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CLINICAL ARTICLE

Short-term changes in the physiology of the primary motor cortex following head impact exposure during a Canadian football game

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OBJECTIVE This study investigated the association between head impact exposure (HIE) during varsity Canadian football games and short-term changes in cortical excitability of the primary motor cortex (M1) using transcranial magnetic stimulation (TMS).

METHODS Twenty-nine university-level male athletes wore instrumented mouth guards during a football game to measure HIE. TMS measurements were conducted 24 hours before and 1–2 hours after the game. Twenty control football athletes were submitted to a noncontact training session and underwent identical TMS assessments. Between-group changes in short-interval intracortical inhibition (SICI) ratios over time were conducted using two-way ANOVAs. The relationship between HIE (i.e., number, magnitude, and cumulative forces of impacts) and SICI (secondary outcome) was also investigated using Pearson correlations.

RESULTS Relative to controls, the group of athletes who had played a full-contact football game exhibited a significant intracortical disinhibition ($p = 0.028$) on the SICI 3-msec protocol (i.e., short interstimulus interval of 3 msec) within hours following the game. Moreover, exposure to ≥ 40*g* hits positively correlated with SICI disinhibition ($p < 0.05$).

CONCLUSIONS Athletes exposed to subconcussive hits associated with Canadian football exhibit abnormal M1 corticomotor inhibition function, particularly when the recorded impact magnitude was ≥ 40*g*. Given the deleterious effects of decreased inhibition on motor control and balance, systematically tracking head impact forces at each game and practice with contacts could prove useful for injury prevention in contact sports.

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KEYWORDS sport concussions; subconcussion; head impact exposure; intracortical inhibition; contact sports; football; transcranial magnetic stimulation; trauma; traumatic brain injury

Sport-related concussions are a growing concern as recent estimates suggest that 85% of Canadian university-level football players will sustain at least one concussion throughout their career.[1] Typically, concussions result in chemical and metabolic changes that manifest as somatic, cognitive, or emotional symptoms; physical signs; and sleep disturbances, in the absence of visible macrostructural damage on routine clinical scans.[2]

Subconcussive head impacts involve insufficient forces or accelerations to produce clinical signs and symptoms that characterize a concussion.[3,4] It has been demonstrated that repeated exposure to both concussive and subconcussive head impacts can affect short-term and long-term brain health (e.g., motor control and memory performance).[5,6] These subconcussive impacts are extremely common in contact sports. In Canadian football, they range from a few times per game for receivers to nearly every play for linemen,[7] potentially leading to adverse outcomes. For in-

ABBREVIATIONS FDI = first dorsal interosseus; HIE = head impact exposure; iMG = instrumented mouth guard; M1 = primary motor cortex; MEP = motor evoked potential; SICI = short-interval intracortical inhibition; TMS = transcranial magnetic stimulation.

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stance, reduced performance in attention and memory was found in university rugby players postseason, with only 3% experiencing a diagnosed concussion.[4] Furthermore, extended exposure to repetitive head impact was demonstrated to alter the neurochemistry of athletes.[8,9] These findings are particularly concerning given that some athletes playing contact sports, including varsity football players, can be exposed to nearly 1400 impacts per year,[10] which could lead to subclinical brain alterations.

Current methods to assess head impact–related outcomes are mostly based on self-report, which can lead to underreporting of symptoms, particularly in athletes eager to return to play.[11] Considering the major shortcomings associated with medical decisions based on such subjective measures, coming up with objective measures to assess acute neurological dysfunctions associated with head impacts in contact sports is of paramount clinical importance. In terms of injury mechanism characterization, validated sensor technologies can measure head impact exposure (HIE) and its biomechanical profile (velocity, direction, magnitude, etc.).[12] Biomechanical factors such as magnitude and linear acceleration have been shown to reflect the severity and extent of alterations in brain integrity following both concussion and subconcussive impacts.[13]

In parallel, transcranial magnetic stimulation (TMS) applied over the dominant primary motor cortex (M1) is particularly appealing in the context of contact sports as it serves the dual purpose of objectively probing motor system physiology changes with exercise as well as HIE.[14] In previous dose-response pharmacological studies,[15] short-interval intracortical inhibition (SICI) has been shown to reflect $GABA_A$ receptor subtype inhibitory function within M1.[16] The latter measure of GABA-mediated inhibition of M1 was suggested to underlie acute and long-term increases in neuroplasticity associated with physical activity[17] and is thereby reduced following aerobic exercise.[18] Given its high test-retest reliability[19] and well-described mechanisms of action as well as its high sensitivity to subconcussive head impacts, sport concussions, and postconcussion syndrome,[20–22] SICI modulation represents a particularly insightful neuromarker to characterize. The GABA-mediated disruptions were shown to persist even years following a concussion and have been linked to reduced motor learning in otherwise asymptomatic concussed athletes who had successfully returned to play football.[22,23]

