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Repetitive Subconcussive Head Impacts in Sports and Their Impact on Brain Anatomy and Function: A Systematic Review

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ABSTRACT

Repetitive subconcussive head impacts occur regularly in sports. However, the exact relationship between their biomechanical properties and their consequences on brain structure and function has not been clarified yet. We therefore reviewed prospective cohort studies that objectively reported the biomechanical characteristics of repetitive subconcussive head impacts and their impact on brain anatomy and function. Only studies with a pre- to post-measurement design were included. Twenty-four studies met the inclusion criteria. Structural white matter alterations, such as reduced fractional anisotropy and an increase in mean diffusivity values, seem to be evident in athletes exposed to repetitive subconcussive head impacts exceeding 10 g. Such changes are observable after only one season of play. Furthermore, a dose-response relationship exists between white matter abnormalities and the total number of subconcussive head impacts. However, functional changes after repetitive subconcussive head impacts remain inconclusive. We therefore conclude that repetitive subconcussive head impacts induce structural changes, but thus far without overt functional changes.

Introduction

Sports participation has been associated with valuable outcomes, including physiological health promotion and psychosocial benefits [1]. However, there are growing concerns about the potential adverse effects of sports participation due to repetitive subconcussive head impacts (RSHIs) observed in contact and collision sports (i. e. ice hockey, American football, and soccer) [2, 3]. A subconcussive head impact is characterized by a cranial impact that does not result in overt symptoms, such as dizziness, headaches, or short-term memory loss [4], which are present in diagnosed concussions [5]. Head impacts appear particularly during body checks in ice hockey [6], tackles/blocks in American football (FB) [7] or headers in soccer [8]. A head impact event leading to potential meaningful clinical changes has been defined as an event that is caused by a minimum of 10 g linear acceleration (LA) [9, 10]. Still, the brain injury threshold seems to be individualized, as each person's brain (anatomy / function) reacts differently to external impact forces [9, 11, 12]. Subconcussive head impact events are believed to have the most negative impacts when they occur in a cumulative manner [4]. Exposure to such events may be linked with long-term clinical malfunctions, such as chronic traumatic encephalopathy (CTE) [13], neurodegenerative diseases [14], and acute cognitive deficits [15–17]. However, it is still unclear whether a threshold of 10 g is plausible and/or if there exists a dose-response relationship between subconcussive head impacts and brain anatomy and function.

RSHIs may cause structural white matter alterations, such as axonal swelling, axonal integrity reduction, and neuroinflammation [4, 18, 19], mostly in the absence of overt functional (cognitive) symptoms [20, 21]. Structural brain alterations following RSHIs have also been linked with acute and chronic signs of axonal injury [20] as well as neuronal loss [21]. Despite the fact that most researchers did not find overt cognitive symptoms, or report mixed findings [21, 22], some cognitive changes have been detected after RSHIs. For example, reduced reaction time [23] and impaired processing speed abilities [24] have been reported after recent repetitive heading exposure in soccer. RSHIs, just like sport-related concussions (SRCs), are characterized by central accelerative biomechanics (i. e. number of impacts, linear and rotational accelerations, impact duration, and impulse) acting on the head [25, 26]. Therefore, they could potentially share some common symptoms of brain injury. In fact, SRCs may also lead to alterations on the white matter microstructural level, as indicated by axonal integrity loss and axonal swelling [27, 28]. In contrast to RSHIs, SRCs are often accompanied by observable changes, such as loss of consciousness, balance deficits, and sleep disturbances [5, 29]. SRCs are characterized by high acceleration values that surpass acceleration thresholds of 10 g, as it is assumed for RSHIs. In fact, linear accelerations of SRCs have been reported as high as 98 g for male collision sports [30] or 43 g for female ice hockey [12]. Compared to males, females seem to sustain SRCs at lower acceleration magnitudes [11, 12, 31].

The central biomechanical features in sport-related subconcussive head impacts may be induced by direct or inertial (i. e. whiplash) loadings on the head and be either linear or rotational in nature [32]. Despite the acceleration metrics, the impulse and the duration of the impact seem to critically contribute to the injury mechanism [26]. Linear acceleration of the head has been associated with an increase in intracranial pressure, while rotational acceleration (RA) is thought to produce more diffuse injury, due to induced shear forces [32]. Thus far, only head acceleration events, which are induced by a minimum of 10 g LA, are expected to have negative impacts on brain health [9]. This might happen in situations with head-to-head contact in football, during headers in soccer, or while receiving a punch in boxing [8, 33]. In fact, male high school football athletes are exposed to an average magnitude of 26.3 ± 2.8 g LA during head impacts [34] and the same-aged female soccer players to 16.1 ± 3.6 g LA [35]. Another investigation of women soccer players reported a median LA of 12.51 (range 10.0–66.06 g) during games [36]. Despite the magnitude of the impact, the frequency of RSHIs sustained across a career or season has also been potentially linked with adverse brain health effects [9, 37]. Male youth football athletes experience on average 582.8 ± 444.3 (range of 86–1996) subconcussive head impacts over a single season [38] while another investigation of 95 male high school footballers revealed a mean number of 652 impacts (range of 5–2235) across a single season of play [39]. The average number of subconcussive head impacts across one season of play has been reported in women soccer players with 142.9 ± 118.8 (range of 86.9–189.3) [35] and 79 in male soccer [40]. Thus, we assume that a combination of high acceleration events in sports and its repetitive occurrence may have negative consequences on brain function and anatomy.

