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Abstract

Purpose: To investigate how hamstring injuries affect brain functional connectivity (FC) and identify potential biomarkers for injury assessment and rehabilitation. Methods: Brain activity was recorded during a rigorous motor task using electroencephalography in 129 footballers. Demographic, anthropometric, injury, and football-related data were also collected. Brain FC was calculated separately for the rest and activity periods. A 2-way mixed analysis of variance was conducted for group comparisons, and a partial correlation analysis examined links between FC and injury parameters. Results: The execution of the motor task led to a significant decrease in alpha-band FC during activity compared with rest (injured: $P < .0001$, $\eta p2 = .38$; control: $P < .001$, $\eta p2 = .11$). Injured players showed significantly lower FC during activity ($P = .006$, $\eta p2 = .07$) as well as a greater decrease from rest to activity ($P < .001$, $\eta p2 = .13$), particularly in frontal ($P < .001$, $\eta p2 = .17$) and temporal ($P = .03$, $\eta p2 = .08$) regions. There were significant inverse correlations between the injury severity index and global ($P = .003$, $r = -.58$), frontal ($P < .001$, $r = -.72$), and parietal ($P = .015$, $r =$

−.59) connectivity. Conclusion: Reduced FC in footballers with previous hamstring injury suggests an increased cognitive effort required for task execution, namely, in regions associated with motor planning and movement sequencing. The correlation analysis results point to a relationship between the age and severity of the injury and the degree of this cognitive effort increase.

Keywords: electroencephalography, fast movement, hamstring injuries, football

The incidence of hamstring injuries (HIs) continues to grow among professional football players.[1] This injury is known to negatively impact player and team performance[2–4] and to have a high financial cost.[5] Hamstring injuries are defined as a sudden onset of posterior thigh pain during activity that is reproduced with hamstring stretching and/or activation,[6] which lead to training or match time loss,[7] and often occur during actions involving fast muscle actions.[8]

Hamstring injuries have also been reported to have a high recurrence rate,[1] and parameters such as injury severity on physical examination or imaging and strength measurements have shown a poor or uncertain association with recurrence.[9] Conversely, persistent neuromuscular inhibition has been strongly suggested to contribute to recurrence.[10,11] This inhibition is thought to arise from local damage and to serve as a protective mechanism to reduce pain and tissue load after injury.[10] However, local muscle pain may cause chronic supraspinal adaptations,[12] affecting the voluntary recruitment ability in the long term.[13] Di Trani[14] suggested that HIs may damage mechanoreceptors, leading to postdeafferentation cortical remodeling. Such cortical changes could significantly impact the integration of proprioceptive input, which is crucial for muscle control and coordination, especially during rapid actions such as sprinting.[15] This notion is supported by studies indicating that

proprioceptive, tactile, and spatial deficits derived from hamstring pain are associated with cortical reorganization in regions processing lower-limb sensory information.[16–18] Moreover, Australian football athletes with a previous HI have shown impaired joint position sense and leg swing movement discrimination.[19] Despite these findings suggestive of post-HI cortical adaptations across multiple regions, research on the relationship between HIs and changes in brain activity is scarce. Accordingly, the recent London International Consensus on "Hamstring Injuries: Rehabilitation, Running and Return to Sport" emphasized that, beyond peripheral factors, further research is needed to clarify the role of central nervous system changes in HIs.[20] Existing research on electroencephalography (EEG) signal changes after musculoskeletal injuries does, however, provide encouraging evidence of how these adaptations may also be present in HIs.[21–24] Zhang et al[21] investigated cortical activity changes in soccer players with chronic ankle instability and found differences in frontal theta (but not in alpha) power during drop-jump landing compared with healthy controls, suggesting that lower-limb joint instability induces band-specific adaptations. Similarly, Baumeister et al[22,23] studied altered EEG activity after ACL reconstruction during force control and joint position sense tasks and found higher frontal theta power in both tasks and lower parietal alpha power during force reproduction tasks in the affected knee of injured individuals.[22,23] Finally, a previous study using the same task identified frontal and central EEG power differences in the theta and alpha bands in footballers with previous HI.[24] These studies highlight the task dependency of cortical activation changes following lower-limb injuries, emphasizing the role of theta and alpha oscillations in compensatory neural mechanisms. However, these studies primarily used power analysis and did not specifically examine the relation among brain activity in different regions, that is, functional connectivity (FC).

