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Brain Connectivity, Acute Post-Concussion Symptoms, and Cognition in Adults With Concussion: A Quantitative Electroencephalography Study

Francesca Buhagiar[1], Melinda Fitzgerald[2,3], Sarah C. Hellewell[2,3], Jason Bell[1], Samantha Moore[1], Aleksandra K. Gozt[2,3], Jacinta Thorne[2,3,4], Elizabeth Thomas[5,6], Antonio Celenza[6], Dan Xu[5,7,9], Suzanne Robinson[10,11], Gill Cowen[7], Michael Bynevelt[8], Daniel M. Fatovich[12], and Carmela F. Pestell[1,2]*

[1]School of Psychological Science, University of Western Australia, Perth, Western Australia, Australia
[2]Curtin Health Innovation Research Institute, Curtin University, Bentley, Western Australia, Australia
[3]Perron Institute for Neurological and Translational Science, Nedlands, Western Australia, Australia
[4]School of Allied Health, Faculty of Health Sciences, Curtin University, Bentley, Western Australia, Australia
[5]School of Population Health, Curtin University, Perth, Western Australia, Australia
[6]Medical School, The University of Western Australia, Perth, Western Australia, Australia
[7]Curtin Medical School, Faculty of Health Sciences, Curtin University, Bentley, Western Australia, Australia
[8]Neurological Intervention and Imaging Service of Western Australia, Western Australia, Australia
[9]The First Affiliated Hospital, Sun Yat-Sen University Guangzhou, China
[10]Deakin Health Economics, Institute for Health Transformation, Deakin University, Victoria, Australia
[11]enAble Institute, Curtin University, Bentley, Western Australia, Australia
[12]Emergency Medicine, Royal Perth Hospital, Perth, Western Australia, Australia

Abstract

Mild traumatic brain injury (mTBI) accounts for 80–90% of all TBI. Post-mTBI symptoms are measured using the Post-Concussion Symptom Scale (PCSS); however, symptom heterogeneity limits specificity. Better understanding of the neuropathophysiology underlying post-concussion symptoms could enhance diagnostic accuracy. We explored the association between network connectivity, PCSS and neuropsychological functioning within 7 days post-mTBI. We hypothesized that network dysregulation would (a) correlate positively with PCSS scores and (b) correlate negatively with cognitive performance; and that (c) cognitive performance would correlate negatively with PCSS scores. Network activity was measured in 19 participants aged 21 to 65, following a medically diagnosed mTBI. Quantitative electroencephalography (qEEG) measured default mode, salience, and frontoparietal networks, while cognition was measured via neuropsychological assessment. Hypothesis (a) was not supported. Of the cognitive domains, support was only found for an association between network dysfunction and immediate memory. There was no association between neuropsychological performance and PCSS scores. PCSS scores were not a sensitive indicator of neuropsychological status and did not reflect the status of underlying brain network regulation. This study provides preliminary evidence for immediate memory as an indicator of altered network connectivity in acute mTBI. Evaluating neurophysiological and cognitive impacts of mTBI may improve understanding of individual recovery needs.

Keywords: default mode network; salience network; frontoparietal network; post-concussion symptoms; quantitative electroencephalography

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***Address correspondence to**: Francesca Buhagiar, School of Psychological Science, The University of Western Australia, 35, Stirling highway, Perth, WA 6009 Australia. Email: francesca.buhagiar@research.uwa.edu.au

Edited by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

Reviewed by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
Tanya Morosoli, MSc: 1) Clínica de Neuropsicología Diagnóstica y Terapéutica, Mexico City, Mexico; 2) ECPE, Harvard T. H. Chan School of Public Health, Boston, MA, USA