However, there is still a lack of understanding of the relationship between specific head acceleration metrics in RSHIs and their consequences on brain health in athletes. RSHIs and SRC may share some common structural changes, such as axonal integrity loss and axonal swelling [4, 18, 19, 27, 28]. In contrast to SRCs [5, 29], functional deficits seem not present after RSHI exposure [21, 22]. Whether RSHIs affect specific cognitive domains, which should be detectable, is yet to be fully clarified. Additionally, the concrete structural changes induced by RSHIs have not been linked with specific head acceleration metrics. In fact, the threshold of 10 g is still debated [9, 11, 22]. To our knowledge, no recent systematic review has been conducted exploring the specific head acceleration metrics (i. e. number of subconcussive impacts, average linear acceleration, mean rotational acceleration) in RSHIs and their consequences on brain anatomy and function. Therefore, the aim of this work is to provide a systematic review of head acceleration metrics in sports and their functional and anatomical consequences on brain health in athletes exposed to RSHIs.

Materials and Methods

This systematic review is reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) 2020 guidelines [41].

Search strategy

A two-fold literature search was conducted in the following electronic databases up until July 25, 2023: Web of Science, PubMed, and SPORTDiscus. The search strings were developed using keywords and by applying the ‘OR’ and ‘AND’ Boolean operators. The specific search string for the cognitive research domain is listed in ▶ **Table 1** and the one for the anatomical facet in ▶ **Table 2**.

Inclusion criteria

The eligibility criteria are described in line with the PICOS guidelines [42, 43] and can be found in ▶ **Table 3**. As the literature search was conducted in a two-fold way, we incorporated common inclu-

▶ **Table 1** Search terms cognitive changes and RSHIs.

Boolean operator	Search terms
	(‘collision sport’ OR ‘contact sport’ OR ‘athlete*’ OR ‘sport’ OR ‘performance’ OR ‘player’)
AND	(‘head impact*’ OR ‘head kinematic*’ OR ‘head acceleration’ OR ‘head biometric*’ OR ‘sub-concussive’ OR ‘repetitive head impact’)
AND	(‘neurocogn*’ OR ‘cognit*’ OR ‘neurolog*’ OR ‘function’ or ‘executive function’)

Note: Utilized in the databases PubMED, SPORTDiscus, and Web of Science for cognitive change outcomes; the asterisk is used as a wildcard symbol that broadens a search.

sion and exclusion criteria, which are further specified according to the two research questions, i. e. cognitive and anatomical changes.

Common inclusion criteria were: (1) written in English language, (2) peer-reviewed full-text articles, (3) experimental prospective study design, (4) quantitative in-vivo assessment of head acceleration exposure > 10 g, (5) report of at least one head acceleration metric over the course of the observation period (e. g. LA, RA, number of subconcussive head impacts).

The subsequent inclusion criteria were chosen to identify articles with a focus on cognitive changes after RSHIs: (1) utilization of a validated neurocognitive assessment tool, and (2) report of the repeated measures pre- to post neurocognitive test performance. Articles targeting anatomical change parameters were identified according to the following criteria: (1) using a validated structural brain-imaging method (i. e. DTI or MRI), (2) report of the repeated measures pre- and post-structural changes of the brain.

Exclusion criteria

In general, studies with one of the following criteria have been excluded: (1) self-reported head acceleration assessment, (2) retrospective study design, (3) no in-vivo head acceleration assessment, (4) no respective acceleration data reported, (5) did not exclude concussed subjects from their analysis, (6) no report of the repeated measures change parameters (cognitive / anatomical), and (7) in case of anatomical changes, if they only used functional imaging methods (i. e. Electroencephalography (EEG), Functional near-infrared spectroscopy (fNIRS), or Functional magnetic resonance spectroscopy (fMRS)).

▶ **Table 2** Search terms anatomical changes and RSHIs.

Boolean operator	Search terms
	('collision sport' OR 'contact sport' OR 'athlete*' OR 'sport' OR 'performance' OR 'player')
AND	('head impact*' OR 'head kinematic*' OR 'head acceleration' OR 'head biometric*' OR 'sub-concussive' OR 'repetitive head impact')
AND	('brain imag*' OR 'structural change' OR 'MRI' OR 'DTI' OR 'anatomic* change' OR 'white matter' OR 'diffuse axonal injury' OR 'gray matter')
Note: Utilized in the databases PubMED, SPORTDiscus, and Web of Science for anatomical change outcomes; the asterisk is used as a wildcard symbol that broadens a search.	

▶ **Table 3** PICOS criteria for study inclusion.

PICOS	Inclusion criteria
Population	Active contact sport athletes of all ages and gender
Intervention	Repetitive subconcussive head impact exposure (> 10 g)
Comparison	With or without control group
Outcomes	Cognitive: Head acceleration metrics, changes in cognitive domains (e. g. processing speed, working memory, attention, reaction time, auditory verbal learning, arithmetic tasks, task switching)
	Anatomical: Head acceleration metrics, changes in anatomical metrics (e. g. fractional anisotropy, mean diffusivity, radial diffusivity, axonal radiation)
Study type	Prospective cohort studies

Study selection

Record screening was processed using the reference management software Rayyan [44]. In the beginning, all duplicates were removed from the literature via automatic detection. The studies were then reviewed by title and labelled as relevant, irrelevant, or unclear. Records that were deemed not relevant to one of the research questions have been removed from further screening. Then, abstracts and titles of articles with relevance or unclear relevance were screened, and if deemed not relevant, they got excluded. In the final step, the remaining articles were screened in full-text version and, if necessary, removed.

Risk of bias assessment

We applied the Newcastle-Ottawa Scale (NOS) for prospective cohort studies [45] to identify the risk of biased results within the included studies. This checklist includes eight items, grouped into three categories: (1) group selection, (2) comparability, and (3) outcome. The assessment was done by two independent raters.

Data extraction

The relevant data from the included studies was extracted and used to populate summary outcome tables. Extracted information included: the first name of the author, publication year, sample size, participant information, existence of a control group, the lengths of exposure, utilized cognitive assessment, accelerometer device, recording threshold, imaging technique, head acceleration metrics (either expressed as Median (*Mdn*) or Mean (*M*) values), cognitive/anatomical outcome data, and follow-up measurement timing.

