An Initial Assessment of Head Acceleration Events in Rugby League using Instrumented Mouthguards and Qualitative Video Analysis

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The candidate confirms that the work submitted is his own and that appropriate credit has been given where reference has been made to the work of others.

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Publications

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Abstract

There is a growing concern that the long-term effects potentially associated with contact sport are not only caused by concussions, but by the accumulation of subconcussive impacts. It is therefore important to understand the head acceleration event (HAE) exposure sustained by contact sports individuals. In this thesis, a combination of qualitative video analysis and biomechanical instrumented mouthguard data is used to quantify and characterise HAE sustained by professional male rugby league players during competitive matches. Peak linear acceleration (PLA), peak angular acceleration (PAA) and peak change in angular velocity (ΔPAV) were collected using custom-fit instrumented mouthguards with an 8 g or 800 rad/s^2 resultant data acquisition threshold. A total of 725 HAE were collected from 31 player matches throughout 10 male players. Multiple HAE often occurred in the same tackle phase and 47.6% of HAE occurred after the initial contact between a tackler and the ball-carrier. Indirect HAE accounted for around half of HAE from carries (50.4%) and a quarter of HAE from tackles (23.8%), they also led to significantly greater ΔPAV than direct HAE ($p < 0.001$, ES = 0.69, interpretation = moderate). Each kinematic was also skewed to lower values, therefore applying a data acquisition threshold of 10 g led to an exposure rate of around half (13.8 ± 6.9 HAE per player, per match) than the exposure rate reported using an 8 g or 800 rad/s^2 threshold (28.0 ± 10.6 HAE per player, per match). The results and methodology from this study demonstrate the efficacy of a combination of biomechanical data with qualitative video analysis in quantifying and characterising HAE exposure in rugby league and will form the basis for a future league wide instrumented mouthguard study.

Abbreviations

Abbreviation	Phrase
ATD	Anthropometric Test Dummy
BAM	Brain Angle Metric
BIC	Brain Injury Criteria
BRIC	Kinematic Brain Injury Criterion
BrIC	Brain Injury Criterion
CG	Centre of Gravity
CIR	Concussion Interchange Rule
CISG	Concussion in Sports Group
CP	Combined Probability
CSDM	Cumulative Strain Damage Measure
cSP	Cortical Silent Period
CTE	Chronic Traumatic Encephalopathy
DTI	Diffuse Tensor Imaging
ES	Effect Size
ESL	European Super League
FE	Finite Element
fMRI	Functional Magnetic Resonance Imaging
GRTP	Graduated Return to Play
GSI	Gadd's Severity Index
HAE	Head Acceleration Event
HIA	Head Injury Assessment
HIC	Head Injury Criterion
HIP	Head Impact Power
ImPACT	Immediate Post Concussion Assessment and Cognitive Testing
KLC	Kleiven's Linear Combination
LICI	Long Interval Intracortical Inhibition
LOC	Loss of Consciousness
MPS	Maximum Principle Strain
MRS	Magnetic Resonance Spectroscopy
NFL	National Football League
NRL	National Rugby League of Australia
PAA	Peak Angular Acceleration
PAV	Peak Angular Velocity
PCS	Principle Component Score
PLA	Peak Linear Acceleration
PRHIC	Power Rotation Head Injury Criterion
PTC	Post Tackle Contact
RIC	Rotational Injury Criterion
ROI	Region of Interest
SCAT-5	Sports Concussion Assessment Tool 5
ΔPAV	Peak Change in Angular Velocity

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1 Introduction

Originating in the north of England following the rugby split in August 1895 (Collins, 2013), rugby league is a popular sport played around the world at different competition levels (King and Gissane, 2009). The team collision sport is extremely physical, with thirteen players on each side trying to carry the ball over the opposition's touchline. Progress down the field is limited to kicking and carrying, whilst passes must not be thrown in a forward direction. Each team is allowed to be tackled six times in possession before returning the ball to the opposition and resuming play immediately, as such the same players are involved in offence and defence (Gabbett, 2004). Games are played for eighty minutes over two halves, with no breaks between offense and defence. Inherently, due to the high number of collisions players are involved in (Naughton et al., 2020) rugby league participation implies a risk of both musculoskeletal and head injuries (Gabbett, 2004). Recently, increased media coverage of the potential short- and long-term effects of concussions and repetitive head impacts has been met by rule changes and increased awareness within rugby league, as well as a growing body of research into the mechanisms of head injuries. Biomechanical studies have identified that head accelerations can be caused by inertial loading from body impacts as well as direct head impacts, as such the term head acceleration event (HAE) is used henceforth. Research quantifying and characterising the exposure of HAE and concussions in rugby league is less established when compared with other sports such as American Football, however advancements in wearable head impact sensor technology have given rise to many studies across non-helmeted sports measuring the exposure and magnitude of HAE sustained by players during games. Therefore, it is the aim of this study to use a combination of biomechanical instrumented mouthguard data and qualitative video analysis to quantify and characterise HAE sustained in professional male rugby league matchplay.

2 Literature Review

2.1 Head Injuries in Rugby League

2.1.1 Incidence of concussion

Unlike in rugby union (Kemp et al., 2019), there are no standardised and regulated injury surveillance systems in place to form a more reliable concussion incidence rate in professional rugby league. Instead, incidence rates are reported in systematic reviews (Gardner et al., 2015a, Koh et al., 2003) and papers reporting the incidence rates of individual teams (Savage et al., 2013, Hinton-Bayre et al., 2004) and leagues (Gardner et al., 2017a, Fitzpatrick et al., 2018, Stephenson et al., 1996). This has meant that studies reporting the incidence of concussion within rugby league are hampered by inconsistencies in the injury definitions used as well as the time periods measured.

Within the European Super League (ESL), the concussion incidence rate increased from 2 and 3 concussions per 1000 player hours in 2013 and 2014 respectively, to 8 concussions per 1000 player hours in 2015 (Fitzpatrick et al., 2018). This increase coincided with the introduction of concussion reporting rules in 2014 and the HIA protocol in 2015, so the increase likely signifies increased compliance in reporting concussions rather than an actual change in rate of the concussions. Higher concussion rates in professional rugby league have been reported from studies of the NRL, with Gardner et al. (2015b) reporting the incidence rate of three NRL clubs over the 2013 season at 14.8 concussions per 1000 player hours (Gardner et al., 2015b). Interestingly, a lower incidence rate in the 2014 NRL season across all teams was reported at 8.9 concussion per 1000 player hours (Gardner et al., 2016). This variability across seasons and samples demonstrates the need for a standardised and regulated injury surveillance system within rugby league. Moreover, the concussion rate should be monitored closely in order to identify increases or decreases that may arise following the introduction of new rules or new coaching techniques.

A pooled analysis of concussion rates in different levels of rugby league reported that concussion incidence rates were greater in studies of amateur rugby league than studies of professional, semi-professional and junior rugby league players (King et al.,

2017b). Research reporting the incidence rates of concussions sustained during rugby league training is extremely limited, with just one study reporting any concussions from professional training (Gissane et al., 2012). The study reported data from training and games from 1990 to 2003 and reported one case of concussion from 161,700 hours of training. This may suggest that the risk of concussion from training exposures is low, however more research is needed in order to gauge the training exposure in the modern game.

2.1.2 Concussion Mechanisms in Rugby

Tackling and carrying have consistently been identified as the most common cause of concussion within rugby league (Gabbett and Domrow, 2005, Gissane et al., 2003, Norton and Wilson, 1995, Gardner et al., 2016, King et al., 2012). It is unclear whether ball-carriers or tacklers are at a greater risk of concussion. A greater number of tacklers were removed from play than ball-carriers using the concussion interchange rule in the NRL (Gardner et al., 2016), however an older study reported that ball-carriers were not concussed more often than tacklers (Hinton-Bayre et al., 2004). Increased concussion rates have also been reported in forwards (Gardner et al., 2016) which may be explained by forwards being involved in more tackles than backs (McLellan et al., 2011, Cummins and Orr, 2015, Naughton et al., 2020).

Tackle height has been consistently identified as a risk factor (King et al., 2012, Gardner et al., 2016, Gardner et al., 2015b). Consistently, head-high tackles are the predominate cause of concussions in ball-carriers (Hinton-Bayre et al., 2004, Gardner et al., 2015b). However, tackle height appears to be less of a risk factor in tacklers. A similar number of concussion interchanges occurred after both upper body tackles and lower body tackles in the NRL in 2013 (Gardner et al., 2015b), whilst upper body tackles led to 67% of concussion interchanges in 2014 (Gardner et al., 2016). Given that lower body tackles are more common than upper body tackles in professional rugby league (King et al., 2010), this may suggest that upper body tackles pose a greater risk of concussion than lower body tackles for both tacklers and ball-carriers. More directed research in this area is required to inform potential rule changes and coaching techniques to reduce the risk of concussions. The proportion of illegal high tackles that led to concussions increased from 12% to 25% between 2013 and 2014 in the NRL (Gardner et al., 2016, Gardner et

al., 2015b). This may reflect stricter officiating in light of an increased awareness of concussion. Furthermore, this supports the notion that tougher sanctions for higher tackles could help to reduce the concussion incidence rate.

There is a lack of literature utilising qualitative video analysis to assess the risk factors and characteristics of tackles resulting in concussion in rugby league. Using literature from rugby union may be useful for generalising findings across to rugby league as a first step, and guiding research design. Within rugby union, it has been reported that active shoulder tackles, front-on tackles and higher speed tackles were all associated with an increased propensity to trigger a HIA, further, tacklers were involved in more HIAs than ball-carriers in head to head contact cases (Tucker et al., 2017). The authors also found that tacklers bent at the waist were 1.5 times less likely to trigger an HIA than tacklers that were upright, this supports the idea that tackle technique can influence the number of HIAs experienced (Tierney et al., 2018a). Technical, evidence-based coaching cues have been developed following an analysis of HIA cases in rugby union (Tierney et al., 2018a), a similar study is necessary for rugby league in order to help to improve technique to reduce the number of head injuries and concussion within the sport.

2.1.3 Subconcussion

Subconcussive impacts have been defined as cranial impacts which do not result in a diagnosed concussion (Bailes et al., 2013). They can be caused through indirect (inertial) loading of the head following rapid acceleration or deceleration of the body, which has been termed as the slosh phenomenon (Smith et al., 2012). The term HAE is more appropriate to incorporate both direct head impacts and head accelerations which are caused by indirect head loading through contact with the body.

The concept of subconcussion is problematic due to difficulty in defining the upper and lower boundaries of a subconcussive impact. The upper boundary of a subconcussive HAE relies on an accurate diagnosis and sensitive detection of concussive HAE. Many potentially concussive HAE will not be diagnosed due to officials not spotting the HAE and also misdiagnoses of concussions by the officials due to players ‘sandbagging’ the concussion assessments to avoid missing games (Higgins et al., 2017). Similarly, the

lower boundary of subconcussion is also unclear. In practice, the lower boundary of subconcussive impacts (and indeed all HAE) is governed by data acquisition thresholds employed within biomechanical studies measuring HAE.

The rationale for setting a data acquisition threshold is often to remove any HAE caused by non-contact events such as running, or jumping (King et al., 2016). This is because the clinical effects of lower magnitude HAE are not well-understood. Whilst comparisons of head kinematics and exposure rates between studies would benefit from a universal data acquisition threshold, a universal data acquisition threshold across different devices is not appropriate, given that different wearable HAE sensors report kinematics differently due to different sources of error (Wu et al., 2016, Kieffer et al., 2020a) (Section 2.4.1.). Similarly, previous studies (King et al., 2017c, King et al., 2018) have used head kinematics of non-contact events measured by a biteplate system (Ng et al., 2006) in order to set data acquisition thresholds in wearable patch devices, which may lead to under- or overreporting of HAE exposure. A more appropriate method of setting a data acquisition threshold would be to measure the head kinematics of non-contact events using the same device.

More recently, an envelope of head kinematics sustained during daily activities was published using a variety of devices (Miller et al., 2019). Figure 1 shows that all but one study reported peak accelerations caused by running to be under 4 g and 200 rad/s^2. The one study which reported the maximum accelerations sustained during running to be around 8 g and 800 rad/s^2 used an instrumented headband (Bussone and Prange, 2014). These results may suggest that studies using a 10 g threshold are likely to ignore HAE which are caused by contact events, and therefore underreport the subconcussive load. Whilst a 4 g and 200 rad/s^2 data acquisition threshold may include a greater number of HAE caused by contact events, this would lead to limitations in data collection by the wearable devices (Discussed in Section 5).

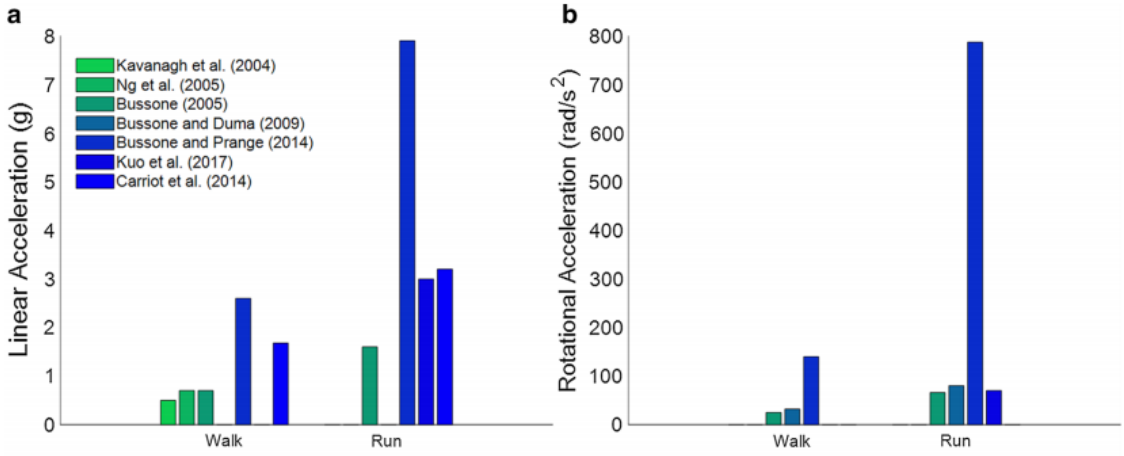

Figure 1. Comparison of linear acceleration (a) and rotational acceleration (b) for walking and running. Taken from Miller et al. (2019).

Until more is understood about the kinematics of HAE that are non-injurious, even with repetitive and prolonged exposure, then all HAE which exceed those caused by non-contact events should be included in studies investigating subconcussive exposure, without compromising the function of wearable devices in detecting higher magnitude HAE (Section 5). Furthermore, whilst appropriate thresholds must be set for specific devices, similar data acquisition thresholds should be used between studies to allow the comparison between sports and over time. Research elucidating the lower-bound of head kinematics that can injure axonal or neuronal integrity over time is required in order to set an appropriate threshold, as well as sport-specific studies elucidating the maximum head kinematics sustained from non-contact events such as juking and cutting manoeuvres. Another approach to this would be to measure the maximum head kinematics sustained in non-contact sports which are not currently associated with long-term effects to brain health, such as gymnastics.

2.1.4 Head Acceleration Events in Rugby League

There are a limited number of studies measuring HAE in rugby league players. Currently no studies have reported the kinematics sustained by elite professional rugby league players, with studies including amateur male players (King et al., 2017c, Carey et al., 2019), amateur female players (King et al., 2018), adolescent male players (Carey et al., 2020) and junior male players (King et al., 2017a). Existing studies in rugby league are limited by the use of the XPatch device, which is a wearable patch instrumented with

an accelerometer and gyroscope, that has a tendency to overestimate head acceleration (Wu et al., 2016). A detailed review of wearable impact sensors is included in Section 2.4.1. Furthermore, different linear acceleration data acquisition thresholds of 10 (King et al., 2017a, King et al., 2017c, King et al., 2018) and 20 *g* (Carey et al., 2019, Carey et al., 2020) limit comparison between these studies. No studies currently report the HAE exposure from training. This is an important area to understand in future studies as training hours will be far greater than game hours over the course of the career of a professional rugby league player, and so a large proportion of an athlete's cumulative HAE may be from training exposures.

All studies found that forwards sustained a greater number of HAE than backs (King et al., 2017a, King et al., 2020, King et al., 2018, Carey et al., 2019, Carey et al., 2020, King et al., 2017c). These findings are consistent with previous studies which reported that forwards were involved in a greater number of collisions per game than backs (Cummins and Orr, 2015, Naughton et al., 2020). Two studies reported higher peak linear and angular acceleration values amongst backs when compared with forwards (King et al., 2017a, King et al., 2018), whilst another study reported higher peak linear and angular acceleration values amongst forwards (King et al., 2017c).

Carey et al. (2020) combined qualitative video analysis with kinematic data and provided analysis of different HAE characteristics. The authors found that direct HAE accounted for around three quarters of all HAE above 20 g. The authors found that 46.7% of HAE from carries (ball-carrier impacted whilst carrying the ball into a tackle) were caused by the arm of the tackler, whilst just under a quarter of indirect HAE were caused by contacts between the shoulders of the ball-carrier and tackler, or the shoulder of the ball-carrier and the torso of the tackler. It was also reported that 46.1% of HAE occurred after the initial carry/tackle contact.