Introduction

An estimated 99 to 275 out of 100,000 (incidence) Australians suffer a traumatic brain injury (TBI; James et al., 2019; Pozzato et al., 2019). On a global level, incidence rates range from 331 to 412 per 100,000 (James et al., 2019). Mild traumatic brain injury (mTBI) makes up 80–90% of all traumatic brain injuries (Gardner & Yaffe, 2015; Jungfer, 2017), with an incidence of mTBI in people aged 16 to 59 years reported to be 302 per 100,000 person-years (Skandsen et al., 2019). The terms concussion and mTBI are often used interchangeably (Patricios et al., 2023). While some suggest that concussion is a less severe type of mTBI, others do not differentiate between the two terms (Mayer, Quinn, et al., 2017). The position statement on concussion in sport by the American Medical Society for Sports Medicine (Harmon et al., 2019) described concussion as a "traumatically induced transient disturbance of brain function that involved a complex pathophysiological process." Importantly, mTBI can result in diffuse axonal injury and disruption of the long white matter tracts, which connect various regions within brain networks resulting in alterations in function (Bai et al., 2022). The types of events resulting in mTBI include sports injuries, cycling accidents, assaults, falls, and motor vehicle accidents (Jagnoor & Cameron, 2014; Langer et al., 2020). The different mechanisms of injury have common biomechanical forces including linear and rotational acceleration (Rowson et al., 2016), with rotational forces being more likely to cause diffuse axonal injury (Gennarelli, 1971; Zhang et al., 2006). Individuals with diffuse axonal injury are three times more likely to have unfavorable outcome, than those with focal brain injuries (van Eijck et al., 2018).

Symptoms experienced following mTBI include anxiety/mood, cognitive, headache/migraines, vestibular, ocular, and fatigue (Harmon et al., 2019). While symptoms in adults last 2 weeks on average, up to 50% experience persistent post-concussion symptoms (PPCS) beyond the expected 2-week timeframe [17–19 (L. J. Carroll et al., 2014; de Freitas Cardoso et al., 2019; McInnes et al., 2017; Rivara & Graham, 2014)]. Studies have reported that 50% of participants experienced cognitive dysfunction at 3 months postinjury (Theadom et al., 2016), with deficits in executive function, working memory, and memory recall seen up to 24 months post-mTBI (Bai et al., 2022; Bedard et al., 2020; E. L. Carroll et al., 2020; de Freitas Cardoso et al., 2019). Individuals with higher PPCS scores display poorer cognitive performance compared to those with lower PPCS scores (Custer et al., 2016). A complex array of pre- and postinjury biopsychosocial factors influence an individual's post-concussion experience, such as personality characteristics, preexisting psychological disorders, and individual coping strategies (Young, 2020). Postinjury interventions such as rest versus exercise have also been debated (Allen, 2022; Liotta, 2021), with emerging research suggesting that physical activity modulates brain network activity and overall brain health (Dorsman et al., 2020; McFadden et al., 2013; Schmitt et al., 2019).

Network Connectivity

As the knowledge of mTBI pathophysiology develops, there is an emerging understanding that the post-mTBI phenomena are best reframed as a disorder of dysfunctional brain networks (Hayes et al., 2016; Iverson, 2019). In fact, altered network connectivity within the brain has been linked to cognitive function and overall symptom severity following mTBI (Bai et al., 2022; D'Souza et al., 2020), making it a potentially contributing neurophysiological factor to the PPCS phenomenon. Post-concussion symptoms have been associated with altered activity within the salience network (SN), the default mode network (DMN), and the frontoparietal network (FPN) following mTBI (Bonnelle, Leech, et al., 2011; Ham et al., 2014; Han et al., 2016; Jilka et al., 2014; Mayer, Mannell, et al., 2011; Messé et al., 2013; Sharp, Beckmann, et al., 2011; Shumskaya et al., 2012; Sours et al., 2015; Sponheim et al., 2011; Stevens et al., 2012; Tang et al., 2011; Zhou, Lui, et al., 2014; Zhou, Milham, et al., 2012). The DMN plays a central role in focusing our thoughts internally, being inhibited when shifting attention externally, and the network is active at rest (Hayes et al., 2016). Hence, understanding network connectivity post-mTBI may inform the brain–behavior relationship and help with contextualizing symptoms.

Measuring Network Function

Functional magnetic resonance imaging (fMRI) and diffusion weighted imaging (DWI) are typically used to measure brain network function in a research setting (Mortaheb et al., 2021; Pavlovic et al., 2019). Both these methods are costly and highly technical, limiting accessibility to functional neurophysiological assessment for the general population. Quantitative electroencephalography (qEEG) is a cheaper, less invasive, more portable, and accessible neuroimaging option, offering better temporal resolution than fMRI. Brain biomarkers detected using qEEG such as coherence, amplitude, and power, have been correlated with MRI findings in

TBI, stroke and tumor populations, demonstrating its potential utility as a measure of brain function (Thatcher, Biver, et al., 1998a, 1998b; Thatcher, North, et al., 2001). In mTBI, coherence, phase, and amplitude asymmetry measures using qEEG differentiated controls from mTBI with 95% accuracy (Thatcher, North, et al., 2001). Additionally, this increased brain dysfunction was associated with reduced cognitive performance on measures of verbal comprehension, visuospatial processing, processing speed, object naming, word fluency, inhibition/switching, and visual and verbal memory recall in a sample of mild to severe TBI participants (Thatcher, North, et al., 2001). Decreased coordination of neural functioning within the frontal regions on qEEG have also been observed following blast-related mTBI (Sponheim et al., 2011).