Results

Identification of studies

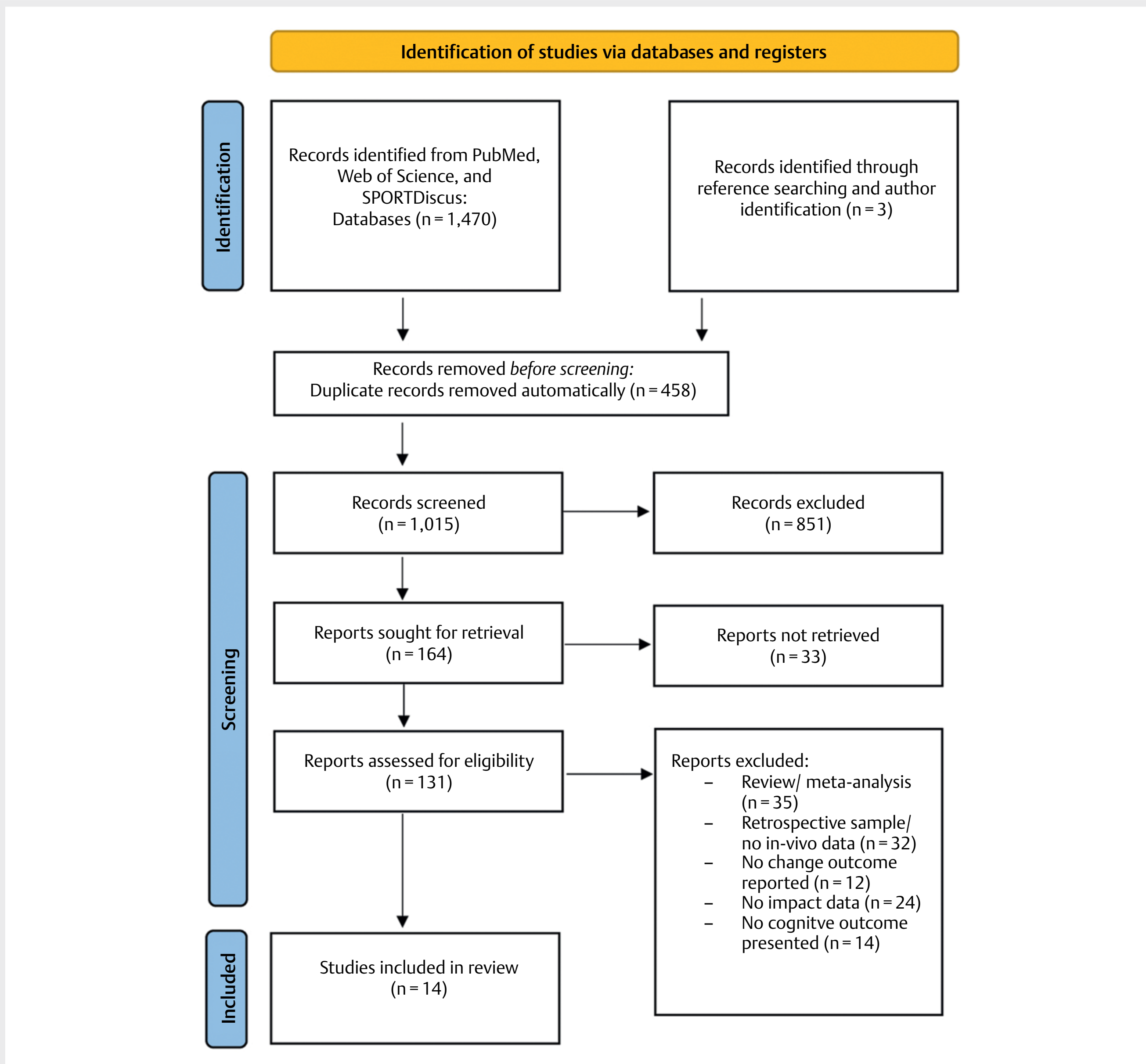
Regarding the cognitive research domain, we identified 1,470 records in the initial database search. Three additional records have been identified through reference searching and author identification. After automatically removing duplicates and an assessment of eligibility, a total of 14 articles have been included. The article identification process is presented in ▶ **Fig. 1**. The literature search targeting anatomical changes resulted in a total of 256 records. Additionally, two records were included from reference searching and author identification. After screening and combined with the additional records, a total of 11 records were eligible. ▶ **Fig. 2** illustrates the full literature identification process.

Risk of bias assessment

The risk of bias assessment of the included studies did not reveal any inter-rater conflicts. Overall, 20 out of 24 included studies hold at least six out of nine possible stars. The individual score of each included record is listed in ▶ **Table 4**. Different scores were mainly identified in the sections of Item 2 and 5, targeting the inclusion of a control group (Item 2) and the existence of control variables in the respective data analysis (Item 5).

Data extraction and synthesis

Data were extracted from the included records and used to provide an overview of all 24 studies that have been identified as relevant