Given the known excitotoxic component driving the metabolic cascade of concussion,[24] authors have suggested that the observed persistent M1 cortical disinhibition could be the result of an imbalance between inhibitory GABAergic and excitatory glutamatergic mechanisms of M1.[22,23] Recently, Di Virgilio et al. found increased intracortical inhibition in both soccer players following a single exposure to repetitive subconcussive impacts[25] and boxers following 3-minute sparring bouts.[26]

The present study therefore combines telemetric sensor technology inserted in mouth guards with TMS to test whether the accumulation and magnitude of head impacts during a varsity Canadian football game modulate M1 intracortical inhibition assessed within a few hours after the sporting event. To this end, TMS assessment was conducted 24 hours before (baseline) and 1–2 hours after (acute changes) a single football game. We first hypothesized that greater changes of M1 cortical inhibition would be found in athletes who sustained hits during a football game than in control athletes who only participated in a noncontact training session. More specifically, we hypothesized that corticomotor inhibition would be significantly reduced (i.e., corticomotor disinhibition) in athletes immediately after (1–2 hours) the football game compared with their baseline levels. Second, we hypothesized that sustaining more high-magnitude head impacts would be associated with accentuated corticomotor disinhibition.

Methods

Participants

University-level male athletes were recruited from two varsity teams (University of Montréal and McGill University) participating in the Canadian Varsity Football League during the 2021 and 2022 fall seasons. The recruitment was based on a convenience sample: all players from the two teams were approached and participation in the study was voluntary. Study participants received financial compensation. Participants were assigned to either the in-season protocol (protocol 1) or the noncontact training session (protocol 2).

Participants were excluded if they were left-handed or if they had a history of neurological conditions, cognitive deficiencies such as memory loss, trouble concentrating, or a history of attention-deficit disorder. All participants were free of contraindications to the use of TMS.[27] The study was approved by the local ethics committee, and written consent from individual participants and the football clubs was obtained prior to data collection.

Study Design

For the in-season protocol (protocol 1), participants were included over the course of 10 games during two consecutive seasons (fall 2021 and 2022). On average, 3 participants were recruited per game. All participants took part in two 20-minute TMS sessions conducted over 2 days: 24 hours before and 1–2 hours after a single football game. All TMS measurements were performed between 3 PM and 9 PM. HIE over the course of the same game for each participant was measured via the telemetric sensor technologies described below.

For the control group (protocol 2), participants (all varsity Canadian football players) were assigned to a noncontact training session conducted over the course of 5 consecutive sessions during the off-season (winter 2023). On average, 4 participants were recruited per session. All participants took part in two TMS sessions conducted 24 hours before and 1–2 hours after physical activity. The noncontact aerobic training session lasted 1.5 hours. The duration and intensity of this aerobic training session were designed by a certified kinesiologist and intended to mimic the running distance and physical exertion of a football game specific to the different football positions.

TMS Recordings

TMS was performed using a figure-of-eight coil po-

TABLE 1. Characteristics of the study sample, with associated Wilcoxon rank-sum test and p values between the in-season and control groups

	Total Sample (n = 49)	In-Season Protocol (n = 29)	Control Protocol (n = 20)	W Statistic	p Value
Age, yrs	23.0 [22.0, 24.0]	23.0 [22.0, 24.0]	23.0 [22.0, 23.3]	297.0	0.891
Height, cm	185.4 [180.3, 190.5]	185.0 [178.0, 190.5]	190 [185.4, 191.0]	407.0	0.017
Weight, kg	94.8 [84.8, 113.4]	90.7 [83.9, 115.6]	97.3 [87.6, 109.3]	310.5	0.684

Values are presented as median [Q1, Q3].

sitioned over M1 at the optimal position to elicit motor evoked potentials (MEPs) in the contralateral first dorsal interosseus (FDI) muscle. The coil was oriented at a 45° angle from the midline over the left M1 hand area. The optimal scalp site for evoking MEPs from the relaxed right FDI muscle was marked with a washable marker on a white bathing cap to make sure that the coil was held in the same position throughout the experiment. The resting motor threshold was calculated as the minimal stimulation intensity evoking an MEP of at least 50 μV in 6 of 10 consecutive trials.[23] EMG responses to paired-pulse SICI stimulation were recorded.