Although power analysis of isolated regions provides valuable information about localized cortical

activity, it offers a limited view of overall brain function. Neural processes underlying motor control and injury adaptation are inherently network-based, depending not only on activity within individual regions but also on the coordination between them.[25] FC captures these relational dynamics by quantifying how signals are synchronized across brain areas,[26,27] providing a closer proxy to how information is processed and transferred through distributed neural circuits. Importantly, FC measures can reveal subtle neurocognitive differences that might remain undetected in power analyses, as shown in studies where connectivity alterations better predicted behavioral and clinical outcomes than regional activation alone.[28,29] By examining FC, we move beyond regional activation patterns to understand how injuries alter the integration of motor, sensory, and cognitive resources across the brain, which is particularly relevant given the brain-wide nature of motor planning and sensorimotor integration.[30,31] In EEG, FC can be derived by estimating statistical dependencies between channels (eg, phase/amplitude coupling), and graph-theoretical measures then summarize network organization (eg, degree) to characterize large-scale brain network dynamics.[27]

To the best of our knowledge, studies examining brain connectivity in athletes with previous HIs are nonexistent, and those involving other injuries do not assess FC during movement and rely on task-based functional MRI.[32,33] Given the scarcity of research relating brain FC to lower-limb injuries (including HIs), the primary objective of the present study was to investigate whether footballers with and without an HI history show differences in FC during a maximum-speed knee flexion–extension task. We hypothesized that FC metrics would differ between these groups, reflecting potential long-term impacts of HIs on brain function. In addition, we explored whether these changes correlated with injury severity (ie, time loss due to injury) and the injury age (ie, time difference between testing and injury date), based on the assumption that both the magnitude of the injury and the time since it occurred may influence brain adaptive responses. By addressing these hypotheses, we aim to improve the

understanding of how HIs can affect brain FC and to identify potential biomarkers for injury assessment and rehabilitation.

Material and Methods

Study Design and Participants Recruitment

A retrospective cross-sectional study design was employed at the start of the 2021/2022 football season to accomplish the study objectives. The study was advertised among local football male teams. Players with at least 5 years of football practice, a minimum of 3 training sessions plus a match per week, and no active injury limiting performance were invited to participate in this study. Goalkeepers and players with a history of knee, thigh, hip, or central nervous system structural injury or surgery; who practiced other sports or structured physical activity twice per week or more in the last 2 years; or who had any condition preventing the player from completing the study protocol were excluded. All participants provided a signed written consent form before performing the tests. This study was approved by the local ethics committee (#15/2021).

Protocol

Prior to testing, all participants completed a standardized warm-up consisting of 5 minutes of stationary cycling at a comfortable pace (~70 rpm). Individuals were then familiarized with the maximum knee flexion–extension movement rate (MR) task in a prone position, which has been described previously.[34] Participants were instructed to perform alternating repeated flexion–extension movements with both legs as fast as they could between 45° and 90° of knee flexion (Figure 1A). The task consisted of eight 10-second blocks of fast bilateral alternating knee flexion–extension movements with a 5-second rest between blocks. The 10-second duration was chosen because this period has been

found to show the greatest decrease in MR.[35] The knee flexion–extension maximal MR was measured in hertz by an accelerometer placed at the ankle,[34] and the rating of perceived exertion (RPE) was measured by a modified 0 to 10 Borg scale.

Clinical Anamnesis

A sports physiotherapist with more than 10 years of professional practice questioned all participants regarding their demographic, anthropometric, injury, and football-related data. Regarding the HIs specifically, information on the date of injury occurrence, context, mechanism, time loss (days away from play due to injury), injury age (days between injury date and testing), and injured limb were obtained. An injury severity score was defined based on the time loss: 1: minimal, 1–3 days; 2: mild, 4–7 days; 3: moderate, 8–28 days; and 4: severe, >28 days.[7] To encompass both measures in a single variable, we defined an injury severity index as the injury severity score minus the injury age times an arbitrary constant chosen such that the index would be strictly positive (C = 0.1). A retrospective period of 2 seasons has previously been used in studies of football-related HIs.[36,37] In addition, the accuracy of HI self-reporting has been previously confirmed.[38]

Brain Electrical Activity

Electroencephalography data were collected during the whole task using a Vertex SC823 device (Meditron Eletromedicina Ltda). Cz alignment was performed using the midpoints of the inion and nasion in the sagittal plane and the 2 preauricular points in the coronal plane as reference, with the remaining electrodes placed according to the international 10 to 20 system. A total of 24 channels were used. Online referencing to 2 mastoid electrodes was performed, and the sampling rate was 250 Hz.

A circuit impedance of 10 kΩ was ensured in all electrodes prior to starting data collection, and a 0.1 to 70 Hz analog band-pass filter was applied by the amplifier.

EEG Signal Analysis

The EEG signal underwent preprocessing using the HAPPILEE pipeline, a standardized software for low-density EEG data processing.[39] A 50-Hz line noise reduction was performed using the CleanLine method. Subsequently, data were filtered with a 1to 100-Hz band-pass filter using EEGLAB's FIR filter (https://sccn. ucsd.edu/eeglab/), ensuring the removal of slow drifts and fast noise components. The pipeline also incorporated wavelet thresholding with default settings to denoise the EEG signals in the time– frequency domain. The MuscIL feature of HAPPE[40] was utilized to specifically address and remove muscle artifacts that frequently contaminate EEG recordings. Finally, the EEG data were rereferenced to the average of all electrodes, a standard procedure that offers a neutral reference and improves the clarity of the EEG signal.

