players over a domestic competition season. Impacts thresholds were reviewed (Chapter 8) and the identification of common reporting modalities was identified for future studies reporting on impact biomechanics.

***Instrumented mouthguard acceleration analyses for head impacts in amateur rugby union players over a season of matches***

In Chapter 7 a prospective observational (field based) study was undertaken to record the magnitude and frequency of impacts to the head with moulded instrumented mouthguards worn by thirty eight premier amateur senior rugby players participating in the 2013 domestic season of matches.

The incidence of concussion in sports provides a unique opportunity to collect biomechanical data on head accelerations involved in mild traumatic brain injury (mTBI).[528] By instrumenting athletes with accelerometer instrumented headbands[457] and helmets,[524] head acceleration data have provided proof of concept and laid the groundwork for future research.[141, 528] By quantifying the head impacts experienced by professional football players, the National Football league (NFL) reconstructed injurious game impacts based on video

analysis.[498] By recreating 31 impacts, 25 of which were concussive, injury risk curves were developed for mTBI's.[528] Nominal values for an injury were a peak linear acceleration of 98*g* and peak rotational acceleration of 6,432 rad/s$^2$.[498] Although this study did characterize concussive biomechanics, the injury risk curves likely over-estimate the risk as head impacts that did not result in concussions were not accounted for.[530]

Non-helmeted sports have also utilised accelerometers in measuring impacts from sports participation. Heading the ball by female soccer youths resulted in peak accelerations of 63*g* and 8,869 rad/s$^2$.[261] No concussions were reported,[261] as no injury nor injury risk was assessed, even though some of the rotational accelerations were within the nominal values for an injury to the head when compared with NFL data[498] and injury risk tolerance levels.[632] Although data are accumulating for soccer and NFL there are no published data for non-helmeted collision sports such as rugby union.

A total of 20,687 impacts >10*g* (range 10.0-164.9*g*) were recorded over the duration of the study. The mean number of impacts per player over the duration of the season of matches was 563 ±618 resulting in a mean of 95 ±133 impacts to the head per player, per match over the duration of the season of matches. The mean number of impacts per player over the duration of the season of matches was 564 ±618 resulting in a mean of 95 ±133 impacts to the head per player, per match over the duration of the season of matches. The mean impacts per match were 1,379 ±578 resulting in a mean of 77 ±42 impacts to the head per player position, per match. Forwards sustained more impacts than backs (RR: 10.89; [95% CI: 1.85 to 1.93]; $p$<0.001) over the duration of the season of matches.

The back left side of the head sustained the highest linear acceleration (27 ±4*g*) for player position, for the forwards and per player. The top right side of the head sustained the highest linear acceleration for the backs (28 ±11*g*). The top right side of the head sustained the highest rotational accelerations (6,160 ±2,908 rad/s$^2$) for player positions and this was similar for backs (7,273 ±3,379 rad/s$^2$) and per players (5,333 ±3,673 rad/s$^2$).

Players in our study recorded a mean of 77 impacts to the head per game or 1,379 impacts per player, per season of matches which was higher than any other published study to date. American high school football studies had a mean impact per player of 16 to 29 per game or 520 to 652 per season.[52, 538] American collegiate football impacts studies varied from 9 to 13 impacts per session (practice and games combined) or 414 to 1,400 impacts per season (practice and game combined).[109, 141, 538]

The mean linear acceleration value of impacts (22*g*) measured over the course of the season of matches was higher than the mean value reported for studies in youth football (15*g*),[115] but similar to high school (21-26*g*),[49, 50, 150, 596] some collegiate football (18-27*g*),[28, 528] but lower than female youth soccer (25-63*g*),[261] some American collegiate (32*g*)[141] and professional American football (60*g*)[497] mean values. The mean rotational acceleration for impacts (3,990 rad/s$^2$) measured over the course of the season of matches was higher than American youth (671 rad/s$^2$),[115] and American high school football (973-1,711 rad/s$^2$),[49, 50, 596] American collegiate football (848-1,107 rad/s$^2$),[28, 528] subconcussive American collegiate impacts (1,230 rad/s$^2$),[531] but less than female youth soccer (8,889 rad/s$^2$),[261] concussed American collegiate players (5,022 rad/s$^2$),[531]

non-concussed (4,043-4,446 rad/s$^2$)[497] and concussed professional American football reconstructions (4,870-7,173 rad/s$^2$).[497]

The distribution of impacts varied by location, player position, player group and by players. Impacts were more commonly recorded on the side of the head (n=9,232; 45%) with the right side (23%) more commonly impacted. The distribution of the impacts recorded is similar to male collegiate ice hockey (30%)[616] but different from American high school and American collegiate football where up to 45% of impacts occurred in the front.[49, 50, 449, 596] Impacts to the top of the head (n=539; 3%) were less commonly recorded. Backs recorded the highest mean linear (28*g*) and rotational (7,273 rad/s$^2$) accelerations on the top-right side. This was similar to high school and collegiate football where impacts to the top of the head varied from 19 to 38*g*.[49, 50, 449, 596] Differences between the impact locations could be related to the different sporting codes and the protective equipment utilised.

The relationships between linear and rotational acceleration and injury tolerance level for impacts have been previously described.[49, 52, 254, 498] The injury tolerance level for impacts are impacts greater than 95*g* and 5,500 rad/s$^2$ and impacts to the front, top or back of helmets,[49, 52, 254, 498] but this is primarily limited to helmeted sports studies. There were 181 impacts greater than 95*g* and 4,452 impacts greater than 5,500 rad/s$^2$ recorded in our study indicating that there are some impacts above the injury tolerance level for impacts of concussion occurring in rugby union. The number of impacts above these levels varied by player group, player position and players enrolled in the study. This was similar for impacts in the low, moderate and severe injury risk data acquisition limits for both linear and rotational accelerations with the majority of these being recorded in the low injury severity limits previously identified.[264, 478, 632]

Monitoring head impacts in contact sports has been suggested to assist in the identification of high risk events and to alert medical personnel of the need to perform a concussion evaluation of the identified player.[42, 240] Monitoring may reduce the incidence and severity of concussions by reducing subsequent impacts that may cause impact brain injury.[240]

***The influence of threshold on the reporting of impact data in contact and collision sports: Consensus needed***

In Chapter 8 a review was undertaken on the impact thresholds utilised to report data in sport from accelerometer systems. Head impact data are essential to help understand the biomechanics of head injury to develop potential injury prevention strategies. There are currently no standard for reporting head impact data to enable inter-study comparisons. Currently the use of accelerometers may not necessarily provide the meaningful inter-study comparisons that are sought due to data collection, processing and methodologies not being standardized.[495] Several studies have undertaken to report impacts to the head in sports such as American football,[28, 29, 49, 50, 52, 53, 110, 111, 115, 141, 261, 265, 449, 464, 528, 531, 538, 596, 626] ice hockey,[451, 452, 521] soccer[261] and rugby union[330] but these have all utilised different data impact thresholds and reported different results. As such, studies utilising different impact thresholds have proposed varying conclusions based on the methodological and reporting approaches undertaken.

This study was undertaken as there are currently no standards identified for the reporting of head impact data in sports to enable comparison between studies. As a result of this, the conclusions drawn from data using different impact thresholds have resulted in substantially different conclusions.

To outline head impact thresholds, and their potential effects on impact data in sport, calculations were undertaken on the number of impacts reported based on the percentage of impacts removed compared with a 10*g* impact threshold using data from 38 senior rugby players in New Zealand. Of 43 studies identified, 16 (37.2%) reported impacts using >10*g* threshold. Application of the varied impact thresholds resulted in 20,687 impacts >10*g*; 11,459 (44.6% less), impacts >15*g*; and 4,024 (80.5% less) impacts >30g. Studies reported descriptive statistics as mean (±SD), median, 25$^{th}$ to 75$^{th}$ interquartile range, and 95$^{th}$ percentile.

Based on the differences observed in this study, at the 14.4*g* threshold there could have been as many as 42% of the impacts recorded not being reported. As a result, studies[28, 29, 44, 47, 49, 50, 84, 116, 150, 531, 596, 626, 631] using impact thresholds above 10*g* may have removed 2,100 to 206,573 impacts. The discussion surrounding subconcussive impacts has become popular.[13, 43, 132, 257, 398, 566] Initially the term subconcussive impact described an impact that did not result in severe, noticeable symptoms, especially loss of consciousness.[132] However, recently subconcussive is a term utilised to describe an asymptomatic non-concussive impact to the head.[13, 43, 257, 398, 566] The issue relating to the effects of subconcussive impacts is controversial as researchers and clinicians are divided on the true effects.[21, 209, 257, 435, 455, 581]

The Head Impact Criterion (HIC) and Gadd Severity Index (GSI) are the most commonly utilised head injury assessment functions, particularly in safety standards,[632] however this was not reflected in the sport head impact studies reviewed. Based on the Wayne State University tolerance curve,[604] the HIC and GSI criteria do not account for the complex motion of the brain, or the contribution of resultant rotational acceleration to the head.[471, 472, 632] The inclusion of these parameters may be more historical and provide the ability for inter-study comparisons with previous studies. However, as HIC and GSI are not commonly reported, the inclusion of these parameters in future studies needs to be standardised.

The most commonly reported head impact biomechanics were the resultant linear and rotational accelerations, although not all studies reviewed reported both. Some studies reported only resultant linear accelerations[53, 141, 174, 254, 257, 403, 449, 538, 626] or resultant rotational accelerations[168, 531] which may limit their inter-study comparison usability. It has been suggested that both resultant linear and rotational accelerations should be reported with head impact metrics.[481] More recently[530, 596] resultant linear and rotational acceleration results have been combined into a risk weighted exposure (RWE) metric. This metric can be beneficial for fully capturing the linear ($RWE_{Linear}$), rotational ($RWE_{Rotational}$) and combined probability (from linear and rotational) ($RWE_{CP}$) of the risk of a concussion as it accounts for the frequency and severity of each players impacts.

More than half (52%) of the studies[26, 28, 49, 52, 53, 141, 150, 168, 254, 265, 330, 398, 403, 449, 451-453, 464, 478, 498, 528, 596, 626, 631, 632] reported the impact biomechanics data as mean ± standard deviation (±SD). Some studies[28, 150, 464, 596, 631] (10%) also reported the head impacts as median, but not all[464, 631] (4%) included the interquartile ranges (IQR) for the data. Of the studies that reported the impact biomechanics by the median, only 7% reported

the IQR. Most of the studies reporting the median also reported the 95th percentile of the impacts. Other data reporting methodologies utilised within the data sets reviewed were the median of 95th percentile,[110] the 98th,[150, 241] 99th,[150, 241] and 99.5th[150] percentiles.

In addition to the impact biomechanics being presented by various methodologies, some studies[49, 52, 254, 265, 330, 478, 632] also incorporated impact tolerances and impact severity levels. The use of this data may be important if a risk assessment is undertaken for possible long term implications from repetitive head impacts (RHI). Recently in a small sample[26] of collegiate players with no reported concussions after a season of American football, there were white matter changes that correlated with multiple head impact measures. Participants with more than 30-40 RHI's with peak rotational accelerations >4,500 radians per second per second (rad/s$^2$) per season ($r$=0.91; $p$<0.001), and more than 10-15 RHI's >6,000 rad/s$^2$ ($r$=0.81; $p$<0.001), were significantly correlated with post-season white matter changes.[26]

The issues identified with the use of accelerometers for physical activity[495] may be similar for head impact biomechanics and include affordability of the accelerometers,[495] and the administration burden[495] to the participants and researcher(s) given post data collection analysis. The choice of accelerator brand,[493] generation[62] and firmware version,[293] wearing position[630] based on the sports code requirements (i.e. helmet mounted vs. headband mounted vs. mouthguard embedded vs. patch), specifics of the research being undertaken such as the epoch length[151, 479] (match vs. training vs. combined), data imputation methods,[364] dealing with spurious data[161] and the reintegration of smaller epochs into larger epochs[307] are all considerations for use of accelerometers.

The differing descriptive statistics utilised for reporting head impacts in sports limits the use and availability of inter-study comparisons. A consensus on methods of data analysis, including the thresholds to be used in sports impact assessment is needed. Based on the data available to date, the 10*g* threshold is the most commonly reported impact threshold. Validation studies are required to determine the best threshold for impact data collection in sport. Until in-field validation are completed, it is recommended that data should be reported as mean ± standard deviation, median and interquartile ranges using the 10*g* impact threshold.

**Thesis limitations**

The quality of the epidemiological studies contained in this thesis were dependant on the data quality utilised for analysis.[217] In Chapter 3, the data provided for the analysis was from the ACC database and is dependent on several data input factors:

(1) The correct data code being used on the data collection sheet.
(2) People going to a registered health professional for treatment of their injury.
(3) Underreporting of costs due to patients undertaking private medical care.
(4) People making a claim for the injury (there is no time limit on when a patient can make an acute injury claim to ACC).[217]

The data retrieved from the ACC database were protected to ensure client confidentiality by limiting access to low level results of under four injury claims. Consequently any data that was less than, or equal to, three

injury claims were rounded to represent three claims only. The net result of these limitations may have seen a downwards bias in the injury entitlement claim incidence and, some of the calculations were not equal depending on the way the data were rounded.

Another limitation to Chapter 3 was that the data were limited to just the ACC database. Currently there is no other recording system available for analysis of concussion injuries in areas such as:

(1) Numbers participating in sporting activities.
(2) Age of players participating.
(3) Identification of the ethnicity of players.
(4) Number of matches played and trainings completed enabling match and training exposure hours.
(5) How the injuries occurred.
(6) What participation level the injuries occurred in.
(7) Whether the injuries occurred as a result of match or training activities.
(8) At what stage of the match or training activity the injuries occurred.

In conducting a survey of rugby league players (Chapter 4) there were some limitations identified with the use of a survey questionnaire. In particular:

(1) Some players did not return the questionnaires and these were excluded from the data set;
(2) Some of the questionnaires were incomplete and required contacting these players (where the contact details were available) for the information to be completed, and in the case where the player was uncontactable, these were excluded; and
(3) Players who were reviewed in consecutive years had only their first questionnaire results enrolled in the study as there had been player education undertaken when these were first screened.

The use of the King-Devick required that all players were assessed with a baseline and were available for post-match assessment. In conducting the research for Chapters 5 and 6 the limitations identified were:

(1) Not all players made themselves available post-match for the evaluation via the King-Devick test. Some players actively avoided being tested and this necessitated assessments at the first training session following the match; and
(2) Some players declined the invitation to participate and not all members of the amateur senior rugby team were enrolled.

In conducting the observational study recording the magnitude and frequency of head impacts (Chapter 7) there were some limitations identified. In particular:

(1) The effects of saliva from the mouth of some players resulted in some of the activation contacts in the mouthguard becoming inactive and the impact data were not able to be downloaded. As a result the data reported were incomplete and the number of impacts would be more than have been reported;
(2) The correlation of the All-In-Mouth mouthguard with laboratory head-forms is good (peak linear acceleration (PLA) ($r^2$=0.937), peak rotational velocity (PRV) ($r^2$=0.966), and peak rotational

acceleration (PRA) ($r^2$=0.882) when compared with the head's centre of gravity)[63] but the impact measurements should be assumed to have some form of error that is dependent on impact conditions, the measure of interest and the variability tested;

(3) The percentage of impacts identified at the 10*g* inclusion limit that were able to be visualised by video review and analysis was 65% to 85% of the total impacts recorded per match.

**Model of concussion**

In undertaking this research, it soon became apparent that there was not a model in which to base the identification, assessment and management of concussion. The initial concept for this thesis was based around the recursive, dynamic model of etiology of sports injury[446] where it has been reported that if a player is injured they either undergo recovery towards adaption or they do not recover and are removed from participation. In the case of sport-related concussion these injuries may not be identified or reported and the participants are re-exposed to the same extrinsic factors that lead to the first concussion. In attempting to research sideline assessment tools for the identification of concussion and removal from play the complexity of the management of concussion became obvious.

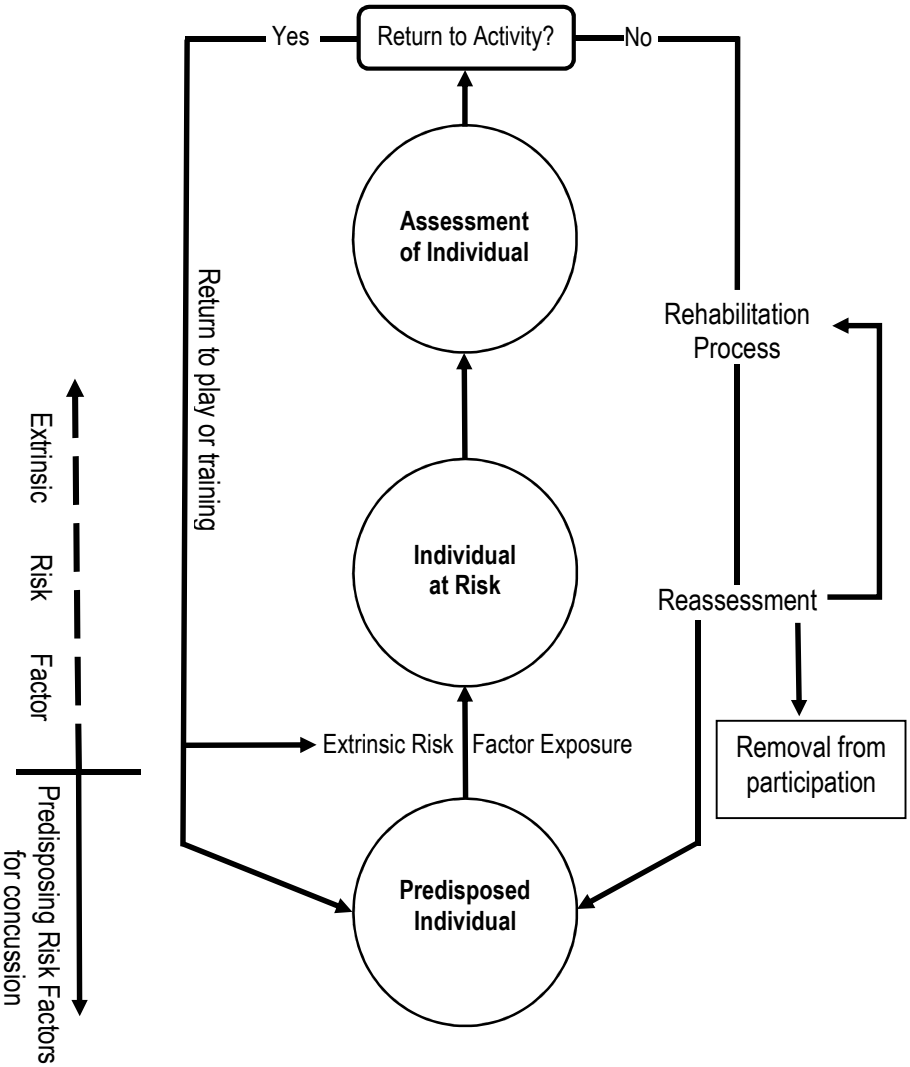


**Figure 9:** The cyclic model for concussion management.

Based on the recursive, dynamic model of etiology of sports injury,[446] and incorporating the neurobiopsychosocial model for concussion,[407] the cyclic model for concussion management was developed to standardise the identification, assessment and management of concussion (see Fig 9).

Similar to the recursive, dynamic model of etiology of sports injury,[446] the cyclic model for concussion management highlights how in the case of sports participants, athletes can repeatedly go through the cycle depending upon the outcome for return to play. If the player is assessed as having a concussion then they undertake a rehabilitation process and move towards a reassessment. If, during the reassessment, they are identified as still having a concussive injury they can either recycle through the rehabilitation process and undertake another reassessment or be removed from participation. If a player is assessed to have no active signs of concussion, then they can return to the activity or are able to return to training activities.

Although the athlete may not be obviously injured with a concussive injury, the risk of injury occurring will depend upon the amount of extrinsic exposure that has occurred from activity participation combined with the aspects of the neurobiopsychosocial model and intrinsic risk factors that the athlete may already have. The combined value of these factors will vary by individual athlete and the extent of this predisposition to a concussive event is, to date, unknown.

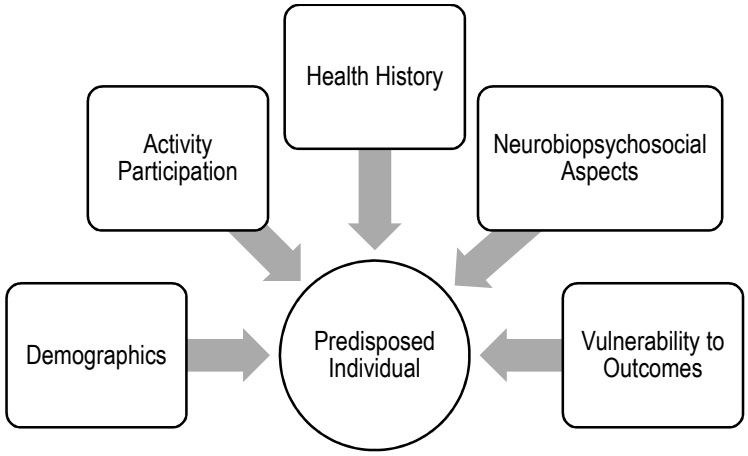


**Figure 10:** Possible predisposing influences for a head injury of the individual.

This predisposition (see Fig 10) may include, but is not limited to, aspects such as the co-modifiers identified by the Concussion in Sports Group,[392, 423, 543] neurobiospychosocial aspects (e.g. history of attention deficit disorder or learning disability; tress and resilience Levels, etc.),[407] demographics (e.g. age,[407] gender,[99, 135, 407, 509] race[407]), health history (e.g. prior concussion history, co-morbid conditions, etc.),[407] vulnerability to outcomes (e.g. physiologically-compromised brain and heightened vulnerability to repeat injury)[407] and possibly other factors[543] yet to be identified such as genetic predisposition to concussion.[167, 474]

When the predisposed individual becomes exposed to the extrinsic risk factors they become at risk of incurring a concussive injury. The extrinsic risk factors are multifactorial and, in terms of sports participation, may include all or some of the factors previously identified (see Table 1 in Chapter 1). These risk factors may exert an influence on the predisposed individual increasing the risk of an injury occurring. In terms of concussion, this may be in the form of repetative head trauma[407] (e.g. subconcussive impacts). The impacts

may be cumulative.[291, 464, 585] with possible longitudinal effects[436, 437, 572, 573] but, to date, there have been no direct causal effects identified.[315]

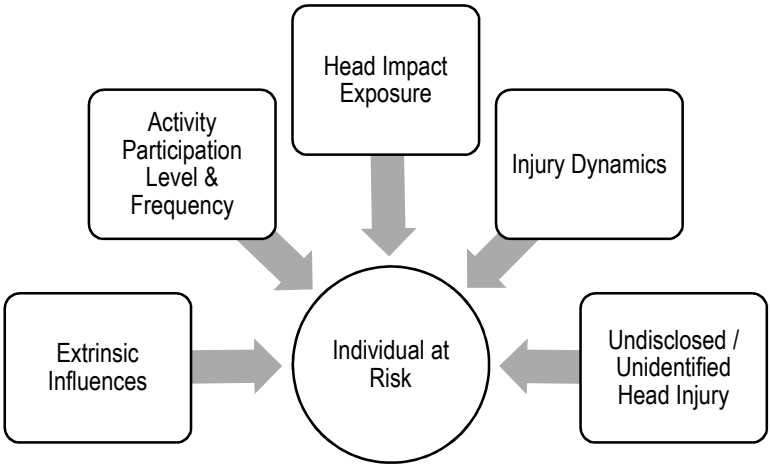


**Figure 11:** Possible risk factor influences for head injury of individual.

In addition to the extrinic risk factors the individual is exposed to, there are other aspects that can influence the risk (see Fig 11). These include, but are not limited to, activity participation type/nature, level and frequency (e.g. one exposure vs multiple exposures per day, etc.),[28, 84, 407, 464] head impact exposure (e.g. cumulative subconcussive impacts, previous head injuries etc.),[28, 84, 407, 464] injury dynamics (resultant linear and rotational forces, impact location, etc.)[50, 241, 407, 411, 530, 616] and undisclosed or unidentified head injury (e.g. risk of second impact syndrome).[38, 73, 407, 436, 543]

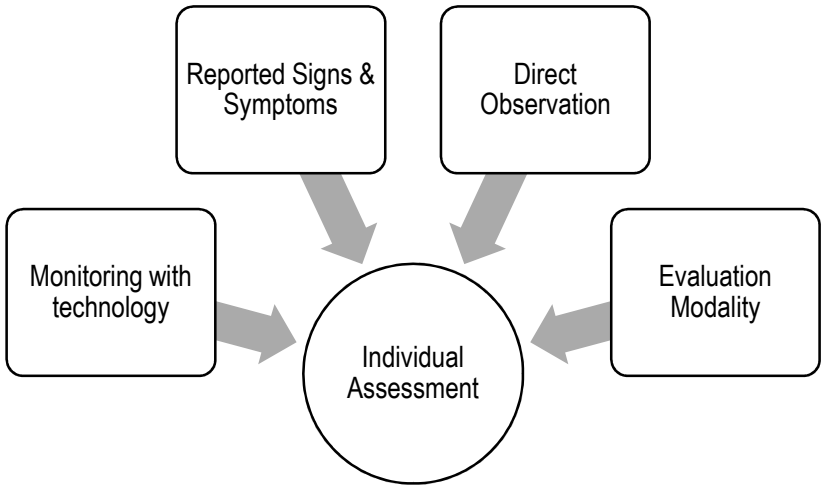


**Figure 12:** Possible influencing factors on the assessment of the individual for a head injury.

As seen in Figure 12, factors that may impact on the decision as to when assessment should occur may include direct observation (e.g. showing signs and symptoms, loss of consciousness etc.), reported signs and symptoms (e.g. 'bell rung', 'shaking out cobwebs', 'seeing stars', etc.),[4, 247] assessment of the athlete as part of sideline or post-match routine screening (King-Devick saccadic reading test,[139, 204, 205, 367, 462, 480] SCAT3,[255, 423] ImPACT®,[588] etc.), identification of at-risk aspects through monitoring technology (e.g. HITS,[52,

[616] XGuard,[330] XPatch[467, 629] etc.) or through the use of other possible clinical evaluation modality.[10, 30, 37, 239, 515] The assessment of the individual at risk may occur on the sideline in the sporting environment during the sporting activity, after the activity has occurred, as part of a pre-organised screening program, as a result of the individual presenting with symptoms or as an incidental finding following assessment of other injuries that have occurred.

Once the individual assessment is completed, the next phase of the cyclic model is the return to activity. Individuals with changes identified through the assessment process, proceed through to the rehabilitation process which ideally should be individualised.[144] The ultimate goal of rehabilitation is to undertake a graduated stepwise process[65, 78, 79, 117, 130, 144, 163, 262] and return to their activity symptom free.[423] In the case of the individual not passing reassessment criteria they can either be returned to the rehabilitation phase,[79, 144] and in the case of sporting activities this may involve being removed from the activity altogether.[72, 74, 414, 415, 423] The cyclical aspect of the reassessment and rehabilitation has no set time frame,[144] incorporates academic,[122, 131, 399] employment[66] and sporting[65, 78, 79, 163, 262, 407, 465] restrictions and is dependent on several factors. These include, but are not limited to, psychological function (post-injury comorbidities, premorbid level of functioning, etc.),[14, 92, 248, 407] environmental factors (e.g. social support, life stressors, etc.),[407] and motivational factors (e.g. expectations of self and others, secondary gain, etc.).[407] All these aspects may have some impact on the stage in which the individual moves through or becomes stagnated within.

In terms of the individual who is cleared to return to their activity they can either recycle back into the activity or return to a pre-activity stage. Although the individual assessment has identified no concussive injury, they may be at an increased risk of a concussive injury if they are repeatedly exposed to subconcussive impacts, or deny any concussive symptoms and have not reported them.

Further research is warranted on the cyclic model of concussion management in terms of interventional modalities being identified for future management of concussive injuries, the prevention of concussive injuries and the identification through newly developed tools for the identification of concussive injuries.

**Future directions**

This thesis reported several aspects of epidemiology, identification and assessment of concussions in amateur rugby union and rugby league. In addition this thesis also explored the frequency, duration and magnitude of impacts to the head in amateur senior rugby union league players. The published articles resulting from this thesis, combined with the increased public, media and sports medicine awareness of these injuries has resulted in the development of strategies for the protection of rugby union and rugby league participants.

Future research should focus on:

***The King-Devick (K-D) saccadic reading test***

1. The majority of studies have reported on the use of the K-D in male orientated sporting activities. No studies, to date, have reported on the assessment and identification of concussions in female orientated sporting activities. It is recommended that future studies consider:

   a. The assessment, identification and management for sports-related concussion in female sporting activities; and
   b. The effects of fatigue on the results of the King-Devick test in female sports participants.
2. The K-D test is designed to be age appropriate with participants aged 5 to 7 years reading only test card I, ages of 8 and 9 reading test cards I and II and over the age of 9 reading all three test cards. To date there have been no published studies reporting on the use of the K-D test with participants in the youth age groups for rugby union. Recently a study[335] has been published reporting on the use of the K-D in junior (U11) rugby league participants. It is recommended that future studies consider:
   a. The use of the K-D test in the assessment and management of concussions recorded in youth sporting activities;
   b. The effects of fatigue on youth sports participants and changes in the K-D test; and
   c. Differences in the results of the K-D in male and female youth sports participants.
3. The K-D test has reported to have significant correlations ($p<0.0001$) with the visual motor speed (VMS), reaction time (RT), verbal memory (VEM) and visual memory (VIS) of the Immediate Post-concussion Assessment Cognitive Test (ImPACT®)[588] computerised concussion evaluation system. No other neurocognitive computerised concussion evaluation system has undergone the same correlation comparisons. It is recommended that future studies consider:
   a. The comparison and utility of the K-D and Cognitive Status Sport (Cog Sport),[88] composite scores in concussed patients; and
   b. The comparison and utility of the K-D and CNS Vital Signs (CNSVS) composite scores in concussed patients; and
   c. The comparison and utility of the K-D and Axon Sports Cognitive assessment composite scores in concussed patients.
4. The K-D test has been reported[314, 317, 329] to have been used in the return-to-play assessment of concussed players. Although this has been identified, no study, to date, has specifically reported on the use of the K-D in return-to-pay assessment. It is recommended that future studies consider:
   a. Use of the K-D in the management of return-to-play activities of concussed sports participants;
   b. Use of the K-D in the management of return-to-work activities of concussed patients; and
   c. Use of the K-D in the management of return-to-school/academia activities in concussed student.

***Impact biomechanics***

1. Only one study[330] has been published on using instrumentation on un-helmeted players to record and report impacts to the head in amateur senior rugby union. Although this study highlighted the number of impacts that can occur during match participation, as measured by an instrumented mouthguard, further studies are warranted. It is recommended that future studies consider:

   a. Impacts to the head that occur from match and training participation in women's rugby union;
   b. Impacts to the head that occur from match and training participation in junior rugby union; and
   c. Impacts to the head that occur from match and training participation in professional rugby union.
2. As reported in Chapter 3, the sporting activities in New Zealand that have the highest number of Accident Compensation Corporation moderate-to-severe concussion claims are rugby union, rugby league, soccer, touch rugby, hockey, softball and netball. Although there are studies reporting the impact biomechanics in senior amateur rugby union (Chapter 7) and women's soccer (heading drill),[261] no other studies have reported on the other sporting codes identified. It is recommended that future studies consider:
   a. Impacts to the head that occur from match and training participation in amateur rugby league;
   b. Impacts to the head that occur from match and training participation in amateur soccer;
   c. Impacts to the head that occur from match and training participation in touch rugby;
   d. Impacts to the head that occur from match and training participation in softball; and
   e. Impacts to the head that occur from match and training participation in netball;
      i. These studies should consider youth / age grade, male and female participants.

***Other concussion related research***

As a result of the studies undertaken in this thesis, other aspects of concussion are warranted in the following areas:

1. Use of a biomarker in the assessment and management of sports-related concussion in the Emergency Department;
2. The identification of the most appropriate graduated stepwise process for return to school, academics and work activities;
3. The identification of the appropriate protocol for rest and graded exertion at different age and competition levels for concussed patients;
4. Review of the concussion discharge advice from Emergency Departments and General Practitioners: Evidence based concussion guidelines; and
5. Use of the cyclic model for concussion management in the care and identification of concussion in sports participants in relationship to assessment and management, return-to-play protocols and the identification of risk factors related to concussion.

**Conclusions**

This thesis has provided novel contributions with:

- Identification of the costs of sport-related concussions in New Zealand for seven sports codes. Previous studies had reported an estimated cost in total but had not established the costs per sport, per person and by gender;
- Identification of the number of players who did not report their concussions, the lack of knowledge of the risks and potential consequences of concussion in players in New Zealand for the first time;
- Identification of the 1:3 ratio of witnessed to un-witnessed concussions that occurred in match play for amateur senior rugby union (First published study on sideline assessment of concussion in amateur rugby union);
- Identification that the K-D test can be used as part of a continuum for the assessment and monitoring of players with a concussion, and can assist healthcare providers to evaluate adequate cognitive rest and inform clinical decisions regarding return-to-play and return-to-academic activities (First longitudinal study on the use of the K-D);
- Amateur senior rugby union players recorded more impacts than for American football collegiate and high school players (First publication of impacts to the head in an amateur senior rugby union team. Identification of the frequency and magnitude of head impacts); and
- Identification of the need for consensus on the reporting of impact biomechanics and suggested reporting standards for studies of head impacts in sports;
- A cyclical model for concussion management.

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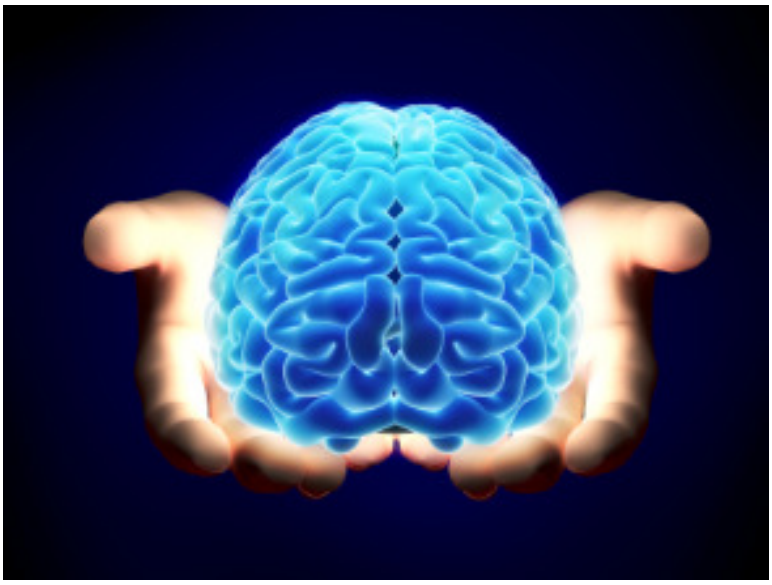
# Appendices

# Appendix I: Technical Report to NZRL

# Concussions in amateur rugby league: Player self-reported history and the incidence of match related concussions 2008 – 2012



## A technical report to the New Zealand Rugby League

15st October 2014

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Reviewed by

Dean Watkins, New Zealand Rugby League

NEW ZEALAND RUGBY LEAGUE

# Introduction

This technical report is supplied to the New Zealand Rugby League as a summary of data obtained through the writers role as a team medic in the Wellington Rugby League catchment region. The information contained in this report is a result of a summarising of personal medical histories provided by players and from matches where the writer was present as the sideline medic.

Mild traumatic brain injury (mTBI), more commonly known as a concussion, has become an increasingly serious concern for all sporting activities worldwide.[100, 416] In the United States of America it has been reported that 1.6 to 3.8 million sports related concussions occur annually.[358] The cumulative (direct and indirect) annual costs of these sport related concussions is nearly $US 17 billion (1999 values). Amongst 15- to 24-yr-olds, sport related concussion is the second to road trauma as most common causes of traumatic brain injury.[564] The awareness, identification, management and prevention of concussion in sport is becoming an ever increasing concern in sporting activities and with growing media awareness there is a focus on how sporting activities management of these injuries is being undertaken.

In establishing the NZRL Concussion Policy it was seen as a step towards assisting the sport of Rugby league in New Zealand towards advancement in the process of the management of these injuries. Additionally there has been the introduction of the rugby league fundamentals where there is a specific focus on concussion injuries aimed at all levels of participation and management. Unfortunately this seems to not have been enough as shown by the results of this technical report. The results of three years of observations and recording of concussive injuries in one district reflects a poor compliance with the policy, minimal knowledge of what a concussion is and limited awareness of the management of players with concussions.

Recent development in sideline tools for the identification of concussions may assist the sport of rugby league in better identification of these injuries but a general cultural shift is required for an appropriate level of management to occur. This shift is seen as required by team management, club and match officials at all levels of involvement and the general rugby league environment. As well the “win at all costs” attitudes and rewarding team management for keeping players on the field needs to be address at all levels of participation before steps to manage rugby league related concussions can be successfully undertaken and the safety of players becomes a priority at all levels of participation.

# Self-reported concussion history of amateur rugby league players: One districts experience

## Background

The topic of sport-related concussion is receiving a growth in published research relating to concussion identification, assessment and management in all sporting environments.[123, 391] As well it has become one of the most troublesome injuries facing the sports medicine professional,[347] especially in regards to the early identification of concussive signs and symptoms and appropriate concussive management facilitation.[273, 409]

Amateur sports such as rugby league in New Zealand do not have the resources available for qualified medical personnel to be at every match every weekend.[331] As such it is often the coach or team manager (if the team has one) to make the decisions in regards to player welfare and this includes concussion identification and management. The awareness of team management in regards to concussion has been previously reported on[331, 477, 601] and highlight the need for concussion education to occur for all people involved in the management level of sports participation.

Players with a concussive injury returning to their sport have been reported to be at a greater risk of complications.[291, 388] These complications are related to subsequent concussive events and may result in prolonged concussive symptoms and cumulative cognitive deterioration.[391] Often the identification of a sport-related concussion is left to the coach of the team as there are often no medical personnel available at the games and training sessions.[347] As a result concussive signs may be missed as the coach may be unable to take the required time to fully assess the player and sometimes concussions may be inadvertently missed. The aim of this study was therefore to review self-reported concussive history of amateur rugby league players to identify concussions within a two year period, concussive events outside of this time frame and medical review and clearances for return-to-play.

## Methods

A retrospective analysis was undertaken on all medical pre-competition questionnaires completed during the 2010 to 2012 competition periods by amateur rugby league players. The pre-competition questionnaires were requested as part of the assessment process for the provision of medical coverage to the players by one of the authors (DK). The questionnaires were reviewed for the concussion history of players for the previous two years, prior history of concussion and the Post-Concussion Symptom Scale that was used for the establishment of baseline data for players competing in various rugby league competitions.

### Subjects

Over the study period a total of 213 male participants from domestic and representative amateur rugby league teams agreed to take part in recording their concussion history as part of studies[317] being conducted. All participants were provided with an information sheet and gave either personal informed consent to participate or had parental informed consent before completing the history questionnaire as part of studies that were conducted.

**Concussion History Questionnaire**

All players were asked about their concussion history. This included history of concussions in the current and previous playing seasons, number of concussions, residual symptoms from any concussions reported and medical clearance to return-to-play. Participants were also asked to list the total number of years they had played rugby league. Participants were also asked to identify whether they considered a mild Traumatic Brain Injury or a Concussion to be more serious, or whether they were the same.

**Post-Concussion Symptom Scale (PCSS)**

The PCSS is a 22-item neuropsychological assessment scale that is a part of the larger Sport Concussion Assessment Tool (SCAT). Developed in the late 1980's,[381] the PCSS was designed to measure the severity of symptoms in the acute phase of recovery from a concussion. Using a 7-point Likert-type scale anchored at 0 (complete absence) to 6 (most severe) players rank each symptom according to the severity that they have experienced. The symptoms are not specific to concussion and even non-injured participants have recorded symptoms on the PCSS[381]. A threshold symptom score of 7 was set for the duration of the study[359]. Any player reporting more than 7 symptoms on any assessment were referred for further medical evaluation. This threshold was adopted as an indicator of a head injury having occurred based on prior studies[359]. The internal consistency reliability (Cronbach's α) for the PCSS has been reported to range from 0.88 to 0.94[381]

**Statistical Analysis**

All the data collected were entered into a Microsoft Excel spreadsheet and analysed with Statistical Package for Social Sciences (SPSS v.19.0; IBM SPSS Inc., Chicago, IL, USA for windows). Data are reported as means and standard deviations (SD) where appropriate. Correlations between concussion history and symptoms reported were analysed using a Spearman's Correlation Coefficient ($r_S$). To ascertain reliability of the questionnaire and player recall of their concussion history, players who were selected in more than one group (n=20) had their concussion history computed with a weighted Cohen $\kappa$ coefficient.[85, 607] A one-way analysis of variance (ANOVA) was used to investigate differences between the reported concussions, previous history of concussions and the symptoms identified. Statistical significance was set at $p<0.05$. All data are reported as mean ± SD.

## Results

Over the duration of the study a total of 213 (mean ± *SD*: age, 19.2 ± 4.4 yr) amateur rugby league players competed in the studies being undertaken. Participants reported an average of 4.3 ± 2.7 yr playing experience in rugby league. There was an average of 4.0 ± 2.6 concussive injuries per participant in the previous two years (see Table 1). There was an average of 5.0 ± 4.6 concussive injuries per participant the period preceding. A few participants (6.1%) reported that they had no previous history of having incurred a concussion, loss of consciousness or "bell-ringer" / "ding" throughout their life.

**Table 1:** Demographics of amateur rugby league participants for age, playing experience, concussive history and associated symptoms for previous two years, concussive history for prior years, return to play requirements and current post-concussion symptoms scores and severity by means, standard deviations and ranges reported.

| | Total | U15 | U17 | Premier |
|---|---|---|---|---|
| **Demographics** | | | | |
| Age, average SD [range] | 19.2 ±4.4 [13-34] | 14.5 ±0.6 [13-15] | 16.5 ±0.5 [16-17] | 21.9 ±3.9 [18-34] |
| Forwards | 119 | 24 | 26 | 69 |
| Backs | 94 | 18 | 21 | 55 |
| Experience, average SD [range] | 4.3 ±2.7 [1-12] | 4.7 ±2.8 [1-9] | 3.4 ±2.5 [1-10] | 4.4 ±2.7 [1-12] |
| **Concussive history in previous two years** | | | | |
| Concussion, average SD [range] | 1.9 ±1.2 [0-5] | 1.8 ±1.2 [0-5] | 2.0 ±1.3 [0-5] | 1.9 ±1.3 [0-5] |
| Loss of Consciousness, average SD [range] | 1.2 ±0.2 [0-2] | 1.3 ±0.5 [0-2] | 1.1 ±0.3 [0-2] | 1.2 ±0.4 [0-2] |
| "Bell Ringer" / "Ding", average SD [range] | 2.2 ±2.0 [0-10] | 2.3 ±2.4 [0-10] | 2.3 ±1.9 [0-10] | 2.2 ±1.9 [0-10] |
| Total, average SD [range] | 4.0 ±2.6 [0-11] | 4.1 ±2.8 [0-11] | 3.8 ±2.5 [0-11] | 4.0 ±2.5 [0-11] |
| **Symptoms previous two years** | | | | |
| Headaches, average SD [range] | 1.9 ±1.4 [0-6] | 2.4 ±1.8 [0-6] | 1.6 ±0.6 [0-3] | 1.9 ±1.4 [0-6] |
| Dizziness, average SD [range] | 2.0 ±1.5 [0-6] | 1.5 ±0.7 [0-3] | 2.2 ±1.6 [1-6] | 2.0 ±1.5 [0-6] |
| Memory Difficulties, average SD [range] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] |
| Visual Disturbances, average SD [range] | 1.3 ±0.4 [0-2] | 1.0 ±0.0 [0-1] | 1.3 ±0.5 [0-3] | 1.3 ±0.4 [0-2] |
| Hearing Problems, average SD [range] | 1.4 ±0.8 [0-3] | 1.7 ±1.0 [0-3] | 1.0 ±0.0 [0-1] | 1.4 ±0.8 [0-3] |
| Other Symptoms, average SD [range] | 1.8 ±1.2 [0-4] | 1.8 ±1.2 [0-4] | 1.8 ±1.5 [0-4] | 1.8 ±1.2 [0-4] |
| **Concussive history in previous years** | | | | |
| Concussion, average SD [range] | 2.8 ±2.6 [0-10] | 3.0 ±2.8 [0-10] | 2.5 ±2.6 [0-10] | 2.9 ±2.6 [0-10] |
| Loss of Consciousness | 1.8 ±1.5 [0-6] | 1.8 ±1.4 [0-6] | 1.8 ±1.7 [0-6] | 1.7 ±1.5 [0-6] |
| "Bell Ringer" / "Ding", average SD [range] | 2.4 ±2.3 [0-10] | 1.9 ±1.1 [0-4] | 2.2 ±2.1 [0-10] | 2.5 ±2.4 [0-10] |
| Total, average SD [range] | 5.0 ±4.6 [0-19] | 4.5 ±4.3 [0-19] | 4.5 ±4.7 [0-19] | 5.2 ±4.6 [0-19] |
| **Return to play** | | | | |
| Seen by Dr, n (% of total) | 16 (7.5) | 0 - | 3 (1.4) | 12 (5.6) |
| Stand down (3 weeks) completed, n (% of total) | 11 (5.2) | 0 - | 2 (0.9) | 8 (3.8) |
| Medical clearance to return to play, n (% of total) | 6 (2.8) | 0 - | 2 (0.9) | 4 (1.9) |
| **Post-Concussion Symptoms Scale** | | | | |
| Score, average SD [range] | 7.7 ±6.1 [0-22] | 6.5 ±4.8 [0-22] | 8.4 ±6.9 [0-22] | 7.7 ±6.1 [0-22] |
| Severity, average SD [range] | 10.9 ±17.1 [0-61] | 8.6 ±12.1 [0-57] | 12.0 ±19.5 [0-58] | 10.9 ±17.1 [0-61] |

A total of 7.5% of all participants reported seeing a medical doctor for their reported concussion while only 5.2% completed the required three week return-to-play process. Even fewer players (2.8%) reported seeing a medical doctor for a clearance for return-to-play. No Under 15 yr old player reported seeing a medical doctor or having a stand down period for return-to-play. It was identified that 7.0% (n=27) of the participants had high PCSS scores and were assessed to have an active concussion at the time the assessment was being conducted.

Despite 39.0% of players reporting that they had not had a concussion in the previous two years they did report that they had loss of consciousness and "bell ringer" / "dings" from match participation (see Table 2). Players reporting three or more concussions (n=31) also reported the highest mean number of headaches (2.0 ± 1.0), dizziness (2.9 ± 1.9) and previous concussions (6.5 ± 4.1) than players reporting fewer concussions in the previous two years.

**Table 2:** Demographics of amateur rugby league participants reporting none, one, two, 3 or more concussions in the previous two years by age, playing experience, concussive history and associated symptoms, concussive history for prior years, return to play requirements and current post-concussion symptoms scores and severity by means, standard deviations and ranges reported.

| | Previous 2 yr history | | | |
|---|---|---|---|---|
| | No Concussions (n=83) | 1 concussion (n=66) | 2 concussions (n=33) | 3+ concussions (n=31) |
| **Demographics** | | | | |
| Age, average SD [range] | 18.7 ±4.5 [13-34] | 19.7 ±4.6 [14-32] | 19.2 ±4.5 [14-32] | 19.5 ±3.8 [14-28] |
| Experience, average SD [range] | 4.6 ±2.8 [1-10] | 4.2 ±2.8 [1-12] | 4.0 ±2.9 [1-10] | 3.7 ±2.2 [1-8] |
| **History in previous two years** | | | | |
| Concussion, average SD [range] | 0.0 - | 1.0 - | 2.0 - | 3.8 ±1.5 [3-5] |
| Loss of Consciousness, average SD [range] | 1.0 ±0.0 [0-1] | 1.1 ±0.4 [0-2] | 1.0 ±0.0 [0-1] | 1.5 ±0.5 [0-2] |
| "Bell Ringer" / "Ding", average SD [range] | 3.6 ±2.9 [0-10] | 1.2 ±0.4 [0-2] | 1.5 ±0.5 [0-2] | 2.6 ±1.4 [0-5] |
| Total, average SD [range] | 3.7 ±3.2 [0-11] | 2.8 ±0.9 [1-4] | 3.3 ±0.9 [2-4] | 7.1 ±2.5 [5-10] |
| **Symptoms previous two years** | | | | |
| Headaches, average SD [range] | 0.0 - | 2.1 ±1.7 [0-6] | 1.3 ±0.5 [0-3] | 2.0 ±1.0 [0-3] |
| Dizziness, average SD [range] | 3.3 ±2.1 [0-6] | 1.3 ±0.4 [0-2] | 1.5 ±0.5 [0-2] | 2.9 ±1.7 [0-5] |
| Memory Difficulties, average SD [range] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] | 1.0 ±0.0 [0-1] |
| Visual Disturbances, average SD [range] | 1.5 ±0.5 [0-2] | 1.0 ±0.0 [0-1] | 2.0 ±0.0 [0-2] | 1.0 ±0.0 [0-1] |
| Hearing Problems, average SD [range] | 0.0 - | 1.4 ±0.8 [0-3] | 1.0 ±0.0 [0-1] | 0.0 - |
| Other Symptoms, average SD [range] | 0.0 - | 1.6 ±0.4 [0-2] | 4.0 ±0.0 [0-4] | 0.0 - |
| **Previous history of concussive events** | | | | |
| Concussion, average SD [range] | 1.4 ±0.5 [0-2] | 2.1 ±1.2 [0-5] | 2.5 ±1.4 [0-4] | 6.5 ±4.1 [0-10] |
| Loss of Consciousness | 1.0 ±0.0 [0-1] | 1.4 ±1.0 [0-4] | 2.0 ±0.0 [0-2] | 4.0 ±2.1 [0-6] |
| Bell Ringer / Ding, average SD [range] | 4.0 ±3.8 [0-10] | 1.3 ±0.4 [0-2] | 2.6 ±1.9 [0-6] | 2.5 ±0.5 [0-3] |
| Total, average SD [range] | 3.2 ±3.8 [0-13] | 4.0 ±2.0 [0-9] | 5.5 ±2.9 [2-10] | 10.1 ±7.6 [2-19] |
| **Return to play** | | | | |
| Seen by Dr, n (% of total) | 5 (2.3) | 7 (3.3) | 1 (0.5) | 3 (1.4) |
| Stand down (3 weeks) completed, n (% of total) | 3 (1.4) | 5 (2.3) | 1 (0.5) | 2 (2.9) |
| Medical clearance to return to play, n (% of total) | 3 (1.4) | 2 (0.9) | 0 - | 1 (0.5) |
| **Post-Concussion Symptoms Scale** | | | | |
| Score, average SD [range] | 6.5 ±5.5 [0-20] | 6.1 ±3.0 [0-12] | 7.7 ±7.1 [2-20] | 18.5 ±3.6 [0-22] |
| Severity, average SD [range] | 14.4 ±16.7 [0-57] | 7.4 ±5.4 [0-18] | 14.2 ±22.6 [0-61] | 21.4 ±24.7 [0-57] |

There was a correlation observed between concussions reported in the last two years and previous concussions ($r_s$=0.597; $p$<0.001); loss of consciousness and previous loss of consciousness ($r_s$=0.681; $p$<0.001) and "bell-ringer" / "ding" and dizziness ($r_s$=0.622; $p$<0.001) and these were significant (see Table 4). This was similar for previous loss of consciousness and headaches ($r_s$=0.671; $p$<0.001); previous concussions and headaches ($r_s$=0.627; $p$<0.001) and PCSS score and PCSS severity ($r_s$=0.916; $p$<0.001).

**Table 3:** Spearman's correlations between reported history of concussion, loss of consciousness and bell ringers; symptoms experienced post event in the past two seasons; previous history of concussion, loss of consciousness and bell ringers; post-concussion score and severity of amateur rugby league players in New Zealand.

| | Concussion | LOC | "Bell Ringer" | Headaches | Dizziness | Memory | Visual | Hearing | Other | Previous Concussion | Previous LOC | Previous "Bell Ringer" | PCSS Score | PCSS Severity |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Concussion | - | | | | | | | | | | | | | |
| LOC | *0.30*[a] | - | | | | | | | | | | | | |
| "Bell Ringer" | *0.18*[a] | 0.17[b] | - | | | | | | | | | | | |
| Headaches | *0.45*[a] | *0.51*[a] | 0.06 | - | | | | | | | | | | |
| Dizziness | *0.30*[a] | *0.32*[a] | *0.62*[a] | *0.42*[a] | - | | | | | | | | | |
| Memory | *0.28*[a] | *0.34*[a] | *0.20*[a] | *0.26*[a] | *0.18*[a] | - | | | | | | | | |
| Visual | 0.07 | 0.16[b] | *0.20*[a] | *0.35*[a] | *0.22*[a] | *0.36*[a] | - | | | | | | | |
| Hearing | 0.13 | *0.20*[a] | 0.11 | *0.29*[a] | *0.35*[a] | 0.12 | *0.30*[a] | - | | | | | | |
| Other | 0.14[b] | *0.32*[a] | -0.11 | *0.48*[a] | 0.16[b] | 0.14[b] | 0.10 | *0.47*[a] | - | | | | | |
| Previous Concussion | *0.60*[a] | *0.45*[a] | -0.05 | *0.63*[a] | *0.20*[a] | *0.23*[a] | *0.40*[a] | *0.29*[a] | *0.33*[a] | - | | | | |
| Previous LOC | *0.23*[a] | *0.68*[a] | *0.18*[a] | *0.67*[a] | *0.40*[a] | *0.48*[a] | *0.42*[a] | 0.12 | *0.51*[a] | *0.50*[a] | - | | | |
| Previous "Bell Ringer" | *0.31*[a] | *0.25*[a] | 0.04 | *0.31*[a] | *0.31*[a] | 0.13 | *0.41*[a] | *0.24*[a] | 0.15[b] | *0.49*[a] | *0.22*[a] | - | | |
| PCS | 0.14[b] | *0.34*[a] | -0.02 | *0.21*[a] | *0.24*[a] | *0.30*[a] | 0.14[b] | 0.08 | -0.07 | 0.16[b] | *0.21*[a] | *0.30*[a] | - | |
| PCS Severity | 0.07 | *0.22*[a] | -0.03 | 0.06 | 0.12 | *0.31*[a] | 0.15[b] | -0.12 | -0.26 | 0.01 | 0.07 | *0.29*[a] | *0.92*[a] | - |

LOC: Loss of Consciousness; PCS: Post-Concussion Symptoms Scale; Correlation is significant at (a) $p < 0.01$; (b) $p < 0.05$

The reliability of the self-reported concussion questionnaire yielded a weighted $\kappa$ coefficient with substantial reliability for concussions ($\kappa$=0.797; $p$<0.001), loss of consciousness ($\kappa$=0.816; $p$<0.001) and bell ringer / ding ($\kappa$=0.777; $p$<0.001) in the previous two years. When compared with the players history of previous concussions ($\kappa$=0.864; $p$<0.001) and loss of consciousness ($\kappa$=0.914; $p$<0.001) the results were similar.

## Discussion

The purpose of this study was to report on the concussion history of amateur rugby league players. The results of this study is concerning with an average of four concussive events per player for all age groups reported. Even more concerning is the finding that despite players reporting having had no concussions in the previous two year period they reported an average of nearly four concussive events.

Although the history of concussions reported in this study was undertaken by the use of a self-reporting questionnaire there is some support that the identified history may be reliable.[304] The questionnaire utilised in this study was identical for all groups who were assessed as part of their medical assessment in the pre-competition period. In a study reporting on the reliability of concussion history it was identified that the self-reported history of concussion measure was of moderate reliability.[304]

Players across all age groups were administered the same questionnaire in different settings where they were gathered for the respective age group competition meetings over a 12 month period. Players who were involved in more than one assessment process (n=20) were removed from subsequent analysis to ensure there were no duplication of the results. These players did provide the source for the reliability analysis of the self-reported concussion history indicating the weighted $\kappa$ statistics were in the "substantial" agreement range according to the guidelines of Landis and Loch.[357]

An unexpected finding was that 7% of all players had active concussive symptoms and was a cause for concern. None of the players identified with active concussive symptoms had seen any medical professional for their symptoms nor reported these symptoms to anyone as they felt this was all part of "playing the game". All of these players were advised to seek further medical assistance and required to provide a full medical clearance before they would be allowed to return to match activities. One player was subsequently medically cleared two days later by his own medical practitioner as he "had no signs of a concussion". This player was identified as having recorded a concussive incident in the following match[317] and was advised not to return to match activities for the rest of the competition, to seek medical review and to refrain from training until medically cleared to do so.

A previous study[409] identified that players will not report concussive symptoms because they do not think it is sufficiently serious enough to necessitate reporting. A lack of knowledge of the risks and potential consequences of concussion was reported to be the most common reason for the non-reporting of a possible concussion.[409] It was also identified that more than a third of players failed to report a concussive injury as they did not recognize that they had sustained a concussive injury based on their symptoms.[409] This may have been the case in the current study with 7% of players showing signs of a concussive event. Further

research is warranted to explore whether players in amateur contact sports such as rugby league have concussion awareness.

With only 7.5% of players reporting their concussive history identified they sought medical assistance after having a concussive event is a concern. Similarly the finding that only 5.2% of players with concussive events completed a 3 week stand down and even less (2.8%) sought a medical clearance is also concerning. Although the New Zealand Rugby League has a concussion policy, based on an international consensus agreement,[425] in place for the management of concussive events and provides an annual update for the identification and management of concussion it appears this information is not reaching all members of this sporting environment. Additionally there are no readily available tools for the assessment of concussion on the sideline for the non-medical support person with amateur rugby league teams.

A possible concussion assessment tool that may assist the team medic / trainer is the King-Devick test. Designed as a saccadic reading test,[485] the King-Devick has been utilised with amateur rugby league[317] and has been shown to readily identify concussive events.[202, 203, 317] The King-Devick has also been utilised with the monitoring of mixed martial arts fighters, boxing, basketball and American collegiate football players.[202, 203] Additionally the King-Devick does not require the operator to be a medical professional, requires less than a minute to administer[202] unlike the Sports Concussion Assessment Tool which can take approximately 20 minutes to complete.[149]

Non-compliance with return-to-play protocols is not unique to amateur rugby league players and has been reported in studies on rugby union,[280, 578] ice hockey[1] and US high school athletes.[394] In a study[280] reporting on compliance with return-to-play regulations in schoolboy and community rugby union it was reported that schoolboy rugby had 100% non-compliance with player return-to-play after a concussive event. The current study is similar to suburban rugby non-compliance (95% vs. 97%) but greater than other studies reporting on high school rugby[578] (77.1%) ice hockey (33%) and US high school sports (16.7%) non-compliance and indicates that there are differences in how return-to-play regulations are completed in sporting environments despite international agreement with management of sport-related concussions. Further research is warranted to explore the return to play practices of amateur sports such as rugby union and rugby league.

Players reporting three or more concussions in the previous two years also reported more prior concussions than those players reporting none, one or two concussions. This finding was not unexpected as a prior history of concussion places the brain at risk to repeat injury and symptom exacerbation.[434] As well sports participants with two or more concussions have also been reported to have more neurobehavioral symptoms and impairment on selected neuropsychological tests than players with a history of a single concussion.[90]

# Concussion identification in amateur rugby league in New Zealand by use of the Sport Concussion Assessment Tool (SCAT)

## Background

Played internationally, rugby league is a physical collision sport participated at junior, amateur, semi-professional and professional levels of competition.[185] Similar to rugby union in regards to skills and movement patterns, rugby league does not have line-outs, mauls, consists of 13 players per team (compared to 15 players as seen in rugby union) and requires an immediate play-the-ball after each tackle is completed.[185] Rugby league is an intermittent sport comprising of intense bouts of activity (e.g. tackling and sprinting), interspersed with short bouts of lower intensity activities (e.g. walking and jogging).[185] Divided into two groups, forwards and backs, these players can expect to undergo on average of 29 to 55 physical collisions (tackles and ball-carries) per game.[224] Due to the nature of the game there is a risk of an injury occurring, such as concussion (also known as mild traumatic brain injury), to the players involved.