According to the method of Kujirai and colleagues,[28] a short interstimulus interval of 3 msec was used to test corticomotor inhibition. Twenty consecutive trials were collected for this condition with an interpulse interval of 6–8 seconds. A subthreshold conditioning stimulus set at 80% of the resting motor threshold preceded a supra-threshold test stimulus. This test stimulus was adjusted to produce an averaged MEP of 1 mV of peak-to-peak amplitude.[28] The SICI measure consisted of the ratio of the conditioned test stimulus to the test stimulus alone.[23]

Telemetric Sensor Recordings

Athletes who took part in protocol 1 were equipped with instrumented mouth guards (iMGs; Prevent Biometrics) during a Canadian varsity football game in the 2021 or 2022 regular season (3 players per game) to measure the magnitude of linear accelerations (expressed in gravitational units [*g*]). The specific HIE variables of interest were number of impacts, mean magnitude, and cumulative supra-threshold (i.e., ≥ 10*g*) forces sustained within a game (sum of all impacts in one game per player). Consistent with previous reports,[7,10] only impacts of an intensity ≥ 10*g* were recorded to avoid the detection of false positives due to movement of the head while running or jumping. The iMGs were fitted for each athlete's bite through a standard boil-and-bite process to ensure a secure fit. This telemetric sensor technology had previously been tested and validated during contact sports.[29,30]

Statistical Analysis

Demographic information, HIE data, and TMS variables were subjected to standard descriptive statistics, and potential baseline differences between groups were investigated using the Student t-test or Wilcoxon rank-sum test according to the nature of their distribution. Two-way repeated-measures ANOVAs were used to assess differences in SICI measures between groups (individuals from protocol 1 vs individuals from protocol 2) and across time (24 hours before and 1–2 hours after physical activity) (primary outcome). The threshold for statistical significance was set at $p < 0.05$. Tukey's corrections for multiple comparisons were applied. Two-tailed Pearson correlations were performed to assess the potential relationship between HIE data and SICI (secondary outcome). Finally, we used an iterative approach using arbitrary g-force cutoffs to investigate whether high- versus low-magnitude impacts differently affected the SICI ratio. We set an opportunistic threshold at ≥ 40*g*, allowing an optimal within-group stratification analysis (i.e., players who sustained ≥ 40*g* and those who did not). Potential baseline differences between subgroups were investigated using one-way ANOVAs (three subgroups: players exposed to ≥ 40*g*, players not exposed to ≥ 40*g*, and controls). A series of post hoc analyses were performed to assess whether the number of high-magnitude head impacts (defined as ≥ 40*g*) and cumulative forces from ≥ 40*g* head impacts sustained were linked to SICI modulation measured within 1–2 hours after the game. A false discovery rate correction was applied as follows: with a 0.05 alpha probability for type I error, 3 tests × 0.05 = 0.15 type I errors and 0.85 nonrejections of the null hypothesis; the significance threshold was therefore set at $p = 0.05 \times 0.85 = 0.0425$.

Results

Participants

Forty-nine university-level male athletes (median [IQR] age 23.0 [2.0] years, height 185.4 [10.2] cm, weight 94.8 [28.6] kg) were recruited for the purpose of this study. Twenty-nine athletes took part in the in-season protocol (protocol 1), while 20 athletes were randomly assigned to the control protocol (protocol 2). The two groups were equivalent according to age and weight but differed in height (Table 1). Of the 29 athletes randomly assigned to the in-season protocol, only 1 athlete was diagnosed with a concussion while being equipped with iMG technology. After being evaluated by a member of the football medical team, he was cleared to participate in the postgame TMS session.

