Effects of one season of rugby on the neurological integrity of adolescent players

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Abstract

Background
Rugby union is a popular contact sport where high impact collisions frequently occur. Concern exists regarding concussion and the overall brain health of those playing the game. Repeated sub-concussive collisions may compromise rugby players' neurological integrity. Presently little objective data exists in this research space regarding head acceleration events experienced by adolescent rugby players. More research is justified in this population as adolescents make up the majority of those playing the game.
Methods
Forty-one male participants were recruited from two under-16 rugby teams. Participants underwent pre- and post-season MRI scans (T1-weighted structural imaging and high angular resolution diffusion imaging), neuro-cognitive, and conscious motor control assessments. Participants were fitted with instrumented mouthguards to record head acceleration events experienced during the season. Post-season processing of MRI scans focused on within-subject analysis of pre- to post-season changes in white matter as measured by diffusion tensor imaging. Linear mixed models were used to investigate correlations between neurological changes and cumulative head impact loading recorded by the mouthguards.
Findings
Results from pre- and post-season MRI scans indicated a non-significant relationship between head acceleration event exposure in one season of rugby and changes in white matter microstructure, including for those players exposed to a higher level of impact magnitude.
Interpretation
MRI results suggest exposure to one season of rugby for healthy adolescent male players does not directly result in neurological compromise. There was a non-significant relationship between the primary variable of interest, total cumulative loading, and changes in white matter integrity as measured by diffusion tensor imaging. This non-significance held when controlling for variables such as training age, headgear use, and body mass index. Further research, particularly using longitudinal designs, is needed to further elucidate the potential for microstructural neurological change in adolescent rugby players.
Funding
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Introduction

The neurological integrity of those playing contact sport is an issue of concern worldwide.[1] The majority of such research has taken place in adult, male cohorts, particularly in American football.[2] A lack of data exists for rugby, despite it being a full contact sport with wide global popularity.[3] This lack of data is especially prescient with junior and female players.[2] In New Zealand, where the study took place, 76% of players are under-18 (NZ Rugby, unpublished dataset). If it is accepted that this concern comes from the repeated exposure of players to sub-concussive collisions, and such collisions are inherent in rugby,[4] then one pertinent research approach, described here, is to measure these collisions and their associated head acceleration events (HAEs). Recent technological advances (namely instrumented mouthguards) provide a means though which to quantify the incidence and relative size of HAEs (peak linear acceleration (PLA) and peak rotational acceleration (PRA)) in rugby.[2]

To the best of our knowledge, this study is the first of its kind using a cohort of adolescent rugby players to combine quantification of HAE incidence and magnitude with the use of MRI-based metrics as a primary outcome. This work is essential to more clearly understand the singular and cumulative effect of HAEs on brain integrity. Results from the study could additionally provide critical data to rugby stakeholders (including governing bodies, coaches, and players) which in turn could improve the safety and subsequently the brain health of those playing the game. As a prospective cohort study, this research differs from the mostly retrospective work, conducted almost entirely on adult players, thus far in the field.[5-8]

The aim of the current study was to investigate how cumulative loading for incidence and magnitude intersects with potential changes in white matter (WM) diffusion, based on one season of play in under-16 male rugby players. Pre- and post-season neuro-cognitive assessments and structural and diffusion-based MRI scans were conducted on all participants. We hypothesised those participants exposed to a lower incidence and magnitude of head acceleration events (PLA/PRA) would show fewer changes (pre/post season) in WM fibre cross section and density, neurocognitive testing scores and conscious motor control than those exposed to higher total exposures.

Methods

Study design

This study was a prospective cohort design. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE)[9] guidelines for cohort studies informed the reporting of the study design for our research and were followed for reporting of results. The study was conducted with two junior male (under-16) rugby teams, during the 2022 and 2023 seasons (one in each season). Pre- and post-season testing was undertaken with each participant.

The study was based at two junior rugby clubs in Canterbury, New Zealand. The Canterbury Rugby Football Union (CRFU) and Ellesmere Rugby Sub-union (ERSU) were fully supportive of the research and provided letters to indicate their endorsement of the study. Inclusion criteria was defined as: being a member of U16 male rugby team, able to take full part in training and competitive matches, able to give informed written consent, and willingness to wear HitIQ Nexus A9 mouthguards (HitIQ Ltd, Melbourne, Australia) at all trainings and matches. Exclusion criteria was defined as: declines to take part in study.