International initiatives for sports-related concussions have resulted in the formation of the 'Concussion in Sport Group' (CISG). Following several international meetings involving leading sports organisations the CISG have produced consensus statements on the definition, assessment and management strategies for sports-related concussions.[425] In addition the Sport Concussion Assessment Tool (SCAT and SCAT2)[425] was produced as a comprehensive evaluation tool for assessing sports-related concussion. As a result of the consensus statement and assessment tool, sporting bodies such as New Zealand Rugby League amended their concussion policy to reflect these changes and produced educational packages for team management nationwide (http://www.nzrl.co.nz/media/22461/concussion_policy_sept_2010.pdf). Although there is an increased awareness of the effects of concussion, many concussions are neither recognized by athletes nor observed by coaches or trainers.[114] Consequently a large proportion of concussions remain unreported.[114]

The reported incidence of concussion from rugby league participation has varied from 2.5[228] to 34.8[182] per 1,000 match hours. In an analysis of publicly available broadcast games for the National Rugby League's Telstra Premiership 2010 season in Australasia[440] the incidence rate for probable concussion was 11.1 per 1,000 player hours and was likely to be underestimated. In studies reporting amateur rugby league injuries in New Zealand the incidence of concussions varied from 6.5[322] to 27.2[324] per 1,000 match hours. The identification and recording of concussion at this level of participation has been based on player reports or trainer assessment and no formalized assessment process was undertaken in these studies. To date there have been no published prospective studies reporting the use of the SCAT or SCAT2 in identification of concussions at the amateur level of rugby league participation. This study undertook to describe SCAT and SCAT2 results, concussion incidence, injury mechanisms, and player positions injured during prospective data collection over three seasons for domestic and representative rugby league players.

## Methods

A total of 176 (124 domestic, 52 representative) premier level rugby league players participated in the study during the 2008, 2009 and 2010 competitive seasons. All concussions were assessed with the SCAT[422] in 2008 and SCAT2[425] in the 2009 and 2010 seasons. The mean ±SD age, stature and mass of the players enrolled in the study were 25.3 ±5.5 yr, 1.78 ±0.06 m and 91.9 ±15.2 kg. Injury data were collected from a total of 83 (61 club, 22 representative) amateur rugby league matches played from March to October each year, including all trial, fixtures and finals matches. Matches were played under the rules and regulations of the New Zealand Rugby League (NZRL) that includes the mandatory wearing of mouthguards during match play. All participants were invited to participate in the study and received a clear explanation of the study, including the risks and benefits of participation. Ethics approval was provided (AUTEC 08/44).

### Match exposure and concussion definition

Injury incidence rates expressed as the number of injuries sustained per 1,000 playing hours were determined using previously described methods.[323] Over the duration of the competition, all match injuries were recorded by the team sports medic on a standardized injury reporting form regardless of severity.[323] The definition of a concussion utilised for this study was "*any disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific symptoms and often does not involve loss of consciousness".*[425]

### Sport Concussion Assessment Tool (SCAT)

The SCAT is a paper neurocognitive tool developed from existing concussion assessment tools.[422] Established as having face validity, the SCAT reliability and change scores have not been reported to date.[4] The SCAT consists of subjective and evaluative components. Subjective components consist of 25 symptoms based on a 7-point Likert scale anchored at 0 (no symptoms) to 6 (severe symptoms). The evaluative component consists of signs, modified Maddock's questions, symptom scores, cognitive assessment and neurological screening. The cognitive assessment utilises immediate word recall of five words upon hearing the words and delayed word recall following concentration tasks, with additional stating months of the year in reverse order and repeating single digits in reverse order. The SCAT2[425] is a revised assessment tool that has expanded on the subjective components of the SCAT. Changes to the SCAT2[425] are the deletion of some symptoms, combining dizziness and balance problems symptoms, modified balance error scoring systems (BESS), three trials for immediate word recall and scoring up to 100.

### Balance Error Scoring System (BESS)

The balance error scoring system was developed as a method of evaluating postural stability without the use of complex or expensive equipment.[526] There have been significant correlations between the BESS and force-platform sway measures ($r$=0.42 to 0.79) with inter-rater reliability coefficients of 0.78 to 0.96.[526] Three

different stances (double stance, single stance, and tandem stance) were tested on a firm surface with each test lasting 20 seconds.[526] Players were directed to assume the required stance by placing their hands on their iliac crests and the test would begin when they closed their eyes. During the single-leg stances, players were asked to maintain the contralateral limb in 20° of hip flexion with 30° of knee flexion. Players were asked to stand quietly and as motionless as possible in the stance position, keeping their hands on their iliac crests and their eyes closed. Players who lost their balance were to make any necessary adjustments to return to the testing position as quickly as possible. Performance was scored by adding 1 error point for each error committed to a total of 10 points. Trials were considered incomplete if they could not sustain the stance position for longer than 5 seconds.[526]

**Procedure**

All rugby league match activities were attended by the team sports medic. Any player observed to have received a direct blow to the head, was slow to rise from a collision or appeared unsteady on their feet following a collision were assessed on-field for any injury. Players were removed from the match activity and rested on the sideline if they showed any signs of delayed answering or incorrect answers to questions. A reassessment of the player was undertaken approximately 20 minutes later[619] utilising the SCAT and BESS (2008) or the SCAT2 (2009-10). Players with a total score of less than 93 were ruled out of further participation in any match or training activities and required further medical evaluation. Any player with loss of consciousness was treated for cervical spine injury, immobilized and transferred to a hospital for further management. All suspected concussions were referred to a medical practitioner for further management. All players assessed to have a suspected concussion undertook a graduated return-to-play program[425] and required a full medical clearance before they could return to full training and match activities.

**Statistical analysis**

All data collected were entered into a Microsoft Excel spreadsheet and analysed with MedCalc for Windows version 11.3.8.0 (MedCalc Software, Mariakerke, Belgium http://www.medcalc.be). Given the slight differences between the SCAT and SCAT2, the results from the SCAT collected in the first year of the study were adapted to be consistent with the SCAT2 scores by amending the data from the SCAT to reflect the changes made to the SCAT2. The results were then added up to reflect a score out of 100. The summary results of the SCAT2 were reported as mean ±SD and range of scores and differences were assessed with independent *t*-tests. A one-sample chi-squared ($\chi^2$) test was used to determine whether the observed injury frequency was significantly different from the expected injury frequency. To compare between concussion injury incidence rates risk ratios (RR's) were deemed to be significant at $p<0.05$.

## Results

The overall injury exposure for the three competitive seasons was 1,435 match hours at risk (see Table 4). The match injury exposure for the 2008, 2009 and 2010 seasons was 432.3 (domestic 328.5; representative 103.7); 484.1 (domestic 380.4; representative 103.7) and 518.7 (domestic 345.8; representative 172.9).

Eighteen players recorded a total of 19 concussions over the study period resulting in a total incidence of 13 (95% CI: 8 to 21) concussions per 1,000 match hours. One player recorded a second concussion the same playing season.

**Table 4:** Number of rugby league concussions, hours per concussion, mean time per injury, player appearances and match minutes per concussion by participation level and total per 1,000 match hours with 95% confidence intervals.

| | Domestic Competition (n=124) | Representative Competition (n=52) | Total (n=176) |
|---|---|---|---|
| Concussions Observed | 12 | 7 | 19 |
| Concussions Expected | 14.0 | 5.0 | 19 |
| Injury rates per 1,000 playing hours (95% CI) | 11.4; *6.5 to 20.0* | 18.4; *8.8 to 38.6* | 13.2; *8.4 to 20.8* |
| Number of matches played | 61 | 22 | 83 |
| Exposure hours | 1,055 | 380 | 1,435 |
| Hours per concussion (95% CI) | 87.9; *49.9 to 154.8* | 54.3; *25.9 to 114.0* | 75.5; *48.2 to 118.4* |
| Mean time (hrs) for first concussion (95%CI) | 6.8; *3.9 to 12.0* | 4.2; *2.0 to 8.8* | 5.8; *3.7 to 9.1* |
| Total No. concussions per match (95% CI) | 0.20; *0.11 to 0.35* | 0.32; *0.15 to 0.67* | 0.23; *0.15 to 0.36* |
| Player appearances per concussion (95%CI) | 66.1; *37.5 to 116.4* | 40.9; *19.5 to 85.7* | 56.8; *36.2 to 89.0* |

**Table 5:** Player position, player group, injury cause, match period, concussion severity and protective equipment worn when concussions were recorded for three consecutive seasons for amateur rugby league in New Zealand.

| | Domestic Competition (n=124) | | | Representative Competition (n=52) | | | Total (n=176) | | |
|---|---|---|---|---|---|---|---|---|---|
| | **No.** | **Rate; *95% CI*** | **%** | **No.** | **Rate; *95% CI*** | **%** | **No.** | **Rate; *95% CI*** | **%** |
| Player position | | | | | | | | | |
| No. 1 Fullback | 1 | 0.9; *0.1 to 6.7* | 8.3 | 3 | 7.9; *2.5 to 24.5* | 42.9 | 4 | 36.2; *13.6 to 96.5* | 21.1 |
| No. 2 Wing | 1 | 0.9; *0.1 to 6.7* | 8.3 | 0 | 0.0; - | 0.0 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| No. 3 Centre | 2 | 1.9; *0.5 to 7.6* | 16.7 | 1 | 2.6; *0.4 to 18.7* | 14.3 | 3 | 27.2; *8.8 to 84.3* | 15.8 |
| No. 4 Centre | 0 | 0.0; - | 0.0 | 1 | 2.6; *0.4 to 18.7* | 14.3 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| No. 5 Wing | 0 | 0.0; - | 0.0 | 0 | 0.0; - | 0.0 | 0 | 0.0; - | 0.0 |
| No. 6 Stand off | 0 | 0.0; - | 0.0 | 1 | 2.6; *0.4 to 18.7* | 14.3 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| No. 7 Half back | 1 | 0.9; *0.1 to 6.7* | 8.3 | 0 | 0.0; - | 0.0 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| No. 8 Prop | 1 | 0.9; *0.1 to 6.7* | 8.3 | 0 | 0.0; - | 0.0 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| No. 9 Hooker | 2 | 1.9; *0.5 to 7.6* | 16.7 | 0 | 0.0; - | 0.0 | 2 | 18.1; *4.5 to 72.4* | 10.5 |
| No. 10 Prop | 1 | 0.9; *0.1 to 6.7* | 8.3 | 1 | 2.6; *0.4 to 18.7* | 14.3 | 2 | 18.1; *4.5 to 72.4* | 10.5 |
| No. 11 Second row | 1 | 0.9; *0.1 to 6.7* | 8.3 | 0 | 0.0; - | 0.0 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| No. 12 Second row | 1 | 0.9; *0.1 to 6.7* | 8.3 | 0 | 0.0; - | 0.0 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| No. 13 Loose forward | 1 | 0.9; *0.1 to 6.7* | 8.3 | 0 | 0.0; - | 0.0 | 1 | 9.1; *1.3 to 64.3* | 5.3 |
| Backs | 5 | 4.7; *2.0 to 11.4* | 41.7 | 6 | 15.8; *7.1 to 35.1* | 85.7 | 11 | 14.2; *7.9 to 25.7* | 57.9 |
| Forwards | 7 | 6.6; *3.2 to 13.9* | 58.3 | 1 | 2.6; *0.4 to 18.7* | 14.3 | 8 | 12.1; *6.0 to 24.2* | 42.1 |
| Injury cause | | | | | | | | | |
| Tackler | 3 | 2.8; *0.9 to 8.8* | 25.0 | 1 | 2.6; *0.4 to 18.7* | 14.3 | 4 | 2.8; *1.0 to 7.4* | 21.1 |
| Ball carrier | 9 | 8.5; *4.4 to 16.4* | 75.0 | 6 | 15.8; *7.1 to 35.1* | 85.7 | 15[a] | 10.5; *6.3 to 17.3* | 78.9 |
| Match period | | | | | | | | | |
| 1st quarter | 1[b] | 0.9; *0.1 to 6.7* | 8.3 | 0 | 0.0; - | 0.0 | 1[c] | 2.8; *0.4 to 19.8* | 5.3 |
| 2nd quarter | 2 | 1.9; *0.5 to 7.6* | 16.7 | 2 | 5.3; *1.3 to 21.0* | 28.6 | 4 | 11.1; *4.2 to 29.7* | 21.1 |
| 3rd quarter | 2 | 1.9; *0.5 to 7.6* | 16.7 | 2 | 5.3; *1.3 to 21.0* | 28.6 | 4 | 11.1; *4.2 to 29.7* | 21.1 |
| 4th quarter | 7[a] | 6.6; *3.2 to 13.9* | 58.3 | 3 | 7.9; *2.5 to 24.5* | 42.9 | 10[b] | 27.9; *15.0 to 51.8* | 52.6 |
| 1st half | 3 | 2.8; *0.9 to 8.8* | 25.0 | 2 | 5.3; *1.3 to 21.0* | 28.6 | 5 | 7.0; *2.9 to 17.3* | 26.3 |
| 2nd half | 9 | 8.5; *4.4 to 16.4* | 75.0 | 5 | 13.1; *5.5 to 31.6* | 71.4 | 14 | 19.5; *11.6 to 32.9* | 73.7 |
| Concussion severity | | | | | | | | | |
| No LOC | 10 | 9.5; *5.1 to 17.6* | 83.3 | 6 | 15.8; *7.1 to 35.1* | 85.7 | 16 | 11.1; *6.8 to 18.2* | 84.2 |
| <5 s LOC | 2[c] | 1.9; *0.5 to 7.6* | 16.7 | 1 | 2.6; *0.4 to 18.7* | 14.3 | 3[d] | 2.1; *0.7 to 6.5* | 15.8 |
| Protective equipment | | | | | | | | | |
| Equipment used | 11 | 10.4; *5.8 to 18.8* | 91.7 | 6 | 15.8; *7.1 to 35.1* | 85.7 | 17 | 11.8; *7.4 to 19.1* | 89.5 |
| Mouthguard | 3 | 2.8; *0.9 to 8.8* | 25.0 | 2 | 5.3; *1.3 to 21.0* | 28.6 | 5 | 3.5; *1.5 to 8.4* | 26.3 |
| Headgear | 4 | 3.8; *1.4 to 10.1* | 33.3 | 0 | 0.0; - | 0.0 | 4 | 2.8; *1.0 to 7.4* | 21.1 |
| Shoulder pads | 2 | 1.9; *0.5 to 7.6* | 16.7 | 0 | 0.0; - | 0.0 | 2 | 1.4; *0.3 to 5.6* | 10.5 |
| Strapping | 10 | 9.5; *5.1 to 17.6* | 83.3 | 4 | 10.5; *3.9 to 28.0* | 57.1 | 14 | 9.8; *5.8 to 16.5* | 73.7 |

Rate reported per 1,000 match hours. CI: Confidence Interval. Significant difference (p<0.05) than (**a**)=Tackler; (**b**)=1st quarter; (**c**)= 4th quarter; (**d**)=no loss of consciousness.

**Player position, injury cause, match period, concussion severity and protective equipment worn**

The fullback (36; 14 to 97 per 1,000 match hours), No. 3 centre (27; 9 to 84 per 1,000 match hours), the hooker and No. 10 prop (18; 5 to 72 per 1,000 match hours) were the most common player positions to record a concussion (see Table 5). Ball-carriers sustained significantly more concussions than tacklers (RR: 3.8; 1.3 to 11.3; p=0.012). There were significantly more concussions in the fourth than the first quarter (RR: 10.0; 1.3 to 77.7; p=0.007) of matches (see Table 2) and were significantly more concussions in the second than the first half of matches (RR: 2.8; 1.0 to 7.6; p=0.039). The majority of players who recorded a concussion (74%) were not wearing a mouthguard when the concussion occurred (RR: 3.8; 1.4 to 10.2; p=0.039).

**Sport Concussion Assessment Tool (SCAT) scores**

Table 6 lists the mean ±SD and range of scores of the SCAT2. The mean ±SD sum score for the SCAT2 assessments of concussed players was 66.9 ±14.5. Although domestic players recorded a higher mean subtotal (50.5 vs. 39.9: *t*=1.38; p=0.216), and total score (70.8 vs. 60.7: *t*=1.13; p=0.302) than representative players these were not significant.

**Table 6:** Mean, standard deviations and range of SCAT2 concussion assessment component scores for 176 amateur rugby league players and for club (n=124) and representative (n=52) playing levels in New Zealand.

| | Domestic Competition (n=124) | | Representative Competition (n=52) | | Total (n=176) | |
|---|---|---|---|---|---|---|
| | **Mean; ±*SD*** | **Range** | **Mean; ±*SD*** | **Range** | **Mean; ±*SD*** | **Range** |
| **Domain area (maximum score)** | | | | | | |
| Symptom score (22) | 11.9; *2.9* | 6 to 16 | 10.9; *4.0* | 8 to 18 | 11.5; *3.3* | 6 to 18 |
| Physical signs score (2) | 1.2; *0.8* | 0 to 2 | 1.0; *0.8* | 0 to 2 | 1.1; *0.8* | 0 to 2 |
| Glasgow Coma Scale (15) | 14.1; *0.8* | 13 to 15 | 11.4; *5.8* | 3 to 15 | 13.1; *3.7* | 3 to 15 |
| Balance examination (30) | 22.5; *2.7* | 19 to 28 | 16.0; *11.2* | 0 to 25 | 19.9; *7.7* | 0 to 28 |
| Coordination score (1) | 0.8; *0.4* | 0 to 1 | 0.6; *0.5* | 0 to 1 | 0.7; *0.5* | 0 to 1 |
| **Subtotal (70)** | **50.5; *2.0*** | **47 to 53** | **39.9; *18.7*** | **13 to 59** | **46.3; *12.4*** | **13 to 59** |
| Orientation score (5) | 3.5; *1.0* | 2 to 5 | 3.3; *1.6* | 0 to 5 | 3.4; *1.3* | 0 to 5 |
| Immediate memory score (5) | 3.9; *0.8* | 3 to 5 | 3.9; *1.8* | 0 to 5 | 3.9; *1.2* | 0 to 5 |
| Concentration score (15) | 9.6; *1.6* | 8 to 13 | 11.3; *2.4* | 8 to 14 | 10.3; *2.0* | 8 to 14 |
| delayed recall score (5) | 3.3; *1.7* | 0 to 5 | 2.4; *1.7* | 0 to 5 | 2.9; *1.7* | 0 to 5 |
| **SAC subtotal score (30)** | **20.4; *3.0*** | **15 to 27** | **20.9; *6.2*** | **8 to 26** | **20.6; *4.4*** | **8 to 27** |
| **SCAT2 total (100)** | **70.8; *3.7*** | **67 to 79** | **60.7; *22.3*** | **21 to 79** | **66.9; *14.5*** | **21 to 79** |
| Maddocks Score (5) | 3.2; *1.0* | 2 to 5 | 3.4; *1.0* | 2 to 5 | 3.3; *1.0* | 2 to 5 |

SD= Standard Deviation**Discussion**

The finding of 13 concussions per 1,000 match hours is more than some,[273, 322] but not all[320] published studies reporting concussions as a result of participation in rugby league. There have been changes to the rules in regards to tackles involving contact with the head and tackling styles, such as spear tackling and head-slamming into the ground being outlawed in rugby league. Not recorded in this study was if the

concussion occurred in the tackle by direct contact to the head region or through contact with the ground. This is seen as a limitation to this study. Head high tackles and head contact with the ground have been reported to be the most common recorded mechanisms of injury when a concussion occurs.[273] Unfortunately there was no multi-view video camera footage to enable post injury analysis. Future studies investigating concussive injuries in rugby league should consider video analysis as part of the study protocol.

The use of the SCAT2 as part of concussion assessment for rugby league has never been reported. It has been recommended that the SCAT2 be not only utilised as a concussion assessment tool but also to identify -normative values of athletes before the competition starts.[539] This recommendation was made after the present study commenced, and thus was not incorporated in the protocol. In addition, many of the symptoms listed on the SCAT2 are not concussion specific. Uninjured players may also record these symptoms (i.e. fatigue, low energy) and an understanding of these symptoms when assessing a player with concussion is important. The most common reported symptoms reported in the current cohort of players were fatigue, low energy and headache which is similar to other studies[539] reporting the use of the SCAT2 as a sport concussion assessment tool in other sporting codes.

The SCAT2 is a longer sideline concussion tool and, although is an improvement over the original SCAT, will require additional time to complete on the sideline making it more of a training room assessment tool as opposed to a readily available sideline assessment tool.[149]. The ability to be able to conduct the complete test on the sideline was limited due to the team having a medic-trainer who was also required to interact with the players on the field and to manage any medical event that occurred. Consequently the assessments were done after the match had finished. In addition, the SCAT2 was utilised for the assessment of the concussed player. It were not utilised as part of an on-going review and comparison for the return-to-play process. A medical practitioner, not part of rugby league nor in contact with the team sports-medic, would clear the concussed player to return-to-play without having discussed the initial assessment. Some players were cleared to return before the graduated return-to-play protocol (< 5 days post-incident) was completed. This often resulted in a conflict between the team medic, the coach and the player. This is seen as a limitation to the use of the SCAT2 in amateur sports. Until the wider health care community encourages the facilitation of information such as SCAT2 for medical assessment of amateur players, use of concussion assessment tools at the sideline is limited to individuals doing their own recording.

Similar with a previous study[273] on injuries in professional rugby league, the fullback recorded the most concussions. This may be related to the nature of rugby league where the fullback has to take the ball forward usually into the larger forwards. If the fullback has a previous history of concussion then the risk of concussion increases two-fold compared with players with no previous history.[544] A limitation of the present study was that the players medical history including previous concussions was not recorded. Future studies should consider including medical history assessment in their data collection. One player had a recurring concussion in the same playing year resulting is a loss of the rest of the season. Another player suffered a neck injury the same time he sustained a concussion, resulting in his decision to terminate his participation

in rugby league. Both of these players reported a previous history of concussions after they were diagnosed during the current study.

The tackle situation is reportedly the most common cause of concussions in rugby league.[338] The tackle is also the most important skill in rugby league.[188] Success in the tackle is dependent on tackling ability, ability to tolerate the associated physical collisions and the capacity to 'win' the tackle contest.[188] With the emphasis on increased play-the-ball speed and dominance of the ruck, the tackle is an important area to focus on for injury prevention. Most tackle-related injuries occur when the ball carrier is tacked at the shoulder height / mid-torso region, in the blind vision area, with two or more tacklers.[339] Injury prevention programs designed to highlight these areas may be useful to assist in reducing the incidence of concussive injuries in tackle situations. In addition the development of correct tackling technique (defence) and falling in the tackle (attack) programs may also assist in reducing the incidence of concussions, as well as other injuries, in the tackle.[338] Further studies reporting on the tackle and the incidence of concussions are warranted.

Although 18 players reported they had a concussion, there is a tendency by players to under-report or hide the symptoms of concussion so they can return-to-play the following week.[409] Considering that more than 50% of concussions may not be reported[409] then the incidence of concussions in amateur rugby league may be more likely 27 per 1,000 match hours. This incidence rate is similar to a previous study[324] reporting division 2 amateur rugby league injuries. This highlights the need for more amateur rugby league clubs to have appropriate medical coverage for teams to ensure timely identification and management of concussions.[278] Although players in this study had a trained sports-medic, no other teams involved in the competitions had any formal medical coverage. Further longitudinal studies at amateur level sporting participation are warranted to explore team medical coverage and the incidence of concussions.

Studies have highlighted the concern surrounding reporting the recognition and management of concussion. In the analysis of broadcast rugby league game[440] it was identified that most injured players continued playing or returned–to-play despite being visibly concussed and described as such by the commentary team. They surmised that although 'return-to-play decisions' for professional players were guided by medical assessment, the way concussion is portrayed by media might affect public awareness of appropriate concussion management. In reviewing the knowledge of youth coaches[601] 42% of identified loss-of-consciousness as the hallmark of concussion, 26% of coaches would permit a player exhibiting signs of concussion to return-to-play. This was similar in New Zealand,[331] with 39% reporting loss-of-consciousness as the hallmark of concussion; 68% would leave a player with signs of concussion to continue to play and 68% would permit a player to return-to-play while still symptomatic. Of concern was 56% of respondents knew how to recognise a concussion, 38% knew the management of concussion and 31% could identify concussion prevention strategies. Further research is warranted to continue to evaluate concussion awareness in team and match officials at all levels of sports participation.

Personal protective equipment such as strapping, head-gear and shoulder pads are worn at the discretion of the player as long as they comply with rugby league safety requirements.[469] Mouthguards are a mandatory requirement for all rugby league players in New Zealand entering the field of match play.[469] The finding that 74% of players who were concussed were not wearing a mouthguard is a concern. This may be reflective of the level of monitoring of players in contact sports by team management and match officials and the need for further education of players. Although mouthguards have been shown to have a protective benefit for orofacial injuries, the evidence for protection against concussions is based on limited case series studies and cross sectional non-randomised surveys.[113] The use of properly fitted pressure laminated mouthguards has been suggested to reduce the severity of concussions. Mouthguards with a density that produces a posterior jaw separation of 3 mm to 4 mm have been suggested that they reduce the impact forces involved in concussions.[621] To date there are no published studies providing evidence that standard or fitted pressure laminated mouthguards decrease the rate, or severity, of concussions.[113] Further research is warranted to examine the effects of mouthguards on concussion incidence and severity with players that wear mouthguards in comparison with those that don't.

In rugby union[578] nearly a third of players surveyed were aware of situations where players continued to play on due to peer pressure; 76% believed a team mate had been concussed and stayed on and 31% believing that this was due to pressure. When returning-to-play from a concussion 52% of players surveyed reported they did not adhere to a return-to-play protocol nor sought medical clearance to return-to-play.[578] This may be similar to the amateur rugby league community with limited resources available to assess and manage concussions. Further studies are warranted to explore the concussion knowledge of amateur sports participants.

## Conclusion

The information contained in this technical report addresses two main areas in relationship to sport-related concussion in amateur rugby league. Firstly the knowledge and management of concussion of amateur rugby league players is a concern. The finding that less than 10% of players reporting a concussion from rugby league activities are seen by a health professional raises concerns about the management by team management and match officials. Of greater concern is the finding that even less players reporting having had a concussion undertakes the identified mandatory stand down period and are medically cleared to return to match activities.

The second area addressed is the reporting of the incidence of concussions in amateur rugby league team over three consecutive seasons. No published study to date has undertaken to report specifically on rugby league concussions and the finding of an incidence of 13 per 1,000 match hours is within the range of previous studies. By utilising the SCAT2 as the sideline paper neurocognitive assessment tool for the concussions identified or reported the management of these injuries was able to be commenced. Ball

carriers recorded more concussions than the tacklers. Tackle technique training skills may be useful in reducing the incidence of concussion in amateur rugby league.

As shown by this report, the incidence of reported concussion is within the range of published studies but when reviewing the player concussion data there is an identified lack of knowledge of what a concussion is. Concerning was the finding that players reporting never having a concussion did have loss of consciousness, “dings” and had concussion related symptoms. This may be reflective of the lack of awareness of what a concussion is at both the player and the team management level in amateur rugby league.

# Appendix II: Technical Report to NRL / NZRL


# Sports-related concussions in rugby league: The magnitude of the problem and evidence for impact assessment

**A report for the National Rugby League and New Zealand Rugby League**

22nd March 2015

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# Overview

This technical report is provided to the National Rugby League (NRL) and the New Zealand Rugby League (NZRL) to enable consideration of the magnitude of the concussion problem and consideration of the evidence for impact assessment. Specifically the research evidence for the following topics are covered:

1. Literature analysis of the magnitude of the concussion problem in rugby league.
2. Field research data describing the frequency, magnitude and distribution of impacts to the head in junior and senior rugby league measured via instrumented patches.
3. Field research data describing the sideline concussion assessment in junior rugby league using the King-Devick test.

**Key findings are:**

- Rugby league has the highest percentage of sport code total costs and mean costs per ACC claim for concussion (92.2%; $25,545).
- Semi-professional participants had nearly a two-fold greater concussion injury rate than professional participants and a three-fold greater concussion injury rate than amateur participants.
- Throughout the 2014 match competition season, there were 1,977 impacts recorded via accelerometer patches applied behind the ear of 19 junior players. The youth players were exposed to impacts that can be considered high-magnitude at any level of participation. Despite the number of high-magnitude impacts recorded, there were no witnessed concussive events throughout the duration of the study.
- There were 13,895 impacts recorded via accelerometer patches applied behind the ear of 38 senior players. There was an average of 56 impacts over 10*g* per game over the course of the season. The majority (41%) of impacts occurred to the side of the head. The cumulated impacts per player, per match were 56 ±22 and 731 ±289 respectively.
- The King-Devick test was able to identify junior players with functionally-detected cognitive impairment without any observable concussion symptoms.

**Based on these findings we recommend:**

- Player and team management education surrounding prevention, identification and assessment of concussion is essential.
- Baseline and post-match assessment of all players using the King-Devick is required.
- Prospective studies to determine the cumulative impact loads on players using the accelerometer patches applied behind the ear and concussion outcomes is required.
- Validation studies using the accelerometer patches are needed.
- Studies using players at all levels of participation are needed (particularly juniors, females and Maori).

# Study summaries

**A brief history of rugby league and the magnitude of the concussion problem**

Rugby league in New Zealand was the catalyst for the establishment of the game internationally from the 1905 'All Gold's' tour through Australia, England and Wales. Although different to rugby union in the modern version, league draws its roots from the game. As a result of breaking away from rugby union, league has rapidly expanded in New Zealand as a sport, as people saw it as a faster, skilful and entertaining form of rugby. Given the increasing participation in league it is important to ensure performance can be improved whilst minimising risk of injury. Several epidemiology studies have been conducted to determine the magnitude of the sport-related concussion problem in New Zealand. Rugby league (92.2%; $25,545) recorded the highest percentage of sport code total costs and mean costs per ACC claim for concussion. Males participating in rugby league recorded the highest mean costs per moderate-to-severe claim ($26,182). Over the 2001-11 period, New Zealand Māori recorded the highest mean costs per moderate-to-severe claim for rugby league ($43,604). People participating in rugby league activities in the 30-39 age group recorded the highest total ($2,268,398) and mean cost ($48,211) per moderate-to-severe claim.

**Concussions as a result of participation in rugby league activities**

A systematic literature analysis of the magnitude of the concussion problem in rugby league was performed. Data were extracted and pooled from 23 studies that reported the number and incidence of concussions in rugby league match and training activities. Amateur rugby league players had the highest incidence of concussive injuries in match activities while semi-professional players had the highest incidence of concussive injuries in training activities. Semi-professional participants had nearly a two-fold greater concussion injury rate than professional participants and a three-fold greater concussion injury rate than amateur participants.

**Frequency, magnitude and distribution of head impacts in junior and senior rugby league measured via an instrumented patch**

Head impacts and concussive injuries have become a growing interest in the media, sports medicine and academic environments. Repetitive impacts have been reported to be associated with long term consequences such as Dementia, Alzheimer's and Chronic Traumatic Encephalopathy. We wanted to quantify the frequency, magnitude and distribution of head impacts in junior rugby league. A junior rugby league team (19 players) was monitored with the X2Patch behind the left ear of every player during match activities. The number of impacts and the linear and rotational forces involved were used to calculate the risks associated with every impact, and the cumulated impacts per player, per match and per season. Calculations of injury risk, injury severity and risk weighted exposure were provided. Throughout the 2014

match competition season, there were 1,977 impacts recorded. There were 28 impacts above 80g. This impact magnitude is high and highlighted that, although these were youth players, they were exposed to impacts over a competition duration that can be considered high-magnitude at any level of participation. Despite the number of high-magnitude impacts recorded, there were no witnessed concussive events recorded throughout the duration of the study. Mean linear acceleration measured over the season of matches was similar to mean linear accelerations reported in studies for American Pop Warner and Youth football. Mean rotational accelerations measured were higher than American Pop Warner, youth and high school football. Further studies should explore impacts at the senior, female and professional levels of participation. Although we reported the data by means (±SD), median and 95$^{th}$ percentile to enable comparisons across published studies, there is a need for standardization of reporting head impact biomechanics to enable accurate comparisons.

Senior rugby league players are stronger and faster and likely to impact other players with more force in tackles than junior players. Therefore given our work with junior players, we wanted to extend this work to quantify the frequency, magnitude and distribution of head impacts in senior rugby league. Thirty eight players wore the XPatch behind the left ear for every match competed over the pre-season and competition season. The number of impacts and the linear and rotational forces involved were used to calculate the risks associated with every impact, and the cumulated impacts per player, per match and per season. There were 13,895 impacts recorded. There was an average of 56 impacts over 10*g* per game over the course of the season. The majority (41%) of impacts occurred to the side of the head which is not the same as other studies reporting impacts in American high school and collegiate football. Median resultant linear accelerations were lower than high school and collegiate American football while the median resultant rotational accelerations were higher. The cumulated impacts per player, per match were 56 ±22 and 731 ±289 respectively. Although we reported the data by means (±SD), median and 95$^{th}$ percentile to enable comparisons across the studies to be conducted, the need for standardization of reporting head impact biomechanics is needed.

**Use of the King-Devick test in sideline assessment of concussion in junior rugby league**

The King-Devick test is a rapid number naming reading timed-test that assesses changes in the saccades or fast eye-movements of the player based on previous baseline assessments. Areas of the brain involved in saccades are widespread and can be affected without any observable concussion signs and symptoms being recorded. To date, there has been no published research surrounding the use of the King-Devick test in junior sports participants. Therefore we wanted to quantify King-Devick base line values for junior rugby league players. Nineteen junior rugby league players were assessed pre-season and after each match or when there was parental concern of a concussive injury. The King-Devick test was able to identify players with functionally-detected cognitive impairment without any observable concussion symptoms.

# Introduction

Participation in rugby league activities in New Zealand is largely amateur, with only one professional team (The New Zealand Warriors). Rugby league unfortunately has a high injury rate. Recent Accident Compensation Corporation (ACC) reports have identified that the rugby league injury rate is increasing. This has socioeconomic ramifications in terms of familial support, social interactions and employment responsibilities. There are also financial implications for the individual, the community, society and the government through lost income and rehabilitation costs. To minimize the incidence and severity of injuries there is a need for education of participants on the nature and cause of injuries, and evaluation of injury prevention strategic that could be put in place.

This technical report is provided to the National Rugby League (NRL) and the New Zealand Rugby League (NZRL) to enable consideration of the magnitude of the concussion problem and consideration of the evidence for impact assessment. Specifically the research evidence for the following topics are covered:

1. Literature analysis of the magnitude of the concussion problem in rugby league.
2. Field research data describing the frequency, magnitude and distribution of impacts to the head in junior and senior rugby league measured via instrumented patches.
3. Field research data describing the sideline concussion assessment in junior rugby league using the King-Devick test.

This technical report is part of the ongoing PhD research being undertaken by Dr Doug King on concussion and head impacts through the Sport Performance Research Institute New Zealand at Auckland University of Technology.

# Part 1: A brief history of rugby league and the magnitude of the concussion problem

## Overview

Rugby league in New Zealand was the catalyst for the establishment of the game internationally from the 1905 'All Gold's' tour through Australia, England and Wales. Although different to rugby union in the modern version, league draws its roots from the game. As a result of breaking away from rugby union, league has rapidly expanded in New Zealand as a sport, as people saw it as a faster, skilful and entertaining form of rugby. Given the increasing participation in league it is important to ensure performance can be improved whilst minimising risk of injury. Several epidemiology studies have been conducted to determine the magnitude of the sport-related concussion problem in New Zealand. Rugby league (92.2%; $25,545) recorded the highest percentage of sport code total costs and mean costs per ACC claim. Males participating in rugby league recorded the highest mean costs per moderate-to-severe claim ($26,182). Over 2001-11 period, New Zealand Māori recorded the highest mean costs per moderate-to-severe claim for rugby league ($43,604). People participating in rugby league activities in the 30-39 age group recorded the highest total ($2,268,398) and mean cost ($48,211) per moderate-to-severe claim.

## Introduction

Following the now historic meeting in 1895 at the George Hotel in Huddersfield, a schism ensued with the Rugby Football Union (RFU). This schism occurred when 22 clubs broke away from RFU control and established the Northern Rugby Football Union.[162, 242, 627] The development of the 'Northern Union' (NU) was the start of professionalism in sport. Under NU rules players were compensated for wages lost as a result of sports participation.[162, 242, 534, 627] The NU implemented new rules resulting in changes such as: (1) the number of players reduced to 13 on the field; (2) elimination of the line out; (3) implementation of a different scoring structure; and (4) introduction of a 'play the ball' stopping the use of 'rucking' and 'mauling' for possession of the ball as in RU.[383] These changes continued over time into the game now known as rugby league.[196] Played by both junior and senior level competitors, rugby league is played in countries such as Australia, New Zealand, Scotland, Ireland, France, Russia, United States of America, Wales, Papua New Guinea, Fiji, Samoa and South Africa.[41, 448] Participants range in age from six to sixty years in amateur (male[187, 319, 322, 518] and female[186, 318]), semi-professional [312, 313, 322] and professional (male[218, 224, 275, 484, 571]) level competitions.

## Rugby league in New Zealand

In 1907 Albert Henry Baskerville, a postal clerk, organised a Professional All Black tour to go to England to play Northern Union rules against Northern Union sides.[162, 242, 627] Known as the "Pro-Blacks" [162, 242, 627] or "All Gold's",[534] the tour team of consisted of 27 players from around New Zealand, including nine current All Black rugby union internationals.[534] The team travelled to Australia and played three matches in New South

Wales before heading to England with Australian Henry Herbert (Dally) Messenger who was invited to tour with the team.[534] Competing in 29 club based matches, three test matches and two international matches in England and Wales, the All Gold's returned to Australia leaving four players behind in England, having secured professional club contracts.[534]

Returning to Australia the All Gold's played three more club based matches before they played Australia in the first test match.[534] Following the win (11-10) by the All Gold's they travelled to Brisbane for the second test. Unfortunately, on route to Brisbane, Baskerville developed a "chill" and subsequently died from complications.[534] Despite the loss of their team mate and friend, the team played Australia in Brisbane winning 24 -12 before departing back to Sydney for the third test.[534] Losing this test (14-9) the All Gold's headed back to New Zealand having brought Northern Union rules to the international domain in England, Wales and Australia.

The first rugby league match played in New Zealand was on Saturday 13th June 1908 at Wellington's Athletic Park.[162, 242, 534] Organised as a benefit match for the widowed mother of Albert Henry Baskerville, the match was promoted as "rugby as it is played in England".[242] The match was watched by between 6-7,000 spectators and drew praise by newspapers as an improvement on rugby union rules, producing a more interesting, skilful and much faster game.[242]

Following this match, groups of enthusiasts attempted to start the new game in towns and cities throughout New Zealand and, by the end of 1908 representative teams had been in organised in Southland, Otago, South Canterbury, Wellington, Taranaki and Auckland.[162, 242] A Maori team was also formed and they competed in matches in New South Wales and Queensland, Australia in 1909.[162, 242] The regional districts also formulated governing bodies to cater for the games in their districts.[162, 242] But it was not until 1910 that the national governing body, the New Zealand Rugby League was formulated.[162, 242]

The New Zealand Rugby Union reacted to the tour, its own players defection and the development of rugby league in New Zealand in several ways.[162, 242] The first reaction was to disqualify players who partook in rugby league from any rugby union activity, building or sports ground. Rugby union also endeavoured to stifle the development of rugby league by trying to have municipally own sports grounds denied to rugby league.[162, 242] By pressuring municipal councils into believing that they would disqualify a ground as professional and not allow rugby union to be played there, some councils denied requests by rugby league clubs to play in municipal areas.[162, 242] Despite these endeavours, in the early years rugby league grew as a sport, with people seeing it as a faster, more skilful and entertaining form of rugby.

### What is rugby league?

Rugby league is a team sport that consists of 13 players. Junior and amateur rugby league matches are typically (but not always) played under an unlimited interchange rule whereas professional/elite and semi-professional/sub-elite rugby league teams utilise a limited interchange rule. Up to 12 interchanges are permitted in these competitions. Each team is permitted six tackles with the ball and they must advance

down the field into the opposition's territory and score a try.[185, 218, 225] The ball must be passed backwards but can be carried or kicked into the opposition's territory.[185, 225] At the completion of the six tackles, the ball is immediately given to the opposition team to commence their set of six tackles.[185, 218] The same players are therefore involved in both attack and defence.

The game is played under different rules depending upon participation age. Children aged less than nine years old play under mini-modified rules requiring a half sized field of play. There is no tackling, no kicking of the ball and no contested scrums in this age group.[95, 302] Children between the ages of 10 and 12 years old also participate in a modified rules version of the game. This requires that the matches are played on three-quarter sized fields, allow tackles to be made, have limited kicking and they can contest for the ball in a scrum as in the full version of rugby league.[95, 302] For participants over the age of 13 years of age, the game is participated under the international rules.

Similar to rugby union, the rugby league team consists of two main groups of participants (six forwards and seven backs) on the field and four reserves.[82, 184, 448] The demands on the participants vary according to the specific positions played[82, 184, 448] with forwards (prop n=2, hooker n=1, second row n=2 and lock n=1) more predominately involved in large numbers of physical collisions and tackles.[180] Backs (half-back n=1, stand-off n=1, centre n=2, wing n=2 and fullback n=1) spend more time in free running but are involved in tackles and collisions.[180] These are sometimes classified into four subgroups reflecting positional commonality (i.e. props, hookers and halves, back-rowers and outside backs).[82, 196, 448] The demands placed on the participants vary according to the specific position played during match participation.[184, 230]

As with all sport, participating in rugby league activities carries injury risk. The game is intermittent in nature requiring participants to compete in a challenging contest. Players competing in rugby league often undergo frequent bouts of high intensity activity (e.g. tackling, sprinting, running and passing) interspersed with short bouts of low intensity activity (e.g. jogging, walking and standing).[41, 97, 196, 447] Because of the intermittent nature of the game, the physiological demands of rugby league are complex. Players are required to have maximal aerobic power, speed, muscular strength and power and agility developed appropriately to compete in the match environment.[41, 97, 196, 447] As a result of the physical requirements and intense nature of the game, musculoskeletal injuries are commonly reported.[179, 185, 196]

### The magnitude of the sport-related concussion problem in New Zealand

Several epidemiology studies have been conducted to determine the magnitude of the sport-related concussion problem in New Zealand. Moderate-to-severe (MSC) Accident Compensation Corporation (ACC) claims and costs for concussion have been quantified for 2001 to 2011.[326] Of the seven sports codes, rugby league recorded a low number of MSC claims but the highest mean cost per claim. Although MSC injury claims were only 6.4% of total claims, they accounted for 79.1% of total costs indicating that although the majority of sport-related concussions may be minor in severity, the related economic costs associated with more serious sport-related concussion can be high.

Rugby league (92.2%; $25,545) and netball (86.6%; $11,371) recorded the highest percentage of sport code total costs and mean costs per ACC claim. Males participating in rugby league recorded the highest mean costs per MSC claim ($26,182). Over 2001-11 period, New Zealand Māori recorded the highest mean costs per MSC claim for rugby league ($43,604). People participating in rugby league activities in the 30-39 age group recorded the highest total ($2,268,398) and mean cost ($48,211) per MSC claim.

A reason for high concussion claim numbers in sport may be related to the focus on concussion identification and management education resulting in increased reporting. Rugby league had the third highest number of claims (n=179) for reported concussions over the ten years. However, the overall knowledge level of concussion identification and management was only 42% for rugby league team coaches and trainers in New Zealand which was lower than other studies reporting concussion knowledge of team coaches (62%[601] to 84%[477]). Of 95 rugby league coaches and team managers, 26% reported they would not remove a player with symptoms of concussion, 20% would return a symptomatic player to participation and 39% reported that concussion only occurred when a player lost consciousness.[331] With the number of reported concussions in this study, and given the lack of knowledge surrounding concussion identification and management previously reported there is possibly an under-reporting of concussion in rugby league. Recently it has been reported that un-witnessed concussive events were identified with the use of a saccadic reading test in rugby league[317] and rugby union.[314] The number of un-witnessed to witnessed concussions occurred at a 3.4 to 1 ratio.[314] If this is indicative of the true incidence then the reported numbers, and costs in this study may only be a third of the actual incidence and economic costs associated with sport-related concussions.

Of concern was that rugby league recorded a low number of MSC claims but the highest mean cost per claim. In particular, males and Māori participating in rugby league activities recorded the highest mean costs of all sports. This may be reflective of the nature of rugby league when compared with rugby union or the management of these types of injuries. For example, the differences in the types and numbers of tackles between the two sports may expose players in rugby league to more head impacts resulting in more severe head trauma and therefore more cost per claim. Studies have reported that contact with the head during the tackle occurred at a rate of 0.6 per 1,000 tackles in rugby union[516] but at a rate of 89.9 per 1,000 tackles in rugby league.[332] This higher rate of contact with the head in rugby league may be reflected in the higher costs recorded in the current study. Further studies are warranted to explore the incidence of impacts that occur and the management of these injuries in rugby union and rugby league. A conclusion from this study was that Māori rugby league males aged between 30 and 39 years, and female players need to be targeted for injury prevention initiatives.

Epidemiology evaluation by ethnic groups has also been conducted[337] for New Zealand rugby league injuries requiring medical treatment and associated costs using ACC data for 1999 to 2007. New Zealand Maori accounted for 39.8% of the number of total injury claims and 43.5% of the total injury entitlement costs but were recorded as only 13.2% of the total New Zealand population. Accounting for only 3.2% of the population

distribution living in Auckland, New Zealand Maori recorded 11.7% of the total injury claims in the Auckland district. Soft tissue injuries accounted for 10.6% (±8.5%) of injury claims and 7.9% (±6.7%) of injury entitlement costs for all ethnic groups. New Zealand Maori recorded more injury claims for the knee than all other ethnic groups. Injury claims for New Zealand Europeans recorded more trade occupations, New Zealand Maori more plant and machinery occupations and Pacific peoples more elementary occupations. New Zealand Maori recorded significantly more injury claims for both males and females than all other ethnic groups over the study period. This study identified that NZ Maori are disproportionately participating in rugby league in NZ, but the proportions injured are consistent with reported proportions playing the game.

# Part 2: Concussion in rugby league: A pooled analysis

## Overview

A systematic literature analysis of the magnitude of the concussion problem in rugby league was performed. Data were extracted and pooled from 23 studies that reported the number and incidence of concussions in rugby league match and training activities. Amateur rugby league players had the highest incidence of concussive injuries in match activities (22.9 per 1,000 match hours) while semi-professional players had the highest incidence of concussive injuries in training activities (3.1 per 1,000 training hours). Semi-professional participants had nearly a two-fold greater concussion injury rate than professional participants and a three-fold greater concussion injury rate than amateur participants.

### Background

Rugby league has a high incidence of injury, especially when compared with rugby union.[545] Injury incidence in rugby league increases with increasing participation level.[180] In reviews of match and training injuries in rugby league,[180, 185, 338] match injuries varied from 1[517] to 825[179, 180] per 1,000 match hours while training injuries ranged from 12.2[276] to 106[182] per 1000 training hours. Limitations to these individual studies are that they rely on small numbers of players, typically are only from one club or competition, utilize different injury definitions and methodological approaches and are usually conducted over a short duration.[323] The generalizability of these individual studies for the identification of the injury incidence in rugby league is limited.[225]

A strategy previously utilised[225, 328] is to combine the information provided by epidemiological studies into a single estimate,[33, 428] termed a pooled analysis.[76] To achieve this technique it is recommended that all the studies included in the analysis be on common grounds, have similar definitions, have a comparable population and have adequacy and specificity of exposure data.[625] By pooling the data, the information provided can then be statistically re-analysed providing more precise injury data.[33] This has been undertaken on professional rugby league studies[225] and more recently[328] by pooling all the published studies reporting injury incidence at all levels of participation. The professional study[225] only reported the injury incidence for professional rugby league participation was 40 per 1,000 player hr. whereas the more recent study[328] reported both match and training injuries for all levels of rugby league participation. This study[328] reported an injury incidence of 148 per 1,000 match hr. and a training injury incidence of 12.6 per 1,000 training hr.

A systematic qualitative review of concussions in rugby league[207] identified that the incidence of concussion varied widely from 0.0 to 40.0 per 1,000 playing hours depending on the injury definition utilised (time loss vs. no time loss). The incidence rates varied between match and training activities, playing positions and the season (winter vs. summer).[207] Of all concussive injuries, 29% were associated with illegal match activities.[207] Although this review did summarise the available literature qualitatively, no study to date has

undertaken a pooled analysis of concussions in rugby league. Therefore the purpose of this study was to review all published rugby league studies reporting injuries from match and training participation and report the pooled data estimates on rugby league concussion injury epidemiology. In addition this study added information for professional, semi-professional, amateur and junior levels of participation in both the match and training environments. Specifically, this included estimates of concussion injury incidence for the different rugby league participation levels.

## Methods

The methodology utilised in this study was similar to previous pooled analysis studies reporting rugby league injuries[225, 328] and using the steps as described by Friedenreich.[33, 169]

### Search strategy for identification of databases

Searches of PubMed, CINHAL, Ovid, MEDLINE, SCOPUS, ScienceDirect and SportDiscus® databases were performed to identify studies published in English prior to December 2014. The computer databases provided access to sports-oriented and biomedical journals, serial publications, books, theses, conference papers, and related research published since 1948. Terms utilized for the search of relevant research studies included rugby AND league AND injur*. Bibliographies of all papers retrieved for evaluation were also examined for additional references. All articles that met the inclusion criteria were entered in an EndNote X6.0.1 database.

### Inclusion criteria

Published studies that reported the incidence of injury in rugby league match and training activities were collated. These studies were included in the analysis if they were:

(1) Published later than 1990;
(2) Reported the match or training time exposure enabling calculation of player time injury rates;
(3) Reported concussions;
(4) More than one study reporting injuries at the identified participation level.

### Procedures

As all of the studies included in the pooled analysis were observational in design, two reviewers extracted the study characteristics, numerical data and assessed the quality, by adhering to the protocol for systematic review of observational studies (MOOSE).[575] This approach was utilised as it enables a more precise estimate of effects of influential factors, takes into account confounding factors (participation level and age) and the heterogeneity of the studies.[214] An additional advantage to utilising a pooled analysis approach is that the same statistical model can be utilised with data from methodologically diverse studies.[214]

A total of 1,422 studies were initially identified through the database searches. This consisted of 333 studies identified in PubMed, 43 in CINHAL, 679 in Ovid Medline, 186 in SCOPUS and 181 in Sports Discus (see

Fig 1). Some studies reviewed were initially identified as being eligible for inclusion in the pooled analysis but were excluded due to a variety of reasons. The exclusion of these studies were identified as being: (a) Older than 1990;[2, 3, 611] (b) Reporting of data as means;[197, 309] (c) Non reporting of all injuries or total exposure times;[192-194, 197, 198, 200, 333, 334] (d) Use of a previously reported injury database;[227] (e) A review paper;[180, 185, 207, 219, 284, 336-338, 385] (f) Self-reported injuries;[475] and (g) not reporting concussions.[160, 175, 176, 178, 179, 181, 187, 191, 199, 275, 276, 306, 483, 517]

A total of 23 studies were identified that reported data collection of rugby league injuries. This consisted of 17 studies[182, 184, 187, 189, 218, 223, 226, 228, 312, 316, 318, 320-322, 324, 545, 571] reporting rugby league match injuries and six studies[83, 182, 183, 190, 223, 319] reporting rugby league training injuries (see Table 2.1). Data pertaining to the participation level were reviewed and the studies were grouped into professional, including reserve and elite;[218, 223, 226, 228, 545, 571] semi-professional, including sub-elite;[182-184, 189, 190, 321] amateur[83, 316, 318-320, 322, 324] and junior.[187, 312]

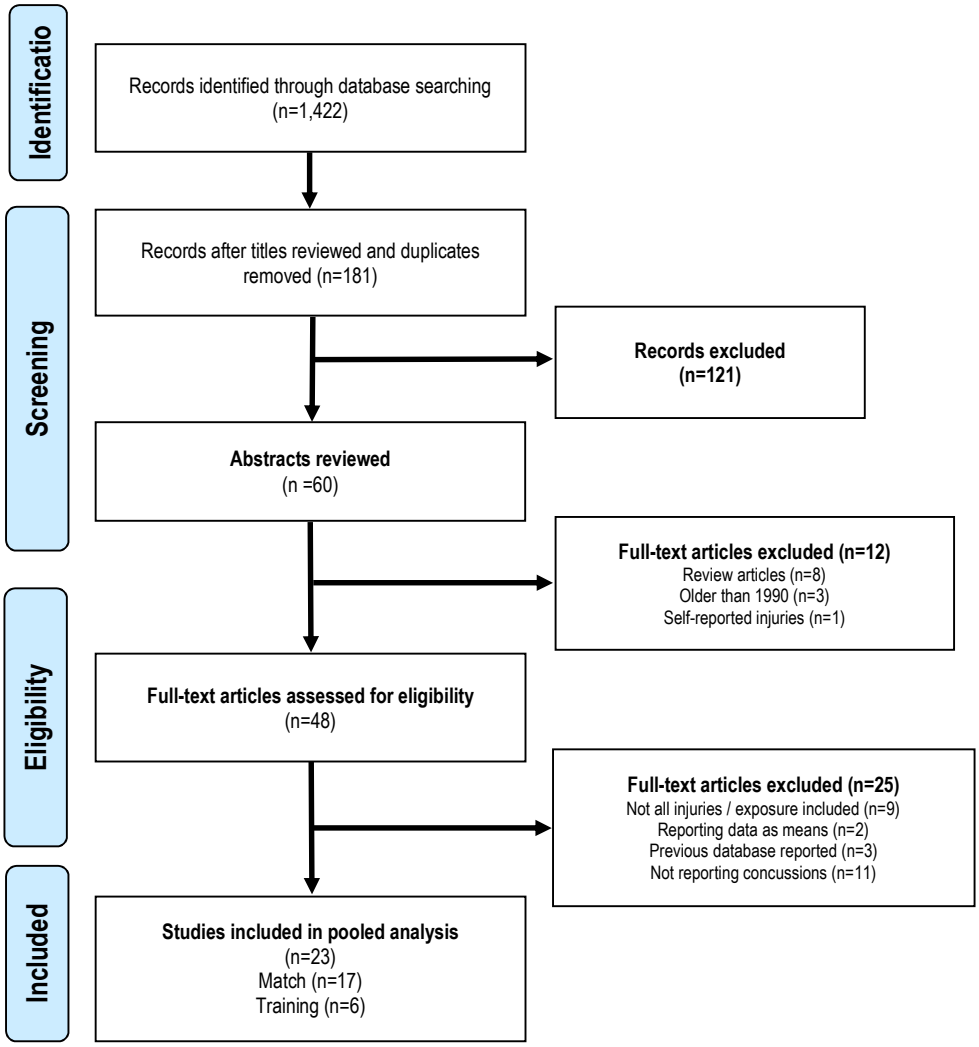

**Figure 2.1:** Flow of identification, screening, eligibility and inclusion for the pooled analysis of match and training rugby league concussion injuries.

Many of the identified studies did not include all the areas of interest for the pooled analysis. So, specific information was extracted from individual studies on an 'as-required' basis throughout the analytical process and to exclude data if there were less than two studies reporting at the same participation level. Not all studies reported concussions limiting the evaluation of these areas to the total match and training injuries recorded. Injury severity was pooled for the number of injuries recorded in both match and training session studies in this analysis.

**Assessment of study quality**

All studies meeting the selection criteria were assessed for quality based on modified previously published checklists.[575] Quality was described as the confidence that the design, conduct and analysis of the study was such that it minimised bias in the estimation of the factors associated with injury on the outcome measures.[377] The quality assessment criteria utilised in this study have been previously identified.[377]

**Statistical analysis**

The data from the individual studies were combined as previously described.[225] Incidence rates and 95% confidence intervals (CI) were calculated. To compare between injury rates, risk ratios (RRs) were used. The RRs were assumed to be significant at $p<0.05$. To test for significant difference, chi-squared ($\chi^2$) goodness-of-fit tests were utilised.

## Results

The studies reported match injury data from 2,431 match events totalling 40,425.6 match exposure hr. (Professional: 1,658 matches; 28,728.5 match hr; Semi-professional: 561 matches; 8,779.9 match hr; Amateur: 114 matches; 1,484.7 match hr; Junior: 98 matches; 1,432.5 match hr). Studies reporting training injury data were drawn from 196,524.7 training exposure hr. (Professional: 167,793.5 training hr; Semi-professional: 14,202.4 training hr; Amateur: 14,528.8 training hr). There were no published studies that reported junior training injuries. Overall quality of the studies included in the pooled analysis was good (median 5/6; range 4-5). Not all studies were prospective and no study had a blinded outcome.

The pooled analysis concussion injury incidence for match injuries was 8.1 (95% CI: 7.3 to 9.0) per 1,000 match hr. (see Table 2.1). More concussions were recorded in amateur than professional (RR 2.9: 95% CI: 2.0 to 4.1; $p<0.0001$), semi-professional (RR: 3.9; 95% CI: 2.5 to 5.9; $p<0.0001$) and junior (RR: 3.3; 95% CI: 1.6 to 6.6; $p=0.0005$) studies. The pooled analysis concussion incidence for training injuries was 0.2 (95%CI: 0.2 to 0.3) per 1,000 training hr. (see Table 2.1). There were more concussions recorded in semi-professional than amateur (RR: 15.0; 4.7 to 48.3; $p<0.0001$) and professional (RR:519.8; 95% CI: 71.6 to 3,772.8; $p<0.0001$) training session studies. Semiprofessional rugby league participants recorded a two-fold

risk ratio (RR: 1.9; 95% CI 1.3 to 2.9; *p*=0.0013) when comparing match and training concussion incidence (see Table 2.2).

**Table 2.1:** Pooled analysis of concussions in rugby league for match and training exposure activities by participation level by number of concussions reported, total exposure hours and rate per 1,000 hours with 95% confidence interval (CI).

| | No. | Hours | Rate (95% CI) |
|---|---|---|---|
| **Match reported concussions[a]** | | | |
| **Total** | **327** | **40,425.6** | **8.1 (7.2 to 9.0)** |
| Gender | | | |
| Male[182, 184, 187, 189, 218, 223, 226, 228, 312, 316, 320-322, 324, 545, 571] | 325 | 40,096.5 | 8.1 (7.2 to 9.0) |
| Female[318] | 2 | 329.2 | 5.1 (1.3 to 20.4) |
| Level of participation | | | |
| Professional[218, 223, 226, 228, 545, 571] | 231 | 28,728.5 | 8.0[c] (7.1 to 9.2) |
| Semi-Professional[182, 184, 189, 321] | 52 | 8,779.9 | 5.9[c] (4.5 to 7.8) |
| Amateur[316, 318, 320, 322, 324] | 34 | 1,484.7 | 22.9[abd] (16.4 to 32.0) |
| Junior[187, 312] | 10 | 1,432.5 | 7.0[c] (3.8 to 13.0) |
| **Training reported concussions[b]** | | | |
| **Total** | **48** | **196,524.7** | **0.2 (0.2 to 0.3)** |
| Gender | | | |
| Male[83, 182, 183, 190, 223, 319] | 48 | 196,524.7 | 0.2 (0.2 to 0.3) |
| Level of Participation | | | |
| Professional[223] | 1 | 167,793.5 | 0.01[b] (0.00 to 0.04) |
| Semi-Professional[182, 183, 190] | 44 | 14,202.4 | 3.1[ac] (2.3 to 4.2) |
| Amateur[83, 319] | 3 | 14,528.8 | 0.2[b] (0.1 to 0.6) |

CI = Confidence interval; (a) = rate reported per 1,000 match hr.; (b) rate reported per 1,000 training hr. Significant difference ($p<0.05$) than (a) = Professional; (b) = Semi-professional; (c) = Amateur; (d) = Junior.

**Table 2.2:** Risk ratio of match to training concussion injuries for professional, semi-professional and amateur rugby league participants with 95% confidence intervals.

| Participation level | RR (95% CI) | $\chi^2$; (df=1), | *p* value |
|---|---|---|---|
| Professional[218, 223, 226, 228, 545, 571] | 1,349.2 (189.3 to 9,618.66) | 1,341.4 | <0.0001 |
| Semi-Professional[182-184, 189, 190, 321] | 1.9 (1.28-2.85) | 10.36 | 0.0013 |
| Amateur[83, 316, 318-320, 322, 324] | 110.9 (34.1 to 360.7) | 300.3 | <0.0001 |

CI = Confidence interval

## Discussion

The aim of this study was to review and update the analysis of rugby league injuries specifically looking at the incidence of concussion injuries. The current study encapsulates a broad spectrum of published rugby league studies and incorporates both match and training concussion injuries at professional, semi-

professional and amateur levels of participation. Studies reporting women's[318] match and junior training rugby league injuries would have been included, but there was a paucity of published studies in these areas suitable for inclusion to enable a pooled analysis. Future studies are warranted to explore these groups of rugby league participants.