Head Impact Exposure

For the entire sample (n = 29), the median [IQR] number of impacts (≥ 10*g*) per player during a football game was 11.6 [15.0], the magnitude of each head impact was 18.9*g* [7.1*g*], and the cumulative force sustained per player

TABLE 2. Head impact exposure: number of impacts, impact magnitude, and cumulative forces for the in-season group (n = 29)

Impact Magnitude Range, *g*	No. of Impacts per Player	Magnitude of Head Impacts per Hit, *g*	Cumulative Forces Sustained, *g*
10–24 (n = 29)	8.6 [5.0, 18.0]	15.0 [14.2, 15.7]	127.8 [87.0, 294.3]
25–39 (n = 25)	2.0 [1.0, 4.0]	30.0 [28.8, 31.3]	61.0 [35.1, 128.7]
40–59 (n = 14)	0 [0, 1]	0 [0, 45.0]	0 [0, 45]
≥60 (n = 5)	1 [1, 1]	71.0 [70.0, 73.0]	71.0 [70.0, 73.0]
≥40 (n = 16)	1 [1, 2]	45.9 [44.9, 77.0]	54.7 [44.9, 111.1]

Values are presented as median [Q1, Q3].

was 234*g* [312.9*g*]. All 29 players were exposed to impacts between 10*g* and 24*g*, 25 players experienced impacts between 25*g* and 39*g*, 14 players experienced impacts between 40*g* and 59*g*, and 5 participants were exposed to impacts ≥ 60*g*. Overall, 16 players were exposed to head impacts ≥ 40*g*, while the remaining 13 did not sustain such impacts. The categorization of HIE according to impact magnitude ranges (i.e., 10*g*–24*g*, 25*g*–39*g*, 40*g*–59*g*, ≥ 60*g*, and ≥ 40*g*) is shown in Table 2.

TMS Results

SICI Following a Football Game

The baseline SICI ratios did not significantly differ between the football group and the control group ($t = -1.33$, $p = 0.189$). Descriptive SICI statistics (Table 3) show that whereas the SICI ratio increased by a mean ± SE of 0.054 ± 0.0614 in the football group in the hours following the football game, it decreased by 0.0704 ± 0.0352 in the control group within the same time interval following the noncontact training session (Fig. 1). A 2 × 2 mixed ANOVA on SICI showed a significant time × group interaction [$F(1,44) = 5.192$, $p = 0.028$, $\eta^2 = 0.106$]. Neither the main effect of groups (football game vs noncontact training) [$F(1,44) = 3.788$, $p = 0.058$, $\eta^2 = 0.079$] nor the main effect of time (24 hours before sport vs 1–2 hours after sport) [$F(1,44) = 0.098$, $p = 0.756$, $\eta^2 = 0.002$] on SICI measures reached statistical significance.

SICI and Head Impact Magnitude

Two-tailed Pearson correlations between HIE (number and cumulative forces) and SICI modulation following a football game were computed for each participant. SICI modulation following the game was found to be unrelated to the number of impacts across low-magnitude impact ranges [< 25*g*: $r(29) = 0.151$, $p = 0.451$; < 40*g*: $r(29) = -0.182$, $p = 0.363$; < 60*g*: $r(29) = -0.253$, $p = 0.202$]. SICI modulation was also unrelated to the cumulative forces of impacts across low-magnitude impact ranges [< 25*g*: $r(29) = 0.128$, $p = 0.526$; < 40*g*: $r(29) = -0.198$, $p = 0.322$; < 60*g*: $r(29) = -0.242$, $p = 0.223$]. However, the observed SICI disinhibition following the game was significantly related to the number of high-magnitude head impacts ≥ 40*g* [$r(29) = -0.397$, $p = 0.041$] and the cumulative forces ≥ 40*g* [$r(29) = -0.468$, $p = 0.014$] after false discovery rate corrections for multiple comparisons were applied (Fig. 2). Furthermore, the observed SICI disinhibition following the game strongly correlated with the number of head impacts ≥ 60*g* [$r(29) = -0.629$, $p < 0.001$] and cumulative forces ≥ 60*g* [$r(29) = -0.648$, $p = 0.014$].

Exploratory descriptive statistics show that, on average, participants who sustained ≥ 40*g* impacts (n = 16) exhibited a mean ± SE SICI disinhibition ratio of −0.112 ± 0.0333, while participants who had not been exposed to ≥ 40*g* (n = 13) during the course of a football game exhibited, on average, a slight SICI inhibition increase (mean 0.0311 ± 0.0477), which better aligned with data collected in the noncontact athlete group (mean 0.0711 ± 0.0503) (Fig. 3). The baseline differences in SICI ratios between these three groups were not significant ($F = 0.798$, $df = 2$; $p = 0.457$). A post hoc between-athlete ANOVA on SICI modulation (SICI 24 hours before the game − SICI within 1–2 hours after the game) with group as the independent variable (football players who sustained ≥ 40*g* hits [n = 16] vs athletes not exposed to ≥ 40*g* hits [n = 13]) reached statistical significance [$F(1,27) = 6.452$, $p = 0.018$, $\eta^2 = 0.205$].