Participants

Ethical approval for all procedures and experimental design was obtained in full from the University of Canterbury Human Ethics Committee prior to undertaking the study. Ethics approval for the study was obtained on 13 July 2021 (Ref: HEC 2021/26). The study was approved by New Zealand Rugby's (NZR) Medical Science and Advisory Panel (MSAP).[10]

Forty-two (n = 42, age 15·02 ±0·76) under-16 male rugby players provided informed written assent and signed up to participate in this observational cohort study. All participants' parents provided written consent. Forty participants (n = 40) completed a full season of rugby with full sets of data associated with their participation, with two participants withdrawing. Five concussions (n = 5, one in the first season, four in the second season)

were diagnosed by a medical doctor. All concussed players were compliant in completing post-concussion neuro-cognitive assessments and MRI scans.

Head acceleration events (HAEs) were recorded by the instrumented mouthguards and video verified by trained researchers. Incidence was calculated via the number of HAEs in total, and per player-hour.[5] Impact rate was calculated by the sum of verified HAEs ≥ 8 g divided by the sum or player-hours.[11] Magnitude was reported as peak linear acceleration (PLA) and peak rotational acceleration (PRA). Total cumulative impact loading was calculated per player, in both game-play and training scenarios, by summing PLA and PRA values across the whole season.[12] To date, no accepted procedure exists for calculating cumulative impact burden.[12]

Procedures

Participants underwent a comprehensive MRI scan protocol pre- and post-season. Neuro-cognitive assessments (NIH Toolbox, Movement Specific Reinvestment Scale, Decision Specific Reinvestment Scale) were performed at the MRI appointment.[13, 14] The MRI protocol included a T1-weighted image to measure tissue volumes and cortical thickness and a high angular resolution diffusion imaging (HARDI) to measure white matter integrity. This MRI protocol was a non-invasive and safe imaging technique for repeat scans in this cohort.

MR imaging was performed on a 3T Siemens (Siemens Healthcare, Erlangen, Germany) Skyra scanner (Pacific Radiology, St George's Hospital, Christchurch, New Zealand) using a 64-channel head and neck coil. The primary acquisition was (a) multi-shell HARDI, 2-dimensional diffusion-weighted imaging, with uniformly distributed directions (25, 50, and 75 volumes each with: b=1000/2000/2700 s/mm^2 respectively) and 10 acquisitions without diffusion weighting (b = 0 s/mm^2):TE/TR =92/3600 ms, flip angle=78°, acquisition matrix=110×110, FOV =220 mm, 72 slices, slice thickness 2 mm, multi-band factor=3, ungated, phase encoding anterior-posterior (plus flipped phase encoding images to enable distortion correction), voxel=2×2×2 mm^3. Other parameters were as follows: (b) Structural T1-weighted MPRAGE (TE/TR/TI=2.85/2000/880 ms, Flip angle=8°, FOV=256 mm, bandwidth=240 Hz/pixel, voxel=1×1×1 mm^3).

T1-weighted images were processed to calculate estimates of cortical thickness, surface area, and volumes, using Freesurfer (v.7.2.0; http://surfer.nmr.mgh.harvard.edu). Pre- and post-season T1 scans were processed using the longitudinal pipeline. In brief, pre- and post-season T1 scans ($T1F_0$ and $T1F_1$) were used to create an unbiased within-subject template.[15] Amalgamation of subject space into midspace, using inverse consistent registration,[15] allowed extraction of global metrics in each participant at each timepoint. Images were visually inspected for reasonable representations of grey matter (GM) and white matter (WM) using spherical surface maps and parcellations.[16] The unbiased midspace T1 image was then normalised (warped to fsaverage space and smoothed) to MNI space. The difference in values between the two timepoint scans in MNI space allowed for calculation of within-subject structural change. T1-weighted images are often used in diffusion weighted imaging (DWI) studies to provide anatomic reference.[17, 18] The following values for global metrics were then isolated for analysis: cerebral WM volume (mm^3), subcortical GM volume (mm^3), total GM volume (mm^3), cortical surface area (left and right hemispheres, mm^2), and cortical mean thickness (left and right hemispheres, mm). Total intracranial volume (mm^3) was used as a co-variate while total cumulative loading for each player (in both peak linear and peak rotational acceleration) was the primary variable of interest.

For the diffusion-weighted images, preprocessing in MRTrix3 (version 3.0.3, https://mrtrix.readthedocs.io/en/3.0/3/) included denoising and Gibbs-ringing artefact removal. The images then underwent susceptibility-induced distortion correction (using the reversed phase-encoding volumes) using FSL 'topup'. Diffusion-weighted volumes then underwent eddy current and motion correction using FSL 'eddy'.[19] No participants were excluded after motion correction using this procedure. Excessive motion was defined as mean relative motion greater than three times the standard deviation of the group. Brain mask estimations were then improved using bias field correction. Using commands from MRTrix3 shells were extracted using the 'dwiextract' command, then undergoing diffusion tensor estimation using the 'dwi2tensor' command.[20] Images for the following DTI metrics in subject space were created: fractional anisotropy (FA), mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD).

