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Player and match characteristics associated with head acceleration events in elite-level men's and women's rugby union matches

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ABSTRACT

Objective To examine the likelihood of head acceleration events (HAEs) as a function of previously identified risk factors: match time, player status (starter or substitute) and pitch location in elite-level men's and women's rugby union matches.

Methods Instrumented mouthguard data were collected from 179 and 107 players in the men's and women's games and synchronised to video-coded match footage. Head peak resultant linear acceleration (PLA) and peak resultant angular acceleration were extracted from each HAE. Field location was determined for HAEs linked to a tackle, carry or ruck. HAE incidence was calculated per player hour across PLA recording thresholds with 95% CIs estimated. Propensity was calculated as the percentage of contact events that caused HAEs across PLA recording thresholds, with a 95% CI estimated. Significance was assessed by non-overlapping 95% CIs.

Results 29 099 and 6277 HAEs were collected from 1214 and 577 player-matches in the men's and women's games. No significant differences in match quarter HAE incidence or propensity were found. Substitutes had higher HAE incidence than starters at lower PLA recording thresholds for men but similar HAE propensity. HAEs were more likely to occur in field locations with high contact event occurrence.

Conclusion Strategies to reduce HAE incidence need not consider match time or status as a substitute or starter as HAE rates are similar throughout matches, without differences in propensity between starters and substitutes. HAE incidence is proportional to contact frequency, and strategies that reduce either frequency or propensity for contact to cause head contact may be explored.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Head acceleration events (HAEs) mitigation strategies are a priority for rugby union to optimise player welfare.

⇒ Instrumented mouthguards show promise as an accurate method for on-field HAE measurement.

WHAT THIS STUDY ADDS

⇒ HAEs occurred in on-field regions where contact event occurrence was the highest.

⇒ Substitutes had a higher HAE incidence than starters over certain PLA recording thresholds for men.

⇒ No significant differences in match quarter HAE incidence or propensity were found.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Mitigating HAEs will involve reducing exposure to contact events since these are primarily responsible for HAEs.

⇒ Potential mitigation strategies should aim to reduce exposure to specific incidents or the likelihood of HAEs in specific circumstances, particularly where propensity is elevated.

⇒ HAE exposure should be monitored for starters and substitutes, particularly forwards, who experience a higher incidence of HAEs than backs.

INTRODUCTION

Participation in contact sports such as rugby union carries a risk of concussion and head impacts.[1–3] Emerging evidence about the potential medium and long-term health consequences of both concussions and non-concussive impacts necessitates interventions aimed at reducing both the frequency and magnitude of these events.[4–6] Head acceleration events (HAEs) can result from either direct head contact or body contact and thus provide a means to quantify and describe direct and indirect head loading.[1] While the consequences of specific frequency and magnitudes of HAEs on long-term brain health remain unknown, a precautionary approach to reducing HAE exposure is recommended.[1] Accordingly, developing strategies to reduce both population and individual-level HAE exposure, thereby potentially mitigating concussion and long-term risk, is of paramount importance.[7–9]

Instrumented mouthguards (iMG) can quantify head linear and rotational kinematics during on-field HAEs by providing coupling with the skull through the upper dentition.[10] Head kinematics (linear acceleration, angular acceleration and angular velocity) are associated with concussion injury risk and HAEs.[1 11 12] The primary contributor to brain deformation appears to be rotational head kinematics and various biomechanical brain injury mechanisms may exist, including those involving repetitive HAEs.[1]

To develop effective interventions to reduce HAE incidence, it is necessary first to understand the circumstances under which HAEs occur. This identifies risk factors for HAEs, allowing mitigation strategies to be explored to reduce overall HAE incidence, either by reducing exposure to the events more likely to cause HAEs or by reducing the likelihood that a circumstance will cause an HAE. For this reason, an approach that explores both propensity (the likelihood that a given circumstance or behaviour causes an HAE) and incidence (overall HAEs per player hour, a function of propensity and exposure) is necessary. In the present study, the HAE incidence and propensity for three factors previously identified as risk factors for injury in the sport were determined. The study aimed to compare HAE incidence and propensity for starter/substitute players and match quarter and to examine the field location of tackle, carry and ruck HAEs within elite-level men's and women's rugby union matches throughout a season.

METHODS

Study design and participants

A prospective observational cohort study was undertaken with 179 (118 forwards, 60 backs) and 107 (60 forwards, 47 backs) participants from the men's and women's games, respectively. Participants were recruited from elite-level Premiership and Premier 15s clubs, respectively, during the 2022/2023 season, representing the highest club rugby levels in England. Data were collected from domestic league, cup and European cup competitions for men and domestic league and cup competitions for women. All participants underwent three-dimensional dental scans and received a custom-fit iMG (Prevent Biometrics, Minneapolis, Minnesota, USA). The iMGs were equipped with an accelerometer and gyroscope, sampled at 3200 Hz with a measurement range of ±200 g and ±35 rad/s, respectively. Each iMG was also equipped with an embedded infrared proximity sensor to assess the coupling of the iMG to the upper dentition during an HAE. The validity of the Prevent Biometrics (Prevent) iMG has been demonstrated in previous studies both on-field and within laboratory settings.[13–16]

HAEs were identified when a linear acceleration trigger threshold of 8 g measured at the mouthguard was exceeded on a single axis of the iMG accelerometer.[17] HAE kinematics were captured at 10 ms pretrigger and 40 ms post-trigger. Impacts were transformed and recorded at the head centre of gravity (CG) following SAE J211 recommendations.[18] A recording threshold of 400 rad/s^2 and 5 g at the head CG was used to capture and include HAEs from contact events only (positive predictive value >0.99).[17] A trigger threshold of 8 g can record HAEs below 8 g at the head CG.[17 19] Peak resultant linear acceleration (PLA) at the head CG and peak resultant angular acceleration (PAA) were extracted from each HAE.