SICI Modulation in a Concussed Athlete

The athlete who was diagnosed with a concussion was taken out of play immediately after sustaining a hit of 44.7*g* as he displayed multiple signs and symptoms of a concussion. Following the football game, the athlete exhibited an SICI disinhibition ratio of −0.21 (SICI 24 hours before: 0.0576; SICI 1–2 hours after: 0.2632). This ratio deviated by 0.18 from the sample median (−0.03).

TABLE 3. SICI modulation measures for in-season and control groups by time points

	24 Hrs Before	2 Hrs After
In-season group (n = 29)	0.219 ± 0.0558	0.273 ± 0.0670
Control group (n = 19)	0.130 ± 0.0366	0.0596 ± 0.0338

Values are presented as mean SICI ratio ± SE.

Discussion

The present study combined iMG technology with TMS to investigate the association between exposure to repetitive subconcussive head impacts and short-term SICI modulation occurring within a few hours after a Canadian varsity football game. This investigation is novel as no controlled study to date has quantified short-term (1–2 hours) SICI modulation following naturalistic HIE during a football game. Our data revealed two main findings supporting our hypotheses: 1) there was a significant group ×

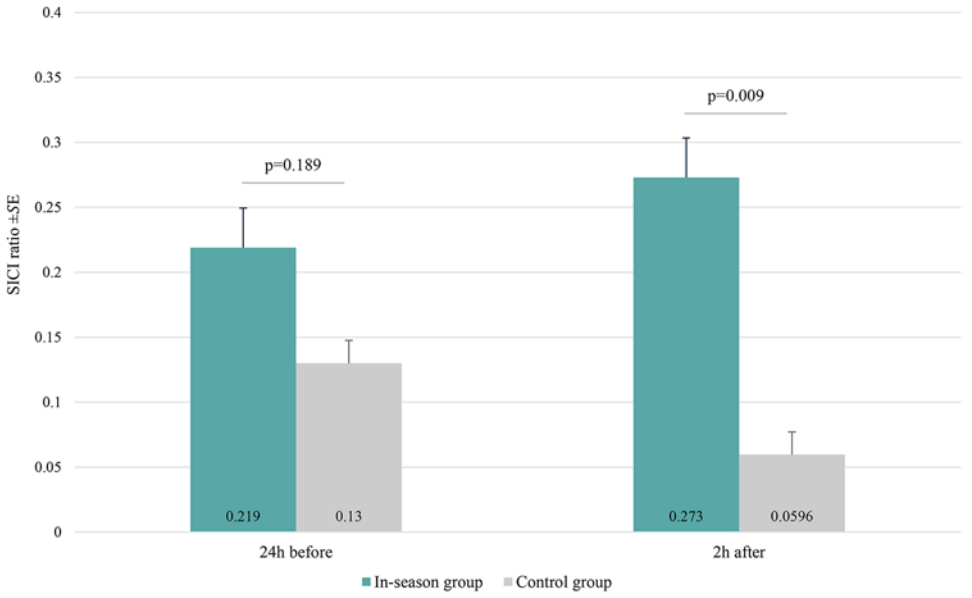

FIG. 1. SICI ratio for in-season and control groups by time points. Between-group p values were calculated using the Student t-test. h = hours. Figure is available in color online only.

time interaction on SICI inhibition in football players, and 2) 1–2 hours after the game, football players exposed to more ≥ 40*g* hits and greater cumulative forces from ≥ 40*g* head impacts exhibited greater postgame SICI disinhibition.

