Sports Medicine
https://doi.org/10.1007/s40279-025-02314-2

ORIGINAL RESEARCH ARTICLE

Contact Role and Tackle Characteristics Shape Head Acceleration Exposure in Male Community Rugby: A Cohort Study Utilising Instrumented Mouthguards

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Accepted: 22 August 2025

Abstract

Background Rugby Union has attracted increased scrutiny because of concerns over head acceleration events (HAEs), particularly regarding their frequency, severity and potential long-term health implications. While substantial efforts by governing bodies have focused on reducing head impact risks through education, regulatory changes and the introduction of instrumented mouthguards, limited data exist for the community rugby context, especially across different age grades and playing positions.

Objective We aimed to quantify HAE across playing positions, age grades and contact phases in community rugby and to identify match scenarios associated with high-magnitude head loading.

Methods A prospective observational cohort study included 259 male players across U13, U15, U19, and Premier senior men's grades. Players were fitted with instrumented mouthguards, and match play was video recorded for verification. Head acceleration events were identified from instrumented mouthguard-triggered sensor acceleration events > 5 g and coded for match context, player position and contact event characteristics. Statistical models evaluated differences in HAE frequency, incidence rates and head kinematics (peak linear acceleration, peak angular acceleration, rotational velocity change index) across grades, positions and contact scenarios.

Results A total of 7358 HAEs were verified from 8593 sensor acceleration events across 72 matches. Tackles and rucks accounted for ~ 60% of all HAEs. High tackles significantly increased head loading in ball carriers (peak linear acceleration: + 4.16 g, $p = 0.02$; peak angular acceleration: + 443 rad/s^2, $p = 0.002$; rotational velocity change index: + 1.87 rad/s, $p = 0.04$), while low tackles elevated head loading in tacklers (peak linear acceleration: + 4.9 g, $p = 0.004$). Upright tacklers were more likely to produce high tackles ($p < 0.001$) and head-to-head contacts ($p = 0.019$). U13 ball carriers showed higher rotational loading than tacklers (rotational velocity change index: + 5.01 rad/s, $p = 0.008$), likely reflecting frequent secondary mechanisms such as head-to-ground and body-to-ground. Defensive rucks carried a greater HAE risk than attacking rucks (all $p < 0.05$), particularly for U19 jacklers (incidence rate ratio = 2.27, $p < 0.0001$).

Conclusions Tackles and rucks are primary sources of HAEs, with risk shaped by posture, tackle height and player role. Lower tackle heights reduce ball carrier load but increase tackler exposure, indicating a potential safety trade-off. Younger players, particularly U13s, were more susceptible to secondary impacts (e.g. head-to-ground), potentially because of limited task-specific experience and underdeveloped control during the tackled phase. Position- and age-specific strategies may be required to optimise safety and reduce HAE risk across all levels of community rugby.

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Published online: 17 September 2025

Key Points

Age-Related Susceptibility

Although younger players (e.g. U13s) sustain fewer high-magnitude head acceleration events, they are more vulnerable to secondary impact mechanisms such as head-to-ground or body-to-ground, which result in higher rotational accelerations. This pattern may reflect age-related factors such as neuromuscular development, limited task-specific experience and less exposure to tackled-phase technique training. These findings highlight the potential value of age-appropriate interventions focused on posture, falling technique and neck control to reduce head acceleration event risk in youth rugby.

Impact of Tackle Technique and Height

The findings reinforce existing evidence that high tackles and upright posture pose a significant risk to ball carriers, and increase the likelihood of head-to-head contacts. However, low tackle events expose tacklers to similar acceleration loads as high tackles. This dual risk outcome highlights the need for regulatory and coaching strategies that promote mid-range tackles and technique training focused on posture and head placement.

Ruck Roles and Defensive Risks

Defensive rucks exhibited a higher propensity for head acceleration events compared with attacking rucks. Players in defensive roles, particularly jacklers and cleaners, were more frequently exposed to high magnitude events (> 30 g), especially in school-age players (U15 and U19). These findings emphasise the importance of role-specific technique training and monitoring to reduce risk in high-contact ruck engagements.

1 Introduction

Over the past decade, Rugby Union has come under increased scrutiny as awareness and concern about the cumulative effects of head acceleration events (HAEs) have grown [1, 2]. Although, not every contact scenario produces a measurable HAE [3], and only a fraction of these HAEs result in a diagnosed concussion [4], every confirmed concussion can be traced to a significant antecedent acceleration event [5]. This knowledge highlights the critical role of instrumented mouthguard (iMG) monitoring in mapping HAEs to contact events whether symptomatic or not [6]. Systematic iMG monitoring provides an essential complement to conventional injury surveillance strategies, a benefit that is especially pertinent to community rugby, where medical staffing and video-based review are less consistent than in the professional space [6].

Despite increasing efforts to monitor and mitigate head acceleration exposure in Rugby Union, most of what is known about game-related HAEs comes from the professional side [3, 7–9]. Community competitions differ markedly from the elite game, with a wider variety in athlete maturation, coaching resources and match tempo, factors that influence both the frequency and biomechanics of contact phases [10]. Evidence from age-grade academy rugby indicates that as players progress from U12 to senior levels, there is a sharp increase in ball-in-play time, collision frequency and the proportion of active shoulder tackles [10, 11]. Tackles and rucks, which dominate contact volume, therefore warrant specific attention in community settings.

Preliminary studies conducted in New Zealand community rugby (U13, U15, U19 and senior Premier men) reinforced these concerns: tackles accounted for roughly 60% of all HAEs, forwards accumulated greater head loading than backs in the older grades [12, 13]. What remains unclear, however, is how specific match events, player roles and playing positional demands interact across the adolescent-to-adult continuum in community rugby. Without this information, targeted prevention strategies such as tackle-technique coaching, modified rules or neck-strength programmes risk being poorly aligned with the true exposure profiles of community players.

The present study contextualises the HAE incidence in male community matches. By linking iMG data to detailed match coding, we examine (i) how HAEs vary by playing position, (ii) which contact phases (tackles, rucks, scrums, mauls) and player roles contribute most to head loading and (iii) whether the age grade modifies these relationships. The findings are intended to guide age- and role-specific injury-mitigation strategies that can be implemented feasibly in the community game.

2 Methods

2.1 Participants

This prospective observational cohort study included 259 male players recruited from local clubs and schools, representing the spectrum of community rugby where contact was permitted. Informed consent was obtained from all players, and legal guardian consent was also obtained from all players under the age of 16 years. This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki, and ethical approval was granted by the University of Otago Human Ethics Committee (approval number H21/056).

As per the "Game On Provisions" within New Zealand Rugby's domestic safety laws, the duration of matches differed depending on the playing grade and number of players per side [14]. The length of a match for school age players may range from 40 min at minimum (10 a side) to 70 min at maximum (15 a side), while the Premier grade played typical 80-min matches. Detailed participant characteristics, including the number of unique matches, total player matches (defined as matches in which players wore iMGs) and match durations by age group, are provided in Table 1.

2.2 Study Equipment

Following established protocols, players were fitted with a boil-and-bite iMG (Prevent Biometrics, Edina, MN, USA), with the fitting performed by a trained dentist to ensure a secure and accurate fit to each player's dentition [13]. The iMG contains an infrared proximity sensor, a 3.2-kHz triaxial accelerometer, and a gyroscope with measurement ranges of 200 g and 35 rads/s. The iMG included a triaxial accelerometer and gyroscope, operating at 3.2 kHz, with measurement ranges of $\pm$200 g and $\pm$35 rad/s. The proximity sensor generated a temporal log period when the iMG was securely "on-teeth". An impact trigger was set to activate when acceleration exceeded 5 g on any axis (x, y, z) [15], capturing a 50-ms recording window (10-ms pre-trigger and 40-ms post-trigger).

Matches were video recorded using multiple camera angles, including fixed cameras positioned end-on and side-on, as well as a third angle provided by a referee head-mounted camera (Hero8; GoPro Inc., San Mateo, CA, USA). The synchronised footage was imported into Hudl Sportscode (v11; Agile Sports Technologies Inc., Lincoln, NE, USA) for detailed analysis.

The iMG data were exported in XML format and aligned with the Sportscode timeline using a clock flash captured in the video. Each player's unique mouthguard identifier was matched to their jersey number, enabling individual event timelines to be synchronised with corresponding video footage. After synchronisation, acceleration events triggered by the iMG within 3 s of a potential contact phase were visually reviewed. If the event was temporally associated with a contact phase and identified as "on-teeth" by the proximity log, it was confirmed as a positive HAE. Further review of the associated video footage was conducted to determine whether a visible head displacement mechanism could be identified (e.g. direct contact, indirect contact or voluntary movements [13]). As recommended by the Consensus for Head Acceleration Measurement Practices (CHAMP) [16], the raw acceleration waveforms were inspected for signal quality before inclusion in the final dataset.