Further research measuring the HAE exposure in elite rugby players is required. Moreover, the combination of qualitative video analysis and kinematic data has so far been underused in rugby league, with just one study using video analysis beyond video verifying impacts (Carey et al., 2020). This combination has been effective in developing the understanding of HAE exposure in American Football (Tierney et al., 2020) and

similar study designs can develop our understanding of the nature of HAE in rugby league.

2.1.5 Direct and Indirect Head Loading

There are a limited number of studies that compare the head kinematics of direct and indirect HAE. Direct HAE are defined as HAE caused by direct contact to the head of the impacted player, and indirect HAE, often called inertial impacts, are caused by contacts with the body that lead to a transmission of forces to the head through the neck. Video analysis studies of HAE resulting in a CIR (Gardner et al., 2015b, Gardner et al., 2016) revealed that all concussions or CIR uses were the result of direct blows to the head, with no discussion in either study about indirect HAE. This suggests that indirect HAE may pose less of a risk of concussion than direct HAE. Despite this, indirect HAE have been shown to account for a large proportion of HAE in previous studies (Tierney et al., 2020, Carey et al., 2020, Kieffer et al., 2020b), so indirect HAE may play a significant role in cumulative head loading. Direct HAE accounted for a greater proportion of all HAE in junior rugby league players (Carey et al., 2020) and collegiate rugby players (Kieffer et al., 2020b) than indirect HAE, whilst in collegiate American Footballers (Tierney et al., 2020) indirect HAE were more common than direct HAE.

Mixed results have also been reported when the head kinematics of direct and indirect HAE were reported (Tierney et al., 2020, Carey et al., 2020, Kieffer et al., 2020b). Direct HAE have been reported to result in greater peak linear acceleration (PLA) (Kieffer et al., 2020b, Carey et al., 2020) and peak angular acceleration (PAA) (Carey et al., 2020), whilst peak change in angular velocity (ΔPAV) was not significantly different between direct and indirect HAE (Kieffer et al., 2020b). Conversely, Tierney et al. (2020) reported that PLA, PAA and peak angular velocity (PAV) were greater in indirect HAE when compared with direct HAE in collegiate American Footballers wearing instrumented mouthguards, with the biggest difference being seen in PAA (55% greater in angular acceleration and moderate differences in PLA and PAV). Typically, we would expect higher head kinematics for a direct HAE than indirect, given the same conditions. The conflicting results in collegiate american footballers (Tierney et al., 2020) may indicate that low magnitude direct HAE are common from light helmet to helmet contacts, which would reduce the median kinematics for direct HAE. The authors also suggested that

occluded direct head contact, or direct head contact occurring between frames may have led to direct HAE incorrectly labelled as indirect HAE. This may indicate that a single camera view may be insufficient to identfiy indirect and direct HAE.

2.1.6 Managing Subconcussive Load

It has been stated that managing player loads is an important factor in ensuring the welfare and minimising the injury of risk to rugby players (McNabb et al., 2020). The effects of HAE exposure are being investigated in a rapidly growing field of research (Section 2.3) but are not currently well understood in rugby league. Managing the HAE load sustained by players may be a proactive approach of mitigating the potential short- and long-term effects associated with repetitive HAE. Detailed biomechanical studies are needed to quantify the magnitude and frequency of HAE sustained in training and game exposures, especially at elite levels of the sport where exposure time will be greatest over a career. The characteristics of high-risk scenarios should be identified and used to inform strategies to reduce the number HAE sustained in training and game exposures. Furthermore, monitoring of HAE exposure sustained by individuals could be a powerful tool for team doctors, officials and coaches to promote the safety and well-being of their players. In the future, directly measuring the cumulative HAE exposure could help to inform team selection, coaching technique and rule changes from the perspective of managing HAE exposure.

2.2 Effects of Head Acceleration Exposure

A growing field of research is investigating the potential short- and long-term effects of repetitive exposure to HAE and concussions. Areas include mental health, cognitive functioning, motor function and neuropathology. Whilst most existing research is from other contact sports such as American Football, research of rugby populations is growing. Until more is understood about the differences in concussion rates and HAE exposure rates between rugby league players and players of other contact sports, findings from other sports may not be applicable to rugby league. Nonetheless, if adverse short- and long-term effects are reported in other contact sports similar study designs using rugby league populations may be necessary.

2.2.1 Concussion or Cumulative Head Accelerations

Montenigro et al. (2017) investigated a range of clinic outcome measures in former high school and college American football players and compared the predictive power of a range of different exposure metrics. The authors investigated later-life depression, apathy, executive dysfunction and cognitive impairment and exposure metrics included concussion history, total seasons played and age at first exposure, as well as a cumulative exposure metric cumulated head impact index (CHII). CHII was calculated for each athlete using individual self-report measures of athletic exposure and extrapolated objective measures from helmet accelerometer studies. There was a significant linear-dose relationship between CHII and each clinic outcome. Furthermore, the authors found that for each clinical outcome, CHII was a greater predictor of risk of clinical impairment than other exposure metrics, with a significant linear-dose relationship. This illustrates the importance of considering the accumulation of subconcussive HAE when assessing the risk of long-term clinical effects in contact sports players.

2.2.2 Mental Health

There is mixed evidence to suggest mental health is poorer in retired rugby populations (McMillan et al., 2017, Decq et al., 2016). A study of retired Scottish international rugby union players reported that there were no significant differences in mental health when compared with controls (McMillan et al., 2017), whilst a larger scale study of retired French national and international rugby union players reported higher rates of depression than controls (Decq et al., 2016). Surprisingly, the Scottish cohort reported a greater median number of concussions than the French cohort. The different results may be explained by different controls being used in either study. McMillan et al. (2017) compared mental health in retired rugby players to non-athlete controls, whilst Decq et al. (2016) compared mental health in retired rugby player to former athlete control. It is possible in the former study, the mental health benefits of participating in rugby may have offset mental health decrements caused by concussions and repetitive HAE, as athletes were compared to non-athlete controls. One study of retired rugby league players found that there were no significant differences in anxiety or depression in retired rugby league players when compared with controls, however this study used a

much smaller cohort when compared with the previously aforementioned studies of rugby populations (Guell et al., 2020). Further prospective group comparison studies with larger cohorts are necessary to elucidate the effect of HAE exposure on mental health in current and retired rugby league players.

2.2.3 Cognitive Functioning

Research of rugby populations has yielded mixed results. Performance on cognitive tests were not significantly different from controls in either retired international rugby union players (McMillan et al., 2017) or retired professional rugby league players (Gardner et al., 2017b). Conversely, the rate of mild cognitive disorders was greater in retired rugby union players than other retired sportsmen (Decq et al., 2016) and cognitive performance was worse in retired professional rugby league players when compared with controls (Pearce et al., 2018). Hume et al. (2017) reported that retired rugby union players with a history of concussion experienced small to moderate neurocognitive deficits when compared with retired rugby union players reporting no concussions. These areas included executive function, cognitive flexibility, and complex attention. Further, retired elite rugby union players performed worse than the retired amateur rugby union players and retired non-contact athletes in cognitive flexibility and complex attention. In retired American Footballers, the number of HAE sustained over an entire career exhibited a dose-response relationship with cognitive functioning (Montenigro et al., 2017). Mixed evidence to support that cognitive functioning is impaired in retired rugby league players (Pearce et al., 2018, Gardner et al., 2017b) may necessitate longitudinal research designs to elucidate the long-term effects of rugby league participation on cognitive functioning.

2.2.4 Motor Control and Function

Impaired Motor Function

Motor function been demonstrated to be impaired in retired rugby league (Gardner et al., 2017b) and rugby union (McMillan et al., 2017) populations. With retired players performing significantly worse on the grooved pegboard test (Matthews and Klove, 1964) when compared with healthy controls (Gardner et al., 2017b, McMillan et al., 2017), however in both studies only a significant difference was only reported in either the dominant (Gardner et al., 2017b) or non-dominant hand (McMillan et al., 2017). In a

study of retired Canadian American Footballers who sustained a median of 5 concussions each, no significant differences in the same test of manual dexterity were reported when compared with controls, despite the retired players reporting significantly more memory, behavioural and executive symptoms than controls (Tarazi et al., 2018). These findings suggests that the long-term effects of concussions and repetitive HAE on motor function may be exist in rugby populations, however more longitudinal research is needed.

Musculoskeletal Injuries

There is also growing evidence to suggest that impairments to motor control following concussion can lead to an increased risk of musculoskeletal injury (Chmielewski et al., 2020). This concept is supported by a retrospective study which found that an increased number of concussions sustained by retried players was associated with an increased incidence of musculoskeletal injuries over an entire career (Mihalik and Guskiewicz, 2015) as well as a study of rugby players which found that players with a history of concussion were twice as likely to have reported a lower extremity injury (Hunzinger et al., 2020). Moreover, athletes within a 90-day period of their return to play following a concussion exhibited almost double the incidence rate of lower extremity musculoskeletal injury of their non-concussed teammates (Brooks et al., 2016). The mechanism of this increased risk has been attributed to neuroanatomical and neurophysiological changes following concussion which lead to altered motor function, namely: compromised balance and postural stability, impaired movement patterns, decreased force production and muscle activation and impaired motor task performance (Chmielewski et al., 2020). It has been suggested that clinical management of concussion should address motor control deficits in order to reduce further risk of injury (Chmielewski et al., 2020).

Transcranial magnetic stimulation

Transcranial magnetic stimulation is a non-invasive technique of assessing cortical function (Martini and Broglio, 2018). Transcranial magnetic stimulation has been performed on a cohort of former professional rugby league players (Pearce et al., 2018) as well as a mixed cohort of former elite and community rugby union players and non-contact athletes (Lewis et al., 2017). These studies reported long-term changes to

corticomotor function following concussion. Both studies reported increased long interval intracortical inhibition (LICI) in the retired professional groups, whilst a reduced cortical silent period (cSP) in retired professional rugby players was reported by Pearce et al. (2018) but not by Lewis et al. (2017). Increases to the resting motor threshold have also been reported in retired Rugby Union players (Lewis et al., 2017), but not in retired rugby league players (Pearce et al., 2018). Reduced cSP and increased LICI have been associated with enhanced inhibition and impaired motor performance (Pearce et al., 2015, De Beaumont et al., 2012), whilst an increased resting motor threshold usually indicates an impaired excitability threshold of cortical motor neurons (Lewis et al., 2017). Therefore, these findings of altered corticomotor excitation and inhibition in retired rugby league and union players may demonstrate that clinically relevant changes may persist in the long-term. Similarly, a recent study measured corticomotor function of ball-carriers and tacklers using transcranial magnetic stimulation following a series subconcussive rugby tackles (McNabb et al., 2020). The authors found that after the tackles, cSP was significantly reduced compared to baselines levels in tacklers but not ball-carriers. This study provides evidence that subconcussive blows to tacklers resulted in acute changes to cSP. These studies provide evidence that short- and long-term changes to altered corticomotor excitability and impaired motor function are observable in rugby league and union populations. Longitudinal research with measures of cumulative HAE exposure are necessary to investigate these findings further.

2.2.5 Neuroimaging

Diffuse Tensor Imaging

Diffuse tensor imaging (DTI) is an magnetic resonance imaging based technique which reveals subtle changes in the integrity of white matter (Mukherjee et al., 2008) by measuring changes in water diffusion (Pierpaoli et al., 1996). Significant changes in the white matter of retired elite rugby league players have been detected (Wright et al., 2020). The authors postulate that the pattern of changes observed indicate a compromised white matter integrity due to damaged axons and myelin. Similarly, significant differences in changes in fractional anisotropy and mean diffusivity have been identified from pre- to post-season assessment between non-contact sport athletes and contact athletes, in the absence of any diagnosed concussions (McAllister et al., 2014, Bazarian et al., 2014, Gajawelli et al., 2013). Changes to fractional anisotropy (Wilde et

al., 2010) and mean diffusivity (Werring et al., 1999) have been associated with demyelination, and are typically ascribed to white matter integrity abnormalities (McAllister et al., 2014). These changes have been demonstrated to persist for up to 6 months despite a withdrawal from any contact sports (Bazarian et al., 2014), which the authors suggest may signify a lack of white matter recovery and may contribute to progressive, cumulative white matter changes with further prolonged exposure.

Objective measures of HAE exposure, as measured by wearable HAE sensors, have been combined with DTI measures and consistently demonstrated a linear relationship between HAE exposure variables and DTI measures (McAllister et al., 2014, Bahrami et al., 2016, Davenport et al., 2014, Kuzminski et al., 2018). This relationship has been demonstrated in college (McAllister et al., 2014), high-school (Davenport et al., 2014, Kuzminski et al., 2018) and youth (Bahrami et al., 2016) American Footballers. Another study reported that heading exposure, as measured by a self-report questionnaire, was correlated with changes to fractional anisotropy in three locations in temporo-cortical white matter in amateur soccer players (Lipton et al., 2013). These studies suggest that in the absence of a clinically diagnosed concussion, a single season of contact sport can elicit white matter changes detectable by DTI, moreover the relationship between HAE exposure and these diffusivity measure changes has been reported to be linear.

2.2.6 Neuropathology

Neurodegenerative Diseases

To date, no study on the cause of mortality or neurodegenerative disease incidence has been conducted in any rugby populations. In retired American Football populations, the total combined neurodegenerative disease mortality was three times greater than the rest of the population (Lehman et al., 2012). Alzheimer's disease and amyotrophic lateral sclerosis death rates were four times greater. Similarly, in a cohort of over 2,500 retired professional American footballers, there was an increased prevalence of Alzheimer's disease when compared with the general male population in the USA (Guskiewicz et al., 2005). The authors found that the greatest differences in Alzheimer's disease prevalence were observed younger ages (Figure 2), which may suggest that participation in American Football was associated with an earlier onset of Alzheimer's disease. The authors also found that a fivefold increase in the prevalence of mild cognitive impairment

was found in retired players that sustained three or more concussions, when compared with athletes with no concussions. Mackay et al. (2019) investigated a cohort of retired soccer players and found that death rates from neurodegenerative mortality in former soccer players was 3 times greater than neurodegenerative mortality in controls. Alzheimer's disease was identified as the greatest cause of neurodegenerative mortality and Parkinson's Disease the lowest. To elucidate the effects of rugby participation on neurodegenerative disease, it is imperative that retired populations and prospective cohorts are investigated.

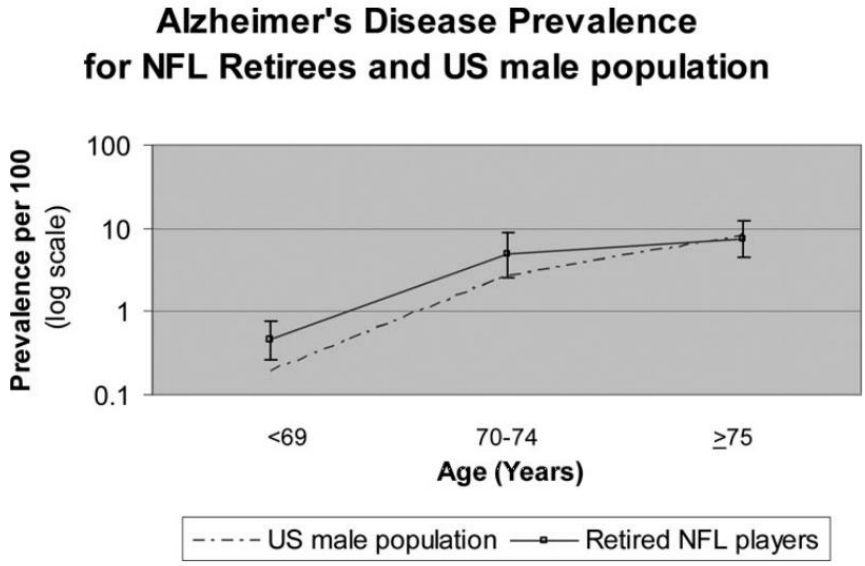

Figure 2. Alzheimer's Disease Prevalence ratios of NFL retirees and general male US population. Taken from Guskiewicz et al. (2005).

Chronic Traumatic Encephalopathy

First diagnosed in a former NFL player (Omalu et al., 2005), chronic traumatic encephalopathy (CTE) has now been reported in former rugby league players (Buckland et al., 2019), amongst other contact sport athletes and non-athletes. CTE is typically characterised as an accumulation of hyperphosphorylated tau in the brain, associated with repetitive brain trauma (McKee et al., 2013). It has been suggested that CTE represents the same condition initially diagnosed in boxers called dementia pugilistica, or punch drunk (Stern et al., 2013). Although a review of both conditions concluded that CTE was associated with fewer reports of motor symptoms and an earlier onset when compared with dementia pugilistica (Gardner et al., 2014) and so separate terminology may be necessary to differentiate symptoms from the conditions. Despite increased

media attention and widespread diagnoses in athlete and non-athlete populations, the evidence to support a causal relationship between the neuropathology of the condition and its clinical features is scarce (Iverson et al., 2015). Due a lack of clinical features associated with CTE, it has been suggested that the neuropathological changes associated with CTE should be regarded as a pattern of pathology, instead of a specific disease (Lee et al., 2019).