The level of noise/artefact in the kinematic signal was classified into three classes (0, 1 or 2) by an in-house Prevent algorithm. Class 0 is determined to contain minimal noise within the recorded accelerometer/gyroscope signals, class 1 for moderate noise or class 2 for severe noise. To further process the signals from the accelerometer/gyroscope, prevent applies a fourth-order (2×2 pole) zero phase, low-pass Butterworth filter to each signal with a cut-off frequency (−6 dB)[20] of 200, 100 and 50 Hz for class 0, 1 and 2 HAE, respectively, similar to previous studies.[16 21–23]

Contact event identification

In-game video timestamps of contact events (specifically tackles, carries and rucks) were captured from commercially available match data (StatsPerform, Chicago, Illinois, USA) using broadcast-quality game footage. These events were linked to HAEs to minimise the occurrence of 'false positives', where an HAE occurs without any head impact during the above-identified contact event types. StatsPerform defined carry match events as 'a *player touching the ball has deemed to make a carry if they have made an obvious attempt to engage the opposition with the ball in hand'*, a tackle as '*a player has attempted to halt the progress or dispossess an opponent in possession of the ball'* and a ruck as '*a player enters a breakdown after it has been set'*. Due to the difficulties in visually identifying players and linking these to HAEs, mauls were excluded from this analysis. Additionally, scrum events were excluded from this analysis due to a small sample size of linked HAEs (n=195). A bespoke MATLAB script was used to link the iMG HAE timestamps to in-game timestamps for tackle, carry and ruck contact events. Five randomly selected matches were used whereby HAEs (n=1210) were manually video analysed to test the accuracy of the MATLAB script. The MATLAB script correctly linked 88% of HAEs to the StatsPerform contact events compared with the manual video analysis approach.

Field location

Each contact event was assigned to a field location, identified using a combination of on-field markings as reference points (eg, 22 m lines, 10 m from halfway, try line), and following typical game analysis practices, where game analysts code activities based on their vertical field location such that attack and defence can readily be distinguished from mid-field play. The horizontal (location relative to the sidelines) location is identified to characterise the width of the play. This results in functional regions on the field and enables contact events to be described as a function of width and nature of attack vs defence.