If a direct or indirect mechanism was observed, further context was recorded as to the manner of contact, for example, head-to-head or body-to-body. A wide range of contextual descriptors was assigned to HAEs during video coding, including player role, tackle height, mechanism of

Table 1 Player match exposure details by grade for iMG participants

Player match exposure details		Grade				Total
		U13 ($n=59$)	U15 ($n=83$)	U19 ($n=58$)	Premier men ($n=59$)	
Players	Forwards	27	49	36	32	**144**
	Backs	27	34	22	27	**110**
	Utility	5	0	0	0	**5**
Matches	Both teams	8	3	3	7	**21**
	Single team	2	14	10	4	**30**
	Total team match events	**18**	**20**	**16**	**18**	**72**
iMG player match details	iMG players per team per match [average (range)]	10 (8–12)	11 (8–13)	8 (6–13)	7 (6–12)	
	Total iMG player matches (n)	**178**	**230**	**133**	**131**	**672**
	Average match time (min)	60.7	77.6	78.0	79.7	
	Average time on teeth (min)/match iMG_forwards	38.3	47.1	42.4	50.0	
	Average time on teeth (min)/match iMG_backs	43.4	43.3	46.2	48.4	
	Total players time (min)	**10,803.7**	**17,840.3**	**10,372.2**	**10,445.7**	**49,461.8**

This table summarizes player and match-level exposure data across four playing grades: U13, U15, U19 and Premier men. Player position breakdown includes forwards, backs and utility players. Match context is presented by team type (single or both teams monitored), along with the number of team match events. For each grade, iMG-specific metrics include the average number of players wearing iMGs per team per match (with range), total iMG player matches, average match duration and average time iMG devices were worn ("on teeth") per match, separately for forwards and backs. Total player time (in minutes) is reported as the cumulative exposure across all matches and grades. Totals are in bold

iMG instrumented mouthguard, *min* minutes contact and situational factors such as whether the player was grounded (prone). These descriptors are illustrated in the example coding window (Fig. 1) and were used consistently throughout the analysis. If an HAE has been confirmed by its temporal association with a contact phase, but the player's head was obscured from all available camera angles, the mechanism was labelled as "unclear". If the player could not be viewed in the footage at all, and the event could not be reliably associated with a contact phase, it was labelled as "off-camera" and excluded from the final analysis.

As shown in Fig. 1, HAEs were contextualised by identifying the match phase in which they occurred (e.g. tackles, ball carries, rucks, mauls, scrums or other phases of play, as defined by World Rugby [22]). Contact event definitions were based on the established literature [3, 17]. Tackles were further framed by direction and height. Tackle height was defined by the first contact made between the tackler and carrier where high tackles were identified as above the sternum, and low tackles were identified as being below the hips (greater trochanter), leaving the space between hips and mid-sternum as mid-range tackles (Fig. 1).

To calculate the propensity of specific match events (e.g. tackles, carries, rucks) resulting in HAEs, we determined both the numerator (number of events associated with HAEs) and denominator (total player involvements in each event type) for each instrumented player (Eq. 1). This process involved aligning the iMG timeline with independently coded match data in Sportscode, following a method similar to that described by Tooby et al. [3]. Only contact events that occurred during verified "on-teeth" periods for the instrumented player were included in the propensity and prevalence calculations. This approach ensured that the propensity analysis was normalised to the individual player's participation in specific match events, allowing for accurate comparisons across event types and players.

$$\text{HAE player prevalence}_{\text{Rucks}} = \frac{\text{Number of ruck involvement causing HAE}}{\text{Total Ruck involvements while instrumented}}. \tag{1}$$

2.3 Data Processing

The raw accelerometer and gyroscope data were retrieved from the Prevent Biometrics server and processed using MATLAB (R2021b; MathWorks Inc., Carlsbad, CA, USA). Custom scripts, Sizewise (School of Physical Education Sport and Exercise Science, University of Otago, Dunedin, New Zealand) were employed to reduce and postprocess the data. Angular acceleration was calculated using a five-point, stencil numerical differentiation method. A 200-Hz,

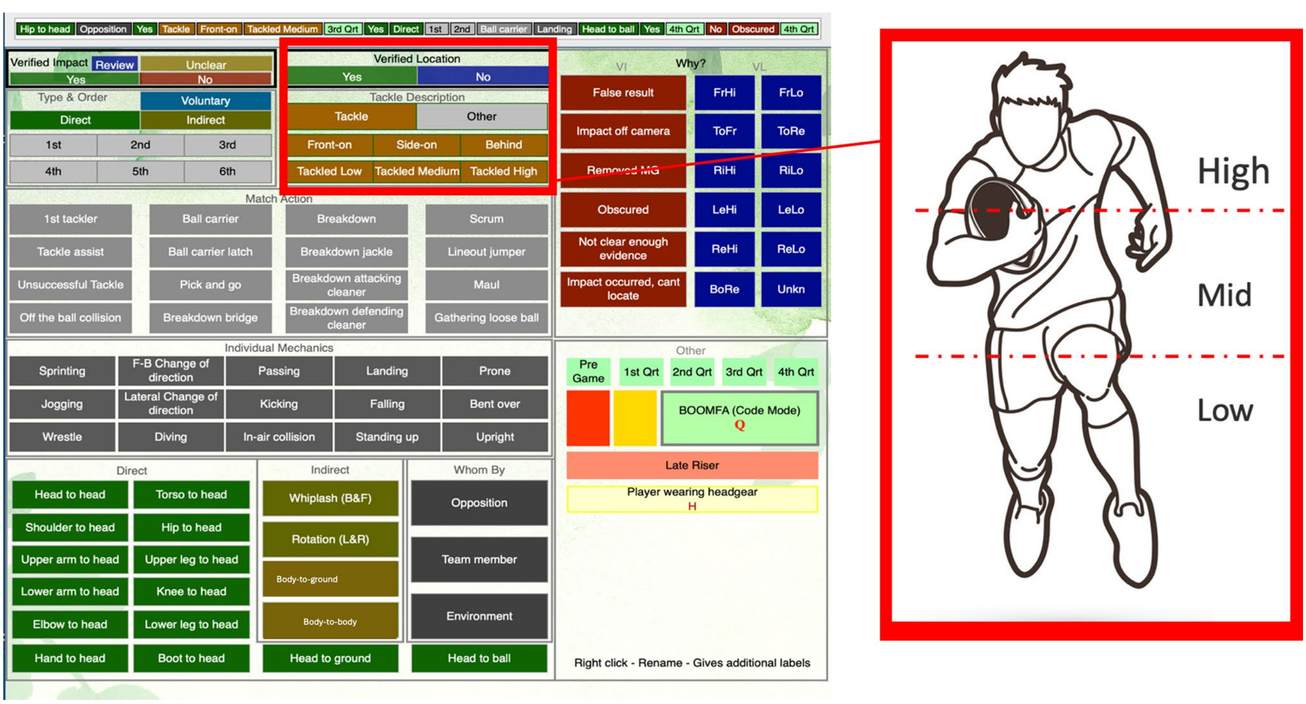


Fig. 1 Coding interface and classification schema used for video review of head acceleration events. Left panel: screenshot of the custom-built coding tool used to classify head acceleration events based on verified impact, contact mechanism, player role, tackle type, individual movement mechanics, impact source (e.g. direct or indirect) and other contextual information (e.g. headgear, game quarter or whether the player rose late). Colour-coded buttons and modular sections support standardised annotation across video reviewers. The right panel is callout related to tackle description: anatomical zones used to classify tackle height: high (shoulder and above), mid (between shoulder and hip) and low (below the hip), as applied in tackle height categorisation during coding

zero-phase, fourth-order Butterworth filter was applied to the data before being transformed into the head centre of mass (COM, Eq. 2) [18]. The head COM location was defined based on population-specific anthropometric data, with coordinates assigned according to either the fifth percentile male (for athletes meeting the fifth percentile criteria for height and weight) or the 50th percentile male (for all other participants) [19–21].

$$a_{\mathrm{h}} = a_{\mathrm{m}} + \alpha \times r_{\mathrm{mh}} + \omega \times \left(\omega \times r_{\mathrm{mh}}\right), \tag{2}$$

where a_{h} is the linear acceleration of the head CoM (g), a_{m} is the linear acceleration measured at the mouthguard (g), α is the angular acceleration vector of the head (rad/s^2), ω is the angular velocity of the head (rad/s) and r_{mh} is the position vector from the mouthguard sensor to the head CoM (m).

