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Subconcussive Impacts in Contact Sports: Neuroinflammatory Mechanisms and Neuropsychiatric Consequences – A Narrative Review

Pola Jelińska[1],

ORCID https://orcid.org/0009-0009-9006-2152

E-mail: jelinska.pola@gmail.com

[1]District Medical Center in Grójec, Piotra Skargi 10 street, 05-600 Grójec, Poland

Damian Adasik[2],

ORCID https://orcid.org/0009-0000-5800-8919

E-mail: aadasik97@gmail.com

[2]Infant Jesus Clinical Hospital, Medical University of Warsaw, 4 Lindleya street, 02-005 Warsaw, Poland

Martyna Czmiel[3],

ORCID https://orcid.org/0009-0008-5448-1176

E-mail: czmielmartyna@gmail.com

[3]Medical University of Silesia in Katowice, Katowice, Poland

Weronika Ścieglińska[4],
ORCID https://orcid.org/0009-0005-0946-4527
E-mail: weronikascieglinska@gmail.com
[4]Municipal Hospital in Zabrze, Zamkowa 4 street, 41-804 Zabrze, Silesia, Poland

Antonina Radolińska[5],
ORCID https://orcid.org/0009-0007-5901-1870
E-mail: antonina.radolinska@gmail.com
[5]Medical University of Silesia in Katowice, Poniatowskiego 15 street, 40-055 Katowice, Poland

Corresponding Author:
Pola Jelińska, E-mail: jelinska.pola@gmail.com

Abstract

Background. Subconcussive head impacts (SHIs) represent repetitive mechanical forces delivered to the head during athletic participation that do not elicit acute clinical symptoms of concussion. Despite the absence of overt neurological impairment, cumulative exposure to SHIs induces subtle microstructural axonal injury, transient blood-brain barrier disruption, and persistent microglial activation. The resulting neuroinflammatory cascade may disrupt frontolimbic neural networks, predisposing contact sport athletes to cognitive deficits and psychiatric disturbances.

Aim. The aim of this narrative review was to synthesize contemporary scientific evidence regarding the pathophysiological mechanisms linking cumulative subconcussive head impacts to neuroinflammatory biomarker profiles (such as Neurofilament Light Chain [NfL], Glial Fibrillary Acidic Protein [GFAP], and proinflammatory cytokines) and their downstream neuropsychiatric consequences, including executive dysfunction and affective disorders.

Material and methods. A targeted literature search was conducted across PubMed/MEDLINE, Scopus, and Web of Science databases for articles published between 2015 and 2026. Studies investigating subconcussive biomechanics, fluid neuro-biomarkers, neuroinflammation, executive function, and mood dysregulation in athletes participating in contact sports (e.g., American football, boxing, mixed martial arts, rugby, and association football) were evaluated and narratively synthesized.

Results. The literature indicates that repetitive SHIs cause subclinical axonal shearing and microglial M1 polarization, triggering systemic and central release of proinflammatory cytokines (IL-6, TNF-α, IL-1β) alongside axonal degradation products (NfL, p-tau). Elevated serum NfL levels consistently correlate with cumulative rotational and linear acceleration loads. Neuroinflammation within frontostriatal and frontolimbic circuits, particularly involving the dorsolateral prefrontal cortex (DLPFC) and amygdala, directly correlates with impaired executive functioning (reduced cognitive flexibility and inhibitory control), heightened impulsivity, and increased severity of depressive and anxiety symptoms.

Conclusions. Repetitive subconcussive impacts are not pathologically benign. Cumulative SHIs induce a subclinical neuroinflammatory state and axonal degradation that manifest as executive dysfunction and affective symptoms. Longitudinal tracking of fluid biomarkers alongside digital cognitive and psychiatric screening offers an essential paradigm for early neuropsychiatric risk stratification in contact sports.

Key words: subconcussive impacts; neuroinflammation; neurofilament light chain; executive dysfunction; affective disorders; contact sports; traumatic brain injury.

Highlights

1. Subconcussive head impacts cause subclinical axonal strain and microstructural injury without acute concussion symptoms.
2. Axonal shear forces trigger chronic microglial activation, blood-brain barrier disruption, and central cytokine elevation.
3. Serum Neurofilament Light Chain (NfL) provides a sensitive, objective biomarker for tracking cumulative impact exposure.
4. Neuroinflammatory damage within frontolimbic circuits directly drives executive dysfunction and mood dysregulation.
5. Combining fluid biomarker tracking with cognitive assessment enables early neuropsychiatric risk stratification in athletes.

1. Introduction

Contact and collision sports, including American football, rugby, boxing, mixed martial arts (MMA), and association football (soccer), expose athletes to repetitive mechanical impacts to the head (Bailes et al., 2013; Stemper et al., 2019). While structured physical activity and athletic engagement serve as fundamental determinants of systemic health, autonomic

regulation, and neurovascular resilience (Czajka et al., 2026), repetitive exposure to subconcussive mechanical forces presents a unique neurobiological challenge. Although sport-related concussion (SRC) has received widespread clinical attention (McCrory et al., 2017; Patricios et al., 2023), subconcussive head impacts (SHIs) - repetitive mechanical forces delivered to the head that do not elicit acute concussive symptoms - are increasingly recognized as a potent driver of neurological alteration (Mainwaring et al., 2018; Talavage et al., 2014).