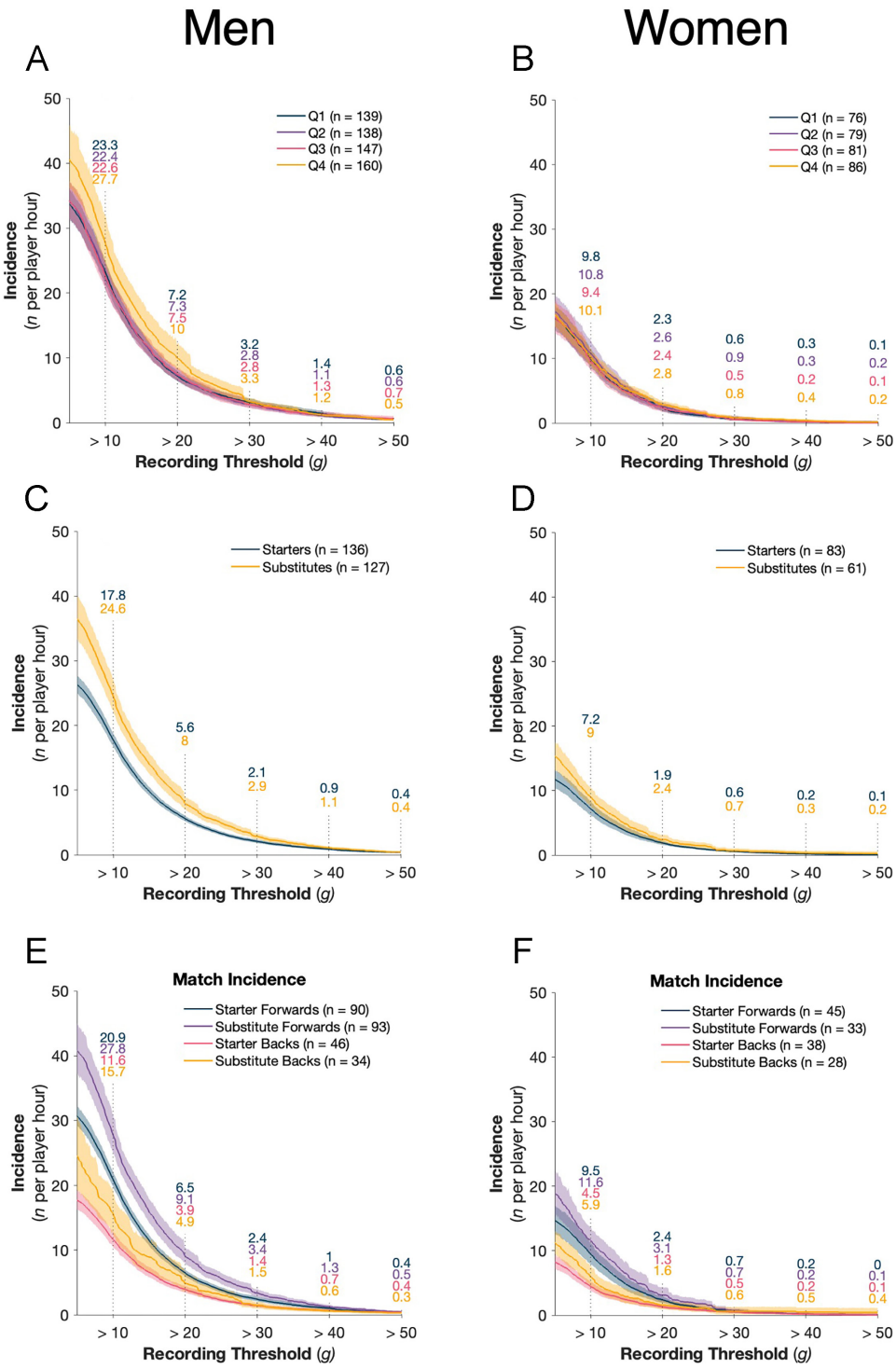

Figure 1 Head acceleration event incidence for (A, B) match quarter, (C, D) starters and substitutes and (E, F) starters and substitutes by player position for men and women across peak linear acceleration recording thresholds. The shaded region indicates 95% CI, and n represents the number of players available for calculation based on compliance requirements (see the 'Methods' section). Online supplemental figure 1 presents the results with a peak angular acceleration recording threshold.

Statistical analysis

29 099 and 6277 HAEs were recorded from 1214 and 577 individual player matches from men's and women's games, respectively. Incidence was calculated as the total number of HAEs per player hour. All HAEs captured during the match period were used for incidence calculations. The player's playing time was obtained from StatsPerform. Subsequently, mean incidence values were calculated across different PLA recording thresholds for playing quarters, starters and substitutes with 95% CIs estimated using a bootstrapping procedure.[17]

Propensity values (with 95% CI) were calculated by dividing the number of tackle, carry and ruck contact events that resulted in an HAE (Men n=18 978, women