The use of a pooled analysis has been previously applied to rugby league injury epidemiological studies.[223, 225, 328] This approach has been shown to produce an overall estimation of the injuries recorded by combining the data provided by the selected studies.[154] The limitations with the use of a pooled analysis methodology have been previously described[33, 546] and issues such as the difference in study design (observation vs. self-reported injury);[137, 507] injury type, site and severity definitions; data collection methods and times; data recording medium and the maintenance of the data medium were considered and addressed through to identification of the data utilised. Despite these limitations, the strength of a pooled analysis is that it provides more accurate estimates of injury rates than individual studies that provided the data,[225] can be utilised as comparisons against other pooled studies and to obtain a combined estimator of the quantitative effect of the relative risk of injuries in rugby league match and training activities.[33, 169]

The major findings of this pooled analysis for studies of concussion occurring in match and training activities were: (i) Semi-professional participants had nearly a two-fold greater concussion injury rate than professional participants and a three-fold greater concussion injury rate than amateur participants; and (ii) Semi-professional participants had a 500-fold higher concussion injury rate than professional participants, and a 15-fold higher concussion injury rate than amateur participants.

It was not unexpected to find that professionals had a 1,300-fold and amateurs had a 110-fold decrease in the incidence of injuries occurring when comparing match and training injury incidence. What was unexpected was the finding that semi-professional participants only had a 2-fold decrease in the incidence of injuries comparing the match and training injury incidence. It has been reported that semi-professional participants have superior physiological capabilities than amateur participants[177, 179] producing a higher playing intensity that may result in a higher injury incidence.[179, 218, 227, 571] However, professional players also have greater physiological attributes than amateur participants and a higher skill level than semi-professional participants[179] and the decrease between match and training injuries is greater than for amateur participants. These differences may be a result of the approach to training activities at the different levels of participation. While professional players may undertake more regular training sessions than amateurs, they likely moderate their training sessions so that injury does not limit their ability to appear in competition. Amateur players partake in less regular training activities but rely on other employment as their source of income,[319] so any injuries that occur may have a direct impact on their financial income. Semi-professional players have a mix of payment for playing and may also utilise another source of employment for income.[179] Over-exposure to a diverse range of physical activities may place them at a higher risk of an injury occurring. Further studies are warranted to explore the issues that surround semi-professional players and the

incidence of both match and training injuries such as employment type, training hours and coaching styles and how these may be reduced.

This pooled analysis showed that in match activities amateur rugby league participants have a higher reported concussion injury rate than professional and semi-professional participants. A limitation with using the pooled analysis for the identification of concussion injuries is that there is no data to further analyse where, when or what player positions are affected when the concussions occur during the match and training activities. Further studies are warranted to further explore the incidence of concussion injuries in all levels of rugby league participation and should include a more detailed analysis of the time, activity and player position to assist with injury prevention programmes.

### Conclusion

A combined estimate of injuries within a specific sport through pooled analysis provides more precise evidence and meaningful information about the sport, whilst controlling for between-study variation due to individual sub-cohort characteristics. This synthesis has illuminated differences in concussion injury rates at several levels in the game, along with information about the differences in the incidence of concussions between playing and training for rugby league. Semi-professional participants had nearly a two-fold greater concussion injury rate than professional participants and a three-fold greater concussion injury rate than amateur participants.

## Part 3: Head impacts in a junior rugby league team measured with an instrumented patch: An exploratory analysis.

### Overview

Head impacts and concussive injuries have become a growing interest in the media, sports medicine and academic environments. Repetitive impacts have been reported to be associated with long term consequences such as Dementia, Alzheimer's and Chronic Traumatic Encephalopathy. We wanted to quantify the frequency, magnitude and distribution of head impacts in junior rugby league. A junior rugby league team (19 players) was monitored with the X2Patch behind the left ear of every player during match activities. The number of impacts and the linear and rotational forces involved were used to calculate the risks associated with every impact, and the cumulated impacts per player, per match and per season. Calculations of injury risk, injury severity and risk weighted exposure were provided. Throughout the 2014 match competition season, there were 1,977 impacts recorded. There were 28 impacts above 80g. This magnitude is high and highlighted that, although these were youth players, they were exposed to impacts over a competition duration that can be considered high-magnitude at any level of participation. Despite the

number of high-magnitude impacts recorded, there were no witnessed concussive events recorded throughout the duration of the study. Mean linear acceleration measured over the season of matches was similar to mean linear accelerations reported in studies for American Pop Warner and Youth football. Mean rotational accelerations measured were higher than American Pop Warner, youth and high school football. Further studies should explore impacts at the senior, female and professional levels of participation. Although we reported the data by means (±SD), median and 95$^{th}$ percentile to enable comparisons across published studies, there is a need for standardization of reporting head impact biomechanics to enable accurate comparisons.

## Introduction

Sports related concussion has received increased media and public awareness with concern for player safety and the risk of injury.[84, 115, 626] Knowledge about the potential metabolic and ultrastructural consequences of impacts to the head has grown, as has the appreciation for repetitive concussive and subconcussive impacts and the possible deleterious effects in some individuals.[626] The use of technology such as accelerometers in the helmets of American football players,[50, 110, 115] the analysis of injury biomechanics has increased the knowledge of the forces, accelerations, frequencies and velocities of head injuries.[626] This knowledge can be applied to circumstances where head injuries can occur.

Youth football players may not have any meaningful impacts to the head because the players are smaller, have a lower impact velocity and participate less.[626] However the risk of a concussive injury, as a result of the impacts that occur from sports activities such as football, for younger players is higher when compared with adults.[116] As well as a higher risk of a concussive event, younger players can also have a prolonged recovery process when compared with adults.[165] In youth American football (6 to 9 yr.) there was a reported average of 107 impacts per-player per-season with an average linear and rotational acceleration of 18g and 901 rad/s$^2$. Most impacts occur during practice (59%) and have a higher magnitude than those recorded in matches.[115] In reporting on Pop Warner "Junior Midgets" football players (12 to 13 yr.), 480 impacts were recorded during matches with an average linear acceleration of 47 ±14g.[626] This was slightly higher than middle school football players (12-14 yr.) where the match median linear accelerations were 21 ±3g.[116] Although the youth players are smaller and had fewer head impacts than the older players, they did record high magnitude impacts and the long term implications of this in an exposure paradigm are unknown.[626]

Despite more studies reporting impacts to the head and concussions, none have identified a data acquisition limit that constitutes a subconcussive impact. Impacts under 10g are reported[473] to be a result of non-contact activities such as walking, jumping, running and sitting.[110, 449] Impacts over 10g that do not result in a participant presenting with acute signs or symptoms of concussion, are identified as subconcussive impacts[13] and repetitive subconcussive impacts may have negative long-term effects.[566] Several studies[50, 84, 626] used data acquisition limits greater than 10g and may have removed subconcussive impacts from their

dataset. The data obtained from these types of impacts is not normally distributed. Using a data acquisition limit greater than 10g may remove a large amount of impacts that could be subconcussive impacts. The data acquisition limit of 10g was therefore selected by us to identify impacts that were considered to have occurred from impact accelerations enabling elimination of activities undertaken in daily living.

Non-helmeted sports have also utilised accelerometers in measuring impacts from sports participation. Heading the ball by female soccer youths yielded peak accelerations of 63g and 8,869 rad/s$^2$.[261] No concussions were reported,[261] as no injury nor injury risk was assessed; even though some of the rotational accelerations were within the nominal values for an injury to the head when compared with NFL data[498] and injury risk tolerance levels.[632] Although data are accumulating for soccer and NFL there are no published data for non-helmeted collision sports such as rugby league. The objective of this study was to investigate the head impact exposure during matches in order to elucidate the frequency, magnitude and distribution of head impacts in junior rugby league players over a junior season of matches in New Zealand.

## Methods

### Experimental approach to the problem

A prospective observational cohort study was conducted on a junior club level rugby league team during the 2014 competition in New Zealand. Nineteen players (males n=14; females n=5) were enrolled in the study with a mean ±SD age, body mass and height of 10 ±1 yr., 55 ±17 kg and 1.5 ±0.1 m. Consent was obtained from the players parents / guardians prior to enrolment. The researchers' University ethics committee approved all procedures in the study (AUTEC 12/156).

All 19 players participated in the modified version of Rugby League (Mod-League) over a single competition season administered through a zonal region under the jurisdiction of the New Zealand Rugby League. The match rules for rugby league have been adjusted to accommodate younger participants (http://www.nzrl.co.nz/pdf/guide_to_the_laws_2010.pdf). The adjustments for this age group (Mods: Under 10 – Under 11 yr.) include a smaller field (80 x 48 m); a smaller ball (270 mm x 162 mm); fewer players (11 per team on field (five forwards (props=2; hooker=1, second row=2) and six backs (Halfback=1, five-eight=1, centre=2, wing=2), or fewer if the opposition has less; two 20 minute halves, no points awarded for a win, no final series and two passes from the play the ball following a tackle. Similar to the adult version of rugby league, the team in possession must carry the ball forward aiming to score a try, but can only pass the ball backwards for a maximum of six tackles before handing the ball to the opposition. Unlike the adult version of rugby league both males and females participated in the same game against teams with a similar mixture of male and female players.

Players wore an instrumented patch behind their ear (XPatch, X2Biosystems, Inc., Seattle, WA, USA) sampling at 1,024 Hz, during every match. The specific details of the accelerometers utilised and the biomechanical measures have been previously reported.[330] The patches recorded head linear and rotational acceleration, impact location and duration. All data were recorded on the X2Biosystem Injury Management

Software (IMS) and transferred to an Excel spreadsheet for further analysis. Over the 2014 domestic junior rugby league match competition season impacts that exceeded our inclusion limit of 10g for a head impact were retained for data analyses. Impacts could be a result of a direct blow to the head, face, neck or elsewhere on the body with an 'impulsive' force transmitted to the head. Each recorded impact was categorised into four general locations (front, side, back and top).[109]

Head impact exposure including frequency, magnitude and location of impacts was quantified using previously established methods.[109, 110] Data were not collected at team trainings due to resource limitations, with priority being quantification of the characteristics of head impacts sustained during rugby league matches in the season. Two measures of impact frequency were computed for each player: player impacts - the total and average number of head impacts recorded for a player during all matches; player position impacts - the total and average number of head impacts recorded for the playing position for all matches. The impact data were analysed by male and female participants.

For the impact to be recorded, 100 ms of data were stored, including 25 ms prior to, and 75 ms following, the impact. Software provided by X2Biosystems calculated the peak linear acceleration, rotational acceleration (x-axis and y-axis angular accelerations), impact location, Head Impact Criterion 15 ms, Gadd Severity Index, and date and time stamp for later download and analyses. Impacts were uploaded and then reviewed to ascertain whether they were from match impacts as verified by video (Sony HDR-PJ540 Camcorder) recorded during the games. Impacts not occurring from match activities were termed 'clacks'. All impacts were assessed through the IMS utilising a "de-clacking algorithm" that involved two methods.[330] All impacts identified as 'clacks' were removed from the dataset prior to downloading for further analysis.

All data were entered into Microsoft Excel and analysed with SPSS V.22.0.0. Impacts were reported as the mean ±standard deviation (SD) per-player per-season of matches, per-player per-match and impact duration (ms). As the impact variables were not normally distributed at all the data acquisition limits (Kolmogorov-Smirnov test; $p<0.001$), the data were expressed as mean ±standard deviation (SD), median and 95$^{th}$ percentile value for impacts recorded for resultant linear and rotational accelerations. These were analysed using a Krusal-Wallis one-way ANOVA with a Dunn's post-hoc test. The risk weighted exposure (RWE) calculations for linear ($RWE_{Linear}$), rotational ($RWE_{Rotational}$) and combined probability (CP) ($RWE_{CP}$)[596] were analysed with a Wilcoxon signed-rank test with a Bonferroni correction applied. Impacts were further divided into one of three categories: (1) Impacts greater than 80g (high); (2) Impacts ranging between 30.0 g and 79.9g (moderate); and (3) Impacts ranging between 10.0g and 29.9g (low).[449].

The impact locations were categorized as front, side, back and top. Impacts to the top of the head were defined as all impacts above an angle 65° from a horizontal plane through the centre of gravity of the head.[241] Impact locations were analysed using a Friedman repeated measures ANOVA on ranks. Post hoc analysis with Wilcoxon signed-rank tests was conducted with a Bonferroni correction applied. Empirical cumulative distributions (CDF) were computed by individual players and total impacts recorded by resultant linear and

rotational accelerations. Our results were then compared to studies quantifying head impact exposure in junior, high school and college American football players.

**Table 3.1:** Impacts to the head greater than 10*g* for total impacts recorded, impacts by forwards and backs in a junior rugby league team (19 players) over a season of matches. Data are presented as mean and standard deviation (±SD), median and 95th percentile for impacts per player per match season, per player per match, impact duration (ms), linear and rotational acceleration and risk weighted exposure by linear, rotational and combined probability.

| Total Impacts | Per-player per-match season | Per-player per-match | PLA (*g*) | | | PRA(rad/s²) | | | $RWE_{Linear}$ | | | $RWE_{Rotational}$ | | | $RWE_{CP}$ | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| n= | Mean ±SD | Mean ±SD | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% |
| **Total Impacts[1]** | | | | | | | | | | | | | | | | | |
| 1,977 | 116 ±74 | 13 ±2 | 22 ±16 | 16 | 57 | 4,041 ±3391 | 2,773 | 11,384 | 0.001 ±0.001 | 0.000 | 0.001 | 0.192 ±0.365 | 0.001 | 1.000 | 0.088 ±0.237 | 0.001 | 0.808 |
| **Forwards Impacts[2]** | | | | | | | | | | | | | | | | | |
| 1511 | 151 ±75 | 15 ±7 | 22 ±16 | 16 | 57 | 3,889 ±3,368 | 2,640 | 11,189 | 0.001 ±0.001 | 0.000 | 0.001 | 0.181 ±0.359 | 0.001 | 1.000 | 0.084 ±0.233 | 0.001 | 0.804 |
| **Backs Impacts[3]** | | | | | | | | | | | | | | | | | |
| 466 | 67±38 | 9 ±5 | 23 ±16 | 17 | 57 | 4,534 ±3,421 | 3,343 | 11,627 | 0.001 ±0.002 | 0.000 | 0.001 | 0.225 ±0.382 | 0.003 | 1.000 | 0.102 ±0.251 | 0.001 | 0.849 |

PLA (*g*) = peak linear acceleration in gravitational force (*g*); PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $RWE_{Linear}$ = Risk Weighted Exposure Linear; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational; $RWE_{CP}$ = Risk Weighted Exposure Combined Probability; (1) = 11 player positions; (2) = 5 players; (3) = 6 players.

**Table 3.2:** Impacts to the head greater than 10*g* for low (<30*g*), moderate (30*g*-80*g*) and high (>80*g*) magnitude impacts for total impacts, impacts by forwards and backs in a junior rugby league team (19 players) over a season of matches. Data are presented as mean and standard deviation (±SD), median and 95th percentile for impacts per player per match season, linear and rotational acceleration and risk weighted exposure by linear, rotational and combined probability.

| | Total Impacts | Per Player Per match season | PLA(*g*) | | | PRA(rad/s²) | | | $RWE_{Linear}$ | | | $RWE_{Rotational}$ | | | RWEcp | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n= | Mean ±SD | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% | Mean ±SD | Median | 95% |
| **Total Impacts[1]** | | | | | | | | | | | | | | | | | |
| <30*g* | 1,601 | 94 ±66 | 16 ±5 | 14.0 | 26 | 2,798 ±1,689 | 2,382 | 6,039 | 0.000 ±0.000 | 0.000 | 0.000 | 0.052 ±0.182 | 0.000 | 0.389 | 0.007 ±0.049 | 0.001 | 0.017 |
| 30*g* – 80*g* | 348 | 22 ±10 | 46 ±12 | 43.0 | 71 | 8,974 ±3,247 | 8,805 | 14,985 | 0.001 ±0.001 | 0.000 | 0.002 | 0.782 ±0.349 | 0.994 | 1.000 | 0.387 ±0.353 | 0.297 | 0.990 |
| >80*g* | 28 | 2 ±1 | 93 ±12 | 90.0 | 121 | 13,930 ±4,991 | 15,117 | 21,764 | 0.008 ±0.006 | 0.005 | 0.025 | 0.906 ±0.269 | 1.000 | 1.000 | 0.844 ±0.312 | 0.996 | 1.000 |
| **Forwards Impacts[2]** | | | | | | | | | | | | | | | | | |
| <30*g* | 1,232 | 123 ±70 | 16 ±5[b] | 14 | 26 | 2,663 ±1,657[b] | 2,198 | 5,660 | 0.000 ±0.000[b] | 0.000 | 0.000 | 0.045 ±0.170[b] | 0.000 | 0.230 | 0.007 ±0.051[b] | 0.000 | 0.012 |
| 30*g* – 80*g* | 258 | 30 ±8 | 46 ±13[b] | 43.0 | 72 | 8,926 ±3,279[b] | 8,730 | 14,758 | 0.001 ±0.001[b] | 0.000 | 0.002 | 0.772 ±0.358[b] | 0.993 | 1.000 | 0.388 ±0.360[b] | 0.295 | 0.988 |
| >80*g* | 21 | 3 ±1 | 92 ±10[b] | 88.0 | 115 | 13,940 ±4,849 | 14,649 | 22,596 | 0.007 ±0.004[b] | 0.005 | 0.019 | 0.931 ±0.219 | 1.000 | 1.000 | 0.846 ±0.292 | 0.994 | 1.000 |
| **Backs Impacts[3]** | | | | | | | | | | | | | | | | | |
| <30*g* | 369 | 53 ±31 | 17 ±5[a] | 15.0 | 26 | 3,190 ±1,665[a] | 2,750 | 6,306 | 0.000 ±0.000[a] | 0.000 | 0.000 | 0.068 ±0.195[a] | 0.001 | 0.520 | 0.007 ±0.029[a] | 0.001 | 0.021 |
| 30*g* – 80*g* | 90 | 15 ±6 | 46 ±12[a] | 44.0 | 70 | 9,433 ±3,315[a] | 9,447 | 15,327 | 0.001 ±0.000[a] | 0.001 | 0.002 | 0.822 ±0.321[a] | 0.998 | 1.000 | 0.438 ±0.360[a] | 0.402 | 0.993 |
| >80*g* | 7 | 2 ±1 | 94 ±15[a] | 88.0 | * | 12,346 ±5,656 | 12,476 | * | 0.009 ±0.009[a] | 0.005 | * | 0.854 ±0.373 | 1.000 | * | 0.764 ±0.370 | 0.978 | * |

PLA (*g*) = peak linear acceleration in gravitational force (*g*); PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $RWE_{Linear}$ = Risk Weighted Exposure Linear; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational; $RWE_{CP}$ = Risk Weighted Exposure Combined Probability; (1) = 11 player positions; (2) = 5 players; (3) = 6 players. Significant difference ($p$<0.05) than: (a) = Forwards; (b) = Backs; * = unable to calculate due to limited numbers

## Results

Twelve matches were completed over the competition season giving a match exposure of 88.0 hours. Both the linear and rotational accelerations were right-skewed and heavily weighted toward low magnitude impacts for impacts greater than the data acquisition limit's utilised (see Figure 3.1). Resultant linear accelerations ranged from 10g to 123g with a mean, median and 95$^{th}$ percentile value of 22g, 16g and 57g respectively (see Table 3.1). Resultant rotational accelerations ranged from 89 rad/s$^2$ to 22,928 rad/s$^2$ with a mean, median and 95$^{th}$ percentile value of 4,041 rad/s$^2$, 2,773 rad/s$^2$ and 11,384 rad/s$^2$ respectively.

A total of 1,977 impacts were recorded (see Table 3.1) for the match competition season. The 19 players experienced a mean of 116 impacts over the duration of the match season resulting in a mean of 13 impacts over the 10g threshold per-match. Although forwards recorded more impacts than backs (1,511 vs. 466; p<0.001) over the duration of the match season, backs recorded a higher average resultant linear (23g vs. 22g; p=0.728) and resultant rotational (4,534 rad/s$^2$ vs. 3,889 rad/s$^2$; p=0.539) accelerations than forwards.

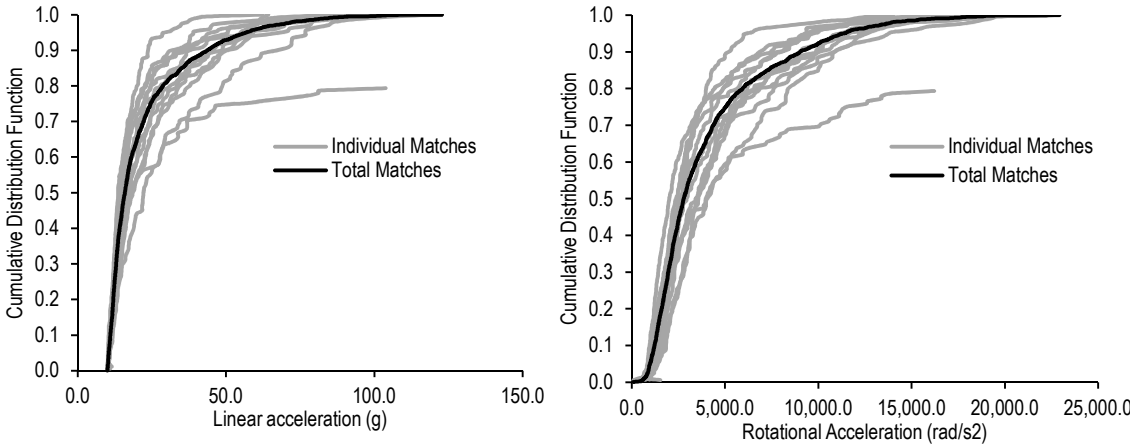


**Figure 3.1:** Cumulative distribution functions for resultant linear (top) and rotational (right) acceleration magnitudes of impacts collected during matches for junior rugby league season 2014.

There were 26 high-magnitude impacts resulting in a mean, median and 95$^{th}$ percentile linear acceleration values of 93g, 90g and 121g respectively (see Table 3.2). The resultant mean, median and 95$^{th}$ percentile rotational accelerations values for high-magnitude impacts were 13,930 rad/s$^2$, 15,117 rad/s2 and 21, 764 rad/s$^2$ respectively. Forwards recorded more moderate-magnitude head impacts than backs (258 vs. 90) resulting in higher resultant linear accelerations (46g vs. 46g; p<0.000).

**Table 3.3:** Impacts to the head greater than 10*g* by impact location for total impacts, impacts recorded by forwards and backs in a junior rugby league team (19 players) for total impacts over a season of matches. Data are presented as mean and standard deviation (±SD), median and 95th percentile for impacts per player per match, impact duration (ms), linear and rotational acceleration and risk weighted exposure by linear, rotational and combined probability.

| | Impacts recorded | | PLA (*g*) | | | PRA(rad/s²) | | | $RWE_{Linear}$ | | | $RWE_{Rotational}$ | | | $RWE_{(CP)}$ | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Location** | **No.** | **%** | **Mean ±SD** | **Median** | **95%** | **Mean ±SD** | **Median** | **95%** | **Mean ±SD** | **Median** | **95%** | **Mean ±SD** | **Median** | **0.950** | **Mean ±SD** | **Median** | **0.950** |
| Total | | | | | | | | | | | | | | | | | |
| Front | 508 | 26 | 25 ±16[bd] | 19 | 59 | 4,915 ±3,513[bd] | 3,686 | 11,948 | 0.001 ±0.001[bd] | 0.000 | 0.001 | 0.261 ±0.405[bd] | 0.006 | 1.000 | 0.117 ±0.265[bd] | 0.002 | 0.898 |
| Side | 944 | 48 | 19 ±15[acd] | 14 | 53 | 3,123 ±2,798[acd] | 2,161 | 8,984 | 0.001 ±0.001[acd] | 0.000 | 0.001 | 0.117 ±0.295[acd] | 0.000 | 0.996 | 0.049 ±0.178[acd] | 0.000 | 0.389 |
| Back | 498 | 25 | 24 ±16[bd] | 18 | 58 | 4,706 ±3,698[bd] | 3,391 | 12,631 | 0.001 ±0.001[bd] | 0.000 | 0.001 | 0.246 ±0.401[bd] | 0.003 | 1.000 | 0.121 ±0.275[bd] | 0.002 | 0.914 |
| Top | 27 | 1 | 37 ±22[abc] | 35 | 87 | 7,425 ±4,711[abc] | 6,104 | 17,896 | 0.001 ±0.002[abc] | 0.000 | 0.005 | 0.490 ±0.476[abc] | 0.420 | 1.000 | 0.299 ±0.401[abc] | 0.029 | 0.999 |
| Forwards | | | | | | | | | | | | | | | | | |
| Front | 341 | 23 | 25 ±16[bd] | 18 | 60 | 4,877 ±3,509[bd] | 3,577 | 11,729 | 0.000 ±0.001[bd] | 0.000 | 0.001 | 0.267 ±0.410[d] | 0.005 | 1.000 | 0.118 ±0.263[bd] | 0.002 | 0.885 |
| Side | 760 | 50 | 19 ±15[bd] | 14 | 54 | 2,976 ±2,795[b] | 2,005 | 8,873 | 0.000 ±0.001[b] | 0.000 | 0.001 | 0.107 ±0.287[b] | 0.000 | 0.995 | 0.046 ±0.173[bd] | 0.000 | 0.359 |
| Back | 394 | 26 | 24 ±16[acd] | 18 | 58 | 4,710 ±3,741[acd] | 3,391 | 12,967 | 0.000 ±0.001[ac] | 0.000 | 0.001 | 0.240 ±0.397[cd] | 0.003 | 1.000 | 0.124 ±0.285[ac] | 0.002 | 0.929 |
| Top | 16 | 1 | 29 ±14[abcf] | 30 | * | 5,981 ±3,764[ab] | 4,669 | * | 0.000 ±0.000[ah] | 0.000 | * | 0.412 ±0.479[ab] | 0.042 | * | 0.168 ±0.292[ac] | 0.008 | * |
| Backs | | | | | | | | | | | | | | | | | |
| Front | 167 | 36 | 24 ±16[d] | 19 | 56 | 4,992 ±3,528[bd] | 3,694 | 13,223 | 0.000 ±0.001[d] | 0.000 | 0.001 | 0.250 ±0.395[bd] | 0.006 | 1.000 | 0.116 ±0.270[bd] | 0.002 | 0.954 |
| Side | 184 | 39 | 21 ±15 | 16 | 52 | 3,730 ±2,735 | 2,838 | 10,278 | 0.000 ±0.000 | 0.000 | 0.001 | 0.156 ±0.323 | 0.001 | 0.999 | 0.060 ±0.199 | 0.001 | 0.741 |
| Back | 104 | 22 | 24 ±15[d] | 18 | 54 | 4,691 ±3,546[ad] | 3,371 | 11,109 | 0.000 ±0.000[d] | 0.000 | 0.001 | 0.269 ±0.417[ad] | 0.003 | 1.000 | 0.001 ±0.235[ad] | 0.002 | 0.726 |
| Top | 11 | 2 | 49 ±27[abe] | 57 | * | 9,526 ±5,317[ab] | 9,556 | * | 0.001 ±0.002[abg] | 0.001 | * | 0.603 ±0.470[ab] | 0.999 | * | 0.489 ±0.471[ab] | 0.609 | * |

PLA (*g*) = peak linear acceleration in gravitational force (*g*); PRA (rad/s²) = peak rotational acceleration in radians/second²; $RWE_{Linear}$ = Risk Weighted Exposure Linear; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational; $RWE_{CP}$ = Risk Weighted Exposure Combined Probability; Significant difference ($p<0.05$) than: (a) = Front; (b) = Side; (c) = Back; (d) = Top; (e) = Forwards; (f) = Backs; * = unable to calculate due to limited numbers

Impacts to the side of the head (48%) were the most common (see Table 3.3). The distribution of total resultant linear accelerations to the side of the head had an average, median and a 95$^{th}$ percentile value of 19g, 14g and 53g respectively. Resultant rotational accelerations to the top of the head had a higher mean (7,425 rad/s$^2$ vs. 3,123 rad/s$^2$; p=0.007) than the side of the head. Forwards recorded more impacts to the back of the head (26% vs. 22%; p<0.001) than backs. Backs recorded higher mean resultant linear accelerations (49g vs. 29g; p=0.021) and had a higher mean $RWE_{Linear}$ (0.001 vs. 0.000; p=0.013) than forwards for impacts to the top of the head.

## Discussion

This study reports, for the first time, the head impact characteristics (magnitude, frequency) experienced by 19 Under 11 junior rugby league players during 12 matches. Surprisingly, high magnitude impacts (>80g) were experienced by this cohort during match participation. This level of severity was similar to impacts reported in American high school[49-51] and collegiate[51, 109, 110] football players; but the New Zealand rugby league players were younger, had less body mass and played at a slower speed than the American players.[115, 631] Unlike American football, rugby league does not have the same protective equipment worn during match activities.

Comparisons of impact data between studies can be difficult given different thresholds used to count an impact. For example, the data acquisition limit utilised for the recording of impacts to the head for Pop Warner youth football[626] players was any linear acceleration greater than 30g, while other studies utilised a 10g[115] and 14.4g[84, 116, 631] limit. No rotational accelerations were reported in Pop Warner youth football.

By using a 30g data acquisition limit for Pop Warner football, it has been estimated that between 80 to 85% of the impacts may have been excluded from the data set.[626] In a study[115] on youth American football players aged 6 to 9 yr. old, approximately 85% of the impacts recorded had a linear acceleration below 30g. This was similar in a study[84] on 9 to 11 yr. old American football players with 80% of impacts recorded had linear accelerations below 30g. The results of these differences is that unless the data are reported at the different data acquisition limits utilised in previous studies (i.e. 10g,[115] 14.4g,[1,6,21] and 30g[626]) resulting in complex tables or large amounts of data presented, then inter-study comparisons are limited.

Although these studies were included in the current study the comparisons should be undertaken with caution as we reported the data utilising a 10g data acquisition limit. A standardised reporting format for impacts needs to be established identifying what parameters should be included (i.e. resultant linear (PLA(g)) and rotational acceleration (PRA(rad/s$^2$)), Head Impact Criterion (15ms) ($HIC_{15}$); Gadd Severity Index (GSI) Head Impact Telemetry severity profile (HITsp) etc.) and at what linear data acquisition limit the data should be reported from. The identification of thresholds for head impacts that are sub-concussive versus non-sub-concussive is needed.

In youth football (6-9 yr.), players recorded an average of 44 impacts during matches or 5.8 impacts to the head per-player per-match.[115] In a slightly older youth football team (12-14 yr.)[116] players recorded an average of 112 impacts during matches or 12 impacts per-player per-match. These study had less impacts on average than our study with players recording an average of 116 impacts to the head during matches or 13 per-player, per-match. Similar to other studies reporting head impact biomechanics, the magnitudes recorded were characterized by a skewed frequency distribution with the majority (64%) of impacts having a linear acceleration of between 10-20g. There were, however,

28 impacts recorded over the duration of the study above 80g. This magnitude has been previously described[449] as "high" and highlights that, although these are youth players, they can be exposed to impacts over a competition duration that can be considered high-magnitude at any level of participation. Despite the number of high-magnitude impacts recorded, there were no witnessed concussive events recorded throughout the duration of the study.

Previous studies reporting impacts for youth American football players have shown that the median linear accelerations were lower when compared with other levels of participation. Observations of 7-8 yr. old American football players[631] showed a median linear acceleration of 16g which was similar to the current study. This was lower than 9-12 yr. old youth American football players with a median head impact of 18g[84] while 12-14 yr. old[116] recorded a slightly higher median linear acceleration of 22g. Although it appears that the players in the current study may have a lower resultant linear acceleration that similar aged American football players, this may be reflective of how these games differ. American football players utilise full protective equipment, including rigid helmets and padding whereas the players in the current study are only required to wear a fitted mouthguard and any other protective equipment must be soft in nature to reduce the risk on possible injury to other players.

When comparing the resultant rotational accelerations with youth American football players, the median rotational acceleration (4,041 rad/s$^2$) were higher than the median and 95$^{th}$ percentiles reported for 7-8 yr. old (686 rad/s$^2$; 2,052 rad/s$^2$),[631] 9-12 yr. old (829 rad/s$^2$; 1,884 rad/s$^2$)[84] and 12 to 14 yr. old (987 rad/s$^2$; 2,769 rad/s$^2$)[116] American youth football players. When compared with high school[50, 51] (median: 903 rad/s; 95% 2,527 rad/s2) and collegiate American football players[110, 529, 531] (median 904-981 rad/s2; 95% 2,787-2,975 rad/s2) this was similar with the current study recording a higher median and 95$^{th}$ percentile (11,143 rad/s$^2$) resultant rotational acceleration. This may be related to the type of activity undertaken in junior rugby league, when compared with American football, where players are required to tackle the player to the ground and use a different tackle technique which may include a rotational type of movement increasing the rotational forces recorded at the head. No other study has reported how the different tackle techniques seen in rugby league differ from tackles in American football, and if these have an effect on the forces recorded at the head. Further research is warranted to explore these differences.

By incorporating the RWE for linear, rotational and combined probability, the variability of exposure due to linear and rotational accelerations can be identified.[596] Only one study[596] has undertaken to report these at the high school (14-18 yr.) level in American football. Participants recorded a median value for rotational accelerations of 22g and a 95$^{th}$ percentile value of 62g. The median rotational acceleration values reported were 1,013 rad/s$^2$ and a 95$^{th}$ percentile of 2,743 rad/s$^2$. The linear accelerations were similar to the current study while the rotational accelerations were lower at the same data acquisition limit. When reviewed by RWE, the median value for $RWE_{Linear}$ (0.053[596] vs. 0.000), $RWE_{Rotational}$ (0.051[596] vs. 0.001) and $RWE_{CP}$ (0.194[596] vs. 0.001) were lower. The differences seen here may be related to the use of the regression coefficients utilised and the exposure time differences between the different sporting codes. The use of the RWE may be beneficial but may be limited to the same sporting codes comparisons. Further research is warranted to identify if there are differences between the different sporting codes and if the regression coefficients utilised are appropriate for non-helmeted sporting activities.

### Limitations

The current study has reported the linear and rotational accelerations by the mean, median and 95th percentile results recorded and this was utilised for comparisons with limited previous studies reporting on American Pop warner, youth and high-school football. Other studies have utilised either a median and/or 95th percentile result format or only reported on linear accelerations which has limited inter-study comparisons. This has resulted in the information provided by these studies being left to stand-alone awaiting future studies reporting similar data acquisition limits and formats to enable comparisons to be completed.

The players in this study were aged between 9 and 11 years old and the impact characteristics maybe specific to this age group. The head impact exposure experienced by this cohort of players may vary when compared with other junior rugby league players at different age group levels as the head impact exposure likely varies by age.

The accelerometers utilised have a 10% error for linear and rotation acceleration and for angular velocity with an average offset of 2° for azimuth and elevation impact location.[63, 401] The correlation of the patch accelerometers with laboratory head-forms is good, however, the impact measurements should be assumed to have some error that is dependent on impact conditions and the measure of interest and the variability tested.[27, 140] It is unlikely that the XPatch (X2Biosystems, Seattle, USA) was tested under all of the conditions seen in rugby league matches and how these conditions would correlate to the laboratory conditions remains unknown. The large percentage errors reported for the reliability of the impact variables mean that the results presented in this study should be interpreted with some caution. Validation studies need to be conducted to ensure we are confident of the meaning of the results.

### Conclusion

By utilising accelerometer fitted patches worn behind the ear of participants in a single rugby league junior team throughout the 2014 match competition season, there were 1,977 impacts recorded. Mean linear acceleration measured over the season of matches was similar to mean linear accelerations reported in studies for American Pop Warner and Youth football. Mean rotational accelerations measured were higher than American Pop Warner, youth and high school football. Further studies should explore impacts at the senior, female and professional levels of participation. Although we reported the data by means (±SD), median and 95th percentile to enable comparisons across published studies, there is a need for standardization of reporting head impact biomechanics to enable accurate comparisons.

## Part 4: Head impacts in a senior amateur rugby league team measured with an instrumented patch: An exploratory analysis

### Overview

Senior rugby league players are stronger and faster and likely to impact other players with more force in tackles than junior players. Therefore given our work with junior players, we wanted to extend this work to quantify the frequency,

magnitude and distribution of head impacts in senior rugby league. Thirty eight players wore the XPatch behind the left ear for every match competed over the pre-season and competition 2014 season. The number of impacts and the linear and rotational forces involved were used to calculate the risks associated with every impact, and the cumulated impacts per player, per match and per season. There were 13,895 impacts recorded via accelerometer fitted patches applied to behind the ear of players. There was an average of 56 impacts over 10*g* per game over the course of the season. The majority (41%) of impacts occurred to the side of the head which is not the same as other studies reporting impacts in American high school and collegiate football. Median resultant linear accelerations were lower than high school and collegiate American football while the median resultant rotational accelerations were higher. The cumulated impacts per player, per match were 56 ±22 and 731 ±289 respectively. Although we reported the data by means (±SD), median and 95$^{th}$ percentile to enable comparisons across the studies to be conducted, the need for standardization of reporting head impact biomechanics is needed.

## Introduction

Rugby league is a popular collision sport played throughout the word that originated in the north of England in the late 19$^{th}$ century.[338] Played at junior, amateur, semi-professional and professional levels of participation,[338] rugby league is an intermittent collision sport involving numerous collisions and tackles.[338] At the professional level of participation it has been reported[332] that players can experience from 34 to 72 tackle situations per match either as the tackler or the ball carrier. As a result of the tackle situations, many impacts to the body and head occur from match participation in rugby league. Knowledge about the potential metabolic and ultrastructural consequences of impacts to the head has grown, as has the appreciation for repetitive concussive and subconcussive impacts and the possible deleterious effects in some individuals.[626] The use of technology such as accelerometers in the helmets of American football players,[50, 110, 115] the analysis of injury biomechanics has increased the knowledge of the forces, accelerations, frequencies and velocities of head injuries.[626]

Despite more studies reporting impacts to the head and concussions, none have identified a data acquisition limit that constitutes where a subconcussive impact occurs. Impacts under 10*g* are reported[473] to be a result of non-contact activities such as walking, jumping, running and sitting.[110, 449] Impacts over 10*g* that do not result in a participant presenting with acute signs or symptoms of concussion, have been identified to be subconcussive impacts[13] and repetitive subconcussive impacts may have negative long-term effects.[566] Several studies have utilised data acquisition limits greater than 10*g* (i.e. 14.4*g*,[84, 531, 596] 15*g*[49, 50, 150] and 30*g*[626]) to report their findings and may have removed a large number of subconcussive impacts from their dataset. The data obtained from these types of impacts is not normally distributed. Using a data acquisition limit greater than 10*g* may remove a large amount of impacts that could be subconcussive impacts. The data acquisition limit of 10*g* was therefore selected by us to identify impacts that were considered to have occurred from impact accelerations enabling elimination of activities undertaken in daily living.

Non-helmeted sports have also utilised accelerometers in measuring impacts from sports participation. Heading the ball by female soccer youths yielded peak accelerations of 63*g* and 8,869 rad/s$^2$.[261] No concussions were reported,[261] as no injury nor injury risk was assessed; even though some of the rotational accelerations were within the nominal values for an injury to the head when compared with NFL data[498] and injury risk tolerance levels.[632] Although data are

accumulating for soccer and NFL there are no published data for non-helmeted collision sports such as rugby league. To date, no study has reported the frequency, magnitude, duration and distribution of the linear and rotational forces that occur from match participation in amateur senior rugby league. This study quantified the impacts to the head via an instrumented patch worn behind the ear for amateur rugby league players over a premier season of domestic matches in New Zealand.

## Methods

A prospective observational cohort study was conducted on a senior club level amateur rugby league team during the 2014 competition in New Zealand. Thirty eight players were enrolled in the study with a mean ±SD age, body mass and height of 23.3 ±4.3 yr. 93.6 ±14.4 kg and 1.80 ±0.05 m. The players were considered amateur receiving no remuneration for participating in rugby league activities. The matches were played under the laws of the New Zealand Rugby League. Players were placed into three groups: (1) hit-up forwards (prop, second row); (2) outside backs (centre, wing and fullback); and (3) adjustables (hooker, halfback, five-eight, and loose forward).[195] The researchers' University ethics committee approved all procedures in the study (AUTEC 12/156) and all players gave informed consent prior to participating in the study.

Players wore an instrumented patch behind their ear (XPatch, X2Biosystems, Inc., Seattle, WA, USA) sampling at 1,024 Hz, during every match. The specific details of the accelerometers utilised and the biomechanical measures have been previously reported.[330] The patches recorded head linear and rotational acceleration, impact location and duration. All data were recorded on the X2Biosystem Injury Management Software (IMS) and transferred to an Excel spreadsheet for further analysis. Over the 2014 senior amateur rugby league match competition season impacts that exceeded our inclusion limit of 10*g* for a head impact were retained for data analyses. Impacts could be a result of a direct blow to the head, face, neck or elsewhere on the body with an 'impulsive' force transmitted to the head. Each recorded impact was categorised into four general locations (front, side, back and top).[109] Over the duration of the study one concussion was witnessed (143.3*g*; 23,133.7 rad/s$^2$) and confirmed by medical personnel including the witnessing of the event resulting in the concussion. The impact data from this concussive event was included in the dataset utilised for analysis.

For the impact to be recorded, 100 ms of data were stored, including 25 ms prior to, and 75 ms following, the impact. Software provided by X2Biosystems calculated the peak linear acceleration, rotational acceleration (x-axis and y-axis angular accelerations), impact location, Head Impact Criterion 15 ms (HIC$^{15}$), Gadd Severity Index (GSI), and date and time stamp for later download and analyses. Impacts were uploaded and then reviewed to assess whether they were from match impacts. Impacts not occurring from match activities were termed 'clacks'. All impacts were assessed through the IMS utilising a "de-clacking algorithm" that involved two methods.[330] An example of the impacts recorded for a single player in a single amateur rugby union match can be seen in Figure 4.1. All impacts identified as 'clacks' were removed from the dataset prior to downloading for further analysis. All matches were videotaped (Sony HDR-PJ540 Camcorder) to enable verification of the impacts recorded.

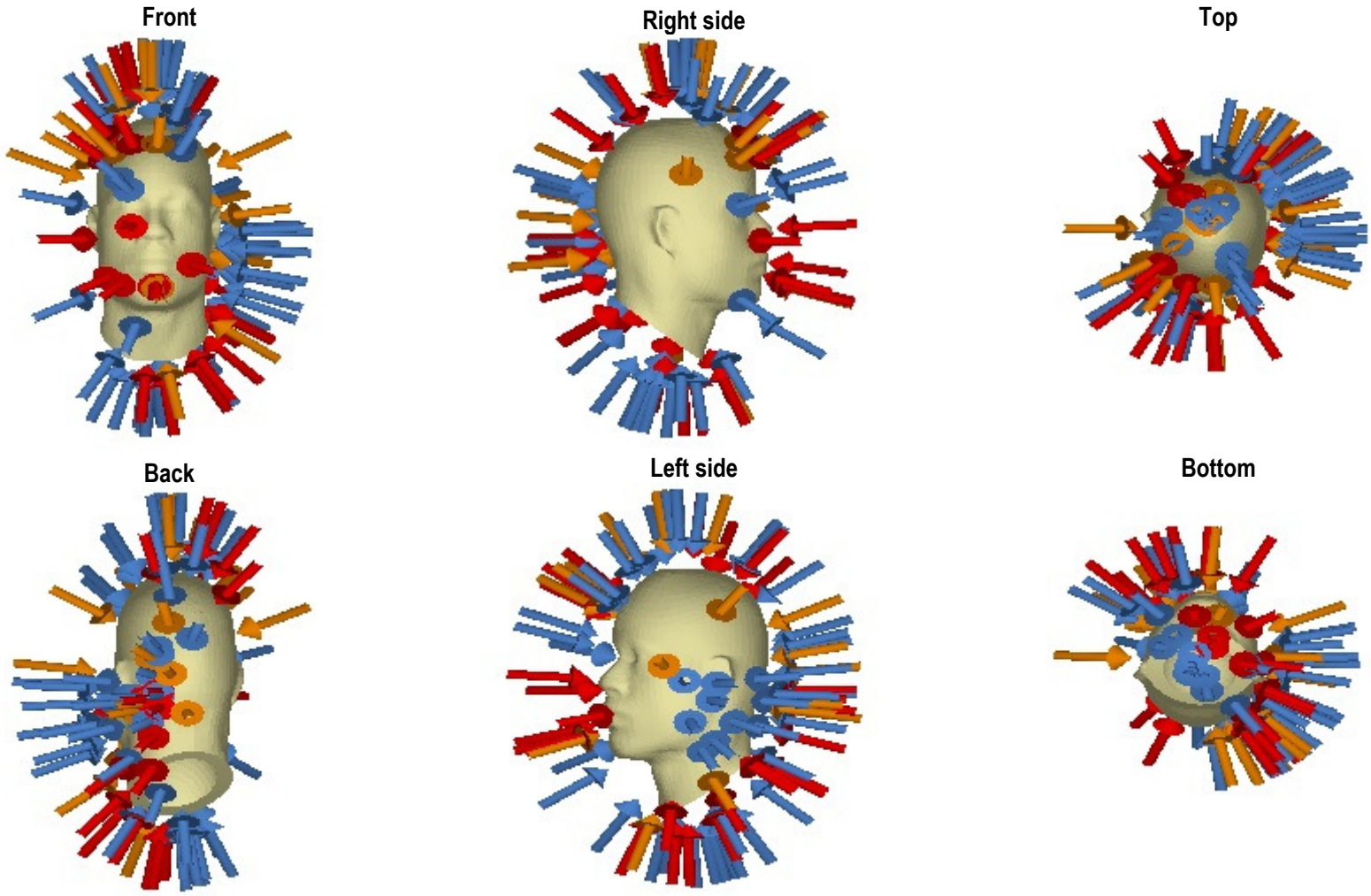


**Figure 4.1:** Example of the locations of impacts for one monitored player with the X2Patch from a single player in a single senior amateur rugby league match on the Impact Management System (X2Biosystems, Seattle, WA, USA). Blue = 10-30g, orange = 40-60g and red = 70g+.

All data were entered into Microsoft Excel and analysed with SPSS V.22.0.0. Impacts were reported as the mean ±standard deviation (SD) per-player per-season of matches, per-player per-match and impact duration (ms). As the impact variables were not normally distributed at all the data acquisition limits (Kolmogorov-Smirnov test; $p<0.001$), the data were expressed as mean ±standard deviation (SD), median [25$^{th}$ to 75$^{th}$ percentile] and 95$^{th}$ percentile value for impacts recorded for resultant linear and rotational accelerations. These were analysed using a Krusal-Wallis one-way ANOVA with a Dunn's post-hoc test.

Head impacts were assessed for injury tolerance level for a concussion occurring using previously published injury tolerance levels[49, 52] for linear (>95*g*) and rotational acceleration (>5,500 rad/s$^2$). Head impacts were assessed for impact severity using previously published levels for linear acceleration (mild <66*g*, moderate 66-106*g*, severe >106*g*) and rotational acceleration (mild <4,600 rad/s$^2$, moderate 4,600-7,900 rad/s$^2$, severe >7,900 rad/s$^2$).[264, 478, 632] Both injury tolerance and impact severity levels were analysed using a Friedman repeated measures ANOVA on ranks. Post hoc analysis with Wilcoxon signed-rank tests was conducted with a Bonferroni correction applied. The risk weighted exposure (RWE) calculations for linear ($RWE_{Linear}$), rotational ($RWE_{Rotational}$) and combined probability (CP) ($RWE_{CP}$)[596] were analysed with a Wilcoxon signed-rank test with a Bonferroni correction applied.

The impact location was categorized as front, side, back and top. Impacts to the top of the head were defined as all impacts above an angle 65° from a horizontal plane through the centre of gravity of the head.[241] Impact locations were analysed using a Friedman repeated measures ANOVA on ranks. Post hoc analysis with Wilcoxon signed-rank tests was conducted with a Bonferroni correction applied. Statistical significance was set at $p<0.05$. Our results were then compared to studies quantifying head impact exposure in junior, high school and college American football players.

## Results

Nineteen matches were completed over the competition season resulting in a match exposure of 328.5 match hours. A total of 13,895 impacts were recorded that were above the 10*g* data acquisition limit (see Table 4.1). Impacts were non-normally distributed, with skewness of 3.09 (*SE* = 0.04) and kurtosis of 13.07 (*SE* = 0.09). Players recorded a mean (±SD), median and 95$^{th}$ percentile linear (23 ±16 *g*, 17 [12-28] *g* and 56*g)*, and rotational (4,098 ±3,669 rad/s$^2$, 3,071 [1,662-5,466] rad/s$^2$ and 11,734 rad/s$^2$) accelerations over the duration of the study.

Hit-up forwards recorded more total impacts (6,966) than adjustables (4,434; $p<0.001$) and outside backs (2,492; $p<0.001$). Hit-up forwards recorded a higher mean and median linear acceleration (26 ±17*g*; 21 [14-33] *g*) than outside backs (20 ±15*g*; 15 [12-23] *g*; $p<0.001$) and adjustables (20 ±14*g*; 14 [11-22] *g*; $p<0.001$). Adjustables recorded a lower mean rotational acceleration (3,513 ±3,317 rad/s$^2$; 2,525 [1,307-4,643] rad/s$^2$) than adjustables (3,726 ±3,349 rad/s$^2$; 2,833 [1,413-4,926] rad/s$^2$; $p<0.001$) and hit-up forwards (4,646 ±3,660 rad/s$^2$; 3,462 [2,133-6,022] rad/s$^2$ $p<0.001$).

The side of the head was the most common impact location (41%) (see Table 4.2). This was similar for outside backs (43%) and adjustables (55%) but hit-up forwards recorded more impacts at the front of the head (36%) when compared with outside backs ($p<0.001$) and adjustables ($p<0.001$). The top of the head recorded the highest mean linear and rotational accelerations for hit-up forwards (31 ±14*g*; 6,082 ±3,578 rad/s$^2$) and outside backs (27 ±16*g*; 5,162 ±3,440

rad/s$^2$) but adjustables recorded the highest mean linear and rotational accelerations (24 ±15*g*; 4,816 ±3,526 rad/s$^2$) on the back of the head.

There were 71 impacts (0.5%) recorded above the linear injury risk limit and 3,377 impacts (24.3%) above the rotational injury risk limit (see Table 4.3). Adjustables recorded higher mean resultant linear accelerations (112 ±18*g*) than outside backs (109 ±9*g*; $p$=0.058) and hit-up forwards (106 ±11*g*; $p$<0.001) for impacts above linear injury risk limit (>96g). Hit-up forwards recorded higher mean resultant rotational accelerations (9,333 ±3,250 rad/s$^2$) than adjustables (8,962 ±3,148 rad/s$^2$; $p$<0.001) and outside backs (9,169 ±3,446 rad/s$^2$; $p$<0.001) for impacts above the rotational injury risk limit (>5,500 rad/s$^2$). Hit-up forwards recorded more rotational accelerations in the moderate rotational impact severity limit (20%) than outside backs (16.8%; $p$=0.002) and adjustables (15.4%; $p$<0.001).

The calculated RWE metric for each risk function are shown in Table 4.3. Total impacts over the duration of the study had a mean calculated $RWE_{Linear}$, $RWE_{Rotational}$ and $RWE_{CP}$ values of 0.000 ±0.001, 0.210 ±0.372 and 0.091 ±0.242 respectively. Hit-up forwards recorded a higher mean $RWE_{CP}$ over the duration of the study (0.108 ±0.259) than outside backs (0.073 ±0.220; $p$<0.001) and adjustables (0.068 ±0.211; $p$<0.001).

**Table 4.1:** Impacts to the head greater than 10*g* in senior amateur rugby league (38 players) for total impacts recorded and impacts per player positional group[195] over a season of matches for total impacts, impacts per match, impacts per player per match, linear and rotational accelerations. Data are presented as mean (±SD), median, 25-75th interquartile range and 95th percentile.

| | Impacts | | | Resultant Linear Acceleration | | | Resultant Rotational Acceleration | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total | Average per match | Average per player | Duration (ms) | PLA (g) | | | PRA (rad/s²) | | |
| | | Mean ±SD | Mean ±SD | Mean ±SD | Mean ±SD | Median [25-75 IQR] | 95th | Mean ±SD | Median [25-75 IQR] | 95th |
| Total Impacts[1] | 13,895 | 731 ±289 | 56 ±22 | 12 ±9 | 23 ±16 | 17 [12-28] | 56 | 4,098 ±3,669 | 3,071 [1,662-5,466] | 11,734 |
| Hit-Up Forwards[2] | 6,966[bc] | 367 ±257 | 92 ±39 | 14 ±9[bc] | 26 ±17[bc] | 21 [14-33] | 60 | 4,646 ±3,660[c] | 3,462 [2,133-6,022] | 12,056 |
| Outside Backs[3] | 2,495[ac] | 131 ±94 | 33 ±12 | 9 ±7[ac] | 20 ±15[ac] | 15 [12-23] | 52 | 3,726 ±3,439[c] | 2,833 [1,413-4,926] | 11,042 |
| Adjustables[4] | 4,434[ab] | 233 ±172 | 19 ±32 | 10 ±8[ac] | 20 ±14[ab] | 14 [11-22] | 48 | 3,513 ±3,317[ab] | 2,525 [1,307-4,643] | 10,807 |

PLA (*g*) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); IQR = interquartile range; (1) = 13 player positions; (2) = 4 player position; (3) = 5 player position; (4) = 4 player positions; Significant difference ($p<0.05$) than (a) Hit-Up Forwards; (b) = Outside backs; (c) = Adjustables

**Table 4.2:** Impacts to the head greater than 10*g* in senior amateur rugby league (38 players) by player positional group[195] over a season of matches for impact location, total impacts recorded, linear accelerations, rotational accelerations and impact duration. Data are presented as mean (±SD), median, 25-75th interquartile range and 95th percentile.

| | | Impact | Resultant Linear Acceleration PLA(*g*) | | | Resultant Rotational Acceleration PRA (rad/s²) | | | Impact Duration (ms) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | **Location** | **Total (%)** | **Mean (±SD)** | **median [25-75 IQR]** | **95%** | **Mean (±SD)** | **median [25-75 IQR]** | **95%** | **Mean (±SD)** | **median [25-75 IQR]** | **95%** |
| Total Impacts[1] | Front | 4,046 (29.1)$^{bcd}$ | 25 ±16$^{bcd}$ | 20 [14-31] | 60 | 4,679 ±3,454$^{cd}$ | 3,612 [2,375-5,807] | 12,110 | 13 ±9$^{bcd}$ | 10 [7-18] | 31 |
| | Back | 3,798 (27.3)$^{acd}$ | 25 ±16$^{acd}$ | 20 [13-31] | 57 | 4,652 ±3,703$^{cd}$ | 3,481 [2,014-6,205] | 11,978 | 12 ±9$^{acd}$ | 10 [6-16] | 32 |
| | Side | 5,639 (40.6)$^{abd}$ | 20 ±15$^{abd}$ | 14 [11-23] | 50 | 3,250 ±3,326$^{abd}$ | 2,229 [1,132-4,098] | 10,416 | 11 ±9$^{abd}$ | 8 [4-14] | 30 |
| | Top | 412 (3.0)$^{abc}$ | 29 ±15$^{abc}$ | 25 [17-36] | 59 | 5,582 ±3,589$^{abc}$ | 4,532 [2,947-7,459] | 12,207 | 16 ±9$^{abc}$ | 14 [8-20] | 35 |
| Hit Up Forwards[2] | Front | 2,484 (35.7)$^{bcdfg}$ | 28 ±16$^{cdfg}$ | 22 [15-35] | 61 | 4,808 ±3,558$^{df}$ | 3,744 [2,394-6,082] | 12,343 | 14 ±9$^{bcdfg}$ | 12 [8-20] | 32 |
| | Back | 2,124 (30.5)$^{adfg}$ | 27 ±16$^{cdfg}$ | 22 [15-34] | 59 | 4,808 ±3,676$^{dfg}$ | 3,648 [2,262-6,262] | 12,090 | 14 ±9$^{adfg}$ | 11 [7-18] | 34 |
| | Side | 2,110 (30.3)$^{adfg}$ | 26 ±18$^{abdg}$ | 20 [13-32] | 61 | 4,608 ±3,704$^{dfg}$ | 3,385 [2,069-6,017] | 11,926 | 13 ±10$^{adfg}$ | 11 [5-19] | 35 |
| | Top | 248 (3.6)$^{abcg}$ | 31 ±14$^{abcf}$ | 28 [20-40] | 61 | 6,082 ±3,578$^{abc}$ | 5,391 [3,213-8,851] | 12,228 | 17 ±10$^{abcf}$ | 15 [9-23] | 35 |
| Outside Backs[3] | Front | 734 (29.4)$^{bcdeg}$ | 23 ±15$^{cde}$ | 17 [13-26] | 55 | 4,663 ±3,309$^{cde}$ | 3,582 [2,509-5,711] | 12,166 | 11 ±8$^{cde}$ | 8 [6-14] | 27 |
| | Back | 615 (24.6)$^{acde}$ | 23 ±15$^{cde}$ | 17 [13-28] | 52 | 4,577 ±3,684$^{ce}$ | 3,394 [1,846-6,197] | 11,777 | 10 ±8$^{cde}$ | 8 [5-13] | 25 |
| | Side | 1,073 (43.0)$^{abdeg}$ | 19 ±14$^{abdg}$ | 13 [11-20] | 42 | 3,223 ±3,188$^{abdeg}$ | 2,216 [1,231-4,119] | 8,947 | 8 ±7$^{abdeg}$ | 6 [3-10] | 23 |
| | Top | 73 (3.0)$^{abcg}$ | 27 ±16$^{abce}$ | 22 [16-33] | 67 | 5,162 ±3,440$^{ac}$ | 4,124 [2,780-6,524] | 15,233 | 14 ±9$^{abce}$ | 12 [8-18] | 27 |
| Adjustables[4] | Front | 828 (18.7)$^{bcdef}$ | 22 ±15$^{bcde}$ | 17 [13-27] | 57 | 4,613 ±3,236$^{bc}$ | 3,696 [2,410-5,758] | 11,644 | 11 ±8$^{cde}$ | 8 [6-14] | 30 |
| | Back | 1,059 (23.9)$^{acde}$ | 24 ±15$^{acde}$ | 19 [13-31] | 52 | 4,816 ±3,526$^{ace}$ | 3,855 [2,088-6,585] | 11,791 | 11 ±8$^{cde}$ | 9 [5-15] | 28 |
| | Side | 2,456 (55.4)$^{abdef}$ | 17 ±12$^{abdef}$ | 12 [11-17] | 40 | 2,537 ±2,930$^{abdef}$ | 1,703 [807-3,067] | 8,826 | 10 ±8$^{adef}$ | 7 [4-13] | 27 |
| | Top | 91 (2.1)$^{abcef}$ | 23 ±11$^{abc}$ | 20 [15-28] | 47 | 4,674 ±2,621$^{c}$ | 3,936 [2,854-6,160] | 10,815 | 14 ±9$^{abc}$ | 13 [8-18] | 34 |

PLA (*g*) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); ms = milliseconds; IQR = interquartile range; (1) = 13 player positions; (2) = 4 player position; (3) = 5 player position; (4) = 4 player positions; Significant difference ($p$<0.05) than (a) = Front; (b) = Back; (c) = side; (d) = Top; (e) = Hit-up Forwards; (f) = Outside backs; (g) = Adjustables

**Table 4.3:** Impacts to the head greater than 10*g* over a season of matches for injury tolerance level,[7, 9, 23, 38] and impact severity limits[264, 478, 632] by resultant linear and rotational accelerations and risk weighted cumulative exposure of impacts to the head for impacts per player positions and per player group in amateur senior rugby league (38 players) by total impacts recorded, percentage of impacts recorded (%), mean, standard deviation (±SD) and median [25th to 75th interquartile range].

| | Total | Total Impacts | | Total | Hit-Up Forwards[1] | | Total | Out-Side backs[2] | | Total | Adjustables[3] | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n= *(%)* | Mean ±SD | Median [25-75 IQR] | n= *(%)* | Mean ±SD | Median [25-75 IQR] | n= *(%)* | Mean ±SD | Median [25-75 IQR] | n= *(%)* | Mean ±SD | Median [25-75 IQR] |
| **Injury tolerance level** | | | | | | | | | | | | |
| >95*g* | 71 *(0.5)* | 108 ±12 | 105 [98-115] | 46[c] *(0.7)* | 106 ±11[bc] | 104 [98-114] | 10 *(0.4)* | 109 ±9[a] | 107 [102-118] | 15[a] *(0.3)* | 112 ±18[a] | 107 [102-115] |
| >5,500 rad/s$^2$ | 3,377 *(24.3)* | 9,211 ±3,418 | 8,203 [6,589-10,940] | 1,984[bc] *(28.5)* | 9,333 ±3,520[bc] | 8,302 [6,703-11,018] | 510[a] *(20.5)* | 9,169 ±3,446[ac] | 8,222 [6,444-10,955] | 883[a] *(19.9)* | 8,962 ±3,148[ac] | 7,943 [6,512-10,837] |
| **Impact severity level** | | | | | | | | | | | | |
| **Resultant PLA(*g*)** | | | | | | | | | | | | |
| <66*g* | 13,542 *(97.5)* | 22 ±12 | 17 [12-27] | 6,737 *(96.7)* | 24 ±13[bc] | 21 [14-31] | 2,442 *(97.9)* | 19 ±11[ac] | 15 [12-22] | 4,363 *(98.4)* | 19 ±11[ab] | 14 [11-21] |
| 66*g*-106*g* | 319 *(2.3)* | 79 ±11 | 76 [70-86] | 208[bc] *(3.0)* | 79 ±11[bc] | 79 [69-88] | 48[a] *(1.9)* | 81 ±11[ac] | 80 [71-89] | 63[a] *(1.4)* | 77 ±10[ab] | 73 [69-83] |
| >106*g* | 34 *(0.2)* | 117 ±11 | 115 [110-121] | 21 *(0.3)* | 116 ±9[bc] | 114 [109-121] | 5 *(0.2)* | 117 ±5[ac] | 117 [113-122] | 8 *(0.2)* | 119 ±19[ab] | 114 [108-120] |
| **Resultant PRA(rad/s$^2$)** | | | | | | | | | | | | |
| <4,600 rad/s$^2$ | 9,585*(69.0)* | 2,237 ±1,195 | 2,204 [1,353-3,156] | 4,468[bc] *(64.1)* | 2,522 ±1,044[bc] | 2,469 [1,718-3,356] | 1,810[a] *(72.5)* | 2,063 ±1,286[ac] | 2,059 [1,005-3,060] | 3,307[a] *(74.6)* | 1,947 ±1,247[ab] | 1,848 [1,030-2,856] |
| 4,600-7,900 rad/s$^2$ | 2,496 *(18.0)* | 5,993 ±940 | 5,876 [5,155-6,473] | 1,394[bc] *(20.0)* | 6,014 ±958[bc] | 5,887 [5,153-6,802] | 420[a] *(16.8)* | 5,907 ±909[ac] | 5,773 [5,137-6,614] | 682[a] *(15.3)* | 6,001 ±919[ab] | 5,292 [5,191-6,735] |
| >7,900 rad/s2 | 1,814 *(13.1)* | 11,484 ±3,190 | 10,729 [9,117-12,791] | 1,104[bc] *(15.8)* | 11,515 ±3,337[bc] | 10,670 [9,069-12,867] | 265[a] *(10.6)* | 11,613 ±3,163[ac] | 10,717 [9,211-12,819] | 445[a] *(10.1)* | 11,334 ±2,811[ab] | 10,797 [9,201-12,759] |
| **Risk Weighted Exposure (RWE)** | | | | | | | | | | | | |
| $RWE_{Linear}$ | 13,985 (*100*) | 0.000 ±0.001 | 0.000 [0.000-0.000] | 6,966[bc] (*50.1*) | 0.000[bc] ±0.002 | 0.000 [0.000-0.000] | 2,495[ac] (*18.0*) | 0.000[a] ±0.001 | 0.000 [0.000-0.000] | 4,434[ab] (*31.9*) | 0.000[a] ±0.003 | 0.000 [0.000-0.000] |
| $RWE_{Rotational}$ | 13,985 (*100*) | 0.210 ±0.372 | 0.002 [0.000-0.168] | 6,966[bc] (*50.1*) | 0.243[bc] ±0.393 | 0.004 [0.000-0.380] | 2,495[ac] (*18.0*) | 0.171[ac] ±0.342 | 0.001 [0.000-0.064] | 4,434[ab] (*31.9*) | 0.165[ab] ±0.338 | 0.000 [0.000-0.038] |
| $RWE_{CP}$ | 13,985 (*100*) | 0.091 ±0.242 | 0.001 [0.000-0.013] | 6,966[bc] (*50.1*) | 0.108[c] ±0.259 | 0.002 [0.001-0.025] | 2,495[ac] (*18.0*) | 0.073[c] ±0.220 | 0.001 [0.000-0.007] | 4,434[ab] (*31.9*) | 0.068[ab] ±0.211 | 0.001 [0.000-0.005] |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); IQR = interquartile range; $RWE_{Linear}$ = Risk weighted cumulative exposure linear acceleration; $RWE_{Rotational}$ = Risk weighted cumulative exposure rotational acceleration; $RWE_{CP}$ = Risk weighted cumulative exposure combined probability from linear and rotational acceleration; (1) = 4 player positions; (2) = 5 player positions; (3) = 4 player positions; Significant difference ($p$<0.05) than (a) = Hit-Up Forwards; (b) = Out-Side backs; (c) = Adjustables

## Discussion

In this study 38 players received an average of 56 impacts over 10*g* per game over the course of a season. As there are, to date, no studies reporting impact biomechanics in rugby league, the inter-study comparisons are limited to American high-school and collegiate football studies. Not every study reviewed utilised the same reporting format, or data acquisition limit, necessitating the reporting of the data as mean (±SD), median [25$^{th}$-75 IQR] and 95$^{th}$ percentile at the 10*g* data acquisition limit. Even using these reporting formats, some studies utilised the 75$^{th}$ percentile, the median of the 95$^{th}$ percentile,[109] while other studies did not report rotational accelerations[449] limiting inter-study comparisons. Some studies also combined matches and practices and only reported the total biomechanical data while others incorporated both match and practice biomechanical data,[109] again limiting potential comparisons. A consensus needs to be established on what the reporting formats for impact biomechanical data (i.e. match vs practice and not combined) should be, and what parameters should be reported (i.e. PLA(*g*), PRA(rad/s$^2$), HITsp, GSI, $HIC_{15}$) to enable future inter-study comparisons to be completed.