Functional Connectivity

Pairwise connectivity metrics were calculated for all electrodes for each epoch in the following frequency bands: theta (4–7 Hz), alpha (8–13 Hz), and beta (13–30 Hz). In this analysis, we employed the Weighted Phase Lag Index to estimate the FC between the neural activities of each brain region.[41] The Weighted Phase Lag Index is a measure of phase synchronization between a pair of signals. Connectivity matrices were derived for each individual and epoch, where each matrix element represented the Weighted Phase Lag Index value between a pair of EEG channels.

Graph Analysis

Graph theory allows for a deeper understanding of the dynamic architecture of neural networks.25 By transforming the connectivity matrices into weighted graphs (nodes representing electrodes and edges depicting the FC value of a given pair), we can derive quantitative measures that reveal important features of brain-wide neural connectivity. For a weighted graph, the nodal degree measures the connectivity strength of a given node.25 The degree can be assessed both globally (average degree across all nodes in the network) and locally (average degree across a subselection of nodes; Figure 1B), providing a comprehensive perspective of the graph's topology. We calculated the rest and activity degrees by averaging the degree from each corresponding epoch (5-s blocks for both rest and activity), and the degree difference was determined as the average value of the percent degree change from each transition between a rest to activity epoch.

Statistical Analysis

All statistical analyses were conducted using Python libraries such as SciPy and Statsmodels. A 2-way mixed analysis of variance (2 [HI and control] × 2 [activity and rest]) was conducted to compare the global degree between injured athletes and the control group for each EEG frequency band of interest. To identify spatial characteristics and differences between groups, a 2-way mixed analysis of variance (2 [HI and control] × 6 [networks]) was conducted to compare local degree changes across 6 subnetworks (frontal, prefrontal, parietal, central, occipital, and temporal; Figure 1B) and between the injured and control groups. Normality tests were performed for all distributions to ensure the validity of parametric analyses. A partial correlation analysis was conducted to examine the relationship between 4 injury parameters (time loss, injury age, severity score, and severity index) and global degree change, as well as degree changes across all networks, while controlling for the knee task MR, RPE, and age. Bonferroni correction

was applied for multiple comparisons, with significance set at $P < .05$. Effect sizes were calculated using partial eta-squared ($\eta p2$) values and classified as small (.01–.06), medium (.06–.14), or large (>.14).[42]

Results

A total of 129 individuals (24.3 [4.2] y old) were recruited to participate in the study, of which 21 did not meet the study criteria (13 goalkeepers and 8 players with previous structural knee injury) and 4 did not complete the experimental task. After preprocessing the data, 15 subjects did not meet a satisfactory signal-to-noise ratio and were excluded due to excessive noise in the EEG recordings. Thus, a total of 89 players (24.1 [4.0] y old) were included in the study, from which 30 players (42.7%) had a history of HI in the previous 2 seasons and 59 acted as controls. Shapiro–Wilk normality tests indicated that all reported dependent variables did not significantly deviate from normality (all $P > .05$). Therefore, parametric analyses (analyses of variance and Pearson correlations) were considered valid.

Task-Related FC Changes

The execution of the knee flexion-extension task generally led to a significant decrease in global degree at the alpha band during activity compared with rest for both groups (injured: $P < .0001$, $\eta 2 = .38$; control: $P = .0003$, $\eta 2 = .11$; Figure 2A). There were no significant spatial characteristics associated with this decrease ($P > .05$), and it was not correlated with task performance or reported perceived fatigue (see Supplementary Figure S1 in Supplementary Material [available online]). We found no significant changes in the theta or beta frequency bands.