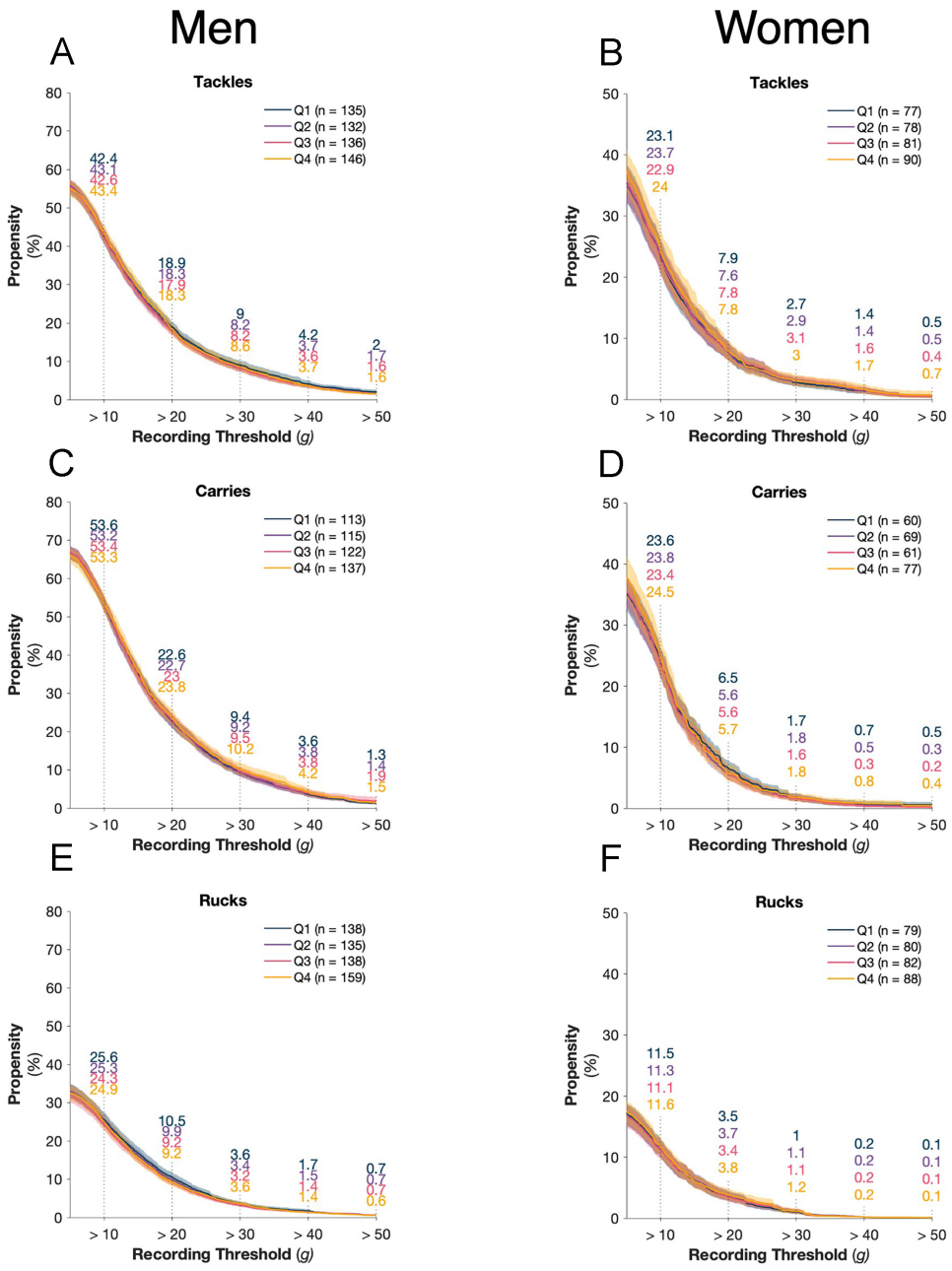

Figure 2 Head acceleration event propensity for tackles, carries and rucks, broken down by match quarters across peak linear acceleration recording thresholds. The shaded region indicates 95% CI. n represents the number of players available for calculation. Online supplemental figure 2 presents the results with a peak angular acceleration recording threshold.

n=3665) by the total number of events the player was involved in while wearing an iMG.[17] A single contact event may result in multiple HAEs due to multiple collisions during the event. In these instances, propensity was calculated using the HAE with the greatest magnitude. Only contact events that corresponded with an on-the-teeth period (based on the iMG proximity sensor) for the instrumented player were used in propensity calculations, and only player matches where the instrumented player wore their iMG for a minimum of 90% of their contact events were used in the incidence calculations.[17]

For statistical comparison of HAEs, a ratio between two HAE propensities was calculated to allow comparisons of HAE risk by match time and in starters vs substitutes. Propensity ratios were explored in three magnitude bands for PLA and PAA thresholds: low magnitude (PLA <10 g and PAA <1.0 krad/s^2), medium magnitude (PLA >10 g and <30 g and PAA >1.0krad/s^2 and <2.0krad/s^2) and high magnitude (PLA >30 g and PAA >2.0 krad/s^2).[17] Significance was determined based on 95% CI not overlapping across recording thresholds.

RESULTS

Match time

There was no significant difference in HAE incidence between match quarters for men (figure 1A). Overall,

Table 1 Propensity ratios of tackles, carries and rucks to result in a maximum-magnitude HAE within low, medium and high HAE magnitude bands as a function of match quarters and starter versus substitutes

		Men			Women		
		Low <10 g	Medium 10 g <30 g	High >30 g	Low <10 g	Medium 10 g<30 g	High >30 g
Tackles	Q1 vs Q2	1.01	0.96	1.10	1.01	0.98	0.95
	Q1 vs Q3	1.00	0.97	1.09	1.00	1.02	0.90
	Q1 vs Q4	1.02	0.96	1.05	1.01	0.97	0.91
	Q2 vs Q3	0.99	1.02	0.99	0.90	1.05	0.95
	Q2 vs Q4	1.00	1.00	0.95	1.00	0.99	0.96
	Q3 vs Q4	1.01	0.99	0.96	1.02	0.94	1.02
Carries	Q1 vs Q2	0.99	1.00	1.02	1.00	0.99	0.96
	Q1 vs Q3	1.00	1.01	0.99	1.00	1.00	1.10
	Q1 vs Q4	0.99	1.02	0.92	1.10	0.97	0.97
	Q2 vs Q3	1.00	1.00	0.97	0.99	1.01	1.14
	Q2 vs Q4	1.00	1.02	0.90	1.01	0.97	1.01
	Q3 vs Q4	1.00	1.02	0.93	1.01	0.96	0.88
Rucks	Q1 vs Q2	1.00	1.00	1.06	1.00	1.03	0.92
	Q1 vs Q3	0.98	1.05	1.11	1.00	1.05	0.91
	Q1 vs Q4	0.99	1.03	0.99	1.00	1.00	0.89
	Q2 vs Q3	0.99	1.04	1.05	1.00	1.02	0.99
	Q2 vs Q4	0.99	1.03	0.94	1.00	0.97	0.96
	Q3 vs Q4	1.00	0.99	0.90	1.01	0.95	0.97
Tackles	Starter vs Substitute	1.05	0.96	0.85	1.03	0.89	0.91
Carries	Starter vs Substitute	0.98	1.00	1.08	1.06	0.81	1.88
Rucks	Starter vs Substitute	1.01	1.00	0.83	1.02	0.85	1.43

Ratios are calculated based on the text labels at each interval presented in figure 2. Q1–4 represent match quarters, with Q1 and Q2 representing the first half, with Q3 and Q4 representing the second half. If no HAEs were recorded during a contact event, the maximum HAE was deemed to be within the low band (<10 g). No significant differences were found across the propensity ratios.
HAE, Head Acceleration Event.

there were 27.7 HAEs per player hour above 10 g in quarter 4 compared with 23.3, 22.4 and 22.6 in quarters 1, 2 and 3, respectively (figure 1A). HAE incidence was similar across quarters in the women's game (9.8, 10.8, 9.4 and 10.1 HAEs per player hour above 10 g in quarters 1, 2, 3 and 4, respectively, figure 1B). The propensity of contact events to result in HAEs was also unchanged over time (figure 2, table 1).