All participants' fractional anisotropy (FA) data was used to create an unbiased, within-subject FA median template using Freesurfer (v7.2.0; surfer.nmr.mgh.harvard.edu), as well as registering FA/MD/AD/RD images from the two time-points (F_0 and F_1) into this within-subject template.[15] The template was then used to create a mean FA skeleton which represents the centres of all tracts common to the cohort, using tract-based spatial statistics (TBSS) via the ENIGMA-DTI skeletonization process (enigma.ini.usc.edu/protocols/dti-protocols/#eDTI). The FA data for each participant was then projected onto the skeleton. The skeleton

projection was then applied to non-FA data, therefore creating skeletons for all DTI metrics. For each measure, the two scans (FA F_0 and FA F_1) were merged into midpoint-space, normalised to MNI space, and projected back onto the skeleton as F_0 MNI and F_1 MNI. Average DTI metrics along the skeleton and within white matter (WM) tracts (defined by the JHU ICBM-DTI-81 white-matter-labels atlas)[21] were extracted for each participant at each timepoint (F_0 and F_1). This allowed the difference between the two values (e.g. FA F_0 MNI and FA F_1 MNI) to be measured for each DTI metric.[16]

Data was displayed on spreadsheets covering sixty-three white matter (WM) tracts. Five regions of interest (ROIs), along with average FA, were selected *a priori* based on previous research in the sports-related concussion (SRC) and/or repeated head impact exposure (RHIE) fields:[22]

- Corpus callosum (CC): genu (GCC), body (BCC), splenium (SCC)
- Superior longitudinal fasciculus (SLF): right (SLF-R), left (SLF-L)
- Corticospinal tract (CST): right (CST-R), left (CST-L)
- Sagittal stratum (including inferior longitudinal fasciculus and inferior fronto-occipital fasciculus) (SS): right (SS-R), left (SS-L)
- Inferior fronto-occipital fasciculus (IFO): right (IFO-R), left (IFO-L)

Statistical analysis

For the processed MRI results, so-called spaghetti plots were drawn to visually ascertain the patterns and diversity among individuals. Models for structural analysis were generated by using multivariate modelling, while models for diffusion metrics (FA, MD, AD, RD) were generated by using the linear mixed models analysis in R (R Foundation for Statistical Computing, Vienna, Austria; https://www.R-project.org/), utilizing the 1me4 package described by Bates (2015).[23] The mixed linear models allowed individual effects as random variables and other variables as fixed effects to partition the variance of the outcome variables. The associated R codes with comments are posted below for further reference. To conduct these analyses, the data were converted from a wide format to a long format and then each of the outcome variables were regressed on the following variables:

- Training age
- Body mass index (BMI)
- Headgear use
- Time between scans (in weeks)
- Changes in NIH Toolbox fluid composite standard scores between sessions
- Quartiles of PRA and PLA loading across the season
- DWI average relative motion between sessions

R code for fractional anisotropy:

```
1mer(values ~ TrainingAge + BMI + Headgear + `TimeBtwScansWksSes1-2` +  `FluidCompSSchangeSes1-2` + QuartileLoadPLA + QuartileLoadPRA + `DWI_Relative_motionSes1-2` + (1 | PartID), data = fa_ss_nc_long)
```