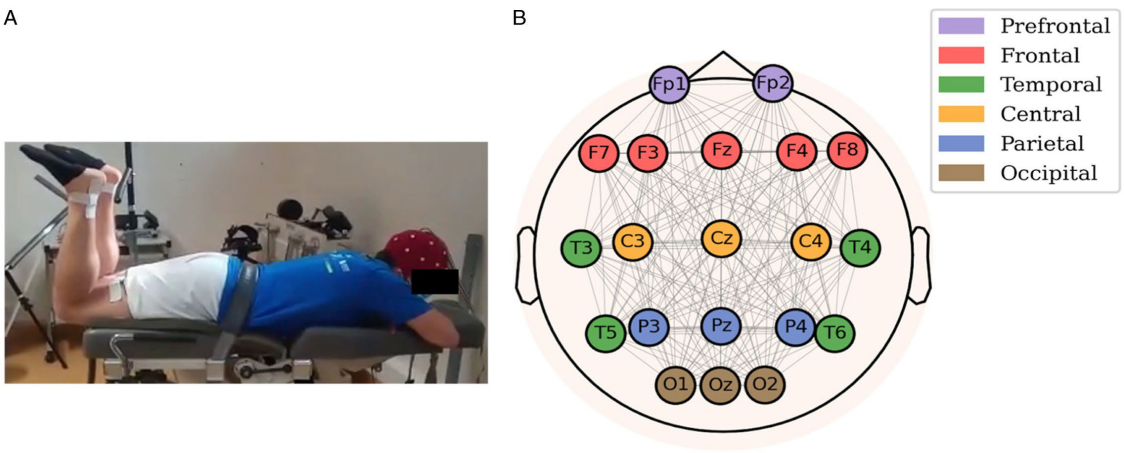

Figure 1 — (A) Experimental setup utilized in the maximum knee flexion–extension motor task protocol employed in the study. (B) Illustration of the electrode layout showing the electrode grouping into different networks used for statistical analysis.

HI Versus Control Groups

When comparing the injured and control groups, there was a significant difference in the percentage change in global degree from rest to activity at the alpha band, with the injured group showing a greater decrease in connectivity ($P = .0006$, $\eta p2 = .13$; Figure 2B). For the rest epochs, there were no significant differences between groups ($P = .2$, $\eta p2 = .02$; Figure 2A), whereas for the activity epochs, the injured group presented a significantly lower connectivity ($P = .006$, $\eta p2 = .07$; Figure 2A). When comparing the subnetworks, we found that the injured group presented a significantly greater decrease in connectivity in the frontal ($P = .0004$, $\eta p2 = .17$; Figure 2C) and temporal ($P = .03$, $\eta p2 = .08$; Figure 2C) regions when compared with the control group. We found no significant group differences in the theta or beta frequency bands. We also found no significant group differences in task performance (MR) or perceived fatigue (RPE) (see Supplementary Figure S2 in Supplementary Material

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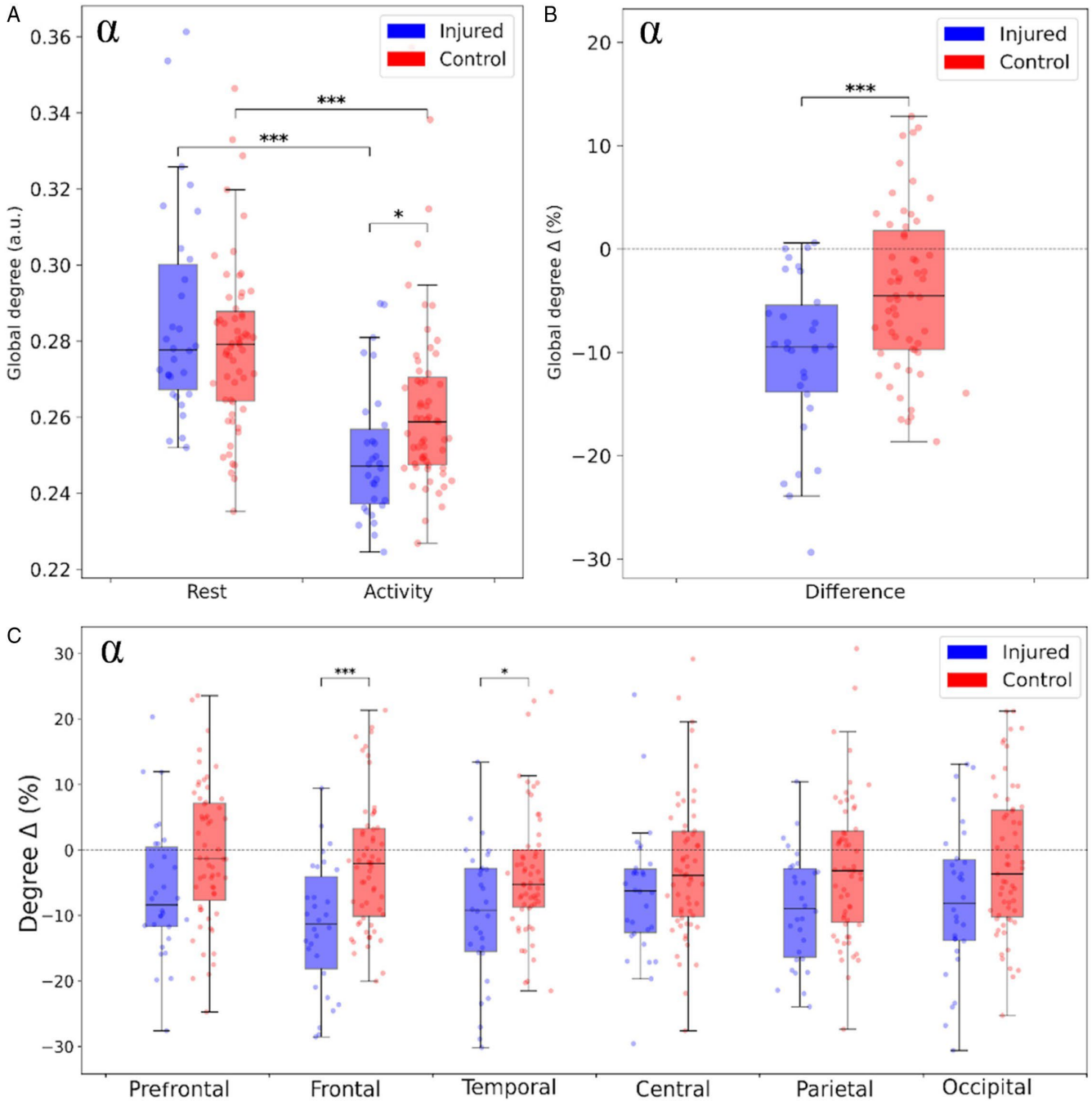