2.3 Biomechanics of Head Acceleration Events

2.3.1 Wearable Sensors

The advent of low-cost micro-electro-mechanical system accelerometers and gyroscopes affords the opportunity to measure head kinematics through wearable head impact sensors (Wu et al., 2016). Accelerometers and gyroscopes instrumented to wearable devices measure linear acceleration and angular velocity, respectively. Angular velocities are differentiated to acquire angular acceleration, and peak values for each kinematic are often calculated to describe a HAE. As a result, the most common kinematics reported when using these devices are PLA, PAA and PAV. These devices have been instrumented onto various wearable devices, including helmet-based sensors, skin patch sensors, skull cap sensors and mouthguard sensors. Practically, non-helmeted sports such as rugby league are limited to instrumented mouthguards, skin patches, headband and skull cap devices. The ability to measure head kinematics during games and practice has an obvious potential for monitoring the cumulative HAE exposure that players of contact sports sustain across games, training and entire seasons.

Wu et al. (2016) evaluated different wearable HAE sensors in measuring head kinematics in vivo. The authors found that wearable patches and skull caps overpredicted peak kinematics when compared with instrumented mouthguards. This overprediction was underpinned by poor skull coupling caused by soft-tissue elasticity. Moreover, the skin patch and skull cap devices showed a tendency to measure accelerations in different vector directions when compared with mouthguards. These measurement errors led the authors to conclude that raw data from these devices should not be used directly to study injury risks. Conversely, instrumented mouthguards demonstrated tight skull coupling, with displacement from the ear canal in all trials being under 0.5mm. Similarly, on-field

validation of devices found that instrumented mouthguards performed the best for measuring head kinematics during HAE (based on positive predictive value) (Kieffer et al., 2020a). Studies have shown rigid skull coupling and accurate peak kinematics in mouthguards when the mandible is tightly clenched (Kuo et al., 2016, Wu et al., 2016). Concerns over the effect of the mandible on the accuracy of mouthguard measures have been raised in previous studies (Kuo et al., 2018, Liu et al., 2020). Kuo et al. (2016) demonstrated that testing with an unconstrained mandible can significantly reduce the accuracy of instrumented mouthguards in anthropometric test dummies (ATDs). In ATD sensors, the mandible is often constrained by springs which can lead to a whipping effect that causes an impact to the underside of the mouthguard, this effect is termed the mandible strike (Liu et al., 2020). Efforts in validation studies have been made to account for this when using ATDs with articulating mandibles (Liu et al., 2020, Camarillo et al., 2013, Rich et al., 2019), however the implications of mandible action in human studies remains unclear. Given that unconstrained mandibles can lead to poorer accuracy (Kuo et al., 2016), further research is required to ascertain whether an unclenched jaw behaves in the same way in vivo as an unconstrained mandible on an ATD, and how often this type of impact occurs on the field. Moreover, the effect of direct contact to the mouthguards on the accuracy of reported kinematics has not been investigated.

The accuracy of instrumented mouthguards varies with which kinematic is being considered (Liu et al., 2020). Liu et al. (2020) identified that algorithms fitting data to the head's centre of gravity (CG) and differentiation calculations amplify noise, which can lead to decreased accuracy when reporting PLA at the head's CG and PAA, respectively, when compared with non-CG data and PAV. The authors also found that at high angular velocities, instrumented mouthguards suffer from gyroscope saturations. Gyroscope saturations occur when the signal (angular velocity) exceeds the dynamic range of the sensor (Dang and Suh, 2014), and this impairs the accuracy of the devices in reporting PAV. Liu et al. (2020) state that gyroscope saturation does not affect the accuracy of PAA as the peak occurs earlier than the angular velocity peak. Inaccuracies were also observed in HAE to the front of the head; however, the authors postulate that these are typically caused by propagation of loading through the facemask of American Football helmets and so similar inaccuracies are less likely in non-helmeted sports. Indeed, a similar study

design to that of Liu et al. (2020) is necessary to identify any inaccuracies in instrumented mouthguards associated with non-helmeted sports.

2.3.2 The Evolution of Kinematic Metrics

Linear vs Angular Acceleration

Earliest research debates focussed on the clinical significance of linear and angular acceleration in brain injury. Gurdjian and Webster (1945) postulated linear acceleration as the primary mechanism of concussion, whilst Holbourn (1943) posited that shear-induced damage caused by angular motion was more important, due to the low shear modulus of the brain (resistance to change in shape). By the same logic, Holbourn (1943) posited that the brain's high bulk modulus (resistance to a change in volume) meant linear acceleration was less likely to lead to brain deformation and therefore injury. Later research postulated that linear and rotational motion predominantly resulted in different types of injuries; linear acceleration was associated with pressure gradients whilst angular acceleration leads to shear stress as a result of rotation of the skull relative to the brain (Unterharnscheidt, 1971). Work by Gennarelli and colleagues subsequently established that angular motions elicited concussive symptoms to a greater degree than did linear motions (Gennarelli et al., 1981, Gennarelli et al., 1972, Gennarelli et al., 1982, Gennarelli et al., 1987). However, both linear an angular accelerations are now associated with a risk of concussion through transient intracranial pressure gradients and strain responses, respectively (Ommaya, 1985, King et al., 2003).

Finite Element Based Kinematic Metrics

Finite Element (FE) head models are detailed models of the brain which utilise advanced neuroimaging techniques to model the anisotropic material properties of white matter (Zhao and Ji, 2019). FE head model simulations are used to estimate brain strain metrics for the whole brain or specific regions of interest (ROIs). Brain strain is a mechanical parameter which describes the deformation of the brain, and brain strain rate describes the rate of change of brain strains. Maximum principle strain (MPS) and cumulative strain damage measure (CSDM) are the most widely used response samplings from FE head modelling (Takhounts et al., 2008). FE-based metrics have also now been used as an alternative to kinematics to report the HAE exposure of youth American footballers (Miller et al., 2020). CNNs are deep learning neural networks that allow for

the faster computation of brain strain than FE head models (Wu et al., 2019a). Kinematic data from instrumented mouthguards can be used as an input for FE head models and CNNs (Liu et al., 2020). Liu et al. (2020) reported that mouthguards with a short sampling time windows did not fully capture the deceleration phase of angular velocity and suggested that sampling time windows of around 100 ms were appropriate for use with CNNs and FE head models. Since then, 20 ms of pre-trigger data and 70 ms of post-trigger data was found to yield brain strain and strain rate calculations that were not significantly different from 200 ms time windows (Liu et al., 2021).

2.3.3 Limitations of Scalar and Peak Values

Peak scalar kinematics such as PLA, PAA and PAV as well as FE-based metrics MPS and CDSM of the whole brain may not be effective in understanding and predicting brain injury, owing to the fact that they effectively treat the whole head as a single unit (Wu et al., 2019b). Some research has focused on using FE-based metrics to investigate brain ROIs such as the brainstem, midbrain, white or grey matter, or more targeted areas such as the thalamus or corpus callosum and found that some areas are more susceptible to brain strain (Zhao et al., 2019, Zhao et al., 2017). Specific FE-based metrics for brain ROIs have also been suggested to better predict injury (Zhang et al., 2004, Giordano and Kleiven, 2014, Kleiven, 2007, Wu et al., 2019b).

Scalar kinematics PLA, PAA and PAV also ignore directional data. Bian and Mao (2020) simulated HAE using the Global Human Body Model Consortium head model (Mao et al., 2013) and found that rotational directions had differential effects on brain strain in different ROIs. Areas of the brain were affected differently by rotation directions (shown in figure 9): lateral bending elicited the lowest MPS distribution, but the highest strain in the thalamus and corpus callosum; the basal ganglia were strained the most by flexion and extension rotations; and axial rotations resulted in the greatest MPS distributions, causing the second highest strains in the corpus callosum and thalamus and the third highest strain in the basal ganglia. Whilst these findings suggest that different directions of rotation affect areas of the brain differently, the clinical significance of these differences have not been investigated.

Impact duration and deceleration times are not captured by peak kinematics. Bian and Mao (2020) simulated HAE across a range of durations and found that shorter deceleration times (<20 ms) were associated with a decreased brain strain, whilst deceleration times greater than 20 ms were associated with increased brain strain. The authors found that short deceleration periods can cancel the initial brain strain, whilst longer deceleration periods cancelled brain strain to a lesser degree than short deceleration periods, and also introduced a new brain strain later on in the impact.

2.3.4 Injury Prediction using Kinematics and Injury Metrics

Wu et al. (2019b) compared the ability of kinematics PLA, PAA and PAV and scalar metric MPS of the whole brain as well as a network-based injury metric to predict concussions from three different datasets. No scalar metric consistently outperformed the others (Figure 3). MPS performed worse than PLA, PAA and PAV which supports that FE-based metrics may not be better in injury prediction than kinematics (Beckwith et al., 2018). PAA outperformed other scalar metrics in two datasets, but performed poorly in the combined dataset, whilst PAV and PLA performed similarly. Rowson and Duma (2013) found PLA was marginally more effective than PAA in predicting concussions in both of their datasets, whilst a combined linear and angular acceleration metric outperformed PAA but was not significantly different from PLA.

The network-based injury metric devised by Wu et al. (2019b) characterises the distribution and magnitude of brain strains. Following HAE simulations using the Worcester head injury model (Zhao and Ji, 2019), the risk of injury is encoded from grey matter ROIs and their white matter interconnections, which make up 312 features of the network. Different features were selected when the metric was trained in order to achieve either the greatest accuracy or sensitivity. Figure 3 shows the positive predictive value of the network-based injury metric that was trained to achieve the greatest accuracy. The authors postulate that this network-based injury metric is beneficial when compared with scalar kinematics and metrics because they provide more complete information about regional brain injury risks, rather than treating the whole head as a single unit. Indeed, Figure 3 shows that the network-based injury metric outperformed all scalar metrics in predicting concussion. The authors suggest that whilst their metric

can be used for binary injury prediction (concussion or non-injury), new possibilities of investigating which brain ROIs are most vulnerable following a HAE may also be possible using the metric. Furthermore, their metric may allow for the relationship between brain strains in specific ROIs and neuroimaging alterations, or subsequent brain dysfunction to be investigated.

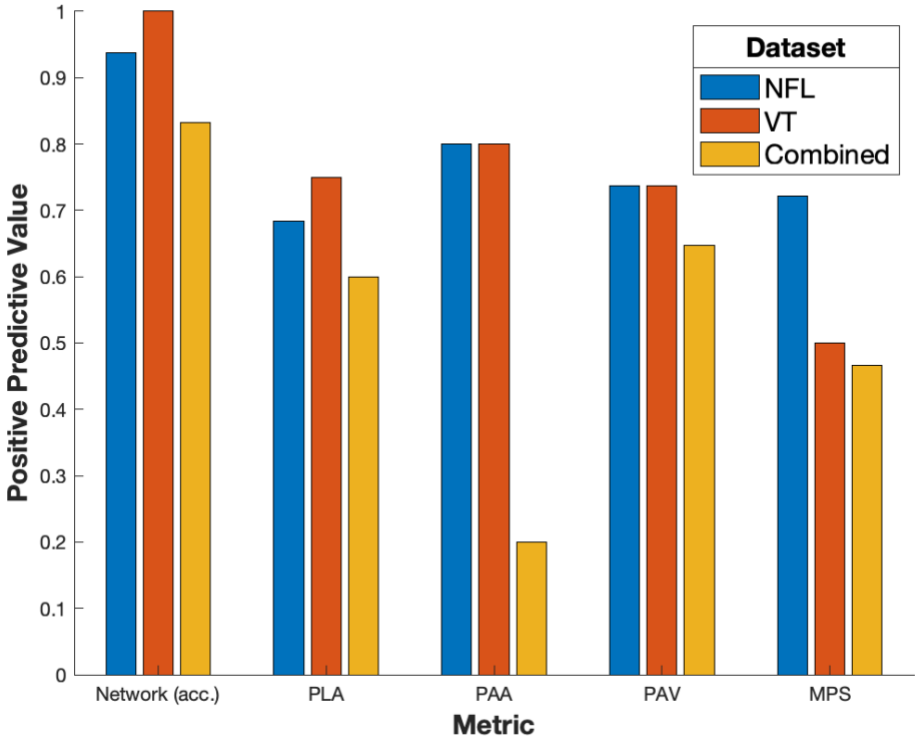

Figure 3. Positive predictive values for kinematic metrics: peak linear acceleration (PLA), peak angular acceleration (PAA) and peak angular velocity (PAV) and FE-based metric maximum principle strain (MPS) of the whole brain, as well as a network based "response feature matrix" trained to achieve the best accuracy (Wu et al., 2019b). Datasets included laboratory reconstructed HAE from the National Football League (NFL), laboratory reconstructed HAE from Virginia Tech (VT), and a combined dataset including NFL and VT datasets as well as instrumented mouthguard HAE from Stanford University.

2.3.5 Elusiveness of Concussion Threshold

Attempts to identify a robust concussion threshold using PLA, PAA and PAV have been mostly unsuccessful (Guskiewicz and Mihalik, 2011). Wu et al. (2019b) identified large differences between injury thresholds between their datasets: PLA thresholds varied from 83.1 to 110.1 g; PAA thresholds varied from 5068.8 to 9461 rad/s^2; and PAV

thresholds varied from 31.1 to 41.2 rad/s. These thresholds resulted in positive predictor values of 0.2 to 0.8 (Figure 3). Similarly, Rowson et al. (2018) found that less than half of diagnosed concussions were associated with the greatest magnitude acceleration for each player, whilst 90% of concussions were associated with acceleration magnitudes within the top five for each player (Figure 4). These findings suggest that a global concussion threshold is likely unattainable, due to personal differences in tolerance to head accelerations.

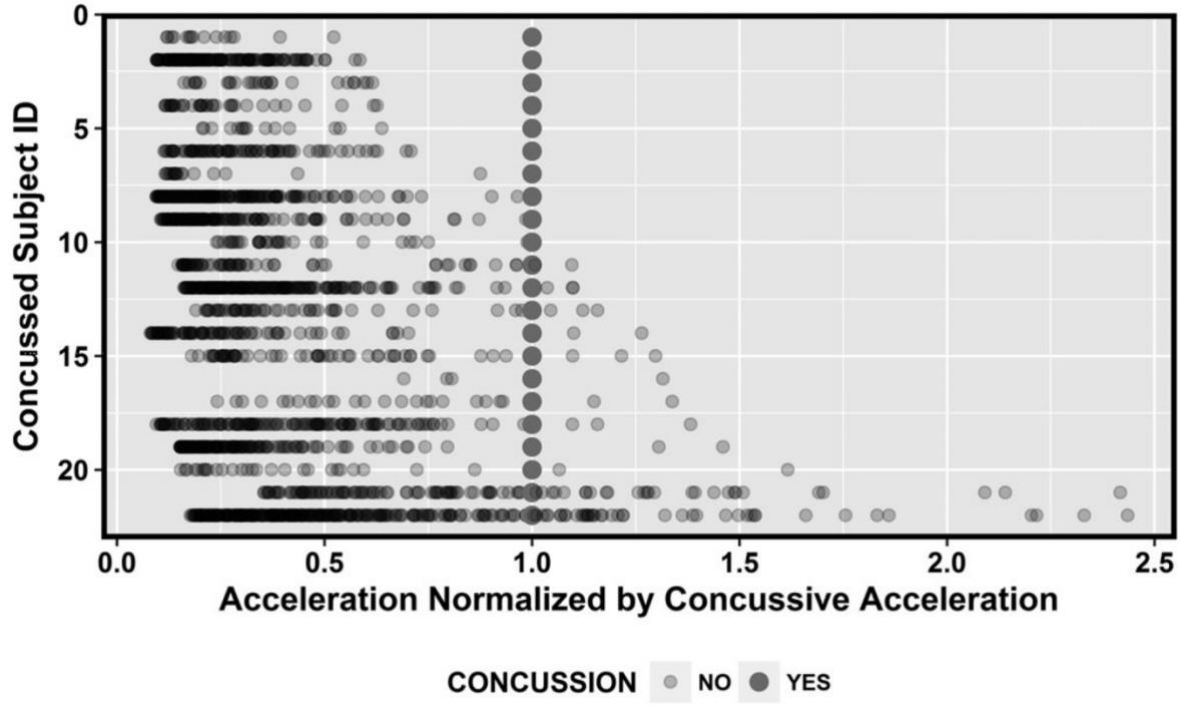

Figure 4. Acceleration magnitudes normalised by the concussive acceleration for each player. Concussed subjects 0-10 experienced the highest acceleration magnitude during the concussive HAE, whilst the rest of the subjects experienced higher acceleration magnitudes from non-concussive HAE. Taken from Rowson et al. (2018).

The elusiveness of a threshold using these kinematics is likely caused by a plethora of intrinsic and extrinsic factors that will influence an individual's tolerance to a HAE. Head size has been positively correlated with injury risk in animal studies, that is the greater the size of the head the lower the tolerance to acceleration (Ommaya et al., 1967), this is because more brain strain is produced in a larger head, mainly due to its greater mass. Furthermore, head shape (Danelson et al., 2008) and soft tissue differences in the brain (Miller and Chinzei, 2002) have also been demonstrated to influence an individual's

tolerance to acceleration. Individuals previously diagnosed with a concussion are also more likely to experience future concussions than those without a diagnosis (Guskiewicz et al., 2003). Similarly, Broglio et al. (2017) compared the degree of HAE exposure in concussed and non-concussed high-school American footballers with similar HAE histories. Concussed individuals were matched with a non-concussed controls based on the HAE histories in the season leading up to the concussion. Non-concussed controls were matched with concussed individuals such that they experienced a similar: magntiude HAE as the concussive HAE (termed the final HAE); number of HAE in the same seasons leading up to the final HAE; and a similar number of HAE in the 24-hour period leading up to the final HAE. HAE density was calculated from the 20 HAE prior to the final/concussive HAE using equation 1.

$$\text{Impact Density} = \sum_{i=1}^{20} \frac{\text{Acceleration}_i}{\text{Time from previous impact}_i} \quad (1)$$

The authors found that concussed individuals experienced a significantly greater number of HAE in the final 3 hours prior to final HAE, and experienced a higher HAE density. Futhermore, Rowson et al. (2018) reported that concussed individuals reported a greater frequency and magnitude of HAE than non-concussed controls. These studies support that sustaining subconcussive HAE proximal to the concussive HAE can lower the tolerance to accelerations (Broglio et al., 2017, Rowson et al., 2018). These findings suggest that tolerance to accelerations is influenced by multiple individual factors, consequently an individual approach is likely most appropriate for identifying tolerance thresholds using head kinematics.