SICI as a Measure of Cortical Excitability Imbalances From In-Game Repetitive Head Impacts

In the present study, football players who sustained ≥ 40*g* hits exhibited similar SICI disinhibition as athletes experiencing postconcussion symptoms.[20] Given robust

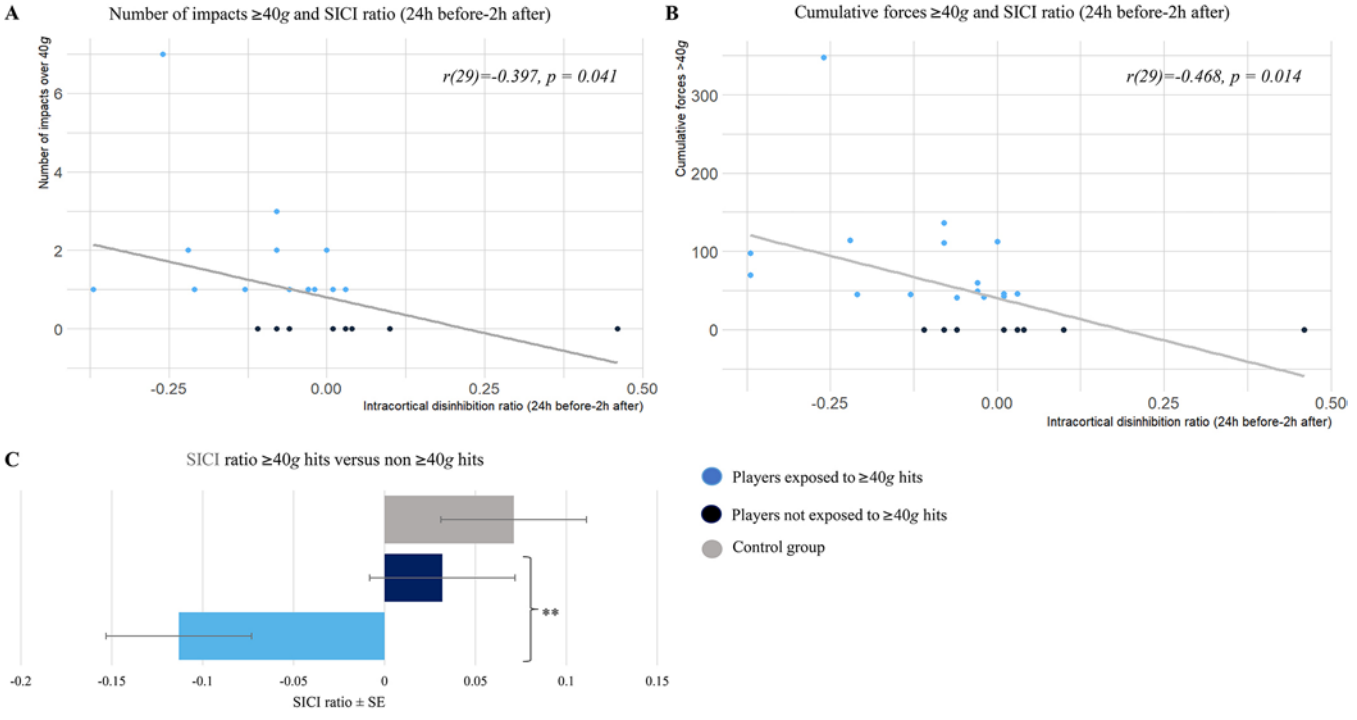

FIG. 2. A: Correlation between the number of impacts ≥ 40*g* and the SICI ratio (24 hours before the football game − 2 hours after the football game). **B:** Correlation between the cumulative forces ≥ 40*g* and the SICI ratio (24 hours before the football game − 2 hours after the football game). **C:** SICI ratio (24 hours before the football game − 2 hours after the football game) of players exposed to ≥ 40*g* hits and players not exposed to ≥ 40*g* hits during a football game. Figure is available in color online only.