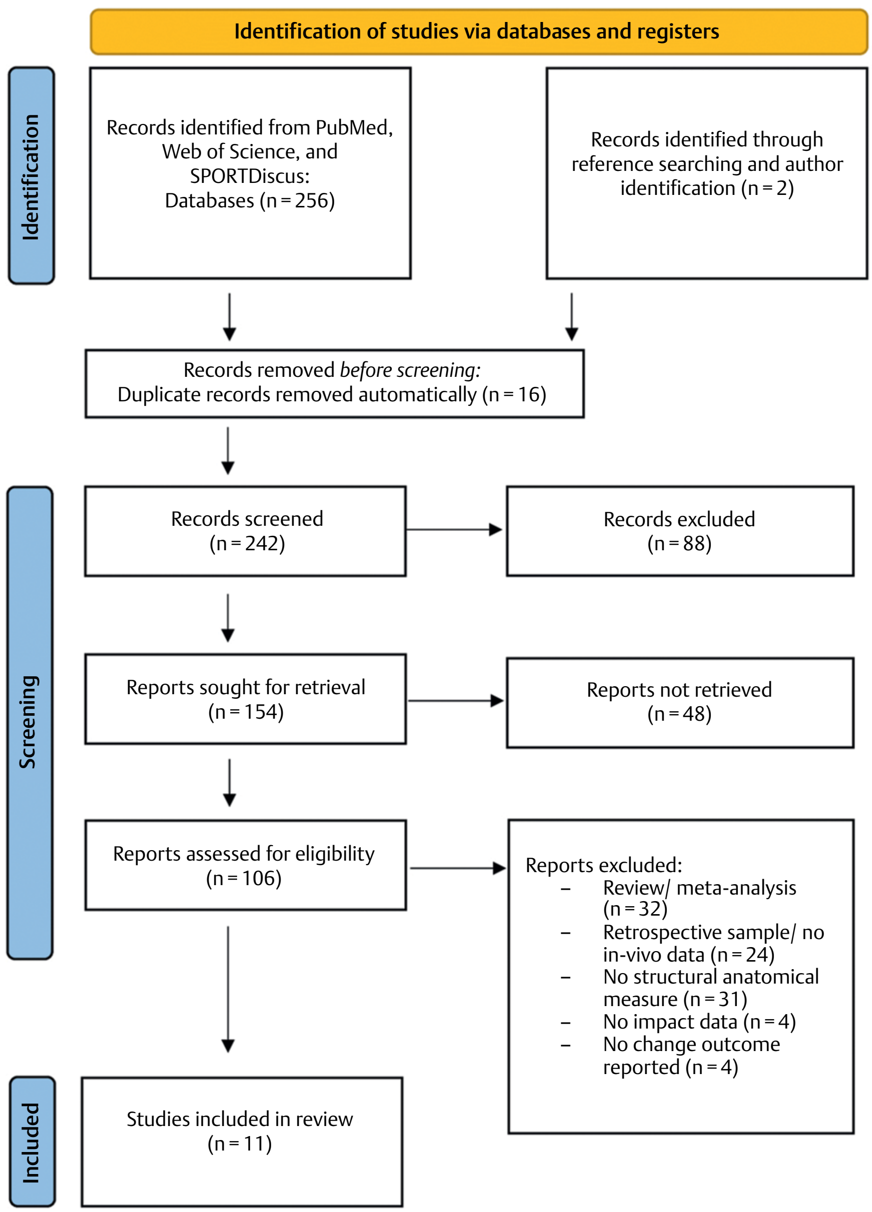


▶ **Fig. 1** PRISMA flow diagram of study identification and selection process for cognitive change parameters [41].

for one of the research domains. ▶ **Table 5** provides an outcome summary for all 14 articles investigating cognitive changes. The detailed outcomes of the 11 included articles for the anatomical changes are listed in ▶ **Table 6**.

Functional outcomes and RSHIs

Four out of 14 included articles did not find any significant changes from pre- to post testing in cognitive performance of the athletes after head impact exposure [35, 46–49]. All of the investigated subjects competed at an amateur level. A total of ten articles did identify significant cognitive changes in cognitive performances from the baseline to the post-testing after head impact exposure. All results from these ten studies showed mixed outcomes regarding improved, decreased, and no change in cognitive performance. Four of the studies identified improved outcomes and no change compared to the baseline [34, 35, 50, 51]. Two of the 14 studies found both improved and decreased cognitive performances across different domains [52, 53]. One study reported increased, decreased, and no change in performance [54], and three found decreased and no change in performance in post testing [38, 55, 56]. Improved performance has been obtained in the following specific cognitive domains: learning and working memory (WM) speed, reaction time, arithmetic processing, processing speed, visual attention, and coding [34, 35, 52–54]. The overall cognitive performance increased in the Comprehensive Trail-Making Test (CTMT), California Verbal Learning Test (CVLT-II), Paced Auditory Serial Addition Test (PASAT), and the Child and Adolescent Memory Profile (ChAMP) [50, 54, 57]. Decreased cognitive performance was re-



▶ **Fig. 2** PRISMA 2020 flow diagram of study identification and selection process for anatomical change parameters [41].

ported in the following distinct domains: memory functioning, processing speed, and response time [52–56], as well as in the composite score [38]. No change compared to the baseline measurement was reported in the overall score of the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT), CogState, Stroop task, Wechsler Intelligence Scale (WISC-V), and Test of Variables of Attention (TOVA) [35, 38, 46–50, 54, 56, 57], as well as in these specific domains: processing speed, attention, WM accuracy [34], verbal and visual memory, visual motor speed and reaction time [55]. In total, seven of the 14 included articles reported the respective statistical association between the obtained head impact metrics and cognitive change parameters from pre to post. Seven out of these 14 investigation did not find any significant relationship between cumulative head impact metrics (i. e. total number of impacts sustained) and cognitive change characteristics [38, 49, 52–56]. Another investigation with college-aged FB and Ice Hockey athletes identified a significant relationship between the peak linear acceleration and the composite score reaction time of the ImPACT, indicating worsened cognitive performance after greater impact exposure [57]. A groupwise analysis revealed that players with detected performance reductions sustained more subconcussive impacts than groups with no changes [56].

▶ **Table 4** Newcastle-Ottawa Quality Assessment Scale scores for included cohort studies.