2.3.6 Brain Injury Criteria and Kinematics

Brain injury criteria (BIC) have been developed to estimate the risk of injury (Table 2). By looking at how PLA, PAA and PAV are incorporated into each BIC, we can draw conclusions as to how they are used to estimate injury risk.

Table 1. Brain injury criteria for sports-related head injuries. Modified from Zhan et al. (2020).

Brain Injury Criteria	Author(s)	Equation
Peak Linear Acceleration (PLA)	-	$\mathrm{PLA} = \lvert a(t) \rvert$
Peak Angular Acceleration (PAA)	-	$\mathrm{PAA} = \lvert \alpha(t) \rvert$
Peak Angular Velocity (PAV)	-	$\mathrm{PAV} = \lvert \omega(t) \rvert$
Head Injury Criterion (HIC)	Versace (1971)	$\mathrm{HIC} = \max_{t_1, t_2} \left\{ \left(\int_{t1}^{t2} \lvert a(t) \rvert \, \mathrm{d}t \right)^{2.5} (t_2 - t_1) \right\}$ Where t_1, t_2 are chosen to maximise HIC and $t_2 - t_1 \leq 15$ ms.
Gadd Severity Index (GSI)	Gadd (1966)	$\mathrm{SI} = \int \lvert a(t) \rvert \, 2.5 \mathrm{d}t$
Principle Component Score (PCS)	Greenwald et al. (2008)	$\mathrm{PCS} = \beta_o + \beta_1 \lvert a(t) \rvert + \beta_2 \, \mathrm{SI} + \beta_3 \, \mathrm{HIC} + \beta_4 \lvert \alpha(t) \rvert$ Coefficients are determined by empirical research.
Kleiven's Linear Combination (KLC)	Kleiven (2007)	$\mathrm{KLC} = 0.00471 \max_t \lvert \omega(t) \rvert + 0.000224 \mathrm{HIC}$ Where HIC $t_2 - t_1 \leq 36$ ms.
Combined Probability (CP)	Rowson and Duma (2013)	$\mathrm{CP} = \beta_o + \beta_1 \lvert a(t) \rvert + \beta_2 \lvert \alpha(t) \rvert + \beta_3 \lvert a(t) \rvert \, \lvert \alpha(t) \rvert$ Coefficients are determined by logistic regression.
Rotational Injury Criterion (RIC)	Kimpara and Iwamoto (2012)	$\mathrm{RIC} = \max_{t_1, t_2} \left\{ \left(\int_{t1}^{t2} \lvert \alpha(t) \rvert \, \mathrm{d}t \right)^{2.5} (t_2 - t_1) \right\}$ Where t_1, t_2 are chosen to maximise HIC and $t_2 - t_1 \leq 36$ ms.
Head Impact Power (HIP)	Newman and Shewchenko (2000)	$\mathrm{HIP} = \max_t \left\{ m \sum a_i(t) \int a_i(t) \mathrm{d}t + \sum I_{ii} \alpha_i(t) \int \alpha_i(t) \mathrm{d}t \right\}$ Where m is mass, I_{ii} is the principle moments of inertia and i denotes the three directions of rotation
Power Rotation Head Injury Criterion (PRHIC)	Kimpara et al. (2011)	$\mathrm{PRHIC} = \max_{t_1, t_2} \left\{ \left(\int_{t1}^{t2} \lvert \mathrm{HIP_{rot}}(t) \rvert \, \mathrm{d}t \right)^{2.5} (t_2 - t_1) \right\}$ Where $\mathrm{HIP_{rot}}$ is the second part of HIP and t_1, t_2 are chosen to maximise HIC, where $t_2 - t_1 \leq 36$ ms.
Kinematic Brain Injury Criterion (BRIC)	Takhounts et al. (2011)	$\mathrm{BRIC} = \frac{\lvert \omega \rvert}{\omega_{cr}} \frac{\lvert \alpha \rvert}{\alpha_{cr}}$ Where $\omega_{cr} = 42.05$ rad/s and $\alpha_{cr} = 363{,}268$ rad/s^2 as estimated from on-field American Football data.
Brain Injury Criterion (BrIC)	Takhounts et al. (2013)	$\mathrm{BrIC} = \sqrt{\left(\frac{\omega_x}{\omega_{xcr}}\right)^2 + \left(\frac{\omega_y}{\omega_{ycr}}\right)^2 + \left(\frac{\omega_z}{\omega_{zcr}}\right)^2}$ Where $[\omega_x, \omega_y, \omega_z]$ are maximum angular velocity in each directional component and $[\omega_{xcr}, \omega_{ycr}, \omega_{zcr}]$ are the critical values found by experimental data [66.2,59.1,44.2] rad/s.
Brain Angle Metric (BAM)	Laksari et al. (2020)	$I\left(\ddot{\theta}_{\mathrm{brain}} + \ddot{\theta}_{\mathrm{skull}}\right) = k\theta_{\mathrm{brain}} - c\dot{\theta}_{\mathrm{brain}}$ Where I is the moment of inertia of the mass, k and c are the stiffness and damping values of the system and the angles of the brain and skull are noted by θ_{brain} and θ_{skull}, respectively.

PLA is not as widely used in BIC as angular measures (PAV, PAA). Only, two of the BIC included incorporate PLA without an angular input (HIC and GSI), whilst in more

modern BIC, linear acceleration is used only alongside an angular component. PLA is used as an input alongside PAA or PAV in four BIC (PCS, KLC, CP and HIP). In KLC and PCS, PLA is incorporated by the incorporation of HIC and/or GSI into the equations, whilst PLA is directly implemented into CP and HIP. Interestingly, the coefficient used for PLA in CP is greater than the coefficient for PAA, which suggests that Rowson and Duma (2013) placed a greater importance on PLA than PAA in concussion prediction. Angular inputs are used without PLA in five BIC (RIC, PRHIC, BRIC, BrIC and BAM). In terms of angular kinematics, acceleration is more widely used than velocity. Angular acceleration is used as an input for six BIC (PCS, CP, HIP, PRHIC, RIC, and BRIC) whilst angular velocity is used in three (KLC, BRIC and BrIC). BAM uses a 3 degree-of-freedom mass-spring-damper brain model with real-word HAE angular acceleration profiles to classify between injurious and non-injurious HAE. HAE duration is factored into all BIC, except CP, BRIC and BrIC which only combine peak kinematics. HIP and PRHIC are the only BIC to incorporate values for the mass and principle moments of inertia of the brain. BIC incorporating values for each directional components are HIP, PRHIC and BrIC, whilst resultant values are used in the rest. Critical values for kinematic components are used within BrIC and BRIC and are calculated from on-field and experimental data respectively.

The ability of these BIC to predict concussions have not been compared independently, however Zhan et al. (2020) conducted analysis on how each BIC correlated with brain strain predictions, as predicted by an FE head model (Ho and Kleiven, 2007). The authors found that BRIC, BrIC, PAV and BAM predicted brain strain the most accurately. These results support previous findings that PAV is a good predictor of brain strain (Bian and Mao, 2020). Further work is required to identify which metric is the most accurate in predicting concussions and brain injury.

2.3.7 Kinematic Metrics and Physiological Changes

Whilst the clinical significance of kinematic metrics has previously been determined by their correlation with diagnosed injuries, recent studies have been able to measure direct physiological changes and their relationship with kinematic metrics (Hajiaghamemar et al., 2020, O'Keeffe et al., 2020). Hajiaghamemar et al. (2020) directly measured acute traumatic axonal injury in a diffuse traumatic brain injury pig model and

compared how different kinematics (PAA and PAV) and FE-derived metrics correlated with the extent of acute traumatic axonal injury. Acute traumatic axonal injury was measured through histopathology, was reported as axonal injury volume, and angular velocity and acceleration kinematics were extracted from the non-contact head rotations. The authors found that axonal injury volume correlation was higher with PAA than with PAV. Similarly, strain-rate metrics were shown to have a higher correlation with axonal injury volume than strain metrics. These findings suggest that angular acceleration and strain rate-based tissue injury metrics are the most useful in predicting the degree of acute traumatic axonal injury. O'Keeffe et al. (2020) combined instrumented mouthguard data with dynamic contact enhanced-MRI images in mixed martial arts fighters and rugby players. The authors measured the degree of dysfunction to the blood-brain barrier (BBB), which is associated with the pathology of CTE (Doherty et al., 2016). FE based metrics first principle strain, first principle strain rate and first principle stress all correlated well with both measures of BBB disruption. The authors suggest that these correlations need validation due to the small dataset. Both studies are examples of exciting new possibilities of directly measuring mechanisms associated with brain injury and concussion, whilst investigating the effect of different head kinematic metrics. Furthermore, they demonstrate how brain strain, as predicted by FE head models, is correlated with physiological changes to the brain following HAE. Similar study designs should aim to quantify how physiological changes correlate with PLA, PAA and ΔPAV.

2.4 Conclusion and Aims

Concussions are an inherent risk of rugby league, however the incidence of concussions is not well understood in the professional game. A variety of potential short- and long-term consequences of conucssions and repetitive HAE have been idenfitied in the literature, however there is a lack of longitudinal research using a rugby league population. As such, the long-term effects of rugby league participation are not established. Research from other sports suggests that the potential long-term consequences associated with contact sports participation are not limited to retired players with a concussion history, and so there is also a concern that an accumulation of subconcussive HAE may have an effect. Despite this, there is a lack of an understanding as to the HAE exposure rate sustained by professional rugby league players. Moreover,

there is a lack of research utilising qualitative video analysis alongside biomechanical measures, so the nature of HAE exposure in rugby league is not well-characterised.

Wearable HAE sensors are being used to monitor HAE sustained during games in contact sports, including rugby league. In non-helmeted sports, only instrumented mouthguards are supported for reporting head kinematics during games, with other devices suffering inaccuraries resulting from poor skull coupling. Currently, no studies have used instrumented mouthguards in rugby league populations. Whilst attempts have been made to identify concussion thresholds using kinematics collected by instrumented mouthguards, due to multiple intrinsic and extrinsic factors that influence an individual's tolerance to HAE and a lack of understanding as to the relationship between kinematics and brain injury, these injury thresholds remain intangible. Consequently, a more approprate application of instrumented mouthguards is to quantify the HAE sustained by players, and qualitatively analyse the associate contact events.

This study will use instrumented mouthguards and qualitiative video analysis to quantify and characterise HAE sustained in professional male rugby league matchplay. Therefore, the aims of the study are: i) to describe the kinematics of HAE; ii) to identify the contact event and impact characteristics for each HAE; iii) to compare the incidence and kinematics of HAE from different contact events and impact characteristics; and iv) to report the HAE exposure sustained by male professional rugby league players in this study.

3 Methodology

3.1 Participants

Ten males from a Super League team for ten games including pre-season friendlies gave written consent and Ethical approval was given for the study by the Faculty of Biological Sciences Ethical Review Committee (#BIOSCI 18-023). The cohort consisted of two backs (both centres) and eight forwards (two props, two second row, two loose forwards a hooker and a back row). Data were collected for a total of 31 player matches. Due to instrumented mouthguards not collecting data for the entire match following improper charging in some cases (n = 4), active player minutes were calculated for each player. Active player minutes were defined as the amount of time from either the start of the half or the player entering the pitch after an interchange, up until either the end of the half or the player leaving the pitch after an interchange, whilst the instrumented mouthguard was collecting data. In the cases where data stopped being collected, active minutes were counted up until the final HAE. Each player's active game minutes and number of HAE are provided in Table 2. No concussions were diganosed in any of the participants.

Table 2. Each participant's position, number of player matches and active player minutes and HAE count above 8 *g* or 800 rad/s^2.

Player	Position	Player Matches	Active Player Minutes	HAE Count
1	Forward	2	96	81
2	Forward	1	26	12
3	Forward	5	344	157
4	Forward	1	77	26
5	Forward	8	280	175
6	Forward	1	46	20
7	Forward	4	104	82
8	Forward	2	119	48
9	Back	6	422	95
10	Back	1	82	29
Total		31	1596	725

3.2 Instrumented Mouthguards

The Prevent Biometric Custom Fit Mouthguard (Prevent Biometrics, Minneapolis MN) was fitted to each player. Dental impressions were taken for each instrumented player and custom-made mouthguards were manufactured to ensure a rigid fit to the upper dentition. The reliability and validity of instrumented mouthguards has been demonstrated in previous studies (Kieffer et al., 2020a, Liu et al., 2020) and discussed in section X. A two-phased approach of assessing the accuracy of a range of wearable head sensors was conducted by Kieffer et al. (2020a). The Prevent Biometric custom fit mouthguard was the highest performing device in both lab-based impact testing on a crash test dummy headform and on the field evaluation, recording a concordance correlation coefficient value of 0.97 in lab-based impact testing and positive predictive value of 96% in active on-field minutes (i.e. periods of open play, not including substitutions, half-time and injury breaks). Similarly, when comparing the accuracy of a range of mouthguards, Liu et al. (2020) identified that the Prevent Biometric Custom Fit Mouthguard was the best performing device with a mean relative error of 4.9%, 4.6% and 2.5% for PLA, PAA and PAV.

The mouthguards are instrumented with gyroscopes and acceleromters sampling at 3200 Hz with measurement ranges of ± 35 rad/s and ± 200 g respectively. The mouthguards were set with a single axis trigger threshold of 5 g, i.e., HAE were captured when linear acceleration exceeded 5 g on a single axis of the accelerometer. The mouthguards captured 10 ms of pre-trigger data and 40 ms of post trigger data. Each HAE consisted of linear acceleration and angular velocity time-traces. Each HAE was processed by Prevent Biometrics. This included the transformation of linear acceleration time-traces to the head centre of gravity (CG) and the application of a 4-pole, zero phase, low-pass Butterworth filter with a corner frequency of 400Hz. Noise can be introduced into time-traces when the mouthguard's adherence to the teeth is poor, therefore Prevent Biometrics ran HAE through a machine learning model which determined whether the HAE contained minimal noise (class 0), moderate noise (class 1) or severe noise (class 2). Another 4-pole, zero phase, low-pass Butterworth filter was applied to class 1 (n = 32) and class 2 (n = 6) HAE at lower corner frequencies of 100 Hz and 50 Hz to reduce the nosie. The present dataset was used to develop Prevent Biometrics' false positive

algorithm, therefore only HAE deemed to be true positives during this process were considered in the analysis. True positives were identified by a combination of video verification and a false postive algorithm.

3.3 Data Processing

For each HAE, peak angular acceleration and peak linear acceleration were calculated from resultant time-traces. A data-acquisition threshold of 8 *g* or 800 rad/s^2 was set to include any impacts above those induced by running and jumping (Miller et al., 2019). Peak change in angular velocity (ΔPAV) was calculated by zeroing the angular velocity time-trace to the onset of the impact (t=0 ms) and taking the peak value from the resultant time-trace (Figure 5). ΔPAV was calculated instead of PAV in order to measure the change in angular velocity due to the impact, to avoid peak values being taken from pre-trigger data. The predominant direction of rotation and translation were defined as the directional component with the greatest magnitude at the index of peak angular acceleration and linear acceleration, respecively (Figure 7D).

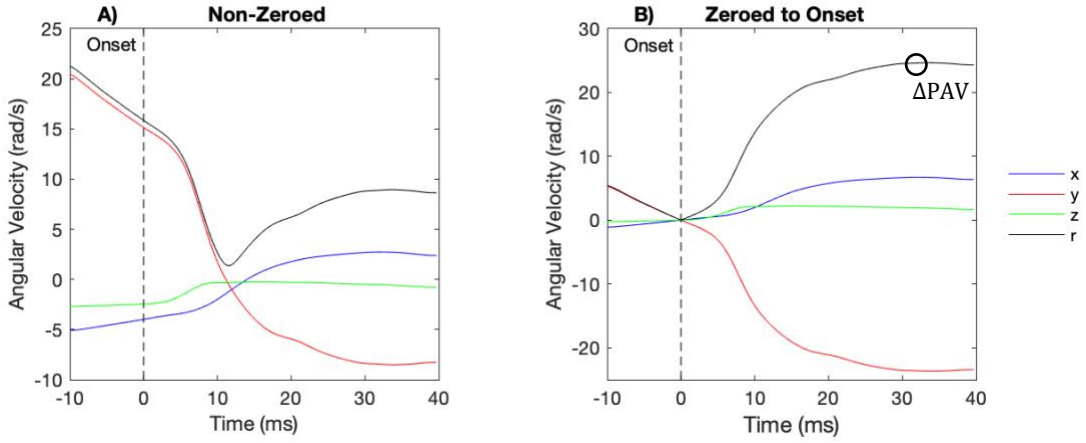

Figure 5. Non-zeroed angular velocity time-trace (A) and angular velocity time-trace zeroed to the onset of impact (B) to calculate peak change in angular velocity (ΔPAV). The onset was defined as the trigger of the impact (t=0 ms).

