

Exploring the link between acute symptom changes and repetitive head impacts in boxing sparring

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Research Article

Keywords: Concussion, Recovery, Combat sports, Neurocognitive assessment, Longitudinal monitoring

Posted Date: September 26th, 2025

DOI: <https://doi.org/10.21203/rs.3.rs-7715343/v1>

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Additional Declarations: The authors declare no competing interests.

Version of Record: A version of this preprint was published at Journal of Science and Medicine in Sport on January 31st, 2026. See the published version at <https://doi.org/10.1016/j.jsams.2026.01.018>.

Abstract

Objectives: The acute dose-response relationship between head impact exposure and the development of concussion remains elusive. Previous research has suggested that individual-specific approaches and the cumulative effects of head impacts should be investigated. This study aimed to monitor acute changes in concussion-related symptoms resulting from boxing sparring, and assess how they relate to head impact exposure.

Design: Observational cohort study

Methods: Seven competitive boxers participated in this exploratory study. Symptoms of concussion were captured via the SCAT5 symptom scale upon recruitment, immediately before, immediately after, and 48 hours after a sparring session. Head impact data were collected with instrumented mouthguards. Each session's mean, median, highest-magnitude impact, cumulative sum, and time-weighted exposure for several injury severity metrics were qualitatively analyzed with respect to acute changes in symptom score.

Results: Group-based analyses did not show any differences in symptom scores pre- and post-sparring; Three participants reported symptom changes in four instances (25% of all sessions). There was no strong association between session-specific exposure and change in symptoms, but time-weighted exposure metrics showed a better alignment than other metrics.

Conclusions: Symptoms may have resulted from the combination of several factors, namely a high density of head impacts, several above-average-magnitude impacts, or pre-existing dispositions. Our results support the concept of individual-specific analyses, as this approach allowed us to identify patterns emerging from a few athletes that were not visible in cohort-averaged summaries. Understanding the relationships between head impact exposure, individual pre-disposition, and signs and symptoms of concussion is essential to designing risk reduction strategies.

Introduction

During sparring, a common form of boxing training typically performed multiple times a week (1, 2), competitive boxers can sustain between 2 and 8 impacts to the head per minute (3–5). This rate exceeds that of American football (0.1 to 0.5 impacts per minute in games or full-contact drills)(6) and the average peak linear acceleration (PLA) measured by head impact sensors during sparring is as high as other contact sports (14–30 g)(3–5). Neuropsychological and cognitive testing conducted on boxers before and after a sparring session show impairments in delayed and verbal memory (3, 4) and in reaction time (4). The number and intensity of head strikes sustained as part of normal training may therefore lead to brain trauma, which may in turn affect both the athlete's immediate performance and the risk of subsequent or more severe injury. The development of brain trauma, which refers to the disruption of brain function and/or the onset of neurological signs and symptoms, such as a concussion, has not been assessed in the context of sparring. There is limited information about how participation in sparring affects an athlete's neurologic and cognitive status during the training week.

Combat sport athletes often avoid medical care after a concussion and return to contact quickly (1, 2, 7). These behaviors stem from a limited understanding of symptoms and consequences (2, 7) and an unwillingness to report their symptoms (7). Therefore, there is a need for tools that allow an objective and accurate monitoring of an athlete's state with respect to brain trauma. Particularly, instrumented mouthguards (IMGs) allow the investigation of the possible dose-response relationship between repetitive head impacts (RHIs) and the signs and symptoms of concussion. If such a relationship exists, it could be a valuable tool to manage an athlete's impact load, enabling individualized informed training and recovery practices aimed at improving athlete performance and health in the short and long terms.

The dose-response link between RHIs and brain trauma remains unclear due to individual variability and metric limitations. Previously, specific head acceleration events (HAEs) have been linked to concussions when an athlete immediately displayed signs of an altered state (8). Concussions were found to occur across a wide range of HAE magnitudes (9, 10) and often attributed to the highest-magnitude impact recorded for an individual (6, 10). These findings suggest that individual predispositions and risk factors (e.g., history of brain trauma, genetics) may affect individual tolerance, resulting in varied outcomes from similar HAEs. In addition, there are several reports of concussions with delayed-onset symptoms, where a single impact could not be identified as the sole cause of injury (11, 12). In such cases, athletes sustained twice as many RHIs on days with injury than on days without injury (12). Studies have also measured acute changes in concussion-related symptoms (13), neurocognitive performance (3), and blood biomarkers (14) following a single full-contact session without a concussion diagnosis. Collectively, these studies show that brain trauma may result from a combination of low magnitude and/or moderate-to-high magnitude RHIs, and that individual-specific analytical approaches may be more appropriate (9–11, 13).

Identifying metrics that have utility in representing RHI exposure to better understand the effects on brain health is challenging. Taking the magnitude of the highest recorded HAE reflects only one impact, ignoring the number of lower-magnitude events. On the other hand, the cumulative sum of all impacts' magnitude cannot differentiate between one large event and a multitude of smaller ones. Cumulative metrics assume linear accumulation without accounting for recovery, which newer time-weighted models address (15–17). There is a strong need to investigate metrics that account for the role of frequency of RHIs and recovery on the occurrence of concussion-related symptoms.

The goal of this pilot observational study was to evaluate whether athletes experience acute changes in concussion-related symptoms after a session of boxing sparring, and how these symptoms evolve over the subsequent 48 hours. The study also used an individual-specific approach to examine RHI exposure, focusing on video-verified high-quality IMG measurements, and changes in self-reported symptoms. As the sample size was too small to formally test the hypothesis that individual-specific exposure was associated with acute changes in self-reported symptoms, we qualitatively examined whether highest-severity impacts or cumulative exposure could be linked to such changes.

Methods

Overview

This study explores acute self-reported symptoms of concussion and RHI exposure data resulting from boxing sparring. Data were collected over one to three sparring sessions for seven competitive boxers (4 females, 3 males, mean age 23 ± 3 years) sparring against various opponents. Concussion symptoms were recorded at recruitment, pre- and post-sparring, and 48 hours later. RHI exposure was quantified from video-verified HAEs recorded by iMGs. Head impact data collection and processing are described elsewhere (5, 18). This study was approved by the Auckland University of Technology Ethical Committee (Approval # 20/153) and all participants provided informed consent.