The median linear acceleration value recorded (17*g*) was lower than the medians reported in American high school (20.5*g*, 21*g*, and 21.9*g*)[49, 150, 596] and collegiate (15*g*, 21*g*, 22g)[53, 109, 449] football players. The mean linear acceleration (23*g*) was also lower than the mean of some (32*g*),[141] but not all, studies (21*g*)[53] providing data for American collegiate football players. The median rotational acceleration value recorded (3,045 rad/s$^2$) is higher than the medians reported in American high school football (671 rad/s$^2$; 1,013 rad/s$^2$)[596] but closer to the 95$^{th}$ percentile reported (2,743 rad/s$^2$; 2,347 rad/s$^2$).[115, 596] When compared with American collegiate football, the median rotational acceleration value recorded was higher than the median of impacts reported (1,392 rad/s$^2$),[111] subconcussive rotational accelerations (872-981 rad/s$^2$),[531] but similar to concussive rotational accelerations recorded (4,948 rad/s$^2$).[531] The mean rotational acceleration (4,119 rad/s$^2$) was also higher (1,670 rad/s$^2$) than values for American high school players. Compared with the median of the 95$^{th}$ percentile of American collegiate football players (4,378 rad/s$^2$)[109] the findings were similar to the mean of the impacts recorded but the 95$^{th}$ percentile of the linear accelerations recorded in this study were higher (11,562 rad/s$^2$). The higher extreme values could be reflective of the activities undertaken in rugby league when compared with American football and further studies are warranted to identify these differences.

The distribution of impacts by impact location showed that 41% of impacts occurred to the side of the head which is not the same as other studies reporting impacts in American high school[596] and collegiate[449] football. When viewed by positional groups the front of the head was the most common impact location for hit-up forwards (36%) which is similar for studies reporting American high school (46%)[596] and collegiate (36%)[449] football impacts in games. The differences in the impact locations when viewed by positional groups may be reflective to the roles these groups play during rugby league matches. Hit-up forwards are more involved in taking the ball straight into the opposition defensive line and are involved in more front on tackle situations whereas the outside backs and adjustables are involved in wider roaming role to either go outside the

defensive line or to attack the defensive line on an angle with the aim of breaking through the line to score a try.

The use of the X2Patch was novel in that the patch incorporates the accelerometer and gyroscope into a reusable monitor adhered to the side of the head behind the ear over the mastoid process of the player. The patches were applied utilising an adhesive patch to hold the patch to the adhesive and this was applied to the side of the head behind the ear. The effects of sweating by the player, and some extreme weather conditions, resulted in some of the adhesives losing their adhesion and falling off the player during the match activities. As well, players who were grabbed around the head during the tackle sometimes had the patch pulled off. As a result the data reported was incomplete and the number of impacts would be more than have been reported.

The accelerometers in the X2Patch have a 10% error for linear and rotation acceleration and for angular velocity with an average offset of 2° for azimuth and elevation impact location.[63, 401] The correlation of the accelerometers with laboratory head-forms is good but the impact measurements should be assumed to have some form of error that is dependent on impact conditions and the measure of interest and the variability tested.[140] It is unlikely that the X2Patch was tested under all of the activities seen in rugby league matches such as the tackle and scrum situations and how these correlate to the laboratory conditions results. As such, the results presented in this study should be interpreted with some caution.

All games were videotaped to enable verification of the impacts recorded. The percentage of impacts identified that were able to be visualised by video review and analysis was 95% of the total impacts recorded per match as only one video recorder was used. The ball carrier with possession of the ball is required to be tackled to the ground and, in most cases, this required two or more defenders to be involved in the tackle. The aim of the tackle was to cause the ball carrier to lose possession of the ball resulting in the defence to regain possession, or to be taken to the ground and, ideally, turned on their back to slow down the play-the-ball situation. Defending players joining the tackle in an attempt to put the ball carrier on the ground would result in multiple of impacts to the ball carrier being recorded. As a result not all the impacts recorded were able to be identified with the use of only one camera.

## Conclusion

Our study was conducted to explore and describe the magnitude, frequency and distribution of head impacts sustained by a single amateur senior premier rugby league team of 38 players over a 2014 match competition season. There were 13,895 impacts recorded via accelerometer fitted patches applied to behind the ear of players. There was an average of 56 impacts over 10*g* per game over the course of the season. The majority (41%) of impacts occurred to the side of the head which is not the same as other studies reporting impacts in American high school and collegiate football. Median resultant linear accelerations were lower than high school and collegiate American football while the median resultant rotational accelerations were higher. The cumulated impacts per player, per match were 56 ±22 and 731 ±289 respectively. Although we reported the data by means (±SD), median and 95$^{th}$ percentile to enable comparisons across the studies to be conducted, the need for standardization of reporting head impact biomechanics is needed.

# Part 5: Use of the King-Devick test for sideline assessment of concussion in junior rugby league

## Overview

The King-Devick test is a rapid number naming reading timed-test that assesses changes in the saccades or fast eye-movements of the player based on previous baseline assessments. Areas of the brain involved in saccades are widespread and can be affected without any observable concussion signs and symptoms being recorded. To date, there has been no published research surrounding the use of the King-Devick test in junior sports participants. Therefore we wanted to quantify King-Devick base line values for junior rugby league players. Nineteen junior rugby league players were assessed pre-season and after each match or when there was parental concern of a concussive injury. The King-Devick test was able to identify players with functionally-detected cognitive impairment without any observable concussion symptoms.

## Introduction

Once trivialised by coaches, sport-related concussions were viewed as a transient injury void of long term consequences.[43] Clashes to the head who were often thought of as "just a ding"[595] and "playing through the pain" was regarded as a sign of the individual's toughness and commitment to the team.[43] All sports participants are at risk of receiving a concussive event.[434] This risk is increased in players with a history of a previous or multiple concussive events.[291] Concussion have become one of the most troublesome injuries facing the sports medicine professional,[347] especially in regards to early identification of concussive signs and symptoms and appropriate concussive management facilitation.[409] Often the identification of a sport-related concussion is left to the team coach as trained health care professional are rarely available at amateur games and training sessions.[347] Concussive signs may be missed, as the coach may be unable to take the required time to fully assess the player and may overlook the signs of a concussion.[327]

Eye function movements may become impaired following brain trauma.[81, 236] In acute traumatic brain injuries latency and inaccuracy of saccades occur following the injury.[269] This can remain in people with post-concussion syndrome where there are a higher number of saccades occurring and poor motor movement timings with longer durations and slower velocities of movement.[270] Poor oculomotor function is reported as one of the most robust discriminators for the identification of,[270] and one of the most widely reported visual problems in,[81, 236] a mild-traumatic brain injury.

The King-Devick was originally developed as a reading tool to assess the relationship between poor oculomotor functions and learning disabilities. The test utilises a series of charts of numbers that progressively become more difficult to read in a flowing manner.[352] The K-D test requires eye movements, language function and attention in order to perform tasks shown to be reflective of suboptimal brain function in hypoxia,[570] extreme sleep deprivation,[120] multiple sclerosis,[462, 508] Parkinson's[370] and concussion.[134, 202, 203, 206, 314, 317, 367, 395] The K-D has been utilised in representative rugby league[317] and domestic rugby union[314]

and identified non-witnessed or 'incidental' concussions. Players identified with changes in their baseline K-D were further assessed with the Sports Concussion Assessment Tool v. 2 (SCAT2) and the identification of a sport-related concussion was confirmed. This studies have highlighted the potential of the K-D to detect sub-concussive impacts that may accumulate over a period and can lead to neurological changes.[317]

Although the K-D test has been utilised as a sideline concussion screening tool in several contact sports,[134, 202, 203, 206, 314, 317, 367, 395] no study, has reported the use of the K-D test with junior players in any sporting environment. This study used the K-D test as a sideline assessment in junior rugby league players over a domestic competition season to identify: (1) the test-retest reliability of the K-D test in junior players; (2) if the K-D test could identify concussive events that occurred from match participation; (3) to identify the over-season learning effect of utilising the K-D test; and (4) if the changes observed in the K-D test were clinically meaningful.

## Methods

A prospective observational cohort study was conducted on a junior club level rugby league team during the 2014 competition in New Zealand. Nineteen players (males n=14; females n=5) were enrolled in the study with a mean age, mass and height of 10.4 ±0.9yr; 54.9 ±17.2kg and 1.52 ±0.11m. Parental consent was obtained from the players parents / guardians prior to enrolment in the study. All players participated in the modified version of Rugby League (Mod-League) over a single competition season administered through a zonal region under the jurisdiction of the New Zealand Rugby League (http://www.nzrl.co.nz/pdf/guide_to_the_laws_2010.pdf). The match rules for rugby league have been adjusted to accommodate for younger participants. The adjustments for this age group (Mods: Under 10 – Under 11 yr.) include a smaller field (80 x 48 m); fewer players participating (11 per team on field (five forwards (props=2; hooker=1, second row=2) and six backs (Halfback=1, five-eight=1, centre=2, wing=2), or fewer if the opposition has less); two x 20 minute halves, no points awarded for a win, no final series and two passes from the play the ball following a tackle utilizing a smaller ball (270 mm x 162 mm). Similar to the adult version of rugby league, the team in possession must carry the ball forward to attempt to score a try, but can only pass the ball backwards for a maximum of six tackles before handing the ball to the opposition. Unlike the adult version of rugby league, both males and females participated in the same game against teams with a similar mixture of male and female players. The researchers' University ethics committee approved all procedures in the study (AUTEC 12/156).

### King-Devick (KD) tool

Based on the time to perform rapid number naming, the King-Devick (K-D) test took less than two minutes to administer.[202, 203] The K-D test involved the players reading aloud a series of random single-digit numbers from left to right. The K-D test included one practice (demonstration) card and three test cards varied in format (see Figure 5.1) on either a moisture-proof 6x8 inch spiral bound physical test or as an application on a IPad platform. Players were asked to read the numbers from left to right across the card as quickly as

they could without making any errors using standardized instructions. Time was kept for each test card, and the K-D summary score for the entire test was based on the cumulative time taken to read all three test cards. The number of errors made in reading the test cards was recorded. Baseline K-D times for all participants were established either preseason (n=11) or when participants joined the team after the season had commenced (n=8). The best time (fastest) of the two trials without errors became the established baseline K-D test time.[202] When head trauma was suspected the K-D test is used as a screening tool. The test is administered once using the same instructions and the time and errors are recorded then compared to the subject's baseline. Worsening of time and/or errors identified on the sideline or post-match K-D test have been associated with concussive injury.[134, 202, 203, 206, 314, 317, 395] K-D test performance has been previously shown to be unaffected in various noise levels and testing environments.[567] The K-D has been reported to have significant correlations ($p$<0.0001) with the visual motor speed (VMS), reaction time (RT), verbal memory (VEM) and visual memory (VIS) of the Immediate Post-concussion Assessment Cognitive Test (ImPACT®)[588] computerised concussion evaluation system. The K-D has been reported to have an inter-class correlation for test-retest reliability of 0.96[367] and 0.97.[202] The K-D tests utilised were v2.2.0 (http://www.kingdevicktest.com) on an IPad2. The IPad2 version enables the use of the K-D test with two different test platforms and these were varied over the duration of the study.

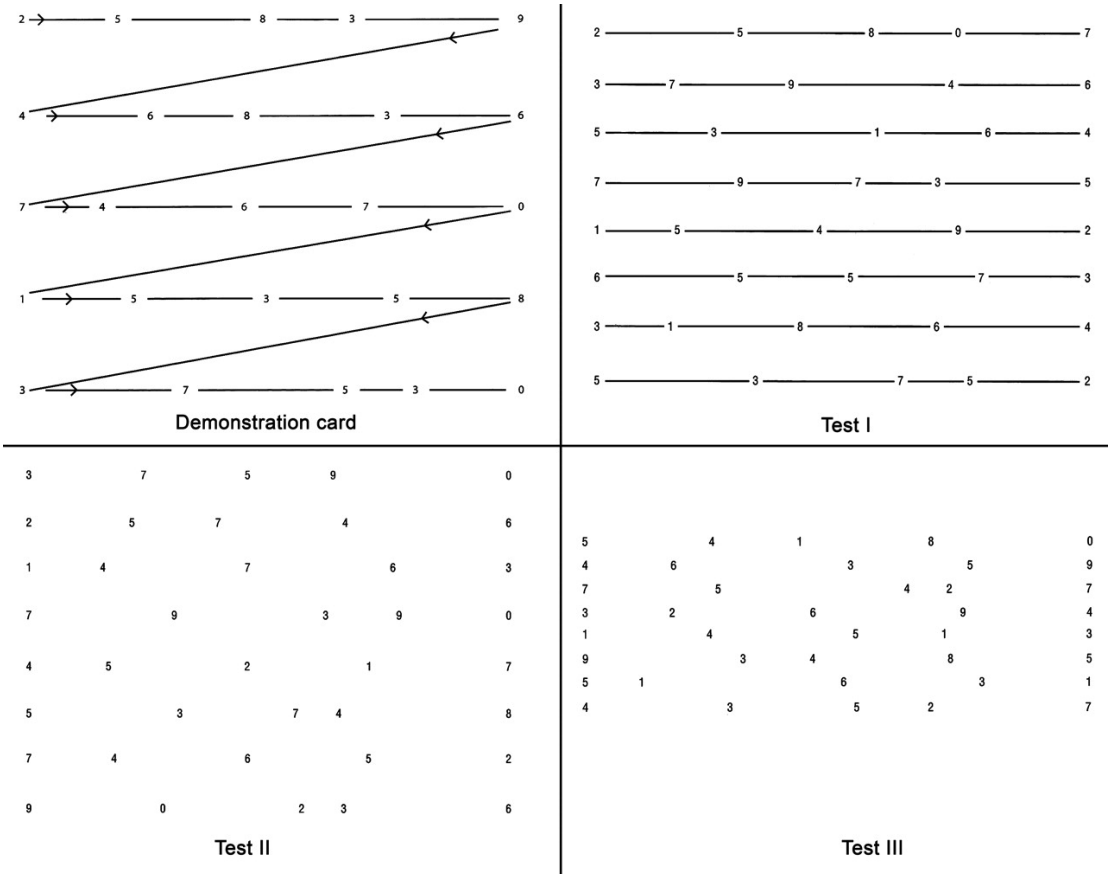


**Figure 5.1:** Practice (demonstration) and test cards for the King-Devick test.

### Testing procedures

Players were tested on the side of the training field twice within a 10 minute period before a training session by the same examiner. No player undertook any match activities until they had established a baseline K-D test. All players were asked to read aloud the practice (demonstration) card before reading aloud all three test cards. Only the three test cards times were recorded. The number of errors made in reading the test cards were recorded.[206] The fastest error-free time of the K-D test was recorded as the baseline score.[317] Players were asked to additionally complete the K-D test after each match they participated in and at the end of the competition season (post-season K-D test).

During matches, the team medic (and lead researcher), observed players for any signs of a direct blow to the head, for being slow to rise from a tackle or collision, or for being unsteady on their feet following a collision. If this occurred the players were assessed on-field. If there were any signs of delayed answering, incorrect answers to questions, or if the player appeared to be impaired in any way, the player would be removed from the match activity and rested on the sideline. Players who reported any sign(s) of a concussion, suspected to have incurred a concussion or who were removed from match participation were assessed with the K-D on the sideline, not allowed to return to play on the same day and referred for further medical assessment. No player identified with delayed (worsening) post-match K-D times was allowed to return to training or match activities without a full medical clearance.

### Statistical analyses

All data collected were entered into a Microsoft Excel spread sheet and analysed with SPSS v22.0.0. Data are presented as mean (±SD) for player data and median values [$25^{th}$-$75^{th}$ interquartile range] for K-D scores. Given the small sample size (n=19), non-parametric statistical tests were utilised. Differences in K-D scores from pre-season (baseline establishment) and post-match K-D scores or post-season K-D scores, were compared using the Wilcoxon signed-rank test. Statistical significance was set at $\alpha=0.05$.

## Results

Twelve matches were competed in resulting in a match exposure of 88.0 match hours. Throughout the competition, no on-field concussions were observed. The characteristics and K-D testing data for the study cohort are show in Table 5.1. There were improved K-D time scores for the second K-D time test when compared with the first time test during baseline testing (67.7 s vs 62.2 s; $p=0.001$). Post season testing of the players demonstrated improvement of the time scores which is likely consistent with learning effects of using the K-D (62.0 s vs. 54.6s; $p=0.002$). Six players were identified post-match with K-D test times greater than 3 s from their baseline with a mean change of 7.4 s (±7.0 s; $p=0.018$). One player recorded post-match K-D test times greater than 3 s from their baseline twice throughout the study period. In addition, one player with a post-match delay score (101.4 s vs. 78.4 s) when compared with his baseline also had two errors on post-match K-D testing. No other player enrolled in the study had errors on their post-match K-D testing.

**Table 5.1:** Player characteristics and the scores, changes and differences in the results of the King-Devick test for junior rugby league players.

| | All players (n=19) | Players with no post-match K-D changes (n=13) | Players with post-match K-D changes (n=6)*c |
|---|---|---|---|
| Age, y ±SD | 10.4 ±0.9 yr. | 10.5 ±0.8 | 10.2 ±1.2 |
| Pre-season K-D test 1, s, mean (±SD) | 67.7 (±10.1) | 64.6 (±9.3) | 74.4 (±8.8) |
| Pre-season K-D test 2, s, mean (±SD) | 62.2 (±10.2)[a] | 59.4 (±9.8) | 68.2 (±9.0) |
| Difference test 1 vs. test 2, s, mean (±SD) | -5.5 (±6.1) | -5.2 (±4.7) | -6.1 (±8.8) |
| Baseline K-D score, s, mean (±SD) | 62.0 (±9.8) | 59.4 (±9.8) | 67.4 (±7.9) |
| Post-match K-D test, s, mean (±SD) | 58.8 (±10.0) | 55.5 (±9.2) | 70.0 (±15.4)[b] |
| K-D change baseline vs. post-match, s, mean (±SD) | -1.2 (±5.0) | -3.9 (±3.3) | 7.4 (±7.0)‡ |
| Post season K-D test, s, mean (±SD) | 54.6 (±10.5)[b] | 51.8 (±9.3) | 60.7 (±11.0) |
| K-D change pre-to post season, s, mean (±SD) | -7.4 (±5.4) | -7.7 (±5.2) | -7.6 (±5.7) |
| ICC (95% CI), K-D baseline 1 vs. baseline 2 | 0.92 (0.79 to 0.97) | - | - |
| ICC (95% CI), K-D baseline vs. post-season | 0.92 (0.80 to 0.97) | - | - |
| Sensitivity (95% CI) K-D test | 1.00 (0.73 to 1.00) | - | - |
| Specificity (95% CI) K-D test | 0.85 (0.42 to 0.97) | - | - |

Abbreviations: K-D = King-Devick test; SD = Standard Deviation; IQR = Inter-Quartile Range; CI = Confidence Interval; s = seconds; Significant difference ($p$<0.05) than (a) = test 1 of baseline; (b) = established baseline; * = one player recorded post-match changes >3 sec twice throughout the competition; (c) = all players with post-match K-D changes were medically assessed and diagnosed with a concussion by their own health practitioner ‡ = positive numbers for change in K-D score indicates longer (worsening) than baseline score

## Discussion

This study is the first to report the results for K-D test monitoring for junior rugby league players. Although no witnessed concussions were recorded over the duration of the study, seven players recorded post-match changes with a mean delay of 4 s from their baseline. All players who recorded a delay from their baseline and their parents were advised of their results and referred for further medical assessment for a possible concussion. Signs and symptoms of concussion can develop over time[140, 423] and, as some players, coaches and parents may not be fully unaware of the actual signs and symptoms of a concussion[315] the requirement for a full medical clearance was to ensure that these players were medically assessed. All seven players referred for further medical assessment were identified to have had a concussion and were medically managed for a return to sports participation. No player with a delay in their post-match K-D score was allowed to return to match or training activities until medically cleared.

In the absence of concussion, the K-D test has been shown to have learning effects associated with repeat testing.[203] This finding is commonly associated with repeated performance measures, or timed testing[203] and can be seen by the mean improvement of 5.5 s between the two tests undertaken to establish the individual players baseline score. This was similar to previous values for the K-D in sports activities with improvements of 1.9 s to 3.1 s[202, 203, 206, 314, 317, 367] for establishing the baseline score of participants. The use of dual baseline assessments allows familiarization of the test and can help alleviate any possible confounding factors[600] such as previous exposure to similar tests. Although employing serial K-D assessments has a learning effect, there should be an improvement in the score upon each subsequent test administration.[600] The IPad version of the K-D test (v2.2.0) utilised in our study had two sets of demonstration and test cards with different numbers along the same pattern. The post-match K-D tests were varied weekly to eliminate any possibility of players learning the numbers. Although tests were varied, players showed an increase in the

scores from their baseline. Any worsening of the scores is likely to be a reliable indicator that players should be evaluated further for a possible concussive event.[203]

The procedures utilised for the assessment and management of players identified to have a worsening of their K-D test score are identical to a previous study.[317] The post-match testing was undertaken at least 15 minutes after the game had finished to allow for any of the complex cascade of ionic, neurometabolic, neurochemical and physiologic events to occur.[233] This is reported to occur in the first 10 min from a concussive event and then a neuronal depression period occurs where cognitive dysfunction manifests.[233] Previous studies[19-21,23-25] have reported K-D test times of participants with a concussive injury having a median of 5 s slower (range 3.2 s to 18.0 s) than their baseline. Our study had a mean of 7.4 s slower than baseline for players who were either removed from match participation or tested post-match and all these players underwent medical evaluation through their own medical practitioner. All players with a worsening of their K-D test score were diagnosed with a concussion by their medical practitioner and underwent the required return to sport evaluations and clearances before they were allowed to participate in training and match activities.

The detection, and removal from play, of players with a possible concussion may help minimize the deleterious outcomes of concussion. The use of a practically implemented, rapid assessment tool for sideline evaluation of sports participants is vital in the detection of concussive injuries. The K-D test is a portable tool that can be utilised on the sideline and is simply based on the time taken to complete a rapid number naming task.[142] The K-D test requires the coordination of visual processes, eye movements (saccades, convergence and accommodation), attention and language function to complete this rapid number naming task.[142] This task requires the integration of functions of the cerebral cortex, cerebellum and brainstem and this has been shown to correlate with suboptimal brain function.[142] It does not require a medical professional to administer[367] making this a realistic sideline assessment tool for amateur sporting activities at all levels of participation, especially where there is minimal or no access to on-site medical personnel.

## Conclusion

The finding of unwitnessed, or incidental, medically assessed and confirmed concussions in the current study was unexpected, but similar to previous studies.[206, 314, 317, 395] A benefit of conducting this research in the current cohort of junior players was the ability to discuss concussions with the parents on an informal basis resulting in some parents presenting their child for assessment after they felt there was a possible concussive event that was not identified during the game. Concussed professional athletes were identified with worsening in the K-D test performances testing.[206] The variety of symptoms that may go unreported by athletes, and undetected by trained observers, emphasize the need for a quick and reliable sideline screening tool to assist team management and parents to accurately detect if a concussive type event has occurred. The results of this study further validate the K-D test as a reliable and rapid sideline tool that provides supportive evidence of a concussive event and assists with removal from play decisions. The K-D

can be quickly and easily administered by non-medically trained lay-persons making this a practical sideline tool as part of the continuum of concussion assessment tools.

## Key findings and recommendations

**Key findings are:**

- Rugby league has the highest percentage of sport code total costs and mean costs per ACC claim for concussion (92.2%; $25,545).
- Semi-professional participants had nearly a two-fold greater concussion injury rate than professional participants and a three-fold greater concussion injury rate than amateur participants.
- Throughout the 2014 match competition season, there were 1,977 impacts recorded via accelerometer patches applied behind the ear of 19 junior players. The youth players were exposed to impacts that can be considered high-magnitude at any level of participation. Despite the number of high-magnitude impacts recorded, there were no witnessed concussive events throughout the duration of the study.
- There were 13,895 impacts recorded via accelerometer patches applied behind the ear of 38 senior players. There was an average of 56 impacts over 10*g* per game over the course of the season. The majority (41%) of impacts occurred to the side of the head. The cumulated impacts per player, per match were 56 ±22 and 731 ±289 respectively.
- The King-Devick test was able to identify junior players with functionally-detected cognitive impairment without any observable concussion symptoms.

**Based on these findings we recommend:**

- Player and team management education surrounding prevention, identification and assessment of concussion is essential.
- Baseline and post-match assessment of all players using the King-Devick is required.
- Prospective studies to determine the cumulative impact loads on players using the accelerometer patches applied behind the ear and concussion outcomes is required.
- Validation studies using the accelerometer patches are needed.
- Studies using players at all levels of participation are needed (particularly juniors, females and Maori).

# Appendix III: Chapters 5, 6, and 7 ethics approval from the AUTEC

**M E M O R A N D U M**
Auckland University of Technology Ethics Committee (AUTEC)

| | |
|---|---|
| To: | Matt Brughelli |
| From: | Rosemary Godbold, Executive Secretary, AUTEC |
| Date: | 26 July 2012 |
| Subject: | Ethics Application Number **12/156 Impact forces associated with match participation in rugby league and rugby union and the identification of sport related concussion.** |

Dear Matt

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC) at their meeting on 9 July 2012 and I have approved your ethics application. This delegated approval is made in accordance with section 5.3.2.3 of AUTEC's *Applying for Ethics Approval: Guidelines and Procedures* and is subject to endorsement by AUTEC at its meeting on 13 August 2012.

Your ethics application is approved for a period of three years until 25 July 2015.

I advise that as part of the ethics approval process, you are required to submit the following to AUTEC:

- A brief annual progress report using form EA2, which is available online through http://www.aut.ac.nz/research/research-ethics/ethics. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 25 July 2015;
- A brief report on the status of the project using form EA3, which is available online through http://www.aut.ac.nz/research/research-ethics/ethics. This report is to be submitted either when the approval expires on 25 July 2015 or on completion of the project, whichever comes sooner;

It is a condition of approval that AUTEC is notified of any adverse events or if the research does not commence. AUTEC approval needs to be sought for any alteration to the research, including any alteration of or addition to any documents that are provided to participants. You are reminded that, as applicant, you are responsible for ensuring that research undertaken under this approval occurs within the parameters outlined in the approved application.

Please note that AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

To enable us to provide you with efficient service, we ask that you use the application number and study title in all written and verbal correspondence with us. Should you have any further enquiries regarding this matter, you are welcome to contact me by email at ethics@aut.ac.nz or by telephone on 921 9999 at extension 6902. Alternatively you may contact your AUTEC Faculty Representative (a list with contact details may be found in the Ethics Knowledge Base at http://www.aut.ac.nz/research/research-ethics/ethics).

On behalf of AUTEC and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Dr Rosemary Godbold
Executive Secretary
**Auckland University of Technology Ethics Committee**
Cc:Doug King doug.king@xtra.co.nz;dking@aut.ac.nz, Patria Hume

# Appendix IV: Sample of a participant consent form.

## Parent/Guardian Consent to Participation in Research

AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

Title of Project: **Impacts that occur in rugby union and rugby league matches and the identification of sport-related concussions.**

Project Supervisor: **Dr Matt Brughelli**

Researcher: **Dr Doug King**

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- I have read and understood the information provided about this research project (Information Sheet dated 17th July 2012).
- I have had an opportunity to ask questions and to have them answered.
- I agree to participate in the research and to have myself videoed during matches.
- I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
- I understand that in the event of a medical situation the information obtained as part of this research project may be used to assist in my medical care and that my identified legal guardian, next-of-kin or parent will be informed of the situation.
- If I withdraw, I understand that all relevant information will be destroyed.
- I wish to receive a copy of the report from the research: tick one: Yes O No O

Participant's Name: .....……

Parent / Guardian's Name: …… Parent / Guardian's Signature: ……..……

Contact Details (if appropriate): .....

.....

.....

Date: ……

***Project Supervisor Contact Details:***
Dr Matt Brughelli,
Sports Performance Research Institute New Zealand,
School of Sport and Recreation,
Auckland University of Technology.
Private Bag 92006
Auckland 1020
64 9 921 9999 ext .7025
mbrughelli@aut.ac.nz

***Approved by the Auckland University of Technology Ethics Committee on 16th July 2012 AUTEC Reference number* 12/156**

# Appendix V: Sample of a participant information pack.

## Participant Information Sheet

**Date Information Sheet Produced:** 12th November 2012

**Project Title**

### Impacts that occur in rugby union matches and the identification of sport-related concussions

***Introduction***

My name is Dr Doug King and I am a PhD student at Auckland University of Technology (AUT) in Auckland. I am also a senior Clinical Nurse Specialist employed in the Emergency Department of the Hutt Valley District Health Board. I am looking at doing research on sport-related concussion and the impacts that occur during rugby union and rugby league match activities.

***Invitation to participate***

- You are invited to take part in the above mentioned research project. Your participation in this research is voluntary. You are free to withdraw consent and discontinue participation at any time without influencing any present and/or future involvement with the Auckland University of Technology.
- Your consent to participate in this research will be indicated by your signing and dating the consent form. Signing the consent form indicates that you have freely given your consent to participate, and that there has been no coercion or inducement to participate by the researchers from AUT.

***What is the purpose of the study?***

- The purpose of the study is to monitor and record the impacts that occur from participation in rugby union and rugby league match activities and when a sport-related concussion occurs over a two year period.
- This study is being conducted as part of a PhD degree. The results of this study will be presented at national / international conferences and submitted to peer-reviewed journals.

***How was I chosen to be asked to participate in the study?***

- Those people who participate in rugby union and rugby league match activities will be invited to participate in the research over two years.

***What happens in the study?***

- You will be asked to complete a pre-competition concussion history questionnaire, a sports concussion assessment baseline evaluation, undertake a measurement of your head and neck and to complete a number reading test. These questionnaires and tests will be used to evaluate you against in the event of a suspected concussion occurring. This information will be collected at the beginning of your enrolment in the study. This information may also be passed onto other healthcare services should there be a medical situation where you are required to be referred for further care and management.
- You will be asked to wear an acelerometer fitted mouthguard while undertaking match activities while you participate in rugby union or rugby league match activities over the next two years. This accelerometer fitted mouthguard will be monitored from the sideline.
- You will be videoed during matches to enable the impacts that occur to you while you play to be correlated with the data that is collected. This video is to be viewed by the researchers only for the

research analysis and will not be used for any rugby related assessment by anyone not directly identified as part of the research team.

***What are the discomforts and risks?***

- Only those discomforts and risks that normally occur from participating in rugby union or rugby league match activities. This includes the risk of a sports-related concussion. This risk can be increased if you have had a previous concussion and this will be discussed with you as part of the concussion history assessment. You may be asked to see another health care professional for further assessment and clearance to play as part of this process

***What are the benefits?***

- Information gained from this research has potential to help shape training strategies, and develop prognostic indicators of value to athletes, clinicians, physical conditioners and coaches.

***What compensation is available for injury or negligence?***

- In the unlikely event of a physical injury as a result of your participation in this study, rehabilitation and compensation for injury by accident may be available from the Accident Compensation Corporation, providing the incident details satisfy the requirements of the law and the Corporation's regulations.

***How is my privacy protected?***

- The data from the project will be coded and held anonymously in secure storage under the responsibility of the principal investigator of the study in accordance with the requirements of the New Zealand Privacy Act (1993).
- All reference to participants will be by code number only in terms of the research thesis and publications. Identification information will be stored on a separate file and computer from that containing the actual data.
- Only the investigators will have access to computerised data.
- Should a situation occur where you become injured then your identified next-of-kin / legal guardian / parent that has been recorded and/or signed the consent form will be contacted to advise them of the injury, the care provided and where you have been transferred to. The information obtained will also be passed onto the healthcare service as part of the on-going management of your medical care.

***What are the costs of Participating?***

- Participating in this research project will not cost you apart from your time that you normally provide for participating in rugby union and rugby league activities.

***Opportunity to consider invitation***

- Please take the necessary time you need to consider the invitation to participate in this research.
- It is reiterated that your participation in this research is completely voluntary.
- If you require further information about the research topic please feel free to contact Doug King (details are at the bottom of this information sheet).
- You may withdraw from the study at any time without there being any adverse consequences of any kind.
- You may ask for a copy of your results at any time and you have the option of requesting a report of the research outcomes at the completion of the study.

***How do I join the study?***

- If you are interested in participating in this research please feel free to contact Dr Doug King (details are at the bottom of this information sheet).

***Participant concerns***

If you have any questions please feel free to contact Dr Doug King or Dr Matt Brughelli. Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor – Dr Brughelli. Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, madeline.banda@aut.ac.nz , 917 9999 ext 8044.

***Researcher Contact Details:*** Dr Doug King, Sports Performance Research Institute New Zealand, School of Sport and Recreation, Auckland University of Technology. Email: dking@aut.ac.nz or phone +64 27 257 9098.

***Project Supervisor Contact Details***

Dr Matt Brughelli, Sports Performance Research Institute New Zealand, School of Sport and Recreation, Auckland University of Technology. Email: mbrughelli@aut.ac.nz or phone +64 9 921 9999 ext .7025

**Approved by the Auckland University of Technology Ethics Committee on** 9th July 2012. **AUTEC Reference number** 12/156

# Appendix VI: Player Concussion Questionnaire

*1*) How old are you?

( ) 15 ( ) 16 ( ) 17 ( ) 18 ( ) 19 ( ) 20
( ) 21 ( ) 22 ( ) 23 ( ) 24 ( ) 25 ( ) 26 ( ) 27 ( ) 28 ( ) 29
( ) 30 ( ) 31 ( ) 32 ( ) 33 ( ) 34 ( ) 35 ( ) 36 ( ) 37 ( ) 38

2) What ethnic group do you primarily associate yourself with? Please choose one group only

( ) New Zealand European ( ) New Zealand Maori
( ) Pacific Peoples ( ) Asian ( ) Other

3) What is your primary playing position? Please choose one group only

( ) Fullback ( ) Wing ( ) Centre ( ) Stand-off ( ) Half-Back
( ) Prop ( ) Hooker ( ) Second Row ( ) Loose Forward

4) How many years have you been playing Rugby League

( ) < 1 yr. ( ) 1 - 5 yr. ( ) 5 - 10 yr. ( ) > 10 yr.

***The following questions refer to symptoms that you may have experienced from ALL sporting activities you have participated in over the last two years***

***Note:***

***a. "After being hit in the head playing sports" refers to any contact with your head; either from another player, yourself, the ground or another object that may have occurred while participating in any of your sporting activities.***

***b. Duration in symptoms for the following questions can be listed in number of seconds, minutes, hours, days, weeks, etc.***

*5*) In the past **two** years, after being hit in the head playing sports, did you ever suffer a **concussion**?*

( ) Yes ( ) No

6) In the past **two** years, after being hit in the head playing sports, were you ever "**knocked out**" or "**knocked unconscious"**?

( ) Yes( ) No

7) In the past **two** years, after being hit in the head playing sports, did you ever feel **confused** for a period of time? This feeling is sometimes referred to as getting "**dinged**" or having your "**bell rung**." You may have felt lightheaded, not remembered the play, or not known where you were.

( ) Yes( ) No

8) In the past **two** years, after being hit in the head playing sports, did you ever experience **headaches**?

( ) Yes( ) No

9) In the past **two** years, after being hit in the head playing sports, did you ever experience **dizziness** or **balance problems**?

( ) Yes ( ) No

10) In the past **two** years, after being hit in the head playing sports, did you ever have **memory difficulties** (difficulty remembering things)? This may have included not being able to remember the plays that were called, forgetting where you were, forgetting the score, etc.

( ) Yes ( ) No

11) In the past **two** years, after being hit in the head playing sports, did you ever have **blurred** or **abnormal vision**? This may have included a feeling of having tunnel vision, difficulty focusing on objects, seeing abnormal colours, double vision, etc.

( ) Yes ( ) No

12) In the past **two** years, after being hit in the head playing sports, did you ever experience **nausea** (feeling sick in your stomach or wanted to vomit or vomited)?

( ) Yes ( ) No

13) In the past **two** years, after being hit in the head playing sports, did you ever experience **hearing problems** (including ringing in your ears)?

( ) Yes ( ) No

14) Are there **any other symptoms** you experienced in the past **two** years, after being hit in the head playing sports? These may include inability to tolerate bright lights, loud noises, irritability, emotional changes, slurred speech etc.

( ) Yes ( ) No

***The following questions refer to symptoms that you may have experienced from** ALL **sporting activities you have participated in before the last two years***
***Note:***

***"After being hit in the head playing sports" refers to any contact with your head; either from another player, yourself, the ground or another object that may have occurred while participating in any of your sporting activities.***

15) ***Prior to** the past **two** years, after being hit in the head playing sports, did you ever suffer a **concussion**?**

( ) Yes ( ) No

16) ***Prior to** the past **two** years, after being hit in the head playing sports, were you ever "**knocked out**" or "**knocked unconscious"**?*

( ) Yes( ) No

17) ***Prior to** the past **two** years, after being hit in the head playing sports, did you ever feel **confused** for a period of time? This feeling is sometimes referred to as getting "**dinged**" or having your "**bell rung**." You may have felt lightheaded, not remembered the pl**ay, or not known where you were?***

( ) Yes( ) No

18) If you have had a concussion, after being hit in the head playing sports, did you undergo any form of medical assessment by either your own General practitioner, a Doctor at an after-hours facility or through the Emergency Department?

( ) Yes( ) No

19) If you have had a concussion, after being hit in the head playing sports, did you undergo the recommended three (3) week stand-down period as described by the NZRL concussion policy?

( ) Yes( ) No

20) If you have had a concussion, after being hit in the head playing sports, did you complete a medical clearance to return to play through either your General practitioner, a Doctor at an after-hours facility or through the Emergency Department?

( ) Yes( ) No

21) Do you currently have any of the following symptoms that may be as a result of having a **concussion**, **being knocked out or loss of consciousness**, having been "**dinged**" or had your "**bell rung**". Please indicate the symptom and the severity of the symptom as identified.

0 = No symptoms 1 - 2 = Mild/Sometimes; 3 - 4 = Moderate; 5 - 6 = Severe/Always

| | Symptom | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|---|---|
| 1) Headache | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 2) "pressure in the head" | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 3) Neck Pain | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 4) Nausea or vomiting | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 5) Dizziness | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 6) Blurred Vision | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 7) Balance problems | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 8) Sensitivity to light | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 9) Sensitivity to noise | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 10) Feeling slowed down | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 11) Feeling like "in a fog" | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 12) "Don't feel right" | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 13) Difficulty concentrating | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 14) Difficulty remembering | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 15) Fatigue or low energy | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 16) Confusion | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 17) Drowsiness | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 18) Trouble falling asleep | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 19) More emotional | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 20) Irritability | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 21) Sadness | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |
| 22) Nervous or anxious | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | ( ) |

# Appendix VII: Equations and Calculation formulas utilised

## Gadd Severity Index (GSI)

In 1966, Gadd[201] proposed a head injury severity index based on the Wayne State Tolerance Curve (WSTC) and it was named the Gadd Severity Index (GSI).[439] Developed from animal and cadaver impact data, the GSI simplified the WSTC by taking into consideration the shape of the linear acceleration time history, providing a weighting factor of 2.5 enabling the whole body acceleration data to be plotted on log-log coordinates along a straight line. The critical value of the GSI is 1,000, that is, if the GSI is less than 1,000 then the head impact is considered probabilistically safe. The GSI is used to quantify severe skull fractures and brain injury risk but is not recommended for use to quantify a risk of concussion.[19] A concern of the GSI is that it can give unrealistically high values for impacts that have a much longer pulse duration.[368] The mathematical expression for the GSI is:

$$GSI = \int_0^T a(t)^{2.5} dt$$

where *a* is the 'effective' acceleration (thought to have been the average linear acceleration) of the head measured in terms of *g*, the acceleration of gravity, and *t* is the time in milliseconds from the start of the impact.[439]

The range of GSI scores for the three cohorts studied were

| | |
|---|---|
| Amateur Senior Rugby Union: | 1.2 to 2,277.3 |
| Amateur Senior Rugby League: | 1.5 to 1,494.6 |
| Junior Rugby League: | 1.6 to 1,311.9 |

The witnessed concussions (n=3) GSI values were

| | |
|---|---|
| Amateur Senior Rugby Union (2 witnessed) | 152.9; 361.2 |
| Amateur Senior Rugby League (1 witnessed) | 1,494.6 |

## Head Impact Criterion (HIC)

In 1971 a modification of the Gadd Severity Index, the Head Injury Criterion (HIC), was proposed[604] to focus the severity index on that part of the impact that was likely to be relevant to the risk of injury to the brain. This was done by averaging the integration of the resultant acceleration/time curve over whatever time interval yielded the maximum value of HIC. Because this varies from one impact to another, the expression for the modified index simply refers to times *t*1 and *t*2. The HIC is computed based on the following expression

$$HIC = \left[\frac{1}{t_2 - t_1}\int_{t_1}^{t_2} a(t)dt\right]^{5/2}(t_2 - t_1)$$

where $t_2$ and $t_1$ are any two arbitrary time points during the acceleration pulse. Acceleration is measured in multiples of the acceleration of gravity [g] and time is measured in seconds. The resultant acceleration is used for the calculation. The US National Highway Traffic Safety Administration (NHTSA) requires $t_2$ and $t_1$ not to be more than 36 ms apart (thus called $HIC_{36}$) and the maximum $HIC_{36}$ not to exceed 1000. In 1998[342] the NHTSA introduced the $HIC_{15}$ where $t_2$ and $t_1$ was not to be more than 15 ms apart and the maximum HIC15 was not to exceed 700. In a numerical study[310] it was estimated that a mild Traumatic Brain Injury (mTBI) tolerance for the $HIC_{15}$, where there is a 25%, 50% and 75% of an mTBI occurring values were 136, 235 and 333 respectively. Although the HIC was assessed for 15 and 36 ms throughout the research undertaken, only the $HIC_{15}$ was reported

The range of HIC scores for the three cohorts studied were

| | **$HIC_{15}$** | **$HIC_{36}$** |
|---|---|---|
| Amateur Senior Rugby Union: | 0.7 to 1,736.2 | 0.8 to 2,142.8 |
| Amateur Senior Rugby League: | 1.0 to 1,086.8 | 1.0 to 1,206.2 |
| Junior Rugby League: | 1.0 to 937.5 | 1.0 to 1,112.7 |

The witnessed concussions (n=3) HIC values were

| | **$HIC_{15}$** | **$HIC_{36}$** |
|---|---|---|
| Amateur Senior Rugby Union (2 witnessed) | 81.0; 265.6 | 84.1; 368.2 |
| Amateur Senior Rugby League (1 witnessed) | 1,052.5 | 1,052.5 |

**It must be noted that the $HIC_{15}$ and $HIC_{36}$ only account for linear and NOT rotational accelerations**

## Head Impact Telemetry Severity Profile ($HIT_{SP}$)

In 2008,[241] the principal component score (PCS), a weighted sum of linear acceleration, rotational acceleration, HIC and GSI, with objectively defined weights, was published. Now more commonly referred to as the Head Impact Telemetry Severity Profile ($HIT_{SP}$), the $HIT_{SP}$ is a weighted composite score including linear and rotational accelerations, impact duration, as well as impact location. The resulting formula is:

$$HIT_{SP} = 10x([0.4718\ x\ sGSI + 0.4742\ x\ sHIC + 0.4336\ x\ sLIN + 0.2164\ x\ sROT] + 2)$$

where sX = (X-mean[X])/(SD[X]), LIN = linear acceleration, ROT = rotational acceleration, HIC = head injury criterion, and GSI = Gadd Severity Index. The offset by 2 and scaling by 10 generates $HIT_{SP}$ values greater than 0 and in the numerical range of the other classic measures studied. A $HIT_{SP}$ score of 63 or greater is reported to be an indication there is a 75% risk of a concussive injury occurring.[241]

The range of $HIT_{SP}$ scores for the three cohorts studied were

| | |
|---|---|
| Amateur Senior Rugby Union: | 10.9 to 243.0 |
| Amateur Senior Rugby League: | 6.3 to 201.5 |
| Junior Rugby League: | 10.5 to 194.1 |

The witnessed concussions (n=3) $HIT_{SP}$ values were

| | |
|---|---|
| Amateur Senior Rugby Union (2 witnessed) | 54.4; 66.7 |
| Amateur Senior Rugby League (1 witnessed) | 201.5 |

## Generalised Acceleration Model for Brain Injury Threshold (GAMBIT)

In an attempt to combine translational and rotational acceleration, the Generalized Acceleration Model for Brain Injury Threshold (GAMBIT) was proposed.[537] This Assumed that a combined load case of equal contributions of translational and rotational accelerations can cause head injury, and the following relationship was proposed:

$$GAMBIT = \left[\left(\frac{a(t)}{a_c}\right)^n + \left(\frac{\varphi(t)}{\varphi_c}\right)^m\right]^k$$

Here $a(t)$ and $\varphi(t)$ denote the translational and rotational acceleration, respectively. $a_c$ and $\varphi_c$ represent critical tolerance levels for those accelerations and *n*, *m* and *k* are constants. Through statistical analysis and computer simulations to available data, the following solution was presented[537]

$$GAMBIT = \left[\left(\frac{a(t)}{250}\right)^{2.5} + \left(\frac{\varphi(t)}{25}\right)^{2.5}\right]^{\frac{1}{2.5}}$$

with $a(t)$ and $\varphi(t)$ given in [*g*] and [krad/s$^2$]. Under the curve of the GAMBIT, the value of 1.000 (250 *g*; 25 krad/s$^2$) was determined to represent a 50% probability of an irreversible head injury while a non-contact head impact accounted for GAMBIT values below 0.620 (150 *g*; 20 krad/s$^2$).

To date the GAMBIT still lacks validation and is therefore hardly ever used.

The range of GAMBIT scores for the three cohorts studied were

| | |
|---|---|
| Amateur Senior Rugby Union: | 0.012 to 1.323 |
| Amateur Senior Rugby League: | 0.015 to 0.987 |
| Junior Rugby League: | 0.034 to 0.780 |

The witnessed concussions (n=3) GAMBIT values were

| | |
|---|---|
| Amateur Senior Rugby Union (2 witnessed) | 0.351; 0.292 |
| Amateur Senior Rugby League (1 witnessed) | 0.891 |

## Risk Weighted Cumulative Exposure

In 2013, a novel cumulative exposure metric, the Risk Weighted Cumulative Exposure (RWE) equation was developed[596] with four previously published analytical risk functions. The four previously published analytical risk functions were the linear resultant acceleration,[498, 529] rotational resultant acceleration[531] and the combined probability (linear and rotational) resultant accelerations.[530] These risk functions were utilised to elucidate individual player and team-based exposure to head impacts. The RWE equations comprise of $a_L$ as the measured peak linear acceleration, $a_R$ as the measured peak rotational acceleration, and $n_{hits}$ as the number of head impacts in a season for a given player.

| Risk function(s) | Equation |
|---|---|
| Linear[498, 529] | $RWE_{Linear} = \sum_{i=1}^{n_{hits}} \boldsymbol{R}(\boldsymbol{a}_L)i$ |
| Rotational[531] | $RWE_{Rotational} = \sum_{i=1}^{n_{hits}} \boldsymbol{R}(\boldsymbol{a}_R)i$ |
| Combined Probability[530] | $RWE_{CP} = \sum_{i=1}^{n_{hits}} \boldsymbol{CP}(\boldsymbol{a}_L, \boldsymbol{a}_R)i$ |

Logistic regression equations and regression coefficients of the injury risk functions utilised in the prediction of injury, where *α* and *β* are the regression coefficients and *x* is the measured acceleration for the linear and rotational risk functions.[596]

| Logistic Regression equation | Risk Function | Regression coefficients |
|---|---|---|
| $R[a] = \frac{1}{1 + e^{-\alpha+\beta x}}$ | Linear[498, 529] | $\alpha$ = -9.805, $\beta$ = 0.0510 |
| | Rotational[531] | $\alpha$ = -12.531, $\beta$ = 0.0020 |
| $\mathrm{CP} = \frac{1}{1 + e^{-(\beta_0+\beta_1 a+\beta_2 \alpha+\beta_3\, a\alpha}}$ | Combined Probability (CP)[530] | $\beta_0$ = -10.2, $\beta_1$ = 0.0433, $\beta_2$ = 0.000873, $\beta_3$ = -9.2E-07 |

$\beta_0, \beta_1$, $\beta_2$ and $\beta_3$ are regression coefficients, *a* is the measured linear acceleration, and *α* is the measured rotational acceleration for the combined probability risk function

The range of RWE scores for the three cohorts studied were:

| | **$RWE_{Linear}$** | **$RWE_{Rotational}$** | **$RWE_{CP}$** |
|---|---|---|---|
| Amateur Senior Rugby Union: | 0.000 to 0.038 | 0.000 to 1.000 | 0.000 to 1.000 |
| Amateur Senior Rugby League: | 0.000 to 0.074 | 0.000 to 1.000 | 0.000 to 1.000 |
| Junior Rugby League: | 0.000 to 0.027 | 0.000 to 1.000 | 0.000 to 1.000 |

The witnessed concussions (n=3) RWE values were

| | **$RWE_{Linear}$** | **$RWE_{Rotational}$** | **$RWE_{CP}$** |
|---|---|---|---|
| Amateur Senior Rugby Union (2 witnessed) | 0.001; 0.007 | 0.979; 0.721 | 0.250; 0.310 |
| Amateur Senior Rugby League (1 witnessed) | 0.092 | 0.994 | 0.937 |

# Appendix VIII: Supplemental data for Chapter 6

## Measurement of the frequency, magnitude, and distribution of head impacts in a senior amateur rugby union team recorded by an instrumented mouthguard

## Total, Match and Player Positional Group Impact Biomechanics Summaries

# Total Match Impact Biomechanics

**Table 1:** Summary of total match impact biomechanics for impacts to the head (n=20,687) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.5 ±14.4 | 14.4 [11.6-21.2] | 45.6 | 84.8 | 10.0 to 164.9 | 402,953.3 |
| **PRA (rad/s$^2$)** | 3,085.7 ±3,010.4 | 2,167.2 [1,106.7-3,980.8] | 9,390.8 | 14,771.5 | 0.4 to 23,397.1 | 63,824,769.6 |
| **$HIC_{15}$** | 15.4 ±81.7 | 3.4 [1.7-8.6] | 54.7 | 206.8 | 0.5 to 5,900.4 | 317,626.3 |
| **GSI** | 0.05 ±0.51 | 0.00 [0.00-0.01] | 0.15 | 0.89 | 0.00 to 40.30 | 1,054.12 |
| **HITsp** | 16.9 ±11.1 | 15.0 [13.1-17.8] | 29.5 | 56.5 | 5.8 to 356.8 | 349,510.3 |
| **GAMBIT** | 0.085 ±0.066 | 0.063 [0.042-0.105] | 0.220 | 0.322 | 0.010 to 1.828 | 1,744.20 |
| **ms** | 7.6 ±5.7 | 6.0 [4.0-10.0] | 19.0 | 30.0 | 2.0 to 40.0 | 157,388.0 |
| **$RWE_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.193 | 8.802 |
| **$RWE_{Rotational}$** | 0.129 ±0.306 | 0.000 [0.000-0.010] | 0.998 | 1.000 | 0.000 to 1.000 | 2,663.838 |
| **$RWE_{CP}$** | 0.048 ±0.175 | 0.000 [0.000-0.003] | 0.352 | 0.988 | 0.000 to 1.000 | 988.846 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of total match impact biomechanics for impacts to the front of the head (n=6,678) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.1 ±13.0 | 13.6 [11.4-19.2] | 40.6 | 78.1 | 10.0 to 163.2 | 120,768.8 |
| **PRA (rad/s$^2$)** | 2,904.3 ±2,794.1 | 2,046.3 [1,031.1-3,760.8] | 8,611.9 | 13,303.9 | 3.6 to 23,228.7 | 19,394,599.6 |
| **$HIC_{15}$** | 12.9 ±103.0 | 2.9 [1.6-6.9] | 40.4 | 138.1 | 0.7 to 5,900.4 | 86,340.2 |
| **GSI** | 0.04 ±0.68 | 0.00 [0.00-0.01] | 0.10 | 0.56 | 0.00 to 40.30 | 280.10 |
| **HITsp** | 18.2 ±11.2 | 15.6 [14.1-18.5] | 30.6 | 56.1 | 11.4 to 356.8 | 121,806.8 |
| **GAMBIT** | 0.083 ±0.069 | 0.058 [0.040-0.102] | 0.225 | 0.323 | 0.013 to 1.828 | 550.47 |
| **ms** | 7.1 ±5.1 | 5.0 [4.0-9.0] | 17.0 | 26.0 | 2.0 to 40.0 | 47,537.0 |
| **$RWE_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.180 | 2.443 |
| **$RWE_{Rotational}$** | 0.116 ±0.290 | 0.000 [0.000-0.007] | 0.991 | 1.000 | 0.000 to 1.000 | 776.663 |
| **$RWE_{CP}$** | 0.037 ±0.152 | 0.000 [0.000-0.002] | 0.188 | 0.961 | 0.000 to 1.000 | 250.283 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of total match impact biomechanics for impacts to the back of the head (n=5,932) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.6 ±15.8 | 15.9 [12.4-24.7] | 51.3 | 90.2 | 10.0 to 164.1 | 128,370.6 |
| **PRA (rad/s$^2$)** | 4,307.8 ±3,601.7 | 3,174.1 [1,782.9-5,723.5] | 11,876.0 | 16,792.8 | 2.9 to 23,397.1 | 25,554,115.7 |
| **$HIC_{15}$** | 19.2 ±64.9 | 4.2 [2.0-12.0] | 80.3 | 249.9 | 0.6 to 2,096.4 | 113,902.4 |
| **GSI** | 0.07 ±0.37 | 0.00 [0.00-0.02] | 0.26 | 1.30 | 0.00 to 14.32 | 399.25 |
| **HITsp** | 13.4 ±8.0 | 11.2 [9.7-14.1] | 24.8 | 44.3 | 7.3 to 168.6 | 79,385.8 |
| **GAMBIT** | 0.105 ±0.070 | 0.083 [0.055-0.135] | 0.245 | 0.340 | 0.010 to 0.588 | 608.37 |
| **ms** | 8.5 ±6.5 | 6.0 [4.0-11.0] | 22.0 | 35.7 | 2.0 to 40.0 | 50,494.0 |
| **$RWE_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.187 | 2.948 |
| **$RWE_{Rotational}$** | 0.224 ±0.381 | 0.002 [0.000-0.253] | 1.000 | 1.000 | 0.000 to 1.000 | 1,328.577 |
| **$RWE_{CP}$** | 0.090 ±0.238 | 0.001 [0.000-0.014] | 0.831 | 0.998 | 0.000 to 1.000 | 531.151 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of total match impact biomechanics for impacts to the side of the head (n=7,364) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.0 ±14.3 | 14.1 [11.4-20.6] | 44.2 | 84.7 | 10.0 to 164.9 | 139,881.3 |
| **PRA (rad/s$^2$)** | 2,292.9 ±2,260.1 | 1,731.1 [852.5-2,988.7] | 6,896.6 | 10,893.4 | 0.4 to 18,435.9 | 16,877,727.8 |
| **$HIC_{15}$** | 14.2 ±71.9 | 3.3 [1.6-8.0] | 45.4 | 213.1 | 0.5 to 3,181.5 | 104,879.3 |
| **GSI** | 0.04 ±0.44 | 0.00 [0.00-0.01] | 0.11 | 0.84 | 0.00 to 21.73 | 329.07 |
| **HITsp** | 19.2 ±12.4 | 16.1 [14.7-19.3] | 33.1 | 68.1 | 11.5 to 252.6 | 141,093.0 |
| **GAMBIT** | 0.072 ±0.055 | 0.055 [0.038-0.086] | 0.176 | 0.286 | 0.010 to 0.835 | 527.28 |
| **ms** | 7.3 ±5.4 | 6.0 [4.0-9.0] | 18.0 | 27.0 | 2.0 to 40.0 | 54,107.0 |
| **$RWE_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.193 | 3.039 |
| **$RWE_{Rotational}$** | 0.065 ±0.221 | 0.000 [0.000-0.001] | 0.779 | 1.000 | 0.000 to 1.000 | 477.280 |
| **$RWE_{CP}$** | 0.024 ±0.120 | 0.000 [0.000-0.001] | 0.061 | 0.841 | 0.000 to 1.000 | 173.616 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of total match impact biomechanics for impacts to the top of the head (n=713) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.5 ±15.5 | 13.9 [11.6-21.2] | 43.3 | 100.1 | 10.0 to 152.2 | 13,932.6 |
| **PRA (rad/s$^2$)** | 2,802.7 ±3,138.7 | 1,505.3 [853.9-3,799.8] | 9,293.7 | 15,910.4 | 2.5 to 22,989.2 | 1,998,326.5 |
| **$HIC_{15}$** | 17.5 ±74.2 | 2.9 [1.6-7.9] | 47.1 | 352.2 | 0.8 to 991.8 | 12,504.4 |
| **GSI** | 0.06 ±0.42 | 0.00 [0.00-0.01] | 0.10 | 1.94 | 0.00 to 6.77 | 45.70 |
| **HITsp** | 10.1 ±9.6 | 7.8 [7.0-9.7] | 17.6 | 46.7 | 5.8 to 178.6 | 7,179.8 |
| **GAMBIT** | 0.083 ±0.074 | 0.053 [0.034-0.106] | 0.245 | 0.350 | 0.011 to 0.476 | 58.08 |
| **ms** | 7.4 ±5.7 | 6.0 [4.0-8.0] | 19.0 | 34.4 | 2.0 to 40.0 | 5,250.0 |
| **$RWE_{Linear}$** | 0.001 ±0.005 | 0.000 [0.000-0.000] | 0.000 | 0.009 | 0.000 to 0.112 | 0.370 |
| **$RWE_{Rotational}$** | 0.114 ±0.290 | 0.000 [0.000-0.007] | 0.998 | 1.000 | 0.000 to 1.000 | 81.318 |
| **$RWE_{CP}$** | 0.047 ±0.181 | 0.000 [0.000-0.002] | 0.297 | 0.994 | 0.000 to 1.000 | 33.796 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