First author (year)	Item #1	Item #2	Item #3	Item #4	Item #5	Item #6	Item #7	Item #8	Total (out of 9)
Asselin (2020)	★	–	★	★	★	★	–	–	5
Bazarian (2014)	★	★	★	★	★★	★	★	★	9
Breedlove (2014)	★	–	★	★	★	★	★	★	7
Broglio (2018)	★	★	★	★	★★	★	★	–	8
Caccese (2019)	★	–	★	★	★	★	★	★	7
Chrisman (2016)	★	–	★	★	★★	★	–	–	6
Chrisman (2019)	★	–	★	★	★	★	★	★	7
Chun (2015)	★	★	★	★	★	★	★	★	8
Doan (2022)	★	–	★	★	★	★	–	–	5
Gong (2018)	★	–	★	★	★	★	★	★	7
Kelley (2021)	★	–	★	★	–	★	★	★	6
Manning (2020)	–	★	★	★	★	★	★	★	7
Marchesseault (2018)	★	–	★	★	★	★	★	★	7
McAllister (2012)	★	★	★	★	★★	★	★	★	9
McAllister (2014)	★	★	★	★	★	★	★	★	8
Myer (2016a)	–	★	★	★	★	★	★	★	7
Myer (2016b)	–	★	★	★	★★	★	★	★	8
Myer (2019)	–	★	★	★	★★	★	★	★	8
Rose (2019)	–	–	★	★	★	★	–	★	5
Rose (2021)	–	–	★	★	–	★	–	★	4
Slobounov (2017)	★	–	★	★	★	★	★	★	7
Stojsih (2010)	★	★	★	★	★	★	–	★	7
Talavage (2014)	★	–	★	★	★	★	★	★	7
Yuan (2017)	–	★	★	★	★★	★	★	★	8

Anatomical outcomes and RSHIs

A total of nine out of 11 articles reported significant structural changes after measuring subconcussive head impact exposure over time. On the other hand, only one out of 11 identified structural changes after the first season of subconcussive head impact exposure but not after the second season [58]. Two studies did not find any significant anatomical changes in after RSHIs [51, 59]. The included studies only investigated amateur athletes. The changes were related to decreased fractional anisotropy (FA) values compared to the baseline measurement in three studies [46, 60–62]. Four out of 11 studies found increased mean diffusivity (MD) values [60, 61, 63, 64]. One study reported increased FA values (which was only identified in one of the two observed teams) [62]. A total of three out of 11 included articles found reduced MD values over time [58, 65, 66]. The change outcomes of axial diffusivity (AD) showed the following: Three studies reported reduced AD values over time [58, 65, 66], and one study found increased AD values in their sample [61]. Only one author reported increased AD values over time [61]. Lastly, radial diffusivity (RD) decrease was identified in three articles [58, 65, 66]. A subset of two articles highlighted increased RD values over time in the corpus callosum and the brainstem [61, 64]. A total of seven out of 11 included studies in this section reported a significant relationship between cumulative head impact metrics (i. e. number of hits sustained) and anatomical changes, detected as white matter changes [46, 51, 60, 62, 63, 65, 66]. They investigated FB athletes in college-age [46], high school-age [62, 63, 65] and youth-age [60], collegiate FB and ice hockey [51] and high school soccer athletes [66]. Only one study, which studied high school FB players, did not identify a significant association between the obtained head impact metrics and the white matter changes (i. e. reduced MD values, reduced AD values, reduced RD values) [58].

Discussion

The aim of this systematic review was to answer the question of whether specific head acceleration metrics in RSHIs affect the brain's anatomy and function. We therefore reviewed prospective cohort studies that examined pre- and post-changes in cognitive functions as well as anatomical changes in the athletes' brains exposed to RSHIs.

Brain function and RSHIs

According to this research, the findings on functional (cognitive) changes after RSHIs are mixed. Additionally, mixed findings are obtained regarding the causal relationship between the total number of subconcussive hits and functional changes. In fact, some of the reviewed studies did not find an association between functional changes and the total number of RSHIs. This has been reported after a whole season of RSHI exposure [53, 54], a weekend tournament [49], and a set of sparring bouts [52]. In addition, it seems to be independent of the type of sport [49, 52–54]. This indicates that

▶ **Table 5** Cognitive changes after RSHI exposure in athletes.

First author (Year)	Participant information					Exposure to RSHIs	Specific measurement information	Impact sensor (threshold)	Mean follow-up time interval	Significant cognitive outcomes (pre- to post)
	Sample size (sex)	Mean Age (*SD*)	Sport	Competitive level	Control group					
Asselin (2020)	*n* = 28 (M)	19.8 [a]	FB	NCAA III	No	One season	ImPACT	HITS (> 10g)	1 week	↔
Bazarian (2014)	*n* = 10 (M)	20.4 (NR)	FB	NCAA III	Yes	One season	ImPACT	HITS (> 10g)	NR	↔
Breedlove (2014)	*n* = 13 (M)	14–18 [b]	FB	High school	No	One season	ImPACT	HITS (NR)	NR	↓ and ↔
Broglio (2018)	*n* = 46 (M)	15.9 (0.8)	FB	High school	Yes	One season	CCAT	HITS (NR)	1–2 months [b]	↑ and ↔
Caccese (2019)	*n* = 38 (M, F)	M: 19.7 (1.2), F: 19.5 (1.0)	Soccer, FB	NCAA Division I	No	One season	ImPACT, K-D	HITS (> 10g)	Within 1 week	↑ and ↔
Chrisman (2016)	*n* = 17 (M, F)	12.6 (1.0)	Soccer	NR	No	Weekend tournament	ImPact, K-D	xPatch (> 10g)	2 days	↔
Chrisman (2019)	*n* = 46 (M, F)	12.27 (NR)	Soccer	NR	No	1 month	ImPACT, K-D	xPatch (> 15g)	NR	↔
Doan (2022)	*n* = 27 (M)	20.7 (1.8)	Boxing	NCBA	No	2x 2 min sparring bouts	ImPACT, ANAM	IBH (> 9.6g)	39.50 ± 2.50 minutes	↑ and ↓
Marchesseault (2018)	*n* = 15 (M)	21.1 (1.5)	Lacrosse	NCAA III	No	One season	CTMT, SCWT	SIM (> 15g)	1 week	↑ and ↔
McAllister (2012)	*n* = 45 (M, F)	19.0 (1.3)	FB, Ice Hockey	Collegiate	Yes	One season	ImPACT, WRAT-IV, PASAT C, CVLT-II	HITS (> 14.4g)	25.0 ± 31.0 days	↑ and ↔
Rose (2019a)	*n* = 55 (M)	P.S.: 10.8 (0.5) H.S.: 15.9 (0.6)	Youth Tackle FB	Varsity	No	Two seasons	CCAT, MSVT, WISC-V, ChAMP, TMT, WASI-IV, WASI-II, T.O.V.A.	Ridell InSite (S1: > 10g, S2: > 15g)	NR	↑, ↓, and ↔
Rose (2021)	*n* = 18 (M)	10.6 (0.64)	Youth Tackle FB	NR	No	Four seasons	CCAT, MSVT, WISC-V, ChAMP, TMT, WASI-IV, WASI-II, T.O.V.A.	Ridell Insite (S1: > 10g, S2-S4: > 15g)	NR	↑ and ↓
Stojsih (2010)	*n* = 55 (M, F)	M:22.0 (NR) F:24.0 (NR)	Boxing	Amateur	No	4x 2min sparring bouts	ImPACT	IBH (> 9.6g)	Post: 30 min Post24: 24h	↓ and ↔
Talavage (2014)	*n* = 10 (M)	15–19 [b]	Football	Varsity, High school	No	One season	ImPACT	HITS (> 14.4g)	1–3 months [b]	↓ and ↔