Role of the funding team

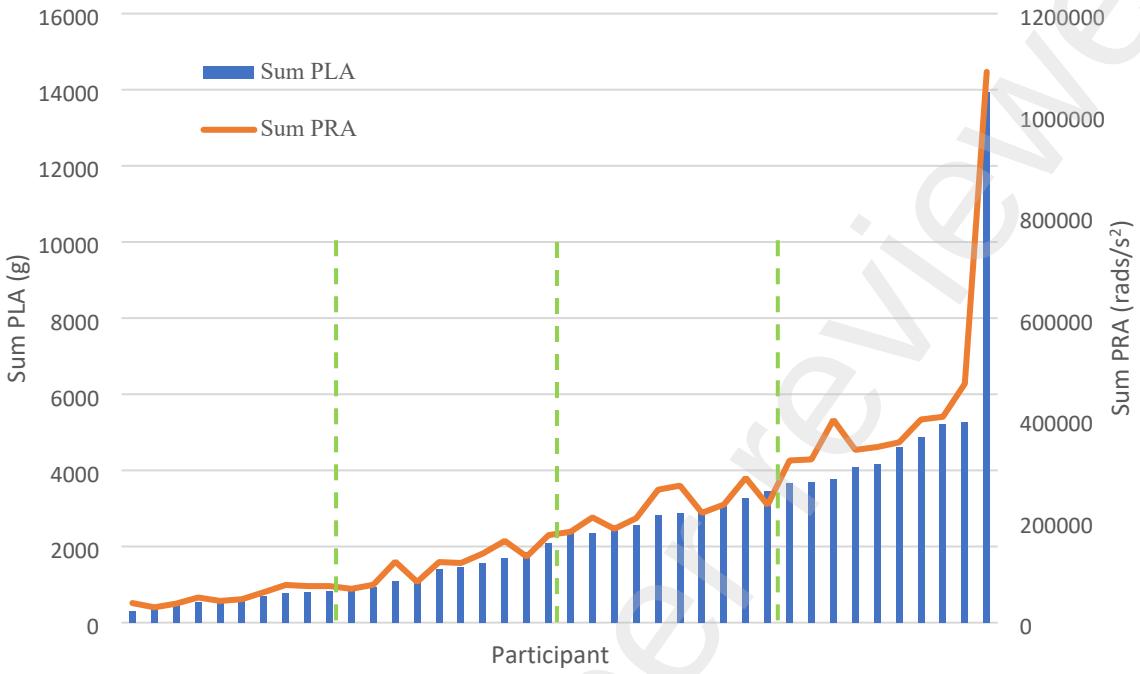
Study sponsors were not directly involved in collection, analysis, or interpretation of data, in the writing of this article, or in the decision to submit the paper for publication. One funder, Pacific Radiology, conducted the MRI scans for all participants. Pacific Radiology technicians followed the scan protocol provided by the medical physicist on the team but provided no interpretation of results. Raw MRI scans were housed on University of Otago servers, before processing took place at the New Zealand Brain Research Institute.

Results

Total cumulative loading for all players

The primary metric for testing this hypothesis was the analysis of processed MRI images from both structural and diffusion-based scans. Total cumulative loading (TCL) for PLA and PRA were calculated and organised by quartile. Overall means (±SD) for TCL were 2,538·65 g (±2,379·76) for PLA and 210,497·23 rads/s^2 (±189,748·35) for PRA. Distribution of TCL for all players can be seen in Figure 1. Regression analysis was performed to investigate the relationship between TCL experienced by the players and any changes noted in the MRI data. The concussion cohort (n = 5) was analysed separately from the non-concussion cohort (n = 34 (structural); n = 28 (diffusion)).

Figure 1. Total cumulative load distribution per player for PLA and PRA. Vertical green bars represent quartiles.



Neuro-cognitive results

Means and standard deviations for NIH Toolbox (fluid composite standard score, Table 1) and Reinvestment Scales (decision-specific and movement-specific, Table 2) are shown in the tables below. Means for the NIH Toolbox fluid composite standard score increased at post-season relative to pre-season, and increased in the concussion cohort relative to pre-season. Non-significant findings were noted in comparison of means for NIH Toolbox fluid composite standard scores for all participants who completed pre-post season testing (n = 40): t_{78} = 1.187, p = 0.239, d = -0.265. Of further note, post-concussive neuro-cognitive scores were more likely to increase relative to pre-season. Values for the reinvestment scales were more likely to increase than decrease. Scores for movement self-consciousness and conscious motor planning generally increased, while scores for decision reinvestment and decision rumination generally decreased.

Table 1. Means (±SD) for NIH Toolbox fluid composite standard scores.

Pre-season (n = 41)	Post-season (n = 40)	Pre-to post-season change	Concussion (n = 5)	Pre-season to concussion change
101.71 (±18.16)	106.93 (±18.49)	+4.88 (±12.79)	110.20 (±10.38)	+8.60 (±14.84)

Table 2. Means (±SD) for movement-specific and decision-specific reinvestment scales.

MSRS			DSRS		
	Pre- to post-season change	**Pre-season to concussion change**		**Pre- to post-season change**	**Pre-season to concussion change**
Factor 1: movement self-consciousness	0.60 (±6.11)	1.60 (±4.98)	Factor 1: decision reinvestment	-0.70 (±4.35)	0.20 (±4.55)
Factor 2: conscious motor processing	0.35 (±5.72)	0.60 (±3.13)	Factor 2: decision rumination	-0.36 (4.05)	-0.80 (±4.92)

T1-weighted structural results

T1-weighted images were analysed, focusing on sub-cortical and total grey matter (GM) volume, white matter (WM) volume, cortical thickness, and cortical surface area. Images for structural and diffusion scans are displayed in Fig 2. T1-weighted images were investigated for pre- to post-season change in the participants and whether this change correlated with TCL. Intracranial volume (ICV) was the same for both time points and was utilised as a co-variate for the cortical values. Means (±SD) were calculated for all participants (Table 3). Fixed effects multivariate results are summarised in Table 4. Results indicate no significant change in any of the measured metrics (volume, surface area, cortical thickness), from pre- to post-season, in the non-concussion cohort (n = 34). Results indicating non-significant structural change held when controlling for the primary variable of interest (TCL) in both summed PLA and summed PRA. Overall, results appear to show no change in neurological structure, as measured by T1-weighted scans, in this cohort of non-concussed male adolescent rugby players exposed to one season of play.