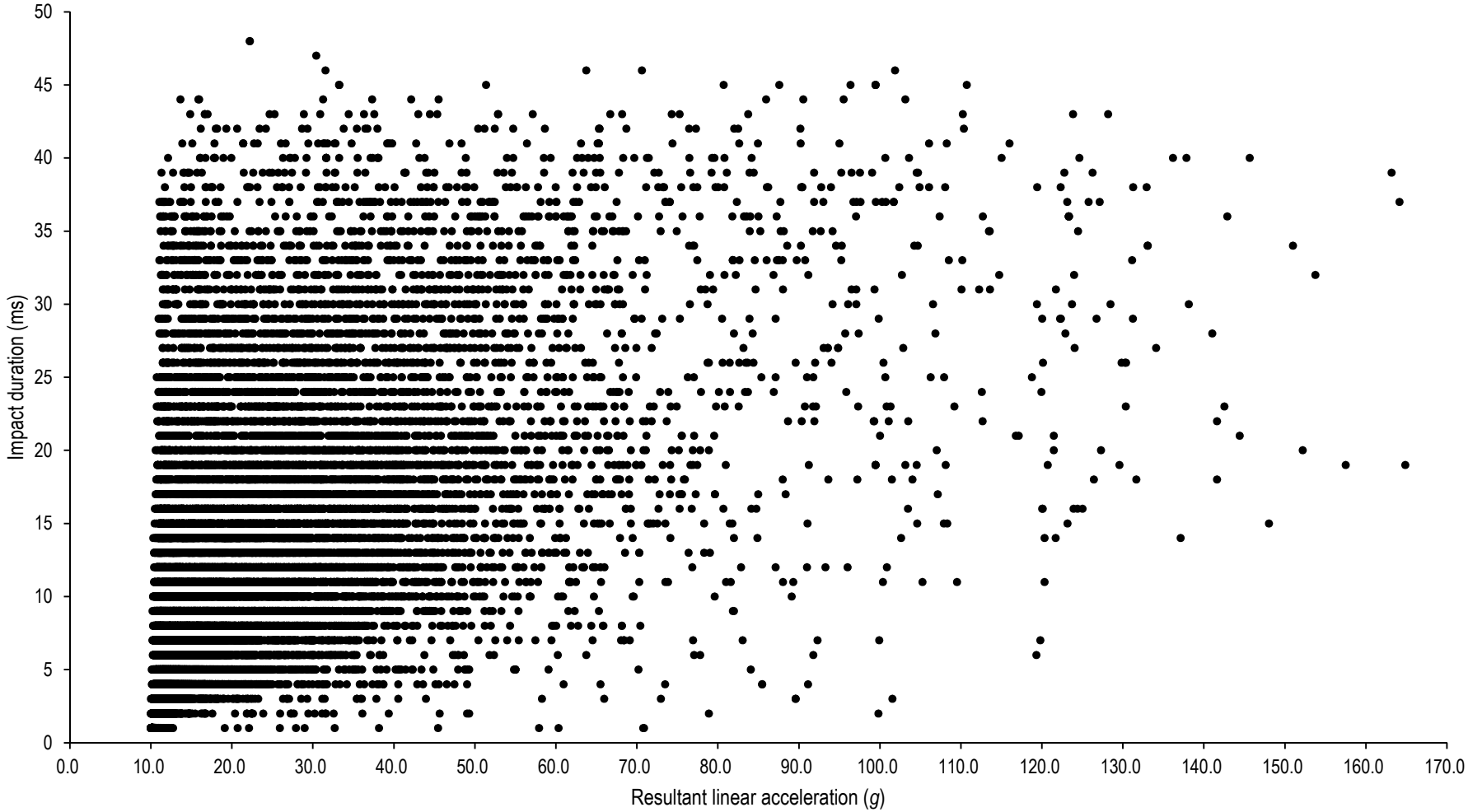


**Figure 1:** Resultant linear accelerations (*g*) by impact duration in milliseconds (ms) for all matches competed in a senior amateur rugby union team over a competitive season

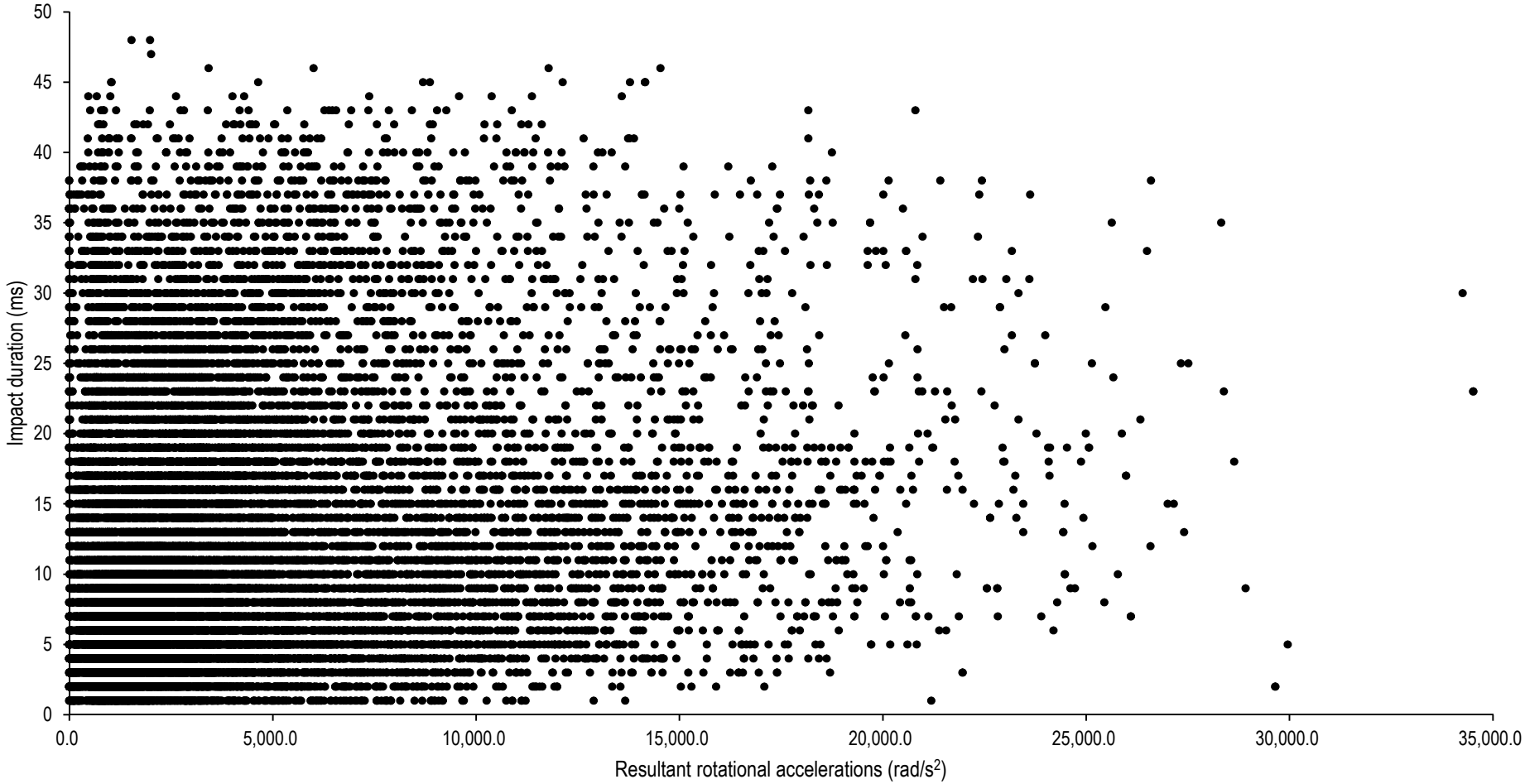


**Figure 2:** Resultant rotational accelerations (rad/s$^2$) by impact duration in milliseconds (ms) for all matches competed in a senior amateur rugby union team over a competitive season

# Individual Match Impact Biomechanics

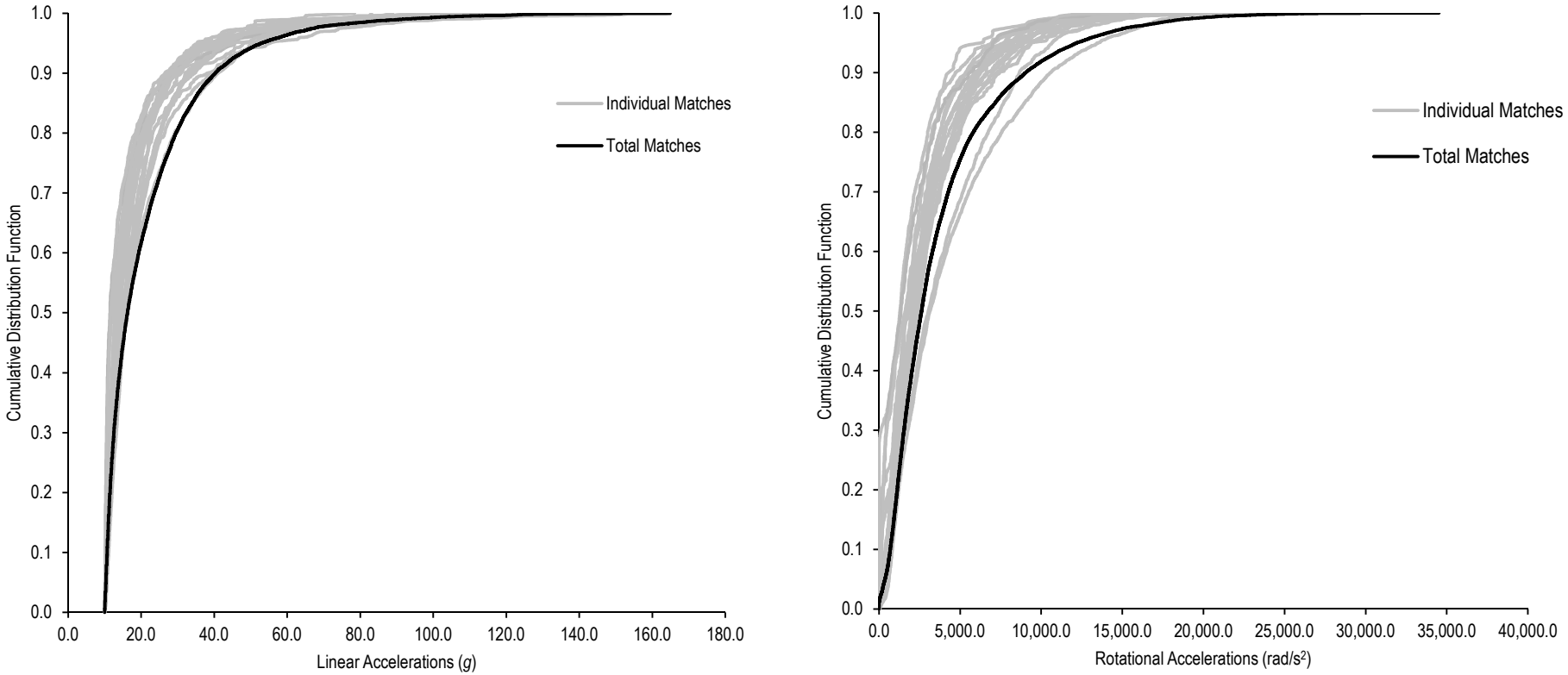


**Figure 3:** Cumulative distribution functions for resultant linear (left) and rotational (right) acceleration magnitudes of impacts collected during matches for senior amateur rugby union season 2013.

**Table 1:** Summary of preseason match 1 impact biomechanics for impacts to the head (n=204) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.7 ±11.4 | 15.0 [12.3-20.8] | 42.5 | 83.2 | 10.0 to 88.9 | 3,816.1 |
| **PRA (rad/s²)** | 2,236.2 ±2,003.9 | 1,709.2 [939.0-2,968.1] | 6,449.1 | 11,728.7 | 6.1 to 11,977.1 | 456,189.0 |
| **$HIC_{15}$** | 12.4 ±54.6 | 3.4 [1.6-7.6] | 32.9 | 289.4 | 0.7 to 706.1 | 2,537.9 |
| **GSI** | 0.05 ±0.35 | 0.00 [0.00-0.01] | 0.06 | 1.30 | 0.00 to 4.82 | 9.26 |
| **HITsp** | 18.1 ±13.9 | 15.9 [13.7-19.0] | 26.1 | 93.0 | 6.9 to 176.3 | 3,702.2 |
| **GAMBIT** | 0.062 ±0.050 | 0.051 [0.038-0.071] | 0.150 | 0.311 | 0.014 to 0.496 | 12.72 |
| **ms** | 6.7 ±5.2 | 5.0 [4.0-8.0] | 15.8 | 33.0 | 2.0 to 39.0 | 1,360.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | 0.004 | 0.000 to 0.005 | 0.042 |
| **$RWE_{Rotational}$** | 0.051 ±0.200 | 0.000 [0.000-0.001] | 0.591 | 1.000 | 0.000 to 1.000 | 10.323 |
| **$RWE_{CP}$** | 0.020 ±0.111 | 0.000 [0.000-0.001] | 0.027 | 0.835 | 0.000 to 0.879 | 4.095 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of competition match 1 impact biomechanics for impacts to the head (n=986) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.3 ±17.6 | 15.3 [11.9-22.8] | 55.7 | 100.2 | 10.0 to 155.5 | 21,035.2 |
| **PRA (rad/s²)** | 2,639.4 ±2,445.6 | 2,005.0 [1,032.5-3,314.4] | 7,714.7 | 12,835.6 | 3.9 to 19,878.4 | 2,602,479.4 |
| **$HIC_{15}$** | 23.3 ±88.6 | 4.0 [1.9-10.1] | 99.3 | 423.0 | 0.7 to 1,566.5 | 23,018.8 |
| **GSI** | 0.09 ±0.51 | 0.00 [0.00-0.01] | 0.36 | 1.89 | 0.00 to 10.70 | 87.23 |
| **HITsp** | 20.0 ±14.2 | 16.3 [14.8-19.3] | 40.4 | 91.0 | 13.0 to 228.8 | 19,720.0 |
| **GAMBIT** | 0.081 ±0.062 | 0.060 [0.041-0.099] | 0.202 | 0.287 | 0.015 to 0.503 | 78.27 |
| **ms** | 8.4 ±6.6 | 6.0 [4.0-11.0] | 22.0 | 39.0 | 2.0 to 40.0 | 8,289.0 |
| **$RWE_{Linear}$** | 0.000 ±0.006 | 0.000 [0.000-0.000] | 0.001 | 0.009 | 0.000 to 0.130 | 0.717 |
| **$RWE_{Rotational}$** | 0.081 ±0.248 | 0.000 [0.000-0.003] | 0.948 | 1.000 | 0.000 to 1.000 | 79.747 |
| **$RWE_{CP}$** | 0.034 ±0.150 | 0.000 [0.000-0.001] | 0.114 | 0.955 | 0.000 to 1.000 | 33.627 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of competition match 2 impact biomechanics for impacts to the head (n=2,639) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 22.1 ±16.8 | 15.6 [12.0-26.0] | 52.7 | 97.9 | 10.0 to 164.9 | 58,301.1 |
| **PRA (rad/s²)** | 4,576.5 ±4,138.1 | 3,141.9 [1,455.9-6,456.9] | 13,575.2 | 17,758.8 | 3.0 to 23,397.1 | 12,063,524.5 |
| **$HIC_{15}$** | 25.1 ±134.2 | 4.4 [2.0-14.7] | 98.8 | 389.7 | 0.6 to 5,900.4 | 66,192.7 |
| **GSI** | 0.10 ±0.87 | 0.00 [0.00-0.02] | 0.39 | 2.01 | 0.00 to 40.30 | 274.06 |
| **HITsp** | 15.8 ±11.1 | 14.3 [12.0-17.1] | 26.6 | 46.6 | 6.4 to 457.2 | 41,621.0 |
| **GAMBIT** | 0.107 ±0.082 | 0.083 [0.049-0.146] | 0.262 | 0.328 | 0.014 to 1.828 | 269.34 |
| **ms** | 9.5 ±7.3 | 7.0 [4.0-12.0] | 25.0 | 38.0 | 2.0 to 40.0 | 24,946.0 |
| **$RWE_{Linear}$** | 0.001 ±0.006 | 0.000 [0.000-0.000] | 0.000 | 0.008 | 0.000 to 0.193 | 1.487 |
| **$RWE_{Rotational}$** | 0.264 ±0.410 | 0.002 [0.000-0.592] | 1.000 | 1.000 | 0.000 to 1.000 | 697.870 |
| **$RWE_{CP}$** | 0.120 ±0.277 | 0.001 [0.000-0.026] | 0.959 | 0.999 | 0.000 to 1.000 | 316.759 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of competition match 3 impact biomechanics for impacts to the head (n=2,595) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.3 ±13.6 | 16.0 [12.3-22.5] | 45.3 | 80.8 | 10.0 to 157.9 | 52,700.9 |
| **PRA (rad/s²)** | 2,714.5 ±2,195.2 | 2,140.9 [1,268.2-3,355.6] | 7,087.1 | 11,160.0 | 3.5 to 17,664.3 | 7,044,164.2 |
| **$HIC_{15}$** | 14.6 ±39.9 | 4.6 [2.0-12.6] | 51.5 | 167.1 | 0.7 to 781.6 | 37,905.5 |
| **GSI** | 0.04 ±0.21 | 0.00 [0.00-0.02] | 0.15 | 0.72 | 0.00 to 5.34 | 108.85 |
| **HITsp** | 18.1 ±12.8 | 15.3 [13.0-19.3] | 34.4 | 67.9 | 5.7 to 258.6 | 46,952.6 |
| **GAMBIT** | 0.080 ±0.049 | 0.067 [0.047-0.098] | 0.176 | 0.262 | 0.016 to 0.595 | 206.18 |
| **ms** | 8.9 ±5.9 | 7.0 [4.0-12.0] | 20.0 | 28.0 | 2.0 to 40.0 | 23,001.0 |
| **$RWE_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.144 | 0.885 |
| **$RWE_{Rotational}$** | 0.074 ±0.230 | 0.000 [0.000-0.003] | 0.838 | 1.000 | 0.000 to 1.000 | 191.102 |
| **$RWE_{CP}$** | 0.023 ±0.117 | 0.001 [0.000-0.002] | 0.067 | 0.882 | 0.000 to 0.999 | 60.166 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of competition match 4 impact biomechanics for impacts to the head (n=2,949) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.4 ±13.5 | 15.9 [12.5-23.5] | 43.9 | 80.5 | 10.0 to 152.3 | 60,062.3 |
| **PRA (rad/s²)** | 2,852.6 ±2,717.4 | 2,014.5 [1,170.7-3,525.4] | 8,297.1 | 13,591.6 | 0.4 to 21,979.5 | 8,412,447.1 |
| **$HIC_{15}$** | 17.0 ±129.8 | 3.7 [1.8-10.0] | 45.5 | 183.6 | 0.7 to 3,586.1 | 50,204.5 |
| **GSI** | 0.06 ±0.87 | 0.00 [0.00-0.01] | 0.12 | 0.64 | 0.00 to 24.49 | 188.01 |
| **HITsp** | 16.6 ±12.2 | 15.2 [12.3-17.8] | 27.4 | 43.9 | 6.6 to 301.1 | 48,873.6 |
| **GAMBIT** | 0.083 ±0.061 | 0.066 [0.044-0.100] | 0.197 | 0.321 | 0.010 to 0.835 | 241.13 |
| **ms** | 7.6 ±5.4 | 6.0 [4.0-10.0] | 19.0 | 26.5 | 2.0 to 40.0 | 22,352.0 |
| **$RWE_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.112 | 1.048 |
| **$RWE_{Rotational}$** | 0.097 ±0.266 | 0.000 [0.000-0.004] | 0.983 | 1.000 | 0.000 to 1.000 | 284.803 |
| **$RWE_{CP}$** | 0.038 ±0.159 | 0.000 [0.000-0.002] | 0.158 | 0.977 | 0.000 to 1.000 | 110.965 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 6:** Summary of competition match 5 impact biomechanics for impacts to the head (n=1,008) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.0 ±16.7 | 15.0 [11.7-23.0] | 52.1 | 110.7 | 10.0 to 142.5 | 21,162.4 |
| **PRA (rad/s²)** | 2,952.4 ±2,670.7 | 2,210.4 [1,118.2-3,701.5] | 8,984.2 | 11,783.0 | 4.4 to 17,604.3 | 2,976,002.0 |
| **$HIC_{15}$** | 17.0 ±50.9 | 3.6 [1.7-9.7] | 72.1 | 241.5 | 0.8 to 640.3 | 17,173.3 |
| **GSI** | 0.06 ±0.25 | 0.00 [0.00-0.01] | 0.28 | 0.97 | 0.00 to 3.56 | 55.79 |
| **HITsp** | 20.0 ±14.3 | 15.8 [14.1-19.5] | 40.1 | 86.9 | 12.3 to 178.5 | 20,153.5 |
| **GAMBIT** | 0.078 ±0.053 | 0.061 [0.041-0.097] | 0.190 | 0.267 | 0.015 to 0.336 | 77.53 |
| **ms** | 7.6 ±6.4 | 6.0 [4.0-9.0] | 21.6 | 34.0 | 2.0 to 40.0 | 7,688.0 |
| **$RWE_{Linear}$** | 0.001 ±0.004 | 0.000 [0.000-0.001] | 0.001 | 0.015 | 0.000 to 0.071 | 0.597 |
| **$RWE_{Rotational}$** | 0.117 ±0.295 | 0.000 [0.000-0.006] | 0.996 | 1.000 | 0.000 to 1.000 | 117.716 |
| **$RWE_{CP}$** | 0.043 ±0.159 | 0.000 [0.000-0.002] | 0.335 | 0.941 | 0.000 to 1.000 | 43.803 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 7:** Summary of competition match 6 impact biomechanics for impacts to the head (n=1,224) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.0 ±16.3 | 12.0 [10.7-17.9] | 43.8 | 102.6 | 10.0 to 164.1 | 22,019.1 |
| **PRA (rad/s$^2$)** | 1,923.6 ±2,512.8 | 1,297.1 [5.4-2,643.0] | 6,953.5 | 11,668.0 | 2.1 to 20,543.0 | 2,354,456.9 |
| **HIC** | 16.8 ±82.8 | 2.8 [1.5-6.3] | 59.0 | 326.3 | 0.6 to 2,096.4 | 20,568.9 |
| **GSI** | 0.06 ±0.48 | 0.00 [0.00-0.01] | 0.22 | 1.02 | 0.00 to 14.32 | 70.67 |
| **HITsp** | 17.7 ±12.0 | 15.1 [14.8-17.3] | 28.7 | 79.4 | 7.2 to 204.0 | 21,656.7 |
| **GAMBIT** | 0.062 ±0.043 | 0.043 [0.037-0.070] | 0.160 | 0.220 | 0.010 to 0.406 | 74.62 |
| **ms** | 6.9 ±5.7 | 5.0 [3.0-8.0] | 19.0 | 27.0 | 2.0 to 40.0 | 8,465.0 |
| **RWE$_{Linear}$** | 0.001 ±0.007 | 0.000 [0.000-0.000] | 0.001 | 0.010 | 0.000 to 0.187 | 0.850 |
| **RWE$_{Rotational}$** | 0.063 ±0.220 | 0.000 [0.000-0.001] | 0.798 | 1.000 | 0.000 to 1.000 | 77.147 |
| **RWE$_{CP}$** | 0.027 ±0.134 | 0.000 [0.000-0.001] | 0.071 | 0.927 | 0.000 to 1.000 | 33.086 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 8:** Summary of competition match 7 impact biomechanics for impacts to the head (n=1,065) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.6 ±13.0 | 15.1 [12.0-21.3] | 44.4 | 83.2 | 10.0 to 94.6 | 20,865.1 |
| **PRA (rad/s$^2$)** | 2,928.5 ±2,860.5 | 2,133.4 [1,184.9-3,616.7] | 8,956.4 | 14,621.6 | 4.2 to 21,528.1 | 3,118,886.9 |
| **HIC** | 11.3 ±29.5 | 3.6 [1.8-7.9] | 46.6 | 168.2 | 0.6 to 463.2 | 11,982.0 |
| **GSI** | 0.03 ±0.15 | 0.00 [0.00-0.01] | 0.09 | 0.39 | 0.00 to 3.16 | 27.96 |
| **HITsp** | 17.3 ±12.0 | 14.5 [12.8-18.1] | 34.3 | 66.2 | 7.6 to 218.6 | 18,432.3 |
| **GAMBIT** | 0.072 ±0.055 | 0.055 [0.040-0.084] | 0.190 | 0.317 | 0.011 to 0.426 | 76.59 |
| **ms** | 6.7 ±4.6 | 5.0 [4.0-8.0] | 15.7 | 26.0 | 2.0 to 39.0 | 7,123.0 |
| **RWE$_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.007 | 0.256 |
| **RWE$_{Rotational}$** | 0.099 ±0.274 | 0.000 [0.000-0.005] | 0.995 | 1.000 | 0.000 to 1.000 | 105.961 |
| **RWE$_{CP}$** | 0.043 ±0.168 | 0.000 [0.000-0.002] | 0.305 | 0.975 | 0.000 to 1.000 | 45.417 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 9:** Summary of competition match 8 impact biomechanics for impacts to the head (n=812) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.0 ±11.6 | 14.3 [11.5-19.5] | 37.2 | 73.1 | 10.0 to 102.1 | 14,581.7 |
| **PRA (rad/s$^2$)** | 2,804.9 ±2,562.8 | 2,206.8 [865.3-4,143.2] | 7,811.1 | 12,052.7 | 3.5 to 16,782.6 | 2,277,555.3 |
| **HIC** | 9.0 ±24.9 | 3.2 [1.6-6.8] | 30.2 | 131.2 | 0.5 to 330.5 | 7,274.8 |
| **GSI** | 0.02 ±0.12 | 0.00 [0.00-0.01] | 0.05 | 0.30 | 0.00 to 2.26 | 15.77 |
| **HITsp** | 16.9 ±11.9 | 14.3 [12.6-17.6] | 29.8 | 71.3 | 6.5 to 184.4 | 13,716.6 |
| **GAMBIT** | 0.080 ±0.053 | 0.062 [0.043-0.102] | 0.199 | 0.270 | 0.013 to 0.371 | 65.10 |
| **ms** | 6.3 ±4.2 | 5.0 [3.0-8.0] | 14.0 | 21.7 | 2.0 to 36.0 | 5,083.0 |
| **RWE$_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.000 | 0.002 | 0.000 to 0.010 | 0.175 |
| **RWE$_{Rotational}$** | 0.104 ±0.262 | 0.000 [0.000-0.014] | 0.957 | 1.000 | 0.000 to 1.000 | 84.077 |
| **RWE$_{CP}$** | 0.024 ±0.112 | 0.000 [0.000-0.003] | 0.090 | 0.834 | 0.000 to 0.998 | 19.338 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 10:** Summary of competition match 9 impact biomechanics for impacts to the head (n=355) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.3 ±11.3 | 13.1 [11.2-18.5] | 40.2 | 81.1 | 10.0 to 83.1 | 6,142.2 |
| **PRA (rad/s$^2$)** | 2,469.1 ±2,437.9 | 1,658.0 [871.6-3,408.8] | 7,972.6 | 12,200.4 | 6.6 to 15,835.5 | 876,514.7 |
| **HIC** | 9.1 ±30.5 | 2.2 [1.3-6.0] | 26.6 | 185.5 | 0.8 to 331.5 | 3,223.7 |
| **GSI** | 0.02 ±0.08 | 0.00 [0.00-0.01] | 0.05 | 0.51 | 0.00 to 1.02 | 5.82 |
| **HITsp** | 19.8 ±13.9 | 16.0 [14.3-20.4] | 36.1 | 105.0 | 9.1 to 144.9 | 7,024.6 |
| **GAMBIT** | 0.069 ±0.050 | 0.052 [0.037-0.087] | 0.172 | 0.278 | 0.012 to 0.354 | 24.67 |
| **ms** | 5.8 ±3.8 | 4.0 [3.0-7.0] | 14.0 | 20.0 | 2.0 to 28.0 | 2,076.0 |
| **RWE$_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.004 | 0.067 |
| **RWE$_{Rotational}$** | 0.084 ±0.254 | 0.000 [0.000-0.003] | 0.967 | 1.000 | 0.000 to 1.000 | 29.781 |
| **RWE$_{CP}$** | 0.027 ±0.120 | 0.000 [0.000-0.001] | 0.170 | 0.761 | 0.000 to 0.990 | 9.451 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 11:** Summary of competition match 10 impact biomechanics for impacts to the head (n=524) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.3 ±11.1 | 12.8 [11.2-17.8] | 42.1 | 63.4 | 10.0 to 78.4 | 9,088.1 |
| **PRA (rad/s$^2$)** | 2,557.9 ±2,815.9 | 1,860.7 [744.5-3,318.5] | 8,305.7 | 12,916.8 | 5.1 to 18,298.1 | 1,340,333.3 |
| **HIC** | 8.5 ±20.8 | 2.6 [1.3-6.0] | 34.3 | 118.0 | 0.7 to 193.7 | 4,464.2 |
| **GSI** | 0.01 ±0.06 | 0.00 [0.00-0.01] | 0.05 | 0.29 | 0.00 to 0.57 | 7.77 |
| **HITsp** | 17.6 ±11.9 | 14.1 [12.7-17.6] | 37.8 | 74.2 | 6.4 to 134.6 | 9,197.1 |
| **GAMBIT** | 0.071 ±0.062 | 0.047 [0.033-0.083] | 0.212 | 0.313 | 0.015 to 0.383 | 37.08 |
| **ms** | 6.1 ±3.9 | 5.0 [3.0-8.0] | 14.0 | 21.0 | 2.0 to 29.0 | 3,181.0 |
| **RWE$_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | 0.001 | 0.000 to 0.003 | 0.092 |
| **RWE$_{Rotational}$** | 0.104 ±0.276 | 0.000 [0.000-0.003] | 0.983 | 1.000 | 0.000 to 1.000 | 54.333 |
| **RWE$_{CP}$** | 0.032 ±0.138 | 0.000 [0.000-0.002] | 0.139 | 0.906 | 0.000 to 0.999 | 16.738 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 12:** Summary of competition match 11 impact biomechanics for impacts to the head (n=464) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 16.8 ±11.5 | 13.0 [11.3-16.9] | 35.6 | 76.4 | 10.1 to 130.3 | 7,808.7 |
| **PRA (rad/s$^2$)** | 2,903.3 ±2,579.7 | 2,026.6 [1,214.1-3,892.7] | 8,161.6 | 12,033.4 | 5.9 to 16,351.9 | 1,347,145.8 |
| **HIC** | 8.7 ±30.0 | 2.4 [1.4-4.5] | 30.1 | 132.5 | 0.7 to 421.9 | 4,025.1 |
| **GSI** | 0.02 ±0.08 | 0.00 [0.00-0.00] | 0.04 | 0.30 | 0.00 to 0.94 | 7.04 |
| **HITsp** | 17.2 ±12.7 | 14.6 [13.3-17.3] | 31.8 | 56.1 | 6.8 to 165.8 | 7,995.1 |
| **GAMBIT** | 0.081 ±0.062 | 0.057 [0.040-0.098] | 0.223 | 0.310 | 0.012 to 0.434 | 37.53 |
| **ms** | 5.7 ±3.4 | 5.0 [3.0-7.0] | 12.0 | 20.4 | 2.0 to 30.0 | 2,649.0 |
| **RWE$_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.040 | 0.126 |
| **RWE$_{Rotational}$** | 0.118 ±0.285 | 0.000 [0.000-0.009] | 0.978 | 1.000 | 0.000 to 1.000 | 54.710 |
| **RWE$_{CP}$** | 0.028 ±0.119 | 0.000 [0.000-0.002] | 0.105 | 0.766 | 0.000 to 0.992 | 12.870 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 13:** Summary of competition match 12 impact biomechanics for impacts to the head (n=1,161) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.4 ±13.1 | 14.1 [11.6-20.0] | 40.3 | 69.6 | 10.0 to 141.6 | 21,348.4 |
| **PRA (rad/s$^2$)** | 3,274.0 ±2,822.4 | 2,385.7 [1,340.0-4,303.5] | 9,003.9 | 13,766.0 | 12.9 to 23,228.7 | 3,801,167.7 |
| **HIC** | 11.7 ±44.3 | 3.2 [1.7-7.8] | 39.4 | 152.4 | 0.6 to 886.7 | 13,641.6 |
| **GSI** | 0.03 ±0.12 | 0.00 [0.00-0.01] | 0.10 | 0.77 | 0.00 to 1.79 | 31.91 |
| **HITsp** | 16.1 ±13.2 | 14.0 [10.6-17.0] | 30.4 | 62.9 | 6.2 to 214.5 | 18,714.9 |
| **GAMBIT** | 0.090 ±0.065 | 0.070 [0.047-0.115] | 0.224 | 0.328 | 0.017 to 0.577 | 104.93 |
| **ms** | 7.7 ±5.1 | 6.0 [4.0-10.0] | 17.0 | 26.0 | 2.0 to 40.0 | 8,975.0 |
| **RWE$_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.000 | 0.002 | 0.000 to 0.068 | 0.416 |
| **RWE$_{Rotational}$** | 0.136 ±0.308 | 0.000 [0.000-0.019] | 0.996 | 1.000 | 0.000 to 1.000 | 157.569 |
| **RWE$_{CP}$** | 0.040 ±0.154 | 0.001 [0.000-0.003] | 0.202 | 0.974 | 0.000 to 1.000 | 46.570 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 14:** Summary of competition match 13 impact biomechanics for impacts to the head (n=1,393) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.2 ±13.9 | 13.5 [11.4-18.5] | 41.3 | 85.7 | 10.0 to 156.4 | 25,313.6 |
| **PRA (rad/s$^2$)** | 4,018.1 ±3,435.6 | 3,044.4 [1,333.7-5,901.4] | 10,701.7 | 15,179.4 | 5.8 to 21,619.0 | 5,597,166.5 |
| **HIC** | 12.5 ±59.2 | 3.0 [1.6-6.7] | 39.1 | 168.8 | 0.7 to 1,313.8 | 17,372.5 |
| **GSI** | 0.04 ±0.38 | 0.00 [0.00-0.01] | 0.11 | 0.72 | 0.00 to 8.97 | 60.07 |
| **HITsp** | 16.5 ±11.7 | 14.7 [12.2-17.5] | 29.1 | 58.2 | 6.6 to 268.2 | 22,925.3 |
| **GAMBIT** | 0.121 ±0.096 | 0.087 [0.046-0.173] | 0.313 | 0.444 | 0.015 to 0.604 | 168.68 |
| **ms** | 7.6 ±5.3 | 6.0 [4.0-9.5] | 18.0 | 28.1 | 2.0 to 40.0 | 10,566.0 |
| **RWE$_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.000 | 0.004 | 0.000 to 0.134 | 0.655 |
| **RWE$_{Rotational}$** | 0.232 ±0.384 | 0.002 [0.000-0.325] | 1.000 | 1.000 | 0.000 to 1.000 | 322.812 |
| **RWE$_{CP}$** | 0.071 ±0.201 | 0.001 [0.000-0.014] | 0.585 | 0.992 | 0.000 to 1.000 | 98.798 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 15:** Summary of competition match 14 impact biomechanics for impacts to the head (n=284) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.2 ±14.6 | 11.5 [10.4-17.2] | 45.7 | 98.7 | 10.0 to 103.0 | 4,882.7 |
| **PRA (rad/s$^2$)** | 2,145.1 ±2,501.5 | 1,357.7 [373.3-2,940.3] | 7,137.9 | 11,718.4 | 4.8 to 16,335.5 | 609,203.7 |
| **HIC** | 10.7 ±34.4 | 2.4 [1.5-5.2] | 42.9 | 223.8 | 0.7 to 361.6 | 3,035.8 |
| **GSI** | 0.02 ±0.09 | 0.00 [0.00-0.01] | 0.09 | 0.65 | 0.00 to 0.81 | 5.85 |
| **HITsp** | 17.0 ±13.1 | 13.9 [13.0-16.7] | 33.9 | 92.3 | 7.3 to 128.6 | 4,834.5 |
| **GAMBIT** | 0.072 ±0.056 | 0.048 [0.040-0.080] | 0.184 | 0.328 | 0.013 to 0.361 | 20.32 |
| **ms** | 6.1 ±4.0 | 5.0 [3.0-8.0] | 14.0 | 19.3 | 2.0 to 28.0 | 1,724.0 |
| **RWE$_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.008 | 0.000 to 0.010 | 0.080 |
| **RWE$_{Rotational}$** | 0.082 ±0.243 | 0.000 [0.000-0.001] | 0.851 | 1.000 | 0.000 to 1.000 | 23.315 |
| **RWE$_{CP}$** | 0.027 ±0.133 | 0.000 [0.000-0.001] | 0.079 | 0.957 | 0.000 to 0.997 | 7.780 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 16:** Summary of competition match 15 impact biomechanics for impacts to the head (n=691) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.0 ±14.5 | 13.5 [11.3-19.3] | 49.2 | 98.3 | 10.0 to 110.2 | 13,100.4 |
| **PRA (rad/s²)** | 2,984.7 ±2,992.0 | 2,066.7 [986.4-4,073.9] | 9,203.3 | 13,046.0 | 3.0 to 22,989.2 | 2,062,397.1 |
| **HIC** | 14.7 ±56.5 | 3.1 [1.6-7.8] | 45.1 | 340.2 | 0.5 to 961.5 | 10,143.8 |
| **GSI** | 0.04 ±0.28 | 0.00 [0.00-0.01] | 0.09 | 0.83 | 0.00 to 5.25 | 28.61 |
| **HITsp** | 16.4 ±11.1 | 14.1 [12.0-16.5] | 31.7 | 76.2 | 6.7 to 125.2 | 11,311.6 |
| **GAMBIT** | 0.086 ±0.067 | 0.060 [0.040-0.108] | 0.230 | 0.347 | 0.014 to 0.468 | 59.65 |
| **ms** | 7.0 ±5.1 | 6.0 [4.0-9.0] | 17.0 | 29.0 | 2.0 to 40.0 | 4,864.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.008 | 0.000 to 0.015 | 0.205 |
| **$RWE_{Rotational}$** | 0.126 ±0.307 | 0.000 [0.000-0.012] | 0.997 | 1.000 | 0.000 to 1.000 | 87.130 |
| **$RWE_{CP}$** | 0.047 ±0.167 | 0.000 [0.000-0.003] | 0.376 | 0.942 | 0.000 to 1.000 | 32.465 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 17:** Summary of competition match 16 impact biomechanics for impacts to the head (n=449) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.0 ±15.7 | 13.8 [11.7-18.6] | 48.6 | 101.6 | 10.0 to 157.5 | 8,540.1 |
| **PRA (rad/s²)** | 3,315.5 ±3,076.9 | 2,407.9 [1,182.2-4,270.2] | 9,902.7 | 15,163.8 | 20.3 to 17,445.9 | 1,488,659.0 |
| **HIC** | 13.5 ±65.4 | 3.1 [1.6-6.3] | 44.7 | 191.1 | 0.6 to 1,254.2 | 6,074.1 |
| **GSI** | 0.04 ±0.23 | 0.00 [0.00-0.01] | 0.11 | 1.20 | 0.00 to 3.82 | 16.28 |
| **HITsp** | 16.3 ±13.8 | 14.4 [10.6-16.9] | 29.0 | 74.9 | 6.8 to 222.0 | 7,327.5 |
| **GAMBIT** | 0.089 ±0.068 | 0.067 [0.044-0.114] | 0.221 | 0.394 | 0.015 to 0.455 | 39.92 |
| **ms** | 6.6 ±4.8 | 5.0 [3.0-8.0] | 15.0 | 29.5 | 2.0 to 38.0 | 2,978.0 |
| **$RWE_{Linear}$** | 0.001 ±0.007 | 0.000 [0.000-0.000] | 0.001 | 0.010 | 0.000 to 0.141 | 0.275 |
| **$RWE_{Rotational}$** | 0.144 ±0.324 | 0.000 [0.000-0.018] | 0.999 | 1.000 | 0.000 to 1.000 | 64.852 |
| **$RWE_{CP}$** | 0.057 ±0.193 | 0.001 [0.000-0.003] | 0.555 | 0.988 | 0.000 to 0.999 | 25.566 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 18:** Summary of competition match 17 impact biomechanics for impacts to the head (n=527) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.3 ±13.1 | 12.8 [11.4-17.3] | 40.6 | 79.2 | 10.0 to 130.2 | 9,141.2 |
| **PRA (rad/s²)** | 2,802.9 ±2,432.7 | 2,149.2 [1,128.4-3,561.5] | 8,816.7 | 10,963.9 | 2.8 to 15,303.3 | 1,477,105.9 |
| **HIC** | 10.1 ±35.0 | 2.3 [1.4-5.3] | 35.4 | 158.3 | 0.7 to 461.8 | 5,327.9 |
| **GSI** | 0.03 ±0.18 | 0.00 [0.00-0.01] | 0.07 | 0.85 | 0.00 to 2.62 | 17.19 |
| **HITsp** | 16.7 ±12.1 | 14.8 [13.1-17.1] | 28.5 | 74.7 | 6.4 to 179.0 | 8,823.8 |
| **GAMBIT** | 0.080 ±0.067 | 0.057 [0.039-0.096] | 0.209 | 0.377 | 0.011 to 0.453 | 42.27 |
| **ms** | 6.7 ±5.2 | 5.0 [3.0-8.0] | 16.0 | 31.6 | 2.0 to 37.0 | 3,537.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.040 | 0.168 |
| **$RWE_{Rotational}$** | 0.098 ±0.270 | 0.000 [0.000-0.004] | 0.994 | 1.000 | 0.000 to 1.000 | 51.875 |
| **$RWE_{CP}$** | 0.031 ±0.130 | 0.000 [0.000-0.002] | 0.186 | 0.840 | 0.000 to 0.999 | 16.273 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 19:** Summary of competition match 18 impact biomechanics for impacts to the head (n=798) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 16.6 ±13.0 | 11.7 [10.5-16.3] | 43.2 | 78.3 | 10.0 to 151.0 | 13,208.9 |
| **PRA (rad/s$^2$)** | 2,770.0 ±3,130.0 | 1,621.5 [331.9-4,032.6] | 9,409.3 | 13,434.4 | 15.4 to 16,737.8 | 2,210,489.0 |
| **HIC** | 9.1 ±28.1 | 2.5 [1.4-4.9] | 39.3 | 135.8 | 0.6 to 455.6 | 7,233.0 |
| **GSI** | 0.02 ±0.13 | 0.00 [0.00-0.00] | 0.07 | 0.53 | 0.00 to 2.86 | 16.77 |
| **HITsp** | 17.3 ±12.5 | 14.3 [13.5-16.9] | 30.9 | 67.6 | 7.0 to 227.8 | 13,833.5 |
| **GAMBIT** | 0.081 ±0.066 | 0.049 [0.040-0.101] | 0.226 | 0.324 | 0.018 to 0.668 | 64.48 |
| **ms** | 6.2 ±4.1 | 5.0 [3.0-8.0] | 14.0 | 24.0 | 2.0 to 34.0 | 4,961.0 |
| **RWE$_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.106 | 0.272 |
| **RWE$_{Rotational}$** | 0.136 ±0.316 | 0.000 [0.000-0.011] | 0.998 | 1.000 | 0.000 to 1.000 | 108.409 |
| **RWE$_{CP}$** | 0.044 ±0.163 | 0.000 [0.000-0.003] | 0.291 | 0.983 | 0.000 to1.000 | 35.184 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 20:** Summary of Semi-Final match 1 impact biomechanics for impacts to the head (n=560) greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.8 ±14.3 | 13.2 [11.5-17.5] | 39.6 | 74.5 | 10.0 to 153.8 | 9,942.9 |
| **PRA (rad/s$^2$)** | 3,068.9 ±2,666.1 | 2,289.6 [1,285.5-3,941.5] | 8,085.5 | 14,039.3 | 4.0 to 17,409.1 | 1,718,601.2 |
| **HIC** | 11.7 ±53.1 | 2.3 [1.4-5.8] | 36.4 | 171.1 | 0.6 to 843.0 | 6,537.8 |
| **GSI** | 0.04 ±0.30 | 0.00 [0.00-0.01] | 0.06 | 0.78 | 0.00 to 5.76 | 20.45 |
| **HITsp** | 16.7 ±13.1 | 15.0 [12.9-17.1] | 28.0 | 62.2 | 6.8 to 232.6 | 9,346.6 |
| **GAMBIT** | 0.078 ±0.057 | 0.058 [0.040-0.096] | 0.197 | 0.294 | 0.017 to 0.420 | 43.80 |
| **ms** | 6.4 ±4.4 | 5.0 [3.0-8.0] | 14.0 | 25.2 | 2.0 to 38.0 | 3,570.0 |
| **RWE$_{Linear}$** | 0.001 ±0.008 | 0.000 [0.000-0.000] | 0.000 | 0.002 | 0.000 to 0.120 | 0.402 |
| **RWE$_{Rotational}$** | 0.110 ±0.277 | 0.000 [0.000-0.009] | 0.974 | 1.000 | 0.000 to 1.000 | 61.518 |
| **RWE$_{CP}$** | 0.038 ±0.160 | 0.000 [0.000-0.002] | 0.128 | 0.971 | 0.000 to1.000 | 21.088 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

# Positional Group Match Impact Biomechanics

**Table 1:** Summary of competition matches impact biomechanics for impacts to the head (n=13,533) greater of forwards than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.6 ±14.4 | 14.4 [11.6-21.7] | 45.7 | 84.2 | 10.0 to 164.9 | 265,078.6 |
| **PRA (rad/s$^2$)** | 3,234.5 ±3,169.4 | 2,258.9 [1,114.3-4,151.0] | 9,905.0 | 15,474.8 | 2.3 to 23,397.1 | 43,772,515.5 |
| **$HIC_{15}$** | 15.7 ±78.6 | 3.5 [1.7-9.0] | 56.1 | 198.2 | 0.5 to 3,586.1 | 212,429.6 |
| **GSI** | 0.05 ±0.49 | 0.00 [0.00-0.01] | 0.16 | 0.93 | 0.00 to 24.49 | 719.24 |
| **HITsp** | 16.9 ±10.9 | 15.0 [13.1-17.8] | 29.3 | 54.6 | 5.9 to 273.3 | 228,267.8 |
| **GAMBIT** | 0.089 ±0.069 | 0.065 [0.042-0.110] | 0.233 | 0.331 | 0.010 to 0.835 | 1,184.84 |
| **ms** | 7.8 ±5.9 | 6.0 [4.0-10.0] | 19.0 | 32.0 | 2.0 to 40.0 | 105,833.0 |
| **$RWE_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.193 | 5.764 |
| **$RWE_{Rotational}$** | 0.142 ±0.320 | 0.000 [0.000-0.014] | 0.999 | 1.000 | 0.000 to 1.000 | 1,927.602 |
| **$RWE_{CP}$** | 0.054 ±0.188 | 0.001 [0.000-0.003] | 0.455 | 0.993 | 0.000 to 1.000 | 735.488 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of competition matches impact biomechanics for impacts to the front of the head (n=4,642) of forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.3 ±13.0 | 13.7 [11.4-19.6] | 40.5 | 79.4 | 10.0 to 157.5 | 90,317.7 |
| **PRA (rad/s$^2$)** | 2,937.2 ±2,831.7 | 2,097.0 [1,005.3-3,814.6] | 8,690.9 | 13,446.9 | 3.8 to 23,228.7 | 14,515,506.0 |
| **$HIC_{15}$** | 12.6 ±82.7 | 3.1 [1.6-7.4] | 41.4 | 137.9 | 0.7 to 3,586.1 | 62,375.4 |
| **GSI** | 0.04 ±0.53 | 0.00 [0.00-0.01] | 0.10 | 0.56 | 0.00 to 24.49 | 192.26 |
| **HITsp** | 18.2 ±10.4 | 15.6 [14.1-18.7] | 30.9 | 54.5 | 11.4 to 273.3 | 90,070.6 |
| **GAMBIT** | 0.085 ±0.068 | 0.060 [0.040-0.106] | 0.233 | 0.329 | 0.013 to 0.668 | 417.19 |
| **ms** | 7.3 ±5.2 | 6.0 [4.0-9.0] | 17.0 | 26.0 | 2.0 to 40.0 | 35927.0 |
| **$RWE_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.141 | 1.754 |
| **$RWE_{Rotational}$** | 0.120 ±0.295 | 0.000 [0.000-0.007] | 0.992 | 1.000 | 0.000 to 1.000 | 591.980 |
| **$RWE_{CP}$** | 0.039 ±0.155 | 0.000 [0.000-0.002] | 0.203 | 0.967 | 0.000 to 1.000 | 192.981 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of competition matches impact biomechanics for impacts to the back of the head (n=4,030) of forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.9 ±15.8 | 16.0 [12.4-25.4] | 51.7 | 88.0 | 10.0 to 164.1 | 88,397.3 |
| **PRA (rad/s$^2$)** | 4,548.8 ±3,788.7 | 3,391.9 [1,868.5-6,132.2] | 12,590.7 | 17,156.0 | 2.9 to 23,397.1 | 18,331,618.4 |
| **$HIC_{15}$** | 20.1 ±66.5 | 4.4 [2.0-13.0] | 86.5 | 264.0 | 0.6 to 2,096.4 | 80,906.9 |
| **GSI** | 0.07 ±0.40 | 0.00 [0.00-0.02] | 0.30 | 1.57 | 0.00 to 14.32 | 300.04 |
| **HITsp** | 13.4 ±8.3 | 11.2 [9.7-14.2] | 24.5 | 43.2 | 7.5 to 168.6 | 54,018.8 |
| **GAMBIT** | 0.109 ±0.073 | 0.086 [0.056-0.141] | 0.254 | 0.347 | 0.011 to 0.588 | 427.79 |
| **ms** | 8.9 ±6.8 | 7.0 [4.0-11.0] | 23.0 | 36.0 | 2.0 to 40.0 | 35,699.0 |
| **$RWE_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.187 | 2.003 |
| **$RWE_{Rotational}$** | 0.246 ±0.396 | 0.003 [0.000-0.433] | 1.000 | 1.000 | 0.000 to 1.000 | 993.263 |
| **$RWE_{CP}$** | 0.103 ±0.255 | 0.001 [0.000-0.019] | 0.898 | 0.999 | 0.000 to 1.000 | 414.880 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of competition matches impact biomechanics for impacts to the side of the head (n=4,166) of forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.9 ±14.1 | 14.0 [11.3-20.6] | 44.1 | 84.1 | 10.0 to 164.9 | 78,651.6 |
| **PRA (rad/s$^2$)** | 2,330.2 ±2,337.4 | 1,745.8 [872.9-2,960.7] | 7,041.0 | 11,480.9 | 2.3 to 18,435.9 | 9,707,797.9 |
| **HIC$_{15}$** | 14.7 ±83.5 | 3.3 [1.6-8.0] | 44.4 | 207.1 | 0.5 to 3,181.5 | 61,411.3 |
| **GSI** | 0.05 ±0.52 | 0.00 [0.00-0.01] | 0.11 | 0.84 | 0.00 to 21.73 | 196.06 |
| **HITsp** | 19.2 ±12.9 | 16.1 [14.7-19.2] | 32.0 | 69.9 | 12.0 to 252.6 | 79,831.6 |
| **GAMBIT** | 0.074 ±0.057 | 0.055 [0.039-0.087] | 0.181 | 0.293 | 0.010 to 0.835 | 304.46 |
| **ms** | 7.5 ±5.4 | 6.0 [4.0-10.0] | 18.0 | 27.0 | 2.0 to 40.0 | 31191.0 |
| **RWE$_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.193 | 1.777 |
| **RWE$_{Rotational}$** | 0.069 ±0.229 | 0.000 [0.000-0.001] | 0.825 | 1.000 | 0.000 to 1.000 | 285.859 |
| **RWE$_{CP}$** | 0.025 ±0.124 | 0.000 [0.000-0.001] | 0.073 | 0.902 | 0.000 to 1.000 | 102.849 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of competition matches impact biomechanics for impacts to the top of the head (n=395) of forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.5 ±15.9 | 13.4 [11.5-21.4] | 46.2 | 98.9 | 10.0 to 152.2 | 7,712.0 |
| **PRA (rad/s$^2$)** | 3,082.5 ±3,530.2 | 1,472.0 [814.8-4,392.6] | 10,635.5 | 16,399.9 | 2.8 to 22,989.2 | 1,217,593.1 |
| **HIC$_{15}$** | 19.6 ±84.8 | 2.7 [1.5-8.0] | 59.8 | 427.0 | 0.8 to 991.8 | 7,736.1 |
| **GSI** | 0.08 ±0.49 | 0.00 [0.00-0.01] | 0.20 | 2.03 | 0.00 to 6.77 | 30.88 |
| **HITsp** | 9.8 ±7.3 | 7.6 [7.0-9.6] | 17.4 | 47.1 | 5.9 to 95.4 | 3,851.7 |
| **GAMBIT** | 0.091 ±0.085 | 0.053 [0.034-0.122] | 0.276 | 0.391 | 0.011 to 0.476 | 35.39 |
| **ms** | 7.6 ±6.1 | 6.0 [4.0-9.0] | 20.0 | 37.0 | 2.0 to 40.0 | 3,016.0 |
| **RWE$_{Linear}$** | 0.001 ±0.006 | 0.000 [0.000-0.000] | 0.001 | 0.008 | 0.000 to 0.112 | 0.230 |
| **RWE$_{Rotational}$** | 0.143 ±0.321 | 0.000 [0.000-0.022] | 1.000 | 1.000 | 0.000 to 1.000 | 56.500 |
| **RWE$_{CP}$** | 0.063 ±0.210 | 0.000 [0.000-0.004] | 0.672 | 0.999 | 0.000 to 1.000 | 24.779 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 6:** Summary of competition matches impact biomechanics for impacts to the head (n-7,154) of backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.3 ±14.5 | 14.3 [11.6-20.6] | 45.3 | 89.1 | 10.0 to 163.2 | 137,874.7 |
| **PRA (rad/s$^2$)** | 2,804.1 ±2,661.3 | 2,006.8 [1,089.9-3,621.2] | 8,345.8 | 12,686.7 | 0.4 to 21,979.5 | 20,052,254.1 |
| **HIC$_{15}$** | 14.7 ±87.2 | 3.2 [1.6-8.0] | 50.6 | 214.3 | 0.5 to 5,900.4 | 105,196.7 |
| **GSI** | 0.05 ±0.55 | 0.00 [0.00-0.01] | 0.12 | 0.87 | 0.00 to 40.30 | 334.88 |
| **HITsp** | 17.0 ±11.4 | 15.0 [13.1-17.7] | 30.2 | 59.9 | 5.8 to 356.8 | 121,454.7 |
| **GAMBIT** | 0.079 ±0.061 | 0.060 [0.040-0.097] | 0.193 | 0.296 | 0.010 to 1.828 | 559.36 |
| **ms** | 7.2 ±5.4 | 5.0 [4.0-9.0] | 18.0 | 27.0 | 2.0 to 40.0 | 51,555.0 |
| **RWE$_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.180 | 3.038 |
| **RWE$_{Rotational}$** | 0.103 ±0.274 | 0.000 [0.000-0.005] | 0.985 | 1.000 | 0.000 to 1.000 | 736.236 |
| **RWE$_{CP}$** | 0.035 ±0.147 | 0.000 [0.000-0.002] | 0.189 | 0.942 | 0.000 to 1.000 | 253.358 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 7:** Summary of competition matches impact biomechanics for impacts to the front of the head (n=1,736) of backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.5 ±12.9 | 13.1 [11.3-18.2] | 41.3 | 74.3 | 10.0 to 163.2 | 30,451.1 |
| **PRA (rad/s$^2$)** | 2,810.5 ±2,682.7 | 1,921.5 [1,088.0-3,557.9] | 8,305.7 | 12,847.8 | 3.6 to 21,528.1 | 4,879,093.5 |
| **$HIC_{15}$** | 13.8 ±146.2 | 2.6 [1.4-5.7] | 37.2 | 153.5 | 0.7 to 5,900.4 | 23,964.8 |
| **GSI** | 0.05 ±0.98 | 0.00 [0.00-0.01] | 0.08 | 0.65 | 0.00 to 40.30 | 87.84 |
| **HITsp** | 18.3 ±13.3 | 15.4 [14.2-18.1] | 30.0 | 68.4 | 11.9 to 356.8 | 31,745.6 |
| **GAMBIT** | 0.077 ±0.072 | 0.054 [0.039-0.095] | 0.200 | 0.293 | 0.014 to 1.828 | 133.27 |
| **ms** | 6.7 ±4.8 | 5.0 [4.0-8.0] | 16.0 | 26.0 | 2.0 to 40.0 | 11,610.0 |
| **$RWE_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.000 | 0.002 | 0.000 to0.180 | 0.689 |
| **$RWE_{Rotational}$** | 0.106 ±0.277 | 0.000 [0.000-0.004] | 0.983 | 1.000 | 0.000 to 1.000 | 184.683 |
| **$RWE_{CP}$** | 0.033 ±0.141 | 0.000 [0.000-0.002] | 0.148 | 0.934 | 0.000 to 1.000 | 57.302 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 8:** Summary of competition matches impact biomechanics for impacts to the back of the head (n=1,902) of backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.0 ±15.7 | 15.7 [12.3-23.1] | 51.0 | 92.4 | 10.0 to 155.5 | 39,973.3 |
| **PRA (rad/s$^2$)** | 3,797.3 ±3,109.1 | 2,867.4 [1,663.6-4,971.9] | 10,025.9 | 15,448.8 | 4.8 to 21,979.5 | 7,222,497.3 |
| **$HIC_{15}$** | 17.3 ±61.3 | 3.9 [1.9-10.0] | 69.9 | 237.0 | 0.8 to 1,006.5 | 32,995.6 |
| **GSI** | 0.05 ±0.30 | 0.00 [0.00-0.01] | 0.18 | 0.94 | 0.00 to 6.40 | 99.21 |
| **HITsp** | 13.3 ±7.4 | 11.2 [9.8-13.9] | 26.0 | 45.2 | 7.3 to 94.7 | 25,307.3 |
| **GAMBIT** | 0.097 ±0.062 | 0.077 [0.054-0.121] | 0.224 | 0.324 | 0.010 to 0.524 | 180.58 |
| **ms** | 7.8 ±5.7 | 6.0 [4.0-10.0] | 19.0 | 29.0 | 2.0 to 40.0 | 14,795.0 |
| **$RWE_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.130 | 0.946 |
| **$RWE_{Rotational}$** | 0.176 ±0.344 | 0.001 [0.000-0.070] | 0.999 | 1.000 | 0.000 to 1.000 | 335.314 |
| **$RWE_{CP}$** | 0.061 ±0.192 | 0.001 [0.000-0.007] | 0.481 | 0.989 | 0.000 to 1.000 | 116.270 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 9:** Summary of competition matches impact biomechanics for impacts to the side of the head (n=3,198) of backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.1 ±14.5 | 14.3 [11.4-20.6] | 44.2 | 88.0 | 10.0 to 144.4 | 61,229.7 |
| **PRA (rad/s$^2$)** | 2,244.1 ±2,154.6 | 1,705.4 [804.5-2,999.5] | 6,630.2 | 10,521.9 | 0.4 to 15,763.9 | 7,169,929.9 |
| **$HIC_{15}$** | 13.6 ±53.1 | 3.2 [1.6-8.1] | 48.4 | 218.3 | 0.5 to 1,566.5 | 43,468.0 |
| **GSI** | 0.04 ±0.30 | 0.00 [0.00-0.01] | 0.11 | 0.88 | 0.00 to 10.70 | 133.01 |
| **HITsp** | 19.1 ±11.5 | 16.1 [14.7-19.3] | 34.0 | 64.0 | 11.9 to 239.3 | 61,051.0 |
| **GAMBIT** | 0.070 ±0.051 | 0.054 [0.038-0.084] | 0.167 | 0.280 | 0.010 to 0.595 | 222.82 |
| **ms** | 7.2 ±5.4 | 5.0 [3.0-9.0] | 18.0 | 28.0 | 2.0 to 40.0 | 22,916.0 |
| **$RWE_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.078 | 1.263 |
| **$RWE_{Rotational}$** | 0.060 ±0.211 | 0.000 [0.000-0.001] | 0.672 | 1.000 | 0.000 to 1.000 | 191.421 |
| **$RWE_{CP}$** | 0.022 ±0.114 | 0.000 [0.000-0.001] | 0.049 | 0.768 | 0.000 to 0.997 | 70.767 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 10:** Summary of competition matches impact biomechanics for impacts to the top of the head (n=318) of backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.6 ±15.1 | 14.8 [12.1-21.1] | 42.6 | 114.2 | 10.1 to 122.9 | 6,220.6 |
| **PRA (rad/s$^2$)** | 2,455.1 ±2,533.5 | 1,539.9 [914.0-3,181.7] | 7,224.8 | 14,927.8 | 2.5 to 17,346.9 | 780,733.3 |
| **HIC$_{15}$** | 15.0 ±58.3 | 3.1 [1.7-7.7] | 40.9 | 352.2 | 0.8 to 640.3 | 4,768.3 |
| **GSI** | 0.05 ±0.30 | 0.00 [0.00-0.01] | 0.09 | 1.02 | 0.00 to 3.62 | 14.82 |
| **HITsp** | 10.5 ±11.9 | 7.9 [7.1-10.2] | 19.7 | 48.1 | 5.8 to 178.6 | 3,344.1 |
| **GAMBIT** | 0.072 ±0.057 | 0.052 [0.034-0.094] | 0.188 | 0.303 | 0.014 to 0.412 | 22.69 |
| **ms** | 7.0 ±5.0 | 5.0 [4.0-8.0] | 17.0 | 27.6 | 2.0 to 37.0 | 2,234.0 |
| **RWE$_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.000 | 0.018 | 0.000 to 0.028 | 0.141 |
| **RWE$_{Rotational}$** | 0.078 ±0.242 | 0.000 [0.000-0.002] | 0.866 | 1.000 | 0.000 to 1.000 | 24.818 |
| **RWE$_{CP}$** | 0.028 ±0.135 | 0.000 [0.000-0.002] | 0.111 | 0.980 | 0.000 to 1.000 | 9.017 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 11:** Summary of competition matches impact biomechanics for impacts to the head (n=6,530) of front row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total..