Self-reported concussion history and symptoms

Each participant completed a questionnaire about their experience in combat sports, training habits (7), and concussion history. Specifically, Participants reported instances of knockouts, loss of consciousness, or being declared unfit to continue by themselves or others, and noted whether these led to diagnosed concussions. Subsequently, the following definition of concussion was adapted (19, 20) and provided to the participants: *"Some people have the misconception that concussions only happen when you black out after a hit to the head or when the symptoms last for a while. In reality, a concussion has occurred anytime you have had a blow to the head that caused you to have symptoms for any amount of time. These include: blurred or double vision, seeing stars, sensitivity to light or noise, headache, dizziness or balance problems, nausea, vomiting, trouble sleeping, fatigue, confusion, difficulty remembering, difficulty concentrating, or loss of consciousness. Whenever anyone is being 'rocked', 'wobbled' or 'fazed', that too is a concussion."* After being given this definition, participants were asked again to recall how many concussions they may have sustained while fighting or sparring. They were asked how often they receive light to heavy head strikes during sparring (2), and how frequently they experience heavy blows (*"a blow to the head that 'rocks' or 'fazes' them, or leaves them feeling dizzy, off-balanced or with blurry vision, even for a short time?"*). Participants also reported how frequently they sustain concussion-related symptoms within 24 hours of sparring using the symptom scale of the Sport Concussion Assessment Tool (SCAT5).

Acute symptoms from sparring

Most athletes sparred twice weekly, with live sparring on Saturday morning (no instructions from the coaching staff), and technical sparring on Wednesday evening (focusing on specific actions each round). Data collection occurred on Saturday mornings, except for one Wednesday evening session. Sessions lasted 40–60 minutes with multiple 3-minute rounds and 30-second breaks. Participants typically sparred at a self-selected intensity against an opponent who may or may not have been part of the study. They completed questionnaires before their warm-up (PRE, mean: 30 ± 10 minutes before the first round, range: 11–45), after their cool-down (POST, mean: 32 ± 24 minutes after the last round, range: 10–111), and approximately 48 hours after (mean 53 ± 7 hours, range 47–69). Each questionnaire asked for the intensity (0 – None, to 6 – Severe) for the 21 or 22 symptoms of the SCAT5 ("Trouble falling asleep" was excluded at PRE and POST). The 48-hour questionnaire also asked the participants to recall how they felt at their worst (WOR), defined as the time when they experienced symptoms at the highest intensity. As a retrospective subjective recall, this may be interpreted differently by each participant, and some might have considered how they felt immediately after sparring, others how they felt later that day. POST questionnaires also asked about heavy blows and provided video-identifiable details. The participants completed all questionnaires with no intervention from the researcher, either on a tablet at the gym or on their own device.

The total number of self-reported symptoms was calculated, as well as the total symptoms severity score (TSSS, the sum of all symptoms' severity). Symptoms were grouped into clinical profiles: ocular-vestibular (Dizziness, Blurred vision, Balance problems, Nausea or vomiting), cognitive-fatigue (Feeling slowed down, Feeling like "in a fog", "Don't feel right", Difficulty concentrating, Difficulty remembering, Fatigue or low energy, Confusion, Drowsiness), post-traumatic migraine (Headache, "Pressure in head", Sensitivity to light, Sensitivity to noise) and anxiety/mood (More emotional, Irritability, Sadness, Nervous or Anxious), except for neck pain and sleep-related issues as per their role as modifiers in concussion assessments (21). We calculated the PRE-POST, PRE-WOR, and PRE-48H changes in the number of symptoms and TSSS. By comparing post- to pre-sparring scores rather than comparing to a baseline report or normative data, we aimed to account for pre-existing symptoms on the day of data collection, thus reducing both within- and between-subject variability (22).

Head impact kinematics

Participants were equipped with Prevent Biometrics Hybrid iMGs (V1, boil-and-bite, Prevent Biometrics Inc., Edina, MN), instrumented with a triaxial linear accelerometer and a triaxial angular rate sensor sampled at 3200 Hz. The sensors recorded a 50-ms event (10 ms pre- and 40 ms post-trigger) when the linear acceleration reached ± 10 g on any axis. We obtained the raw time series data (unfiltered at the sensor location) and the processed time series data (filtered and transformed to the head's center of gravity by Prevent Biometrics' proprietary algorithms) for each HAE, independent of the Prevent Biometrics' classification as true or false positive.

Sparring was filmed using two or three cameras (GoPro Hero 3 + to Hero 7 Black, GoPro, Inc., San Mateo, CA, USA), set up with a frame rate of 60 fps, a shutter speed of $1/120$ s, and a resolution of 1080p. An iMG calibration impact was performed at the start of the session in the view of one camera to align the HAEs' timestamps to the video timeline. Acceleration events recorded outside of sparring rounds were excluded, and the remaining HAEs were verified on video with Nacsport Elite 6.0.0 (Nacsport, Canary Islands, Spain) by one rater (ELF)(18). Raw data were screened for signal quality using previously established criteria (5). Only high-quality HAEs were included for further analysis (482 out of 959, or 50%), independent of their magnitude or the manufacturer-defined quality class. For every included HAE, nine kinematic metrics were calculated from the processed 3DOF time series data (Table 1). All processing steps were performed in MatLab (R2019a, MathWorks, Natick, MA).