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Figure 2. Axial views of T1-weighted image (left), colour-coded diffusion map (FA in subject space, centre), and TBSS white matter skeleton (in MNI space, overlaid on the average cohort FA, right).

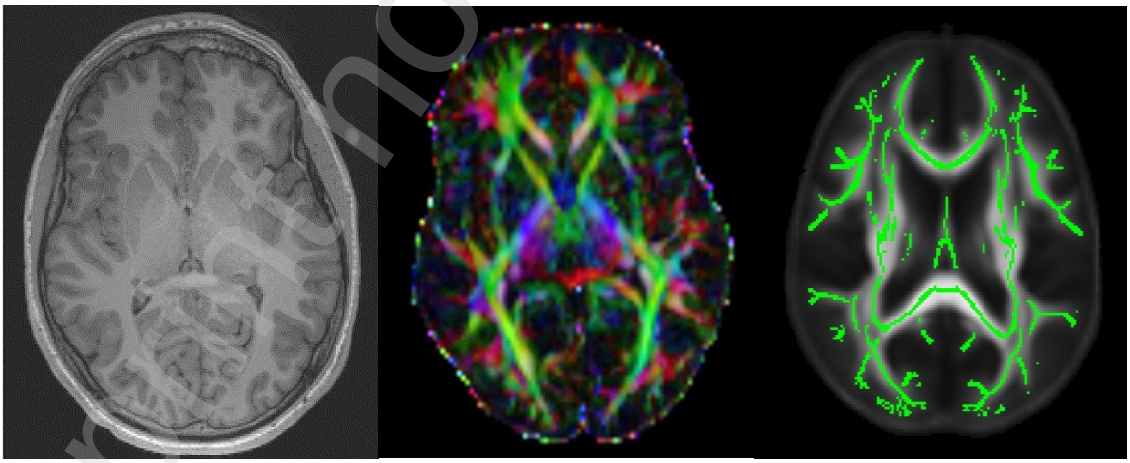

Table 3. Means (±SD) for structural values for non-concussion cohort (n = 34).

	Pre-season value	Post-season value	Change in value
Intracranial vol (mm^3)	1623607.63 (±136918.70)	1623607.63 (±136918.70)	--
Cerebral WM vol (mm^3)	455637.65 (±63973.49)	451138.24 (±77531.05)	-4499.41 (±31379.53)
Subcortical GM vol (mm^3)	70510.41 (±5128.30)	69904.85 (±5655.97)	-605.56 (±2079.91)
Total GM vol (mm^3)	771402.64 (±92043.56)	755388.91 (±108538.33)	-16013.72 (±54331.88)
Surface area R (mm^2)	95817.94 (±10269.59)	94909.32 (±11368.45)	-908.62 (±4238.26)
Thickness R (mm)	2.60 (±0.15)	2.56 (±0.19)	-0.04 (±0.15)
Surface area L (mm^2)	96408.58 (±10534.58)	95508.87 (±11818.55)	-899.71 (±4549.36)
Thickness L (mm)	2.61 (±0.14)	2.56 (±0.19)	-0.05 (±0.14)

vol: volume; WM: white matter; GM: grey matter; R: right; L; left; mm: millimetre

Table 4. Multivariate modelling of global structural MRI values.

	Variable	Estimate	Std. Error	df	*t* value	Pr(>|t|)
Cerebral WM volume	TCL sum PLA	3.832e+01	4.916e+01	3.100e+01	0.779	0.442
	TCL sum PRA	-3.830e-01	6.252e-01	3.100e+01	-0.613	0.545
Subcortical GM volume	TCL sum PLA	2.948e+00	3.745e+00	3.100e+01	0.787	0.437
	TCL sum PRA	-2.928e-02	4.763e-02	3.100e+01	-0.615	0.543
Total GM volume	TCL sum PLA	9.275e+01	6.418e+01	3.100e+01	1.445	0.158
	TCL sum PRA	-9.745e-01	8.163e-01	3.100e+01	-1.194	0.242
Surface area R	TCL sum PLA	1.054e+01	7.420e+00	3.100e+01	1.420	0.166
	TCL sum PRA	-1.190e-01	9.438e-02	3.100e+01	-1.261	0.217
Thickness R	TCL sum PLA	3.286e-05	1.079e-04	3.100e+01	0.305	0.763
	TCL sum PRA	-1.225e-07	1.372e-06	3.100e+01	-0.089	0.929
Surface area L	TCL sum PLA	10.8198	7.6581	3.100e+01	1.413	0.168
	TCL sum PRA	-0.1221	0.0974	3.100e+01	-1.253	0.219
Thickness L	TCL sum PLA	5.906e-05	1.079e-04	3.100e+01	0.547	0.588
	TCL sum PRA	-4.756e-07	1.372e-06	3.100e+01	-0.347	0.731