3.4 Protocol

Broadcast quality video footage was available for each game and each mouthguard-triggered HAE was timestamped and synchronised with video footage to a 40-millisecond resolution to allow for qualitative analysis. The contact event was defined by the stage of the tackle between players: HAE occuring from the initial contact between players was deemed as a tackle or carry, depending on whether the instrumented player was a tackler or ball-carrier, respectively; any HAE occuring between two players that had already made a tackle/carry contact in the same phase and before the ball-carrier was grounded was defined as a post tackle contact (PTC); any HAE caused by players falling to the ground was defined as a ground impact and wrestle HAE were defined as any HAE occuring after the ball-carrier has been grounded before the commencement of the play-the-ball (Figure 6). A qualitative video analysis framework was created from applicable criteria from a consensus framework developed by the Rugby Union Video Analysis Consensus Group (Table 3) (Hendricks et al., 2020). Any descriptors that were not visible in footage were labelled as occluded. HAE were labelled with a phase ID, such that HAE occuring for the same player in the same tackle phase were given the same phase ID but different event IDs. Cohen's Kappa (Cohen, 1960) was used to assess the inter-rater relability for each descriptor and the results are shown in Table 3. In conjuction with the video footage, a customsied Matlab script was developed that utilised the kinematic time-

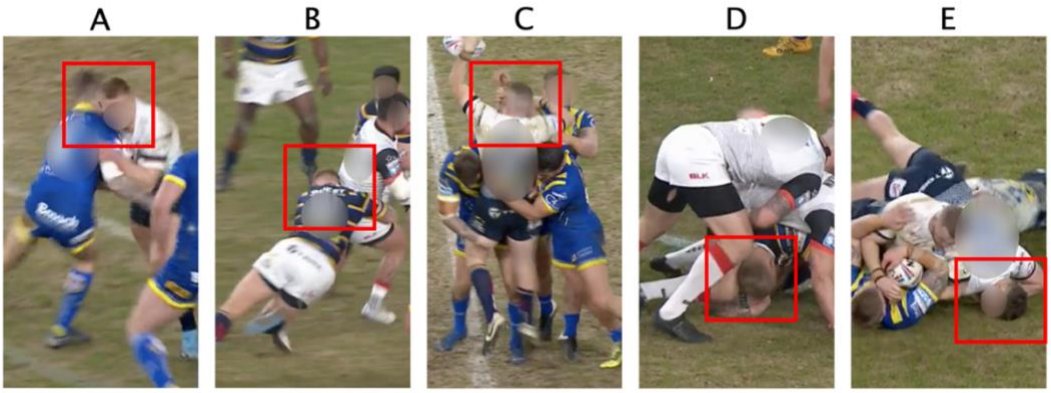

Figure 6. Contact events labelled as (A) carry, (B) tackle, (C) post-tackle contact, (D) ground impact and (E) wrestle. An initial tackle/carry contact between the two impacting players had already been made prior to the arm-to-head HAE pictured in the post-tackle contact (C). Red squares indicate the impacted player.

traces from the instrumented mouthguard to simulate the head motion during the HAE to aid the identification of the cause of each HAE (Figure 7).

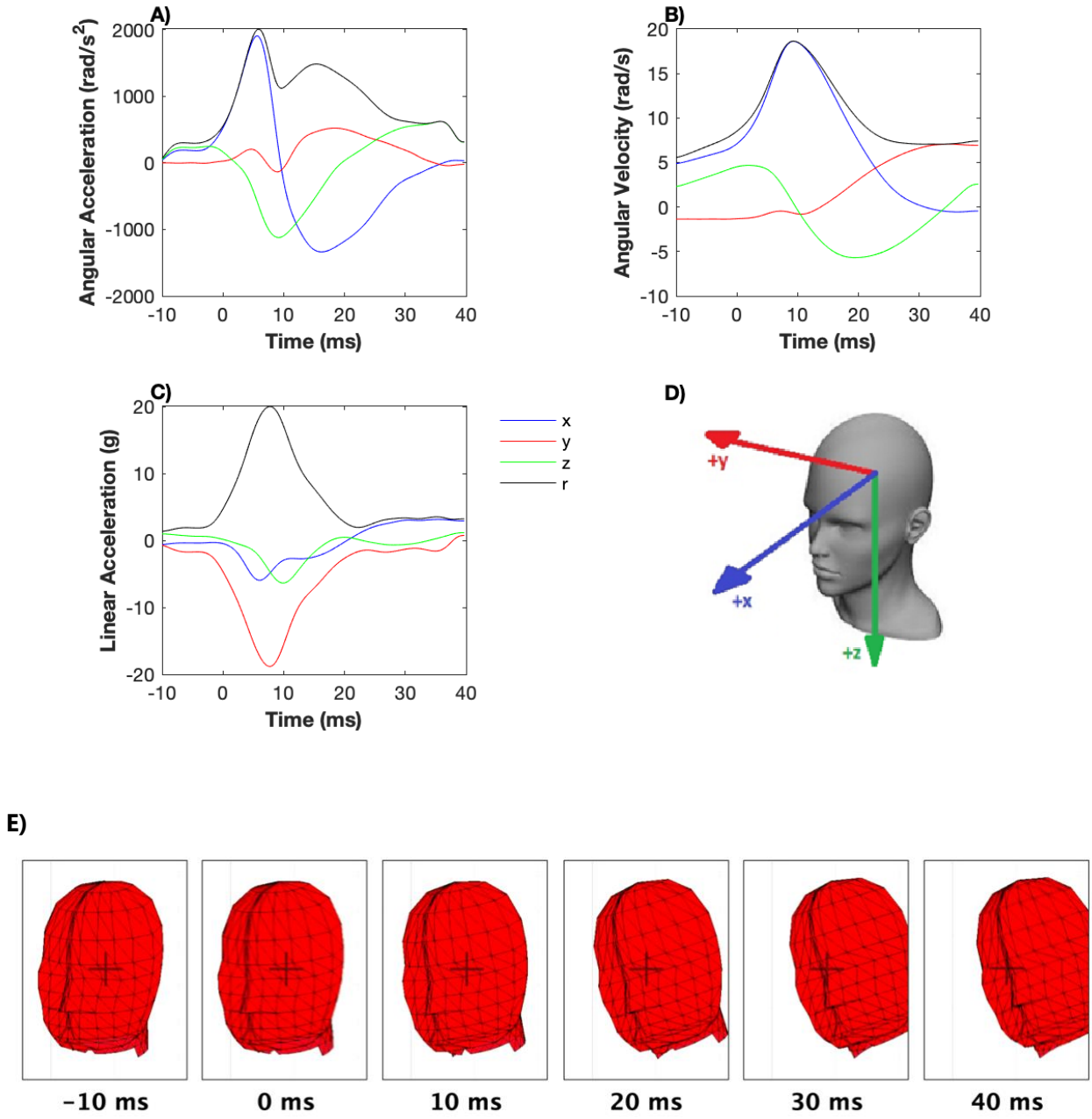

Figure 7. Exemplar HAE with angular acceleration (A), angular velocity (B) and linear acceleration (C) time-traces, and a video simulation (E). The axis system used once kinematics are transformed to the head CG (D).

Table 3. Qualitative analysis framework adapted from an existing framework from Rugby Union (Hendricks et al., 2020).

Characteristic	Cohen's Kappa (Cohen, 1960)	Definition
Contact Event *(Figure 6)*	0.87	**Tackle** – HAE occurs in the tackle and the impacted player is the tackler. **Carry** – HAE occurs in the tackle and the impacted player is the ball-carrier. **Post Tackle Contact (PTC)** – HAE occurs as a secondary contact after the initial one-on-one, simultaneous, sequential, or dual-sequential tackle impact. **Ground Impact** – Any HAE caused by players falling to the ground. **Wrestle** – Any HAE which occurs after the ball-carrier has been grounded before the commencement of the play-the-ball.
The following characteristics were analysed for HAE from tackles, carries, PTCs, ground impacts and wrestles only.		
Impacted Player	1	**Ball-Carrier** – Impacted player is in possession of the ball. **Tackler –** Impacted player does not have possession of the ball and it attempting to stop a ball-carrier.
Head Loading Type	0.82	**Direct** – Head loading is through direct contact with the head. **Indirect** – Inertial head loading transmitted through the neck from an impact to the body.
The following characteristics were analysed for tackle, carry, PTC and wrestle HAE only.		
Impacting body part (Direct HAE only)	0.88	**Lower Leg** – Area below the base of the knee contacts the impacted player. **Upper Leg** – Area above the base of the knee, below the shorts. **Hip** – Area covered by the shorts. **Torso** – Area above short line to base of armpit. **Arm** – Area below armpit level on the upper limb. **Shoulder** – Area above the armpit level to the base of the neck. **Head and Neck** – Area above base of neck including head.
Impacting Player	0.87	**Teammate** – Contact is made with a teammate. **Opposition** – Contact is made with an opposition player.
The following characteristics were analysed for HAE from tackles and carries only.		
Tackle Type	0.82	**Arm** – Tackler attempts to impede the ball-carrier with use of upper limbs. **Smother** – Tackler attempts to impede the ball-carrier with the use of their chest and by wrapping both arms around. **Shoulder** – Initial contact is made by tackler's shoulder. **Tap** – Tackler attempts to trip the ball-carrier with a hand on the lower limb or knee.
Tackle Sequence	0.80	**One-on-One** – Tackler that causes HAE is the first player to make contact. **Simultaneous** – Tackler that causes HAE makes contact simultaneously with another player. **Sequential** – Tackler that causes HAE joins tackle shortly after another player has already made contact. **Dual-sequential** – Tackler that causes HAE joins tackle where two or more players have already made contact.
Tackle Height	0.92	**Upper Body** – Above the area covered by the shorts. **Lower Body** – The area covered by the shorts and below.
Tackle Direction	0.85	**Front** – Initial contact made within 30° of hip orientation. **Side** – Initial contact made within 30°-150° of hip orientation. **Behind** – Initial contact made within 150°-180° of hip orientation.
Ball-Carrier Action	0.79	**Carry** – Ball-carrier has control of the ball and is attempting to keep hold of it. **Catch** – Ball-carrier's attention is on catching the ball from the air. **Gather** – Ball-carrier is collecting the ball from the floor. **Kick** – Ball-carrier is attempting to kick the ball. **Pass** – Ball-carrier is attempting to pass the ball.

3.5 Head Acceleration Exposure Rates

HAE exposure was calculated as the number of HAE per player per match, therefore player matches where the instrumented mouthguards only partially captured data were not included (n=4). Exposure rates were calculated for each individual player at the data acquisition thresholds used in this study (8 *g* or 800 rad/s^2) and previous studies using x-patch devices: 10 *g* (King et al., 2017a, King et al., 2017c, King et al., 2018) and 20 *g* (Carey et al., 2019, Carey et al., 2020).

3.6 Statistical Analyses

All statistical analyses were conducted in R Studio using the lme4 package. PLA, PAA and ΔPAV were used as dependent variables. Categorical variables contact event types (tackle, carry, PTC, ground and wrestle), head loading types (direct and indirect), tackle sequences (one-on-one, simultaneous, sequential and dual-sequential), tackle types (shoulder, smother, arm) and tackle height (upper and lower) were used as independent variables. Data were visually inspected for normality using histograms and Q-Q plots. All dependent variables were log-transformed due to non-normal distrubutions in order to reduce the error from non-uniform data. Data was nested in clusters of individual players, therefore a mixed-effect linear model was used (Tierney et al., 2020) and the player was included as a random intercept in all models. The effect size (ES) difference (95% confidence interval) was estimated from the ratio of the observed mean difference to the pooled standard deviation for each comparison. ES differences were interpreted as trivial (<0.2), small (0.2 to <0.6), moderate (0.6 to <1.2), large (1.2 to <2) and very large (≥2). A boneferroni correction was applied to account for repeated measures. Due to the small cohort size, no statistical tests were performed on the exposure rates, or incidence of HAE from different contact events and impact characteristics.

4 Results

4.1 Head Acceleration Event Kinematics

Over ten matches, 725 visible HAE exceeded either 8 *g* or 800 rad/s^2. PLA (median = 10.5 *g*, Q1 = 8.5 *g*, Q3 = 14.0 *g*), PAA (median = 923.5 rad/s^2, Q1 = 681.9 rad/s^2, Q3 = 1209.9 rad/s^2) and ΔPAV (median = 9.3 rad/s, Q1 = 6.5 rad/s, Q3 = 12.9 rad/s) were positively skewed (Figure 8). Lateral translation was the most common direction for linear acceleration, whilst lateral bending was the most common direction of angular acceleration (Figure 9).

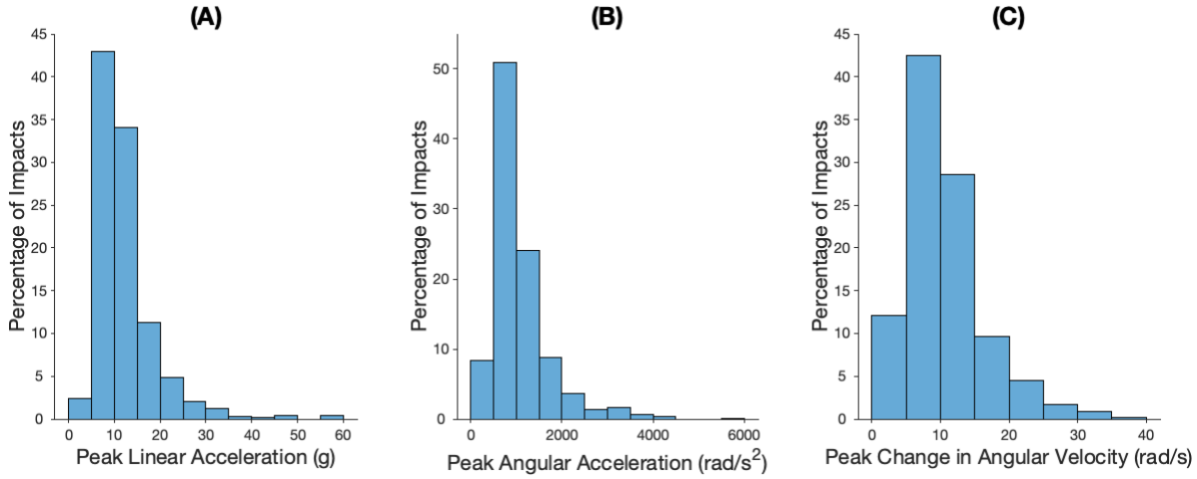

Figure 8. Distribution of kinematics for all HAE above 8 *g* or 800 rad/s^2 (n=725).

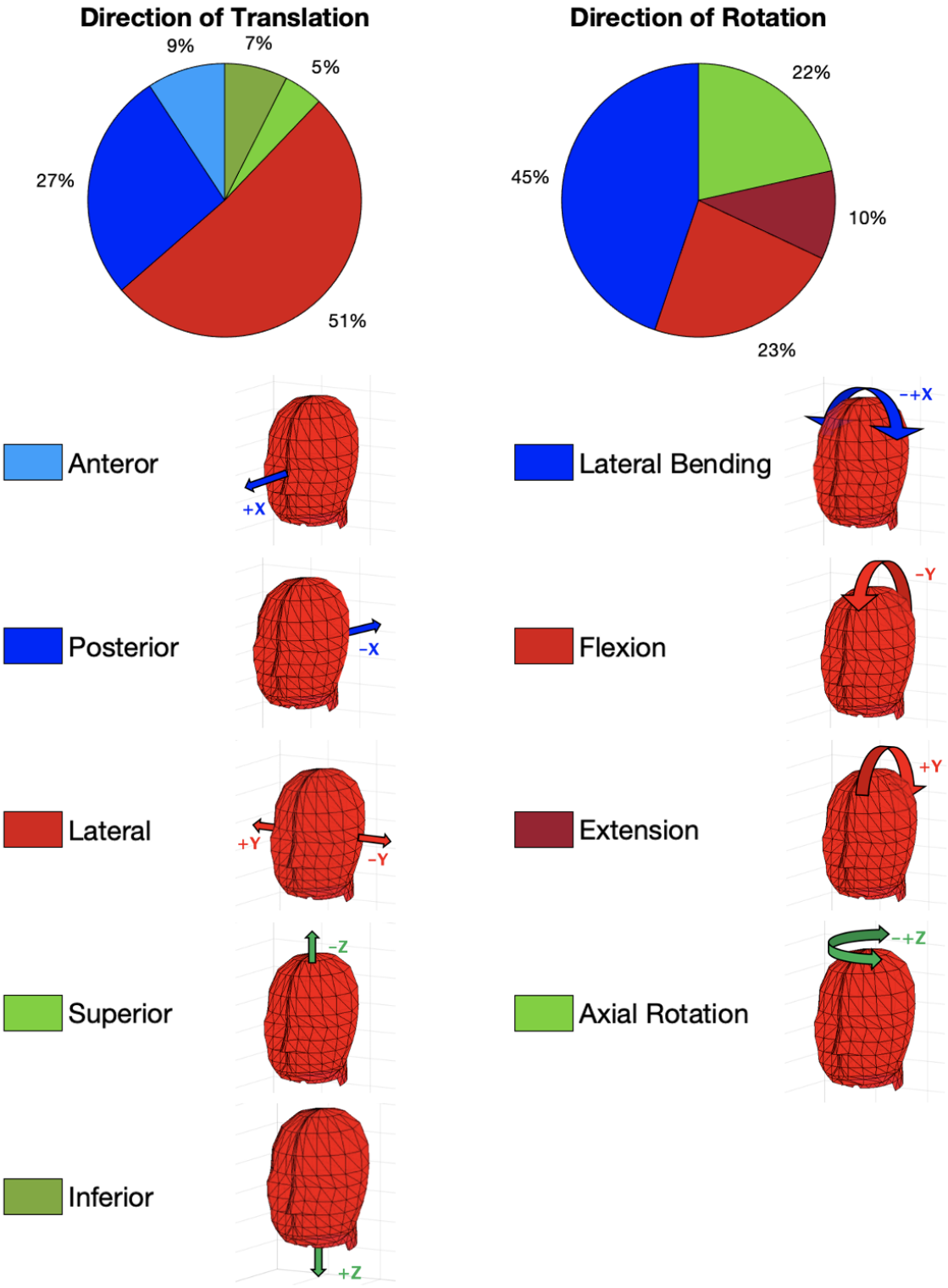

Figure 9. Predominate directions of translation and rotations. Axis directions in the legend refer to Figure 7D.