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223 Figure 2 — Boxplot comparing (A) the global degree at alpha band between the task activity and rest

epochs, (B) the percent change in global degree when transitioning from rest to activity, and (C) the percent change in local degree for each of the 6 networks for both injured (blue, left) and control (red, right) groups. Statistical significance: ***$P < .001$. **$P < .01$. *$P < .05$.

Injury Parameters Correlations

The results from the partial correlation analysis are summarized in Table 1. No significant correlation was found between global alpha degree and either the time loss due to injury ($P = 1$, $r = -.14$; Figure 3A) or the injury age ($P = 1$, $r = .16$; Figure 3B). However, significant negative correlations were observed between global alpha degree change and the severity score ($P = .023$, $r = -.49$; Figure 3C) and the injury severity index ($P = .003$, $r = -.58$; Figure 3D). The most significant correlations were found in the prefrontal (severity score: $P = .045$, $r = -.54$; severity index: $P = .077$, $r = -.52$; Figure 4B), frontal (severity score: $P = .14$, $r = -.49$; severity index: $P < .001$, $r = -.72$; Figure 4A), and parietal (severity score: $P = .011$, $r = -.60$; severity index: $P = .015$, $r = -.59$; Figure 4D) regions. We found no significant correlations in the theta or beta bands.

Discussion

The primary aim of this study was to investigate whether footballers with a history of HI exhibit alterations in brain FC during the execution of a maximum-speed knee flexion–extension task. In line with our hypothesis, we found that alpha-band FC decreased during motor activity compared with rest in all players, but this reduction was significantly greater in those with a previous HI. These differences were most pronounced in the frontal and temporal regions, which are key for motor planning and sequencing. A secondary aim was to assess the relationship between FC alterations and clinical variables related to injury.

Here, we observed significant negative correlations between injury severity indices and FC, particularly within frontal, prefrontal, and parietal networks. Together, these findings indicate that HIs are associated with enduring changes in large-scale brain network dynamics, supporting the idea that long-term cortical adaptations might take place as a consequence of musculoskeletal injuries.

Alpha Connectivity Reduction During Motor Activity

The main FC trend observed in our study was a significant decrease in global degree during activity compared with rest for both groups in the alpha frequency band (Figure 1A), indicating a widespread reduction in alpha connectivity associated with motor activity. Decreased alpha power and FC have been associated with increased perceived mental workload and task demands/complexity.[43,44] The reported mechanisms behind this decreased connectivity include a top-down reduced cortical disinhibition as a response to the need to allocate additional resources to task performance (eg, attentional, visuospatial, and sensorimotor coordination resources).[43]

The injured group exhibited a significantly greater decrease in global alpha connectivity compared with the control group (Figure 2B). When examining the global degree during rest and activity, there was a significant difference only during the activity phase (Figure 2A), indicating that the observed differences were predominantly due to FC variations during the execution of the motor task. Our findings of decreased alpha FC in footballers with an HI history may mean that these players need to use more cortical resources, that is, a greater working memory load, to cope with the demands of this maximal task.

Two hypothetical mechanisms may be behind these differences: (1) players with an HI history show a lower neural efficiency (whether pre or postinjury) and therefore allocate more cortical processing resources to a given task or (2) the injury led to the need to dedicate more attention to spatiotemporal

joint parameters during task execution due to the greater reliance on top-down (internal motor programs) rather than on bottom-up (peripheral proprioceptive afferences) control. Existing evidence suggests that alpha cortical communication is decreased in less proficient motor performance.[45,46] In our study, however, all comparisons were adjusted for task performance and RPE, making it unlikely that differences in execution account for the observed effects. This interpretation is further supported by the absence of any correlation between task performance and global alpha connectivity decrease. It therefore seems more likely that the increased workload shown by these players is due to the need to allocate more resources (perhaps for sensorimotor integration and monitoring of joint parameters) to cope with task demands. This physiological interpretation of FC findings across multiple brain regions in this study reinforces the findings of previous work in footballers that also highlighted the possibility of greater task load and sensorimotor integration resource usage in footballers with previous HI.[24] This is supported by the fact that differences were only seen during activity and were most evident in the frontal and temporal regions, which play an important role in movement planning, inhibition, and timing[47,48] and have been proposed to form a visuospatial processing network.[31] In any case, this greater resource use inevitably leads to a lower motor–cognitive reserve, thus decreasing the ability to cope with additional and/or unexpected demands during athletic performance,[49] which potentially increases the injury risk.~