WM: white matter; GM: grey matter; R: right; L: left; TCL: total cumulative load; PLA: peak linear acceleration; PRA: peak rotational acceleration; df: degrees of freedom

Diffusion-weighted results

Region-of-interest (ROI) analysis using Tract-Based Spatial Statistics (TBSS) was employed. Pre- to post-season changes for the selected ROIs in fractional anisotropy (FA) and mean diffusivity (MD) were quantified for all participants. The primary variable of interest was TCL. Additional co-variates considered in the analysis were: training age, concussion history, body-mass index (BMI), headgear use, time between scans, change in pre- to post-season NIH Toolbox fluid composite standard scores, and average relative motion change between the scans.

Regression modelling was performed for all variables and two diffusion metrics (FA, MD) in the non-concussion cohort (n = 28). Means (±SD) in FA and MD for the five ROIs were calculated. Mean (±SD) relative motion for this cohort was 0.34mm (±0.05, Table 5). No participants were excluded due to excessive relative motion. Pre- to post-season FA values decreased in the CST and IFOF, and increased in the CC, SLF, and SS. No identifiable change was seen in pre- to post-season MD values. No significant relationships were found between any variables.

Table 5. Means (±SD) values for FA and MD for non-concussion cohort (n = 28).

		FA			MD (mm^2/s)		
		Pre-season	**Post-season**	**Change pre to post**	**Pre-season**	**Post-season**	**Change pre to post**
Region of interest	CC	0.72395 (±0.02552)	0.72616 (±0.01556)	0.00221 (±0.01509)	0.00078 (±0.00002)	0.00079 (±0.00003)	0.00000 (±0.00004)
	CST	0.61869 (±0.04112)	0.61265 (±0.04109)	-0.00604 (±0.02115)	0.00070 (±0.00002)	0.00070 (±0.00002)	0.00000 (±0.00002)
	IFOF	0.56589 (±0.04526)	0.56542 (±0.04315)	-0.00047 (±0.01743)	0.00078 (±0.00002)	0.00078 (±0.00002)	0.00000 (±0.00002)
	SLF	0.53675 (±0.02281)	0.53829 (±0.02250)	0.00154 (±0.01605)	0.00073 (±0.00002)	0.00073 (±0.00001)	0.00000 (±0.00001)
	SS	0.56673 (±0.02822)	0.57223 (±0.02074)	0.00549 (±0.01419)	0.00080 (±0.00002)	0.00080 (±0.00002)	0.00000 (±0.00001)

CC: corpus callosum, CST: corticospinal tract, IFOF: inferior fronto-occipital fasciculus, SLF: superior longitudinal fasciculus, SS: sagittal stratum, FA: fractional anisotropy, MD: mean diffusivity
Score decreases are highlighted in blue, increases in orange.

Results for average fractional anisotropy (FA) are shown in Table 6. Average FA is displayed in the table, but relationships were also non-significant for all ROIs selected for analysis (CC, CST, IFOF, SLF, and SS). Results indicate that, when controlling for variables listed in the table, no consistent significant change in FA or MD was noted across the participants, in most white matter (WM) tracts. Statistically significant FA increase was found in CST ~ quartile load PLA ($p = 0{\cdot}027$); FA decrease was found in CST ~ quartile load PRA ($p = 0{\cdot}027$) and splenium of CC ~ headgear ($p = 0{\cdot}031$). Statistically significant MD increase was found in SLF ~ time between scans ($p = 0{\cdot}038$); MD decrease was found in CST ~ headgear ($p = 0{\cdot}022$) and IFOF ~ headgear ($p = 0{\cdot}030$). Results would seem to suggest that, for the primary variable of interest, neither participating in rugby for one season, nor accruing higher cumulative loading within that season, influenced microstructural WM changes in this cohort of adolescent male rugby players.

Table 6. Linear mixed modelling of score changes in average fractional anisotropy.