Starters and substitutes

In men, 86.6% (48 942 min) of the total playing time (56 498 min) was from starters, with 13.4% (7556 min) from substitutes. In women, starters accounted for 86.8% and substitutes 13.2% of the total 23 153 min.

When considered as a single group, substitutes have a higher HAE incidence than starters in men's rugby (24.6 vs 17.8 HAEs per hour, figure 1C). In women, no substitute versus starter difference was found (figure 1D). When considered by the positional subgroup, substitute forwards had a higher lower magnitude HAE (<10 g) incidence than starter forwards (27.8 vs 20.9 for subs vs starters, respectively, figure 1E). At the same time, no differences were found between starter and substitute backs. No differences in incidence were found between positional groups in the women's game (figure 1F).

There were no statistical differences in HAE propensity for the three contact event types between starters and substitutes (table 1), nor when broken down by position group (figure 3).

Field location

Tackler HAE propensity was highest in the defending half of the field for men and women (figures 4 and 5). Carry HAEs, conversely, were more likely when attacking in the opposition half. For both tackle and carry HAEs, there was a slight over-representation of the attacking left side of the midline (figures 4 and 5). Overall, HAEs occurred in on-field regions with high contact event occurrence (figures 4 and 5). For higher magnitude events (>30 g), HAE propensity for tackles and carries was highest between the 5 m line and the try-line in both the men's and women's games (figures 4 and 5). A breakdown of

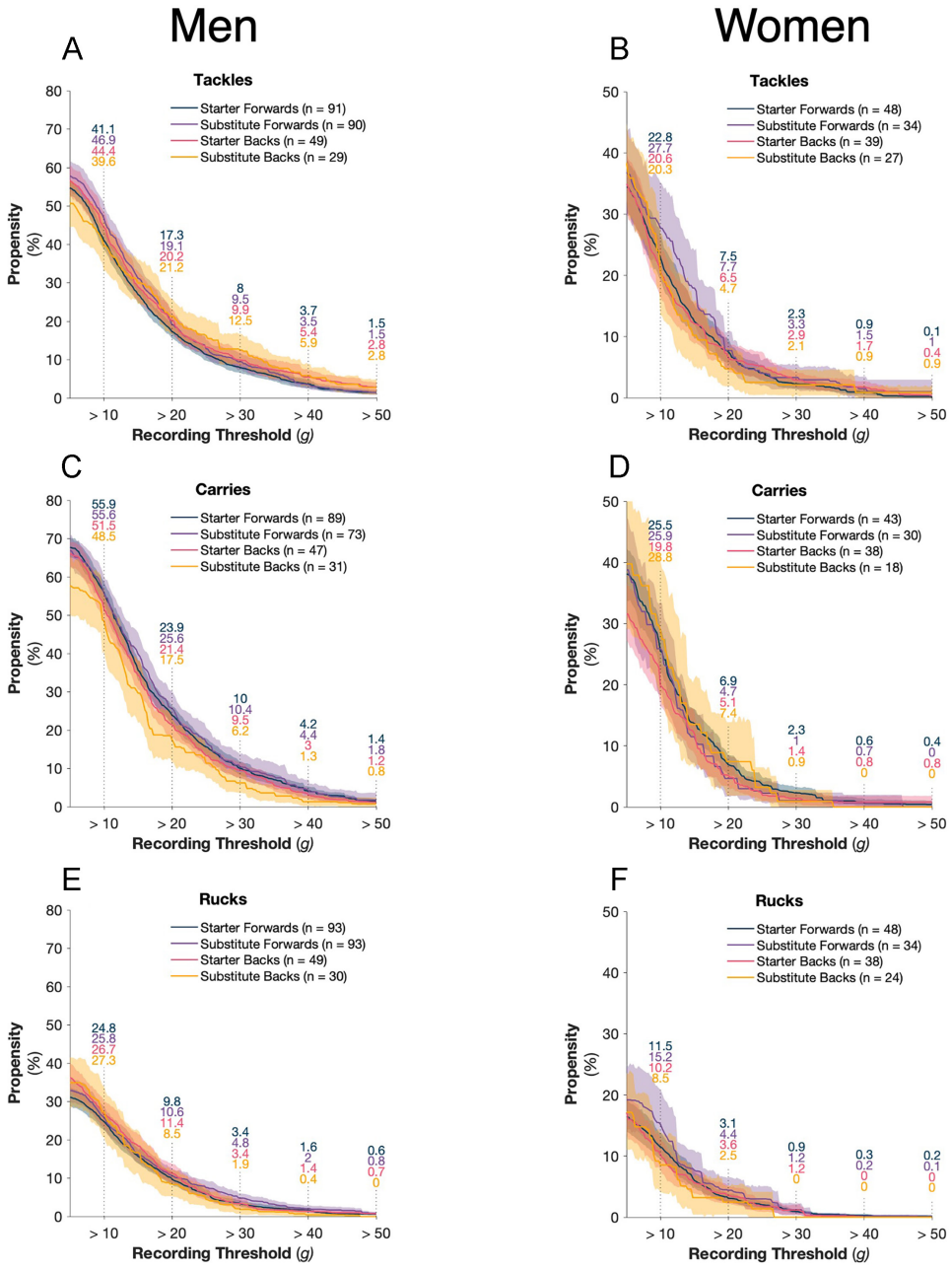

Figure 3 Head acceleration event propensity for tackles, carries and rucks broken down by starters, substitutes and player position across peak linear acceleration recording thresholds. The shaded region indicates 95% CI. n represents the number of players available for calculation. Online supplemental figure 3 presents the results with a peak angular acceleration recording threshold.

the number of events and HAE propensity in each region can be found in online supplemental tables 1 and 2 for the men's and women's games, respectively.