Note. ↓ = sign. decrease; ↑ = sign. increase; ↔ = no sign. change. [a] reported as *Mdn*, [b] reported as range. ANAM = Automated Neuropsychological Assessment Metrics; ANT = Attention Network Test; CCAT = CogState Computerized Cognitive Assessment Tool; ChAMP = Child And Adolescent Memory Profile, CSx = accelerometer (CSx Systems Ltd; Auckland; New Zealand); CTMT = Comprehensive Trail Making Test; CVLT-II = Comprehensive Verbal Learning Test; F = Females; FB = Football; GFT = GForceTracker accelerometers (GForceTracker, Markham, ON, Canada); HITS = Head Impact Telemetry System; IBH = The Impact Boxing Headgear; ImPACT = Immediate Post-Concussion Assessment and Cognitive Tool; H.S. = High School; K-D = King-Devick Test; M = Male; MSVT = Medical Symptom Validity Test; NCAA = National Collegiate Athletic Association; NCBA = National Collegiate Boxing Association; NR = not reported; PASAT C = Paced Auditory Serial Addition Test; P.S. = Primary School; RSHIs = repetitive subconcussive head impacts; S1 = Season 1; S2 = Season 2; SCWT = Stroop Color and Word Test; SIM = Smart Impact Monitors; T.O.V.A. = Test of Variables of Attention; TMT = Delis-Kaplan Executive Function System Trail Making Test; WASI-II = Wechsler Abbreviated Scale of Intelligence 2nd Edition/ 4th Edition ; WISC-V = Wechsler Intelligence Scale for Children 5th Edition; WRAT-IV = The Wide Range Achievement Test 4th Edition.

▶ **Table 6** Anatomical changes after RSHI exposure in athletes.

First author (Year)	Participant information					Exposure to RSHIs	Specific measurement information	Impact sensor (threshold)	Mean follow-up time interval	Significant brain structure alteration (pre- to post)
	Sample size	Mean Age (*SD*)	Sport	Competitive level	Control group					
Asselin (2020)	*n* = 28 (M)	19.8 [a]	FB	NCAA III	No	One season	DTI	HITS (> 10g)	1 week	↓ FA values
Chun (2015)	*n* = 28 (M)	16.7 (0.7)	FB	High school	Yes	Two seasons	DTI	HITS (NR)	3–5 months [b]	↑ FA values ↓ FA values [d]
Gong (2018)	*n* = 16 (M)	16.0 [a]	FB	High school	No	One season	DTI	HITS (> 10g)	10 days [a]	↑ MD values
Kelley (2021)	*n* = 19 (M)	12.1 [a]	FB	Youth FB	No	Two seasons	DTI	HITS (NR)	NR	↓ FA values, ↑ MD values
Manning (2020)	*n* = 60 (F)	20.13 (1.43)	Rugby	Varsity Rugby	Yes	Two seasons	DTI	GFT (> 15g)	2–3 months [b]	↓ FA values, ↑ MD values, ↑ AD values, ↑ RD values
McAllister (2014)	*n* = 80 (M, F)	19.5 (1.3)	FB, Ice Hockey	Collegiate	Yes	One season	DTI	HITS (14.4g)	NR	↔
Myer (2016a)	*n* = 15 (M)	16.3 (1.2)	Hockey	Varsity high school	No	Half a season	DTI	GFT (> 20g)	2.9 ± 1.8 days	↑ MD values, ↑ RD values
Myer (2016b)	*n* = 21 (M)	17.13 (0.66)	FB	Varsity high school	No	One season	DTI	GFT (> 20g)	7.05 ± 4.61 days	↓ MD values, ↓ AD values
Myer (2019)	*n* = 22 (F)	15.93 (1.04)	Soccer	Varsity high school	No	One season	DTI	xPatch (> 10g)	3.73 ± 4.33 days	↓ MD values, ↓ RD values
Slobounov (2017)	*n* = 18 (M)	21.6 (1.28)	FB	Collegiate	No	One season	DTI	BodiTrack (> 25)	1–7 days [b]	↔ FA, MD, AD, RD
Yuan (2017)	*n* (S1) = 10 (M) *n* (S2) = 7 (M)	(S1): 16.90 [a] , (S2): 17.73 [a]	FB	High school	No	Two seasons	DTI	GFT (> 10g)	S1: 12.71 ± 7.87 days S2: 5.18 ± 2.48 days	↓ MD values, ↓ AD values, ↓ RD values