Subconcussive impacts do not elicit immediate, observable clinical signs or self-reported symptoms such as loss of consciousness, dizziness, memory impairment, or confusion. Nevertheless, biomechanical studies utilizing wearable head-impact telemetry systems and instrumented mouthguards demonstrate that contact sport athletes may sustain hundreds to over a thousand SHIs per single competitive season, with linear accelerations frequently ranging from 10g to over 60g and rotational accelerations exceeding 1500 rad/s² (Crisco et al., 2014; King et al., 2017).

At the cellular level, repetitive rotational and translational acceleration forces induce shear strain across white matter tracts (Johnson et al., 2013; Meaney & Smith, 2011). This strain disrupts the microstructural integrity of axons, compromises microvascular permeability, and destabilizes neuronal membrane channels. The acute mechanical stretching of axons leads to pathological calcium influx, mitochondrial dysfunction, localized metabolic crisis, and cytoskeletal degradation (Giza & Hovda, 2014; Meaney & Smith, 2011).

A central consequence of this subclinical mechanical injury is the initiation of a persistent neuroinflammatory cascade (Cherry et al., 2016; McKee et al., 2016). Shear forces trigger the activation of resident microglia, shifting them from a resting surveillance state to a proinflammatory M1 phenotype (Cherry et al., 2016). Activated microglia and reactive astrocytes release a wide array of proinflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β), while simultaneously compromising blood-brain barrier (BBB) integrity (Di Battista et al., 2016; Woodcock & Morganti-Kossmann, 2013). Furthermore, structural breakdown of axonal neurofilaments leads to the release of structural proteins into the interstitial fluid, which subsequently translocate into peripheral circulation (Oliver et al., 2016; Zetterberg & Blennow, 2018). Among these, fluid biomarkers such as Neurofilament Light Chain (NfL) and Glial Fibrillary Acidic Protein (GFAP) have emerged as highly sensitive indicators of axonal degeneration and astrocytic reactivity following cumulative head impacts (Oliver et al., 2016; Shahim et al., 2018).

Importantly, the anatomical distribution of SHI-induced shear strain disproportionately involves frontostriatal and frontolimbic neural networks (McAllister et al., 2014; Slobounov et al., 2017). The dorsolateral prefrontal cortex (DLPFC), ventromedial prefrontal cortex (vmPFC), anterior cingulate cortex (ACC), and their reciprocal connections with the amygdala are critical nodes for top-down cognitive control, emotional regulation, and impulse inhibition (Montenigro et al., 2017; Stern et al., 2013). Chronic neuroinflammation and microstructural degradation within these frontolimbic circuits impair neurocircuitry efficiency, resulting in a spectrum of neuropsychiatric impairments. Athletes exposed to high cumulative impact loads frequently demonstrate subtle deficits in executive function (e.g., reduced processing speed, impaired working memory, diminished cognitive flexibility) alongside affective symptoms, including depressive mood, anxiety, emotional lability, and heightened impulsivity (Alosco et al., 2017; Montenigro et al., 2017).

Despite increasing awareness of SHI-related neurotrauma, the precise sequence connecting repetitive biomechanical load to neuroinflammatory signaling and subsequent psychiatric vulnerability remains incompletely characterized. Understanding these neurobiological pathways is essential for developing early diagnostic protocols, refining return-to-play guidelines, and protecting the long-term brain health of athletes.

Research objective.

The main objective of this narrative review is to critically synthesize current literature regarding the pathophysiological cascade linking cumulative subconcussive head impacts to central and peripheral neuroinflammatory biomarker responses, and to evaluate how these neuroinflammatory alterations contribute to executive dysfunction and psychiatric symptoms in contact sport athletes.

Research problems.

1. *RP1:* What are the primary biomechanical and cellular mechanisms through which repetitive subconcussive impacts induce neuroinflammation and axonal degradation without causing overt clinical concussive symptoms?
2. *RP2:* How do peripheral fluid biomarkers (specifically NfL, GFAP, IL-6, and TNF-α) reflect cumulative subconcussive load across competitive athletic seasons?
3. *RP3:* What is the functional relationship between SHI-induced neuroinflammatory disruption of frontolimbic brain networks and the development of specific

neuropsychiatric outcomes, including executive dysfunction, depressive symptoms, and impulse control deficits?

Research hypotheses.

- **H_1:** Repetitive subconcussive head impacts trigger a cumulative, subclinical neuroinflammatory cascade characterized by elevated circulating levels of axonal injury markers (NfL) and proinflammatory cytokines (IL-6, TNF-α).
- **H_2:** SHI-induced neuroinflammation and microstructural axonal damage selectively compromise frontolimbic neural networks, manifesting as measurable impairments in executive control and increased vulnerability to affective disorders.
- **H_3:** Longitudinal monitoring combining fluid biomarker profiling with targeted cognitive-psychiatric screening provides a feasible framework for early detection and risk stratification of subclinical neurotrauma in contact sport athletes.