	Estimate	Std. Error	df	*t* value	Pr(>|t|)
Training age	0.0004064	0.0011417	19.0000009	0.356	0.726
BMI	-0.0000666	0.0009769	19.0000063	-0.068	0.946
Headgear	-0.0047875	0.0067778	19.0000022	-0.706	0.489
Time btw scans (weeks)	0.0005402	0.0010347	18.9999981	0.522	0.608
Fluid comp SS change	0.0002302	0.0002096	18.9999997	1.098	0.286
Quartile load (PLA, g)	-0.0031370	0.0153461	19.0000037	-0.204	0.840
Quartile load (PRA, rads/s^2)	0.0053285	0.0151399	19.0000031	0.352	0.729
DWI relative motion	0.0139122	0.0755552	19.0000038	0.184	0.856

BMI: body mass index; SS: standard score; PLA: peak linear acceleration; PRA: peak rotational acceleration; DWI: diffusion weighted imaging, df: degrees of freedom; g: gravitational force; rads/s^2: radians per second squared

Discussion

The intersection of head acceleration event magnitude and neurological change

A key methodological innovation embedded in this study was the use of a comprehensive MRI protocol to assess potential neurological change across one season of playing rugby. The primary MRI scan used to detect potential change was diffusion tensor imaging (DTI), which has been extensively used in both the sports-related concussion (SRC)[24] and the repeated head impact exposure (RHIE) populations.[22] This methodology allowed for the analysis of diffusion-based white matter changes and how they may intersect with cumulative HAE burden. This approach has been used with youth American football players,[17, 18] but not with youth rugby players. As such, the study could provide vital baseline data on the effects of one season of rugby exposure on the structure and function of the teenage brain.

The primary variable of interest used in the analysis of the MRI data was total cumulative loading (TCL). This was a simple metric which allowed clear visualisation of each player's head impact burden across one season, summing their PLA and PRA separately. Such measures have been used in rugby studies on adult players[25] but in the diffusion-based studies included in the literature review the measures most often used were total incidence count or measures such as risk weighted exposure (RWE) and head impact telemetry severity profile (HIT_{SP}).[22] Combining PLA and PRA into a single metric has been described as methodologically inappropriate, but single measures of impact burden can be descriptively useful.[12] Cumulative magnitude of this nature allowed the researchers to organise participants by low and high magnitude groups, while detailed pre- and during season record keeping allowed multiple other co-variates, such as training age, body mass index (BMI), concussion history, and neuro-cognitive testing scores (NIH Toolbox fluid composite standard scores) to be considered. The significance of these results follows.

Total cumulative loading (TCL) was calculated for all players. Compared to rugby studies by King et al. the average cumulative values calculated were significantly less (mean PLA 12,029 g; mean PRA 2,101,028 rads/s^2) in a study of a men's amateur team[25] and also less (mean PLA 3,411 g; mean PRA 595,624 rads/s^2) in a study of an under-9 team.[26] Our results were also significantly lower than those reported in high school football (mean

PLA 16,746 g; mean PRA 1,090,698 rads/s^2).[12] Although there was some variation within the quartiles in PLA versus PRA, the same players were represented in each quartile, suggesting a linear relationship between increased PLA burden and increased PRA burden. Whether players ended up in the high load or low load group was based more on variables such as contact disposition, exposure time on the field, and time-loss due to injuries and concussion.

Neuro-cognitive and reinvestment scale results

Non-significant results were found from analysis of both NIH Toolbox fluid composite standard scores and reinvestment scales. These season-based, pre- to post-season non-significant clinical findings reflect other rugby-based studies, conducted at the college and adult level, which similarly showed no significant change in neuro-cognitive functioning based on the tests used (including ImPACT and SCAT assessments).[4, 27, 28] In high school American football, DTI-based changes (such as decreased FA) have been correlated with decreased verbal and visual memory over one season's exposure.[18, 29] This may be reflective of both higher exposure rates within football over rugby, and the relationship between neurological integrity and age of first exposure.[30, 31] Underpinning the need for longitudinal studies, research into university and adult contact sport athletes have associated higher cumulative load with decreased cognitive scores relative to lower FA,[32, 33] whereas other college-level studies found no change.[34, 35] There may be a higher likelihood of neuro-cognitive change related to diffusion-weighted measures in older athletes.

Interpretation of T1-weighted results

Results for structural imaging (T1-weighted images) similarly showed non-significant pre- to post-season changes in the non-concussion cohort based on one season of rugby exposure. This is reflective of findings in multiple diffusion-based studies involving the repeated head impact exposure (RHIE) population, in youth contact sport and/or rugby.[4, 17, 18, 27-29] No significant structural differences in athletes, either within- or between-subject, were reported in any of these studies. Global metrics such as white matter (WM) volume have also been cited in the literature, with Brett et al. (2021) concluding change in gross WM volumes was not associated with higher cumulative load or concussion history.[36] This also reflects our findings.

Interpretation of diffusion-weighted results

Results for diffusion tensor imaging (DTI) in the non-concussion cohort showed non-significant findings in the included metrics (FA and MD) in most white matter (WM) tracts. Although some borderline statistically significant findings were reported in some regions-of-interest (ROIs), these did not follow a consistent pattern and may be considered to be outliers. In contrast to our findings, a systematic review on the use of DTI in the RHIE population found the majority of included studies reported change in WM integrity, most often reported as decreased FA.[22] Three rugby-based studies reported DTI-based changes over the course of one or more season, but all were conducted on adult players.[4, 27, 37] Studies focusing on older contact sport athletes may result in a higher likelihood of detecting DTI-based changes.