Following processing, the peak linear acceleration (PLA) and peak angular acceleration (PAA) were identified as the maximum values of the resultant signal during the recorded window. Further, the rotational velocity change index (RVCI, Eq. 3) was calculated as per Yanaoka et al. [22] Coefficients (c_i) and temporal constraints ($t = 10$ ms) are based on the original reference literature [22]. Rotational velocity change index values are reported in rad/s.

$$\mathrm{RVCI} = \sqrt{c_1\left(\int_{t1}^{t2} \alpha_x \mathrm{d}t\right)^2 + c_2\left(\int_{t1}^{t2} \alpha_y \mathrm{d}t\right)^2 + c_3\left(\int_{t1}^{t2} \alpha_z \mathrm{d}t\right)^2}, \tag{3}$$

where α are the head's angular accelerations about the x, y, z axes (rad/s^2), $\int_{t1}^{t2} \alpha_i \mathrm{d}t$ is the cumulative angular velocity change about axis i during the event window $[t_1, t_2]$, and c_1, c_2, c_3 are weighting coefficients for the three axes, chosen to reflect their relative contributions.

2.4 Statistical Analysis

All statistical analyses were performed using R (v. 4.4.1; R Core Team, 2024) within the RStudio integrated development environment (RStudio Team, 2024). To quantify the frequency and distribution of events, we calculated two key metrics. The first was the incidence rate (IR_{60}), normalised to 60 min of exposure, to standardise event frequency relative to playing time (Eq. 4). The second was the total minutes per acceleration event threshold ($\mathrm{TM}_{\mathrm{HAE}(T)}$), which represents the average time of exposure required to exceed a pre-defined impact event threshold, offering insight into event density and risk over time (Eq. 5). Given the variability in impact occurrences and to address uncertainty in the data, we employed a bootstrap resampling method to improve the robustness of our incidence metrics. This approach involved resampling the impact data five times with replacements to generate stable and reliable estimates of the number of impacts exceeding each defined threshold (T).

$$\mathrm{IR}_{60}(T) = \frac{\mathrm{HAE}(T)}{\text{Total play time}/60} \tag{4}$$

$$\mathrm{TM}_{\boldsymbol{HAE(T)}} = \frac{\text{Total play time (minutes)}}{\mathrm{HAE}(> T)} \tag{5}$$

where HAE(T) is the number of impacts above the PLA threshold (T).

To examine the differences in HAE incidence rates above and below 30 g, a generalised linear mixed-effects model was used. The model included grade and contact event or rugby position as fixed effects. A random intercept for player identification was included to account for repeated measures within players. Because incidence rates were calculated per 60 min of exposure, exposure time was included in the model as a log-transformed offset (log(exposure.time/60). The offset adjusted for differences in total exposure durations across players and events, ensuring appropriate normalisation of HAE incidence rates.

To further estimate the likelihood (propensity) of an HAE occurring relative to a match contact exposure (e.g. total tackles, ball carries, rucks), negative binomial regression models were employed using the glmmTMB package to account for overdispersion in the count data. These models produced incidence rate ratios (IRRs) to compare event rates across conditions. For categorical predictors, where the occurrence of an HAE could be treated as a binary outcome (yes/no), such as tackle direction (front-on, side-on, behind) and ball carrier posture (upright, bent over, other), binomial logistic regression models were used. These models provided odds ratios (ORs) as estimates of relative risk.

A quantile mixed-effects model was used to examine the relationship between contact characteristics and head kinematic outcomes. This modelling approach, which estimates relationships at specific quantiles in the distribution rather than assuming a normally distributed outcome, was chosen because the primary head kinematic variables PLA, PAA and the RVCI were not normally distributed. Quantile modelling is robust to skewed data and provides a more accurate estimate of the median (tau = 0.5) effect of predictors on these outcomes [23, 24].

Three quantile mixed-effects models were conducted. Each model included fixed effects for tackle height (with "tackled low" as the reference category), player role ("1st tackler" vs "ball carrier"), grade level (U13, U15, U19, Premier men) and their interactions. A random intercept for athlete name was included to account for individual variability. To account for the imbalance in the number of observations across player roles, tackle heights, and grades, we applied inverse frequency weighting to the statistical models [25].

These weights were calculated as the inverse of the number of unique athletes in each group, scaled by the maximum group size. This approach ensures that smaller groups contribute proportionally more to the analysis, reducing the potential bias introduced by uneven sample sizes.

3 Results

A total of 8593 sensor acceleration events were identified as potential HAEs by temporal occurrence to a match contact event. Of these, 7358 were able to be visually verified. The breakdown of mechanisms by grade is presented in Table 2.

3.1 Match Exposure by Playing Position (Time-Weighted Generalised Linear Mixed Models)

An overview of player exposure by grade and playing position is reported in Table 1. Instrumented mouthguard data were collected across 72 team matches, totalling 49,461 player-minutes of exposure. Figure 2a illustrates the HAE event density $TM_{HAE(T)}$ (y-axis) across increasing PLA (g) thresholds (x-axis), stratified by player position and grade. When $T > 30$ g, forwards generally exhibit shorter time intervals between impacts than backs, suggesting higher HAE frequency (except for the U13s). Statistically significant differences in $TM_{HAE(T)}$ were observed between Premier men forwards and backs ($p = 0.02$), and between U19 and U13 forwards ($p = 0.04$). Figure 2b shows that Premier locks and U19 outside backs had the highest IR_{60} for impacts below 30 g, compared with their positional peers.

3.2 HAE Incidence Rate by Match Event

Figures 3a and b represent IR_{60} heatmaps by match event, for all HAEs (a) and HAEs > 30 g (b), respectively. Tackle and ruck phases exhibited the highest HAE incidence rates across all playing grades, with tackle events showing a statistically significant elevation in incidence compared with all other match events ($p < 0.05$). Lineouts and non-tackle-related ball carries followed as the next highest HAE incidence phases of the game. Colour intensity reflects the mean incidence rate per 60 min, with higher values shaded in red.

3.3 Tackles

3.3.1 Tackle Roles and HAE Propensity

Across all matches, 10,677 tackles and 6036 ball carries were recorded. Head acceleration events were more prevalent for tacklers overall, with a range from 23 to 37% (Table 3). In

Table 2 Median head kinematic values by mechanism of head acceleration event across playing grades

Grade	Mechanisms	Count	Median PLA (IQR)	Median PAA (IQR)	Median RVCI (IQR)
Premier men	Voluntary	84	7.37 (3.44)	464.21 (552.03)	5.12 (4.39)
	Direct	1161	15.03 (10.58)	1089.05 (965.51)	8.81 (5.94)
	Indirect	175	9.94 (5.79)	778.26 (718.09)	7.26 (5.41)
	Unclear	323	12.37 (10.48)	1013.68 (1368.91)	7.65 (7.3)
U19 boys	Voluntary	85	8.5 (2.7)	714.32 (450.05)	7.44 (4.78)
	Direct	1112	15.33 (12.1)	1153.97 (1109.15)	9.11 (6.22)
	Indirect	158	10.92 (7.34)	869.7 (859.74)	7.78 (6.06)
	Unclear	448	13.04 (10.31)	1124.27 (1344.42)	8.2 (7.01)
U15 boys	Voluntary	121	9.02 (6.47)	786.41 (755.99)	7.61 (4.47)
	Direct	1477	14.91 (11.07)	1201.13 (1117.1)	9.22 (6.43)
	Indirect	223	10.86 (7.92)	1089.8 (1368.43)	9.11 (7.19)
	Unclear	629	10.75 (10.28)	1518.05 (1393.97)	8.81 (6.31)
U13	Voluntary	12	10.45 (5.53)	1284.29 (901.47)	9.75 (6.03)
	Direct	687	13.5 (11.12)	1144.58 (1106.34)	8.66 (6.26)
	Indirect	194	13.68 (10.74)	1675.95 (1626.69)	10.7 (7.32)
	Unclear	469	11.61 (7.64)	1523.18 (1035.43)	8.9 (6.04)

This table presents the count and median values (with IQR) for PLA (in g), RVCI (in rad/s) and PAA (in rad/s^2) across four playing grades (U13, U15, U19, Premier men), stratified by head acceleration event mechanism type. Mechanisms are categorised as voluntary (e.g. purposeful heading), direct (direct contact to the head), indirect (body-to-body transmission of force) and unclear (mechanisms unable to be definitively classified). The table illustrates how both the magnitude and variability of kinematic responses differ by age group and mechanism type

IQR interquartile range, *PAA* peak angular acceleration, *PLA* peak linear acceleration, *RVCI* rotational velocity change index