DISCUSSION

This study explored how previously researched risk factors for injury—field location, time of match and starters versus substitutes—influence both the incidence and propensity of HAEs during contact events and match play in rugby union to provide insight into the timing and circumstances of HAEs to inform potential interventions that reduce the number of head accelerations players will experience in the game.

Accordingly, the first important finding is that HAE incidence and propensity are not affected by the time in the match. No changes were found for the HAE propensity across a spectrum of PLA magnitudes for tackles, carries or rucks as the game progressed and nor was HAE incidence affected by the match quarter, suggesting that both exposure to contact events and the likelihood that those contact events will cause HAEs at any magnitude is not affected by match time. This has implications for how

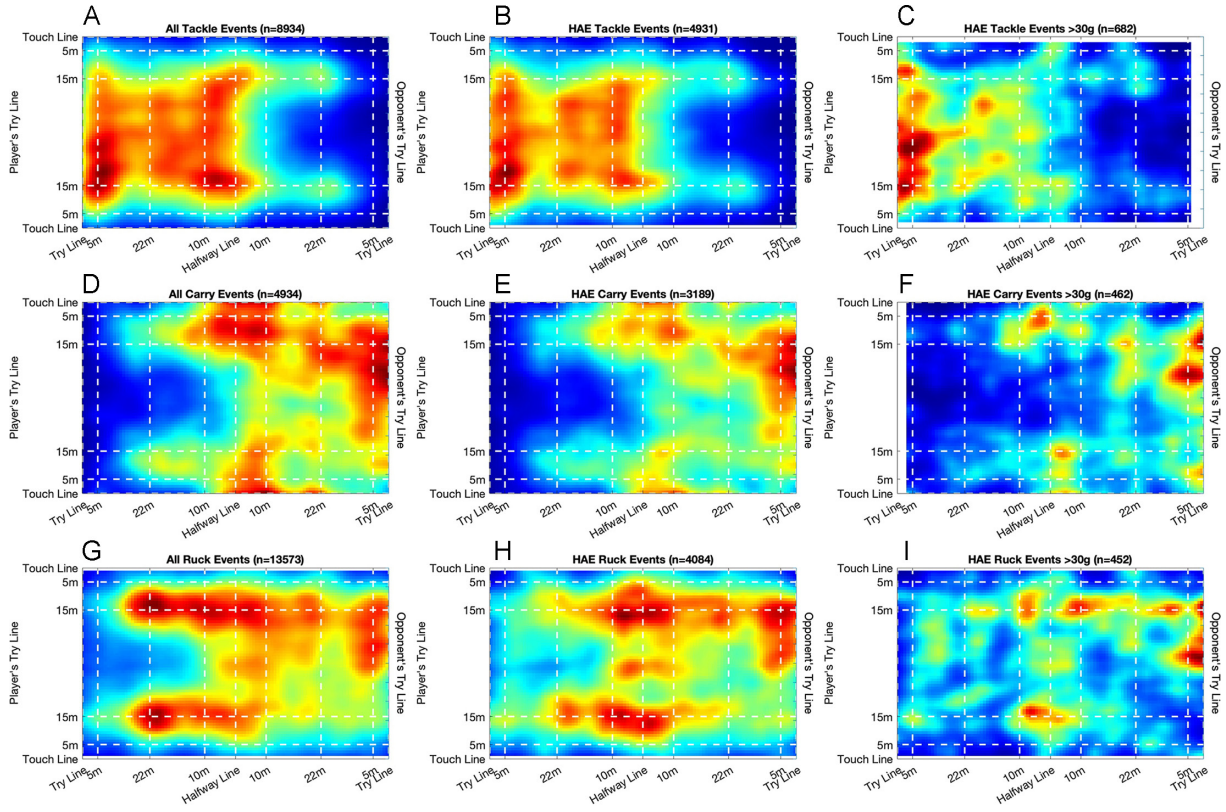

Figure 4 Field location for tackle (A–C), carry (D–F) and ruck (G–I) contact events, head acceleration events (HAE) and HAEs >30 g for the men's game. The colour of the density plot indicates the number of events that occurred in that region. The more red the colour, the more HAEs or contact events occurred in that region.

fatigue and changes in match activities over time affect HAE risk and outcomes. Previous studies have shown that fatigue is a risk factor for injuries,[24–27] though not for HIA removals.[28] We find no such relationship, though, as discussed subsequently, the interaction of accumulated playing time's effects on fatigue and substitutions makes this interpretation complex. It is, of course, not as simple as suggesting that players in Q4 are more fatigued than in Q1 since the introduction of substitutes means that a proportion of players in Q4 are playing their first 20 min.[29]