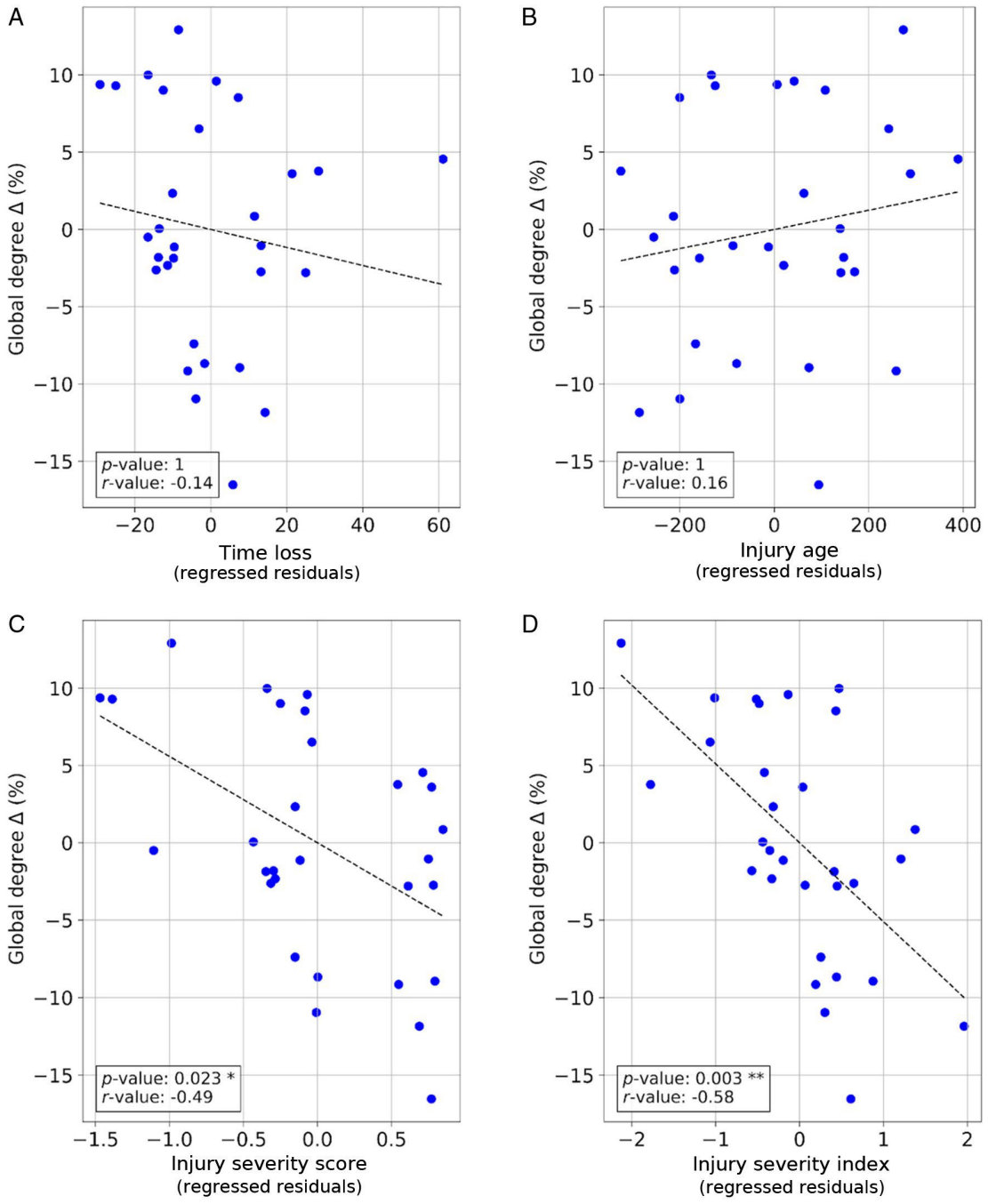
FC and Clinical Variables Correlations

Based on the hypothesis that a more severe injury would lead to greater FC alterations, whereas a longer injury age would diminish

Table 1 Results From the Partial Correlation Analysis of Injury Parameters and Network Degree Changes at Alpha Band (n = 30)