Note. ↓ = sign. decrease; ↑ = sign. increase; ↔ = no sign. change. [a] reported as *Mdn.*,[b] reported as range, [c] Team 1, [d] Team 2. AD = axonal diffusivity; ANAM = Automated Neuropsychological Assessment Metrics; ANT = Attention Network Test; CCAT = CogState Computerized Cognitive Assessment Tool; ChAMP = Child And Adolescent Memory Profile, CSx = accelerometer (CSx Systems Ltd; Auckland; New Zealand); CTMT = Comprehensive Trail Making Test; CVLT-II = Comprehensive Verbal Learning Test; DTI = Diffusion tensor imaging; F = Females; FA = fractional anisotropy; FB = Football; GFT = GForceTracker accelerometers (GForceTracker, Markham, ON, Canada); HITS = Head Impact Telemetry System; IBH = The Impact Boxing Headgear; ImPACT = Immediate Post-Concussion Assessment and Cognitive Tool; H.S. = High School; K-D = King-Devick Test; M = Male; MD = mean diffusivity; MSVT = Medical Symptom Validity Test; NCAA = National Collegiate Athletic Association; NCBA = National Collegiate Boxing Association; NR = not reported; PASAT C = Paced Auditory Serial Addition Test; P.S. = Primary School; RD = radial diffusivity; RSHIs = repetitive subconcussive head impacts; S1 = Season 1; S2 = Season 2; SCWT = Stroop Color and Word Test; SIM = Smart Impact Monitors; T.O.V.A. = Test of Variables of Attention; TMT = Delis-Kaplan Executive Function System Trail Making Test; WASI-II = Wechsler Abbreviated Scale of Intelligence 2nd Edition/ 4th Edition ; WISC-V = Wechsler Intelligence Scale for Children 5th Edition; WRAT-IV = The Wide Range Achievement Test 4th Edition.

immediate cognitive deficits are not associated with RSHI exposure in active collision-sport athletes, which has been supported in studies investigating former [67] and recently active collision sport athletes [68–70]. We could further highlight this in studies using a non-contact control group. They found no group [47] or group by time difference [34, 57] in overall cognitive performance between contact and non-contact athletes. In contrast to this, a cohort of female soccer and male football athletes showed a relationship between impaired visual memory, visual processing speed, and the total number of subconcussive impacts [35]. But only to the total number of subconcussive impacts > 98 g LA [35]. No such relationship was observed between the total number of RSHIs above 10 g. Furthermore, the peak linear acceleration sustained during RSHIs was associated with reduced reaction time in a group of male ice hockey and football players [57]. Together, this indicates that RSHIs above 10 g do not induce cognitive deficits, such as impaired memory and processing speed. Only the maximal peak linear acceleration during RSHIs might induce cognitive deficits. In contrast, reduced psychomotor speed and lower verbal learning abilities related to the frequency of subconcussive impacts > 10 g have been obtained in soccer athletes [71]. Monetenigro et al. (2017) hypothesized that a cumulative number of 7,251 subconcussive impacts needs to be exceeded to induce cognitive impairments in athletes [72]. In fact, two of the included studies reported a pattern between reduced cognitive performance and a high number of subconcussive impacts. Talavage and colleagues (2014) were able to find players with more subconcussive hits to be more likely to show signs of cognitive performance reductions after one season [56], which builds on previous work [73]. Rose et al. (2021) identified deficits in processing speeds in youth tackle footballers after receiving a composite mean number of 15,766 subconcussive head impacts over the period of four consecutive seasons [53]. Thus, longer careers, which are expected to produce higher numbers of subconcussive impacts [22], might be a risk factor for cognitive dysfunctions [74].

Opposing findings of athletes exposed to RSHIs above 10 g and improvements in cognitive domains over time such as learning and working memory (WM) speed, arithmetic processing, and processing speed also exist [34, 35, 52–54]. Actively competing athletes may still benefit from the health effects of sports on cognition [23, 76], nullifying potential cognitive declines. Importantly to note, all subjects investigated in the reviewed studies were non-professional athletes, mostly competing on a collegiate level [35, 46, 47, 50, 52, 57]. The level of play seems to be an important consideration in determining whether athletes are at greater risk of overt cognitive performance deficits. Former amateur athletes, compared to professional ones, do not show signs of neurocognitive changes on a metacognition scale [77], as stated in other retrospective reports [75, 78]. A possible explanation might be the fact that professional athletes are exposed to higher cumulative numbers of subconcussive head impacts [22, 79]. This suggests that the most harmful component for reduced brain function may be the total number of subconcussive hits experienced over time, particularly prevalent among professional athletes. Furthermore, one amateur season of exposure might be too short to induce overt functional changes.

Brain anatomy and RSHIs

The current work shows that exposure to RSHIs exceeding a threshold of 10 g leads to white matter alterations in the athletes' brains [46, 58, 60–66]. Specifically, the structural changes seem to be related to the total number of subconcussive head impacts > 10 g [46, 51, 60, 63, 65, 66]. White matter alterations are mainly characterized by reduced fractional anisotropy (FA) values [46, 60–62]. Reduced FA, which is regularly identified in athletes exposed to repetitive subconcussive head impacts [20–22], is interpreted as a signal of damage to the axons' structure and to the myelin sheath surrounding it [20]. The microstructural damage to the axonal architecture constitutes a marker for neurodegenerative and neuroinflammatory processes in the brain [19]. Axonal damage may negatively affect the later-life vitality of contact-sport athletes [28, 80]. This appears independently of age, type of sport, and even after one single season of play [46, 60, 61]. The reduction of FA values suggests that even young contact-sport athletes, after one season of play, already suffer from detectable structural changes. Additionally, a relationship exists between the total number of RSHIs sustained and the FA value reduction [46, 60, 62]. This dose-response relationship highlights an accumulative risk of structural axonal damage with longer exposure periods [81, 82].