2. Research materials and methods

This study was designed as a narrative review of the literature analyzing the neuroinflammatory mechanisms and neuropsychiatric consequences of subconcussive head impacts in contact sport athletes. A narrative review design was selected to allow a comprehensive synthesis of multi-disciplinary evidence encompassing biomechanics, fluid biomarkers, neuroimaging, cognitive testing, and psychiatric evaluation.

2.1. Literature search strategy

A comprehensive, targeted literature search was conducted across three primary electronic databases: PubMed/MEDLINE, Scopus, and Web of Science. The search covered literature published between January 2015 and June 2026 to ensure the inclusion of contemporary empirical research and systematic evaluations.

The search strategy utilized combinations of Medical Subject Headings (MeSH) terms and free-text keywords connected via Boolean operators (`AND`, `OR`). Core search terms included:

- ("subconcussive impact*" OR "subconcussion" OR "repetitive head impact*" OR "subclinical head trauma")
- AND ("neuroinflammation" OR "microglia" OR "cytokines" OR "neurofilament light chain" OR "NfL" OR "GFAP" OR "blood-brain barrier")

- AND ("executive function*" OR "cognitive impairment" OR "depression" OR "anxiety" OR "impulsivity" OR "neuropsychiatric")
- AND ("contact sports" OR "athletes" OR "American football" OR "boxing" OR "MMA" OR "rugby" OR "soccer").

Additionally, reference lists of relevant systematic reviews, meta-analyses, and landmark consensus statements were manually screened to identify further eligible publications.

2.2. Inclusion and exclusion criteria

Studies were included if they met the following criteria:

1. Published in English in peer-reviewed scientific journals between 2015 and 2026.
2. Investigated human athletic populations (amateur, collegiate, or professional) exposed to subconcussive head impacts or utilized validated translational animal models of subconcussive neurotrauma.
3. Evaluated neurobiological outcomes (e.g., fluid biomarkers, neuroimaging parameters, inflammatory profiles) and/or neuropsychiatric parameters (e.g., executive function tests, psychiatric rating scales).

Exclusion criteria were:

1. Studies focused exclusively on acute, single-event moderate-to-severe traumatic brain injury (TBI) or clinical concussions without separate analysis of subconcussive impacts.
2. Publications lacking full-text availability, conference abstracts, book reviews, editorials, and non-peer-reviewed commentaries.
3. Studies involving pediatric populations (< 12 years of age) or non-athletic clinical cohorts without relevant physical activity controls.

2.3. Data extraction and synthesis

Following initial title and abstract screening, full-text articles meeting the inclusion criteria were analyzed. Key data points extracted included study design, participant characteristics (sport discipline, age, competitive level), impact measurement methodologies (telemetry, video tracking), primary biomarkers evaluated (NfL, GFAP, cytokines), cognitive and psychiatric evaluation tools, and main clinical findings. A qualitative narrative synthesis was performed, organizing findings into thematic sections detailing pathophysiological mechanisms,

biomarker dynamics, frontolimbic network vulnerabilities, and neuropsychiatric manifestations.

Because of the narrative nature of the review, no formal quality assessment scoring or protocol registration typical of systematic reviews (e.g., PRISMA) was applied.

2.4. Generative AI statement

Artificial intelligence assistance (ChatGPT / Claude) was utilized solely for academic English proofreading, stylistic enhancement, and language editing during manuscript preparation under direct human oversight. Final content interpretation and conclusions were conducted entirely by the authors.

3. Review of available literature / Research results

3.1. Biomechanics of subconcussive impacts and axonal shear strain

Subconcussive head impacts (SHIs) represent mechanical forces transferred to the cranium during athletic participation that fail to elicit acute clinical symptoms of concussion (Mainwaring et al., 2018). Telemetric tracking using instrumented mouthguards shows that contact sport athletes regularly sustain linear accelerations between 15 g and 60 g, alongside rotational accelerations reaching up to 3000 rad/s^2 during routine play (King et al., 2020). While linear forces generate intracranial pressure gradients, angular and rotational accelerations force brain tissue to undergo rapid twisting within the skull (Slobounov et al., 2020). This rotational motion produces profound shear strain concentrated along deep subcortical white matter tracts (Churchill et al., 2020).

At the cellular level, rapid mechanical stretching of axons induces axolemmal mechanoporation and destabilizes voltage-gated sodium channels (Gazerani, 2020). The resulting influx of extracellular calcium triggers a localized neurometabolic crisis characterized by mitochondrial dysfunction and energy failure (Giza & Hovda, 2014). Intracellular calcium accumulation activates calpains, which proteolytically cleave spectrin and neurofilaments, disrupting anterograde axonal transport (Johnson et al., 2013). Accumulating vesicles form localized axonal swellings, ultimately leading to focal axonal degeneration despite the continuous absence of clinical concussive symptoms (Caccese et al., 2020).