EEG network region	Injury age (days since injury)	Time loss (days away due to injury)	Severity score	Severity index
Global	.16, (−.21 to .49), 1	−.14, (−.48 to .23), 1	−.49, (−.72 to −.16), .023*	−.58, (−.78 to −.28), .003**
Prefrontal	.08, (−.29 to .43), 1	−.15, (−.48 to .22), 1	−.54, (−.76 to −.23), .045*	−.52, (−.74 to −.2), .077
Frontal	.33, (−.04 to .61), 1	−.12, (−.46 to .25), 1	−.49, (−.72 to −.16), .139	−.72, (−.86 to −.49), .00016***
Parietal	−.01, (−.37 to .35), 1	−.4, (−.67 to −.05), .66	−.6, (−.79 to −.3), .011*	−.59, (−.78 to −.29), .015*
Central	.09, (−.28 to .44), 1	−.19, (−.51 to .18), 1	−.36, (−.64 to .0), 1	−.37, (−.65 to −.02), 1.00
Temporal	.14, (−.23 to .47), 1	.11, (−.26 to .45), 1	−.15, (−.49 to .22), 1	−.27, (−.58 to .1), 1
Occipital	.26, (−.11 to .57), 1	−.03, (−.39 to .33), 1	−.27, (−.58 to .1), 1	−.41, (−.67 to −.06), .55

Abbreviation: EEG, electroencephalography. Note: Data are presented as: *r* value, 95% CI, and *P* value. Bold cells contain significant *P* values ($P < .05$).



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299 Figure 3 — Linear fits between global degree change (%) at alpha band and the regressed residuals of

300 (A) injury time loss (days away due to injury), (B) injury age (days since injury), (C) injury severity

301 score, and (D) injury severity index.

this effect, we proposed an injury severity index that encompasses both variables. Our analysis revealed that the injury severity index demonstrated stronger correlations with the percent global degree change compared with any single metric alone (Figure 3), suggesting that this combined measure serves as a more accurate predictor of the injury's impact on alpha connectivity. These correlations were significant in the frontal, prefrontal, and parietal regions, suggesting that HIs that led to greater time loss and were more recent induced greater adaptations in these regions, which form a crucial network for motor planning, execution, and sensorimotor integration.[30,47] Assuming that HIs could lead to cortical adaptations by damaging muscular mechanoreceptors,[14] then more severe and/or recent injuries could induce more damage and cause greater sensorimotor integration difficulty.[15] This would then lead to a greater need to allocate cortical resources to monitor task performance, as shown by the greater decrease in alpha FC.

This proposed model of increased cortical resource allocation following HIs is supported by our finding that this correlation was most significant in the frontal and parietal networks (Figure 4A and 4D). The frontal network corresponds primarily to the premotor cortex, a region responsible not only for the selection, planning, and execution of movement[50] but also for cognitive functions such