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.8 ±13.2 | 14.1 [11.6-20.5] | 41.9 | 77.0 | 10.0 to 157.9 | 122,609.0 |
| **PRA (rad/s$^2$)** | 2,905.6 ±2,774.3 | 2,073.9 [1,066.5-3,712.8] | 8,679.5 | 13,418.9 | 2.8 to 21,619.0 | 18,973,666.9 |
| **HIC$_{15}$** | 13.8 ±93.5 | 3.2 [1.7-8.0] | 41.4 | 147.8 | 0.6 to 3,586.1 | 89,819.1 |
| **GSI** | 0.05 ±0.61 | 0.00 [0.00-0.01] | 0.11 | 0.61 | 0.00 to 24.49 | 303.70 |
| **HITsp** | 17.3 ±12.1 | 15.1 [13.4-18.1] | 29.7 | 54.7 | 6.4 to 273.3 | 112,928.5 |
| **GAMBIT** | 0.086 ±0.070 | 0.061 [0.041-0.104] | 0.231 | 0.350 | 0.012 to 0.835 | 557.43 |
| **ms** | 7.5 ±5.3 | 6.0 [4.0-10.0] | 18.0 | 27.0 | 2.0 to 40.0 | 48,944.0 |
| **RWE$_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.144 | 2.454 |
| **RWE$_{Rotational}$** | 0.113 ±0.288 | 0.000 [0.000-0.006] | 0.992 | 1.000 | 0.000 to 1.000 | 735.460 |
| **RWE$_{CP}$** | 0.039 ±0.156 | 0.000 [0.000-0.002] | 0.202 | 0.965 | 0.000 to 1.000 | 253.673 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 12:** Summary of competition matches impact biomechanics for impacts to the front of the head (n=3,004) of front row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.2 ±12.9 | 13.9 [11.5-19.5] | 39.8 | 77.7 | 10.0 to 157.5 | 54,818.9 |
| **PRA (rad/s$^2$)** | 2,850.7 ±2,745.4 | 1,989.0 [1,020.1-3,605.5] | 8,606.6 | 13,108.5 | 3.8 to 18,748.8 | 8,563,561.7 |
| **HIC$_{15}$** | 13.1 ±101.2 | 3.1 [1.6-7.4] | 38.2 | 135.7 | 0.7 to 3,586.1 | 39,237.6 |
| **GSI** | 0.04 ±0.66 | 0.00 [0.00-0.01] | 0.09 | 0.49 | 0.00 to 24.49 | 132.50 |
| **HITsp** | 18.1 ±11.2 | 15.5 [14.0-18.5] | 30.1 | 54.6 | 11.4 to 273.3 | 54,509.2 |
| **GAMBIT** | 0.085 ±0.070 | 0.059 [0.040-0.102] | 0.238 | 0.348 | 0.013 to 0.604 | 253.82 |
| **ms** | 7.3 ±5.0 | 6.0 [4.0-9.0] | 17.0 | 25.9 | 2.0 to 40.0 | 22,014.0 |
| **RWE$_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.141 | 1.166 |
| **RWE$_{Rotational}$** | 0.112 ±0.287 | 0.000 [0.000-0.005] | 0.991 | 1.000 | 0.000 to 1.000 | 337.135 |
| **RWE$_{CP}$** | 0.038 ±0.154 | 0.000 [0.000-0.002] | 0.153 | 0.963 | 0.000 to 1.000 | 112.876 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 13:** Summary of competition matches impact biomechanics for impacts to the back of the head (n=1,257) of front row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.7 ±13.7 | 15.1 [12.2-22.4] | 43.5 | 71.9 | 10.0 to 156.4 | 24,821.3 |
| **PRA (rad/s$^2$)** | 4,111.0 ±3,277.7 | 3,202.7 [1,835.9-5,429.6] | 10,806.1 | 15,812.9 | 4.9 to 21,619.0 | 5,167,515.9 |
| **$HIC_{15}$** | 13.4 ±51.7 | 3.6 [1.9-8.9] | 50.8 | 144.7 | 0.6 to 1,242.2 | 16,852.6 |
| **GSI** | 0.04 ±0.31 | 0.00 [0.00-0.01] | 0.13 | 0.62 | 0.00 to 7.79 | 52.54 |
| **HITsp** | 13.3 ±10.3 | 11.1 [9.8-13.8] | 21.9 | 46.5 | 7.7 to 197.3 | 16,766.7 |
| **GAMBIT** | 0.108 ±0.077 | 0.083 [0.054-0.136] | 0.254 | 0.391 | 0.012 to 0.588 | 134.88 |
| **ms** | 7.5 ±5.4 | 6.0 [4.0-9.0] | 18.0 | 29.4 | 2.0 to 40.0 | 9,382.0 |
| **$RWE_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.002 | 0.000 to 0.134 | 0.551 |
| **$RWE_{Rotational}$** | 0.207 ±0.368 | 0.002 [0.000-0.156] | 1.000 | 1.000 | 0.000 to 1.000 | 260.317 |
| **$RWE_{CP}$** | 0.070 ±0.205 | 0.001 [0.000-0.010] | 0.623 | 0.994 | 0.000 to 1.000 | 87.809 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 14:** Summary of competition matches impact biomechanics for impacts to the side of the head (n=2,059) of front row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.1 ±13.3 | 14.4 [11.6-21.3] | 44.7 | 77.7 | 10.0 to 157.9 | 39,292.5 |
| **PRA (rad/s$^2$)** | 2,309.5 ±2,174.8 | 1,812.0 [954.6-2,952.9] | 6,394.3 | 11,224.1 | 2.8 to 17,086.2 | 4,755,263.6 |
| **$HIC_{15}$** | 15.2 ±105.0 | 3.4 [1.7-8.7] | 42.7 | 172.3 | 0.6 to 3,181.5 | 31,304.3 |
| **GSI** | 0.05 ±0.70 | 0.00 [0.00-0.01] | 0.12 | 0.81 | 0.00 to 21.73 | 111.62 |
| **HITsp** | 19.3 ±13.4 | 16.2 [14.5-19.6] | 32.7 | 64.9 | 12.0 to 252.6 | 39,647.6 |
| **GAMBIT** | 0.074 ±0.060 | 0.057 [0.039-0.087] | 0.179 | 0.311 | 0.017 to 0.835 | 151.39 |
| **ms** | 7.8 ±5.7 | 6.0 [4.0-10.0] | 19.0 | 27.0 | 2.0 to 40.0 | 16,147.0 |
| **$RWE_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.144 | 0.681 |
| **$RWE_{Rotational}$** | 0.058 ±0.211 | 0.000 [0.000-0.001] | 0.564 | 1.000 | 0.000 to 1.000 | 118.567 |
| **$RWE_{CP}$** | 0.022 ±0.115 | 0.000 [0.000-0.001] | 0.041 | 0.802 | 0.000 to 0.999 | 44.366 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 15:** Summary of competition matches impact biomechanics for impacts to the top of the head (n=210) of front row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.5 ±13.8 | 12.6 [11.4-16.4] | 43.0 | 97.9 | 10.2 to 100.3 | 3,676.2 |
| **PRA (rad/s$^2$)** | 2,320.6 ±2,894.9 | 1,056.1 [748.8-2,808.5] | 8,599.0 | 14,988.3 | 189.6 to 15,921.6 | 487,325.7 |
| **$HIC_{15}$** | 11.5 ±38.9 | 2.1 [1.4-4.2] | 46.2 | 284.5 | 0.9 to 349.2 | 2,424.5 |
| **GSI** | 0.03 ±0.17 | 0.00 [0.00-0.00] | 0.12 | 1.51 | 0.00 to 1.77 | 7.05 |
| **HITsp** | 9.1 ±5.4 | 7.3 [7.0-8.4] | 17.1 | 43.1 | 6.4 to 47.1 | 1,908.9 |
| **GAMBIT** | 0.083 ±0.088 | 0.040 [0.032-0.091] | 0.280 | 0.448 | 0.018 to 0.476 | 17.34 |
| **ms** | 6.7 ±5.1 | 5.0 [4.0-7.0] | 19.0 | 28.8 | 2.0 to 31.0 | 1,401.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.000 | 0.008 | 0.000 to 0.009 | 0.057 |
| **$RWE_{Rotational}$** | 0.093 ±0.266 | 0.000 [0.000-0.001] | 0.991 | 1.000 | 0.000 to 1.000 | 19.441 |
| **$RWE_{CP}$** | 0.041 ±0.172 | 0.000 [0.000-0.001] | 0.177 | 0.992 | 0.000 to 0.999 | 8.622 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 16:** Summary of competition matches impact biomechanics for impacts to the head (n=7,003) of back row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.3 ±15.4 | 14.7 [11.5-22.7] | 48.4 | 88.7 | 10.0 to 164.9 | 142,454.2 |
| **PRA (rad/s$^2$)** | 3,540.6 ±3,470.0 | 2,468.3 [1,171.3-4,679.6] | 10,799.0 | 16,351.5 | 2.3 to 23,397.1 | 24,794,704.5 |
| **HIC$_{15}$** | 17.5 ±61.5 | 3.7 [1.7-10.1] | 69.8 | 263.9 | 0.5 to 2,096.4 | 122,597.5 |
| **GSI** | 0.06 ±0.34 | 0.00 [0.00-0.01] | 0.22 | 1.14 | 0.00 to 14.32 | 415.52 |
| **HITsp** | 16.4 ±10.2 | 14.7 [12.1-17.5] | 28.8 | 55.1 | 5.9 to 198.3 | 115,187.9 |
| **GAMBIT** | 0.091 ±0.067 | 0.068 [0.044-0.116] | 0.235 | 0.317 | 0.010 to 0.668 | 627.31 |
| **ms** | 8.1 ±6.3 | 6.0 [4.0-10.0] | 20.0 | 35.0 | 2.0 to 40.0 | 56,882.0 |
| **RWE$_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.193 | 3.309 |
| **RWE$_{Rotational}$** | 0.170 ±0.345 | 0.001 [0.000-0.040] | 1.000 | 1.000 | 0.000 to 1.000 | 1,191.746 |
| **RWE$_{CP}$** | 0.069 ±0.212 | 0.001 [0.000-0.005] | 0.657 | 0.998 | 0.000 to 1.000 | 481.796 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 17:** Summary of competition matches impact biomechanics for impacts to the front of the head (n=1,938) of back row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.3 ±13.2 | 13.5 [11.2-19.8] | 41.1 | 83.2 | 10.0 to 141.6 | 35,498.7 |
| **PRA (rad/s$^2$)** | 3,071.2 ±2,956.2 | 2,292.3 [992.5-4,133.4] | 9,037.0 | 14,441.2 | 4.9 to 23,228.7 | 5,951,944.4 |
| **HIC$_{15}$** | 11.9 ±39.7 | 3.1 [1.6-7.4] | 46.9 | 160.9 | 0.7 to 886.7 | 23,137.9 |
| **GSI** | 0.03 ±0.15 | 0.00 [0.00-0.01] | 0.13 | 0.64 | 0.00 to 3.26 | 59.75 |
| **HITsp** | 18.4 ±10.3 | 15.6 [14.0-18.8] | 32.5 | 57.7 | 11.3 to 211.6 | 35,658.5 |
| **GAMBIT** | 0.085 ±0.065 | 0.062 [0.041-0.109] | 0.226 | 0.309 | 0.015 to 0.668 | 163.38 |
| **ms** | 7.2 ±5.3 | 5.0 [4.0-9.0] | 17.1 | 27.6 | 2.0 to 40.0 | 13,913.0 |
| **RWE$_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.000 | 0.004 | 0.000 to 0.068 | 0.589 |
| **RWE$_{Rotational}$** | 0.131 ±0.306 | 0.000 [0.000-0.014] | 0.996 | 1.000 | 0.000 to 1.000 | 254.844 |
| **RWE$_{CP}$** | 0.041 ±0.158 | 0.001 [0.000-0.003] | 0.236 | 0.980 | 0.000 to 1.000 | 80.105 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 18:** Summary of competition matches impact biomechanics for impacts to the back of the head (n=2,772) of back row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 22.9 ±16.6 | 16.6 [12.5-26.8] | 54.5 | 91.5 | 10.0 to 164.1 | 63,548.7 |
| **PRA (rad/s$^2$)** | 4,746.8 ±3,984.3 | 3,477.3 [1,887.9-6,444.5] | 13,459.7 | 17,492.7 | 2.9 to 23,397.1 | 13,158,049.0 |
| **HIC$_{15}$** | 23.1 ±72.0 | 4.8 [2.0-15.6] | 98.9 | 311.8 | 0.7 to 2,096.4 | 64,039.8 |
| **GSI** | 0.09 ±0.44 | 0.00 [0.00-0.03] | 0.38 | 1.72 | 0.00 to 14.32 | 247.48 |
| **HITsp** | 13.5 ±8.5 | 11.2 [9.5-14.4] | 25.3 | 42.9 | 7.3 to 168.6 | 37,334.6 |
| **GAMBIT** | 0.109 ±0.071 | 0.086 [0.056-0.143] | 0.256 | 0.334 | 0.011 to 0.537 | 292.78 |
| **ms** | 9.5 ±7.3 | 7.0 [4.0-13.0] | 25.0 | 37.3 | 2.0 to 40.0 | 26,306.0 |
| **RWE$_{Linear}$** | 0.001 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.187 | 1.452 |
| **RWE$_{Rotational}$** | 0.264 ±0.407 | 0.004 [0.000-0.587] | 1.000 | 1.000 | 0.000 to 1.000 | 732.550 |
| **RWE$_{CP}$** | 0.118 ±0.274 | 0.002 [0.000-0.026] | 0.956 | 0.999 | 0.000 to 1.000 | 327.051 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 19:** Summary of competition matches impact biomechanics for impacts to the side of the head (n=2,108) of back row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.7 ±14.9 | 13.5 [11.1-20.1] | 43.9 | 90.6 | 10.0 to 164.9 | 39,371.0 |
| **PRA (rad/s$^2$)** | 2,350.3 ±2,485.8 | 1,663.5 [781.2-2,992.7] | 7,438.7 | 11,934.2 | 2.3 to 18,435.9 | 4,954,443.7 |
| **HIC$_{15}$** | 14.3 ±54.9 | 3.2 [1.6-7.4] | 47.8 | 272.2 | 0.5 to 938.3 | 30,108.3 |
| **GSI** | 0.04 ±0.26 | 0.00 [0.00-0.01] | 0.10 | 0.92 | 0.00 to 5.76 | 84.45 |
| **HITsp** | 19.1 ±12.3 | 15.9 [14.8-18.9] | 31.7 | 72.7 | 12.0 to 198.3 | 40,193.0 |
| **GAMBIT** | 0.073 ±0.055 | 0.054 [0.038-0.088] | 0.182 | 0.281 | 0.010 to 0.501 | 153.10 |
| **ms** | 7.1 ±5.2 | 6.0 [4.0-9.0] | 17.0 | 27.0 | 2.0 to 40.0 | 15,048.0 |
| **RWE$_{Linear}$** | 0.001 ±0.006 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.193 | 1.096 |
| **RWE$_{Rotational}$** | 0.079 ±0.245 | 0.000 [0.000-0.001] | 0.913 | 1.000 | 0.000 to 1.000 | 167.292 |
| **RWE$_{CP}$** | 0.028 ±0.132 | 0.000 [0.000-0.001] | 0.093 | 0.937 | 0.000 to 1.000 | 58.483 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 20:** Summary of competition matches impact biomechanics for impacts to the top of the head (n=185) of back row forwards greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.8 ±17.8 | 15.3 [11.7-23.7] | 53.0 | 116.1 | 10.0 to 152.2 | 4,035.8 |
| **PRA (rad/s$^2$)** | 3,947.4 ±3,968.9 | 2,727.1 [1,048.3-5,350.2] | 12,006.7 | 19,332.0 | 2.8 to 22,989.2 | 730,267.4 |
| **HIC$_{15}$** | 28.7 ±116.3 | 3.9 [2.0-12.3] | 84.1 | 965.7 | 0.8 to 991.8 | 5,311.5 |
| **GSI** | 0.13 ±0.69 | 0.00 [0.00-0.02] | 0.31 | 5.46 | 0.00 to 6.77 | 23.84 |
| **HITsp** | 10.5 ±9.0 | 8.3 [7.2-10.2] | 17.8 | 67.5 | 5.9 to 95.4 | 1,944.8 |
| **GAMBIT** | 0.102 ±0.080 | 0.072 [0.042-0.153] | 0.274 | 0.367 | 0.011 to 0.426 | 18.05 |
| **ms** | 8.7 ±7.0 | 6.0 [4.0-10.0] | 20.0 | 39.1 | 2.0 to 40.0 | 1,615.0 |
| **RWE$_{Linear}$** | 0.001 ±0.008 | 0.000 [0.000-0.000] | 0.001 | 0.028 | 0.000 to 0.112 | 0.173 |
| **RWE$_{Rotational}$** | 0.200 ±0.366 | 0.001 [0.000-0.125] | 1.000 | 1.000 | 0.000 to 1.000 | 37.059 |
| **RWE$_{CP}$** | 0.087 ±0.244 | 0.001 [0.000-0.012] | 0.871 | 1.000 | 0.000 to 1.000 | 16.157 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 21:** Summary of competition matches impact biomechanics for impacts to the head (n=4,477) of inside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.7 ±15.1 | 14.6 [11.6-21.0] | 46.9 | 91.3 | 10.0 to 163.2 | 88,195.9 |
| **PRA (rad/s$^2$)** | 2,705.9 ±2,591.2 | 1,948.7 [1,049.5-3,496.0] | 7,984.2 | 12,437.0 | 0.4 to 21,979.5 | 12,106,265.9 |
| **HIC$_{15}$** | 16.4 ±105.4 | 3.3 [1.6-8.4] | 57.6 | 238.1 | 0.6 to 5,900.4 | 73,368.2 |
| **GSI** | 0.06 ±0.68 | 0.00 [0.00-0.01] | 0.16 | 0.94 | 0.00 to 40.30 | 252.92 |
| **HITsp** | 12.4 ±8.1 | 10.7 [9.6-12.6] | 20.9 | 38.0 | 8.6 to 374.7 | 55,510.6 |
| **GAMBIT** | 0.077 ±0.060 | 0.059 [0.040-0.095] | 0.183 | 0.284 | 0.010 to 1.828 | 340.29 |
| **ms** | 7.3 ±5.5 | 5.0 [4.0-9.0] | 18.0 | 28.0 | 2.0 to 40.0 | 32,903.0 |
| **RWE$_{Linear}$** | 0.000 ±0.005 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.180 | 2.134 |
| **RWE$_{Rotational}$** | 0.093 ±0.260 | 0.000 [0.000-0.004] | 0.969 | 1.000 | 0.000 to 1.000 | 417.518 |
| **RWE$_{CP}$** | 0.033 ±0.144 | 0.000 [0.000-0.002] | 0.146 | 0.941 | 0.000 to 1.000 | 149.576 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 22:** Summary of competition matches impact biomechanics for impacts to the front of the head (n=980) of inside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.8 ±14.9 | 13.8 [11.5-19.3] | 46.6 | 96.0 | 10.0 to 163.2 | 18,429.9 |
| **PRA (rad/s$^2$)** | 2,958.7 ±2,808.6 | 2,043.0 [1,121.9-3,779.0] | 8,814.6 | 13,286.6 | 3.6 to 19,878.4 | 2,899,495.8 |
| **$HIC_{15}$** | 18.9±193.1 | 3.0 [1.6-6.9] | 63.1 | 210.5 | 0.7 to 5,900.4 | 18,526.9 |
| **GSI** | 0.08 ±1.30 | 0.00 [0.00-0.01] | 0.16 | 0.95 | 0.00 to 40.30 | 75.63 |
| **HITsp** | 12.4 ±8.0 | 10.8 [9.9-12.5] | 20.3 | 34.1 | 9.0 to 218.7 | 12,152.0 |
| **GAMBIT** | 0.080 ±0.081 | 0.057 [0.040-0.096] | 0.203 | 0.302 | 0.017 to 1.828 | 77.35 |
| **ms** | 7.0 ±5.3 | 5.0 [4.0-9.0] | 18.0 | 27.0 | 2.0 to 40.0 | 6,903.0 |
| **$RWE_{Linear}$** | 0.001 ±0.007 | 0.000 [0.000-0.000] | 0.001 | 0.007 | 0.000 to 0.180 | 0.567 |
| **$RWE_{Rotational}$** | 0.116 ±0.288 | 0.000 [0.000-0.007] | 0.994 | 1.000 | 0.000 to 1.000 | 113.662 |
| **$RWE_{CP}$** | 0.041 ±0.160 | 0.000 [0.000-0.002] | 0.252 | 0.971 | 0.000 to 1.000 | 40.052 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 23:** Summary of competition matches impact biomechanics for impacts to the back of the head (n=943) of inside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.6 ±16.4 | 15.9 [12.2-23.5] | 54.6 | 93.3 | 10.0 to 155.5 | 20,371.2 |
| **PRA (rad/s$^2$)** | 3,660.8 ±3,115.6 | 2,741.7 [1,588.9-4,823.4] | 9,526.1 | 16,095.2 | 4.8 to 21,979.5 | 3,452,172.7 |
| **$HIC_{15}$** | 19.6 ±73.0 | 4.2 [2.0-10.9] | 74.2 | 252.8 | 0.8 to 1,006.5 | 18,452.3 |
| **GSI** | 0.07 ±0.39 | 0.00 [0.00-0.02] | 0.22 | 1.62 | 0.00 to 6.40 | 63.04 |
| **HITsp** | 12.4 ±8.7 | 10.2 [9.0-12.5] | 24.0 | 41.3 | 7.8 to 121.8 | 11,693.2 |
| **GAMBIT** | 0.093 ±0.059 | 0.074 [0.051-0.120] | 0.211 | 0.274 | 0.010 to 0.524 | 85.94 |
| **ms** | 8.0 ±6.1 | 6.0 [4.0-10.0] | 20.0 | 35.1 | 2.0 to 40.0 | 7,528.0 |
| **$RWE_{Linear}$** | 0.001 ±0.006 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.130 | 0.560 |
| **$RWE_{Rotational}$** | 0.165 ±0.333 | 0.001 [0.000-0.053] | 0.999 | 1.000 | 0.000 to 1.000 | 155.780 |
| **$RWE_{CP}$** | 0.057 ±0.186 | 0.001 [0.000-0.006] | 0.470 | 0.998 | 0.000 to 1.000 | 53.765 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 24:** Summary of competition matches impact biomechanics for impacts to the side of the head (n=2,334)of inside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.3 ±14.4 | 14.4 [11.5-20.8] | 44.5 | 87.8 | 10.0 to 144.4 | 44,955.5 |
| **PRA (rad/s$^2$)** | 2,241.0 ±2,112.2 | 1,698.4 [843.7-3,023.9] | 6,409.3 | 10,540.3 | 0.4 to 13,086.4 | 5,223,793.3 |
| **$HIC_{15}$** | 13.9 ±55.4 | 3.2 [1.6-8.2] | 49.3 | 222.2 | 0.6 to 1,566.5 | 32,385.3 |
| **GSI** | 0.04 ±0.33 | 0.00 [0.00-0.01] | 0.12 | 0.89 | 0.00 to 10.70 | 100.77 |
| **HITsp** | 12.4 ±8.5 | 10.4 [9.2-12.6] | 22.0 | 47.1 | 8.2 to 211.0 | 28,937.3 |
| **GAMBIT** | 0.070 ±0.049 | 0.055 [0.038-0.086] | 0.160 | 0.255 | 0.013 to 0.595 | 161.19 |
| **ms** | 7.2 ±5.4 | 5.0 [3.0-9.0] | 18.0 | 27.0 | 2.0 to 40.0 | 16874.0 |
| **$RWE_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.078 | 0.882 |
| **$RWE_{Rotational}$** | 0.057 ±0.204 | 0.000 [0.000-0.002] | 0.569 | 1.000 | 0.000 to 1.000 | 133.442 |
| **$RWE_{CP}$** | 0.021 ±0.113 | 0.000 [0.000-0.001] | 0.045 | 0.777 | 0.000 to 0.988 | 49.161 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 25:** Summary of competition matches impact biomechanics for impacts to the top of the head (n=220) of inside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.2 ±16.8 | 15.3 [12.2-21.4] | 42.4 | 114.2 | 10.3 to 122.9 | 4,439.4 |
| **PRA (rad/s$^2$)** | 2,412.7 ±2,459.6 | 1,552.2 [936.0-3,113.8] | 6,926.4 | 14,896.0 | 2.5 to 17,346.9 | 530,804.1 |
| **HIC$_{15}$** | 18.2 ±69.3 | 3.4 [1.6-7.7] | 46.5 | 492.5 | 0.8 to 640.3 | 4,003.6 |
| **GSI** | 0.06 ±0.35 | 0.00 [0.00-0.01] | 0.09 | 2.85 | 0.00 to 3.62 | 13.48 |
| **HITsp** | 12.4 ±9.1 | 10.2 [9.2-12.1] | 18.2 | 76.6 | 8.5 to 90.3 | 2,728.0 |
| **GAMBIT** | 0.073 ±0.058 | 0.055 [0.034-0.094] | 0.187 | 0.331 | 0.016 to 0.412 | 15.81 |
| **ms** | 7.3 ±5.3 | 6.0 [4.0-8.0] | 17.0 | 33.5 | 2.0 to 37.0 | 1,598.0 |
| **RWE$_{Linear}$** | 0.001 ±0.003 | 0.000 [0.000-0.000] | 0.000 | 0.018 | 0.000 to 0.028 | 0.124 |
| **RWE$_{Rotational}$** | 0.067 ±0.222 | 0.000 [0.000-0.002] | 0.789 | 1.000 | 0.000 to 1.000 | 14.635 |
| **RWE$_{CP}$** | 0.030 ±0.145 | 0.000 [0.000-0.001] | 0.048 | 0.985 | 0.000 to 1.000 | 6.598 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 26:** Summary of competition matches impact biomechanics for impacts to the head (n=2,677) of outside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.6 ±13.7 | 13.9 [11.5-19.8] | 42.9 | 84.8 | 10.0 to 148.0 | 49,787.5 |
| **PRA (rad/s$^2$)** | 2,972.5 ±2,775.6 | 2,132.6 [1,176.0-3,800.2] | 8,952.4 | 13,193.9 | 2.7 to 21,528.1 | 7,957,455.6 |
| **HIC$_{15}$** | 12.1 ±42.0 | 2.9 [1.5-7.1] | 41.0 | 198.3 | 0.5 to 803.8 | 32,392.1 |
| **GSI** | 0.03 ±0.17 | 0.00 [0.00-0.01] | 0.10 | 0.79 | 0.00 to 3.47 | 84.70 |
| **HITsp** | 16.1 ±9.8 | 14.5 [12.1-16.8] | 27.5 | 54.3 | 6.5 to 196.8 | 42,969.0 |
| **GAMBIT** | 0.082 ±0.061 | 0.061 [0.041-0.100] | 0.208 | 0.314 | 0.010 to 0.452 | 218.87 |
| **ms** | 7.0 ±5.1 | 5.0 [4.0-9.0] | 17.0 | 26.0 | 2.0 to 40.0 | 18,691.0 |
| **RWE$_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.000 | 0.004 | 0.000 to 0.092 | 0.976 |
| **RWE$_{Rotational}$** | 0.120 ±0.296 | 0.000 [0.000-0.007] | 0.995 | 1.000 | 0.000 to 1.000 | 319.999 |
| **RWE$_{CP}$** | 0.040 ±0.153 | 0.000 [0.000-0.002] | 0.232 | 0.956 | 0.000 to 1.000 | 105.840 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 27:** Summary of competition matches impact biomechanics for impacts to the front of the head (n=756) of outside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 15.9 ±9.3 | 12.8 [11.2-16.7] | 31.6 | 61.1 | 10.0 to 92.7 | 12,021.2 |
| **PRA (rad/s$^2$)** | 2,618.5 ±2,498.8 | 1,711.3 [1,015.1-3,344.3] | 7,756.9 | 11,518.6 | 5.6 to 21,528.1 | 1,979,597.8 |
| **HIC$_{15}$** | 7.2 ±26.6 | 2.2 [1.4-4.2] | 22.4 | 111.8 | 0.7 to 463.2 | 5,437.8 |
| **GSI** | 0.02 ±0.13 | 0.00 [0.00-0.00] | 0.05 | 0.28 | 0.00 to 3.16 | 12.21 |
| **HITsp** | 17.3 ±10.4 | 15.1 [14.1-17.2] | 26.5 | 48.8 | 12.5 to 196.8 | 13,074.9 |
| **GAMBIT** | 0.074 ±0.057 | 0.051 [0.037-0.094] | 0.191 | 0.288 | 0.014 to 0.426 | 55.93 |
| **ms** | 6.2 ±4.1 | 5.0 [4.0-7.0] | 16.0 | 20.0 | 2.0 to 39.0 | 4,707.0 |
| **RWE$_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | 0.001 | 0.000 to 0.006 | 0.122 |
| **RWE$_{Rotational}$** | 0.094 ±0.263 | 0.000 [0.000-0.003] | 0.952 | 1.000 | 0.000 to 1.000 | 71.021 |
| **RWE$_{CP}$** | 0.023 ±0.110 | 0.000 [0.000-0.001] | 0.082 | 0.768 | 0.000 to 1.000 | 17.250 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 28:** Summary of competition matches impact biomechanics for impacts to the back of the head (n=968) of outside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.4 ±15.0 | 15.2 [12.3-22.3] | 49.3 | 92.6 | 10.0 to 148.0 | 19,769.6 |
| **PRA (rad/s$^2$)** | 3,925.2 ±3,093.3 | 2,987.2 [1,778.2-5,046.0] | 10,413.6 | 15,210.6 | 9.1 to 17,711.2 | 3,799,608.3 |
| **$HIC_{15}$** | 15.1 ±46.7 | 3.6 [1.7-9.3] | 68.5 | 231.5 | 0.8 to 803.8 | 14,650.0 |
| **GSI** | 0.04 ±0.17 | 0.00 [0.00-0.01] | 0.14 | 0.87 | 0.00 to 2.99 | 36.42 |
| **HITsp** | 13.0 ±6.4 | 11.2 [9.7-13.8] | 24.4 | 43.6 | 7.3 to 76.3 | 12,623.7 |
| **GAMBIT** | 0.100 ±0.064 | 0.079 [0.056-0.123] | 0.237 | 0.343 | 0.012 to 0.380 | 95.32 |
| **ms** | 7.6 ±5.3 | 6.0 [4.0-10.0] | 19.0 | 26.0 | 2.0 to 40.0 | 7,329.0 |
| **$RWE_{Linear}$** | 0.000 ±0.003 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.092 | 0.387 |
| **$RWE_{Rotational}$** | 0.187 ±0.354 | 0.001 [0.000-0.079] | 1.000 | 1.000 | 0.000 to 1.000 | 180.661 |
| **$RWE_{CP}$** | 0.065 ±0.198 | 0.001 [0.000-0.007] | 0.547 | 0.985 | 0.000 to 1.000 | 62.961 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 29:** Summary of competition matches impact biomechanics for impacts to the side of the head (n=862) of outside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.9 ±15.3 | 13.7 [11.2-19.8] | 44.3 | 99.6 | 10.0 to 142.5 | 16,326.0 |
| **PRA (rad/s$^2$)** | 2,259.2 ±2,302.0 | 1,730.4 [700.2-2,901.3] | 7,234.9 | 10,736.1 | 2.7 to 15,763.9 | 1,947,458.4 |
| **$HIC_{15}$** | 13.4 ±48.6 | 3.3 [1.5-7.7] | 46.9 | 216.0 | 0.5 to 667.2 | 11,567.4 |
| **GSI** | 0.04 ±0.21 | 0.00 [0.00-0.01] | 0.13 | 1.11 | 0.00 to 3.47 | 34.75 |
| **HITsp** | 19.0 ±11.5 | 15.8 [14.6-18.8] | 35.7 | 82.4 | 12.3 to 135.3 | 16,418.5 |
| **GAMBIT** | 0.072 ±0.058 | 0.052 [0.038-0.081] | 0.194 | 0.304 | 0.010 to 0.452 | 61.32 |
| **ms** | 7.0 ±5.5 | 5.0 [3.0-9.0] | 18.0 | 30.4 | 2.0 to 40.0 | 6,059.0 |
| **$RWE_{Linear}$** | 0.001 ±0.004 | 0.000 [0.000-0.000] | 0.001 | 0.009 | 0.000 to 0.071 | 0.451 |
| **$RWE_{Rotational}$** | 0.069 ±0.233 | 0.000 [0.000-0.001] | 0.874 | 1.000 | 0.000 to 1.000 | 59.160 |
| **$RWE_{CP}$** | 0.027 ±0.126 | 0.000 [0.000-0.001] | 0.108 | 0.787 | 0.000 to 0.997 | 23.318 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted

Exposure Combined (linear and rotational) Probability.

**Table 30:** Summary of competition matches impact biomechanics for impacts to the top of the head (n=91) of outside backs greater than 10*g* recorded by an instrumented mouthguard in a senior amateur rugby union team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.4 ±10.3 | 13.7 [11.7-20.1] | 42.7 | - | 10.1 to 59.6 | 1,670.7 |
| **PRA (rad/s$^2$)** | 2,536.2 ±2,703.4 | 1,500.0 [838.2-3,852.2] | 8,822.3 | - | 3.5 to 15,764.2 | 230,791.1 |
| **$HIC_{15}$** | 8.1 ±14.1 | 3.0 [1.8-8.9] | 31.9 | - | 0.8 to 107.5 | 736.9 |
| **GSI** | 0.01 ±0.04 | 0.00 [0.00-0.01] | 0.08 | - | 0.00 to 0.28 | 1.31 |
| **HITsp** | 9.2 ±4.2 | 7.7 [7.1-9.5] | 15.6 | - | 6.5 to 38.1 | 836.6 |
| **GAMBIT** | 0.070 ±0.053 | 0.048 [0.034-0.093] | 0.194 | - | 0.014 to 0.233 | 6.30 |
| **ms** | 6.5 ±4.3 | 5.0 [4.0-8.0] | 19.0 | - | 2.0 to 24.0 | 596.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | - | 0.000 to 0.001 | 0.015 |
| **$RWE_{Rotational}$** | 0.101 ±0.276 | 0.000 [0.000-0.007] | 0.994 | - | 0.000 to 1.000 | 9.157 |
| **$RWE_{CP}$** | 0.025 ±0.114 | 0.000 [0.000-0.002] | 0.212 | - | 0.000 to 0.983 | 2.311 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

# Appendix IX: Supplemental data for Technical Report (Appendix II)

## Head impacts in a junior rugby league team measured with an instrumented patch: An exploratory analysis.

## Total, Match and Player Positional Group Impact Biomechanics Summaries

# Total Match Impact Biomechanics

**Table 1:** Summary of total match impact biomechanics for impacts to the head (n=1977) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 22.2 ±15.8 | 15.9 [12.4-24.6] | 57.0 | 84.6 | 10.0 to 122.8 | 43,864.1 |
| **PRA (rad/s²)** | 4,040.8 ±3,391.2 | 2,777.3 [1,785.7-5,040.5] | 11,383.8 | 16,105.8 | 88.9 to 22,928.0 | 7,988,660.7 |
| **$HIC_{15}$** | 26.2 ±60.2 | 6.3 [3.7-17.7] | 125.2 | 319.8 | 1.0 to 937.5 | 51,722.0 |
| **GSI** | 40.95 ±91.93 | 10.39 [6.38-27.23] | 190.28 | 475.33 | 1.61 to 1,311.85 | 80,959.12 |
| **HITsp** | 12.4 ±9.4 | 8.9 [7.7-12.4] | 30.8 | 55.3 | 6.5 to 118.8 | 24,514.8 |
| **GAMBIT** | 0.152 ±0.112 | 0.109 [0.079-0.183] | 0.396 | 0.565 | 0.034 to 0.780 | 301.3 |
| **ms** | 10.1 ±8.1 | 8.0 [5.0-12.0] | 29.0 | 39.0 | 1.0 to 44.0 | 19,879.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.027 | 0.655 |
| **$RWE_{Rotational}$** | 0.192 ±0.365 | 0.001 [0.000-0.079] | 1.000 | 1.000 | 0.000 to 1.000 | 378.9 |
| **$RWE_{CP}$** | 0.088 ±0.237 | 0.001 [0.000-0.008] | 0.808 | 0.998 | 0.000 to 1.000 | 173.7 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of total match impact biomechanics for impacts to the front of the head (n=508) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.7 ±15.7 | 18.6 [13.8-30.4] | 59.1 | 81.4 | 10.1 to 102.7 | 12,525.5 |
| **PRA (rad/s²)** | 4,915.0 ±3,512.6 | 3,685.8 [2,432.5-6,443.6] | 11,947.6 | 17,304.0 | 594.1 to 19,603.0 | 2,496,833.6 |
| **$HIC_{15}$** | 31.5 ±56.9 | 9.3 [4.7-28.1] | 147.7 | 290.7 | 1.5 to 471.2 | 15,995.9 |
| **GSI** | 49.6 ±90.7 | 14.6 [7.6-45.0] | 228.6 | 438.1 | 2.6 to 922.2 | 25,176.3 |
| **HITsp** | 12.4 ±9.5 | 8.6 [6.9-14.1] | 32.9 | 53.6 | 5.5 to 81.9 | 6,299.2 |
| **GAMBIT** | 0.182 ±0.117 | 0.139 [0.097-0.232] | 0.424 | 0.563 | 0.042 to 0.729 | 92.4 |
| **ms** | 11.8 ±8.8 | 9.0 [6.0-15.0] | 32.0 | 40.9 | 1.0 to 44.0 | 5,985.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.010 | 0.167 |
| **$RWE_{Rotational}$** | 0.261 ±0.405 | 0.006 [0.000-0.578] | 1.000 | 1.000 | 0.000 to 1.000 | 132.7 |
| **$RWE_{CP}$** | 0.117 ±0.265 | 0.002 [0.001-0.026] | 0.898 | 0.999 | 0.000 to 1.000 | 59.7 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of total match impact biomechanics for impacts to the back of the head (n=498) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.3 ±16.0 | 18.2 [13.4-29.1] | 57.7 | 85.1 | 10.1 to 104.7 | 12,083.5 |
| **PRA (rad/s²)** | 4,705.8 ±3,697.9 | 3,390.9 [2,075.2-6,033.0] | 12,631.1 | 16,101.8 | 494.4 to22,928.0 | 2,343,490.5 |
| **$HIC_{15}$** | 32.1 ±72.9 | 9.6 [4.6-26.4] | 129.5 | 328.8 | 1.0 to 937.5 | 15,985.6 |
| **GSI** | 48.7 ±105.4 | 14.7 [7.3-40.4] | 188.8 | 478.9 | 1.6 to 1,311.9 | 24,243.4 |
| **HITsp** | 12.4 ±9.3 | 9.2 [7.4-13.0] | 28.1 | 50.5 | 6.0 to 104.1 | 6,175.2 |
| **GAMBIT** | 0.172 ±0.115 | 0.133 [0.091-0.220] | 0.407 | 0.587 | 0.036 to 0.716 | 85.7 |
| **ms** | 11.0 ±7.7 | 9.0 [6.0-14.0] | 28.1 | 40.0 | 1.0 to 43.0 | 5,483.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.011 | 0.173 |
| **$RWE_{Rotational}$** | 0.246 ±0.401 | 0.003 [0.000-0.376] | 1.000 | 1.000 | 0.000 to 1.000 | 122.7 |
| **$RWE_{CP}$** | 0.121 ±0.275 | 0.002 [0.000-0.019] | 0.914 | 0.998 | 0.000 to 1.000 | 60.2 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of total match impact biomechanics for impacts to the side of the head (n=944) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.3 ±15.0 | 13.8 [11.8-19.4] | 52.7 | 85.4 | 10.0 to 122.8 | 18,252.4 |
| **PRA (rad/s$^2$)** | 3,122.7 ±2,798.0 | 2,161.3 [1,409.4-3,585.8] | 8,983.7 | 15,073.9 | 88.9 to 19,338.3 | 2,947,857.7 |
| **HIC$_{15}$** | 18.8 ±52.0 | 4.6 [3.1-9.3] | 96.6 | 339.5 | 1.0 to 530.4 | 17,744.9 |
| **GSI** | 30.0 ±81.1 | 8.2 [5.5-15.6] | 138.5 | 495.8 | 1.7 to 785. 9 | 28,355.2 |
| **HITsp** | 12.4 ±9.4 | 9.3 [8.6-11.6] | 30.2 | 64.7 | 7.6 to 85.5 | 11,705.6 |
| **GAMBIT** | 0.123 ±0.098 | 0.089 [0.068-0.133] | 0.327 | 0.541 | 0.034 to 0.780 | 116.0 |
| **ms** | 8.4 ±7.4 | 6.0 [4.0-10.0] | 24.8 | 38.0 | 1.0 to 44.0 | 7944.0 |
| **RWE$_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.027 | 0.294 |
| **RWE$_{Rotational}$** | 0.117 ±0.295 | 0.000 [0.000-0.005] | 0.996 | 1.000 | 0.000 to 1.000 | 110.3 |
| **RWE$_{CP}$** | 0.049 ±0.178 | 0.000 [0.000-0.002] | 0.389 | 0.996 | 0.000 to 1.000 | 45.8 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of total match impact biomechanics for impacts to the top of the head (n=27) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 37.1 ±22.0 | 34.5 [16.6-57.2] | 86.6 | - | 12.5 to 97.9 | 1,002.8 |
| **PRA (rad/s$^2$)** | 7,425.1 ±4,711.4 | 6,104.2 [3,602.9-10,795.5] | 17,896.2 | - | 1,487.0 to18,056.2 | 200,478.9 |
| **HIC$_{15}$** | 73.9 ±84.1 | 46.7 [6.0-115.9] | 258.6 | - | 3.0 to 258.9 | 1,995.6 |
| **GSI** | 117.9 ±133.5 | 64.0 [10.1-165.6] | 399.9 | - | 6.8 to 400.1 | 3,184.2 |
| **HITsp** | 12.4 ±9.5 | 10.1 [4.0-20.5] | 32.3 | - | 3.0 to 33.5 | 334.8 |
| **GAMBIT** | 0.268 ±0.148 | 0.242 [0.133-0.358] | 0.632 | - | 0.079 to 0.654 | 7.2 |
| **ms** | 17.3 ±10.8 | 13.0 [8.0-28.0] | 36.6 | - | 2.0 to 37.0 | 467.0 |
| **RWE$_{Linear}$** | 0.001 ±0.002 | 0.000 [0.000-0.001] | 0.005 | - | 0.000 to 0.008 | 0.021 |
| **RWE$_{Rotational}$** | 0.490 ±0.476 | 0.420 [0.005-1.000] | 1.000 | - | 0.000 to 1.000 | 13.2 |
| **RWE$_{CP}$** | 0.299 ±0.401 | 0.029 [0.001-0.629] | 0.999 | - | 0.000 to 0.999 | 8.1 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); HIC$_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; RWE$_{Linear}$ = Risk Weighted Exposure Linear accelerations; RWE$_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; RWE$_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

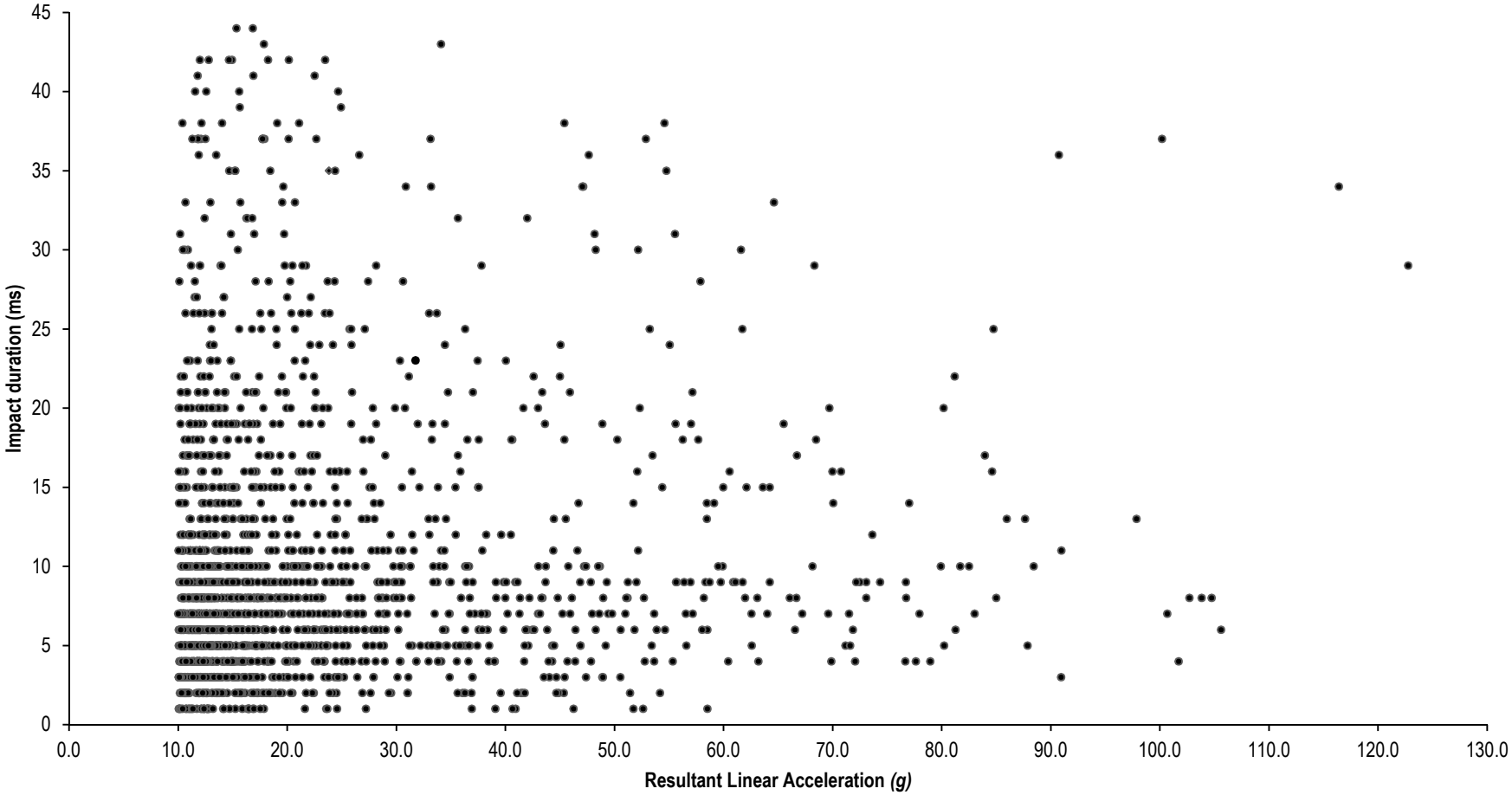


**Figure 1:** Resultant linear accelerations (*g*) by impact duration in milliseconds (ms) for all matches competed in a U11 junior rugby league team over a competitive season

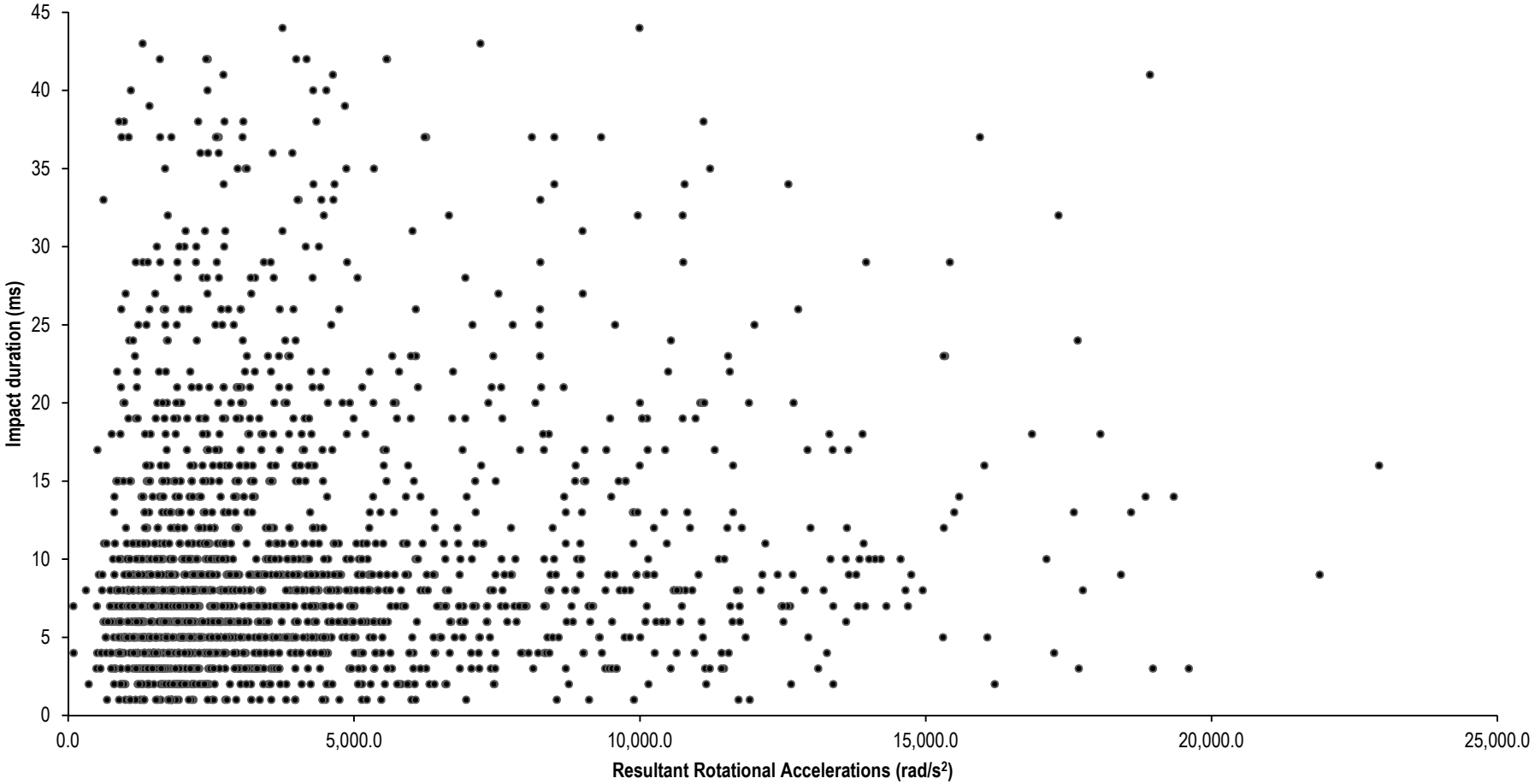


**Figure 2:** Resultant rotational accelerations (rad/s$^2$) by impact duration in milliseconds (ms) for all matches competed in a U11 junior rugby league team over a competitive season

# Individual Match Impact Biomechanics

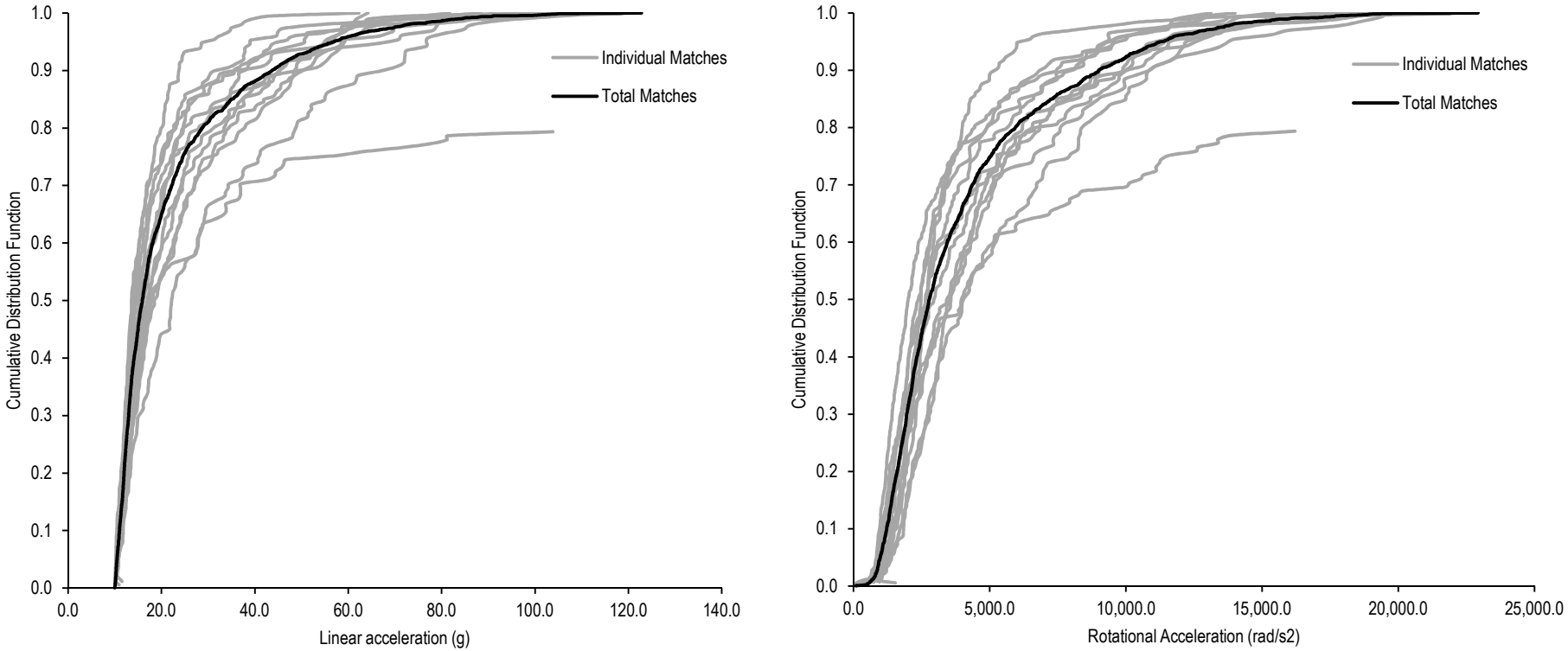


**Figure 3:** Cumulative distribution functions for resultant linear (left) and rotational (right) acceleration magnitudes of impacts collected during matches for junior rugby league season 2014.