4.2 Qualitative Video Analysis

Contact Event Type and Tackle Phases

The combination of qualitative video analysis with timestamped HAE revealed that HAE occurred over multiple different contact events (Figure 10). For both the ball-carrier (57.5%) and tackler (43.9%), around half of all HAE occurred after the initial tackle or carry contact. Multiple HAE often occurred in the same contact phase, such that 501 tackle phases contained 725 HAE, with 64.9% of tackle phases containing a single HAE, 25.6% containing two HAE and 9.6% containing three or more. A greater proportion of HAE were sustained by tacklers (56.1%) than ball-carriers (43.9%).

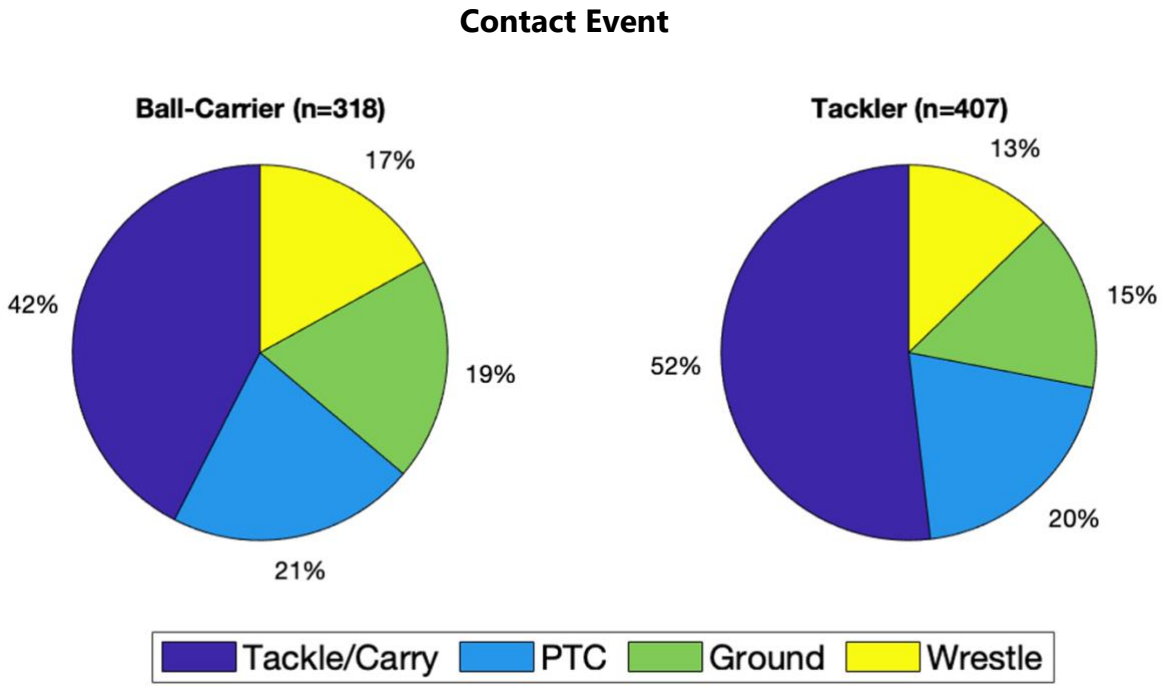

Figure 10. Pie charts showing proportions of contact events for ball-carrier and tackler HAE.

Direct and Indirect Head Loading

Indirect HAE accounted for a greater proportion of HAE for the ball-carrier (34.9%) than the tackler (18.3%). Indirect head loading was most prevalent in HAE from carries (50.4%), and ground impacts (44.7%), and accounted for 23.8% of HAE from tackles. No indirect HAE were recorded from PTCs or wrestles. In ball-carrier HAE, 70.5% of HAE from ground impacts were caused by indirect head loading, whilst in tacklers ground impacts were mostly direct (80.7%).

Tackle Height

All direct HAE from carries were caused by upper body tackles. Upper body tackles also caused a majority of indirect HAE from both carries (98.5%) and tackles (94.0%). Conversely, lower body tackles accounted for a greater proportion of direct HAE from tackles (52.8%).

Impacting Body Part

In direct HAE from lower body tackles, the impacting body part was mostly the hip of the ball-carrier in HAE from tackles (61.2%), whilst contact with the ball-carrier's shoulder (48.0%), arm (21.3%) or torso (20.0%) caused most of the HAE from tackles following upper body tackles. During an upper body tackle, the impacting body part was mostly the arm (43.1%) or shoulder (37.9%) of the tackler in direct HAE from carries. In HAE from PTCs, the most common impacting body parts were the arm (ball-carrier HAE = 44.9%, tackler HAE = 22.7%) and head (ball-carrier HAE = 36.7%, tackler HAE = 33.3%). In HAE from wrestles, the head (42.2%) and arm (22.2%) of the tackler were the most common impacting body parts for ball-carrier HAE, whilst the arm (37.0%), head (32.6%) and shoulder (19.6%) of the ball-carrier most commonly impacted the tackler in HAE from wrestles.

Tackle Type

The shoulder tackle was most common in HAE from tackles and carries, followed by the smother tackle (Figure 11). Shoulder tackles led to indirect head loading in 28.3% of HAE from tackles and 75.8% of HAE from tackles and smother tackles led to indirect head loading in 18.5% of tackles and 33.3% of HAE from tackles, whilst arm tackles led to mostly direct HAE (97.3%).

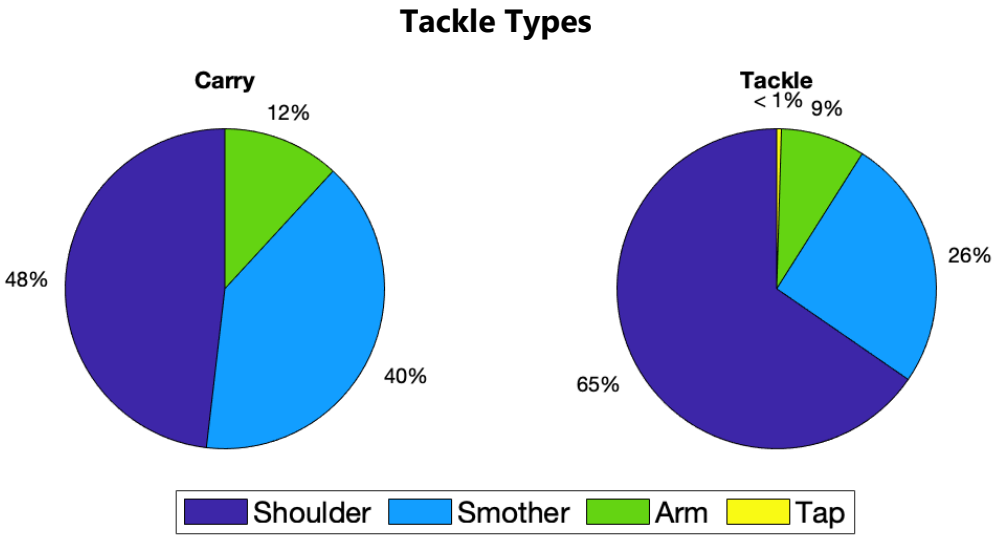

Figure 11. Pie charts showing proportions of tackle types for HAE from tackles and carries.

Tackle Sequence

HAE from different tackle sequences appear similar in both HAE from tackles and carries (Figure 12). Indirect HAE from carries and tackles were caused mostly by one-on-one (45.6% of HAE from carries, 42.0% of HAE from tackles) and simultaneous (41.2% of HAE from carries, 54% of HAE from tackles) tackle sequences.

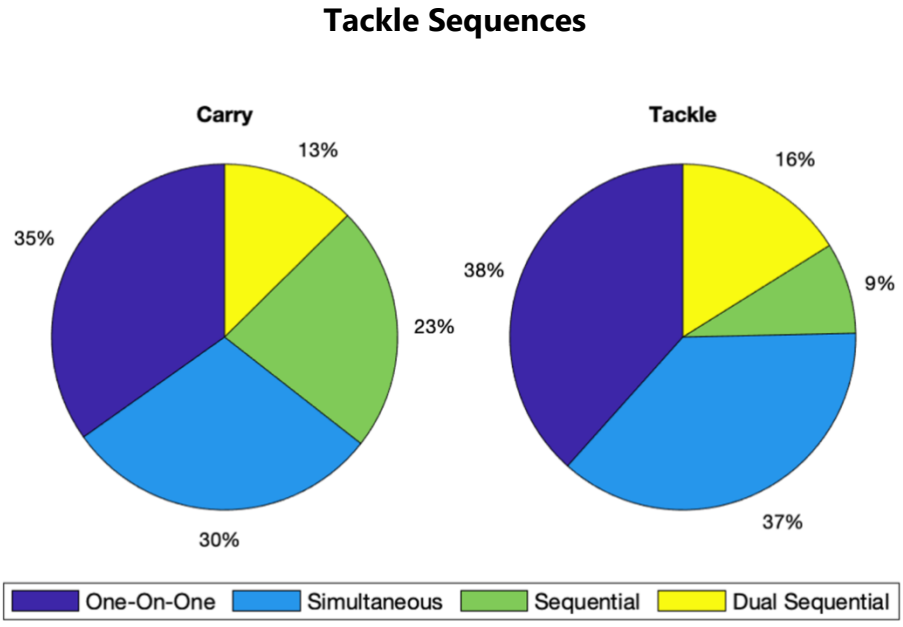

Figure 12. Pie charts showing proportions of tackle sequences for HAE from tackles and carries.

Tackle Direction

Tackles to the front of the tackler were most common in both tackles and carries (Figure 13).

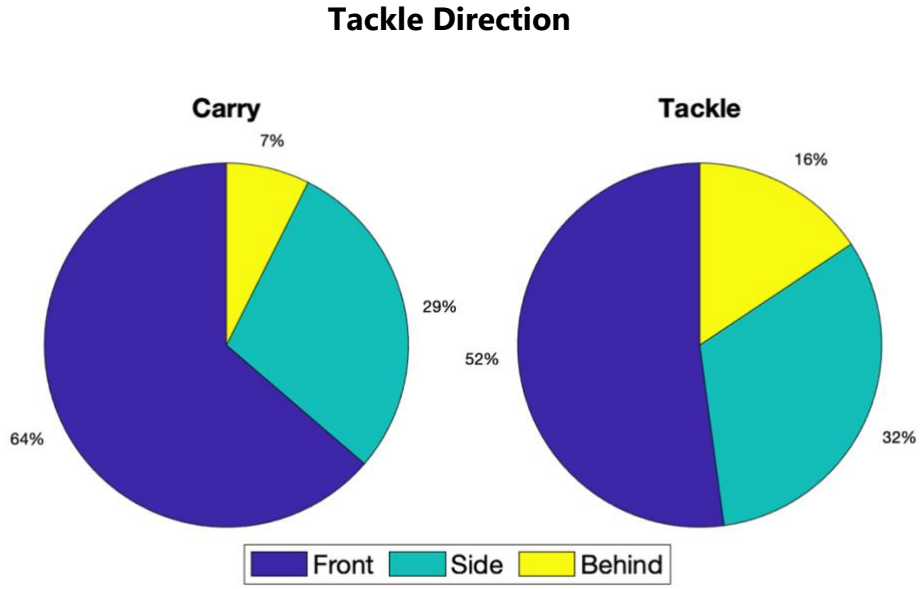

Figure 13. Pie charts showing proportions of tackle directions for HAE from tackles and carries.

Ball-Carrier Action

The ball-carrier's attention was on carrying the ball in 95.4% of HAE from carries and tackles, whilst the ball-carrier was focusing on catching the ball and passing the ball in 1.7% and 2.6% of all HAE from carries and tackles, respectively. No HAE were recorded whilst the ball-carrier was kicking or gathering the ball.

Impacting Player

Impacts with opposition players made up 100% of HAE from carries, 98.6% of HAE from tackles. Impacts with teammate accounted for 19.6% and 16.2% of HAE from PTCs and wrestles, respectively.

4.3 Comparing the Kinematics of Contact Events

Contact Events

PLA was not significantly different between contact events; however significant differences were observed between contact events for PAA and ΔPAV in the pairwise comparison of a mixed effect linear model (Figure 14). HAE from tackles resulted in significantly greater PAA than from carries, PTCs, ground impacts and wrestles (Figure 10B). HAE from PTCs and wrestles resulted in significantly lower ΔPAV than tackles, carries and ground impacts, whilst HAE from tackles led to a significantly greater ΔPAV than ground impacts (Figure 14C). Effect sizes, p values and pairwise comparisons are shown in Table 4.

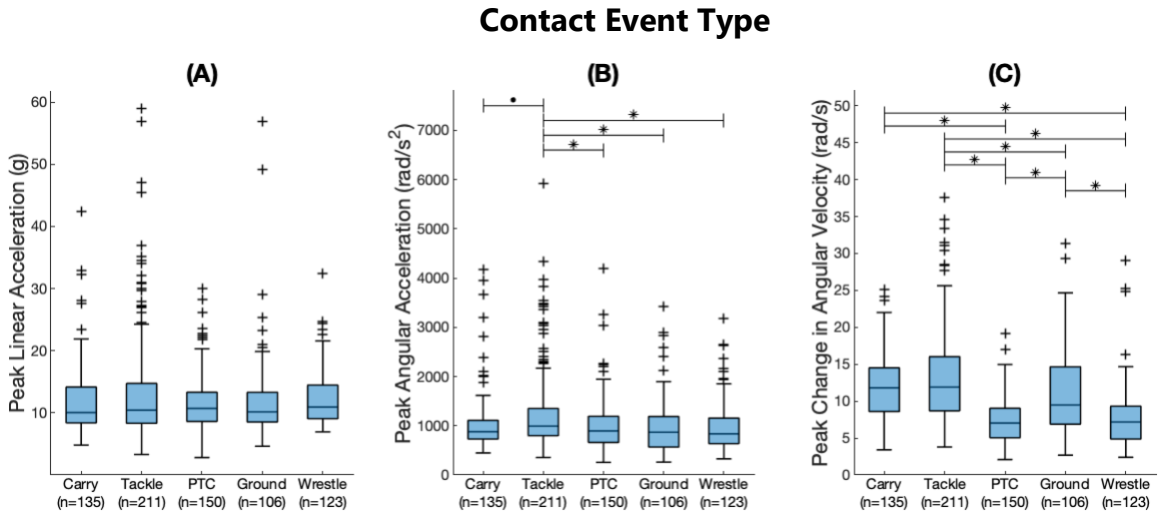

Figure 14. Box plots illustrating median and interquartile range (and outliers as crosses) head kinematics for carry, tackle, PTC, ground and wrestle contact events. Significantly different pairwise comparisons $p<0.05$ (•) and $p<0.01$ (∗) are indicated by whiskers terminating at each comparison.

Table 4. p values and effect size (with interpretation) of pairwise comparisons between contact events from the linear mixed-effects model. Asterisks indicate statistically significant comparisons ($p<0.05$).