Additionally, microstructural brain alterations after RSHIs have been identified as an increase in mean diffusivity (MD) [60, 61, 63, 64] values over time. This represents a chronic irreversible injury state of the brain's tissue [19, 83]. Chronic axonal injury has been previously observed in youth athletes across different types of contact-sports, including American football, hockey, and rugby [60, 61, 63, 64]. Compared to a non-contact control group, increased MD values were only visible in contact-sport athletes [51, 61]. Increased MD values remained after a non-exposure period of three months [61]. This could highlight the danger of long-lasting irreversible structural changes in contact-sport athletes exposed to RSHIs [3]. Furthermore, the number of subconcussive head impacts seems to be related to this MD increase [60, 63], suggesting a dose-response relationship between impact frequency and white matter alteration (i. e. MD increase) [81, 82]. In contrast, decreased MD values are also reported in athletes exposed to RSHIs [58, 65, 66], which are related to the number of subconcussive head impacts [65, 66]. A decrease in MD values over time has been associated with an ongoing recovery phase of the brain's tissue [84], as well as a marker of extracellular space compression, axonal swelling, and inflammatory processes [83]. This injury pattern has already been reported after one season of RSHI exposure across youth soccer and football athletes [58, 65, 66]. Even at a young age, athletes' brains seem to show critical signs of structural alterations [20]. Of note, the variability in MD changes identified in our review might be explained by the variation in follow-up test timing after the athletes' last impact exposure [19], as well as the differing severity and chronicity of brain injury mechanisms potentially accompanying RSHIs [83]. The white matter alterations after RSHIs, indicated by reduced FA [46, 60, 61] and increased MD [60, 61, 63, 64] values, are similarly reported in retired concussed athletes [27, 28]. These signs of brain injury might represent a common structural injury symptomatology in SRCs and RSHI exposure in collision sport athletes.

Lastly, athletes did show brain tissue changes in terms of reduced axial diffusivity (AD) and radial diffusivity (RD) values after RSHIs [58, 65, 66]. Reduced AD and RD values have been generally interpreted as a sign of axonal dysfunction and a loss of axonal membrane integrity [19]. Thus, repeated subconcussive head impacts above 10 g induce white matter alterations. Furthermore, the white matter alterations are related to the total number of sustained RHSIs.

Linkage between functional and anatomical changes

As functional deficits are hypothesized to be a symptom of structural impairments [21], the current work aims to investigate this neurocognitive interplay. However, the results of the association between structural and functional changes are mixed. As RSHIs above 10 g alter white matter structures, such as the corpus callosum (CC) [58, 64, 65], they should induce functional deficits [85]. In turn, this would be critical in complex neural execution situations like motor-control and highly relevant in dynamic sports situations [4]. However, there was no such correlation reported between structural and functional impairments. We could show that no overt cognitive deficits can be expected after one single season of RSHI exposure > 10 g in amateur athletes [49, 52–54]. In contrast, there might be a dose-response relationship between the total number of hits and the occurrence of cognitive deficits [53, 56, 71]. First, findings from cognitive neuroscience propose beneficial effects of sport and exercise on cognition [23, 86], which might positively contribute to the prevention of functional deficits after RSHIs > 10 g. In addition, the brain's ability to compensate for structural alterations (i. e. neural plasticity) [87, 88] might be another explanation for the absence of noticeable cognitive impairments in the present study. For this reason, the brain of active amateur sports athletes might benefit from its compensatory and repair mechanisms in order to prevent the onset of functional impairments after structural brain damage. As discussed previously, amateur collision sport athletes, compared to their professional counterparts, sustain a lower number of subconcussive head impacts over time [22, 79]. Only former professional collision sport athletes show signs of clinical symptoms (incl. overt functional deficits) after their active careers [75]. The higher number of subconcussive impacts experienced by professionals suggests that functional symptoms slowly develop over time. In line with the dose-response hypothesis, it appears that structural damage is only severe enough to cause noticeable functional changes if a certain number of impacts are sustained. However, it is yet to be clarified which specific threshold of structural damage is critical to producing visible functional impairments. Lastly, if the structural changes to the white matter structure should, contrary to the results, affect cognition [85], the cognitive tests utilized (i. e. ImPACT, ANAM, or CogState) may lack sensitivity to detect overt cognitive changes after one single season of play [5, 21, 89]. We also obtained a high level of methodological heterogeneity in the timing of the follow-up cognitive assessments across the included studies. This makes it difficult to draw a causal conclusion between the possible onset and persistence of functional changes. As a result, we conclude that the brain's ability to compensate for structural brain changes, especially those present in active populations, may protect amateur athletes from suffering from overt functional deficits. However, to what specific extent this preventive effect may last is yet unknown. Future research must therefore focus on the neurocognitive interplay between structural alterations and brain functionality.

Conclusion

It is evident that amateur athletes exposed to RSHIs above 10 g suffer from structural brain alterations. This is even present after a single season of play. Structural alterations are mostly found within the white matter by reduced FA [46, 60, 61], reduced AD [58, 65], reduced RD [58, 66], and increased MD values [60, 61, 63, 64]. These alterations display signs of neurophysiological brain injury, such as damage to the axons' structure, the myelin sheath surrounding them [20], compromised axonal integrity [19], and signs of chronic axonal injury [19, 83]. There exists a dose-response relationship between such white matter abnormalities and the sustained cumulative number of RSHIs [46, 51, 60, 63, 65, 66]. This implies that the cumulative number, rather than a single impact event, is causing structural damage. Mixed findings are obtained regarding the presence of functional (cognitive) changes after one season of RSHIs. A relationship pattern for cognitive changes over time and the total number of RSHIs > 10 g seems to be present [53, 56]. Nonetheless, one season of play might not be severe enough to clearly produce detectable functional changes. Thus, we conclude that RSHIs above 10 g after one season of play induce neurophysiological (i. e. white matter) changes that are not displayed in overt functional (cognitive) symptoms.

Conflict of Interest

The authors declare that they have no conflict of interest.

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