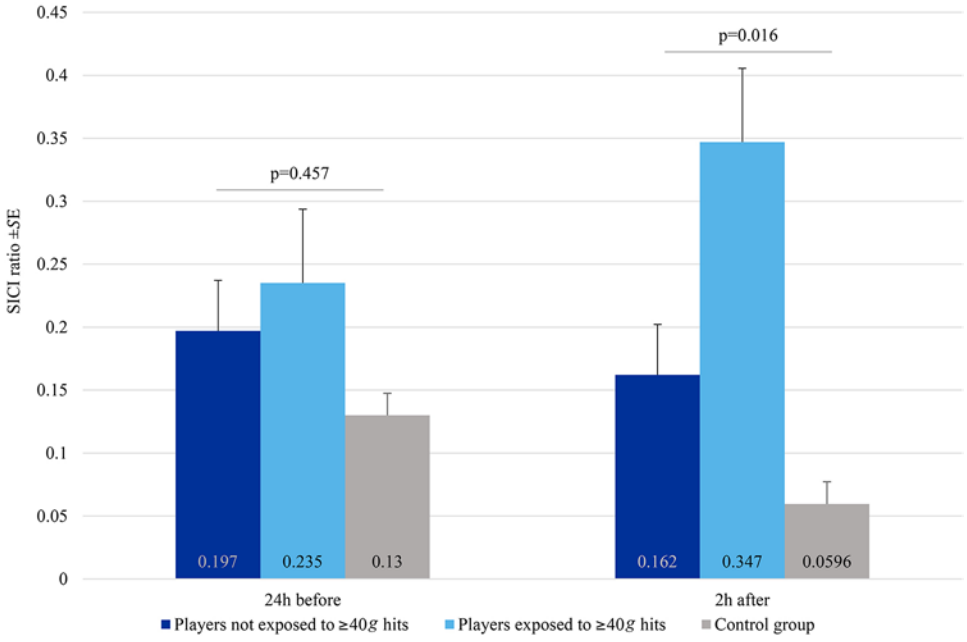

FIG. 3. SICI measures for each group by time points with a stratification in the player group for the players exposed or not to ≥ 40*g* hits during the football game. The p values were calculated using ANOVA. Figure is available in color online only.

evidence suggesting that glutamate/GABA homeostasis underlies SICI modulation, the latter finding raises the possibility that high-magnitude head impacts set off an excitotoxic neurometabolic cascade similar to that classically found following a concussion but without generating observable symptoms.[31] In the acute stages of a concussion, the delicate balance between GABA and glutamate is disrupted by the excessive release of glutamate and a lack of compensation by inhibitory mechanisms including GABA receptor activation. This upsurge in glutamate can overstimulate neurons and cause excitotoxicity that leads to neuronal injury, cell death, and dysfunction of the surviving neurons.[24] The current findings of potentially similar perturbations of this homeostatic balance following repetitive, high-magnitude subconcussive head impacts are important as they provide mechanistic support for the observed cognitive and motor dysfunction associated with prolonged and repeated exposure to subconcussive head impacts. Taken together, these findings identify SICI modulation as an objective physiological marker of HIE repercussions on brain health.

SICI Changes Over Time Following Physical Activity

In the minutes following physical activity, TMS studies have linked significant SICI reduction to M1 plasticity promotion and sport performance improvements.[32] Very little was known, however, about whether such SICI modulation following physical activity was maintained over time. Contrary to our expectations, at 1–2 hours following noncontact aerobic exercise, SICI was found to be slightly increased based on descriptive statistics, a finding that closely resembled what was found in athletes who had not been exposed to ≥ 40*g* head impacts during a football game. The observed SICI disinhibition ratio in the ≥ 40*g* group (n = 16) 1–2 hours following a football game contrasted markedly with results from both the control (n = 20) and < 40*g* groups (n = 13), which therefore accounted for the significant group × time interaction found herein. Taken together, these study findings point to the specific contribution of high-magnitude head impacts, as opposed to exercise or low-magnitude head impacts, to SICI disinhibition found at 1–2 hours following a football game. Moreover, with current evidence that high-magnitude subconcussive head impacts alter M1 intracortical inhibition, the present study suggests that a significant proportion of football players from this study (16 of 29) continued playing with a potentially unfavorable excitotoxic brain state, which could have predisposed them to additional brain sequelae. Once replicated in a much larger sample and validated with alternative neuroimaging tools, the potential glutamate/GABA homeostasis imbalance resulting from high-magnitude head impacts would support the use of iMGs to assist sideline clinical management of head injury risks in contact sports. Among alternative neuroimaging tools, proton MR spectroscopy represents a particularly appealing option, given that it allows the extraction of concentration ratios of neurometabolites of interest, including glutamate/GABA, from any given brain area.[32]