	Peak Linear Acceleration		**Peak Angular Acceleration**		**Change in Angular Velocity**	
Comparison	**p value**	**effect size**	**p value**	**effect size**	**p value**	**effect size**
Carry - Tackle	0.33	-0.11 (Trivial)	0.02*	-0.27 (Small)	0.24	-0.13 (Trivial)
Carry - PTC	0.98	0.00 (Trivial)	0.40	0.10 (Trivial)	<0.001*	1.06 (Moderate)
Carry - Ground	0.79	-0.03 (Trivial)	0.06	0.24 (Small)	0.05	0.24 (Small)
Carry - Wrestle	0.17	-0.18 (Trivial)	0.23	0.16 (Trivial)	<0.001*	0.98 (Moderate)
Tackle - PTC	0.32	0.11 (Trivial)	<0.001*	0.37 (Small)	<0.001*	1.19 (Moderate)
Tackle - Ground	0.51	0.07 (Trivial)	<0.001*	0.51 (Small)	<0.001*	0.37 (Small)
Tackle - Wrestle	0.54	-0.07 (Trivial)	<0.001*	0.42 (Small)	<0.001*	1.11 (Moderate)
PTC - Ground	0.80	-0.03 (Trivial)	0.25	0.14 (Trivial)	<0.001*	-0.82 (Moderate)
PTC - Wrestle	0.16	-0.18 (Trivial)	0.65	0.06 (Trivial)	0.53	-0.08 (Trivial)
Ground-Wrestle	0.26	-0.15 (Trivial)	0.54	-0.08 (Trivial)	<0.001*	0.74 (Moderate)

Head Loading Type

Direct HAE resulted in significantly greater PLA ($p < 0.001$, ES = 0.67, interpretation = moderate) and PAA ($p<0.001$, ES = 0.39, interpretation = small) than indirect HAE, but indirect HAE resulted in significantly greater ΔPAV than direct HAE ($p < 0.001$, ES = 0.69, interpretation = moderate) (Figure 15).

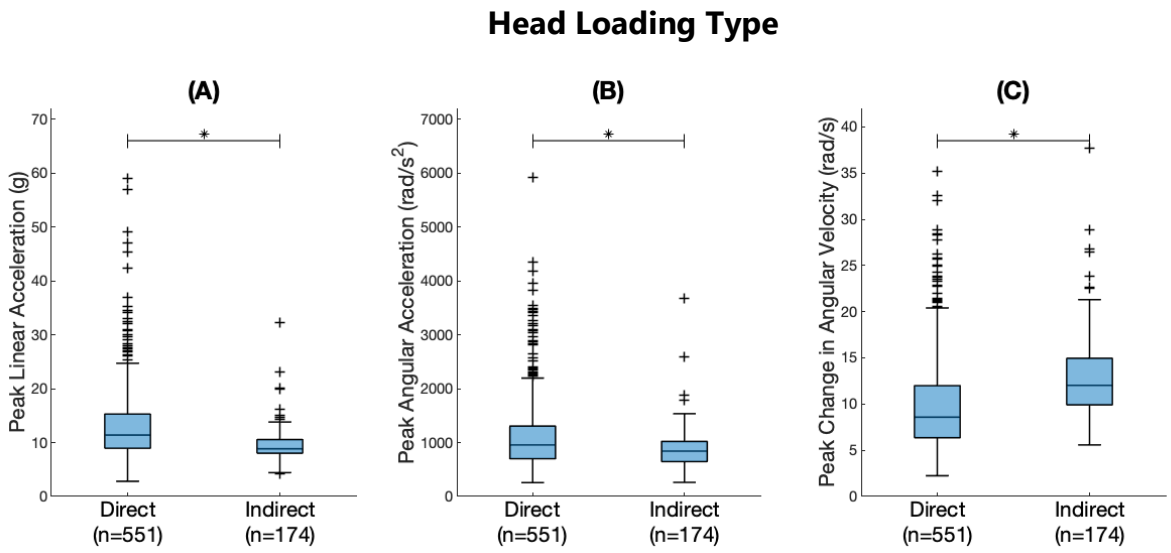

Figure 15. Box plots illustrating median and interquartile range (and outliers as crosses) head kinematics for direct and indirect HAE. Significantly different pairwise comparisons $p<0.05$ (•) and $p<0.01$ (∗) are indicated by whiskers terminating at each comparison.

Tackle Sequences

In HAE from tackles, dual-sequential tackle sequences led to significantly greater PLA than simultaneous tackle sequences ($p = 0.048$, ES = 0.5, interpretation = small), conversely simultaneous tackle sequences led to significantly greater ΔPAV than dual-sequential tackles sequences ($p = 0.016$, ES = 0.55, interpretation = small) (Figure 16). Figure 17 shows that in HAE from carries, dual-sequential tackle sequences caused a significantly greater PLA than one-on-one ($p < 0.01$, ES = 0.8, interpretation = moderate), simultaneous ($p < 0.01$, ES = 0.93, interpretation = moderate) and sequential ($p < 0.01$, ES = 0.93, interpretation = moderate) tackle sequences, whilst one-on-one tackle sequences led to significantly greater ΔPAV in HAE from carries than sequential tackle sequences ($p = 0.03$, ES = 0.45, interpretation = small).

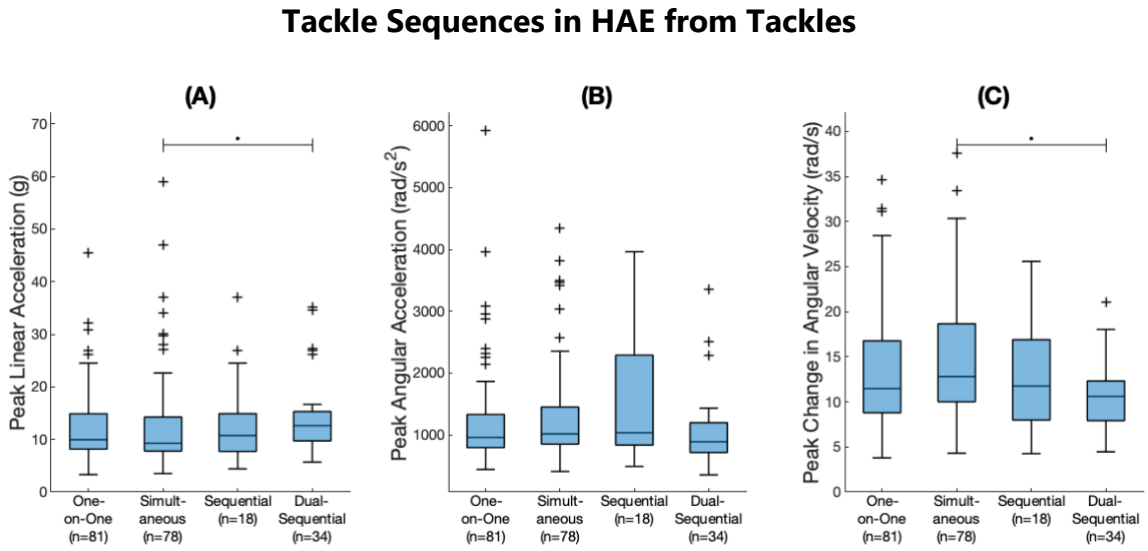

Figure 16. Box plots illustrating median and interquartile range (and outliers as crosses) head kinematics for tackle sequences in HAE from tackles. Significantly different pairwise comparisons $p<0.05$ (•) and $p<0.01$ (∗) are indicated by whiskers terminating at each comparison.

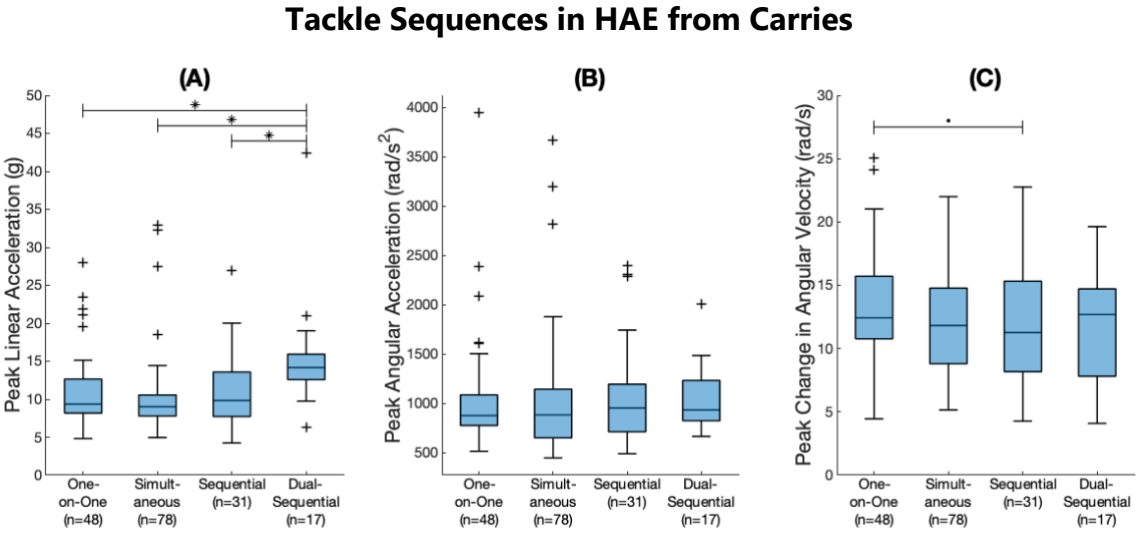

Figure 17. Box plots illustrating median and interquartile range (and outliers as crosses) head kinematics for tackle sequences in HAE from carries. Significantly different pairwise comparisons $p<0.05$ (•) and $p<0.01$ (∗) are indicated by whiskers terminating at each comparison.

Tackle Types

There were no significant differences between tackle types in PLA, PAA or ΔPAV in HAE from tackles (Figure 18). Similarly, in HAE from carries, there were no differences in PLA or PAA between tackle types, however shoulder tackles led to a significantly

greater ΔPAV than both smother (p = 0.048, ES = 0.33, interpretation = small) and arm tackles (p < 0.01, ES = 0.64, interpretation = moderate) (Figure 19).

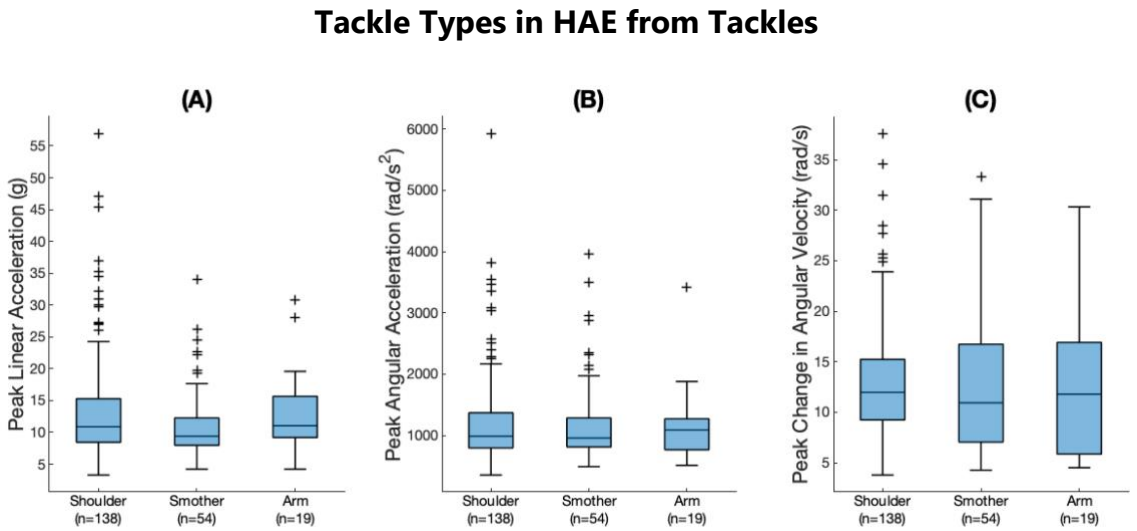

Figure 18. Box plots illustrating median and interquartile range (and outliers as crosses) head kinematics for tackle types in HAE from tackles. Significantly different pairwise comparisons p<0.05 (•) and p<0.01 (∗) are indicated.

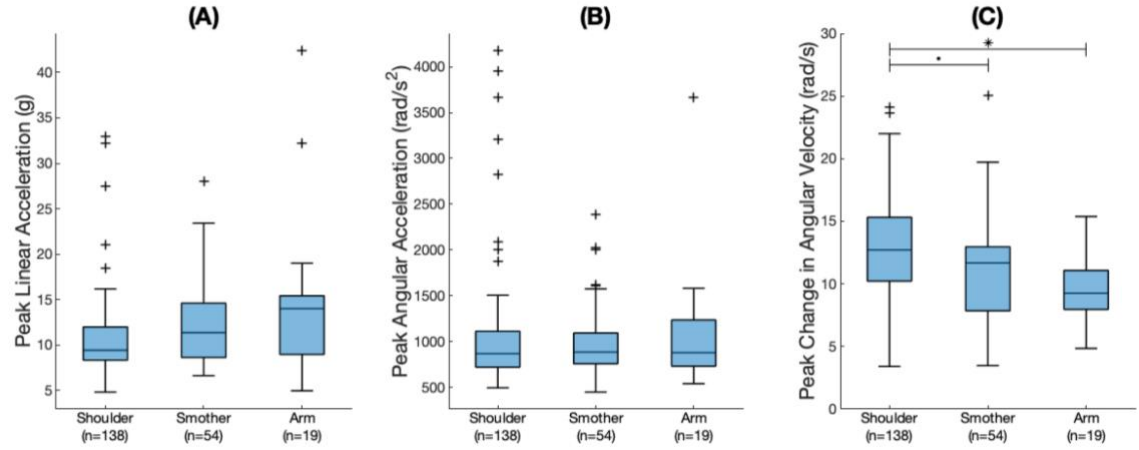

Figure 19. Box plots illustrating median and interquartile range (and outliers as crosses) head kinematics for tackle types in HAE from carries. Significantly different pairwise comparisons p<0.05 (•) and p<0.01 (∗) are indicated.

Tackle Height in Direct HAE from Tackles

There was no significant difference in PLA (p = 0.13), PAA (p = 0.19) or ΔPAV (p = 0.51) between direct HAE from tackles to the upper body and direct HAE from tackles to the lower body (Figure 20). There was insufficient data to compare the effect of tackle height in HAE from carries.

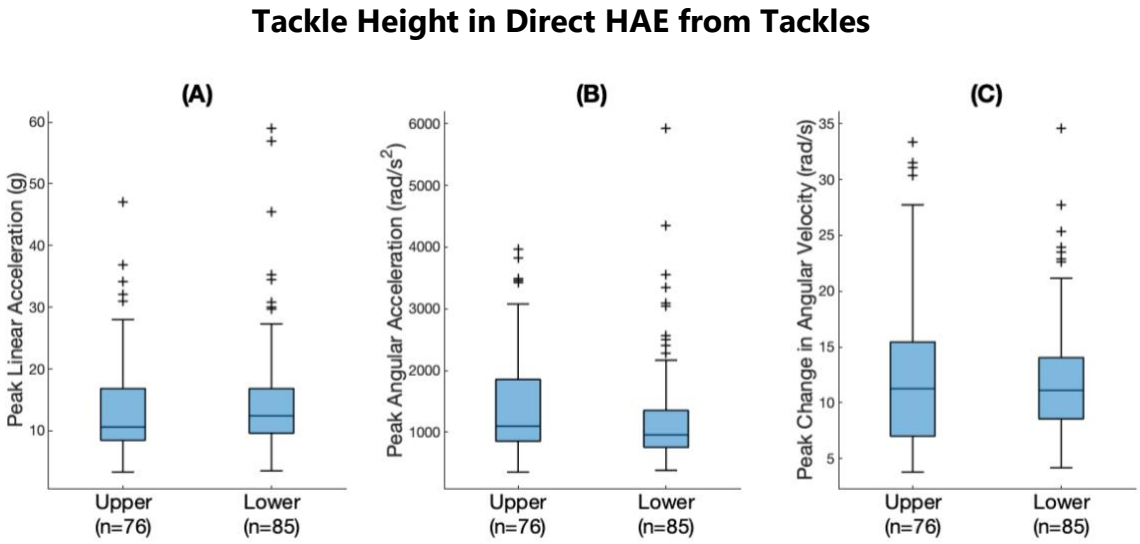

Figure 20. Box plots illustrating median and interquartile range (and outliers as crosses) head kinematics for direct HAE from tackles to the upper and lower body.

4.4 Head Acceleration Exposure Rates

The exposure rates of forwards, backs and the entire cohort are shown in Figure 21. A total of 27 player matches were captured by the instrumented mouthguards. Across the 21 player matches in forwards, the average active player minutes was 49.6 (SD = 17.0), whereas in the six player matches in backs the average active player minutes was 78.8 (SD = 4.1). This was due to forwards being interchanged more often than backs across the games in this study.