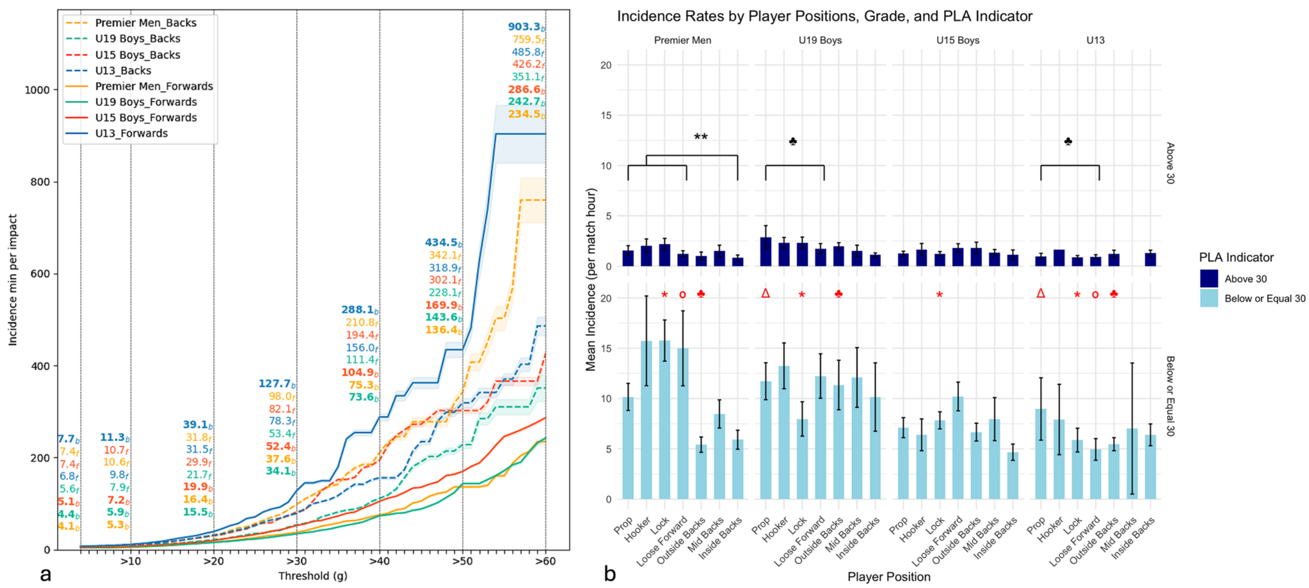


Fig. 2 **a** Head acceleration event (HAE) event density $TM_{HAE(T)}$ (y-axis) across increasing peak linear acceleration (PLA, g) thresholds (x-axis). Total minutes per of exposure by HAE threshold by playing position, forwards represented by solid lines, while backs are represented by broken lines; **b** bar plot representing the incidence per player hour for HAE < 30 g (bottom panel) as light blue bars and HAE > 30 g (top panel) as dark blue bars. Red symbols indicate between-grade differences for HAE < 30 g ($p < 0.05$). Black bars and symbols represent significant HAE > 30 g position comparisons ($p < 0.05$)

fact, the HAE outcome was significantly more likely during tackles than ball carries across all grades (IRRs: 1.24, 1.23, 1.32, 1.22, Premier to U13 respectively; all $p < 0.0001$). Among U19 players, the incidence of HAEs > 30 g was higher for ball carriers (3.22 per 100 ball carries) than for first tacklers (2.44 per 100 tackle events), IRR = 1.04, 95% confidence interval (CI) 1.01–1.06, $p < 0.001$.

3.3.2 Tackle Behaviour and HAE Mechanisms

Relative to front-on tackles, side-on and tackles from behind were significantly less likely to cause a high tackle outcome ($OR_{side\text{-}on} = 0.77$, 95% CI 0.61–0.97, $p = 0.021$; $OR_{behind} = 0.19$, 95% CI 0.09–0.40, $p < 0.001$). Whereas, an upright tackler more than tripled the odds of a high tackle event (OR = 3.29, 95% CI 2.78–3.80, $p < 0.001$) and further increased the likelihood of a head-to-head contact (OR = 1.79, 95% CI 0.30–3.28, $p = 0.019$).

Figures 4a and b illustrate the distribution of high-magnitude head acceleration mechanisms observed within tackle phases across player roles and age grades, highlighting both the frequency (per 100 tackles) and severity [(a) median PLA, (b) median RVCI]. Examining these figures, it can be seen that hip-to-head and shoulder-to-head events are the most severe head acceleration (PLA > 25 g) mechanisms in Premier and U19 tacklers, occurring with the highest propensity (0.5–3.1 events per 100 tackles). These mechanisms match closely with events associated with high rotational velocity (Fig. 4b, RVCI > 14 rads/s), with the addition of lower frequency mechanisms such as knee-to-head and elbow-to-head. Notably, mechanisms associated with the highest severity and frequency in the tackler were not linked to high-tackle events. Considering the outcomes for the Premier and U19 ball carriers, it is clear that there are fewer severe events and again that shoulder-to-head and high tackle outcomes are predominant.

Examining the younger tacklers, U13 and U15, a higher frequency of secondary mechanisms is seen, such as hip-to-head in high tackle or elbow-to-head low tackles (Fig. 4a). There is also a higher frequency of head-to-ground mechanisms in both tackler and ball carriers, particularly in the U13s. In Fig. 4b, there is a marked difference in the frequency and variety of mechanisms triggering severe rotational outcomes for the younger players. The younger tackler appears to experience very similar outcomes to their senior counterparts, with shoulder-to-head and hip-to-head dominating in both severity and propensity. Notably, the most severe outcomes in the U15 tacklers are the head-to-head contacts (~ 0.15/100 tackles). However, in the younger ball carriers, body-to-ground contact occurred with similar frequency for all tackle heights; moreover, the severity of these events increased with tackle height. In addition, head-to-ground was the most frequent mechanism in U13 carriers and the second most frequent in U15s, highlighting the prominence of these secondary events in the younger cohorts. These mechanism-specific patterns dovetail with

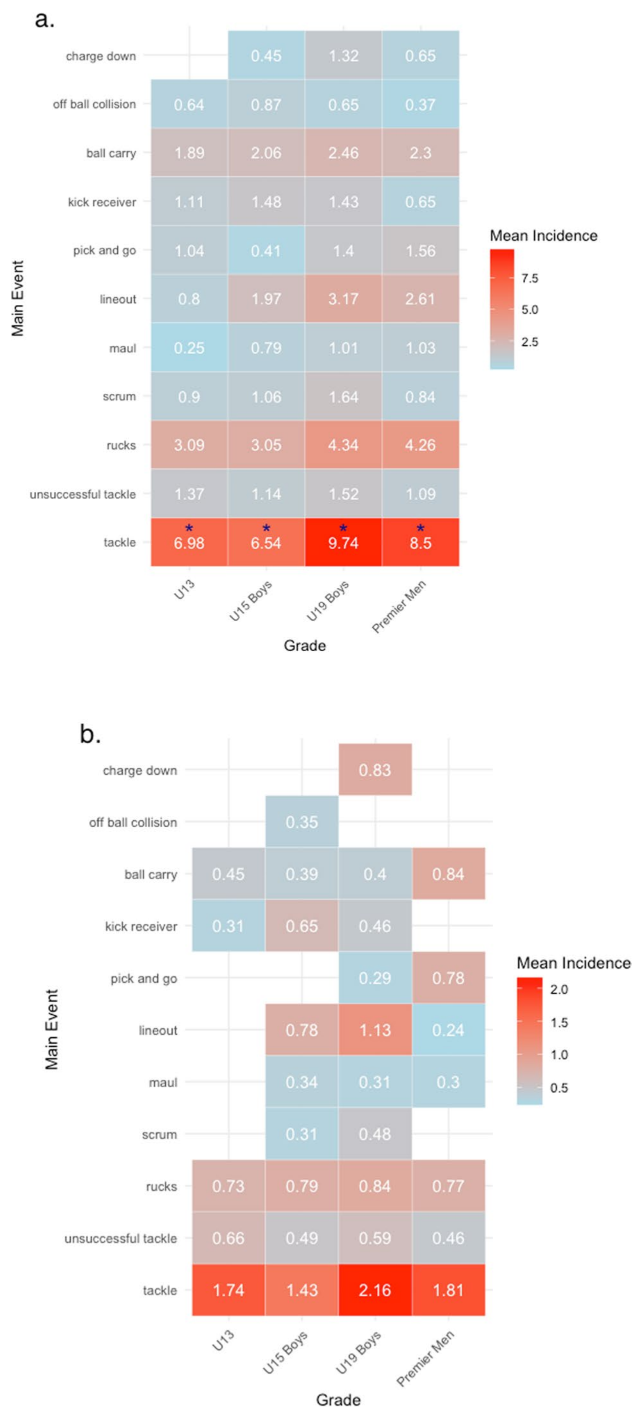


Fig. 3 Heatmaps of mean head acceleration event (HAE) incidence per player hour (IR_{60}) by contact type across playing grades: **a** IR_{60} for all HAE; **b** IR_{60} for HAE > 30 g. Tackle events were statistically higher than all other contact events ($p < 0.05$), indicated by an asterisk (*). Colour intensity reflects the mean IR_{60}, with higher values shaded in red

the age effects in Table 3, where U13 and U15 players exhibited significantly greater rotational motion when grounded (Table 3).