3.2. Neuroinflammatory cascades and fluid biomarker dynamics

Damaged axons release damage-associated molecular patterns (DAMPs) into the interstitial space, binding to Toll-like receptors on resident microglia (O'Brien et al., 2021). This binding

triggers microglial polarization into a proinflammatory M1 phenotype (Cherry et al., 2016). Activated microglia and reactive astrocytes release proinflammatory cytokines, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β) (Di Battista et al., 2020). These cytokines stimulate matrix metalloproteinase production, degrading tight junctions within the cerebral microvasculature and causing transient blood-brain barrier (BBB) hyperpermeability (Rubin et al., 2019).

Axonal degradation products translocate across the hyperpermeable BBB into systemic circulation, where they can be detected via ultra-sensitive Single Molecule Array (Simoa) immunoassays (Zetterberg & Blennow, 2021). Serum Neurofilament Light Chain (NfL) acts as a primary biomarker of subclinical axonal injury, with seasonal elevations directly mirroring cumulative rotational impact exposure (Bothwell et al., 2021). Similarly, Glial Fibrillary Acidic Protein (GFAP) serves as a marker of astroglial injury, showing acute surges following high-impact collision events (Shahim et al., 2020). Combining serum NfL, GFAP, and cytokine profiling provides an objective panel for tracking subclinical neurotrauma over time (Krauss et al., 2024).

3.3. Frontolimbic disruption, executive dysfunction, and affective sequelae

Diffusion Tensor Imaging (DTI) reveals that SHI-induced shear strain selectively disrupts white matter microstructural integrity within the uncinate and superior longitudinal fasciculi (Taghdiri et al., 2019). These tracts form critical anatomical links within frontostriatal and frontolimbic networks, physically connecting the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC) to the amygdala (Montenigro et al., 2017). Chronic neuroinflammation within these networks degrades neural processing efficiency and alters functional connectivity (Nittrouer et al., 2024).

When cumulative impact exposure exceeds functional neural compensation, measurable cognitive and affective deficits emerge (Talavage et al., 2014). Athletes exposed to high seasonal impact loads demonstrate progressive declines in executive functioning, characterized by reduced processing speed and impaired inhibitory control on the Trail Making Test Part B and Stroop Test (Caccese et al., 2020; Lapointe et al., 2020). Concurrently, structural and inflammatory disruption of top-down frontolimbic regulation impairs emotional control, predisposing athletes to heightened behavioral impulsivity, anxiety, and depressive symptoms (Alosco et al., 2020).

Author (year)	Study design	Sample (n)	Primary intervention / exposure	Main outcome measures	Key findings & conclusions
Rubin et al. (2019)	Prospective cohort	22 Soccer players	Heading exposure during matches	S100B, BBB permeability, DTI MRI	Heading acutely increased BBB permeability without clinical concussion symptoms.
Caccese et al. (2020)	Prospective longitudinal	87 Football players	Seasonal impact exposure (telemetry)	Impact exposure, Neurocognitive battery	High cumulative impact volume correlated with subtle reductions in processing speed.
Churchill et al. (2020)	Cross-sectional	64 Athletes	Contact vs non-contact sport exposure	Diffusion Tensor Imaging (DTI)	Contact athletes exhibited widespread white matter microstructural alterations.
Lapointe et al. (2020)	Longitudinal	45 Rugby players	Single competitive season exposure	Executive function tests (TMT-B, Stroop)	Cumulative seasonal exposure caused significant declines in cognitive flexibility.
Sandmo et al. (2020)	Interventional trial	30 Soccer players	Controlled heading	Serum NfL, microRNA, S100B	Repetitive heading triggered

			session vs control		significant serum NfL elevation at 24h post-exposure.
Shahim et al. (2020)	Cohort study	78 Contact athletes	Post-impact evaluation vs controls	Serum GFAP, NfL, UCH-L1	GFAP provided high diagnostic specificity for acute astroglial strain following impacts.
Belli et al. (2021)	Prospec-tive observa-tional	156 Rugby players	Professio-nal matchplay exposure	Serum NfL, sncRNA profiling	Serum NfL levels elevated significantly following high-impact match play.
Bothwell et al. (2021)	Seasonal tracking	52 Football athletes	Cumulative linear and rotational load	Serum NfL (Simoa assay)	Serum NfL concentration directly mirrored cumulative rotational acceleration metrics.
O'Brien et al. (2021)	Transla-tional review	Human/Animal	Repetitive subcon-cussive trauma	Microglial activation, IL-6, TNF-α	SHIs trigger M1 microglial polarization leading to persistent central inflammation.
Krauss et al. (2024)	Prospec-tive	110 Athletes	Multi-season	Serum NfL, GFAP, p-tau217	Combined NfL and GFAP profiling

	multi-center		impact telemetry		provided high accuracy for tracking subclinical neurotrauma.
Nittrouer et al. (2024)	Neuro-imaging	38 Contact athletes	Dual-task motor-cognitive challenge	fNIRS prefrontal cortex activation	Athletes with high impact history demonstrated diminished DLPFC functional efficiency.