Head Impact Magnitude and Neuroimaging Alterations From Subconcussive Impacts

Multiple studies have reported a relationship between HIE in sports and neurobiological marker changes through the evaluation of measures derived from cognitive testing,[33,34] task-driven and resting-state brain behavior,[34,35]

neurovascular coupling,[36,37] white matter integrity,[29,38–40] and gray matter volume.[41] Previous TMS studies have shown changes in M1 excitatory and inhibitory mechanisms following simulated HIE exposure in soccer players,[25] amateur boxers,[26] and rugby players.[4] Moreover, studies in college athletes who play contact sports have revealed microstructural alterations in white matter[42] and white matter diffusivity[43] during a single season in the absence of a history of concussion. Another study showed postseason changes in cerebral blood flow, cerebral connectivity, and the likely presence of microhemorrhages, which were all associated with higher levels of HIE in college soccer players.[44] However, no study to date has suggested the potential existence of a subconcussive susceptibility range beyond which brain damage can be expected. In the present study, the demonstration that head impact magnitude ≥ 40*g* is significantly associated with the observed cortical disinhibition represents preliminary evidence for such a head impact magnitude susceptibility range. Validating ≥ 40*g* as a potential marker of M1 excitability imbalance could justify further research into the functional as well as pathophysiological repercussions of this marker, especially in professional athletes exposed to hundreds or even thousands of such high-magnitude head impacts throughout their long-lasting careers.

Corticomotor Disinhibition of a Concussed Athlete

Only one of the 29 athletes was diagnosed with a concussion after sustaining a 44.7*g* impact during the game; he was wearing an iMG. Interestingly, SICI assessment within 1–2 hours after the game showed a substantial SICI disinhibition ratio (with the concussed athlete deviating by 0.18 from the sample median). Although one should be cautious when interpreting any finding from a single participant, conducting a similar prospective study using SICI and iMGs with a much larger sample could be useful to assess the potential contribution of short-term SICI modulation to the severity and longitudinal evolution of concussion symptomatology.

Limitations

The present work comprises several limitations that should be considered before generalizing the results to the entire population of contact sport athletes. First, as the players were tested following a naturalistic Canadian football game, reproducing the competitive setting and its high-intensity dynamics (including stress, motivation, attention load, and general performance) in our control group was challenging. These differences are likely to have influenced SICI modulation. Second, the size of subgroup samples could have been better harmonized in order to enhance the robustness of the statistical tests conducted. Moreover, future studies could benefit from contrasting football players according to their playing position given that position introduces considerable heterogeneity notably in terms of anthropometric variables (i.e., weight, height, neck circumference, etc.), head impact kinematic characterization, and exposure. Furthermore, collecting data in a larger sample would have likely allowed us to include more concussed athletes in our studied sample so as to improve characterization between subconcussive and concussive impacts on corticomotor disinhibition. However, to our knowledge, this study is among the first to examine the relationship between HIE, including impact magnitude as measured by iMGs, and M1 corticomotor inhibition.

Conclusions

The results of the present study show alterations in corticomotor inhibition within the motor cortex within 1–2 hours after repetitive subconcussive head impacts in a controlled study conducted in varsity football players. Our findings provide evidence that high-magnitude impacts disturb M1 intracortical inhibition. The underlying neurophysiological mechanisms could not be evaluated in the present setting. Future studies are needed to directly investigate whether or how M1 intracortical inhibition may implicate changes in one's brain susceptibility to concussion risks, integrity, or function. Our results collected in a single game raise concerns about the long-term health of athletes exposed to hundreds of head impacts ≥ 40*g* during the course of a single season. The repetitive nature of subconcussive hits beyond this potential ≥ 40*g* head injury susceptibility range is further potentiated in professional contact sport athletes, which could help explain the recently demonstrated association between lifelong repetitive HIE and the development of brain alterations with advancing age. Finally, this study provides additional evidence for the pertinence of implementing sideline iMG tracking in contact sports to assist in the clinical management of injury risks.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Conception and design: De Beaumont, Vinet, Guay, Apinis-Deshaies, Merbah, Caré. Acquisition of data: Vinet, Guay, Apinis-Deshaies, Caré, Corbin-Berrigan, Wagnac. Analysis and interpretation of data: De Beaumont, Vinet, Martens, Guay, Merbah, Caré. Drafting the article: Vinet, Martens. Critically revising the article: De Beaumont, Vinet, Martens, Guay, Apinis-Deshaies, Merbah, Caré, Wagnac. Reviewed submitted version of manuscript: De Beaumont, Vinet, Martens, Guay, Apinis-Deshaies, Corbin-Berrigan, Wagnac. Approved the final version of the manuscript on behalf of all authors: De Beaumont. Statistical analysis: De Beaumont, Vinet, Guay. Administrative/technical/material support: De Beaumont, Vinet, Guay, Apinis-Deshaies, Merbah, Caré, Corbin-Berrigan. Study supervision: De Beaumont, Vinet, Apinis-Deshaies, Corbin-Berrigan.

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