Table 1

Information on the kinematic-based metrics calculated in this study, with reports of magnitude for concussive and non-concussive head acceleration

Metric	General information and calculation method
Peak linear acceleration (PLA)	Maximum value of the resultant linear acceleration time series
Peak angular acceleration (PAA)	Maximum value of the resultant angular acceleration time series
Peak angular velocity (PAV)	Maximum value of the resultant AV time series
Maximal change in angular velocity ($\Delta\omega_{\max}$)	Largest excursion from the AV at the onset of impact. Proposed as a predictor for head injury and brain strains (23, 24). $\Delta\omega_{\max} = \max \left(\sqrt{\sum (\omega_i - \omega_{i0})^2} \right)$ Where ω_i is the AV over time and ω_{i0} the velocity at the onset of impact, measured about the $i = x$-, y- and z-axes. The onset of impact corresponds to the sample that triggered the recording, i.e., when any axis of the raw LA reached ± 10 g.
Head Injury Criterion (HIC)	Modification of the Gadd Severity Index (GSI) focusing on the main LA pulse (25). The GSI originated from the Wayne State Tolerance Curve developed from animal and cadaver research and used to quantify severe brain injury. $HIC = \left[\int_{t_1}^{t_2} a(t) dt \right]^{2.5} (t_2 - t_1)$ Where $a(t)$ is the resultant LA, and t_1 and t_2 are two time points chosen to maximise the value of HIC. Here, we report the HIC_{36}, where $(t_2 - t_1)$ is bounded to ≤ 36 ms.
Rotational Injury Criterion (RIC)	Angular equivalent of the HIC and proposed as a predictor for brain strains (26) $RIC = \left[\int_{t_1}^{t_2} \alpha(t) dt \right]^{2.5} (t_2 - t_1)$ Where $\alpha(t)$ is the resultant AA, and t_1 and t_2 are two time points chosen to maximise the value of RIC. Here, we report the RIC_{36}, where $(t_2 - t_1)$ is bounded to ≤ 36 ms.
Brain Injury Criterion (BrIC)	Based on 3DOF AV, developed from numerical studies and proposed as a predictor for brain strains (27). $BrIC = \sqrt{\sum \left(\frac{\max(\omega_i - \omega_{i0})}{66.3} \right)^2}$ Where ω_i are the AV over time, ω_{i0} the AV at the onset of impact measured about the $i = x$-, y-, and z-axes. We used the AV changes from the impact rather than the absolute maximum velocities to account for the fact that BrIC was developed using head impacts where the initial AV was at or near zero. The onset of impact corresponded to the sample that triggered the recording. ω_{iC} are critical AV and are equal to 66.3, 41.5 rad.s^{-1} about the x-, y-, and z-axes, respectively (27).
Generalized Model for Brain Injury Threshold (GAMBIT)	Developed to consider the combined effects of LA and AA on injury risks (28). The simplified equation was used: $GAMBIT = \frac{a_{peak} \alpha_{peak}}{250 \times 10,000}$ Where a_{peak} and α_{peak} are the peak LA and peak AA, respectively. The 250 g and 10,000 rad.s^{-2} denominators are critical values chosen as thresholds.
Head Impact Power (HIP)	Developed from laboratory reconstructions of American football impacts (29). Represents the head's rate of change of linear and angular kinetic energy. $HIP = \max(m a_x(t) \dot{a}_x(t) + m a_y(t) \dot{a}_y(t) + m a_z(t) \dot{a}_z(t) + I_{xx} \alpha_x(t) \dot{\alpha}_x(t) + I_{yy} \alpha_y(t) \dot{\alpha}_y(t) + I_{zz} \alpha_z(t) \dot{\alpha}_z(t))$ Where m is the mass of the head fixed at 4.5 kg; a_x, a_y, and a_z are the LA (in m.s^{-2}) along the x-, y-, and z-axes, respectively; I_{xx}, I_{yy}, I_{zz} are moments of inertia about the x-, y-, and z-axes, fixed at 0.016, 0.024 and 0.022 N.m.s^2, respectively; and α_x, α_y, α_z are the AV about the x-, y-, and z-axes, respectively.

iMG: instrumented mouthguard; LA: linear acceleration; AV: angular velocity; AA: angular acceleration; 3DOF: 3-degree of freedom; AIS: Abbreviated Injury Scale

Metric	General information and calculation method
	respectively. The LA and AV were integrated from the onset of impact (the sample that triggered the recording, to assume initial velocities of rad.s^{-1}) to the end of the recording, i.e., over 40 ms.
iMG: instrumented mouthguard; LA: linear acceleration; AV: angular velocity; AA: angular acceleration; 3DOF: 3-degree of freedom; AIS: Abbreviated Injury Sc	
Analysis	

Symptom scores and TSSS at POST, WOR, and 48H were compared to PRE using Friedman’s test with Dunn-Sidák corrections (alpha: 0.05). Sessions were treated as independent due to small sample size. A qualitative analysis is provided for the athlete-sessions where an arbitrary increase in TSSS ≥ 5 was reported between PRE and any of the post-sparring time points.

To investigate the association between RHIs and symptoms, the mean and standard deviation (SD), median and inter-quartile range (IQR), maximum value, cumulative sum (the sum of the magnitude of all HAEs included in an athlete-session), and time-weighted exposure were calculated by athlete-session for each of the nine kinematic metrics separately. The time-weighted exposure was originally defined by Merchant-Borna et al. as the cumulative helmet-based impact measure weighted for time between hits (C-HIM_{TBH}) and used by Puvvada et al. as the TWE_{inv} (16, 17). It accounts for both the magnitude and temporal proximity of head impacts, based on the assumption that the effect of a head impact may be amplified when it occurs in close succession to another impact. TWE_{inv} is calculated as the sum of each head impact magnitude X_C , combined with a weighted contribution from prior impacts occurring within a defined time window (Equation below).

$$TWE_{inv} = \sum_{i=1}^n \left[X_c + \sum_{j=1}^m \left[\frac{b}{T_C - T_P} * X_P \right] \right]$$

For each current impact i , the contribution of prior impacts j is scaled inversely by the time elapsed between the current impact timestamp T_C and each prior impact timestamp T_P , expressed in hours. Specifically, the contribution of each prior impact is computed as the product of the prior impact’s magnitude X_P and a scaling factor $\frac{b}{T_C - T_P}$, where b is a time-weighting constant (here, one hour). The outer summation accumulates the total time-weighted exposure across all n head impacts, while the inner summation includes all m impacts that occurred prior to the current impact within the selected time window (here, from the first included impact of the session).

The association between each metric was investigated using Pearson’s correlations after verifying that all data were normally distributed (all $p > 0.04$), and the p - and R^2 values were reported as heatmaps. A Bonferroni correction factor was applied and brought the significance level to $4.8\text{e-}5$. For each metric, the mean, median, maximum value, cumulative sum, and TWE_{inv} of each athlete-session were also normalized over the participant’s average value calculated over two or three athlete-sessions, and plotted to further explore the collinearity between the metrics.