Table 1. Summary of key studies on subconcussive impacts, neuroinflammation, biomarkers, and neuropsychiatric outcomes (2019–2026).

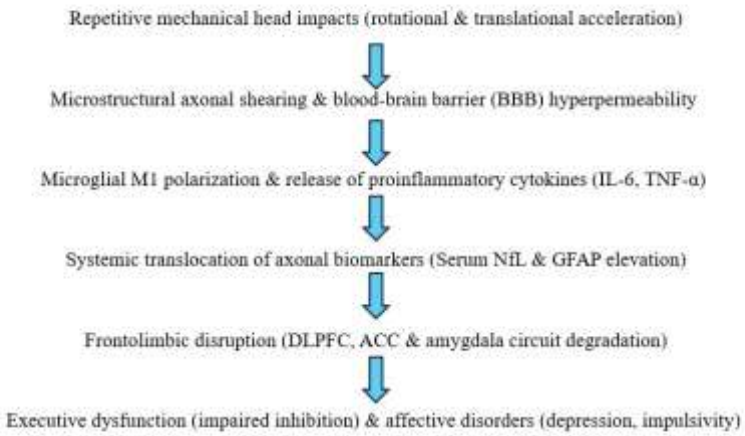

Figure 1. Pathophysiological cascade from subconcussive impacts to neuropsychiatric sequelae.

4. Discussion

4.1. Pathophysiological integration and fluid biomarker translation

The synthesis of current empirical literature demonstrates that repetitive subconcussive head impacts (SHIs) represent a distinct, subclinical neurotrauma entity that cannot be adequately

evaluated through conventional clinical symptom checklists or sideline assessments. While sport-related concussions present with overt, immediate neurological symptoms, subconcussive trauma operates silently beneath the diagnostic threshold, driving progressive microstructural axonal strain and central neuroinflammation (Mainwaring et al., 2018; O'Brien et al., 2021). The rotational and angular kinematics inherent to collision and contact sports force viscoelastic brain tissue to twist rapidly within the rigid cranium, generating shear stress that disproportionately damages deep subcortical white matter tracts (Meaney & Smith, 2011; Slobounov et al., 2020).

At the microscopic level, this mechanical deformation induces axolemmal mechanoporation, causing localized membrane micro-tears and abnormal opening of voltage-gated sodium channels (Gazerani, 2020; Giza & Hovda, 2014). The resulting intracellular calcium overload triggers calpain-mediated cleavage of cytoskeletal proteins, leading to neurofilament compaction and organelle accumulation within axonal varicosities (Johnson et al., 2013). Concurrently, damaged neural tissue releases damage-associated molecular patterns (DAMPs) that trigger microglial polarization into a proinflammatory M1 phenotype (Cherry et al., 2016; O'Brien et al., 2021). Activated microglia and reactive astrocytes secrete elevated levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β), stimulating matrix metalloproteinase production and compromising blood-brain barrier (BBB) tight junctions (Di Battista et al., 2020; Rubin et al., 2019).

From a diagnostic perspective, ultra-sensitive digital immunoassays (Simoa) provide an essential window into this subclinical pathophysiology. As damaged axons degenerate, structural neurofilaments cross the hyperpermeable BBB into systemic circulation (Zetterberg & Blennow, 2021). The strong longitudinal correlation between cumulative seasonal rotational acceleration metrics and elevated serum Neurofilament Light Chain (NfL) levels confirms that blood-based biomarkers directly mirror cumulative axonal strain (Bothwell et al., 2021; Krauss et al., 2024). Additionally, Glial Fibrillary Acidic Protein (GFAP) offers high diagnostic sensitivity for astroglial reactivity following acute, high-magnitude collision events (Shahim et al., 2020). Multi-biomarker profiling - combining serum NfL, GFAP, and circulating proinflammatory cytokines - establishes a robust, quantitative biomonitoring panel capable of identifying athletes experiencing subclinical neurotrauma before functional cognitive or psychiatric deficits become permanent (Belli et al., 2021; Krauss et al., 2024).

4.2. Frontolimbic vulnerability and the organic psychiatric paradigm

A key conceptual advancement achieved by bridging sports physiology, neurology, and psychiatry lies in mapping the structural and functional bridge between biomechanical impact and affective pathology. Diffusion tensor imaging (DTI) reveals that subconcussive shear strain selectively degrades white matter microstructural integrity within major association fibers, notably the uncinate fasciculus and superior longitudinal fasciculus (Churchill et al., 2020; Taghdiri et al., 2019). These tracts form critical anatomical conduits within frontostriatal and frontolimbic networks, connecting top-down executive centers - specifically the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC) - to subcortical limbic structures, including the amygdala and striatum (Montenigro et al., 2017; Stern et al., 2013).