Other region-of-interest (ROI) based studies have found changes in diffusion-based metrics. Schranz et al. (2018) reported small FA increases pre- to post-season in university level female rugby players,[28] but this was not reported as statistically significant. McAllister et al. reported significant differences in MD (in the corpus callosum (CC)) and in FA (in the amygdala) of college contact sport athletes.[33] Most meaningfully for our study, Bahrami et al. (2016) reported correlations between risk-weighted exposure (RWE_{CP}) and decreased FA in the right superior longitudinal fasciculus (SLF) and the left inferior frontal fasciculus (IFOF) in youth football players (aged 8-13 years).[17] Although statistically non-significant, our findings indicated MD changes in the IFOF related to headgear use, and in the SLF related to time between scans. The most analogous measure to RWE, cumulative loading, was associated with FA changes in the corticospinal tract (CST).

Overall, our diffusion-based findings for one season of exposure do not correlate well with published research, highlighting the need for further research into adolescent rugby players. Most published studies using DTI in the RHIE population have reported significant changes in FA and/or MD based on one season of play. Significant results published by other authors may also reflect multiple other variables, such as the type of sport played, age of participants, gender, concussion history, study design (within-subject versus between-subject), and type of analysis used (ROI-based versus voxel-based). Comparison of rugby-playing cohorts versus non-contact sport controls may further elucidate findings.

Summary and future research

In this cohort of adolescent rugby players, exposure to one season of play does not appear to result in measurable macro or microstructural neurological change. Results from pre- and post-season MRI scans indicated a non-significant relationship between head acceleration event exposure in one season of rugby and changes in white matter microstructure, including for those players exposed to a higher level of impact magnitude. This non-significance held when controlling for variables such as training age, headgear use, and body mass index. Results from the study indicated non-significant findings relating to both structural and diffusion-based MRI scans. It should be emphasised these results pertain to one season's exposure, in the absence of concussion, in a cohort of healthy rugby players aged 14-16. Longitudinal studies could provide key data on longer term brain health, the ability of any microstructural white matter damage to repair itself in the off-season, and how this interacts with cumulative loading. We wish for these findings to add value to the field and to provide a key starting and continuation point for researchers to quantify neurological change in young rugby players, and for such data to potentially play a role in making rugby safer.

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Author Contributions

SH, NS, and KM collected and processed field data. NK, DS, and AS provided subject matter expertise in engineering. NK, MH, KA, and ND provided supervisory support and oversight. RM provided subject matter expertise for reinvestment scales. TM provided subject matter expertise for MRI acquisition, pre-and post-season processing, and results interpretation. AB provided statistical analysis and results interpretation. TA provided medical oversight for MRI procedures. DS provided subject matter expertise for NIH Toolbox. AC provided product design expertise for field data collection. SH wrote and revised the manuscript. ND proofread the manuscript and serves as PI for the research group.

Competing Interest Declarations

The authors declare no competing interests.

Research in context

Evidence before this study

Rugby union is a popular contact sport where high impact collisions frequently occur. Increasing concern exists regarding the long-term brain health of those playing the game. Repeated sub-concussive collisions may compromise rugby players' neurological integrity. We searched CINAHL, PubMed, Google Scholar, and Scopus using "adolescent rugby", "head acceleration event", "sports-related concussion", "repeated head impact exposure", "peak linear acceleration", "peak rotational acceleration", "neuro-cognitive testing", and "diffusion tensor imaging" for articles published in English. The search confirmed a lack of published findings on head impact exposure, combined with MRI analysis, in adolescent rugby players. The majority of research in the field had taken place with male adult players, often with retrospective study designs.

Added value of this study

Presently little data exist regarding the brain health of adolescent rugby players, even though adolescents make up the majority of players worldwide. Here we show exposure to one season of rugby for healthy adolescent male players does not appear to directly result in neurological compromise. We found pre- and post-season structural and diffusion-based MRI scans, and neuro-cognitive assessments, did not show significant changes in this cohort of athletes. These results held when controlling for the cumulative head impact burden experienced by the players. There was non-significant change in values of diffusion-based MRI metrics (fractional anisotropy and mean diffusivity) in defined white matter tracts.

Implications of all the available evidence

As a world-first study, we anticipate our findings to be a sound starting point in quantifying how the incidence and magnitude of head acceleration events may impact neurological integrity in young rugby players longitudinally. These findings could be used to inform best practice for rugby coaches, and provide key educational strategies to rugby stakeholders in order to protect the brain health of its players.

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