Nine control charts (one for each metric) were established for each athlete-session. While control charts traditionally represent count data to monitor the incidence of illness or injury (30), we used them to show the magnitude of all HAEs over time and highlight outliers. For each participant, we calculated the mean and SD of all HAEs sustained over all available sessions and plotted the participant’s mean as well as two upper-control limits, i.e., one and two SDs above the mean (31). The HAEs with a magnitude consistently over the upper-control limits across all nine metrics (i.e., outliers) were identified. Such events may be particularly relevant as they reflect a higher-than-average severity on metrics that represent different aspects of head motion (e.g., linear vs. angular, resultant vs. individual components, see Table 1). We qualitatively analyzed such events relative to the presence or absence of self-reported symptoms.

Results

Self-reported history

Initially, none of the seven participants reported diagnosed concussions, despite having been declared unfit to continue fighting/sparring (Table 2). After receiving the definition, they estimated a mean of 3.4 ± 4.0 concussions. Sparring was typically moderate to competition-level intensity, with head strikes of moderate intensity. When reflecting on sparring sessions prior to this study, some participants reported more than 10 symptoms “Sometimes” experienced within 24 hours of sparring (Table 2). The most common symptoms reported were “Headache”, “Neck pain”, “Fatigue or low energy”, and “Trouble falling asleep”, with the latter three sometimes persisting for longer than 24 hours.

Table 2

Participants' details, self-reported history of concussion, and frequency of symptoms experienced within 24 hours of prior sparring sessions, collected via questionnaire upon recruitment.

Participant ID	Sex	Age	Years of boxing	KO or LOC ^{a,b}	Declared unfit ^{a,b}	Diagnosed concussions ^{a,b}	Estimated concussions ^{a,c}	Frequency of heavy blows	Number of symptoms (scale of 0 to 22)	
									"Sometimes"	"About half the time"
A	F	22	5	0/0	1/3	0/0	4/4	Sometimes	11	3
B	F	29	3	0/0	0/0	0/0	0/3	Sometimes	11	0
C	F	22	5	0/0	0/0	0/0	0/0	Never	6	0
D	F	23	1	0/0	0/0	0/0	0/1	Sometimes	13	1
E	M	20	2	-/0	-/1	-/0	-/>10	Sometimes	18	1
F	M	23	8	0/0	0/0	0/0	1/1	Never	1	0
G	M	24	6	0/0	0/0	0/0	0/0	Never	0	0
Mean ± SD		23.3 ± 2.8	4.3 ± 2.4	0/0	0.1 ± 0.4 / 0.6 ± 1.1	0/0	0.7 ± 1.5 / 2.7 ± 3.5		8.6 ± 6.6	0.7 ± 1.1
F: Female, M: Male, KO: knock-out, LOC: loss of consciousness										

^a The numbers of KO, LOC, unfit decision, and concussions are reported as: from fighting / from sparring. Participant E had no fighting experience, therefore only sparring-related numbers are reported.

^b KO/LOC, Declared unfit, and Diagnosed concussion were reported by the participants before being given the definition of concussion.

^c Estimated concussions were reported after being given the definition of concussion.

Sparring-related symptoms

Sixteen athlete-sessions were included (one to three per athlete), with an average number of 9 ± 2 rounds per session (range: 6 to 12), or an average time sparring of 27 ± 7 minutes (18 to 39) (Table 3). Overall, the number of symptoms and TSSS were significantly lower at 48H than at PRE (number: $F_{(3)} = 10.67$, $p = 0.021$; TSSS: $F_{(3)} = 9.07$, $p = 0.028$) (Table 3). No other difference was found. Three participants on four occasions (25% of all athlete-sessions) reported experiencing an increase in TSSS ≥ 5 between PRE and any post-sparring timepoint (Table 3). In three cases, TSSS followed a typical exposure-recovery pattern, increasing from PRE to POST (mean increase of $+14.3 \pm 2.3$), then decreasing from POST to WOR (-9.7 ± 8.6) and from WOR to 48H (-8.7 ± 1.2) (Fig. 1, Supplemental Fig. 1). A decrease from POST to WOR may indicate that boxers started recovering quickly after responding to the POST questionnaire. In the fourth case, the TSSS decreased immediately after sparring (from 7 to 1 at POST), then increased a few hours later (1 to 12 at WOR) before returning to baseline (12 to 7 at 48H). The cognitive-fatigue symptoms cluster was affected in all four instances. In the five instances where symptoms were reported at 48H, participants felt at their worst on average 5 ± 1.5 hours after the end of the sparring session (range 2.7–6.5 hours).

Table 3
Exposure time, high-quality head impacts, number of symptoms and total severity scores for each athlete-session.

Athlete-session	Exposure (minutes)	Head impacts	Number of symptoms				Total symptoms severity score			
		N	PRE	POST	WOR	48H	PRE	POST	WOR	48H
Participant A										
Session 1	30	44	1	0	0	0	1	0	0	0
Participant B										
Session 1*	30	24	9	13 η	4 $\downarrow$	1 $\downarrow$	16	29 η	10 $\downarrow$	2 $\downarrow$
Session 2	27	6	3	4	2	1	5	4	4	1
Session 3	38.5	10	5	3	0 $\downarrow$	0 $\downarrow$	7	3	0 $\downarrow$	0 $\downarrow$
Participant C										
Session 1	33	48	1	1	0	0	2	1	0	0
Session 2	24	33	0	0	0	0	0	0	0	0
Session 3	18	16	0	0	0	0	0	0	0	0
Participant D										
Session 1	30	21	1	0	0	0	1	0	0	0
Session 2*	18	21	3	13 η	7 η	4	5	22 η	14 η	6
Session 3*	30	37	6	1 $\downarrow$	5	5	7	1 $\downarrow$	12 η	7
Participant E										
Session 1*	27	28	1	8 η	6 η	2	1	14 η	12 η	2
Participant F										
Session 1	24	58	0	0	0	0	0	0	0	0
Session 2	36	49	0	0	0	0	0	0	0	0
Session 3	21	6	0	0	0	0	0	0	0	0
Participant G										
Session 1	18	62	1	0	0	0	1	0	0	0
Session 2	35	19	2	0	0	0	2	0	0	0
Median [IQR]	28 [23–31]	26 [18–45]	1 [0–3]	0 [0–3]	0 [0–3]	0 [0–1]	1 [0–5]	0 [0–3]	0 [0–6]	0 [0–1]
Difference with PRE, p-value				0.97	0.16	0.02	0.85		0.34	0.03

PRE: pre-sparring, POST: immediately post-sparring, WOR: self-reported worst time where the participant experienced symptoms at the highest intensity, 48H: approximately 48 hours post-sparring, IQR: inter-quartile range.