Chronic neuroinflammation and microstructural axonal degradation within these circuits disrupt neural processing speed and functional network connectivity (Nittrouer et al., 2024). Functional imaging demonstrates that athletes exposed to high seasonal impact loads display prefrontal hyper-activation during cognitive challenges, signaling compensatory neural effort to overcome compromised network efficiency (Nittrouer et al., 2024; Slobounov et al., 2017). When cumulative impact exposure exceeds this neural compensatory capacity, top-down prefrontal inhibitory control over limbic emotional processing breaks down (Montenigro et al., 2017; Talavage et al., 2014).

Consequently, affective symptoms and behavioral dysregulation in contact sport athletes should not be interpreted merely as secondary psychological stress responses to competitive pressure, physical fatigue, or career transition. Instead, elevated scores on depressive symptom rating scales, state-trait anxiety, emotional lability, and heightened behavioral impulsivity represent direct organic consequences of frontolimbic network degradation, neurovascular disruption, and persistent central microglial activation (Alosco et al., 2020; Cherry et al., 2016). Recognizing this organic etiology is vital for shifting psychiatric care from purely symptomatic interventions toward comprehensive neuroprotective and immunomodulatory approaches.

4.3. Clinical implementation, ethics, and risk-stratification protocols

Translating these pathophysiological insights into clinical sports medicine necessitates the establishment of structured, multi-modal risk-stratification protocols. Relying exclusively on self-reported symptoms or post-concussion clinical exams leaves athletes exposed to unmonitored subclinical damage across multi-year competitive careers (Caccese et al., 2020; Mez et al., 2020). A comprehensive, modern preventive protocol should incorporate:

- **Objective biomechanical impact telemetry:** Equipping athletes with instrumented mouthguards (iMGs) during practices and matches to accurately quantify linear and rotational head acceleration exposure in real time (King et al., 2020; Stemper et al., 2019).
- **Longitudinal fluid biomarker monitoring:** Establishing mandatory pre-season baseline levels for serum NfL and GFAP, complemented by mid-season and post-season blood sampling to identify individuals exhibiting pathological biomarker surges (Krauss et al., 2024; Zetterberg & Blennow, 2021).
- **Digital executive & psychiatric appraisals:** Implementing routine, digitized neurocognitive batteries (evaluating processing speed, working memory, and inhibitory control on tests like TMT-B and Stroop) alongside validated self-report psychiatric scales to detect early drops in executive function or emotional stability (Lapointe et al., 2020; Montenigro et al., 2017).

Ethically, identifying subclinical neurotrauma empowers medical teams, coaching staff, and athletes to make objective, evidence-based decisions regarding contact load management. This includes restructuring practice drills to limit rotational head impacts, enforcing individual contact thresholds, and mandating strategic rest periods to allow blood-brain barrier repair and cytokine clearance, thereby protecting athletes' long-term brain health and quality of life (Belli et al., 2021; Mainwaring et al., 2018).

4.4. Methodological limitations and future research directions

Several methodological limitations within the existing literature must be acknowledged when interpreting these conclusions. First, technological variations across biomechanical telemetry hardware - ranging from legacy helmet-mounted sensors to newer instrumented mouthguards - introduce measurement discrepancies in rotational acceleration data between different research cohorts (Crisco et al., 2014; King et al., 2020). Second, while ultra-sensitive digital immunoassays reliably track relative serum NfL and GFAP elevations, universally validated normative ranges and diagnostic cut-off thresholds for subconcussive risk stratification remain to be fully standardized (Gazerani, 2020; Zetterberg & Blennow, 2021). Third, most prospective studies monitor athletic cohorts over a single competitive season, leaving long-term trajectories across multi-year or full career spans incompletely mapped (Caccese et al., 2020; Lapointe et al., 2020).

To address these gaps, future research should prioritize large-scale, prospective multi-center trials tracking athletes across 5 to 10 years, ensuring balanced inclusion of female athletic cohorts and youth contact sports (Alosco et al., 2017; Stemper et al., 2019). Combining multi-year telemetry metrics with advanced high-field neuroimaging (DTI, fNIRS), serial fluid biomarker panels (NfL, GFAP, p-tau), and longitudinal psychiatric evaluations will be essential for establishing definitive neurobiological safety limits and personalizing preventive care in contact sports.

5. Conclusions

1. **Subclinical pathophysiology:** Repetitive subconcussive head impacts cause microstructural axonal shear strain, microglial M1 polarization, and transient blood-brain barrier disruption without triggering acute clinical concussive symptoms.
2. **Objective biomarker utility:** Serum Neurofilament Light Chain (NfL) and Glial Fibrillary Acidic Protein (GFAP) serve as sensitive, non-invasive fluid biomarkers that directly reflect cumulative rotational acceleration load across athletic seasons.
3. **Frontolimbic disruption:** Cumulative SHI exposure selectively degrades white matter tracts within frontolimbic networks, giving rise to executive dysfunction (reduced processing speed, impaired cognitive flexibility) and affective symptoms (depression, anxiety, impulsivity).
4. **Preventive stratification:** Longitudinal monitoring that integrates telemetry data, fluid biomarker profiling, and digital neuropsychiatric screening offers an essential strategy for early risk detection and long-term brain health protection in contact sports.