The second important finding is that substitutes have the same propensity for HAEs during contact events as starters but have a significantly higher HAE incidence. This is intriguing since propensity findings (figure 3) suggest that the per-contact-event-risk is similar between starters and substitutes regardless of playing position, implying that a substitute's tackle, carry or ruck involvement is as likely to cause an HAE as a starter's. The higher HAE incidence in substitutes must thus be attributable to greater exposure of substitutes to contact events per unit time on the field. We interpret our combination of findings to show that substitutes have higher rates of HAEs because of greater contact frequency, not greater contact risk. This is explained by the positional bias in substitutions, where five or six of the eight available substitutes are usually forwards, whereas starters are split 8/7 between forwards and backs.[29] This accounts for our finding that the HAE incidence in forward substitutes, specifically in the men's game, is considerably higher than for starters, compared with the relatively smaller difference found between starter and substitute backs (figure 3).

Our comparison of starters and substitutes does not enable a direct evaluation of the effects of relative fatigue (in starters) compared with relative freshness (in substitutes). The dataset, for starters, comprises all their contact events, regardless of match time. Second, we compare the HAE propensity in starters and substitutes, but not whether these roles cause HAEs in other players. To tease out the possible influence of freshness on HAE risk, evaluating the HAE propensity in both the tackler and ball carrier is necessary as a function of whether each is a starter or substitute. In future, research should evaluate HAE propensity in the first 10 min of play, irrespective of starter versus substitute roles and compare this to the HAE propensity in players who have played for more than 60 min, for instance. Future research should also explore the possible integration of other game metrics, such as GPS, to understand further how substitutions affect game involvements, with resultant implications for HAE risk.

The final important finding is that HAE propensity is closely linked to players' activities at the time of contact and the location on the field where those activities occur. HAE propensity is highest during tackles when players are defending their try line and during carries when attacking the opposition try line (online supplemental

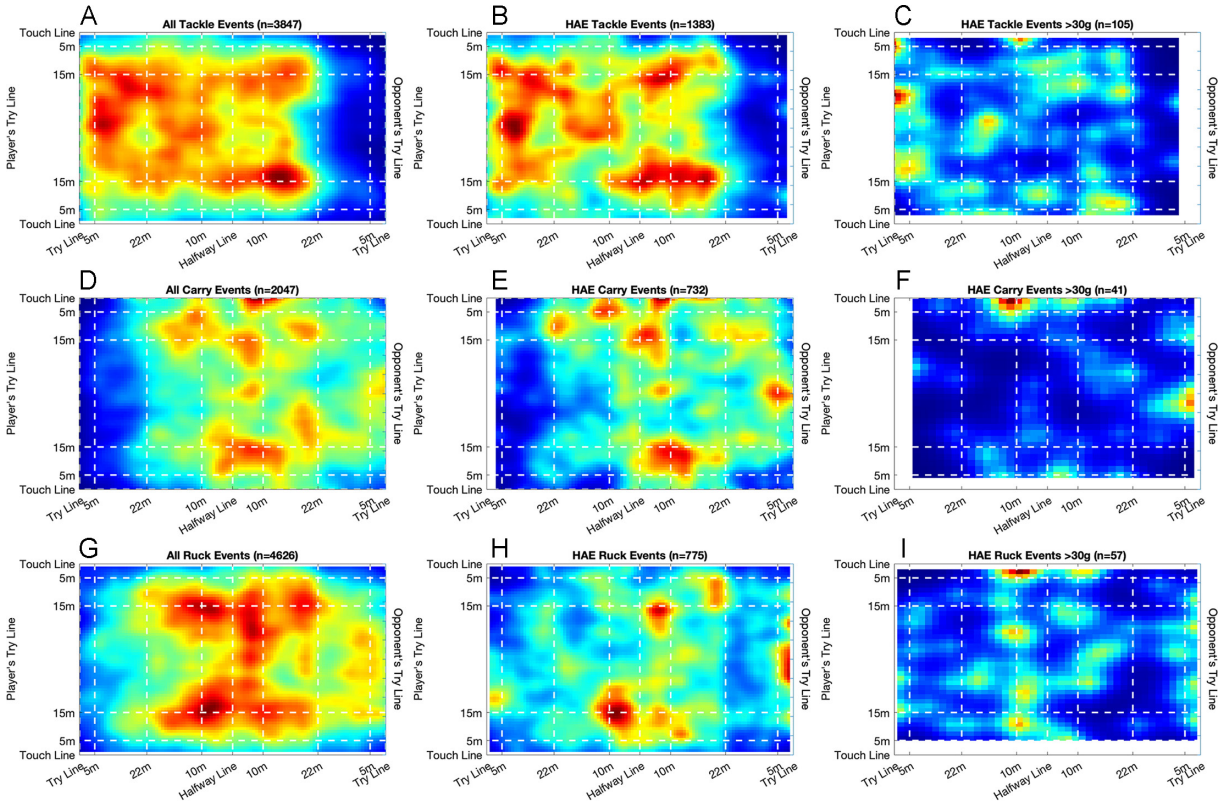

Figure 5 Field location for tackle (A–C), carry (D–F) and ruck (G–I) contact events, head acceleration events (HAE) and HAEs >30 g for the women's game. The colour of the density plot indicates the number of events that occurred in that region. The more red the colour, the more HAEs or contact events occurred in that region.

tables 1 and 2). This is likely a function of the increased intensity of those tackles and carries to prevent or score a try, which may impact the technique used by players in each situation. This would increase the likelihood of significant HAEs, compared with tackles and carries in the midfield, where 'winning the collision' is not as influential. We have not, however, assessed technique in the identified higher-risk situations, but our finding suggests that future research might explore this possibility.