η / $\downarrow$ The arrows indicate an increase or decrease of POST, WOR, or 48H relative to PRE by 3 symptoms or a total symptoms severity score of 5. The difference between PRE and POST, WOR, or 48H was assessed with a Friedman's test and the p-values reported were generated by post-hoc tests with Dunn-Sidak correction; significant differences are highlighted in bold.

* indicates the sessions where the participant reported an increase in total symptom severity score ≥ 5 between pre-sparring and any time point post-sparring.

Kinematics and changes in symptoms

A total of 482 high-quality HAEs were included (50% of all video-verified events), with 6 to 62 HAEs per athlete-session (mean: 30.1 ± 17.9 per session, or 1.2 ± 0.8 per minute of sparring) (Table 3). We observed large variations in mean, median, maximum values, cumulative sum, and time-weighted exposure between individuals and between rounds (Supplemental Tables 1–5, Fig. 2). Sparring partners had a large influence on the number and rate of impacts (Fig. 3, Supplemental Fig. 2).

There were weak-to-strong correlations between all nine metrics for the means (average R^2 : 0.56, range: 0.07–0.97), medians (R^2 : 0.33, 0–0.87), and maximum values (R^2 : 0.58, 0.32–0.99)(Supplemental Fig. 2). The cumulative sums were all significantly and strongly correlated to each other (R^2 : 0.88, 0.56–

1.00); so were the time-weighted exposures (R^2 : 0.96, 0.86–1.00). The cumulative sums all correlated strongly with the number of impacts (R^2 : 0.88, 0.52–0.97), while the TWE_{inv} showed moderate correlations (R^2 : 0.65, 0.56–0.70). No metrics correlated with TSSS changes (R^2 : 0.02, 0.00–0.05).

Participants B and D, who reported symptoms, had three complete sessions each; there was no apparent pattern in the relationship between symptoms changes and the sessions' mean, median, or maximum values (Fig. 2, Supplemental Tables 1–5). The cumulative sums, and particularly the TWE_{inv} , aligned better with the symptom changes. The session with the highest maximum values for participant B (corresponding to the heavy blow reported by the participant) was not associated with a change in TSSS (Fig. 3).

From the control charts, 4 HAEs (0.8% of all high-quality HAEs) were of magnitude higher than two SDs of the participant's mean on all nine metrics (Fig. 3, Supplemental Fig. 2). These four recordings included the heavy blow that was identified on video and met our quality criteria (Participant B, Session 2) and impacts sustained by Participant C (Session 1), Participant D (Session 3), and Participant F (Session 1). Only one of those impacts occurred in a session after which the participant reported an increase in TSSS ≥ 5 (Participant D, Session 3, increase of + 5 between PRE and WOR). Eleven other events (total of 15 or 3.1%) were of magnitude higher than one SD from the participant's mean on all nine metrics, including one recording sustained in an athlete-session after which an increase in TSSS was reported. Other reported heavy blows lacked quality criteria or video confirmation (Participant B, session 3 and Participant E, session 1).

Discussion

We monitored seven boxers to evaluate whether sparring caused acute symptom changes and whether these related to session-specific exposure. Overall, we found that participants self-reported changes in symptoms (TSSS ≥ 5) for 25% of the sparring sessions. In most cases, the symptom severity increased from pre- to post-sparring, then decreased over 48 hours. In our small sample size, there was no consistency between the RHI exposure metrics we calculated and the magnitude of the changes in symptom severity. However, the density of impacts, visible on the control charts and partially represented by the time-weighted exposure parameter, suggests a pattern worth exploring further.

Acute symptoms changes resulting from sparring

When merging the results of the recruitment survey and the data collected over the study, a grouping may emerge. The first group includes participants who reported regularly experiencing heavy blows ("Sometimes" vs. "Never") and symptoms (> 10 symptoms sometimes experienced), and reported acute changes in symptoms and/or heavy blows during the study (participants A, B, D and E). The second group includes participants who did not report anything (C, F and G). Whether the athletes in the first group had greater symptom burden or simply reported more remains unclear. These athletes were newer to the sport; it is possible that experienced boxers habituate to the symptoms, or that a natural selection scenario or survivor bias occurs, where boxers who experience symptoms regularly do not stay in the sport. While the presence of different profiles needs to be further researched, the higher-reporting athletes may benefit from targeted education and monitoring.

Over our limited cohort, there was no significant difference in the number of symptoms or TSSS between pre- and post-sparring, consistent with the work of Kawata et al. on American football players (32). However, when considering only the high-reporting athletes (A, B, D and E), the proportion of athlete-sessions resulting in increases of TSSS ≥ 5 reached 50%. Three of these four participants reported an increase in TSSS ranging from + 5 to + 17 (mean: 12 ± 5) between pre- and post-sparring, resulting in a mean post-sparring TSSS of 19 ± 8 (using the highest value out of POST and WOR). These increases are similar to acute changes from baseline measured in concussed football players (mean change: 13 ± 9 , range: 0–30) (9), and close to TSSS reported after concussion (median: 24, IQR: 11.5–45) (10).