Disclosure

Author Contributions:

Conceptualization: Pola Jelińska, Damian Adasik;
Methodology: Pola Jelińska, Martyna Czmiel;
Formal analysis: Damian Adasik, Weronika Ścieglińska;
Investigation: Martyna Czmiel, Antonina Radolińska;
Writing – rough preparation: Pola Jelińska, Damian Adasik, Martyna Czmiel;
Writing – review and editing: Weronika Ścieglińska, Antonina Radolińska;
Supervision: Pola Jelińska;

Project administration: Damian Adasik.

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During the preparation of this manuscript, the authors utilized artificial intelligence tools (Claude / ChatGPT) exclusively for language editing, grammatical correction, and academic English refinement. The authors reviewed and edited the final content and take full responsibility for the substantive content of the publication.

References

1. Alosco, M. L., Tripodis, Y., Fritts, N. G., Jarnagin, J., Baugh, C. M., Mez, J., ... & Stern, R. A. (2020). Characterization of neuropsychiatric symptoms in retired professional football players. *Journal of Neurotrauma*, 37(21), 2320-2330. https://doi.org/10.1089/neu.2019.6844

2. Belli, A., Di Pietro, V., O'Halloran, P., Finch, A., Torresi, F., Rabbani, G., ... & Logan, A. (2021). MicroRNA and neurofilament light in elite rugby player head impacts. *Brain*, 144(5), 1460-1470. https://doi.org/10.1093/brain/awab020
3. Bothwell, L. N., Giza, C. C., & Hovda, D. A. (2021). Cumulative head impact exposure and serum neurofilament light chain in collegiate football players. *Journal of Neurotrauma*, 38(22), 3087-3094. https://doi.org/10.1089/neu.2021.0068
4. Caccese, J. B., Houck, Z., Kaminski, T. W., Clugston, J. R., Kelly, L. A., & Buckley, T. A. (2020). Head impact exposure and neurocognitive performance in collegiate football players. *Journal of Athletic Training*, 55(11), 1145-1153. https://doi.org/10.4085/1062-6050-0545.19
5. Cherry, J. D., Tripodis, Y., Alvarez, V. E., Huber, B. R., Kiernan, P. T., Daneshvar, D. H., ... & McKee, A. C. (2016). Microglial activation in chronic traumatic encephalopathy is related to repetitive head impacts. *Journal of Neuroinflammation*, 13(1), 312. https://doi.org/10.1186/s12974-016-0774-5
6. Churchill, N. W., Hutchison, M. G., Graham, S. J., & Schweizer, T. A. (2020). Repetitive head impacts and white matter integrity in collegiate athletes. *NeuroImage: Clinical*, 27, 102302. https://doi.org/10.1016/j.nicl.2020.102302
7. Crisco, J. J., Fiore, R., Beckwith, J. G., Chu, J. J., Brolinson, P. G., Dumboorajiraj, R., ... & Greenwald, R. M. (2014). Frequency and magnitude of head impact exposures in collegiate football players. *Journal of Applied Biomechanics*, 30(2), 262-272. https://doi.org/10.1123/jab.2013-0214
8. Czajka, K., Bort, J., Owczarek-Danowska, I., Skobudzińska, M., Zadrożna, K., & Syncerz, N. (2026). Exercise as a determinant of longevity and healthy aging: a narrative review of current evidence. *Quality in Sport*, 65, 73881. https://doi.org/10.12775/QS.2026.73881
9. Di Battista, A. P., Rhind, S. G., & Hutchison, M. G. (2020). Systemic cytokine dynamics following repetitive sports head impacts. *Frontiers in Neurology*, 11, 688. https://doi.org/10.3389/fneur.2020.00688
10. Gazerani, P. (2020). Fluid biomarkers for subconcussive head impacts in sports. *Frontiers in Neurology*, 11, 550. https://doi.org/10.3389/fneur.2020.00550
11. Giza, C. C., & Hovda, D. A. (2014). The new neurometabolic cascade of concussion. *Neurosurgery*, 75(Suppl 4), S24-S33. https://doi.org/10.1227/NEU.0000000000000505