3.3.3 Effects of Tackle Height on Head Kinematics (Quantile Mixed Models)

Tackle height significantly influenced head acceleration, with ball carriers exhibiting a significant stepwise increases in acceleration with tackle height (Fig. 5). For instance, compared with low tackles, mid-range tackles increased the median PLA by + 3.11 g and PAA by + 272 rad/s^2 (95% CI 0.9–6.23, $p = 0.01$; 95% CI 0.8–544.1, $p = 0.049$, respectively) and high tackles further elevate PLA by + 4.16 g, PAA by + 443 rad/s^2 as well as RVCI by + 1.87 rad/s (95% CI 0.5–6.1, $p = 0.02$; 95% CI 1.6–717.7, $p = 0.002$; 95% CI 0.06–3.7, $p = 0.04$, respectively).

Interactions between player role and tackle height suggest that tacklers experienced greater head acceleration than ball carriers in low tackles (Fig. 5, PLA: + 4.9 g, 95% CI 1.6–8.2, $p = 0.004$; RVCI: + 1.6 rad/s, 95% CI 0.3–3.1, $p = 0.03$). This pattern was reversed in the U13 grade, where ball carriers exhibited significantly greater rotational loading than tacklers during low tackles (PAA: + 694.84 rad/s2, 95% CI 158.9–1230.7, $p = 0.012$; RVCI: + 5.01 rad/s, 95% CI 1.3–8.6, $p = 0.008$).

3.4 Ruck Roles and Propensity

A total of 12,900 player ruck involvements were identified, including 9779 attacking rucks and 3121 defending rucks. Across all competition levels, defensive ruck involvements were associated with significantly higher HAE propensity than attacking rucks. The HAE rate ratios for defensive versus attacking rucks ranged from 1.28 to 2.27, with all 95% CIs excluding 1, confirming statistically significant differences ($p < 0.05$ for all grades). The highest relative risk was observed in U19 boys (IRR = 2.27, 95% CI 1.68–3.07, $p < 0.0001$). The lowest relative risk was seen in U15 boys (IRR = 1.28, 95% CI 1.03–1.60, $p = 0.025$). Among ruck roles, the attacking cleaner showed the highest HAE prevalence in attacking rucks, while the jackler was most affected in defensive rucks (Table 4). The U19 jackler had the highest incidence rate for HAE > 30 g, with a rate of 1 such event in every 46 defensive rucks (Table 4).

4 Discussion

This study provides a comprehensive analysis of head acceleration exposure in community rugby, using iMG data collected from 259 players across 72 matches spanning four male age grades. Our findings reveal distinct age- and role-specific patterns in both the frequency and severity of HAEs. Notably, younger players were more susceptible to rotational loading, particularly during low tackles, while older players, especially U19 forwards,

Table 3 Prevalence, incidence and HAE kinematics by tackle role and playing grade

Roles	Grade	Count	HAE prevalence (%)	HAE > 30 g incidence (per 100 tackles)	PLA (IQR)	PAA (IQR)	RVCI (IQR)	p values
1st tackler	Premier men	337	31.9	1.37	13.8 (10.9)	1.091 (1.062)	9.3 (6.2)	
	U19 boys	364	37.8	2.44	14.4 (12.2)	1.147 (1.225)	9.5 (7)	
	U15 boys	541	37.0	2.44	14.6 (12.9)	1.245 (1.183)	9.9 (7.2)	
	U13	300	23.1	1.15	12.4 (8.8)	1.119 (1.017)	8.7 (5.8)[a]	
Ball carrier	Premier men	274	14.0	2.08	14.6 (11)	1.095 (0.941)	9.6 (6.7)	
	U19 boys	272	19.9	3.22	14.4 (12.1)	1.13 (0.902)	9.4 (6)	
	U15 boys	301	22.0	1.79	14.3 (10.1)	1.152 (1.043)	9.4 (6.5)	
	U13	91	11.8	0.82	14.8 (12.2)	1.458 (1.703)	11.2 (9)[a]	0.01
Ball carrier latch	Premier men	22	0.84	0.08	14.7 (6.3)	1.146 (0.802)	8.7 (2.5)	
	U19 boys	20	0.85	0.04	15.6 (7.2)	0.95 (0.913)	7.4 (3.6)	
	U15 boys	4	0.12	0	13.7 (5.6)	0.954 (0.596)	5.7 (1.1)	
	U13	4	0.17	0.04	21.5 (15.3)	1.756 (2.139)	12.8 (7.3)	
Grounded player	Premier men	48	2.80	0.23	12.7 (6.8)	0.991 (0.626)	7.8 (5.1)	U15: 0.018, U13: 0.001, 0.022
	U19 boys	49	3.64	0.48	13.5 (10.4)	1.066 (0.955)	8.2 (5.4)	U13: 0.015
	U15 boys	52	3.02	0.41	14.8 (16)	1.248 (0.928)[b]	8.8 (6.8)	
	U13	21	1.68	0.29	18.8 (16.5)	1.795 (1.852)[b,c]	9.8 (6.4)[b]	
Tackle assist	Premier men	86	3.29	0.31	15.2 (8)	1.042 (0.954)	8.3 (5.6)	
	U19 boys	75	3.18	0.37	15.4 (11.9)	1.188 (1.172)	9.3 (6.9)	
	U15 boys	98	2.97	0.27	15.1 (12.3)	1.251 (1.255)	9.2 (6.2)	
	U13	30	1.25	0.08	16 (7.1)	1.381 (0.934)	9.7 (5.3)	

The table presents the number of HAE observations (count), HAE prevalence (% of events resulting in HAEs) and incidence of high-magnitude HAEs (defined as HAE > 30 g, expressed as the ratio of such events to total tackle events) across player roles and playing grades. Kinematic outcomes include median PLA (in g), PAA (in rad/s^2) and RVCI (in rad/s), all reported with IQRs. Significant p-values indicate between-grade differences within each role. Superscript letters a, b, c denote significant pairwise differences. Superscripts denote significant pairwise differences at $p < 0.05$

HAE head acceleration event, *IQR* interquartile range, *PAA* peak angular acceleration, *PLA* peak linear acceleration, *RVCI* rotational velocity change index

[a]Significantly different from first tackler

[b]Significantly different from Premier men

[c]Significantly different from U19

faced a higher cumulative burden of high-magnitude HAEs. With respect to tackle height, both high and low tackle heights were linked to increased head acceleration, albeit through different mechanisms for ball carriers and tacklers. The results reaffirm the critical influence of tackle height and contact mechanism on head kinematics, highlighting the need for tailored strategies to reduce the head impact risk across developmental stages.

4.1 Match Exposure by Playing Position

Our results demonstrate that forwards experience a higher frequency of HAEs compared with backs, and moreover, that the prop, hooker and lock positions experience the greatest proportion of HAE > 30 g, likely reflecting their involvement in high-contact scenarios such as scrums and rucks. These findings align with Allan et al., who reported higher HAE frequencies in forwards than backs at the elite English level [7]. In contrast, Roe et al. found no positional differences in Southern Hemisphere rugby, suggesting that regional playing styles and tactics may shape positional exposure [7]. These findings reinforce the need to examine individual playing positions within forward and back groups, such as props or inside backs, to identify nuanced risks and refine position-specific training and injury prevention strategies.