Collectively, the three findings invite consideration of potential mitigation strategies that seek to either (a) reduce the exposure to specific incidents or circumstances identified as causing more HAEs or (b) reduce the likelihood of HAEs in specific circumstances, particularly where propensity is noted to be elevated. Either would reduce overall HAE incidence and exposure. Given that no influence of match time on contact event HAE incidence or propensity was found, there is no specific mitigation that would reduce HAE numbers at the level of match time. Our finding that substitutes have the same propensity as starters regardless of playing position does not support the implementation of strategies that may reduce HAE exposure by limiting the number of substitutes unless it can be shown that substitutes change the frequency of exposure to HAE-inducing contact events.

The final finding that the propensity is greatest for tackler defence and ball carrier attacking situations at the try line is also difficult to mitigate without materially affecting how the game is played. As noted, this is likely explained by the urgency and the resultant intensity of the tackles and carries made in these areas since they are point-scoring (or preventing) opportunities. To reduce these propensities without neutralising the contest would be difficult. One approach may be to limit the number of such occurrences, possibly by considering the introduction of time or phase limits for teams in possession in these areas of the field. This would reduce overall HAE numbers through an exposure reduction rather than propensity reduction. It would, however, represent a significant law change for the sport. Alternatively, the possibility that safe technique is compromised in these situations should be explored, as this may invite coaching interventions to reduce HAE risk by reducing propensity while these situations occur at the same frequency.

More generally, mitigation of HAEs will involve reducing exposure to contact events since these are primarily responsible for HAEs.[7 17 30 31] Fatigue remains a potential source of increased HAE risk. Still, the present analysis does not allow the nuance of that potential risk to be explored in a way that may inform whether changes to substitution numbers would increase or decrease overall HAE numbers.

Limitations

One of the main limitations of this study was player compliance with wearing iMG. Where 762 players

consented to participate in this study, only 286 provided data that could be examined. This may create a potential bias in the study results as the players who wore the iMG and are included in the presented dataset may not represent all players in the cohort. The current study may not fully represent the various playing styles and conditions at all rugby levels worldwide. Match HAE characteristics may differ in other rugby cohorts, particularly amateur and youth level.

This study focused on high-level player and match characteristics. It did not look at the effect of tackle, carry, ruck technique or other more detailed characteristics of the contact events on HAEs. Technique is understood to be a significant risk factor for injuries, including concussions, and thus likely plays a role in HAE incidence.[9 32] Further research needs to look at the effect of the technique on HAE incidence. Understanding the effect of the technique on HAE incidence may allow for the enhancement of technical coaching strategies/cues and/or influence possible law changes in the sport to reduce HAEs and, as a result, potential injury risk. Another limitation is the relatively sparse use of iMGs by players in the same match, which prevents exploration of the interactions between players involved in contact situations. In future, when more players are wearing iMG devices, it will be possible to study and compare HAE outcomes for different role players, characteristics and behaviours. Finally, prevent uses in-house algorithms for data processing and filtering. A fully transparent and common signal processing approach, such as the HEADSport filter method,[20] would benefit future study comparisons, particularly if other iMG systems are used.

CONCLUSION

Season-long, competition-wide implementation of iMGs was undertaken to explore how risk factors for injury—time of match, starters versus substitutes and field position—influence both incidence and propensity of HAEs during contact events and match play within elite-level rugby union. HAE incidence and propensity were not affected by match time. Substitutes were found to have a similar propensity yet a higher incidence when compared with starters, likely because the substitute group comprises relatively more forwards who engage in contact activities. Finally, HAE propensity was greatest for attacking (carries) and defending (tackle) situations at the try line. Potential mitigation strategies, therefore, should look to reduce exposure to specific incidents or the likelihood of HAEs in specific circumstances, particularly where propensity is elevated.

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Contributors DA, LS, RT, ECF and GT conceptualised the research project. All authors were involved in design and data collection for the study. DA, JT, LS, RT, ECF and GT were responsible for the analysis and interpretation of the results. DA and GT drafted the manuscript. All authors critically reviewed and edited the manuscript prior to submission. DA is the guarantor for this study and accepts full responsibility for the finished work.

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Competing interests GT and BJ have received research funding from Prevent Biometrics and World Rugby. KAS and MB have received research funding from World Rugby. LS, RT, ECF, DMS and JB are employed by or contracted as consultants to World Rugby. GT previously conducted consultancy work for World Rugby. KAS and SPTK are employed by the Rugby Football Union. SH receives funding for his PhD studies from the Rugby Football Union and Premiership Rugby. BJ is a consultant with Premiership Rugby and the Rugby Football League. MC is employed by Premiership Rugby and was previously employed by the Rugby Football Union. PO'H has previously been contracted by the Rugby Football Union and is employed by Marker Diagnostics UK, a company developing salivary biomarker testing for sport-related concussion.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and this project was approved by the University's Research Ethics Committee (UREC), University of Ulster (#REC-21-0061). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available on reasonable request.

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