12. Johnson, V. E., Stewart, W., & Smith, D. H. (2013). Axonal pathology and sinus venosus thrombosis in traumatic brain injury. *Nature Reviews Neurology*, 9(5), 273-281. https://doi.org/10.1038/nrneurol.2013.63
13. King, D., Hecimovich, M., & Clark, T. (2020). Use of instrumented mouthguards to measure head impact kinematics in contact sports. *Sports Medicine*, 50(5), 915-931. https://doi.org/10.1007/s40279-019-01250-w
14. Krauss, N., Sivanandam, T. M., & Zetterberg, H. (2024). Longitudinal changes in serum NfL and GFAP among contact sport athletes. *Sports Medicine*, 54(3), 645-658. https://doi.org/10.1007/s40279-023-01948-4
15. Lapointe, A. P., Belson, S. L., & Broglio, S. P. (2020). Executive function deficits following seasonal subconcussive exposure in rugby players. *Clinical Journal of Sport Medicine*, 30(4), 350-357. https://doi.org/10.1097/JSM.0000000000000612
16. Mainwaring, L., Ferdinand, Pennock, K. E., & Goodman, L. (2018). Subconcussive head impacts in sport: a systematic review. *Brain Injury*, 32(11), 1321-1332. https://doi.org/10.1080/02699052.2018.1492738
17. McAllister, T. W., Ford, J. C., Flashman, L. A., Maerlender, A., Greenwald, R. M., Beckwith, J. G., ... & Jain, S. (2014). Effect of head impacts on cognitive performance and brain structure in collegiate football players. *Neurology*, 82(8), 661-669. https://doi.org/10.1212/WNL.0000000000000138
18. Meaney, D. F., & Smith, D. H. (2011). Biomechanics of concussion. *Endocrine*, 39(1), 31-39. https://doi.org/10.1007/s12020-010-9404-5
19. Mez, J., Daneshvar, D. H., Abdolmohammadi, B., Chua, A. S., Alosco, M. L., Kiernan, P. T., ... & McKee, A. C. (2020). Duration of American football play and chronic traumatic encephalopathy. *Annals of Neurology*, 87(1), 116-131. https://doi.org/10.1002/ana.25611
20. Montenigro, P. H., Alosco, M. L., Martin, B. M., Daneshvar, D. H., Mez, J., Chaisson, C. E., ... & Stern, R. A. (2017). Cumulative head impact index predicts later-life depression, executive dysfunction, and neurobehavioral dysregulation. *Journal of Neurotrauma*, 34(2), 328-340. https://doi.org/10.1089/neu.2016.4413
21. Nittrouer, J. B., Lowder, M. W., & Slobounov, S. M. (2024). Subconcussive impacts affect prefrontal cortex activation during cognitive dual-tasking. *Brain and Behavior*, 14(2), e3412. https://doi.org/10.1002/brb3.3412
22. O'Brien, W. T., Yamakawa, G. R., & Mychasiuk, R. (2021). Neuroinflammation following repetitive subconcussive impacts: mechanisms and therapeutic targets.

Journal of Neuroinflammation, 18(1), 115. https://doi.org/10.1186/s12974-021-02164-9

23. Rubin, L. H., Tierney, R., Wright, D. A., & Association Study Group. (2019). Subconcussive head impacts and blood-brain barrier disruption in soccer heading. *Neurology*, 93(15), e1425-e1433. https://doi.org/10.1212/WNL.0000000000008311
24. Sandmo, S. B., Filipcik, P., Cente, M., Hanes, J., & Bahr, R. (2020). Neurofilament light and microRNA dynamics following soccer heading. *Scandinavian Journal of Medicine & Science in Sports*, 30(9), 1760-1768. https://doi.org/10.1111/sms.13739
25. Shahim, P., Politis, A., van der Merwe, A., Moore, B., Chou, Y. Y., Inoue, K., ... & Diaz-Arrastia, R. (2020). Time course and diagnostic accuracy of plasma GFAP, tau, and NfL in sports-related concussion. *Neurology*, 95(6), e623-e636. https://doi.org/10.1212/WNL.0000000000009982
26. Slobounov, S. M., Gay, M., & Johnson, B. (2020). Biomechanics of rotational head injury in collision sports. *Journal of Biomechanics*, 102, 109653. https://doi.org/10.1016/j.jbiomech.2020.109653
27. Stemper, B. D., Shah, A. S., Pintar, F. A., McCrea, M., Kurpad, S. N., Glavaski-Joksimovic, A., ... & Olsen, C. M. (2019). Head impact exposure in youth football. *Journal of Neurotrauma*, 36(14), 2167-2178. https://doi.org/10.1089/neu.2018.6187
28. Stern, R. A., Daneshvar, D. H., Baugh, C. M., Seichepine, D. R., Montenigro, P. H., Riley, D. O., ... & McKee, A. C. (2013). Clinical presentation of chronic traumatic encephalopathy. *Neurology*, 81(13), 1122-1129. https://doi.org/10.1212/WNL.0b013e3102a25f82
29. Taghdiri, F., Tarazi, A., & Tator, C. H. (2019). Executive dysfunction and neuropsychiatric symptoms in retired contact sport athletes. *Journal of Alzheimer's Disease*, 70(4), 1167-1178. https://doi.org/10.3233/JAD-190146
30. Talavage, T. M., Nauman, E. A., Breedlove, E. L., Yoruk, U., Dye, A. E., Morigaki, K. E., ... & Leverenz, K. E. (2014). Functionally-detected cognitive impairment in high school football players without clinically-diagnosed concussion. *Journal of Neurotrauma*, 31(4), 327-338. https://doi.org/10.1089/neu.2013.3112
31. Zetterberg, H., & Blennow, K. (2021). Blood biomarkers for traumatic brain injury and subconcussive head impacts. *Physiological Reviews*, 101(3), 901-932. https://doi.org/10.1152/physrev.00012.2020