The data show a clear age-related increase in exposure to high-magnitude HAEs (> 30 g) among community rugby forwards. U13 forwards experience the lowest frequency, one HAE > 30 g every 127 min of play, or roughly once every three to four matches based on their average exposure of 38 min per game. In contrast, U19 forwards sustain one HAE > 30 g every 34 min, resulting in at least one such

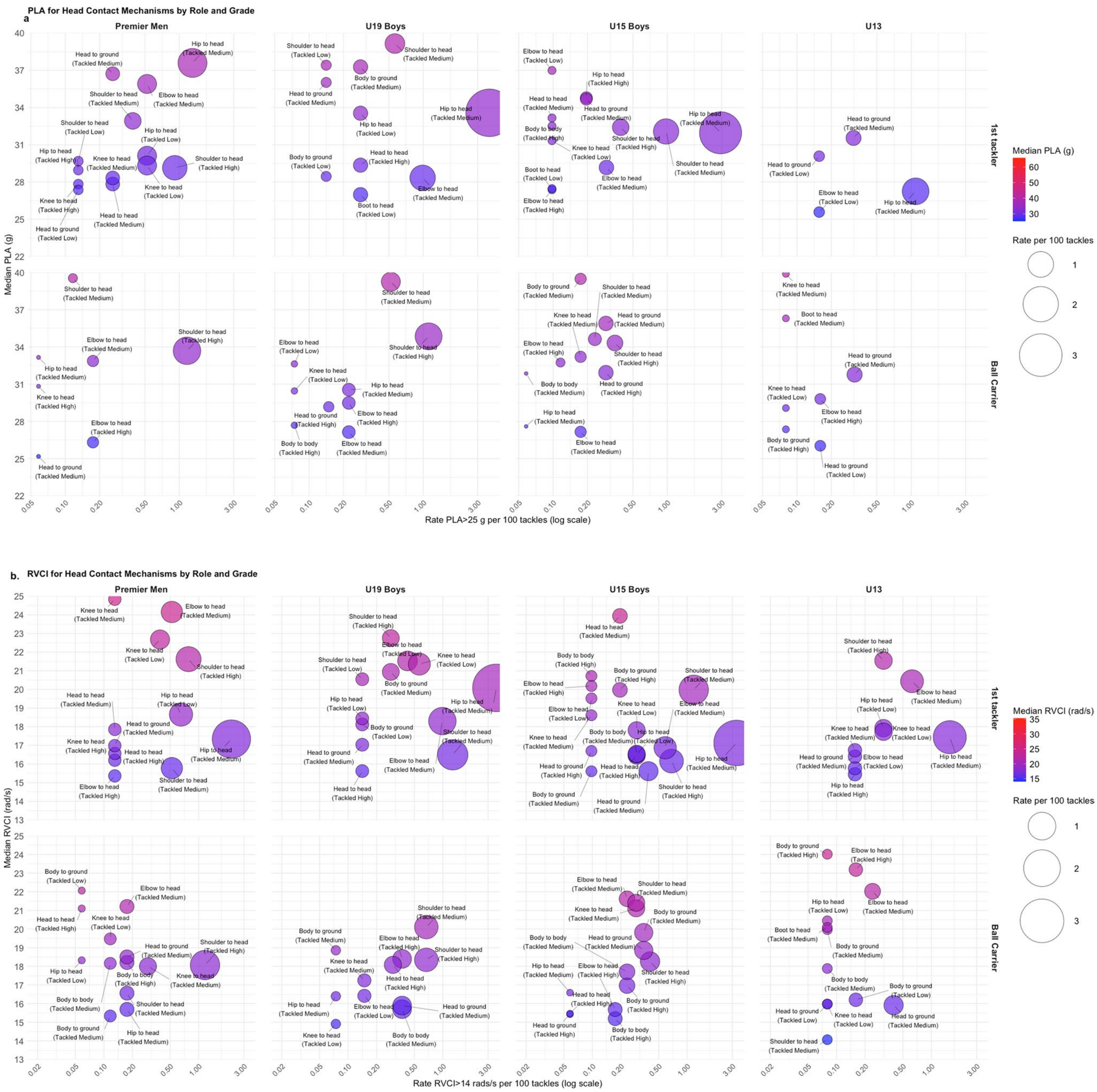


Fig. 4 Bubble plots displaying the severity and frequency of head acceleration events (HAEs) by contact mechanism, stratified by playing grade and tackle role. Panel **a** presents the median peak linear acceleration (PLA) for HAEs exceeding 25 g, while panel **b** shows the median rotational velocity change index (RVCI) for events exceeding 14 rad/s. Each bubble represents a distinct head contact mechanism. The *x*-axis shows the log-scaled frequency of the mechanism per 100 tackle events, and the *y*-axis indicates the median magnitude of the HAE (PLA or RVCI). Bubble size reflects the frequency of the mechanism, with larger bubbles indicating more common occurrences. Bubble colour denotes the severity of the event, with deeper red tones representing higher magnitude impacts. Contact mechanisms are labelled by contact type and tackle height classification (e.g. "Shoulder to head [Tackled High]") and are separated by player role, with tackler data shown in the top row and ball carrier data in the bottom row

event per match, similar to senior adults. This escalating exposure across age grades suggests a growing cumulative load on school-age players as they advance, particularly among forwards. It also raises important concerns for younger athletes who "play up" into older age groups, where thaeymay be exposed to greater biomechanical demands before reaching sufficient physical or neurological maturity. Research has linked cumulative head impacts with adverse long-term neurological outcomes [26], and studies in youth rugby indicate that playing against older, more physically developed opponents increases injury risk [27, 28]. These findings underscore the need for careful consideration of age-grade progression policies and targeted exposure management strategies to protect younger players who may be at elevated risk.

4.2 Contact Phases

Across all playing grades, tackles and rucks stood out as having the highest IR_{60} for both HAE > 30 g and HAE < 30 g. These findings are consistent with recent iMG research in the elite space [7, 8, 29]. Tackles are consistently identified as the most common cause of head injuries and concussions in rugby union. Quarrie and Hopkins reported that tackles were the most dangerous facet of play, with the head being the most common site of injury [30]. Tierney et al. found that only 10 out of 52 significant direct head impacts occurred during rucks, whereas 31 out of 52 significant direct head impacts occurred during tackles [31]. While rucks also pose a risk, they appear to be less dangerous than tackles overall.

4.2.1 Tackles

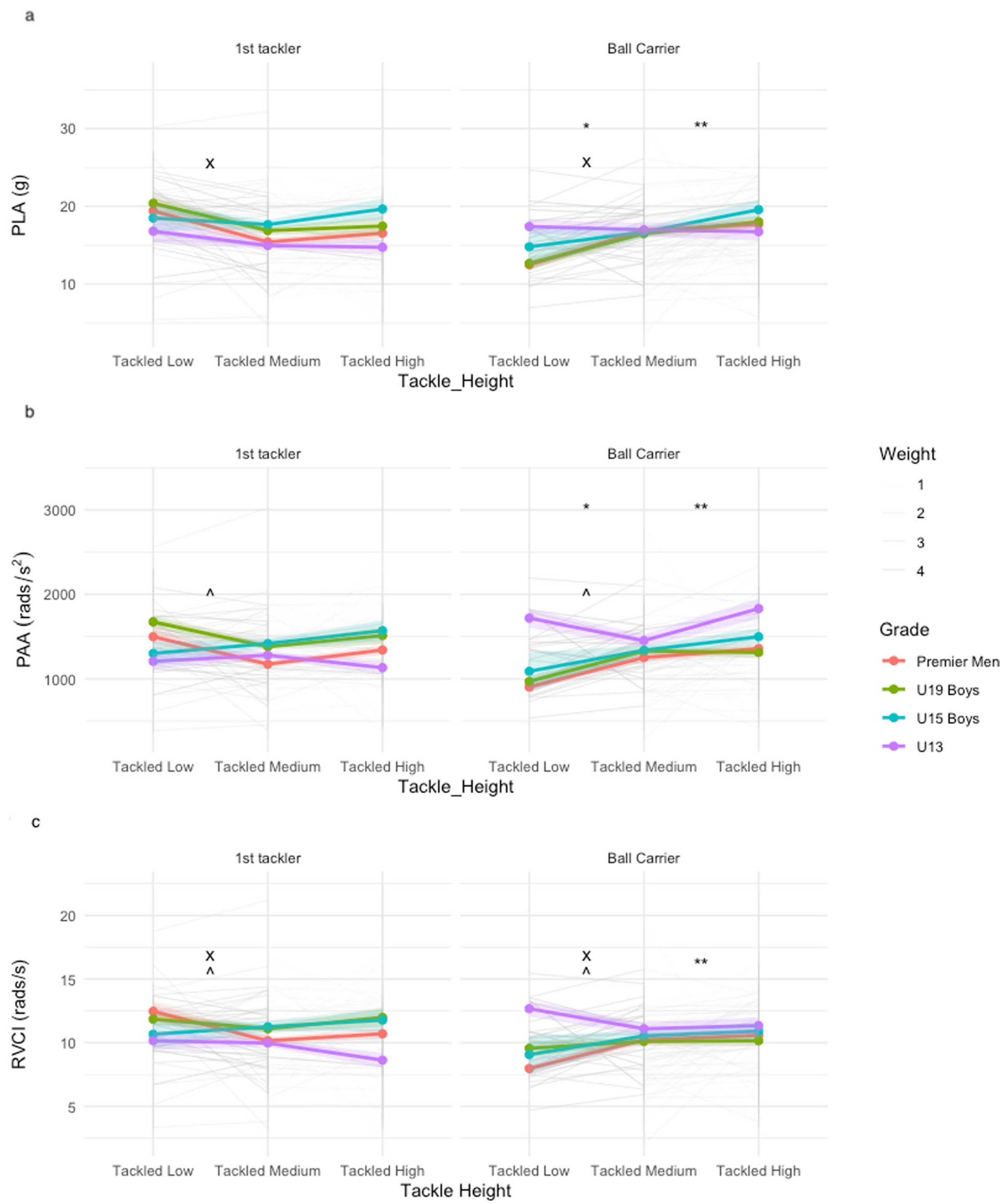
The findings of this study reinforce the growing body of evidence that tacklers face the greatest risk of HAEs during tackle phases, a trend echoed by Tucker et al. and Cross et al., who identified tacklers as the most common recipients of head injury assessments, with up to 78% of head injury assessments occurring in tacklers [32, 33]. However, our results also highlight that tackle direction, body posture and head placement meaningfully modulate this risk. Specifically, front-on tackles were significantly more likely to result in high tackle events than side-on or behind tackles, supporting prior evidence that front-on, active shoulder and high-speed tackles increase the likelihood of injury for both ball carriers and tacklers [32, 34–36]. Moreover, we found that upright tacklers were 27 times more likely to produce high tackles, and six times more likely to involve head-to-head contact, compared with tacklers adopting a bent-at-the-waist posture. While these findings strongly support coaching strategies aimed at encouraging bent postures, our data also show that severe HAEs often arose from poor head placement, particularly when the tackler's head contacted the ball carrier's hip or shoulder. This suggests that attention to posture alone is insufficient and provides support for recommendations like those made by Davidow et al., that safe tackle execution must also incorporate technical elements such as "head up" positioning, correct head placement to the side of the ball carrier, and controlled entry using shortening steps [37].