Establishing whether the symptom score changes resulted directly from RHI exposure is not straightforward. Overall, it is common for athletes to report symptoms in the absence of injury, resulting from exhaustion, dehydration, heat-related illness, or anemia (33, 34). While unlikely to have been an issue for the participants in this study, the question of dehydration is particularly relevant in combat sports, as a result of weight management strategies (34, 35). The participants also sometimes reported, prior to sparring, episodes of fatigue and poor sleep, illness, or neck/shoulder soreness; we eliminated this week-to-week within-subject variability (22) by calculating the session-specific pre/post change. Additionally, because some of the symptoms reported immediately after sparring may be confounded with the effects of physical activity (33, 34), the timing of the assessment, whether it is symptoms, cognition, or physical performance, needs to be thought through in future studies aiming to understand the effects of repetitive head impacts.

Implications for athlete safety

Symptom duration may reflect a window of neurometabolic vulnerability (36, 37). The Association of Ringside Physicians states that "*Under no circumstances should a combat sports athlete compete or engage in sparring activity or competition if he or she is experiencing signs and symptoms of concussion.*" (38) As symptoms appear or get worse while sparring or fighting, the athlete's ability to protect themselves may decrease via a decline in reaction time (4), motor control (39), or information retention (3), consequently increasing the risks of getting hit (40). However, athletes may push through their symptoms while they are sparring, but especially fighting, as well as return to training or competition shortly after having sustained a concussion, often hiding their symptoms and not seeking medical advice (2, 7). Precautions are particularly important to take in the lead-up to competition, where the frequency and intensity of sparring may increase. Additionally, while the International Boxing Association (IBA) rules limit the number of fights to one a day, it is common for amateur boxers to partake in multiple fights over several days (e.g., at any major tournament). Safety recommendations include body sparring and limiting contact, though reduced duration may unintentionally increase intensity. Until there exist objective tools that can assess an athlete's state in real-time, it is key that athletes and trainers are educated on how to recognize the signs of concussion, and that they understand that removal from activity is the best decision for safety and performance.

The quantitative analysis of the association between changes in TSSS and kinematics showed no correlation over the 16 athlete-sessions. Prior studies of concussion cases likewise found no link between symptom changes and head impact metrics (11, 13). To the best of our knowledge, there has not been any previous assessment of the relationship between cumulative sums or time-weighted exposure and self-reported symptoms in the absence of a diagnosed concussion. Our qualitative analysis, starting with the visual representation of the impacts over time (Fig. 3), suggested that some athletes sustained large volumes of head impacts in short periods, and that this seemed to happen to a higher degree during the sessions that resulted in changes in TSSS. Though preliminary, higher TWE_{inv} values during symptom-reporting sessions suggest the potential utility of this measure, warranting further investigation.

Particularly, the choice of the equation parameters, such as the constant b or the unit of time, need to be refined and potentially adapted relative to the frequency of impacts as well as the research question (i.e., if impacts are summed over a session or a season). Other metrics, such as the impact density (15) or metrics accounting for the time until assessment (16), should also be further investigated.

Our visual representation of the impacts over time, inspired by control charts, showed that most outliers were not associated with increased symptoms. However, most of these events occurred to participants who never reported changes in symptoms, suggesting that all impacts sustained may have been below a theoretical individual tolerance threshold that was not reached during this study. Indeed, the choice of using the mean and standard deviations as done in clinical applications of the control charts was arbitrary and not meant to represent an individual's tolerance. Our goal was to investigate the role of individual-specific exposure-based limits to highlight outliers of particularly high magnitude that may be associated with symptoms worsening. However, a future objective would be to personalize the chart's limits based on individual tolerance and response to head impact exposure, as we hypothesize that the same exposure may produce different responses in different people. The current control charts also did not account for a hypothetical decrease of injury tolerance as the number of impacts increases, but we could imagine using evolutive control limits declining over the session, or using time-weighted cumulative measures instead of singular events. Control limits could also be adjusted for pre-existing symptoms due to dehydration, poor sleep, or anxiety. These suggestions are all articulated around finding an athlete-specific injury tolerance threshold that would allow for better identification of injured athletes.

We calculated kinematic-based metrics that utilize different aspects of the kinematic signals (multiple degrees-of-freedom or the resultant, linear or angular, acceleration or velocity), and different impacts were singled out by the control charts analysis depending on the metric used. However, metrics were strongly correlated across sessions, suggesting redundancy in the information they provide. The cumulative sums were also strongly associated with the number of impacts and may therefore not be more informative than a simple count. Puvvada et al. (2021)(17) further reported that the association between changes in white matter integrity and TWE_{inv} over a season of youth American football was not stronger than with the number of impacts. Redundancy may be greater over longer durations, such as seasonal or lifetime accumulation. This highlights the need to continue looking for measures of exposure that have physiological relevance and are appropriate for specific research questions.

Limitations

While our work is one of the first investigations of time-weighted exposure relative to neuropsychological changes, this feasibility study was limited by small sample size, restricting statistical power and generalizability. Additionally, the graded symptom scale is inherently subjective, in contrast to objective tests, such as neurocognitive assessments, that may help identify brain trauma without relying on athletes voluntarily reporting symptoms. The symptom scale was, however, an appropriate way to assess the participant's state over several timepoints, in that it was easy and quick to administer, which likely helped with athlete compliance and retention throughout the study. We applied and evaluated nine metrics used in the prediction of (mild) traumatic brain injury; however, several were developed for more severe injuries, and their relevance to RHIs remains unclear. There is also no consensus on the strongest predictor of brain trauma. More research is needed on the clinical relevance of these metrics in the absence of a diagnosed concussion. Finally, we also reiterate that 50% of the HAEs recorded by the iMGs were excluded from analysis because of data quality issues. The number of HAEs per session was strongly correlated with the number of high-quality HAEs per session ($R^2: 0.87$), therefore, the findings would likely remain unchanged. However, there may have been other impacts that would have reached the control charts limits, or the control limits may have changed, which may have affected outlier detection. Improving iMG data quality remains a key challenge.