**Table 1:** Summary of competition match 1 impact biomechanics for impacts to the head (n=86) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.7 ±12.8 | 16.3 [12.4-22.5] | 57.4 | - | 10.3 to 64.2 | 1,781.3 |
| **PRA (rad/s²)** | 3,575.3 ±2,710.2 | 2,720.7 [1,735.0-4,271.0] | 9,397.7 | - | 735.9 to 13,117.6 | 307,476.8 |
| **$HIC_{15}$** | 19.3 ±32.7 | 8.0 [4.2-14.8] | 112.7 | - | 1.4 to 166.9 | 1,656.5 |
| **GSI** | 28.7 ±47.4 | 12.1 [7.0-23.5] | 173.2 | - | 2.8 to 237.9 | 2,470.8 |
| **HITsp** | 20.0 ±15.4 | 14.3 [11.6-19.5] | 67.4 | - | 9.3 to 80.0 | 1,720.0 |
| **GAMBIT** | 0.140 ±0.085 | 0.113 [0.089-0.159] | 0.361 | - | 0.054 to 0.463 | 12.1 |
| **ms** | 10.3 ±6.9 | 9.0 [5.0-14.0] | 26.7 | - | 2.0 to 34.0 | 886.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.001 | - | 0.000 to 0.001 | 0.019 |
| **$RWE_{Rotational}$** | 0.159 ±0.343 | 0.001 [0.000-0.018] | 0.998 | - | 0.000 to 1.000 | 13.7 |
| **$RWE_{CP}$** | 0.057 ±0.181 | 0.001 [0.000-0.004] | 0.469 | - | 0.000 to 0.952 | 4.9 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of competition match 2 impact biomechanics for impacts to the head (N=130) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 26.0 ±18.3 | 18.8 [13.5-31.9] | 71.1 | 96.6 | 10.1 to 100.7 | 3,384.1 |
| **PRA (rad/s²)** | 5,174.0 ±4,104.5 | 3,575.2 [2,515.9-6,718.1] | 14,563.2 | 19,520.9 | 308.6 to 19,603.0 | 672,617.3 |
| **$HIC_{15}$** | 41.5 ±83.6 | 9.6 [5.0-31.7] | 267.0 | 457.3 | 1.5 to 471.2 | 5,398.1 |
| **GSI** | 65.1 ±130.8 | 14.5 [8.0-48.8] | 407.7 | 814.1 | 2.1 to 922.2 | 8,463.7 |
| **HITsp** | 12.4 ±9.6 | 8.4 [7.2-13.3] | 37.1 | 58.1 | 5.8 to 62.4 | 1,612.0 |
| **GAMBIT** | 0.194 ±0.140 | 0.140 [0.101-0.234] | 0.505 | 0.709 | 0.034 to 0.729 | 25.2 |
| **ms** | 12.6 ±9.4 | 9.0 [6.0-17.0] | 35.5 | 40.8 | 1.0 to 42.0 | 1634.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.002 | 0.008 | 0.000 to 0.009 | 0.055 |
| **$RWE_{Rotational}$** | 0.268 ±0.419 | 0.005 [0.001-0.680] | 1.000 | 1.000 | 0.000 to 1.000 | 34.9 |
| **$RWE_{CP}$** | 0.138 ±.299 | 0.002 [0.001-0.048] | 0.987 | 1.000 | 0.000 to1.000 | 17.9 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of competition match 3 impact biomechanics for impacts to the head (N=190) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 23.3 ±14.9 | 17.0 [13.2-28.4] | 57.1 | 75.5 | 10.1 to 100.2 | 4,428.0 |
| **PRA (rad/s²)** | 4,386.7 ±3,519.9 | 2,967.6 [1,949.6-5,431.6] | 11,538.3 | 15,772.6 | 88.9 to 17,656.2 | 833,464.9 |
| **$HIC_{15}$** | 26.4 ±44.4 | 7.7 [4.1-24.5] | 138.4 | 242.7 | 1.2 to 247.0 | 5,021.2 |
| **GSI** | 41.8 ±70.2 | 11.4 [6.6-39.8] | 182.6 | 399.7 | 2.0 to 400.1 | 7,943.0 |
| **HITsp** | 12.4 ±9.5 | 8.0 [6.8-13.4] | 34.7 | 49.6 | 5.5 to 53.3 | 2,356.0 |
| **GAMBIT** | 0.162 ±0.111 | 0.110 [0.086-0.212] | 0.375 | 0.547 | 0.040 to 0.598 | 30.9 |
| **ms** | 11.3 ±9.0 | 9.0 [6.0-14.0] | 35.9 | 40.2 | 1.0 to 42.0 | 2153.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.009 | 0.056 |
| **$RWE_{Rotational}$** | 0.227 ±0.396 | 0.001 [0.000-0.125] | 1.000 | 1.000 | 0.000 to 1.000 | 43.1 |
| **$RWE_{CP}$** | 0.109 ±0.255 | 0.001 [0.000-0.016] | 0.836 | 0.998 | 0.000 to 0.999 | 20.7 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of competition match 4 impact biomechanics for impacts to the head (N=136) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.1 ±16.8 | 17.4 [13.4-25.9] | 60.1 | 89.8 | 10.1 to 90.9 | 3,280.7 |
| **PRA (rad/s²)** | 4,491.2 ±3,320.3 | 3,467.4 [2,174.1-5,466.0] | 12,943.0 | 14,975.6 | 951.7 to 15,421.9 | 610,806.7 |
| **$HIC_{15}$** | 29.8 ±53.6 | 8.0 [4.4-20.8] | 138.0 | 311.5 | 1.5 to 372.1 | 4,055.9 |
| **GSI** | 48.9 ±92.2 | 13.6 [8.0-33.6] | 248.6 | 593.9 | 3.1 to 722.8 | 6,652.9 |
| **HITsp** | 20.0 ±15.3 | 13.8 [11.2-19.2] | 56.0 | 91.8 | 9.9 to 108.3 | 2,720.0 |
| **GAMBIT** | 0.172 ±0.119 | 0.128 [0.090-0.205] | 0.487 | 0.630 | 0.049 to 0.648 | 23.5 |
| **ms** | 11.3 ±9.2 | 8.0 [5.0-14.0] | 37.2 | 43.3 | 1.0 to 44.0 | 1534.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.006 | 0.048 |
| **$RWE_{Rotational}$** | 0.211 ±0.375 | 0.004 [0.000-0.161] | 1.000 | 1.000 | 0.000 to 1.000 | 28.7 |
| **$RWE_{CP}$** | 0.111 ±0.268 | 0.002 [0.000-0.011] | 0.930 | 0.991 | 0.000 to 0.997 | 15.1 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of competition match 5 impact biomechanics for impacts to the head (N=138) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 31.1 ±22.4 | 22.3 [14.1-41.1] | 77.2 | 110.7 | 10.2 to 116.4 | 4,296.8 |
| **PRA (rad/s²)** | 5,433.7 ±3,863.7 | 4,084.3 [2,407.3-8,031.9] | 12,599.9 | 19,928.5 | 627.3 to 21,891.3 | 749,849.9 |
| **$HIC_{15}$** | 50.4 ±85.1 | 12.8 [5.3-50.9] | 273.7 | 438.0 | 1.4 to 449.4 | 6,949.5 |
| **GSI** | 78.8 ±133.0 | 20.8 [8.7-89.5] | 420.6 | 723.2 | 3.3 to 778.9 | 10,871.4 |
| **HITsp** | 19.0 ±14.3 | 13.4 [9.6-23.1] | 55.9 | 78.0 | 7.8 to 79.9 | 2,622.0 |
| **GAMBIT** | 0.208 ±0.137 | 0.156 [0.105-0.274] | 0.472 | 0.706 | 0.044 to 0.710 | 28.7 |
| **ms** | 12.2 ±8.6 | 9.0 [6.0-16.0] | 31.1 | 37.6 | 1.0 to 38.0 | 1682.0 |
| **$RWE_{Linear}$** | 0.001 ±0.002 | 0.000 [0.000-0.000] | 0.003 | 0.016 | 0.000 to 0.020 | 0.100 |
| **$RWE_{Rotational}$** | 0.337 ±0.439 | 0.013 [0.000-0.966] | 1.000 | 1.000 | 0.000 to 1.000 | 46.6 |
| **$RWE_{CP}$** | 0.168 ±0.311 | 0.004 [0.001-0.112] | 0.954 | 1.000 | 0.000 to 1.000 | 23.2 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 6:** Summary of competition match 6 impact biomechanics for impacts to the head (n=193) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.6 ±15.4 | 14.7 [12.0-23.2] | 59.1 | 80.7 | 10.0 to 88.4 | 4,177.5 |
| **PRA (rad/s²)** | 4,160.5 ±3,650.2 | 2,649.4 [1,605.8-5,650.9] | 12,174.7 | 16,097.2 | 500.2 to 17,245.7 | 802,984.6 |
| **$HIC_{15}$** | 27.5 ±56.7 | 5.6 [3.7-18.1] | 149.2 | 339.1 | 1.2 to 376.6 | 5,302.7 |
| **GSI** | 43.3 ±88.7 | 10.1 [6.0-26.5] | 246.5 | 509.5 | 2.2 to 606.7 | 8,355.0 |
| **HITsp** | 12.4 ±9.6 | 8.3 [7.4-12.0] | 34.4 | 59.8 | 6.6 to 63.5 | 2,393.2 |
| **GAMBIT** | 0.155 ±0.122 | 0.101 [0.074-0.188] | 0.413 | 0.598 | 0.042 to 0.780 | 29.9 |
| **ms** | 10.5 ±9.0 | 8.0 [5.0-12.0] | 33.6 | 43.0 | 1.0 to 43.0 | 2032.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.005 | 0.053 |
| **$RWE_{Rotational}$** | 0.220 ±0.385 | 0.001 [0.000-0.222] | 1.000 | 1.000 | 0.000 to 1.000 | 42.4 |
| **$RWE_{CP}$** | 0.109 ±0.268 | 0.001 [0.000-0.011] | 0.889 | 0.996 | 0.000 to 0.999 | 21.0 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 7:** Summary of competition match 7 impact biomechanics for impacts to the head (n=255) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.9 ±15.2 | 14.5 [12.1-20.5] | 47.0 | 101.3 | 10.0 to 122.8 | 5,066.4 |
| **PRA (rad/s$^2$)** | 3,146.0 ±2,928.4 | 2,010.6 [1,300.8-3,776.1] | 9,614.9 | 14,893.0 | 504.5 to 17,592.4 | 802,238.4 |
| **$HIC_{15}$** | 18.9 ±47.0 | 4.9 [3.4-11.2] | 102.5 | 338.2 | 1.2 to 354.2 | 4,815.2 |
| **GSI** | 29.3 ±67.6 | 9.1 [6.5-17.8] | 146.6 | 458.8 | 1.7 to 504.3 | 7,468.5 |
| **HITsp** | 12.4 ±9.5 | 9.1 [8.3-11.8] | 30.2 | 68.3 | 7.4 to 74.2 | 3,162.0 |
| **GAMBIT** | 0.124 ±0.096 | 0.086 [0.067-0.139] | 0.353 | 0.515 | 0.037 to 0.561 | 31.7 |
| **ms** | 8.6 ±7.0 | 6.0 [5.0-10.0] | 26.0 | 35.3 | 1.0 to 42.0 | 2205.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.001 | 0.010 | 0.000 to 0.027 | 0.097 |
| **$RWE_{Rotational}$** | 0.132 ±0.315 | 0.000 [0.000-0.006] | 0.999 | 1.000 | 0.000 to 1.000 | 33.6 |
| **$RWE_{CP}$** | 0.056 ±0.190 | 0.000 [0.000-0.002] | 0.528 | 0.987 | 0.000 to 1.000 | 14.4 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 8:** Summary of competition match 8 impact biomechanics for impacts to the head (n=114) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 22.2 ±17.3 | 14.8 [11.8-27.7] | 65.2 | 100.4 | 10.1 to 103.8 | 2,532.4 |
| **PRA (rad/s$^2$)** | 4,199.5 ±3,577.2 | 2,776.9 [1,670.5-5,135.6] | 12,599.5 | 15,865.0 | 857.6 to 16,209.0 | 478,744.4 |
| **$HIC_{15}$** | 28.3 ±64.4 | 5.9 [2.8-22.5] | 137.7 | 437.7 | 1.0 to 457.0 | 3,231.5 |
| **GSI** | 42.4 ±98.0 | 9.1 [4.6-33.4] | 191.9 | 692.8 | 1.6 to 731.4 | 4,830.5 |
| **HITsp** | 20.0 ±15.0 | 14.0 [12.3-21.2] | 46.6 | 103.1 | 11.0 to 105.9 | 2,280.0 |
| **GAMBIT** | 0.152 ±0.112 | 0.100 [0.074-0.200] | 0.371 | 0.643 | 0.040 to 0.667 | 17.3 |
| **ms** | 9.8 ±8.3 | 7.0 [4.0-14.0] | 26.0 | 43.1 | 1.0 to 44.0 | 1114.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.002 | 0.010 | 0.000 to 0.011 | 0.043 |
| **$RWE_{Rotational}$** | 0.211 ±0.387 | 0.001 [0.000-0.086] | 1.000 | 1.000 | 0.000 to 1.000 | 24.1 |
| **$RWE_{CP}$** | 0.110 ±0.266 | 0.001 [0.000-0.010] | 0.917 | 0.996 | 0.000 to 0.998 | 12.5 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 9:** Summary of competition match 9 impact biomechanics for impacts to the head (n=148) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.4 ±11.8 | 13.6 [11.9-20.0] | 42.1 | 78.1 | 10.1 to 81.7 | 2,723.8 |
| **PRA (rad/s$^2$)** | 3,530.4 ±2,837.9 | 2,474.9 [1,948.3-3,722.0] | 9,721.9 | 16,459.1 | 735.6 to 18,923.2 | 522,504.0 |
| **$HIC_{15}$** | 13.9 ±27.8 | 4.6 [3.0-10.8] | 59.0 | 174.0 | 1.3 to 176.3 | 2,051.5 |
| **GSI** | 21.6 ±42.9 | 7.3 [5.0-16.9] | 87.0 | 273.8 | 2.4 to 283.8 | 3,194.8 |
| **HITsp** | 12.4 ±9.7 | 8.9 [7.9-12.5] | 28.6 | 65.3 | 6.7 to 67.8 | 1,835.2 |
| **GAMBIT** | 0.125 ±0.083 | 0.097 [0.076-0.128] | 0.318 | 0.444 | 0.043 to 0.459 | 18.6 |
| **ms** | 8.1 ±6.8 | 6.0 [4.0-9.0] | 21.6 | 38.6 | 1.0 to 41.0 | 1206.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | 0.003 | 0.000 to 0.003 | 0.029 |
| **$RWE_{Rotational}$** | 0.130 ±0.306 | 0.001 [0.000-0.006] | 0.999 | 1.000 | 0.000 to 1.000 | 19.3 |
| **$RWE_{CP}$** | 0.052 ±0.185 | 0.001 [0.000-0.002] | 0.416 | 0.991 | 0.000 to 1.000 | 7.7 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 10:** Summary of competition match 10 impact biomechanics for impacts to the head (n=220) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 25.0 ±16.8 | 18.1 [13.1-31.0] | 59.8 | 89.1 | 10.1 to 104.7 | 5,496.2 |
| **PRA (rad/s²)** | 4,475.4 ±3,668.1 | 3,545.3 [1,882.0-5,861.7] | 11,461.3 | 18,679.9 | 494.4 to 22,928.0 | 984,591.1 |
| **$HIC_{15}$** | 35.4 ±95.8 | 8.2 [4.1-29.5] | 130.1 | 672.5 | 1.1 to 937.5 | 7,796.0 |
| **GSI** | 55.6 ±138.7 | 14.5 [6.8-45.7] | 247.5 | 934.8 | 1.7 to 1,311.9 | 12,235.7 |
| **HITsp** | 12.4 ±9.3 | 9.4 [7.7-13.4] | 27.1 | 65.7 | 6.4 to 86.1 | 2,728.0 |
| **GAMBIT** | 0.173 ±0.125 | 0.138 [0.080-0.223] | 0.448 | 0.680 | 0.036 to 0.716 | 38.08 |
| **ms** | 11.2 ±8.7 | 8.0 [5.0-14.8] | 33.0 | 41.2 | 1.0 to 42.0 | 2469.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.011 | 0.081 |
| **$RWE_{Rotational}$** | 0.232 ±0.387 | 0.004 [0.000-0.259] | 1.000 | 1.000 | 0.000 to 1.000 | 51.1 |
| **$RWE_{CP}$** | 0.096 ±0.242 | 0.002 [0.000-0.020] | 0.851 | 1.000 | 0.000 to 1.000 | 21.1 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 11:** Summary of competition match 11 impact biomechanics for impacts to the head (n=173) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.5 ±14.2 | 15.4 [12.0-22.4] | 50.7 | 89.6 | 10.2 to 102.7 | 3,546.4 |
| **PRA (rad/s²)** | 3,772.8 ±3,262.6 | 2,655.5 [1,580.7-4,545.6] | 10,033.3 | 18,558.6 | 597.3 to 18,973.3 | 652,687.6 |
| **$HIC_{15}$** | 21.0 ±45.6 | 6.1 [3.6-12.6] | 111.7 | 299.4 | 1.0 to 314.1 | 3,624.5 |
| **GSI** | 32.2 ±70.9 | 9.3 [6.4-19.2] | 156.6 | 467.3 | 1.9 to 496.7 | 5,577.5 |
| **HITsp** | 12.4 ±9.6 | 9.0 [7.8-11.7] | 31.3 | 66.3 | 6.8 to 67.0 | 2,145.2 |
| **GAMBIT** | 0.138 ±0.098 | 0.103 [0.074-0.154] | 0.322 | 0.549 | 0.039 to 0.698 | 23.9 |
| **ms** | 8.9 ±6.7 | 7.0 [5.0-10.0] | 21.3 | 38.8 | 1.0 to 41.0 | 1538.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.010 | 0.048 |
| **$RWE_{Rotational}$** | 0.169 ±0.343 | 0.001 [0.000-0.030] | 0.999 | 1.000 | 0.000 to 1.000 | 29.2 |
| **$RWE_{CP}$** | 0.061 ±0.193 | 0.001 [0.000-0.004] | 0.481 | 1.000 | 0.000 to 1.000 | 10.6 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 12:** Summary of competition match 12 impact biomechanics for impacts to the head (n=194) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 16.2 ±7.0 | 14.1 [11.8-18.5] | 31.4 | 44.5 | 10.0 to 62.3 | 3,150.5 |
| **PRA (rad/s²)** | 2,941.7 ±2,121.0 | 2,391.2 [1,539.7-3,738.2] | 6,489.3 | 13,000.1 | 95.1 to 14,007.8 | 570,695.0 |
| **$HIC_{15}$** | 9.4 ±17.3 | 4.6 [3.2-8.8] | 26.9 | 100.8 | 1.6 to 190.6 | 1,819.5 |
| **GSI** | 14.9 ±24.5 | 8.2 [6.0-14.1] | 49.8 | 171.7 | 2.4 to 245.1 | 2,895.5 |
| **HITsp** | 12.4 ±9.4 | 9.6 [7.9-13.6] | 25.7 | 61.2 | 6.1 to 93.1 | 2,405.6 |
| **GAMBIT** | 0.112 ±0.060 | 0.095 [0.076-0.130] | 0.242 | 0.410 | 0.036 to 0.417 | 21.7 |
| **ms** | 7.4 ±5.1 | 6.0 [4.0-9.0] | 19.0 | 26.5 | 1.0 to 35.0 | 1426.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | 0.001 | 0.000 to 0.001 | 0.027 |
| **$RWE_{Rotational}$** | 0.063 ±0.207 | 0.000 [0.000-0.006] | 0.610 | 1.000 | 0.000 to 1.000 | 12.3 |
| **$RWE_{CP}$** | 0.024 ±0.128 | 0.001 [0.000-0.002] | 0.026 | 0.915 | 0.000 to 0.959 | 4.6 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

# Male, Female and Player Positional Group Impact Biomechanics

**Table 1:** Summary of competition match impact biomechanics of male participants for impacts to the head (n=1,579) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.7 ±15.6 | 15.4 [12.3-24.2] | 55.5 | 83.2 | 10.0 to 122.8 | 34,309.6 |
| **PRA (rad/s²)** | 3,948.9 ±3,368.1 | 2,674.9 [1,699.8-4,877.5] | 11,224.8 | 15,963.7 | 88.9 to 21,891.3 | 6,235,351.8 |
| **$HIC_{15}$** | 25.0 ±56.5 | 6.0 [3.6-16.7] | 120.8 | 311.4 | 1.0 to 710.3 | 39,410.2 |
| **GSI** | 39.2 ±87.5 | 10.1 [6.3-26.1] | 182.4 | 475.3 | 1.6 to 974.3 | 61,897.7 |
| **HITsp** | 12.4 ±9.4 | 8.8 [7.7-12.3] | 30.6 | 57.5 | 6.7 to 97.2 | 19,579.6 |
| **GAMBIT** | 0.149 ±0.111 | 0.106 [0.076-0.180] | 0.383 | 0.563 | 0.034 to 0.780 | 235.7 |
| **ms** | 9.9 ±8.0 | 7.0 [5.0-12.0] | 29.0 | 38.0 | 1.0 to 44.0 | 15566.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.027 | 0.514 |
| **$RWE_{Rotational}$** | 0.188 ±0.364 | 0.001 [0.000-0.058] | 1.000 | 1.000 | 0.000 to 1.000 | 297.0 |
| **$RWE_{CP}$** | 0.086 ±0.235 | 0.001 [0.000-0.007] | 0.807 | 0.998 | 0.000 to 1.000 | 136.0 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of competition match impact biomechanics of male participants for impacts to the front head (n=389) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.5 ±15.7 | 18.4 [13.6-30.3] | 59.0 | 78.9 | 10.1 to 102.7 | 9,534.2 |
| **PRA (rad/s²)** | 4,946.2 ±3,547.3 | 3,585.0 [2,423.0-6,604.5] | 12,817.3 | 17,365.9 | 594.1 to 19,603.0 | 1,924,070.9 |
| **$HIC_{15}$** | 31.0 ±57.0 | 9.1 [4.5-27.4] | 141.4 | 292.6 | 1.5 to 471.2 | 12,076.2 |
| **GSI** | 49.5 ±93.1 | 14.1 [7.6-44.5] | 223.8 | 440.8 | 2.6 to 922.2 | 19,241.6 |
| **HITsp** | 12.4 ±9.5 | 8.6 [7.0-14.1] | 32.7 | 53.9 | 5.6 to 81.1 | 4,823.6 |
| **GAMBIT** | 0.183 ±0.119 | 0.138 [0.097-0.232] | 0.418 | 0.588 | 0.042 to 0.729 | 71.0 |
| **ms** | 11.7 ±9.1 | 8.0 [6.0-15.0] | 32.5 | 41.1 | 1.0 to 44.0 | 4542.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.010 | 0.129 |
| **$RWE_{Rotational}$** | 0.270 ±0.409 | 0.005 [0.000-0.661] | 1.000 | 1.000 | 0.000 to 1.000 | 105.0 |
| **$RWE_{CP}$** | 0.119 ±0.267 | 0.002 [0.001-0.036] | 0.916 | 0.999 | 0.000 to 1.000 | 46.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of competition match impact biomechanics of male participants for impacts to the back of the head (n=398) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.2 ±15.8 | 17.9 [13.3-29.9] | 55.7 | 84.0 | 10.1 to 103.8 | 9,639.0 |
| **PRA (rad/s²)** | 4,692.3 ±3,653.1 | 3,524.7 [2,020.5-6,172.9] | 12,515.9 | 16,049.6 | 615.2 to 21,891.3 | 1,867,549.7 |
| **$HIC_{15}$** | 31.0 ±62.8 | 9.4 [4.5-29.0] | 122.3 | 328.8 | 1.0 to 710.3 | 12,343.3 |
| **GSI** | 47.2 ±92.0 | 14.2 [7.1-42.8] | 184.5 | 478.9 | 1.6 to 974.3 | 18,782.3 |
| **HITsp** | 12.4 ±9.4 | 9.0 [7.1-13.6] | 29.0 | 54.4 | 5.7 to 90.2 | 4,935.2 |
| **GAMBIT** | 0.172 ±0.115 | 0.133 [0.089-0.228] | 0.392 | 0.587 | 0.036 to 0.700 | 68.5 |
| **ms** | 10.9 ±7.5 | 9.0 [6.0-14.0] | 27.1 | 39.0 | 1.0 to 42.0 | 4357.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.011 | 0.133 |
| **$RWE_{Rotational}$** | 0.254 ±0.407 | 0.004 [0.000-0.444] | 1.000 | 1.000 | 0.000 to 1.000 | 101.2 |
| **$RWE_{CP}$** | 0.123 ±0.274 | 0.002 [0.000-0.026] | 0.909 | 0.994 | 0.000 to 1.000 | 48.9 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of competition match impact biomechanics of male participants for impacts to the side of the head (n=772) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.7 ±14.5 | 13.6 [11.8-18.2] | 48.5 | 83.8 | 10.0 to 122.8 | 14,404.8 |
| **PRA (rad/s²)** | 2,986.4 ±2,752.8 | 2,034.5 [1,373.1-3,420.6] | 8,905.5 | 14,901.7 | 88.9 to 18,973.3 | 2,305,462.5 |
| **$HIC_{15}$** | 17.6 ±50.6 | 4.5 [3.1-8.5] | 85.5 | 335.7 | 1.0 to 530.4 | 13,599.5 |
| **GSI** | 28.0 ±78.6 | 8.1 [5.6-14.2] | 128.8 | 498.5 | 1.7 to 785.9 | 21,647.3 |
| **HITsp** | 12.4 ±9.4 | 9.4 [8.8-11.5] | 27.7 | 65.8 | 7.8 to 88.0 | 9,572.8 |
| **GAMBIT** | 0.118 ±0.094 | 0.086 [0.068-0.123] | 0.310 | 0.530 | 0.034 to 0.780 | 91.1 |
| **ms** | 8.2 ±7.1 | 6.0 [4.0-9.0] | 24.0 | 37.3 | 1.0 to 42.0 | 6334.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.027 | 0.235 |
| **$RWE_{Rotational}$** | 0.106 ±0.285 | 0.000 [0.000-0.003] | 0.995 | 1.000 | 0.000 to 1.000 | 81.7 |
| **$RWE_{CP}$** | 0.046 ±0.175 | 0.000 [0.000-0.001] | 0.348 | 0.995 | 0.000 to 1.000 | 35.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of competition match impact biomechanics of male participants for impacts to the top of the head (n=20) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th* | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 36.6 ±22.7 | 34.1 [15.3-54.6] | 96.3 | - | 12.5 to 97.9 | 731.6 |
| **PRA (rad/s²)** | 6,913.4 ±4,473.1 | 5,782.5 [3,372.5-9,886.7] | 17,510.8 | - | 1,487.0 to 17,656.2 | 138,268.7 |
| **$HIC_{15}$** | 69.6 ±82.6 | 40.5 [5.8-107.3] | 258.8 | - | 4.5 to 258.9 | 1,391.1 |
| **GSI** | 111.3 ±129.7 | 59.9 [10.5-164.5] | 398.8 | - | 7.3 to 399.7 | 2,226.5 |
| **HITsp** | 12.4 ±9.4 | 9.6 [4.4-19.8] | 33.9 | - | 3.4 to 34.2 | 248.0 |
| **GAMBIT** | 0.253 ±0.133 | 0.242 [0.131-0.352] | 0.589 | - | 0.079 to 0.598 | 5.1 |
| **ms** | 16.7 ±11.0 | 12.5 [7.3-27.8] | 36.9 | - | 2.0 to 37.0 | 333.0 |
| **$RWE_{Linear}$** | 0.001 ±0.002 | 0.000 [0.000-0.001] | 0.008 | - | 0.000 to 0.008 | 0.016 |
| **$RWE_{Rotational}$** | 0.458 ±0.468 | 0.293 [0.005-0.999] | 1.000 | - | 0.000 to 1.000 | 9.2 |
| **$RWE_{CP}$** | 0.262 ±0.387 | 0.024 [0.001-0.523] | 0.998 | - | 0.000 to 0.999 | 5.2 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability. * = unable to calculate

**Table 6:** Summary of competition match impact biomechanics of female participants for impacts to the head (n=398) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.0 ±16.8 | 17.6 [13.1-27.4] | 63.1 | 85.0 | 10.1 to 104.7 | 9,554.6 |
| **PRA (rad/s²)** | 4,405.3 ±3,461.8 | 3,262.2 [2,129.0-5,487.4] | 11,637.2 | 18,064.9 | 494.4 to 22,928.0 | 1,753,308.9 |
| **$HIC_{15}$** | 30.9 ±72.9 | 8.2 [4.2-21.4] | 165.3 | 372.3 | 1.4 to 937.5 | 12,311.9 |
| **GSI** | 47.9 ±107.6 | 12.8 [6.6-34.4] | 255.1 | 495.2 | 2.5 to 1,311.9 | 19,061.4 |
| **HITsp** | 12.4 ±9.3 | 9.0 [7.6-12.3] | 31.8 | 52.7 | 6.3 to 103.4 | 4,935.2 |
| **GAMBIT** | 0.165 ±0.115 | 0.128 [0.089-0.196] | 0.429 | 0.648 | 0.040 to 0.716 | 65.6 |
| **ms** | 10.8 ±8.5 | 8.0 [5.0-15.0] | 28.0 | 42.0 | 1.0 to 44.0 | 4313.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.011 | 0.141 |
| **$RWE_{Rotational}$** | 0.206 ±0.368 | 0.002 [0.000-0.169] | 1.000 | 1.000 | 0.000 to 1.000 | 81.8 |
| **$RWE_{CP}$** | 0.095 ±0.247 | 0.001 [0.000-0.011] | 0.814 | 0.999 | 0.000 to 1.000 | 37.7 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 7:** Summary of competition match impact biomechanics of female participants for impacts to the front of the head (n=119) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 25.1 ±15.6 | 19.3 [14.6-31.1] | 60.4 | 82.3 | 10.1 to 82.5 | 2,991.4 |
| **PRA (rad/s²)** | 4,813.1 ±3,409.5 | 3,870.7 [2,457.7-5,577.8] | 11,622.1 | 18,560.8 | 885.1 to 18,923.2 | 572,762.7 |
| **$HIC_{15}$** | 32.9 ±57.0 | 9.5 [5.4-32.7] | 170.4 | 369.2 | 1.7 to 407.4 | 3,919.7 |
| **GSI** | 49.9 ±82.6 | 15.1 [8.3-60.8] | 258.0 | 507.0 | 3.2 to 548.5 | 5,934.7 |
| **HITsp** | 12.4 ±9.6 | 8.6 [6.8-14.0] | 34.9 | 59.8 | 5.3 to 64.1 | 1,475.6 |
| **GAMBIT** | 0.180 ±0.109 | 0.142 [0.102-0.240] | 0.442 | 0.558 | 0.065 to 0.564 | 21.39 |
| **ms** | 12.1 ±7.9 | 9.0 [7.0-16.0] | 28.0 | 41.2 | 2.0 to 42.0 | 1443.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.004 | 0.038 |
| **$RWE_{Rotational}$** | 0.232 ±0.391 | 0.008 [0.000-0.200] | 1.000 | 1.000 | 0.000 to 1.000 | 27.7 |
| **$RWE_{CP}$** | 0.111 ±0.260 | 0.002 [0.001-0.018] | 0.796 | 1.000 | 0.000 to 1.000 | 13.3 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 8:** Summary of competition match impact biomechanics of female participants for impacts to the back of the head (n=100) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.4 ±16.7 | 19.0 [14.3-27.7] | 61.7 | 104.5 | 10.2 to 104.7 | 2,444.5 |
| **PRA (rad/s²)** | 4,759.4 ±3,889.8 | 3,300.5 [2,171.4-5,624.5] | 13,863.3 | 22,859.5 | 494.4 to 22,928.0 | 475,940.9 |
| **$HIC_{15}$** | 36.4 ±104.1 | 11.0 [5.1-21.7] | 168.2 | 931.3 | 1.4 to 937.5 | 3,642.3 |
| **GSI** | 54.6 ±147.6 | 15.8 [7.9-36.8] | 282.4 | 1,303.3 | 2.5 to 1,311.9 | 5,461.1 |
| **HITsp** | 12.4 ±9.4 | 9.6 [8.1-12.0] | 29.1 | 81.5 | 6.6 to 81.9 | 1,240.0 |
| **GAMBIT** | 0.171 ±0.117 | 0.134 [0.099-0.190] | 0.457 | 0.714 | 0.040 to 0.716 | 17.1 |
| **ms** | 11.3 ±8.5 | 9.0 [6.0-14.0] | 35.7 | 43.0 | 1.0 to 43.0 | 1126.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.011 | 0.000 to 0.011 | 0.040 |
| **$RWE_{Rotational}$** | 0.216 ±0.376 | 0.003 [0.000-0.208] | 1.000 | 1.000 | 0.000 to 1.000 | 21.6 |
| **$RWE_{CP}$** | 0.113 ±0.280 | 0.001 [0.000-0.012] | 0.979 | 1.000 | 0.000 to 1.000 | 11.3 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 9:** Summary of competition match impact biomechanics of female participants for impacts to the side of the head (n=172) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 22.4 ±17.1 | 15.0 [12.2-25.1] | 66.3 | 88.7 | 10.1 to 91.0 | 3,847.6 |
| **PRA (rad/s$^2$)** | 3,734.9 ±2,923.0 | 2,764.1 [1,741.0-4,943.2] | 10,209.7 | 16,479.3 | 636.6 to 19,338.3 | 642,395.1 |
| **$HIC_{15}$** | 24.1 ±57.6 | 5.4 [3.4-14.2] | 120.9 | 378.4 | 1.4 to 395.5 | 4,145.4 |
| **GSI** | 39.0 ±91.1 | 9.1 [5.3-24.4] | 238.0 | 556.2 | 2.5 to 722.8 | 6,707.9 |
| **HITsp** | 12.4 ±9.3 | 8.9 [7.9-12.4] | 33.0 | 62.7 | 6.9 to 67.8 | 2,132.8 |
| **GAMBIT** | 0.145 ±0.109 | 0.104 [0.072-0.172] | 0.410 | 0.653 | 0.040 to 0.664 | 24.9 |
| **ms** | 9.4 ±8.5 | 7.0 [4.0-11.0] | 26.7 | 43.3 | 1.0 to 44.0 | 1610.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.002 | 0.005 | 0.000 to 0.006 | 0.059 |
| **$RWE_{Rotational}$** | 0.166 ±0.332 | 0.001 [0.000-0.060] | 1.000 | 1.000 | 0.000 to 1.000 | 28.6 |
| **$RWE_{CP}$** | 0.060 ±0.191 | 0.001 [0.000-0.007] | 0.573 | 0.998 | 0.000 to 1.000 | 10.4 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 10:** Summary of competition match impact biomechanics of female participants for impacts to the top of the head (n=7) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$* | 99$^{th}$* | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (g)** | 38.7 ±21.5 | 36.3 [16.9-63.2] | - | - | 13.3 to 69.7 | 271.2 |
| **PRA (rad/s2)** | 8,887.2 ±5,427.9 | 9,008.7 [3,602.9-12,584.3] | - | - | 3,288.2 to 18,056.2 | 62,210.2 |
| **HIC** | 86.4 ±93.8 | 50.6 [7.5-213.6] | - | - | 3.0 to 226.8 | 604.5 |
| **GSI** | 136.8 ±153.0 | 76.0 [9.4-302.6] | - | - | 6.8 to 400.1 | 957.6 |
| **HITsp** | 12.4 ±9.7 | 9.4 [3.4-23.9] | - | - | 2.8 to 27.6 | 86.8 |
| **GAMBIT** | 0.310 ±0.189 | 0.293 [0.134-0.429] | - | - | 0.117 to 0.654 | 2.2 |
| **ms** | 19.1 ±10.7 | 20.0 [10.0-28.0] | - | - | 5.0 to 36.0 | 134.0 |
| **$RWE_{Linear}$** | 0.001 ±0.001 | 0.000 [0.000-0.001] | - | - | 0.000 to 0.002 | 0.005 |
| **$RWE_{Rotational}$** | 0.580 ±0.523 | 0.996 [0.005-1.000] | - | - | 0.003 to 1.000 | 4.1 |
| **$RWE_{CP}$** | 0.403 ±0.450 | 0.218 [0.001-0.795] | - | - | 0.001 to 0.999 | 2.8 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability. * = unable to calculate

**Table 11:** Summary of competition match impact biomechanics of forwards for impacts to the head (n=1,511) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.8 ±15.7 | 15.5 [12.3-24.0] | 56.8 | 84.7 | 10.0 to 116.4 | 32,934.3 |
| **PRA (rad/s²)** | 3,888.8 ±3,368.5 | 2,642.0 [1,668.0-4,773.8] | 11,189.4 | 16,014.9 | 88.9 to 22,928.0 | 5,876,035.9 |
| **$HIC_{15}$** | 25.6 ±61.9 | 6.1 [3.6-16.0] | 122.6 | 335.7 | 1.0 to 937.5 | 38,725.6 |
| **GSI** | 40.1 ±94.6 | 10.1 [6.4-25.3] | 187.5 | 484.8 | 1.6 to 1,311.9 | 60,638.2 |
| **HITsp** | 20.0 ±15.1 | 14.4 [12.7-19.8] | 49.2 | 90.0 | 11.0 to 188.4 | 30,220.0 |
| **GAMBIT** | 0.148 ±0.112 | 0.105 [0.076-0.175] | 0.392 | 0.569 | 0.034 to 0.780 | 224.2 |
| **ms** | 9.9 ±8.0 | 8.0 [5.0-12.0] | 28.0 | 40.0 | 1.0 to 44.0 | 14,931.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.020 | 0.483 |
| **$RWE_{Rotational}$** | 0.181 ±0.359 | 0.001 [0.000-0.047] | 1.000 | 1.000 | 0.000 to 1.000 | 273.9 |
| **$RWE_{CP}$** | 0.084 ±0.232 | 0.001 [0.000-0.006] | 0.804 | 0.998 | 0.000 to 1.000 | 126.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 12:** Summary of competition match impact biomechanics of forwards for impacts to the front of the head (n=341) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.8 ±15.6 | 18.3 [14.2-31.1] | 60.4 | 77.8 | 10.1 to 100.7 | 8,463.0 |
| **PRA (rad/s²)** | 4,877.5 ±3,509.4 | 3,577.2 [2,401.1-6,614.6] | 11,729.5 | 16,855.2 | 594.1 to 19,603.0 | 1,663,220.2 |
| **$HIC_{15}$** | 31.6 ±57.3 | 9.4 [4.7-31.1] | 151.3 | 276.8 | 1.5 to 471.2 | 10,790.7 |
| **GSI** | 50.0 ±94.0 | 14.7 [7.7-46.4] | 230.7 | 426.6 | 2.6 to 922.2 | 17,064.8 |
| **HITsp** | 20.0 ±15.2 | 13.8 [11.3-23.2] | 53.0 | 75.7 | 8.8 to 130.3 | 6,820.0 |
| **GAMBIT** | 0.183 ±0.119 | 0.138 [0.095-0.246] | 0.426 | 0.584 | 0.042 to 0.729 | 62.3 |
| **ms** | 11.7 ±8.8 | 9.0 [6.0-15.0] | 32.8 | 42.0 | 1.0 to 44.0 | 3,974.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.009 | 0.109 |
| **$RWE_{Rotational}$** | 0.267 ±0.410 | 0.005 [0.000-0.666] | 1.000 | 1.000 | 0.000 to 1.000 | 91.0 |
| **$RWE_{CP}$** | 0.118 ±0.263 | 0.002 [0.001-0.036] | 0.885 | 0.998 | 0.000 to 1.000 | 40.3 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 13:** Summary of competition match impact biomechanics of forwards for impacts to the back of the head (n=394) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.3 ±16.3 | 18.2 [13.3-28.9] | 58.0 | 91.3 | 10.2 to 104.7 | 9,568.4 |
| **PRA (rad/s²)** | 4,709.7 ±3,741.3 | 3,390.9 [2,050.2-5,907.1] | 12,967.3 | 16,202.4 | 615.2 to 22,928.0 | 1,855,602.3 |
| **$HIC_{15}$** | 33.8 ±79.3 | 9.6 [4.5-24.3] | 140.3 | 350.3 | 1.0 to 937.5 | 13,303.9 |
| **GSI** | 51.1 ±114.4 | 14.6 [7.4-38.0] | 202.0 | 553.0 | 1.6 to 1,311.9 | 20,123.6 |
| **HITsp** | 20.0 ±15.1 | 15.0 [12.1-20.5] | 45.1 | 80.2 | 10.0 to 158.0 | 7,880.0 |
| **GAMBIT** | 0.172 ±0.117 | 0.133 [0.091-0.215] | 0.418 | 0.589 | 0.036 to 0.716 | 67.8 |
| **ms** | 11.2 ±7.9 | 9.0 [6.0-15.0] | 29.0 | 41.0 | 1.0 to 43.0 | 4,394.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.011 | 0.143 |
| **$RWE_{Rotational}$** | 0.240 ±0.397 | 0.003 [0.000-0.319] | 1.000 | 1.000 | 0.000 to 1.000 | 94.7 |
| **$RWE_{CP}$** | 0.124 ±0.285 | 0.002 [0.000-0.018] | 0.929 | 0.998 | 0.000 to 1.000 | 48.7 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 14:** Summary of competition match impact biomechanics of forwards for impacts to the side of the head (n=760) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.0 ±15.1 | 13.6 [11.7-18.2] | 54.3 | 86.7 | 10.0 to 116.4 | 14,434.1 |
| **PRA (rad/s$^2$)** | 2,975.7 ±2,795.1 | 2,005.3 [1,336.8-3,361.0] | 8,872.6 | 15,727.6 | 88.9 to 19,338.3 | 2,261,518.3 |
| **$HIC_{15}$** | 18.4 ±52.5 | 4.5 [3.1-8.5] | 97.3 | 346.7 | 1.0 to 530.4 | 13,985.5 |
| **GSI** | 29.3 ±81.5 | 8.2 [5.6-14.0] | 138.0 | 495.4 | 1.9 to 785.9 | 22,244.4 |
| **HITsp** | 20.0 ±15.1 | 15.1 [14.1-18.3] | 47.5 | 107.3 | 12.7 to 137.6 | 15,200.0 |
| **GAMBIT** | 0.119 ±0.098 | 0.085 [0.068-0.124] | 0.326 | 0.559 | 0.034 to 0.780 | 90.5 |
| **ms** | 8.3 ±7.3 | 6.0 [4.0-10.0] | 24.0 | 38.4 | 1.0 to 44.0 | 6,327.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.020 | 0.226 |
| **$RWE_{Rotational}$** | 0.107 ±0.287 | 0.000 [0.000-0.003] | 0.995 | 1.000 | 0.000 to 1.000 | 81.6 |
| **$RWE_{CP}$** | 0.046 ±0.173 | 0.000 [0.000-0.001] | 0.359 | 0.998 | 0.000 to 1.000 | 34.7 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 15:** Summary of competition match impact biomechanics of forwards for impacts to the top of the head (n=16) than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$* | 99$^{th}$* | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 29.3 ±13.9 | 30.2 [14.2-40.3] | - | - | 12.5 to 58.0 | 468.7 |
| **PRA (rad/s$^2$)** | 5,980.9 ±3,764.5 | 4,668.7 [2,959.8-8,740.4] | - | - | 1,487.0 to 14,747.6 | 95,695.1 |
| **$HIC_{15}$** | 40.3 ±39.6 | 40.5 [5.8-55.2] | - | - | 3.0 to 154.5 | 645.5 |
| **GSI** | 75.3 ±98.5 | 59.9 [9.2-93.1] | - | - | 6.8 to 399.7 | 1,205.3 |
| **HITsp** | 20.0 ±15.3 | 20.6 [6.3-27.0] | - | - | 5.0 to 63.2 | 320.0 |
| **GAMBIT** | 0.231 ±0.137 | 0.222 [0.113-0.285] | - | - | 0.079 to 0.598 | 3.7 |
| **ms** | 14.8 ±10.5 | 11.5 [7.3-21.5] | - | - | 2.0 to 37.0 | 236.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | - | - | 0.000 to 0.001 | 0.005 |
| **$RWE_{Rotational}$** | 0.412 ±0.479 | 0.042 [0.001-0.973] | - | - | 0.000 to 1.000 | 6.6 |
| **$RWE_{CP}$** | 0.168 ±0.292 | 0.008 [0.001-0.181] | - | - | 0.000 to 0.988 | 2.7 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability. * = unable to calculate

**Table 16:** Summary of competition match impact biomechanics of backs for impacts to the head (n=466) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 23.5 ±16.1 | 17.2 [13.0-26.4] | 57.2 | 85.2 | 10.1 to 122.8 | 10,929.9 |
| **PRA (rad/s$^2$)** | 4,533.5 ±3,421.5 | 3,343.5 [2,207.2-5,802.1] | 11,626.8 | 17,687.4 | 95.1 to 21,891.3 | 2,112,624.9 |
| **$HIC_{15}$** | 27.9 ±54.5 | 7.9 [3.9-21.6] | 130.4 | 287.1 | 1.1 to 407.4 | 12,996.4 |
| **GSI** | 43.6 ±82.7 | 12.5 [6.2-35.3] | 214.4 | 450.6 | 1.7 to 548.5 | 20,320.9 |
| **HITsp** | 12.4 ±9.5 | 8.8 [7.2-12.6] | 32.1 | 57.1 | 5.7 to 65.5 | 5,778.4 |
| **GAMBIT** | 0.166 ±0.111 | 0.128 [0.090-0.208] | 0.409 | 0.568 | 0.036 to 0.700 | 77.2 |
| **ms** | 10.6 ±8.4 | 8.0 [5.0-13.0] | 30.7 | 38.0 | 1.0 to 43.0 | 4,948.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.027 | 0.172 |
| **$RWE_{Rotational}$** | 0.225 ±0.382 | 0.003 [0.000-0.275] | 1.000 | 1.000 | 0.000 to 1.000 | 105.0 |
| **$RWE_{CP}$** | 0.102 ±0.251 | 0.001 [0.000-0.015] | 0.849 | 0.999 | 0.000 to 1.000 | 47.4 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 17:** Summary of competition match impact biomechanics of backs for impacts to the front of the head (n=167) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.3 ±15.9 | 18.9 [13.2-27.9] | 56.4 | 92.5 | 10.1 to 102.7 | 4,062.5 |
| **PRA (rad/s$^2$)** | 4,991.7 ±3,528.3 | 3,693.5 [2,487.2-6,017.4] | 13,223.4 | 17,962.7 | 885.1 to 18,412.9 | 833,613.4 |
| **$HIC_{15}$** | 31.2 ±56.4 | 8.9 [4.7-25.9] | 151.9 | 328.9 | 1.6 to 407.4 | 5,205.2 |
| **GSI** | 48.6±83.9 | 14.0 [7.6-40.6] | 238.2 | 473.9 | 2.8 to 548.5 | 8,111.5 |
| **HITsp** | 12.4 ±9.6 | 8.7 [6.9-12.4] | 32.9 | 59.2 | 5.8 to 63.9 | 2,070.8 |
| **GAMBIT** | 0.181 ±0.113 | 0.142 [0.102-0.226] | 0.432 | 0.568 | 0.058 to 0.586 | 30.2 |
| **ms** | 12.0 ±8.9 | 8.0 [6.0-16.0] | 32.0 | 38.0 | 1.0 to 38.0 | 2,011.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.010 | 0.058 |
| **$RWE_{Rotational}$** | 0.250 ±0.395 | 0.006 [0.001-0.360] | 1.000 | 1.000 | 0.000 to 1.000 | 41.7 |
| **$RWE_{CP}$** | 0.116 ±0.270 | 0.002 [0.001-0.018] | 0.954 | 1.000 | 0.000 to 1.000 | 19.4 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 18:** Summary of competition match impact biomechanics of backs for impacts to the back of the head (n=104) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.2 ±14.8 | 18.1 [13.6-31.4] | 54.0 | 83.6 | 10.1 to 84.0 | 2,515.0 |
| **PRA (rad/s$^2$)** | 4,691.2 ±3,546.2 | 3,371.3 [2,171.8-6,762.8] | 11,108.8 | 21,417.2 | 494.4 to 21,891.3 | 487,888.3 |
| **$HIC_{15}$** | 25.8 ±39.8 | 9.3 [4.7-32.1] | 106.7 | 277.3 | 1.3 to 284.8 | 2,681.7 |
| **GSI** | 39.6 ±59.7 | 15.2 [7.1-49.2] | 161.6 | 397.9 | 2.4 to 407.3 | 4,119.8 |
| **HITsp** | 12.4 ±9.5 | 8.4 [6.6-15.1] | 32.0 | 64.2 | 4.7 to 65.6 | 1,289.6 |
| **GAMBIT** | 0.172 ±0.111 | 0.137 [0.091-0.244] | 0.376 | 0.686 | 0.040 to 0.700 | 17.9 |
| **ms** | 10.5 ±7.0 | 8.5 [6.0-14.0] | 23.5 | 39.9 | 1.0 to 40.0 | 1,089.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.004 | 0.030 |
| **$RWE_{Rotational}$** | 0.269 ±0.417 | 0.003 [0.000-0.656] | 1.000 | 1.000 | 0.000 to 1.000 | 28.0 |
| **$RWE_{CP}$** | 0.110 ±0.235 | 0.002 [0.000-0.037] | 0.726 | 0.994 | 0.000 to 1.000 | 11.5 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 19:** Summary of competition match impact biomechanics of backs for impacts to the side of the head (n=184) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.8 ±14.9 | 15.8 [12.1-23.1] | 51.9 | 87.4 | 10.1 to 122.8 | 3,818.3 |
| **PRA (rad/s²)** | 3,730.1 ±2,734.6 | 2,837.8 [1,927.2-4,536.4] | 10,277.6 | 13,237.3 | 95.1 to 13,915.7 | 686,339.4 |
| **$HIC_{15}$** | 20.4 ±50.3 | 5.4 [3.2-13.3] | 103.3 | 340.1 | 1.1 to 367.3 | 3,759.4 |
| **GSI** | 33.2 ±79.8 | 9.3 [5.2-22.4] | 152.4 | 505.9 | 1.7 to 514.8 | 6,110.8 |
| **HITsp** | 12.4 ±9.3 | 9.2 [8.0-12.3] | 30.7 | 64.4 | 7.0 to 68.8 | 2,281.6 |
| **GAMBIT** | 0.139 ±0.097 | 0.102 [0.079-0.164] | 0.359 | 0.522 | 0.036 to 0.599 | 25.6 |
| **ms** | 8.8 ±7.8 | 6.0 [4.0-10.8] | 26.8 | 38.8 | 1.0 to 43.0 | 1,617.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.001 | 0.007 | 0.000 to 0.027 | 0.069 |
| **$RWE_{Rotational}$** | 0.156 ±0.323 | 0.001 [0.000-0.030] | 0.999 | 1.000 | 0.000 to 1.000 | 28.7 |
| **$RWE_{CP}$** | 0.060 ±0.199 | 0.001 [0.000-0.005] | 0.741 | 0.958 | 0.000 to 0.984 | 11.1 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 20:** Summary of competition match impact biomechanics of backs for impacts to the top of the head (n=11) greater than 10*g* recorded by an instrumented patch in a junior rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th* | 99th* | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 48.6 ±26.9 | 57.2 [16.6-66.7] | - | - | 13.6 to 97.9 | 534.1 |
| **PRA (rad/s²)** | 9,525.8 ±5,316.5 | 9,555.7 [4,474.9-12,584.3] | - | - | 3,086.6 to 18,056.2 | 104,783.8 |
| **$HIC_{15}$** | 122.7 ±107.7 | 115.9 [8.6-226.8] | - | - | 4.5 to 258.9 | 1,350.1 |
| **GSI** | 179.9 ±157.0 | 161.0 [15.8-348.8] | - | - | 9.4 to 400.1 | 1,978.8 |
| **HITsp** | 12.4 ±9.5 | 15.0 [1.8-21.1] | - | - | 0.9 to 25.5 | 136.4 |
| **GAMBIT** | 0.320 ±0.153 | 0.333 [0.202-0.413] | - | - | 0.130 to 0.654 | 3.5 |
| **ms** | 21.0 ±10.5 | 22.0 [11.0-30.0] | - | - | 6.0 to 36.0 | 231.0 |
| **$RWE_{Linear}$** | 0.001 ±0.002 | 0.001 [0.000-0.001] | - | - | 0.000 to 0.008 | 0.016 |
| **$RWE_{Rotational}$** | 0.603 ±0.470 | 0.999 [0.027-1.000] | - | - | 0.002 to 1.000 | 6.6 |
| **$RWE_{CP}$** | 0.489 ±0.471 | 0.609 [0.004-0.960] | - | - | 0.001 to 0.999 | 5.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability; * = unable to calculate

# Appendix X: Supplemental data for Technical Report (Appendix II)

## Head impacts in a senior amateur rugby league team measured with an instrumented patch: An exploratory analysis.

## Total, Match and Player Positional Group Impact Biomechanics Summaries

# Total Match Impact Biomechanics

**Table 1:** Summary of total match impact biomechanics for impacts to the head (n=greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

**Impact biomechanics of total competition matches impacts (n=13,895 impacts)**

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | Range | Cumulative |
|---|---|---|---|---|---|
| **PLA (*g*)** | 23.1 ±15.6 | 17.1 [12.2-28.0] | 55.6 | 10.0 to 165.9 | 321,078.9 |
| **PRA (rad/s$^2$)** | 4,097.5 ±3,668.7 | 3,071.6 [1,661.1-5,466.0] | 11,734.1 | 4.1 to 32,720.4 | 56,934,631.1 |
| **$HIC_{15}$** | 29.3 ±63.5 | 8.0 [4.8-24.6] | 124.0 | 1.0 to 1,086.8 | 407,065.9 |
| **GSI** | 46.3 ±95.3 | 14.8 [8.5-38.9] | 197.2 | 1.5 to 1,494.6 | 643,235.8 |
| **HITsp** | 20.0 ±15.0 | 14.6 [11.8-21.4] | 47.7 | 6.3 to 201.5 | 277,891.3 |
| **GAMBIT** | 0.155 ±0.118 | 0.120 [0.073-0.201] | 0.397 | 0.015 to 0.987 | 2,155.8 |
| **(ms)** | 12.9 ±9.1 | 10.0 [6.0-18.0] | 33.0 | 1.0 to 46.0 | 178,869.0 |
| **$CER_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.000 to 0.074 | 4.7 |
| **$CER_{Rotational}$** | 0.210 ±0.372 | 0.002 [0.000-0.168] | 1.000 | 0.000 to 1.000 | 2,914.1 |
| **$CER_{CP}$** | 0.091 ±0.242 | 0.001 [0.000-0.013] | 0.848 | 0.000 to 1.000 | 1,267.9 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of total match impact biomechanics for impacts to the front of the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

**Impact biomechanics of total competition matches impacts to the front of the head (n=3,664 impacts)**

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | Range | Cumulative |
|---|---|---|---|---|---|
| **PLA (*g*)** | 26.2 ±16.6 | 20.5 [14.5-32.3] | 60.9 | 10.0 to 127.4 | 95,861.0 |
| **PRA (rad/s$^2$)** | 4,925.9 ±3,544.6 | 3,802.4 [2,547.7-6,103.0] | 12,533.4 | 29.9 to 28,882.6 | 18,048,591.6 |
| **$HIC_{15}$** | 36.4 ±70.6 | 12.1 [5.6-34.9] | 154.3 | 1.0 to 1,086.8 | 133,349.5 |
| **GSI** | 57.0 ±107.4 | 19.9 [9.6-54.5] | 243.9 | 1.7 to 1,371.7 | 209,013.2 |
| **HITsp** | 22.4 ±16.4 | 16.5 [13.3-24.5] | 53.4 | 10.0 to 187.8 | 81,958.1 |
| **GAMBIT** | 0.187 ±0.117 | 0.151 [0.107-0.230] | 0.432 | 0.019 to 0.909 | 685.3 |
| **(ms)** | 13.4 ±8.6 | 11.0 [7.0-18.0] | 32.0 | 1.0 to 44.0 | 48,941.0 |
| **$CER_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.000 to 0.034 | 1.5 |
| **$CER_{Rotational}$** | 0.250 ±0.392 | 0.007 [0.001-0.419] | 1.000 | 0.000 to 1.000 | 917.1 |
| **$CER_{CP}$** | 0.109 ±0.263 | 0.002 [0.001-0.025] | 0.930 | 0.000 to 1.000 | 400.0 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of total match impact biomechanics for impacts to the back of the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

**Impact biomechanics of total competition matches impacts to the back of the head (n=3,694 impacts)**

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | Range | Cumulative |
|---|---|---|---|---|---|
| **PLA (*g*)** | 25.3 ±15.9 | 20.1 [13.8-31.3] | 57.5 | 10.0 to 124.1 | 93,403.4 |
| **PRA (rad/s$^2$)** | 4,758.4 ±3,722.5 | 3,592.0 [2,124.6-6,268.0] | 12,087.8 | 104.3 to 32,720.4 | 17,577,498.4 |
| **$HIC_{15}$** | 34.3 ±71.3 | 10.9 [4.5-32.3] | 132.5 | 1.1 to 879.5 | 126,559.1 |
| **GSI** | 52.9 ±104.3 | 17.7 [7.4-51.8] | 217.0 | 1.6 to 1,316.2 | 195,320.1 |
| **HITsp** | 21.6 ±16.1 | 16.1 [12.6-24.1] | 50.4 | 6.3 to 175.9 | 79,910.7 |
| **GAMBIT** | 0.174 ±0.122 | 0.135 [0.087-0.225] | 0.418 | 0.026 to 0.987 | 641.3 |
| **(ms)** | 12.3 ±8.6 | 10.0 [6.0-17.0] | 31.0 | 1.0 to 44.0 | 45,381.0 |
| **$CER_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.000 to 0.029 | 1.4 |
| **$CER_{Rotational}$** | 0.259 ±0.397 | 0.005 [0.000-0.501] | 1.000 | 0.000 to 1.000 | 955.8 |
| **$CER_{CP}$** | 0.110 ±0.261 | 0.002 [0.000-0.029] | 0.891 | 0.000 to 1.000 | 408.0 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of total match impact biomechanics for impacts to the side of the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Impact biomechanics of total competition matches impacts to the side of the head (n=6,131 impacts) | | | | |
|---|---|---|---|---|---|
| | **Mean ±SD** | **Median [25$^{th}$-75$^{th}$]** | **95%** | **Range** | **Cumulative** |
| **PLA (*g*)** | 19.6 ±14.1 | 13.4 [11.5-21.6] | 49.3 | 10.0 to 165.9 | 120,161.5 |
| **PRA (rad/s$^2$)** | 3,088.1 ±3,441.2 | 2,079.9 [636.9-4,055.8] | 10,486.9 | 4.1 to 30,270.0 | 18,933,173.6 |
| **$HIC_{15}$** | 21.2 ±52.0 | 5.8 [4.6-13.4] | 93.4 | 1.0 to 1,052.5 | 129,800.4 |
| **GSI** | 34.7 ±79.7 | 14.1 [8.2-22.1] | 148.8 | 1.5 to 1,494.6 | 212,688.2 |
| **HITsp** | 17.3 ±13.0 | 12.5 [11.4-17.1] | 41.2 | 9.8 to 201.5 | 106,084.4 |
| **GAMBIT** | 0.122 ±0.108 | 0.085 [0.044-0.152] | 0.343 | 0.018 to 0.956 | 745.0 |
| **(ms)** | 12.8 ±9.6 | 10.0 [5.0-18.0] | 33.0 | 1.0 to 46.0 | 78,173.0 |
| **$CER_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.000 to 0.074 | 1.7 |
| **$CER_{Rotational}$** | 0.146 ±0.327 | 0.000 [0.000-0.012] | 1.000 | 0.000 to 1.000 | 892.4 |
| **$CER_{CP}$** | 0.064 ±0.207 | 0.000 [0.000-0.003] | 0.630 | 0.000 to 1.000 | 393.6 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of total match impact biomechanics for impacts to the top of the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Impact biomechanics of total competition matches impacts to the top of the head (n=406 impacts) | | | | |
|---|---|---|---|---|---|
| | **Mean ±SD** | **Median [25$^{th}$-75$^{th}$]** | **95%** | **Range** | **Cumulative** |
| **PLA (*g*)** | 28.7 ±15.1 | 24.8 [17.4-36.3] | 59.6 | 10.2 to 117.0 | 11,653.0 |
| **PRA (rad/s$^2$)** | 5,850.7 ±3,715.6 | 4,732.6 [3,120.5-7,834.9] | 12,934.0 | 283.9 to 19,995.4 | 2,375,367.6 |
| **$HIC_{15}$** | 42.8 ±61.5 | 19.1 [9.1-52.6] | 170.2 | 1.6 to 501.4 | 17,356.9 |
| **GSI** | 64.6 ±90.6 | 29.6 [14.0-78.4] | 255.7 | 2.5 to 686.0 | 26,214.3 |
| **HITsp** | 24.5 ±14.8 | 19.3 [14.9-29.1] | 56.9 | 9.9 to 112.2 | 9,938.1 |
| **GAMBIT** | 0.207 ±0.112 | 0.180 [0.125-0.268] | 0.425 | 0.015 to 0.720 | 84.2 |
| **(ms)** | 15.7 ±9.3 | 14.0 [9.0-20.0] | 35.0 | 1.0 to 42.0 | 6,374.0 |
| **$CER_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.000 to 0.021 | 0.162 |
| **$CER_{Rotational}$** | 0.366 ±0.436 | 0.045 [0.002-0.958] | 1.000 | 0.000 to 1.000 | 148.7 |
| **$CER_{CP}$** | 0.163 ±0.303 | 0.006 [0.001-0.098] | 0.952 | 0.000 to 1.000 | 66.3 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