This emphasis on tackle posture is further supported by our finding that tackle height is closely linked to head acceleration outcomes, particularly for the ball carrier. From U15 to Premier men, higher tackle heights were associated with stepwise increases in PAA, PLA and RVCI for ball carriers. This reinforces the notion that lowering tackle height could reduce concussion risk in ball carriers, providing further support for the recent regulatory efforts by World Rugby and NZ Rugby relating to this effect [38, 39]. However, our findings also reveal a critical trade-off: while ball carriers benefit from lower tackles, tacklers experience significantly greater head acceleration when executing low tackles, especially those made below hip height. These dual risks underscore the complexity of tackle safety regulations.

Consistent with our observations, Woodward et al. found that both high and low tackles carried a greater risk of HAE > 30 g compared with mid-range tackles in elite rugby [29], while Quarrie and Hopkins similarly identified high tackles as most hazardous for ball carriers and low tackles as particularly risky for tacklers [30]. Together, these findings suggest a need for balance between coaching and regulatory strategies. Coaches and regulators should not rely wholly on regulatory changes to improve the injury trajectory, rather interventions should emphasise and promote the adoption of mid-range tackle height executed with a bent-at-the-waist posture and appropriate head placement. This combined approach may offer the best opportunity to reduce head acceleration exposure for both players involved in the tackle and enhance safety across all levels of the game.

In our study, the youngest age groups, specifically U13 and to some extent U15 athletes, exhibited distinct kinematic responses during contact situations compared with their older counterparts. These younger players demonstrated greater susceptibility to rotational loading across most contact scenarios. Notably, within low tackle events, the youngest (i.e. U13) ball carriers sustained significantly higher rotational acceleration than tacklers, a trend that was contrary to observations in older age groups. This pattern could reflect a limited capacity to resist head motion when the trunk decelerates rapidly that requires a combination of muscle strength and timing of muscle activation to stabilise the head [40, 41].

Anticipatory postural adjustments are a central nervous mechanism for controlling body motion when a perturbation is expected, a reflex that develops in childhood and continues to mature throughout adolescence [41, 42]. Importantly, its refinement is shaped not only by biological maturation, but also by experience and repeated exposure to task-specific perturbations [43, 44]. In the context of rugby, this suggests that playing experience and tackle-specific training may accelerate the development of anticipatory head control. Our



findings may therefore reflect both age-related immaturity and a limited exposure to tackle contexts in younger players [45, 46].

A particularly striking observation was the predominance of head-to-ground and body-to-ground mechanisms in the younger cohorts. These mechanisms were not only more frequent but were also associated with elevated rotational

◂**Fig. 5** Quantile mixed-effects estimates of head-kinematic magnitudes (median peak linear acceleration [PLA], peak angular acceleration [PAA] and rotational velocity change index [RVCI]) across tackle-height categories (low, medium, high) for first tacklers (left panels) and ball carriers (right panels) in Premier men, U19 boys, U15 boys and U13. ** indicates a statistically significant increase from low to high tackles ($p < 0.05$). * indicates a significant increase from low to medium tackles ($p < 0.05$). X denotes a significant player-role × low-tackle interaction across all grades. ^ denotes a significant player-role × low-tackle interaction specifically in the U13 grade. Grey lines represent the distribution of model predictions, incorporating random intercepts per player and inverse frequency weighting across player × grade groups. These were included to visualise the individual-level model variation and the contribution of each observation to the trend

loading, especially among ball carriers. Such patterns suggest a potential breakdown in tackle technique, where younger players are more prone to collapsing or "pancaking" into the ground, leading to secondary whiplash-like impacts. These findings align with those reported in the school-age female cohort by Bussey et al. and reinforce the importance of neuromuscular development and targeted skill acquisition in mitigating rotational head loading during contact [12].

Together these results underscore the need to recognise and address age-specific vulnerabilities in head control and contact response. Interventions aimed at improving tackle technique, neck strength and anticipatory control should be prioritised in youth and school age rugby to reduce injury risk and support safer long-term player development.

4.2.2 Rucks

Rugby rucks are among the most frequent contact events in Rugby Union, occurring approximately every 41 s during match play [47]. Rucks emerged in the current study as a notable contributor to HAEs, with potential variation in risk between attacking and defensive roles. While defensive rucks demonstrated a slightly higher propensity for HAE > 30 g, their relative frequency in match play is lower than attacking rucks. Prior analyses of elite-level matches estimate that defensive contesting roles (e.g. jacklers) occur in approximately 10–20% of ruck situations, with attacking cleaners comprising the majority of ruck involvements [30, 48].

Despite their lower frequency, defensive ruck roles remain a meaningful contributor to overall HAE burden owing to their elevated per-event risk. One study reported that in 75% of ruck-related concussions, the injured player was acting defensively, either as the jackler or as a support player entering the contest [49]. In the current study, Premier-level defensive cleaners experienced a higher frequency of HAE > 30-g events (0.91 per 100 rucks) than their attacking counterparts (0.62 per 100 rucks). More notably, U19 and U15 jacklers experienced the highest frequencies across all ruck roles and grades, 2.17 and 1.02 respectively, highlighting a substantial risk for players in this position. These findings are consistent with the existing literature indicating that defensive ruck actions, particularly jackling, expose players to elevated biomechanical demands, driven by low body positioning, acute entry angles and high-intensity collisions with opposition players [8, 50]. They also corroborate our previous research, which identified U19 forwards as especially susceptible to high-magnitude HAE within community rugby contexts [13].

To more effectively understand and mitigate these risks, future research should investigate the specific biomechanical and situational determinants of head acceleration within ruck engagements. Variables such as player posture, entry trajectory and force profiles warrant closer examination. In particular, school-age rugby may benefit from regulatory changes that limit the number of players permitted to enter the ruck, thereby reducing crowding, secondary impacts and uncontrolled collisions that elevate the risk of HAEs.

4.3 Limitations

This research faced several limitations. The quality of iMG fitting might have varied across different grades. Although smaller iMGs with reduced material volume were provided to younger athletes, achieving an optimal fit remained challenging because of factors such as narrower dental arches, triangular bites, and the presence of missing or misaligned teeth common among younger players. An inadequate fit could result in an increased number of false and voluntary iMG activations. To address the fit issues, we employed qualified dentists for the fitting process and monitored iMG-tooth displacement using a proximity sensor, which allowed us to assign a quality-of-fit score. Further, only sensor acceleration events that were deemed as being "on-teeth" during the contact event were included in the final analysis.

While inverse frequency weighting was applied to reduce bias from unequal group sizes, we acknowledge that differences in outcome frequency across grades reflect genuine variations in gameplay exposure. Therefore, weighting ensures a proportional contribution to model estimation but does not adjust for structural exposure differences between age groups. This should be considered when interpreting cross-grade comparisons.