Conclusion

This study explored sparring-related symptoms and assessed individual-specific approaches to repetitive head impact analysis. Symptoms occurred in 25% of sessions overall but in 50% for athletes in a high-reporting group. The intensity of the symptoms immediately after the end of the sparring session were as high as post-concussion levels, but symptoms were short-lived and resolved within 48 hours. From our preliminary findings, there was limited evidence that the simple accumulation of head impacts based on cumulative sums or the highest-magnitude impact sustained during a session were associated with changes in self-reported symptoms. Time-weighted exposure showed promise in capturing impact density during symptom-provoking sessions. Our results support the concept of individual-specific analyses, which revealed patterns not captured by cohort-averaged summaries. Eventually, symptoms may have resulted from the combination of several factors, namely a high density of head impacts, several above-average-magnitude impacts, and pre-existing dispositions (e.g., personal history, stress, poor-quality sleep, and dehydration). It is essential to better understand these associations to develop informed risk reduction strategies.

Declarations

Pre-print statement

Author contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Enora Le Flao. The first draft of the manuscript was written by Enora Le Flao and all authors commented on subsequent versions of the manuscript. All authors read and approved the final manuscript.

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Figures

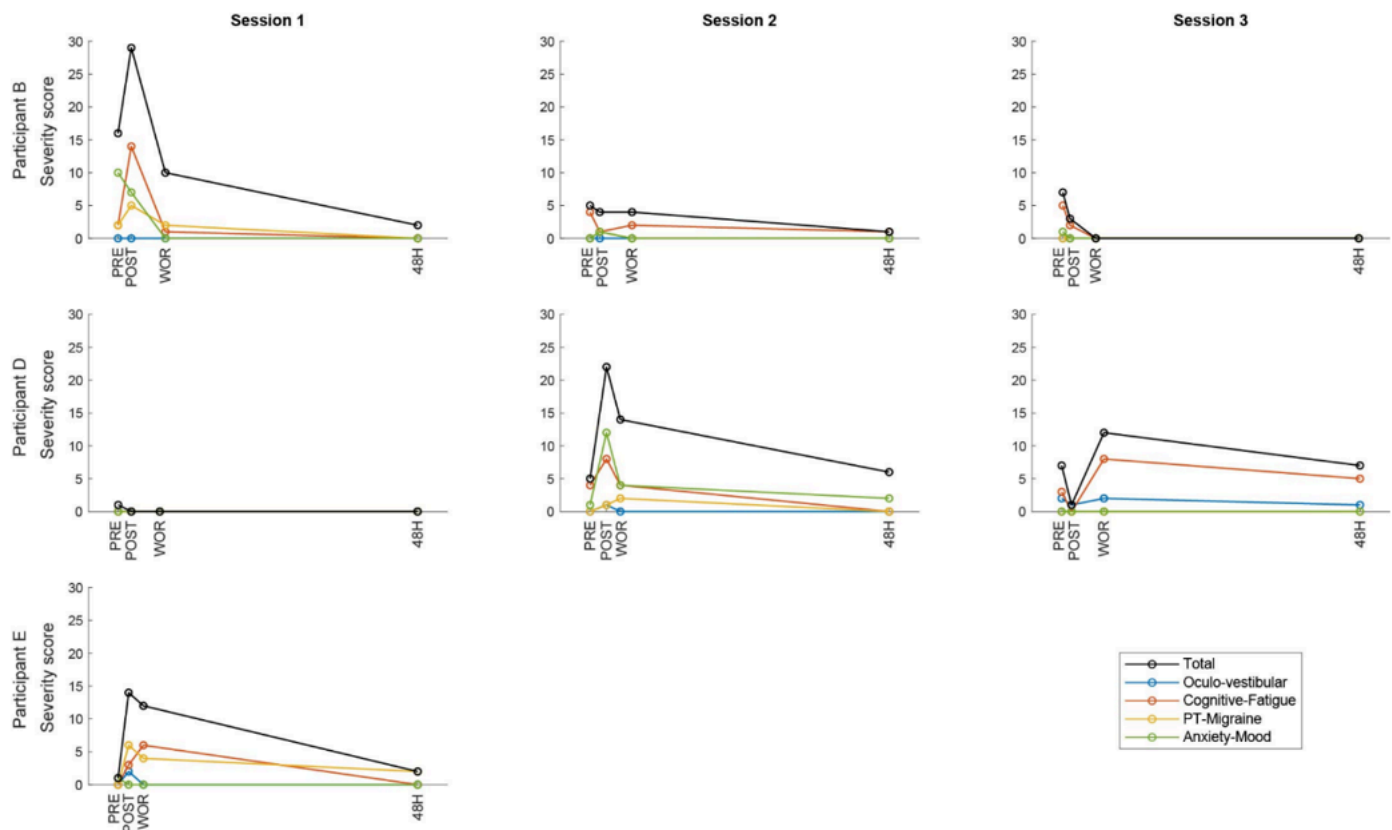


Figure 1

Evolution of the total symptom severity scores (TSSS) for the participants that reported a change in TSSS ≥ 5 at least once over the course of the study. The TSSS (black line), as well as the severity scores by clinical profile (colored lines), are represented for each athlete-session. The difference between the total and the sum of the clinical profiles scores is explained by the exclusion of the “Neck pain” and “Trouble falling asleep” symptoms from the clinical profiles. PRE: pre-sparring, POST: immediately post-sparring, WOR: self-reported worst time where the participant experienced symptoms at the highest intensity, 48H: approximately 48 hours post-sparring.