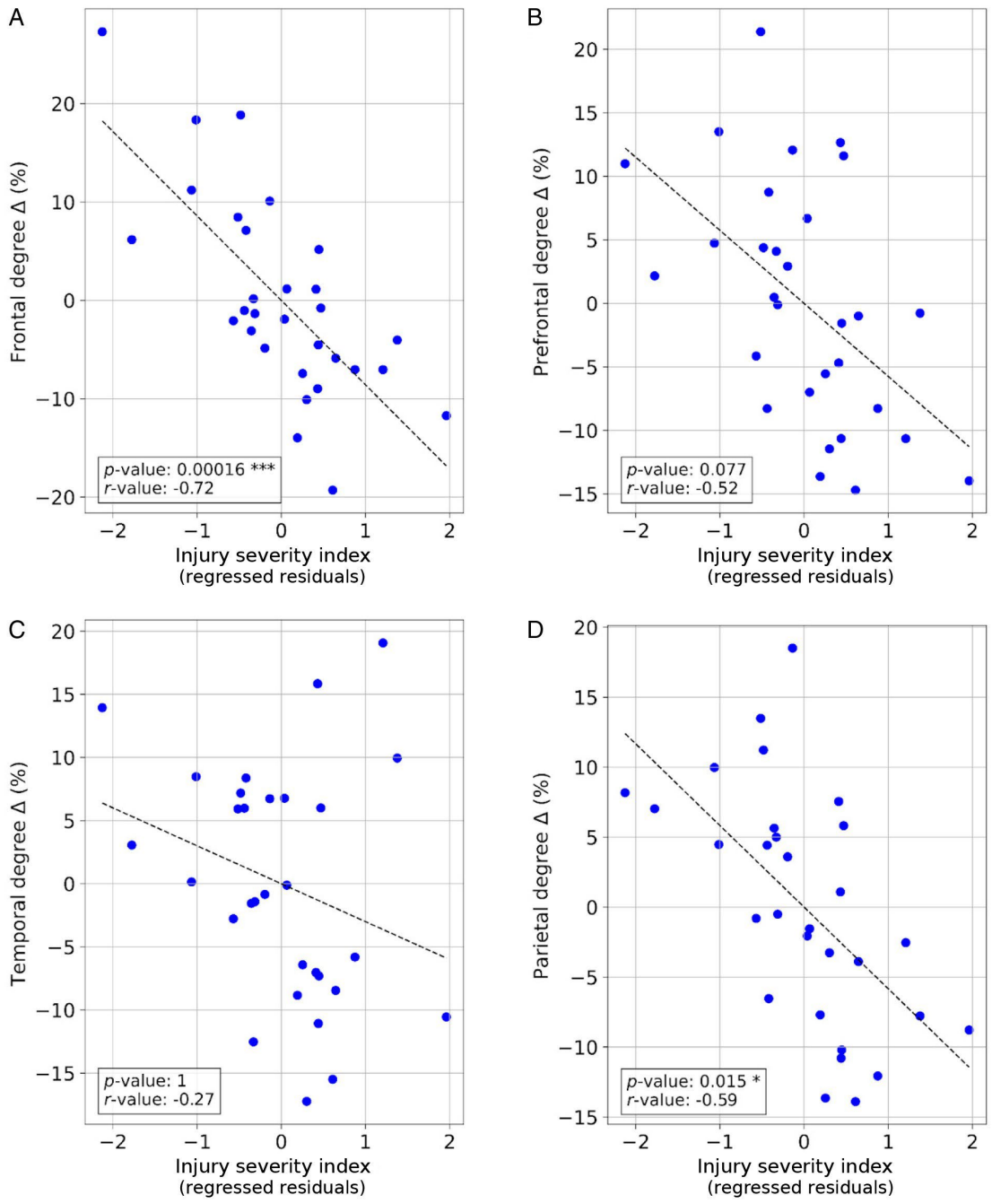

Figure 4 — Linear fits between the regressed residuals of the injury severity index and the (A) frontal, (B) prefrontal, (C) temporal, and (D) parietal degree change (%) at alpha band.

as spatial attention and working memory.[51] The parietal network is mainly comprised of the superior parietal lobe, a region thought to be essential for sensorimotor integration and for maintaining internal representations of the body's current state.[52,53] Overall, the key functions of the main affected regions strongly support the hypothesis that the greater alpha FC reduction seen in the HI group reflects an increased cognitive effort requirement for motor planning and sensorimotor integration during the execution of the motor task.

Alternatively, it is possible that individuals with naturally higher working memory demands during motor activity are more susceptible to injuries. This hypothesis aligns with research suggesting that variations in neural activity and connectivity can influence an individual's susceptibility to injury, as neural mechanisms play a crucial role in maintaining motor coordination and stability.[32,33,54] Further research is needed to explore this potential bidirectional relationship and to determine whether alpha connectivity reduction during motor activity can be used as a predictive marker for injury risk.

Study Limitations

Although our study provides valuable insights into the impact of HIs on brain FC in professional footballers, some limitations must be acknowledged. First, the cross-sectional design of the study limits our ability to draw causal inferences about the relationship between HIs and changes in FC. Longitudinal studies are needed to establish temporal relationships and track changes over time. Second, self-reported injury history may be subject to recall bias, which could impact the accuracy of the injury severity data. Addressing these limitations in future research may allow us to deepen our understanding of the neural consequences of HIs and enhance strategies for injury prevention and rehabilitation.

Practical Applications

Our findings suggest that adding a neurocognitive component in HI rehabilitation and recurrence prevention may be advantageous, especially aiming at promoting effective attentional resource use and at decreasing cognitive load during knee high-speed movements. In addition, the differences in brain FC that we found showcase how the entire motor pathway can contribute to or be affected by HIs, suggesting that any interventions should also target all stages of movement planning and production. Finally, considering the early stages of research on the role of differences in brain activity and connectivity in HIs, our study can also serve as a stepping stone for researchers in this field. Prospective studies on the value of FC as a biomarker of HI risk are particularly warranted. Other aspects that require further investigation in light of our findings also include a more detailed analysis according to injury classification and expanding to female football and other sports where HIs are common (eg, rugby).

Conclusion

For the first time, we have measured FC patterns during the execution of a maximum-speed motor task and were able to detect the influence of an HI on brain-wide and network-specific graph metrics. We suggest that the observed connectivity differences in the injured group reflect an increased cognitive effort requirement to perform the task. Considering this model of HIs, subsequent studies should employ a longitudinal design and include sensory inputs and decision-making components to the motor task, designed to manipulate the working memory load and validate or refute the proposed model. Resolving these questions would represent 1 step further in the direction of using EEG-based connectivity analysis as a tool for developing targeted rehabilitation protocols and injury prevention strategies tailored to the

cognitive profile of each athlete.

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