Additionally, we did not track teams throughout the entire season because of time limitations and the extensive number of athletes involved. Instead, we monitored each team for a minimum of four matches. Although the observation period was standardised, it was staggered by grade throughout the season. Consequently, some grades may have been observed at the season's start, while others were monitored toward the end. Seasonal variations could influence the intensity of game play; however, this was not explicitly modelled in

Table 4 Head kinematics, prevalence and incidence of HAEs by ruck role and playing grade

Ruck description	Player role	Grade	Event count	Prevalence (%)	Median PLA (IQR) g	Median PAA (IQR) krad/s^2	Median RVCI (IQR) rad/s	HAE > 30 g			
								Rate per 100 events	Median PLA	Median RVCI	Mechanism highest % > 30 g
Attacking rucks	Attacking cleaner	Premier men	162	5.93	16.4 (10.0)	1.026 (0.841)	8.4 (5.1)	0.62	41.4	13.3	Head to head
		U19 boys	106	4.97	15.9 (12.6)	1.120 (1.162)	9.1 (5.2)	0.61	34.4	9.6	Shoulder to head
		U15 boys	170	6.16	14.9 (9.6)	1.151 (0.908)	8.8 (5.8)	0.72	43.2	20.8	Torso to head
		U13	52	2.42	14.4 (8.8)	1.123 (1.147)	8.5 (5.5)	0.19	51.5	28.7	Shoulder to head
	Sealer	Premier men	21	0.76	18.9 (12.8)	1.619 (1.112)	9.1 (6.3)	0.11	56.0	20.5	Lower arm to head
		U19 boys	58	2.71	17.8 (12.8)	1.192 (1.520)	8.0 (4.3)	0.33	43.8	29.4	Shoulder to head
		U15 boys	33	1.19	17.8 (10.1)	1.405 (1.028)	9.8 (7.1)	0.22	35.3	15.8	Shoulder to head
		U13	12	0.56	15.8 (9.3)	1.238 (1.329)	6.9 (3.8)	0.09	58.8	33.3	Head to head
Defending rucks	Defensive cleaner	Premier men	30	3.89	14.4 (17.0)	1.362 (1.104)	8.8 (5.2)	0.91	78.8	30.3	Hip to head
		U19 boys	14	2.11	13.6 (5.4)	1.038 (0.804)	7.6 (6.4)	0.15	41.2	22.6	Hip to head
		U15 boys	21	2.14	16.6 (10.9)	1.133 (0.717)	7.9 (3.5)	0.1	20.4	12.6	Lower arm to head
		U13	7	0.99	10.0 (9.9)	1.387 (1.214)	7.7 (4.4)	0	25.8	8.9	Shoulder to head[a]
	Jackler	Premier men	68	8.69	14.4 (9.6)	1.022 (0.784)	8.4 (4.1)	0.78	41.2	18.9	Shoulder to head
		U19 boys	102	15.11	14.6 (9.3)	1.012 (0.962)	8.0 (4.2)	2.17	43.6	13.4	Head to head
		U15 boys	81	8.26	16.9 (11.5)	1.236 (1.113)	8.7 (5.5)	1.02	53.8	23.1	Shoulder to head
		U13	36	5.10	14.0 (7.0)	1.163 (0.630)	7.7 (3.9)	0.14	34.1	11.8	Shoulder to head
Other ruck involvements	Grounded player	Premier men	12	0.34	18.2 (15.5)	1.481 (1.561)	9.3 (9.7)	0.09	43.2	30.0	Boot to head
		U19 boys	12	0.43	17.1 (12.8)	1.269 (0.897)	7.5 (7.4)	0.11	45.9	21.0	Knee to head
		U15 boys	13	0.35	15.8 (6.5)	1.210 (0.502)	9.9 (4.6)	0	21.0	13.2	Lower leg to head[a]
		U13	8	0.29	15.8 (5.0)	1.268 (0.573)	8.0 (5.5)	0	15.8	8.1	Lower leg to head[a]
	Other ruck player	Premier men	59	1.68	10.8 (7.5)	0.785 (0.482)	6.4 (2.6)	0.06	30.9	14.9	Knee to head
		U19 boys	82	2.94	12.4 (12.6)	1.136 (1.265)	8.3 (6.0)	0.32	52.8	22.3	Lower arm to head
		U15 boys	106	2.87	11.0 (9.9)	1.258 (1.339)	8.4 (6.3)	0.3	70.8	52.3	Shoulder to head
		U13	93	3.36	13.1 (12.9)	1.341 (1.691)	8.8 (6.9)	0.4	52.8	22.3	Upper arm to head

This table presents the number of HAEs (event count), prevalence (% of ruck involvements resulting in an HAE) and head kinematic metrics (median PLA, PAA and RVCI) by player role during ruck events, stratified by playing grade. Incidence is expressed as the number of HAEs > 30 g per 100 ruck events. For high-magnitude events (HAE > 30 g), the table also includes the median PLA and RVCI values, and identifies the contact mechanism most frequently associated with > 30 g events for each role

HAEs head acceleration events, *IQR* interquartile range, *PAA* peak angular acceleration, *PLA* peak linear acceleration, *RVCI* rotational velocity change index

[a]Indicates that there were no > 30-g mechanisms for this player role; the mechanism presented the next highest HAE magnitude

our statistical approach. Future studies may benefit from incorporating the time of the season as a covariate to further explore potential temporal effects.

5 Conclusions

This study offers a comprehensive analysis of the match context for HAEs in male community rugby underlining how age, playing position, contact type, and tackle technique shape head kinematics and potential injury risk. While younger cohorts experienced fewer high-magnitude HAEs overall, they were notably more susceptible to rotational loading, which may reflect underdeveloped neck strength and head control mechanisms, factors that may elevate concussion risk during indirect or secondary contact events.

Across all age grades, tackles and rucks consistently emerged as the primary driver of HAE frequency and magnitude. Within these contact phases, several key risk factors for HAE propensity and severity were identified, including upright tackler posture, both high and low tackle heights, and defensive ruck roles. These risks were particularly pronounced in the U19 cohort, where defensive jacklers exhibited the highest rates of high-magnitude HAEs.

These findings underscore the need for a multi-faceted approach to injury mitigation in community rugby. This includes promoting posture adjustments, encouraging balanced tackle strategies (e.g. mid-range tackle height with appropriate head placement) and delivering position-specific skill development to reduce head acceleration loads. In parallel, regulatory measures must evolve to reflect the biomechanical realities of community-level play, with special attention to the risks facing younger and defensively engaged players. Together, these strategies offer a pathway to enhancing player safety and reducing head impact exposure in the community rugby environment.

Acknowledgements This study would not have been possible without the support of a number of organisations and individuals from the Dunedin community rugby family and all the players and coaches who participated in this study. Specifically, we thank Spencer Byers, who completed the impact verification, DM, BRC, JM, and DT who assisted with the mouthguard fitting, and all other research assistants who aided in the data collection. We thank the Otago Rugby Football Union for their support of the project and assistance with team recruitment. We thank Otago Polytechnic, HC, Elevate Analysis, and AS for their support with video capture of trainings and matches and coding. We also thank Prevent Biometrics for their support and cooperation.

Funding World Rugby funded this project. This study builds upon the foundation of our ongoing research under the Otago Rugby Community Head Impact Detection (ORCHID) study series, which focuses on understanding the biomechanics and contextual risks of HAEs in men's and women's community rugby.

Declarations

Conflicts of Interest MDB has received research funding from World Rugby. JR is currently employed part-time by New Zealand Rugby as a Concussion Scientist and is enrolled as a PhD candidate at the University of Otago. RT serves as a consultant for World Rugby. DS was employed by New Zealand Rugby at the time of the study, received research funding from World Rugby and is currently employed by the International Rugby Players Association. EF is the Chief Medical Officer for World Rugby. BN is currently employed by Prevent Biometrics as the Director of Applied Science and Integration, and was a research assistant at the University of Otago during the time of the study. While several authors hold roles within professional rugby organisations, these are disclosed in the interest of transparency. No author received direct compensation for the conduct or reporting of this study. The authors affirm that these affiliations did not influence the study design, data interpretation or manuscript preparation. All other authors declare no competing interests.

Ethics Approval The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. This study involved human participants and was approved by the University of Otago Human Ethics Committee ID:H21_056UOHEC (Health) ID H21_056.

Consent to Participate Written informed consent was obtained from all players; legal guardian consent was also obtained from players under the age of 16 years.

Consent for Publication Not applicable.

Availability of Data and Material Data are available on reasonable request to the corresponding author.

Code Availability Custom codes for data processing are hosted on GitHub and can be shared upon request after all manuscripts have been accepted for publication.

Author Contributions MDB, DS, RT and EF conceived and planned the study. DS, JR, GS and MDB oversaw and carried out the data collection in the field. DT oversaw the iMG fitting. MDB, RMG, BN, and JR oversaw the data cleaning and processing. MDB and RMG wrote the custom codes for data processing and synchronising events and completed the data analysis. MDB took the lead in writing the manuscript. All authors provided critical feedback and helped shape the final manuscript submission.

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