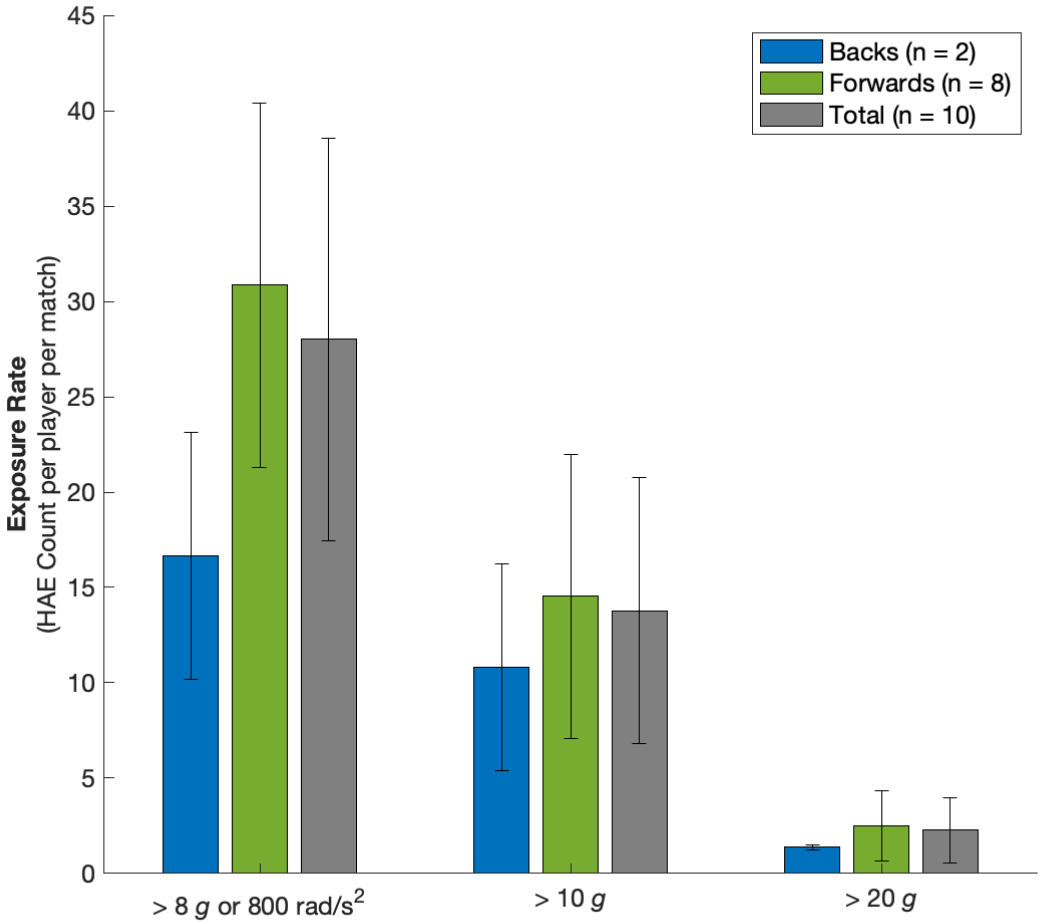

Figure 21. Exposure rates for backs, forwards, and the entire cohort at three different thresholds. Whiskers show standard deviation.

5 Discussion

The aim of this thesis was to quantify and characterise HAE sustained during professional rugby league games. Qualitative video analysis of instrumented mouthguard data allowed for HAE to be characterised. These data can be used to inform HAE reduction strategies, by identifying the contact events and characteristics that lead to HAE. Our results revealed that multiple HAE can occur in the same tackle phase from different contact event and from different tackle sequences. Around half of all HAE occurred after the initial contact between players, which is consistent with previous findings in junior rugby league (Carey et al., 2020), and around two thirds of all HAE from tackles and carries occurred after the one-on-one collision. This suggests that to monitor HAE exposure effectively with video analysis alone, the entire tackle phase should be considered for each player and would therefore be a tedious approach. Consequently, the combination of kinematic data from wearable sensors with qualitative video analysis is the most efficient and comprehensive way to effectively monitor and understand HAE exposure in rugby league and other sports.

Indirect HAE accounted for around a quarter of all HAE, and around half of all HAE from carries. Indirect HAE led to a significantly greater ΔPAV than direct HAE, whilst PLA and PAA were significantly less in indirect HAE than direct HAE. Similar findings have been reported in previous studies (Tierney et al., 2020, Carey et al., 2020, Kieffer et al., 2020b). Conversely, indirect HAE have previously been reported to lead to significantly greater PAA than direct HAE (Tierney et al., 2020), however the authors indicated that direct head contact may have occurred during events erroneously qualitatively labelled as indirect HAE. Typically, we would expect higher head kinematics for a direct HAE than indirect HAE, given the same impact conditions. This suggests that qualitative video analysis alone may be insufficient for labelling HAE and the use of time-traces and video reconstructions (Figure 7) is beneficial for qualitative video analyis, particularly in identifying the head loading type. Given that ΔPAV was greater in indirect HAE in the present study, indirect HAE may play a significant role in the accumulation of brain loading in rugby league players. However, whilst PAV has been shown to correlate well with brain strain (Takhounts et al., 2013, Bian and Mao, 2020), more research is needed to ascertain the relationship between ΔPAV and brain injury. Nonetheless, these findings

clearly illustrate the importance of considering body collisions when assessing the HAE exposure of rugby league players.

Tackle height has been consistently identified as a risk factor of concussions in rugby league (King et al., 2012, Gardner et al., 2016, Gardner et al., 2015b) and a previous study in rugby union demonstrated that lower tackle heights result in significantly lower head kinematics from indirect HAE in ball-carriers (Tierney et al., 2018b, Tierney and Simms, 2017). Indeed, upper body tackles led to virtually all HAE from carrying the ball, with the exception of some lower body tackles leading to indirect HAE in ball-carriers. This could support the notion that encouraging lowering tackle heights could reduce the HAE exposure from carrying the ball. However, this does not include HAE from ground impacts. Tackle height is not thought to be a factor in the risk of concussion for tacklers in rugby league (Gardner et al., 2015b), which is consistent with our finding that direct HAE from tackles did not result in head kinematics significantly different in upper body tackles and lower body tackles. Similarly, tackle type did not appear to influence HAE kinematics. Shoulder tackles led to significantly greater ΔPAV than smother and arm tackles in HAE from carries, however this is likely driven by an increased incidence of indirect HAE from shoulder tackles whilst carrying the ball. It is likely that a more granular approach to investigating the effect of tackle height is necessary to investigate differences in HAE kinematics from different tackle techniques. For example, given that the tackle sequence appears to influence HAE kinematics, tackle technique should be analysed separately for each tackle sequence (e.g., one-on-one tackles only). This data would be valuable for identifying the safest and most dangerous tackle techniques for both the ball-carrier and tackler and could be used to inform rule changes and tackle technique to reduce HAE exposure and risk of concussion.

Our results demonstrate that HAE are skewed to lower kinematics. This suggests that even small changes to data acquisition thresholds can have a large effect on the HAE counts reported in studies. Indeed, when using different data acquisition thresholds to report HAE exposure, an 8 g or 800 rad/s^2 threshold (28.0 ± 10.6 HAE per player per match) reported around double the number of HAE per player, per match than a 10 g threshold did (13.8 ± 6.9 HAE per player per match). By the same logic, small differences in the way different mouthguards report kinematics can also have a large effect on the

HAE count reported in studies. A higher exposure rate of 76 ± 42 HAE above 10 *g* per player, per match has been reported in amateur rugby union players using instrumented mouthguards (King et al., 2015), however given that different instrumented mouthguards were used between studies a direct comparison between HAE exposure in sports is not supported. An independent validation of different instrumented mouthguard devices should be carried out before findings using different devices are compared. Furthermore, a false negative analysis of all instrumented mouthguard devices should be carried out to ensure that exposure rates are not underreported.

Previous studies from amateur rugby league studies using patch devices (King et al., 2017a, King et al., 2020, King et al., 2018, Carey et al., 2019, Carey et al., 2020, King et al., 2017c) have reported greater HAE exposure rates in forwards than backs. GPS and video analysis data revealed that forwards are involved in a greater number of collisions per game than backs (Cummins and Orr, 2015, Naughton et al., 2020), which may explain these findings. Whilst trends in our data may also support these findings, due to a lack of a statistical test comparisons between forwards and backs in this study is not supported. A statistical test was not supported due to a small number of player matches for backs (n = 7). HAE from tackles resulted in a significantly greater PAA than HAE from carries, which may suggest that our results support previous findings that the tackler is at a greater risk than the ball-carrier in a tackle (Gardner et al., 2016), however no differences were seen in PLA or ΔPAV. Given that forwards are involved in more tackles than carries (Naughton et al., 2020) and that tackles can lead to higher HAE kinematics, forwards may suffer from brain injury at a greater rate than backs.

Directional data from instrumented gyroscopes and accelerometers revealed that lateral bending and lateral translation were the most common predominant directions of angular and linear acceleration, respectively. These findings could be used to inform neck strengthening strategies to reduce HAE kinematics. Utilising the directions of translation and rotation during HAE to assess the injury risk may also have benefits over the use of absolute resultant values such as PLA, PAA and ΔPAV that do not account for the direction of acceleration. FE head modelling simulations have revealed that different directions of rotation principally affect different areas of the brain, for example lateral bending elicits the highest strain in the thalamus and corpus callosum when compared with other

directions of angular acceleration (Bian and Mao, 2020). Therefore, Bian and Mao (2020) suggest that the direction of rotation may be an important factor in the mechanism of brain injury and concussion. Despite this, the effect of the direction of angular acceleration on the risk of concussion or brain injury has not been directly investigated. The methodology of this thesis demonstrates that directional data is readily available with the use of instrumented mouthguards and can be investigated alongside clinical measures, though validation as to the accuracy of the directions of rotation and translation may need to be undertaken. Another possibility is to use mouthguard data to predict brain strains in specific ROIs of the brain using an FE head model or a CNN (Liu et al., 2020).

5.1 Limitations and Future Work

PLA, PAA and ΔPAV were used to describe HAE in this study. Peak scalar kinematics such as those used in this study ignore directional and temporal data. Rotation direction and impact duration and deceleration times have been reported to have an effect on the degree of brain strain in HAE (Bian and Mao, 2020). Given that brain strain describes the deformation of the brain, disregarding directional and temporal data may be a reductive approach when describing the magnitude of HAE. Brain strain metrics are a good way of implementing temporal and directional data, however due to an insufficient amount of pre- and post-trigger data collected by the mouthguards used in this study, brain strain metrics were unable to be collected using our instrumented mouthguard data (Liu et al., 2021). Currently the relationship between these kinematics and brain injury is not well understood. These kinematics have been implemented into BIC to assess the risk of brain injury (Section 2.3.6). However, BIC are mainly used to assess the risk of skull fractures and concussion. The magnitude of kinematics that contribute to the long term effects of repetitive HAE exposure (Section 2.2) has not been investigated. Therefore, future studies should incorporate clinical measures alongside HAE kinematics in a longitudinal study design. Different kinematic metrics can be investigated to see which best correlate with various clinical measures to ascertain which kinematics are most appropriate to describe HAE. Furthermore, the minimum kinematics associated with clinical outcomes could be used to set data acquisition thresholds in future biomechanical studies. Previously, HAE kinematic measures have been combined with DTI measures in

American footballers (McAllister et al., 2014, Bahrami et al., 2016, Davenport et al., 2014, Kuzminski et al., 2018), a similar study design should be carried out with rugby league players to reveal the effect of rugby league participation on white matter integrity. Other clinical outcomes could include cognitive and motor function, mental health and the presence of neurodegenerative diseases, which have not been investigated in rugby league players.

As the relationship between brain injury and HAE kinematics is not understood, data acquisition thresholds are selected to remove HAE kinematics sustained from non-contact events. Despite being lower than most data acquisition thresholds employed in studies measuring HAE in contact sport (King et al., 2016), our data acquisition threshold of 8 g or 800 rad/s^2 used in this thesis may also underestimate the number of HAE sustained as a result of contact events. Utilising a lower data acquisition thresholds towards of 4 g or 200 rad/s^2 (Miller et al., 2019) would likely compromise the sensitvity of the instrumented mouthguards and increase their false negative rate. A 5 g single axis trigger threshold is used on the Prevent Biometric Custom Fit Mouthguard and was selected by its manufacturers to minimise the number of HAE triggered by non-contact events and noise. Reducing the trigger threshold below 5 g would cause the mouthguard to trigger more often from noise, as the filtering process occurs after a HAE is triggered. Each time the mouthguard triggers pre- and post-trigger data are collected and following the collection of post-trigger data there is a brief time period where the mouthguard cannot record data. This period is termed the rearming time. In the Prevent Biometric Custom Fit mouthguard, the soonest another HAE can trigger after the end of the previous HAE is 27 ms, this gives a reaming time of 17 ms, as 10 ms of pre-trigger data is recorded in the subsequent HAE. An increased trigger rate of the mouthguard would increase the likelihood of peak kinematics occurring during the rearming time of the mouthguard, which would result in HAE being missed or underreported. Consequently, trigger thresholds below 5 g are not recommended. An appropriate method to determine a data acquisition threshold could be to directly measure the peak kinematics sustained by rugby league players during non-contact events such as running and cutting manoeuvres, to include only the HAE with peak kinematics greater than those that result from non-contact events. The trigger threshold in the Prevent Biometric Custom Fit mouthguard is taken from accelerometer values, therefore a linear acceleration trigger threshold must

be used. However, angular kinematics such as PAA can be incorporated into data acquisition thresholds, applied after the trigger threshold. Currently, studies measuring the head kinematics in contact sport have only implemented linear acceleration data acquisition thresholds (King et al., 2016). Given the importance placed on angular acceleration in the mechanisms of brain injuries and concussion (Section 2.4.2.), a combined linear and angular data acquisition threshold may be more appropriate in future studies, to include impacts with low linear acceleration but high angular kinematics.

Exposure rates are reported in this study may underreport the HAE sustained by participants given that a false negative analysis was not conducted. HAE were all qualitatively analysed and therefore video verified. Quantifying the false negative rate is not possible using video analysis alone because it is not clear whether visible HAE are not collected by the mouthguard due to an insufficient magnitude to trigger the mouthguard or due to device errors. Laboratory based ATD validation with controlled impact magnitudes above the trigger threshold may allow for the false negative rate to be quantified, however this would not account for other variables associated with on-field data collection, such as shouting, variation in adherence to teeth and other voluntary head movements.

Under the current study design, only the tackles and carries that resulted in a mouthguard triggered HAE were qualitatively analysed. Whilst comparisons of the incidence of HAE characteristics can be made between tackler and ball-carrier HAE, it is unclear in the present study whether an increased incidence of specific HAE characteristics is reflective of an increased propensity to result in a HAE, or that they are more common in all tackles. Combining current data with data from the qualitative analysis of all tackles and carries that do not result in a mouthguard triggered HAE would allow us to identify how likely each tackle characteristic is to result in a HAE. Any future studies qualitatively analysing HAE within rugby league and other contact sports should compare the incidence of HAE characteristics with the characteristics of contact events that do not result in sensor triggered HAE, in order to quantify the risk of each characteristic leading to a HAE. This would allow for safer tackle techniques to be identified more effectively and could be used to inform strategies to reduce the HAE

exposure sustained by rugby league players. Performance outcomes should also be investigated alongside HAE kinematics to identify safe tackle techniques that are effective from a performance perspective. Including a performance outcome can lead to a greater impact on the sport as players and coaches are more incentivised to implement them.

The sample in this study included more forwards than backs. This means that a comparison between the incidence of ball-carrier HAE and tackler HAE is not supported, as forwards are involved in more tackles than carries (Naughton et al., 2020), therefore HAE are likely to be skewed towards tackler HAE. Given that the collision load is different between backs and forwards, a separate approach to analysing their HAE may be required. Only male professional players were monitored in this study and therefore results are likely not applicable to female, or amateur players. A future league wide study using instrumented mouthguards has now been approved which will include professional and amateur male and females (BBC Sport, 2021). The methodology used in this thesis will provide the basis for the league wide study and raises important questions throughout the literature review and discussion that the study will need to address. This thesis did not contain data from any training sessions and so the league wide study should quantify and characterise the HAE exposure from training. A more granular analysis of tackle technique will be possible with a larger sample size. Given the openness and dynamic nature of tackles in rugby league, it may be more appropriate to investigate one-on-one, simultaneous, sequential, and dual-sequential tackles and carries separately, as the sample size in the current thesis was insufficient to do so. This would allow for a more detailed understanding of the effect of tackle technique on the magnitude of head kinematics and could be used to inform concussion mitigation and HAE reduction strategies. Furthermore, more robust exposure rates can be calculated to compare the incidence of HAE between playing positions, males and females and professional and amateurs in the sport.

5.2 Conclusion

Results from this study suggest that HAE occur throughout the entire tackle phase, with multiple HAE often occurring in the same phase. Therefore, the use of video analysis alone is an inefficient way to monitor the HAE exposure of rugby league players.

Qualitative analysis also revealed that indirect HAE accounted for around a quarter of all HAE, and half of all HAE to ball-carriers. Indirect HAE also led to a significantly greater ΔPAV than direct HAE. This illustrates the importance of considering body collisions when assessing the HAE exposure of rugby league players. Virtually all HAE from carrying the ball occurred from upper body tackles which supports the notion that encouraging lower tackle height through coaching and rule changes can reduce the HAE exposure to ball-carriers. Tackle height did not significantly affect the kinematics of HAE from tackles. The kinematics of HAE were skewed to lower values. This means that data acquisition thresholds have a large effect on HAE counts reported in studies. A 10 g data acquisiton threshold led to an exposure rate half of an 8 g or 800 rad/s^2 threshold did. By the same logic, this means that if instrumented mouthguard devices report kinematics slightly higher or lower than one another then there will be large differences in the HAE count reported. Consequently, comparison of exposure rates reported using different devices is not supported until the kinematics of on-field HAE are validated against one another.

In order to eludicate which tackle and carry techniques are the safest, the characteristics of contact events that do not result in HAE should be identified and compared to the dataset in this study. Future research should also endeavour to include both clinical and performance outcomes. Including clinical outcomes can allow us to understand which kinematics are most important in brain injury, whilst including performance outcomes can allow for the safest and most effective tackle techniques to identified, thus incesntivising coaches to implement coaching strategies that can help to reduce HAE exposure. The methodology and findings from this thesis will be used to inform future studies arising from the league wide instrumented mouthguard project (BBC Sport, 2021).

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