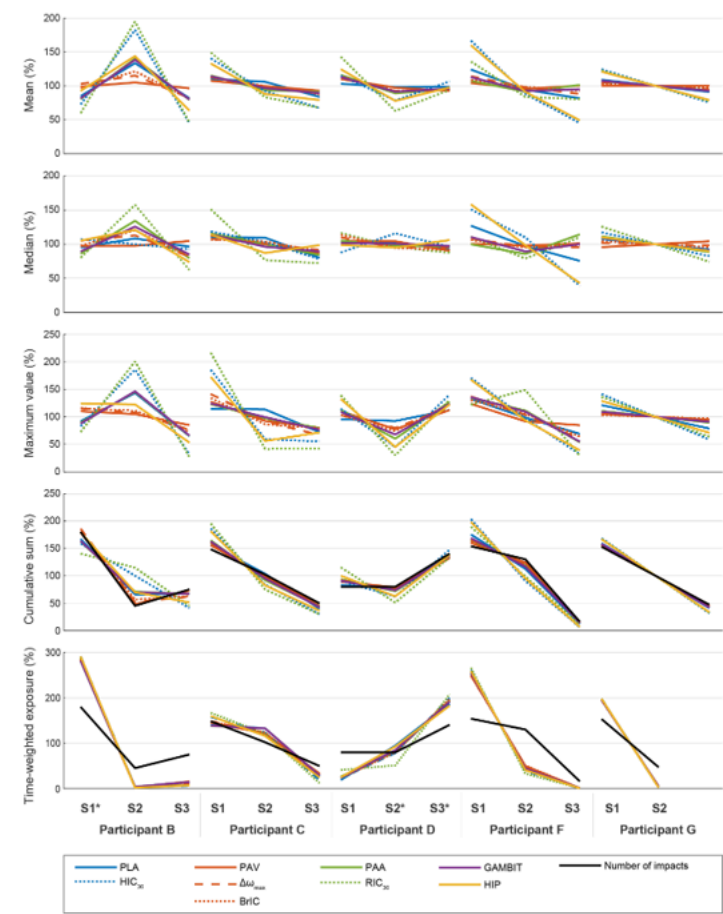


Figure 2

Variation in normalized means, medians, maximum values, cumulative sums, and time-weighted exposures across athlete-sessions for the nine calculated metrics. The number of high-quality head acceleration events is also represented in the bottom two plots. Each value is expressed as a percentage of the participant’s mean value calculated over the two or three athlete-sessions. Only the participants who had more than one complete session are included. The asterisks by the session number (S1 to S3) indicate that the participant reported an increase in total symptom severity score (TSSS) ≥ 5 between pre-sparring and any timepoint post-sparring.

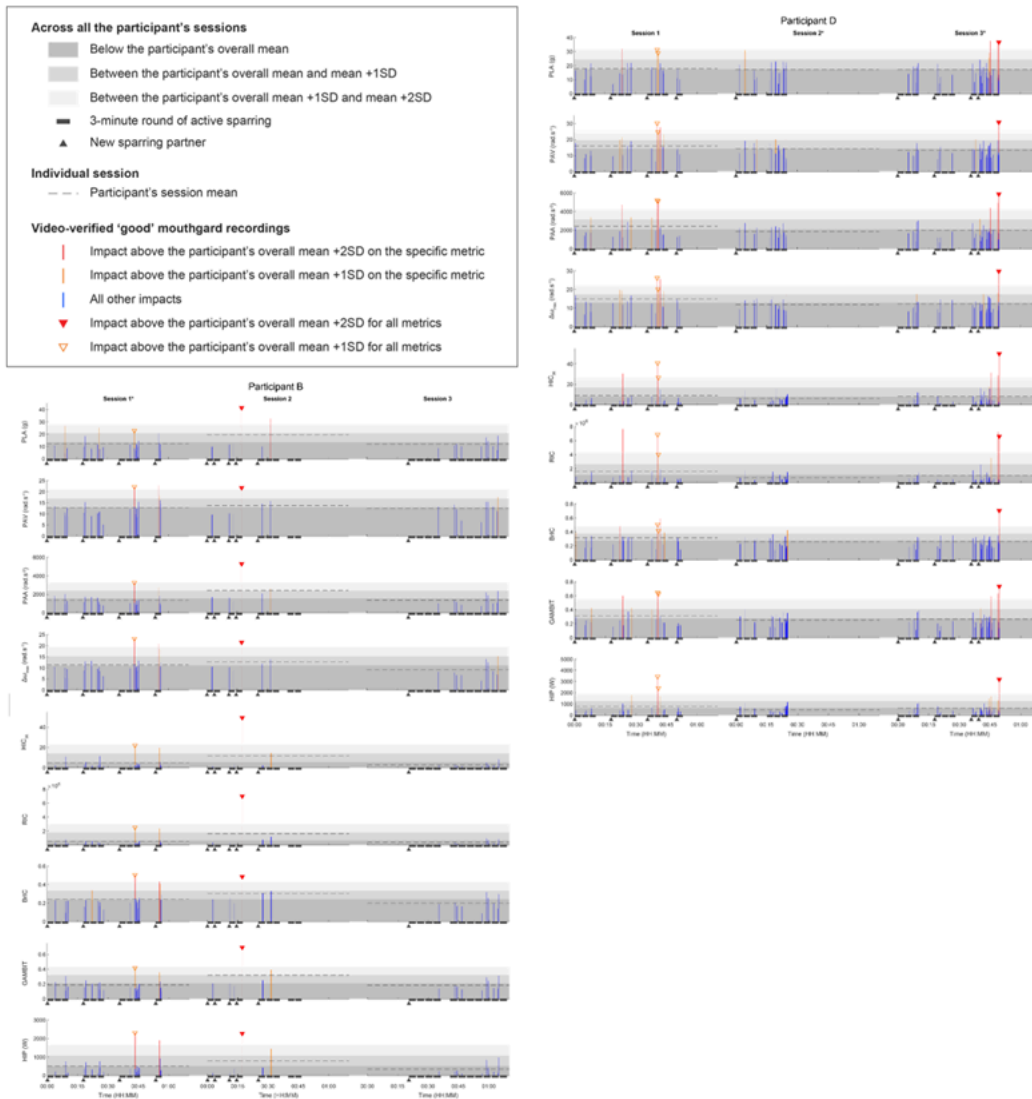


Figure 3

(next pages) Control charts: representation of the magnitude of all video-verified high-quality head acceleration events collected over three full athlete-sessions for participants B and D (the control charts for the other participants are presented in Supplemental figure 2). Each subplot shows 70 minutes and covers the full sparring session, including rest time. The upper border of the shaded areas represents the participants' mean or the mean and one or two standard deviations (SD). Participant's mean and SD values were calculated over all athlete-sessions available for the participant. Each vertical line represents one recording and is blue by default. The orange and red vertical lines represent the recordings for which the magnitude was above the participant's mean +1SD (orange) or mean +2SD (red) values. The triangles highlight the recordings that were above the control limits for all 10 metrics. The asterisks by the session number (above the plots) indicate the sessions where the participant reported an increase in total symptom severity score ≥ 5 from pre- to post-sparring.

Supplementary Files

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