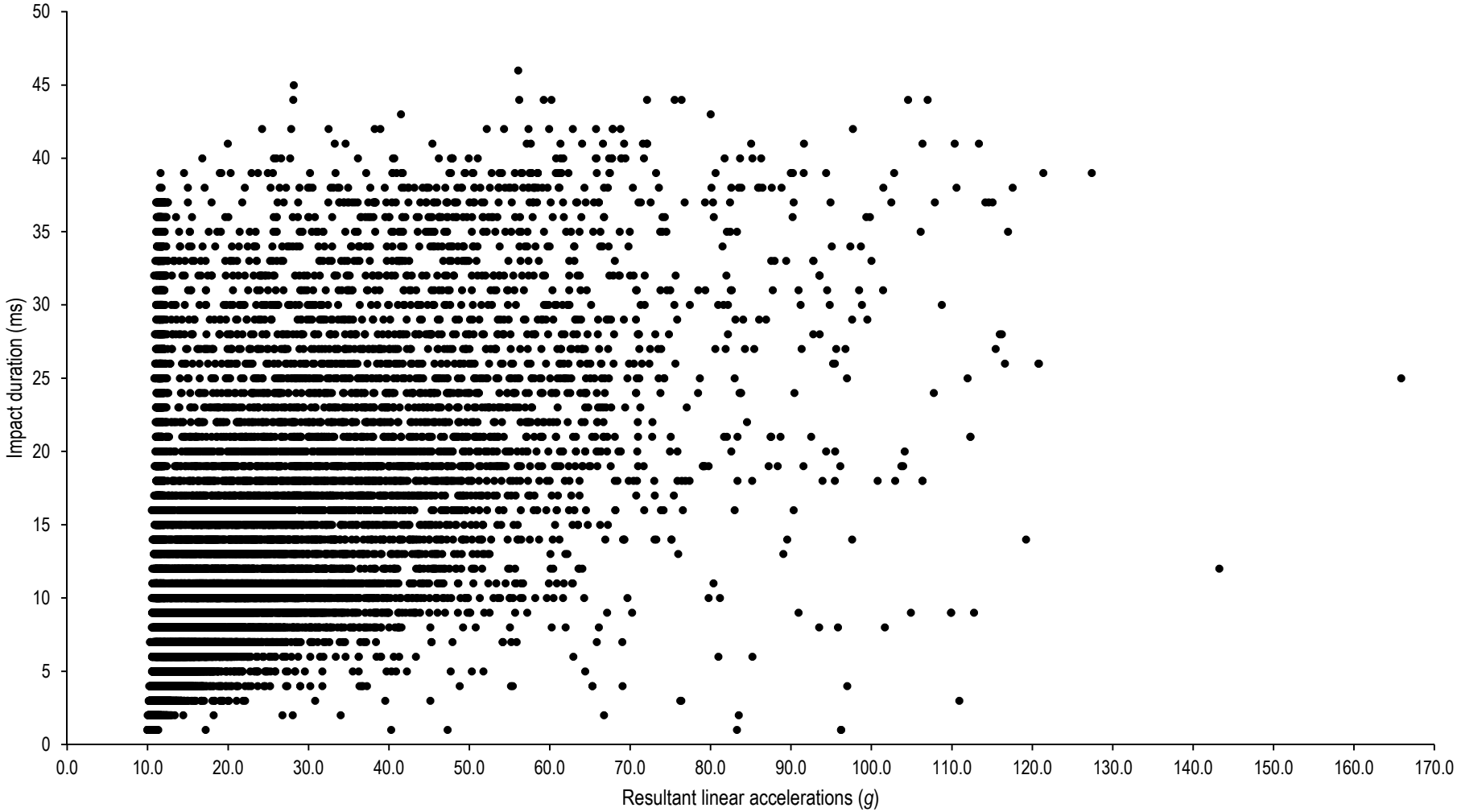


**Figure 1:** Resultant linear accelerations (*g*) by impact duration in milliseconds (ms) for all matches competed in a senior amateur rugby league team over a competitive season

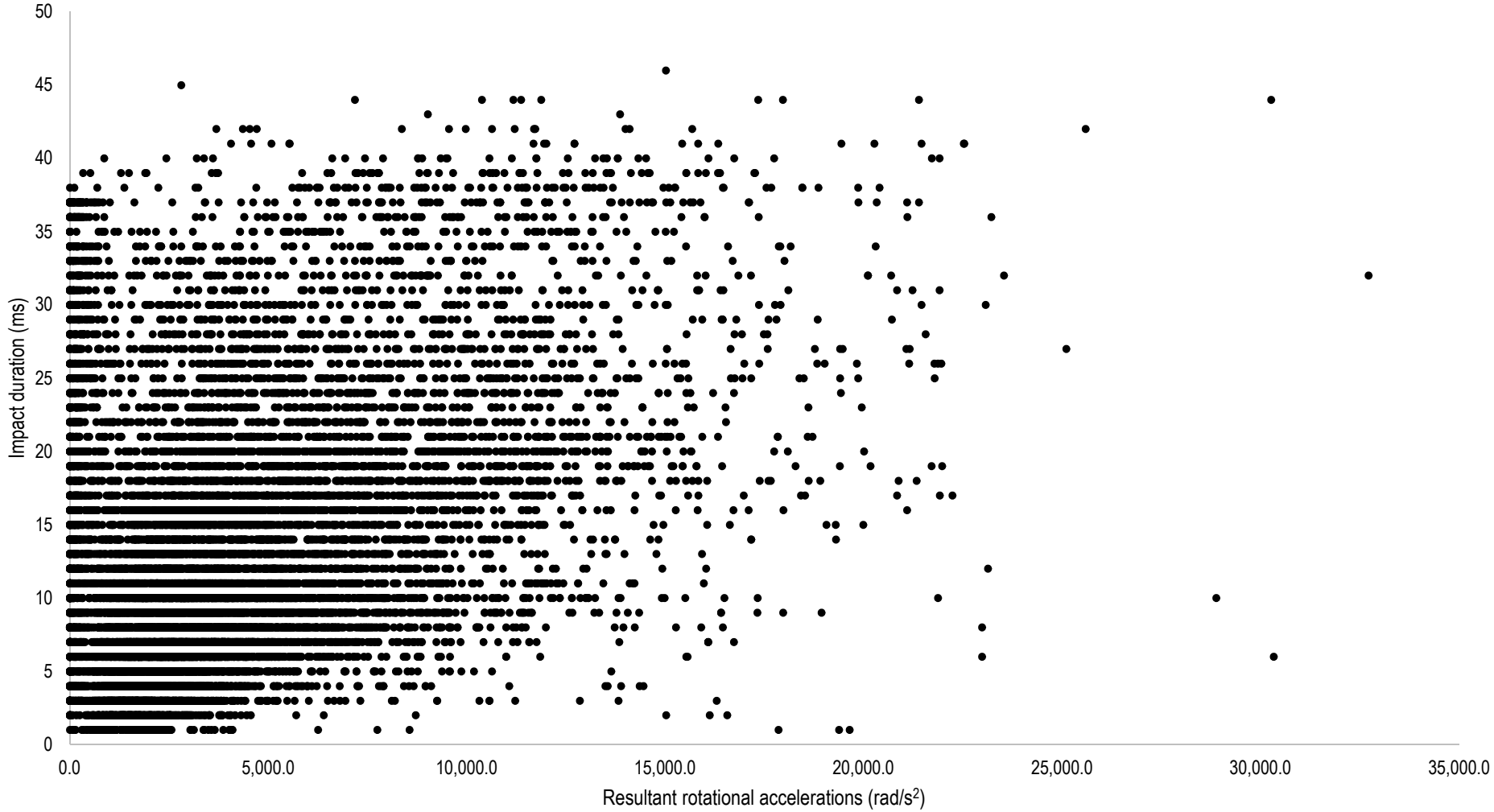


**Figure 2:** Resultant rotational accelerations (rad/s$^2$) by impact duration in milliseconds (ms) for all matches competed in a senior amateur rugby league team over a competitive season

# Individual Match Impact Biomechanics

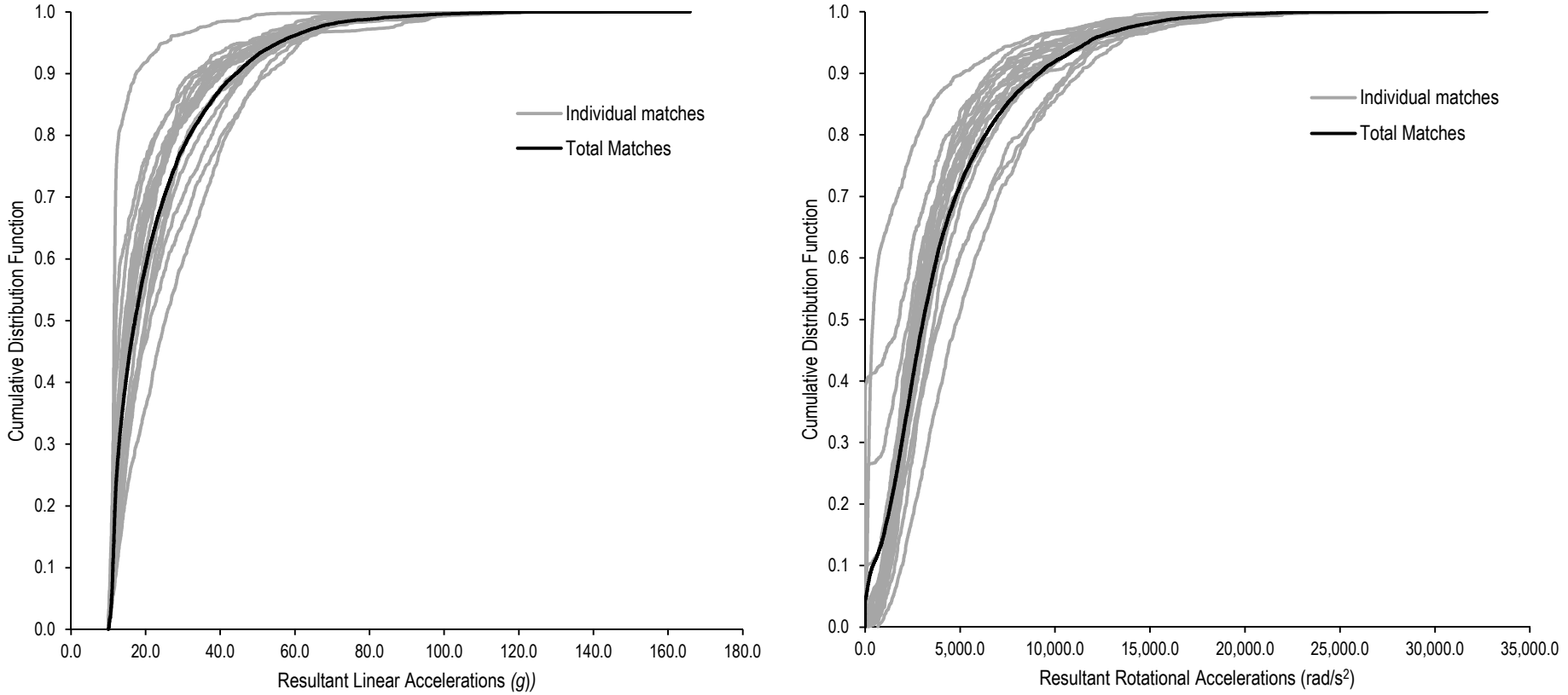


**Figure 1:** Cumulative distribution functions for resultant linear (left) and rotational (right) acceleration magnitudes of impacts collected during matches for senior amateur rugby league season 2014.

**Table 1:** Summary of preseason match 1 impact biomechanics for impacts to the head (n=768) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 13.6 ±7.0 | 11.6 [11.3-12.2] | 25.6 | 48.1 | 10.0 to 165.9 | 17,212.5 |
| **PRA (rad/s²)** | 1,800.4 ±3,064.6 | 419.8 [175.1-2,196.9] | 8,221.6 | 15,528.7 | 14.2 to 22,987.2 | 2,273,867.6 |
| **$HIC_{15}$** | 7.3 ±13.7 | 5.5 [5.1-5.8] | 15.1 | 66.5 | 0.9 to 268.3 | 9,174.6 |
| **GSI** | 14.6 ±18.5 | 14.1 [9.9-14.7] | 23.0 | 90.2 | 1.6 to 360.1 | 18,390.0 |
| **HITsp** | 20.0 ±14.3 | 17.7 [17.1-18.3] | 34.2 | 87.6 | 10.5 to 258.1 | 25,260.0 |
| **GAMBIT** | 0.081 ±0.088 | 0.041 [0.037-0.092] | 0.250 | 0.461 | 0.034 to 0.935 | 102.7 |
| **ms** | 14.3 ±10.3 | 12.0 [5.0-22.0] | 35.0 | 37.0 | 1.0 to 39.0 | 18,020.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.000 | 0.001 | 0.000 to 0.009 | 0.164 |
| **$RWE_{Rotational}$** | 0.079 ±0.252 | 0.000 [0.000-0.000] | 0.980 | 1.000 | 0.000 to 1.000 | 100.2 |
| **$RWE_{CP}$** | 0.033 ±0.154 | 0.000 [0.000-0.000] | 0.084 | 0.989 | 0.000 to 1.000 | 41.1 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of preseason match 2 impact biomechanics for impacts to the head (n=961) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.5 ±15.7 | 19.8 [14.1-28.3] | 56.2 | 95.0 | 10.0 to 165.9 | 23,778.3 |
| **PRA (rad/s²)** | 4,724.9 ±3,666.6 | 3,656.7 [2,351.2-5,885.6] | 12,036.6 | 18,739.1 | 66.2 to 30,270.0 | 4,578,449.8 |
| **$HIC_{15}$** | 32.8 ±73.0 | 11.2 [5.3-24.9] | 128.3 | 437.1 | 1.2 to 801.2 | 31,793.3 |
| **GSI** | 50.9 ±112.4 | 17.4 [9.2-37.7] | 212.1 | 597.8 | 2.0 to 1,232.0 | 49,321.9 |
| **HITsp** | 20.0 ±15.2 | 15.2 [12.3-20.5] | 46.2 | 88.0 | 6.2 to 155.1 | 19,368.3 |
| **GAMBIT** | 0.172 ±0.122 | 0.135 [0.094-0.209] | 0.419 | 0.676 | 0.041 to 0.956 | 166.4 |
| **ms** | 12.2 ±8.2 | 10.0 [7.0-16.0] | 30.0 | 39.0 | 1.0 to 44.0 | 11,819.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.007 | 0.000 to 0.015 | 0.352 |
| **$RWE_{Rotational}$** | 0.234 ±0.382 | 0.005 [0.000-0.319] | 1.000 | 1.000 | 0.000 to 1.000 | 226.4 |
| **$RWE_{CP}$** | 0.096 ±0.248 | 0.002 [0.001-0.018] | 0.882 | 1.000 | 0.000 to 1.000 | 93.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of competition match 1 impact biomechanics for impacts (n=489) to the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 22.0 ±17.2 | 15.6 [12.0-24.3] | 55.7 | 97.6 | 10.0 to 165.9 | 10,743.7 |
| **PRA (rad/s²)** | 4,386.3 ±3,785.5 | 3,125.5 [1,993.0-5,184.2] | 12,695.1 | 18,863.6 | 250.6 to 21,458.6 | 2,144,919.9 |
| **$HIC_{15}$** | 30.5 ±88.4 | 6.2 [3.2-17.1] | 123.2 | 598.7 | 0.9 to 771.5 | 14,918.4 |
| **GSI** | 46.9 ±131.0 | 11.5 [5.1-28.8] | 186.4 | 883.4 | 1.5 to 1,343.8 | 22,909.7 |
| **HITsp** | 17.2 ±13.8 | 13.6 [12.0-17.2] | 37.0 | 92.3 | 6.9 to 136.3 | 8,419.5 |
| **GAMBIT** | 0.158 ±0.126 | 0.115 [0.079-0.185] | 0.420 | 0.592 | 0.032 to 0.973 | 77.2 |
| **ms** | 9.9 ±8.3 | 7.0 [4.0-13.0] | 28.0 | 38.0 | 1.0 to 41.0 | 4,854.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.001 | 0.008 | 0.000 to 0.020 | 0.206 |
| **$RWE_{Rotational}$** | 0.191 ±0.361 | 0.002 [0.000-0.103] | 1.000 | 1.000 | 0.000 to 1.000 | 93.6 |
| **$RWE_{CP}$** | 0.097 ±0.263 | 0.001 [0.000-0.009] | 0.931 | 1.000 | 0.000 to 1.000 | 47.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of competition match 2 impact biomechanics for impacts to the head (n=388) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.5 ±15.2 | 14.8 [11.9-22.7] | 52.3 | 83.0 | 10.0 to 165.9 | 7,953.8 |
| **PRA (rad/s$^2$)** | 3,655.1 ±3,390.4 | 2,552.7 [1,482.8-4,195.3] | 11,181.1 | 17,975.7 | 215.2 to 20,014.5 | 1,418,184.7 |
| **$HIC_{15}$** | 23.8 ±68.4 | 5.3 [2.9-14.0] | 100.7 | 350.1 | 1.3 to 688.6 | 9,236.4 |
| **GSI** | 36.3 ±95.6 | 10.1 [4.2-23.1] | 144.6 | 590.5 | 1.7 to 947.4 | 14,076.9 |
| **HITsp** | 17.2 ±13.0 | 13.9 [12.4-17.1] | 38.3 | 75.1 | 7.6 to 140.1 | 6,665.9 |
| **GAMBIT** | 0.136 ±0.114 | 0.092 [0.065-0.162] | 0.395 | 0.599 | 0.032 to 0.726 | 52.7 |
| **ms** | 9.3 ±7.6 | 7.0 [4.0-12.0] | 25.0 | 38.1 | 1.0 to 41.0 | 3,625.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.034 | 0.136 |
| **$RWE_{Rotational}$** | 0.154 ±0.332 | 0.001 [0.000-0.016] | 1.000 | 1.000 | 0.000 to 1.000 | 59.8 |
| **$RWE_{CP}$** | 0.066 ±0.216 | 0.001 [0.000-0.004] | 0.779 | 1.000 | 0.000 to 1.000 | 25.7 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of competition match 3 impact biomechanics for impacts to the head (n=840) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.0 ±14.4 | 13.2 [11.2-20.2] | 47.8 | 80.6 | 10.0 to 165.9 | 15,974.8 |
| **PRA (rad/s$^2$)** | 3,087.5 ±3,475.0 | 2,332.7 [24.4-4,261.2] | 10,871.2 | 17,095.2 | 4.6 to 21,393.5 | 2,590,438.4 |
| **$HIC_{15}$** | 20.5 ±55.4 | 5.1 [4.4-11.6] | 88.5 | 276.9 | 1.0 to 735.7 | 17,240.6 |
| **GSI** | 33.5 ±82.3 | 12.9 [7.6-18.5] | 145.1 | 425.2 | 1.7 to 1,061.7 | 28,076.1 |
| **HITsp** | 17.1 ±12.9 | 13.5 [12.7-17.1] | 39.7 | 82.6 | 6.9 to 121.4 | 14,335.3 |
| **GAMBIT** | 0.121 ±0.112 | 0.091 [0.036-0.147] | 0.372 | 0.593 | 0.020 to 0.796 | 101.8 |
| **ms** | 9.9 ±7.2 | 8.0 [5.0-12.0] | 25.0 | 38.0 | 1.0 to 44.0 | 8,267.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.023 | 0.249 |
| **$RWE_{Rotational}$** | 0.131 ±0.310 | 0.000 [0.000-0.018] | 1.000 | 1.000 | 0.000 to 1.000 | 110.3 |
| **$RWE_{CP}$** | 0.064 ±0.215 | 0.000 [0.000-0.003] | 0.710 | 0.998 | 0.000 to 1.000 | 54.1 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 6:** Summary of competition match 4 impact biomechanics for impacts to the head (n=633) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.8 ±15.0 | 14.4 [11.5-24.1] | 56.3 | 83.8 | 10.0 to 165.9 | 13,142.0 |
| **PRA (rad/s$^2$)** | 3,623.3 ±3,389.6 | 2,470.4 [1,380.4-4,742.6] | 11,188.6 | 15,354.1 | 4.7 to 21,103.8 | 2,293,542.1 |
| **$HIC_{15}$** | 24.2 ±58.8 | 5.2 [3.7-17.1] | 126.0 | 357.1 | 1.0 to 659.9 | 15,316.8 |
| **GSI** | 38.8 ±88.4 | 11.7 [6.3-26.8] | 192.1 | 543.3 | 1.6 to 867.8 | 24,561.3 |
| **HITsp** | 16.9 ±12.6 | 13.0 [11.8-17.7] | 43.6 | 78.9 | 6.9 to 106.8 | 10,723.2 |
| **GAMBIT** | 0.140 ±0.115 | 0.099 [0.065-0.174] | 0.400 | 0.567 | 0.031 to 0.646 | 88.4 |
| **ms** | 9.7 ±8.4 | 7.0 [4.0-13.0] | 30.0 | 38.0 | 1.0 to 43.0 | 6,126.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.009 | 0.174 |
| **$RWE_{Rotational}$** | 0.176 ±0.348 | 0.001 [0.000-0.045] | 1.000 | 1.000 | 0.000 to 1.000 | 111.2 |
| **$RWE_{CP}$** | 0.078 ±0.227 | 0.001 [0.000-0.006] | 0.805 | 0.996 | 0.000 to 1.000 | 49.5 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 7:** Summary of competition match 5 impact biomechanics for impacts to the head (n=1,128) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 25.5 ±15.8 | 20.1 [13.4-34.0] | 57.3 | 76.3 | 10.0 to 165.9 | 28,811.8 |
| **PRA (rad/s$^2$)** | 5,087.0 ±3,810.5 | 3,955.8 [2,129.4-7,173.7] | 12,893.1 | 16,407.4 | 221.2 to 23,219.2 | 5,738,119.2 |
| **$HIC_{15}$** | 35.1 ±61.0 | 11.7 [4.4-39.1] | 148.7 | 324.4 | 1.1 to 656.4 | 39,602.8 |
| **GSI** | 54.5 ±96.4 | 19.2 [7.4-60.0] | 229.4 | 466.1 | 1.7 to 1,369.5 | 61,530.9 |
| **HITsp** | 20.0 ±15.1 | 14.5 [10.4-24.0] | 48.6 | 78.1 | 8.0 to 183.1 | 22,560.0 |
| **GAMBIT** | 0.178 ±0.123 | 0.140 [0.082-0.249] | 0.421 | 0.559 | 0.029 to 0.925 | 201.2 |
| **ms** | 12.6 ±9.0 | 10.0 [6.0-18.0] | 32.0 | 38.7 | 1.0 to 44.0 | 14,237.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.019 | 0.392 |
| **$RWE_{Rotational}$** | 0.312 ±0.424 | 0.010 [0.000-0.860] | 1.000 | 1.000 | 0.000 to 1.000 | 351.9 |
| **$RWE_{CP}$** | 0.137 ±0.284 | 0.003 [0.000-0.064] | 0.936 | 0.997 | 0.000 to 1.000 | 154.0 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 8:** Summary of competition match 6 impact biomechanics for impacts to the head (n=523) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.9 ±12.3 | 15.1 [11.5-22.7] | 48.7 | 65.8 | 10.0 to 165.9 | 10,392.4 |
| **PRA (rad/s$^2$)** | 3,771.3 ±2,954.7 | 2,916.7 [1,798.0-4,886.9] | 10,568.4 | 14,118.0 | 4.1 to 16,751.2 | 1,972,383.2 |
| **$HIC_{15}$** | 17.3 ±31.0 | 6.4 [3.7-15.5] | 74.0 | 164.5 | 1.0 to 235.1 | 9,048.6 |
| **GSI** | 27.2 ±44.2 | 12.3 [5.8-26.7] | 111.1 | 243.8 | 1.6 to 339.1 | 14,248.2 |
| **HITsp** | 17.7 ±13.8 | 12.9 [10.8-18.7] | 43.7 | 85.9 | 4.3 to 103.5 | 9,274.5 |
| **GAMBIT** | 0.137 ±0.088 | 0.109 [0.071-0.180] | 0.328 | 0.426 | 0.030 to 0.497 | 71.4 |
| **ms** | 9.8 ±7.3 | 8.0 [4.0-14.0] | 25.0 | 30.8 | 1.0 to 39.0 | 5,102.0 |
| **$RWE_{Linear}$** | 0.000 ±0.000 | 0.000 [0.000-0.000] | 0.001 | 0.002 | 0.000 to 0.003 | 0.108 |
| **$RWE_{Rotational}$** | 0.171 ±0.335 | 0.001 [0.000-0.060] | 1.000 | 1.000 | 0.000 to 1.000 | 89.5 |
| **$RWE_{CP}$** | 0.061 ±0.193 | 0.001 [0.000-0.007] | 0.662 | 0.975 | 0.000 to 0.998 | 32.0 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 9:** Summary of competition match 7 impact biomechanics for impacts to the head (n=539) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 23.5 ±15.1 | 18.2 [13.0-28.2] | 54.3 | 81.4 | 10.0 to 165.9 | 12,688.1 |
| **PRA (rad/s$^2$)** | 4,154.3 ±3,553.3 | 3,148.9 [1,806.6-5,411.7] | 11,550.0 | 19,072.9 | 8.3 to 21,791.1 | 2,239,146.4 |
| **$HIC_{15}$** | 28.2 ±58.5 | 8.7 [4.6-25.5] | 119.1 | 319.2 | 0.9 to 613.1 | 15,199.4 |
| **GSI** | 44.6 ±92.0 | 15.4 [8.0-39.4] | 177.2 | 525.8 | 1.4 to 998.3 | 24,022.9 |
| **HITsp** | 16.9 ±12.6 | 12.9 [10.7-18.4] | 39.1 | 81.7 | 5.4 to 125.9 | 9,089.8 |
| **GAMBIT** | 0.158 ±0.120 | 0.122 [0.082-0.198] | 0.377 | 0.619 | 0.018 to 0.987 | 85.0 |
| **ms** | 11.5 ±8.1 | 10.0 [6.0-16.0] | 29.0 | 36.0 | 1.0 to 44.0 | 6,219.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.029 | 0.183 |
| **$RWE_{Rotational}$** | 0.199 ±0.361 | 0.002 [0.000-0.153] | 1.000 | 1.000 | 0.000 to 1.000 | 107.3 |
| **$RWE_{CP}$** | 0.085 ±0.236 | 0.001 [0.000-0.012] | 0.824 | 1.000 | 0.000 to 1.000 | 45.8 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 10:** Summary of competition match 8 impact biomechanics for impacts to the head (n=407) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.4 ±13.6 | 16.3 [12.3-24.6] | 50.7 | 67.7 | 10.0 to 165.9 | 8,721.2 |
| **PRA (rad/s²)** | 3,701.5 ±3,125.6 | 2,883.0 [1,764.8-4,668.5] | 9,894.9 | 16,496.2 | 92.6 to 22,239.4 | 1,506,502.7 |
| **$HIC_{15}$** | 21.7 ±55.7 | 7.7 [3.9-18.6] | 92.3 | 216.2 | 1.3 to 908.5 | 8,849.7 |
| **GSI** | 34.2 ±77.7 | 13.7 [7.1-29.5] | 143.8 | 329.0 | 1.8 to 1,157.9 | 13,902.2 |
| **HITsp** | 17.6 ±13.2 | 14.3 [11.8-19.0] | 39.1 | 66.5 | 5.7 to 195.1 | 7,149.4 |
| **GAMBIT** | 0.139 ±0.092 | 0.114 [0.075-0.178] | 0.330 | 0.522 | 0.030 to 0.552 | 56.7 |
| **ms** | 11.3 ±8.2 | 9.0 [5.0-15.0] | 29.6 | 36.9 | 1.0 to 42.0 | 4,580.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.002 | 0.000 to 0.016 | 0.108 |
| **$RWE_{Rotational}$** | 0.149 ±0.316 | 0.001 [0.000-0.039] | 0.999 | 1.000 | 0.000 to 1.000 | 60.8 |
| **$RWE_{CP}$** | 0.057 ±0.197 | 0.001 [0.000-0.006] | 0.552 | 0.996 | 0.000 to 1.000 | 23.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 11:** Summary of competition match 9 impact biomechanics for impacts to the head (n-=1,209) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 25.4 ±16.1 | 20.2 [14.5-30.4] | 57.9 | 86.1 | 10.0 to 165.9 | 30,732.6 |
| **PRA (rad/s²)** | 3,534.3 ±2,887.5 | 2,800.6 [1,759.1-4,297.6] | 8,686.6 | 15,057.6 | 155.3 to 28,882.6 | 4,273,005.1 |
| **$HIC_{15}$** | 32.1 ±60.7 | 12.4 [6.0-31.0] | 121.8 | 344.5 | 1.0 to 858.9 | 38,852.6 |
| **GSI** | 49.1 ±89.4 | 20.8 [10.5-49.0] | 179.6 | 489.5 | 1.6 to 1,316.2 | 59,357.0 |
| **HITsp** | 19.5 ±14.6 | 14.6 [11.7-21.4] | 45.2 | 89.2 | 5.5 to 186.2 | 23,569.1 |
| **GAMBIT** | 0.156 ±0.096 | 0.129 [0.091-0.193] | 0.336 | 0.538 | 0.029 to 0.708 | 188.9 |
| **ms** | 14.1 ±8.7 | 12.0 [8.0-19.0] | 31.0 | 38.0 | 1.0 to 42.0 | 17,048.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.026 | 0.472 |
| **$RWE_{Rotational}$** | 0.121 ±0.286 | 0.001 [0.000-0.019] | 0.992 | 1.000 | 0.000 to 1.000 | 146.4 |
| **$RWE_{CP}$** | 0.047 ±0.171 | 0.001 [0.000-0.005] | 0.289 | 0.993 | 0.000 to 1.000 | 57.0 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 12:** Summary of competition match 10 impact biomechanics for impacts to the head (n=444) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.1 ±14.0 | 15.5 [12.0-25.1] | 51.0 | 78.9 | 10.0 to 165.9 | 9,379.5 |
| **PRA (rad/s²)** | 3,616.8 ±3,325.4 | 2,559.0 [1,466.4-4,357.5] | 10,758.9 | 16,899.9 | 7.9 to 21,092.7 | 1,605,845.0 |
| **$HIC_{15}$** | 25.6 ±63.9 | 6.7 [3.7-19.7] | 112.9 | 316.6 | 1.1 to 853.5 | 11,377.4 |
| **GSI** | 40.0 ±89.7 | 12.5 [6.3-32.4] | 195.8 | 429.5 | 1.9 to 1,055.4 | 17,743.2 |
| **HITsp** | 17.3 ±12.4 | 13.4 [11.7-18.2] | 39.0 | 77.3 | 6.5 to 101.8 | 7,698.2 |
| **GAMBIT** | 0.145 ±0.111 | 0.110 [0.073-0.175] | 0.386 | 0.562 | 0.022 to 0.806 | 64.4 |
| **ms** | 11.6 ±9.2 | 9.0 [5.0-16.8] | 33.8 | 39.6 | 1.0 to 45.0 | 5,169.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.014 | 0.120 |
| **$RWE_{Rotational}$** | 0.157 ±0.336 | 0.001 [0.000-0.022] | 1.000 | 1.000 | 0.000 to 1.000 | 69.5 |
| **$RWE_{CP}$** | 0.069 ±0.214 | 0.001 [0.000-0.004] | 0.689 | 0.998 | 0.000 to 1.000 | 30.7 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 13:** Summary of competition match 11 impact biomechanics for impacts to the head (n=815) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.5 ±14.9 | 15.8 [12.1-24.2] | 51.7 | 84.9 | 10.0 to 165.9 | 17,489.6 |
| **PRA (rad/s²)** | 3,798.7 ±3,630.8 | 2,681.1 [1,534.5-4,643.6] | 11,761.1 | 17,167.9 | 12.2 to 30,333.2 | 3,095,929.4 |
| **$HIC_{15}$** | 25.7 ±65.2 | 6.3 [3.7-18.1] | 108.1 | 360.6 | 1.2 to 1,086.8 | 20,943.0 |
| **GSI** | 40.2 ±93.5 | 12.7 [5.7-29.5] | 172.5 | 478.5 | 1.8 to 1,371.7 | 32,738.2 |
| **HITsp** | 17.6 ±13.4 | 13.6 [12.0-17.8] | 41.7 | 75.8 | 6.1 to 181.2 | 14,353.5 |
| **GAMBIT** | 0.141 ±0.110 | 0.101 [0.067-0.171] | 0.381 | 0.532 | 0.015 to 0.841 | 114.5 |
| **ms** | 11.1 ±8.5 | 8.0 [5.0-16.0] | 31.0 | 37.0 | 1.0 to 41.0 | 9,085.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.008 | 0.232 |
| **$RWE_{Rotational}$** | 0.170 ±0.347 | 0.001 [0.000-0.038] | 1.000 | 1.000 | 0.000 to 1.000 | 138.4 |
| **$RWE_{CP}$** | 0.080 ±0.235 | 0.001 [0.000-0.006] | 0.796 | 0.999 | 0.000 to 1.000 | 64.9 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 14:** Summary of competition match 12 impact biomechanics for impacts to the head greater (n=301) than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 20.8 ±14.1 | 15.6 [12.1-24.7] | 51.0 | 80.0 | 10.0 to 165.9 | 6,261.3 |
| **PRA (rad/s²)** | 3,484.8 ±3,037.3 | 2,619.7 [1,634.8-4,312.4] | 9,335.1 | 15,503.5 | 35.4 to 20,331.8 | 1,048,933.7 |
| **$HIC_{15}$** | 23.3 ±64.1 | 7.1 [4.2-15.8] | 101.4 | 272.8 | 1.1 to 879.5 | 7,013.0 |
| **GSI** | 36.2 ±89.3 | 13.4 [7.0-27.1] | 139.7 | 411.9 | 1.9 to 1,092.4 | 10,909.7 |
| **HITsp** | 20.0 ±15.0 | 15.6 [13.0-20.6] | 45.3 | 82.6 | 11.4 to 176.0 | 6,020.0 |
| **GAMBIT** | 0.133 ±0.092 | 0.106 [0.074-0.156] | 0.308 | 0.525 | 0.033 to 0.676 | 39.9 |
| **ms** | 11.1 ±8.6 | 8.0 [5.0-15.0] | 32.0 | 39.0 | 1.0 to 43.0 | 3,343.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.018 | 0.087 |
| **$RWE_{Rotational}$** | 0.134 ±0.303 | 0.001 [0.000-0.020] | 0.998 | 1.000 | 0.000 to 1.000 | 40.5 |
| **$RWE_{CP}$** | 0.050 ±0.186 | 0.001 [0.000-0.005] | 0.317 | 0.997 | 0.000 to 1.000 | 15.1 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 15:** Summary of competition match 13 impact biomechanics for impacts to the head (n=556) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.2 ±14.6 | 15.6 [12.0-25.2] | 53.7 | 88.1 | 10.0 to 165.9 | 11,144.0 |
| **PRA (rad/s²)** | 4,020.5 ±3,675.7 | 2,865.1 [1,678.1-5,053.5] | 11,597.3 | 17,818.3 | 7.9 to 32,720.4 | 2,110,746.7 |
| **$HIC_{15}$** | 23.3 ±60.4 | 6.7 [3.5-16.2] | 91.7 | 350.5 | 1.1 to 743.0 | 12,226.3 |
| **GSI** | 37.2 ±94.1 | 12.4 [6.2-28.0] | 146.8 | 597.6 | 1.8 to 1,173.1 | 19,515.9 |
| **HITsp** | 16.9 ±12.6 | 13.5 [11.7-18.0] | 36.3 | 67.5 | 6.5 to 146.9 | 8,897.3 |
| **GAMBIT** | 0.148 ±0.114 | 0.111 [0.070-0.179] | 0.375 | 0.646 | 0.026 to 0.911 | 77.8 |
| **ms** | 10.4 ±7.8 | 8.0 [5.0-15.0] | 26.0 | 37.0 | 1.0 to 42.0 | 5,481.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.013 | 0.153 |
| **$RWE_{Rotational}$** | 0.179 ±0.350 | 0.001 [0.000-0.081] | 1.000 | 1.000 | 0.000 to 1.000 | 94.1 |
| **$RWE_{CP}$** | 0.079 ±0.232 | 0.001 [0.000-0.007] | 0.777 | 0.999 | 0.000 to 1.000 | 41.7 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 16:** Summary of competition match 14 impact biomechanics for impacts to the head (n=983) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 27.4 ±18.0 | 21.2 [13.3-36.2] | 61.5 | 91.6 | 10.0 to 165.9 | 25,797.4 |
| **PRA (rad/s²)** | 5,210.5 ±4,078.5 | 3,919.7 [2,144.1-7,485.9] | 13,468.6 | 17,601.8 | 26.8 to 22,535.7 | 4,913,505.5 |
| **$HIC_{15}$** | 43.9 ±73.0 | 12.5 [4.9-50.3] | 201.1 | 354.0 | 1.0 to 559.9 | 41,408.5 |
| **GSI** | 67.2 ±108.0 | 21.1 [9.5-74.5] | 284.9 | 533.0 | 1.6 to 782.8 | 63,361.6 |
| **HITsp** | 17.7 ±14.2 | 12.5 [9.2-20.5] | 47.4 | 76.8 | 4.9 to 103.8 | 16,720.3 |
| **GAMBIT** | 0.189 ±0.127 | 0.155 [0.090-0.259] | 0.440 | 0.597 | 0.029 to 0.693 | 178.0 |
| **ms** | 13.9 ±9.7 | 11.0 [7.0-20.0] | 35.0 | 40.6 | 1.0 to 44.0 | 13,080.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.001 | 0.006 | 0.000 to 0.025 | 0.439 |
| **$RWE_{Rotational}$** | 0.317 ±0.431 | 0.009 [0.000-0.920] | 1.000 | 1.000 | 0.000 to 1.000 | 298.6 |
| **$RWE_{CP}$** | 0.158 ±0.308 | 0.003 [0.000-0.086] | 0.965 | 0.999 | 0.000 to 1.000 | 148.6 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 17:** Summary of competition match Semi-Final 1 impact biomechanics for impacts to the head (n=681) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 18.5 ±14.2 | 12.0 [11.4-19.1] | 47.9 | 79.1 | 10.1 to 165.9 | 21,437.0 |
| **PRA (rad/s²)** | 2,646.7 ±3,517.8 | 1,779.9 [8.1-3,720.8] | 10,058.4 | 16,030.0 | 4.4 to 20,112.2 | 3,067,528.6 |
| **$HIC_{15}$** | 19.7 ±56.3 | 5.7 [5.3-10.2] | 71.7 | 258.3 | 1.3 to 699.5 | 22,854.7 |
| **GSI** | 32.7 ±80.5 | 14.4 [11.2-17.9] | 117.2 | 359.1 | 2.5 to 984.6 | 37,917.6 |
| **HITsp** | 18.0 ±13.4 | 14.3 [14.0-16.8] | 36.1 | 81.4 | 7.1 to 158.6 | 20,807.7 |
| **GAMBIT** | 0.109 ±0.108 | 0.074 [0.035-0.138] | 0.335 | 0.530 | 0.031 to 0.783 | 126.3 |
| **ms** | 14.2 ±10.1 | 11.0 [6.0-20.0] | 36.0 | 37.0 | 1.0 to 41.0 | 16,513.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.003 | 0.000 to 0.016 | 0.324 |
| **$RWE_{Rotational}$** | 0.125 ±0.308 | 0.000 [0.000-0.006] | 0.999 | 1.000 | 0.000 to 1.000 | 144.7 |
| **$RWE_{CP}$** | 0.060 ±0.210 | 0.000 [0.000-0.002] | 0.551 | 0.996 | 0.000 to 1.000 | 70.0 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 18:** Summary of competition match Semi-Final 2 impact biomechanics for impacts to the head (n=1,212) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 29.3 ±16.4 | 25.3 [15.7-38.6] | 61.0 | 83.5 | 10.0 to 165.9 | 35,492.6 |
| **PRA (rad/s²)** | 5,704.8 ±3,520.9 | 4,871.8 [3,008.3-7,893.6] | 11,989.4 | 16,701.9 | 673.2 to 21,161.1 | 6,914,252.3 |
| **$HIC_{15}$** | 44.0 ±65.3 | 20.5 [6.8-54.2] | 168.2 | 363.6 | 1.1 to 649.4 | 53,305.1 |
| **GSI** | 68.7 ±98.0 | 32.9 [11.0-85.1] | 266.5 | 487.9 | 1.6 to 839.5 | 83,270.3 |
| **HITsp** | 16.0 ±13.2 | 11.6 [7.7-19.6] | 39.7 | 72.9 | 3.2 to 114.5 | 19,448.6 |
| **GAMBIT** | 0.205 ±0.117 | 0.185 [0.117-0.271] | 0.426 | 0.558 | 0.031 to 0.767 | 248.4 |
| **ms** | 14.9 ±9.9 | 12.0 [8.0-21.0] | 36.0 | 39.0 | 1.0 to 46.0 | 18,088.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.004 | 0.000 to 0.020 | 0.497 |
| **$RWE_{Rotational}$** | 0.370 ±0.436 | 0.058 [0.001-0.963] | 1.000 | 1.000 | 0.000 to 1.000 | 448.3 |
| **$RWE_{CP}$** | 0.150 ±0.279 | 0.007 [0.001-0.131] | 0.902 | 0.998 | 0.000 to 1.000 | 181.4 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 19:** Summary of competition Grand Final match impact biomechanics for impacts to the head (n=1,018) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95% | 99% | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 23.2 ±15.6 | 17.5 [12.0-28.3] | 56.0 | 87.3 | 10.0 to 165.9 | 23,425.3 |
| **PRA (rad/s$^2$)** | 4,265.2 ±3,422.4 | 3,295.0 [1,969.7-5,589.5] | 11,458.8 | 15,840.1 | 11.8 to 21,780.8 | 4,312,126.3 |
| **$HIC_{15}$** | 30.2 ±60.7 | 8.3 [4.2-27.6] | 129.9 | 338.3 | 1.0 to 582.0 | 30,552.4 |
| **GSI** | 51.6 ±104.8 | 14.6 [7.1-42.2] | 244.1 | 516.9 | 1.5 to 1,025.3 | 52,172.9 |
| **HITsp** | 18.5 ±14.7 | 13.3 [10.7-19.7] | 47.9 | 82.5 | 5.6 to 137.3 | 18,727.8 |
| **GAMBIT** | 0.166 ±0.128 | 0.125 [0.077-0.208] | 0.441 | 0.634 | 0.025 to 0.763 | 167.7 |
| **ms** | 12.7 ±9.9 | 9.0 [5.0-18.0] | 34.0 | 39.0 | 1.0 to 44.0 | 12,837.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.005 | 0.000 to 0.012 | 0.318 |
| **$RWE_{Rotational}$** | 0.221 ±0.381 | 0.003 [0.000-0.206] | 1.000 | 1.000 | 0.000 to 1.000 | 223.6 |
| **$RWE_{CP}$** | 0.090 ±0.236 | 0.001 [0.000-0.014] | 0.838 | 0.998 | 0.000 to 1.000 | 91.4 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

# Player Positional Group Impact Biomechanics

**Table 1:** Summary of competition match impact biomechanics of forwards for impacts to the head (n=9,875) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 24.6 ±16.2 | 18.9 [12.9-30.4] | 57.4 | 57.4 | 10.0 to165.9 | 242,504.3 |
| **PRA (rad/s$^2$)** | 4,287.2 ±3,651.0 | 3,171.1 [1,820.5-5,706.2] | 11,788.8 | 11,788.8 | 4.1 to32,720.4 | 42,336,421.6 |
| **$HIC_{15}$** | 33.4 ±68.8 | 10.3 [4.8-30.9] | 138.5 | 138.5 | 0.9 to1,086.75 | 330,221.2 |
| **GSI** | 41.0 ±80.6 | 13.6 [7.1-38.5] | 173.1 | 173.1 | 1.0 to1,206.19 | 395,792.5 |
| **HITsp** | 52.0 ±102.6 | 16.9 [8.8-49.1] | 220.7 | 220.7 | 0.0 to1,494.60 | 513,557.8 |
| **GAMBIT** | 0.163 ±0.119 | 0.127 [0.080-0.212] | 0.403 | 0.403 | 0.015 to0.987 | 1610.353 |
| **ms** | 12.9 ±9.1 | 10.0 [6.0-18.0] | 32.2 | 32.2 | 1.0 to46.0 | 127803.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to0.202 | 3.842 |
| **$RWE_{Rotational}$** | 0.223 ±0.382 | 0.002 [0.000-0.246] | 1.000 | 1.000 | 0.000 to1.000 | 2,205.0 |
| **$RWE_{CP}$** | 0.098 ±0.248 | 0.001 [0.000-0.017] | 0.866 | 0.866 | 0.000 to 1.000 | 964.8 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 2:** Summary of competition match impact biomechanics of forwards for impacts to the front of the head (n=2,869) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 26.9 ±16.6 | 21.3 [14.9-33.6] | 61.0 | 61.0 | 10.0 to 127.4 | 77,040.8 |
| **PRA (rad/s$^2$)** | 4,748.7 ±3,545.9 | 3,675.4 [2,347.0-5,980.8] | 12,335.0 | 12,335.0 | 29.9 to 28,882.6 | 13,624,065.1 |
| **$HIC_{15}$** | 38.6 ±72.5 | 13.7 [6.0-38.7] | 163.1 | 163.1 | 0.9 to 1,086.75 | 110,764.0 |
| **GSI** | 41.0 ±80.6 | 13.6 [7.1-38.5] | 173.1 | 173.1 | 1.0 to 1,206.19 | 395,792.5 |
| **HITsp** | 59.6 ±109.1 | 22.2 [10.0-59.3] | 251.7 | 251.7 | 0.0 to 1,371.70 | 170,861.8 |
| **GAMBIT** | 0.184 ±0.117 | 0.149 [0.104-0.230] | 0.424 | 0.424 | 0.019 to 0.909 | 526.934 |
| **ms** | 14.0 ±8.9 | 11.0 [7.0-19.0] | 32.0 | 32.0 | 1.0 to 44.0 | 40137.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.034 | 1.158 |
| **$RWE_{Rotational}$** | 0.241 ±0.387 | 0.006 [0.000-0.361] | 1.000 | 1.000 | 0.000 to 1.000 | 691.6 |
| **$RWE_{CP}$** | 0.104 ±0.256 | 0.002 [0.001-0.023] | 0.914 | 0.914 | 0.000 to 1.000 | 298.4 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 3:** Summary of competition match impact biomechanics of forwards for impacts to the back of the head (n=2,733) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 26.4 ±16.1 | 21.5 [14.4-33.3] | 58.3 | 58.3 | 10.0 to 121.4 | 72,082.8 |
| **PRA (rad/s$^2$)** | 4,796.9 ±3,710.2 | 3,590.0 [2,152.0-6,501.0] | 12,030.5 | 12,030.5 | 104.3 to 32,720.4 | 13,110,042.8 |
| **$HIC_{15}$** | 37.5 ±74.1 | 13.5 [4.9-37.8] | 142.0 | 142.0 | 0.9 to 879.49 | 102,512.4 |
| **GSI** | 41.0 ±80.6 | 13.6 [7.1-38.5] | 173.1 | 173.1 | 1.0 to 1,206.19 | 395,792.5 |
| **HITsp** | 57.6 ±109.6 | 21.1 [7.7-59.5] | 235.2 | 235.2 | 0.0 to 1,316.15 | 157,508.7 |
| **GAMBIT** | 0.177 ±0.123 | 0.141 [0.087-0.231] | 0.422 | 0.422 | 0.026 to 0.987 | 485.079 |
| **ms** | 13.2 ±9.1 | 11.0 [7.0-18.0] | 33.0 | 33.0 | 1.0 to 44.0 | 36082.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.026 | 1.088 |
| **$RWE_{Rotational}$** | 0.270 ±0.404 | 0.005 [0.000-0.616] | 1.000 | 1.000 | 0.000 to 1.000 | 737.9 |
| **$RWE_{CP}$** | 0.115 ±0.263 | 0.002 [0.000-0.035] | 0.888 | 0.888 | 0.000 to 1.000 | 313.2 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 4:** Summary of competition match impact biomechanics of forwards for impacts to the side of the head (n=3,963) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.3 ±15.5 | 15.0 [11.4-24.5] | 53.4 | 53.4 | 10.0 to 165.9 | 84,320.8 |
| **PRA (rad/s$^2$)** | 3,486.1 ±3,532.6 | 2,397.2 [1,221.0-4,379.4] | 11,003.2 | 11,003.2 | 4.1 to 30,270.0 | 13,815,286.4 |
| **$HIC_{15}$** | 26.0 ±61.7 | 6.4 [4.4-18.1] | 115.2 | 115.2 | 1.0 to 1,052.52 | 102,951.2 |
| **GSI** | 41.0 ±80.6 | 13.6 [7.1-38.5] | 173.1 | 173.1 | 1.0 to 1,206.19 | 395,792.5 |
| **HITsp** | 41.4 ±92.3 | 13.5 [8.3-29.1] | 180.4 | 180.4 | 0.0 to 1,494.60 | 164,058.7 |
| **GAMBIT** | 0.135 ±0.112 | 0.096 [0.064-0.164] | 0.366 | 0.366 | 0.018 to 0.956 | 534.654 |
| **ms** | 11.7 ±9.1 | 9.0 [5.0-16.0] | 32.0 | 32.0 | 1.0 to 46.0 | 46447.0 |
| **$RWE_{Linear}$** | 0.000 ±0.004 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.202 | 1.483 |
| **$RWE_{Rotational}$** | 0.166 ±0.347 | 0.000 [0.000-0.022] | 1.000 | 1.000 | 0.000 to 1.000 | 658.9 |
| **$RWE_{CP}$** | 0.076 ±0.225 | 0.001 [0.000-.004] | 0.770 | 0.770 | 0.000 to 1.000 | 302.8 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 5:** Summary of competition match impact biomechanics of forwards for impacts to the top of the head (n=310) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 29.2 ±14.6 | 26.4 [17.6-38.1] | 58.2 | 58.2 | 10.2 to 107.9 | 9,059.8 |
| **PRA (rad/s$^2$)** | 5,764.6 ±3,583.1 | 4,758.4 [3,039.9-8,051.6] | 12,217.4 | 12,217.4 | 283.9 to 17,900.1 | 1,787,027.4 |
| **$HIC_{15}$** | 45.1 ±60.0 | 22.9 [9.6-57.4] | 175.2 | 175.2 | 1.0 to 501.42 | 13,993.6 |
| **GSI** | 41.0 ±80.6 | 13.6 [7.1-38.5] | 173.1 | 173.1 | 1.0 to 1,206.19 | 395,792.5 |
| **HITsp** | 68.2 ±89.4 | 35.2 [14.1-86.4] | 259.9 | 259.9 | 0.0 to 686.02 | 21,128.5 |
| **GAMBIT** | 0.205 ±0.111 | 0.182 [0.121-0.272] | 0.419 | 0.419 | 0.015 to 0.620 | 63.687 |
| **ms** | 16.6 ±9.7 | 15.0 [9.0-23.0] | 35.0 | 35.0 | 1.0 to 42.0 | 5137.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.013 | 0.113 |
| **$RWE_{Rotational}$** | 0.376 ±0.440 | 0.047 [0.002-0.973] | 1.000 | 1.000 | 0.000 to 1.000 | 116.6 |
| **$RWE_{CP}$** | 0.163 ±0.294 | 0.007 [0.001-0.150] | 0.927 | 0.927 | 0.000 to 0.999 | 50.4 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability. * = unable to calculate

**Table 6:** Summary of competition match impact biomechanics of backs for impacts to the head (n=4,020) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95$^{th}$ | 99$^{th}$ | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 19.8 ±13.7 | 14.5 [11.6-22.0] | 48.2 | 48.2 | 10.0 to 124.1 | 79,545.9 |
| **PRA (rad/s$^2$)** | 3,705.7 ±3,267.0 | 2,804.9 [1,564.0-4,814.6] | 10,728.5 | 10,728.5 | 11.5 to 25,108.8 | 14,897,001.2 |
| **$HIC_{15}$** | 24.7 ±59.4 | 5.2 [2.8-13.6] | 79.6 | 79.6 | 0.6 to 735.65 | 76,126.6 |
| **GSI** | 28.4 ±73.7 | 7.7 [3.7-18.4] | 101.6 | 101.6 | 0.9 to 773.25 | 86,092.6 |
| **HITsp** | 18.7 ±14.0 | 8.0 [3.6-20.7] | 124.3 | 124.3 | 0.0 to 1,173.10 | 114,354.6 |
| **GAMBIT** | 0.134 ±0.103 | 0.103 [0.065-0.166] | 0.341 | 0.341 | 0.019 to 0.925 | 537.842 |
| **ms** | 9.1 ±7.1 | 7.0 [4.0-11.0] | 24.0 | 24.0 | 1.0 to 44.0 | 36387.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.029 | 1.075 |
| **$RWE_{Rotational}$** | 0.000 ±0.000 | 0.001 [0.000-0.052] | 1.000 | 1.000 | 0.000 to 1.000 | 647.9 |
| **$RWE_{CP}$** | 0.000 ±0.000 | 0.001 [0.000-0.006] | 0.678 | 0.678 | 0.000 to 1.000 | 266.9 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 7:** Summary of competition match impact biomechanics of backs for impacts to the front of the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.8 ±14.9 | 16.2 [12.7-24.6] | 54.4 | 54.4 | 10.0 to 119.2 | 25,710.2 |
| **PRA (rad/s²)** | 4,509.4 ±3,213.3 | 3,507.1 [2,445.8-5,460.7] | 11,903.9 | 11,903.9 | 161.9 to 21,715.0 | 5,307,590.8 |
| **$HIC_{15}$** | 24.7 ±59.4 | 7.0 [3.7-17.5] | 103.4 | 103.4 | 0.9 to 538.55 | 26,736.0 |
| **GSI** | 35.2 ±80.5 | 7.7 [3.7-18.4] | 101.6 | 101.6 | 0.9 to 773.25 | 86,092.6 |
| **HITsp** | 19.1 ±14.1 | 11.5 [6.0-27.2] | 149.8 | 149.8 | 0.0 to 1,173.10 | 41,484.7 |
| **GAMBIT** | 0.161 ±0.108 | 0.130 [0.092-0.193] | 0.395 | 0.395 | 0.019 to 0.763 | 189.000 |
| **ms** | 10.3 ±7.4 | 8.0 [5.0-13.0] | 26.0 | 26.0 | 1.0 to 44.0 | 12166.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.023 | 0.362 |
| **$RWE_{Rotational}$** | 0.000 ±0.000 | 0.004 [0.000-0.167] | 1.000 | 1.000 | 0.000 to 1.000 | 235.6 |
| **$RWE_{CP}$** | 0.000 ±0.000 | 0.002 [0.001-0.011] | 0.846 | 0.846 | 0.000 to 1.000 | 102.9 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 8:** Summary of competition match impact biomechanics of backs for impacts to the back of the head (n=1,062) greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 21.0 ±14.5 | 15.7 [12.1-23.9] | 49.8 | 49.8 | 10.0 to 124.1 | 22,315.3 |
| **PRA (rad/s²)** | 4,289.3 ±3,662.0 | 3,148.4 [1,727.2-5,564.1] | 11,713.4 | 11,713.4 | 432.1 to 25,108.8 | 4,555,285.6 |
| **$HIC_{15}$** | 24.7 ±59.4 | 5.9 [3.3-15.1] | 87.7 | 87.7 | 0.9 to 735.65 | 24,262.6 |
| **GSI** | 34.1 ±91.6 | 7.7 [3.7-18.4] | 101.6 | 101.6 | 0.9 to 773.25 | 86,092.6 |
| **HITsp** | 19.1±15.2 | 9.0[5.0-23.2] | 135.4 | 135.4 | 0.0 to 1,061.65 | 36,223.9 |
| **GAMBIT** | 0.144±0.112 | 0.107[0.073-0.178] | 0.361 | 0.361 | 0.033 to 0.925 | 153.383 |
| **ms** | 9.0 6.9 | 7.0 [4.0-11.0] | 24.0 | 24.0 | 1.0 to 44.0 | 9608.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.029 | 0.327 |
| **$RWE_{Rotational}$** | 0.000 ±0.000 | 0.002 [0.000-0.197] | 1.000 | 1.000 | 0.000 to 1.000 | 221.5 |
| **$RWE_{CP}$** | 0.000 ±0.000 | 0.001 [0.000-0.011] | 0.855 | 0.855 | 0.000 to 1.000 | 92.1 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 9:** Summary of competition match impact biomechanics of backs for impacts to the side of the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75th interquartile range], 95th percentile, 99th percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25th-75th] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 17.2 ±11.6 | 12.4 [11.2-18.1] | 39.3 | 39.3 | 10.0 to 103.7 | 28,834.7 |
| **PRA (rad/s²)** | 2,693.0 ±2,698.4 | 1,898.6 [1,013.7-3,499.5] | 7,980.0 | 7,980.0 | 11.5 to 19,956.4 | 4,521,544.6 |
| **$HIC_{15}$** | 24.7 ±59.4 | 3.6 [2.3-9.0] | 52.5 | 52.5 | 0.6 to 470.33 | 21,727.7 |
| **GSI** | 18.8 ±49.9 | 7.7 [3.7-18.4] | 101.6 | 101.6 | 0.9 to 773.25 | 86,092.6 |
| **HITsp** | 18.2 ±12.8 | 4.4 [2.4-14.3] | 76.7 | 76.7 | 0.0 to 597.80 | 31,581.6 |
| **GAMBIT** | 0.105 ±0.085 | 0.072 [0.048-0.133] | 0.285 | 0.285 | 0.028 to 0.702 | 176.963 |
| **ms** | 7.9 ±6.7 | 6.0 [3.0-10.0] | 22.0 | 22.0 | 1.0 to 42.0 | 13319.0 |
| **$RWE_{Linear}$** | 0.000 ±0.001 | 0.000 [0.000-0.000] | 0.000 | 0.000 | 0.000 to 0.011 | 0.336 |
| **$RWE_{Rotational}$** | 0.000 ±0.000 | 0.000 [0.000-0.004] | 0.969 | 0.969 | 0.000 to 1.000 | 164.9 |
| **$RWE_{CP}$** | 0.000 ±0.000 | 0.000 [0.000-0.002] | 0.145 | 0.145 | 0.000 to 1.000 | 59.6 |

PLA (g) = peak linear acceleration; PRA (rad/s²) = peak rotational acceleration in radians/second/second (rad/s²); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability.

**Table 10:** Summary of competition match impact biomechanics of backs for impacts to the top of the head greater than 10*g* recorded by an instrumented patch in a senior amateur rugby league team for the mean (±SD), median [25-75$^{th}$ interquartile range], 95$^{th}$ percentile, 99$^{th}$ percentile, range and cumulative (sum of all impacts) total.

| | Mean ±SD | Median [25$^{th}$-75$^{th}$] | 95th | 99th | Range | Cumulative |
|---|---|---|---|---|---|---|
| **PLA (*g*)** | 26.3 ±16.5 | 21.3 [15.8-30.5] | 63.7 | 63.7 | 10.1 to 117.0 | 2,685.8 |
| **PRA (rad/s$^2$)** | 5,025.3 ±3,566.7 | 3,876.5 [2,740.0-6,405.3] | 12,190.9 | 12,190.9 | 832.4 to 18,907.1 | 512,580.2 |
| **$HIC_{15}$** | 24.7 ±59.4 | 13.1 [6.9-26.5] | 138.0 | 138.0 | 1.3 to 471.17 | 3,400.3 |
| **GSI** | 49.7 ±92.3 | 7.7 [3.7-18.4] | 101.6 | 101.6 | 0.9 to 773.25 | 86,092.6 |
| **HITsp** | 21.1 ±16.2 | 18.9 [10.3-40.7] | 212.0 | 212.0 | 0.0 to 633.95 | 5,064.4 |
| **GAMBIT** | 0.181 ±0.112 | 0.152 [0.112-0.217] | 0.381 | 0.381 | 0.023 to 0.720 | 18.496 |
| **ms** | 12.7 ±7.7 | 11.0 [7.0-17.0] | 29.9 | 29.9 | 1.0 to 40.0 | 1294.0 |
| **$RWE_{Linear}$** | 0.000 ±0.002 | 0.000 [0.000-0.000] | 0.001 | 0.001 | 0.000 to 0.021 | 0.050 |
| **$RWE_{Rotational}$** | 0.000 ±0.000 | 0.008 [0.001-0.569] | 1.000 | 1.000 | 0.000 to 1.000 | 25.9 |
| **$RWE_{CP}$** | 0.000 ±0.000 | 0.002 [0.001-0.027] | 0.962 | 0.962 | 0.000 to 1.000 | 12.3 |

PLA (g) = peak linear acceleration; PRA (rad/s$^2$) = peak rotational acceleration in radians/second/second (rad/s$^2$); $HIC_{15}$ = Head Impact Criterion (15 ms); GSI = Gadd Severity Index; HITsp = Head Impact Telemetry severity profile; GAMBIT = Generalised Acceleration Model for Brain Injury Threshold; ms = impact duration in milliseconds; $RWE_{Linear}$ = Risk Weighted Exposure Linear accelerations; $RWE_{Rotational}$ = Risk Weighted Exposure Rotational accelerations; $RWE_{CP}$ = Risk Weighted Exposure Combined (linear and rotational) Probability; * = unable to calculate