Hence, altered qEEG measures like phase, coherence, and amplitude asymmetry (current source density [CSD]) within specific brain regions (e.g., the frontal lobe) have shown diagnostic and prognostic utility in mTBI (Haneef et al., 2013; Thatcher, North, et al., 2001). While phase difference (PD) refers to the temporal synchrony of electrical activation between two brain areas, indicating the data transfer efficiency within a neuron cluster (Fell & Axmacher, 2011; Rabinovich et al., 2012), coherence refers to the degree of EEG frequency correlation between two electrodes, reflecting the activity similarity in the underlying brain areas, quantifying the level of connectivity between the areas of interest (Haneef et al., 2013). Lagged coherence (LC) addresses the impact of volume conduction present with instantaneous coherence (IC; Milz et al., 2014). Although qEEG has been validated as a functional measure for mTBI, its utility for measuring network function post-mTBI remains limited, with one study demonstrating that functional connections detected by qEEG were comparable with DWI findings in mTBI (Sponheim et al., 2011). Research has demonstrated that the integration of brain function occurs at a global level via networks of neurons rather than within a single localized area (Luria, 1973; Mesulam, 2000), highlighting the importance of assessing the brain's global and integrated functions within distinct networks rather than individualized areas of function. Considering that mTBI results in stretching and shearing of the axonal and vascular structures, including disruption of white matter tracts (McKee & Daneshvar, 2015; Mito et al., 2022; Narayana, 2017; Pavlovic et al., 2019), measuring network activity post-mTBI is important to determine whether global, higher order, integrated brain functioning has been impacted.

Quantifying Post-Concussion Sequelae

Post-mTBI symptoms are often measured using a questionnaire such as the Post-Concussion Symptom Scale (PCSS; Lovell et al., 2006). While the PCSS is a valid and reliable measure (Lovell et al., 2006; McLeod & Leach, 2012), the heterogeneity of post-mTBI symptoms offers limited specificity for diagnostic purposes. For example, 45% of a very large sample (n = 11,759) drawn from the general population met the criteria for PPCS in one European study (Voormolen et al., 2019). While the cognitive status of the individuals was not specified, the level of education varied from primary school to college or university level education. Improving our understanding of the neurophysiology underlying post-concussion symptoms is one way to enhance diagnostic specificity, by considering not only clinical presentation or symptom ratings but also an individual's neurophysiological status. The pathophysiology of mTBI has been described as a complex interaction of events caused by both primary and secondary damage, resulting in structural as well as functional changes (Masel & DeWitt, 2010; Young, 2020). Primary damage refers to damage that has occurred at the time of injury, while secondary damage refers to pathophysiology evolving days or even months postinjury (Pavlovic et al., 2019). In fact, changes in brain activity have been observed after mTBI, even when the clinical symptoms have resolved, suggesting that the absence of symptoms is not synonymous with recovery (Barr et al., 2012; McCrea et al., 2010; Prichep et al., 2013).

The present study aimed to measure the association between brain network connectivity, acute PCSS scores, and cognition (processing speed, inhibition/switching, immediate memory, visuospatial/constructional, language, attention, and delayed memory) in Australian adults within 7 days after mTBI. It was hypothesized that network dysregulation (DMN, SN, and FPN) would be (a) positively correlated with acute PCSS scores and (b) negatively correlated with cognitive performance as measured on neuropsychological tests. Additionally, it was hypothesized that (c) cognitive performance would be negatively correlated with PCSS scores. To the best of our knowledge, this study is the first to investigate the utility of measuring network function using qEEG in the acute stages post-mTBI and as such, serves as an initial pilot study of this novel approach in an mTBI sample.

Materials and Methods

Participants

Participants were drawn from the larger ongoing CREST study (Gozt et al., 2021), where all Phase II participants with qEEG and neuropsychological data available at the time of this study were selected for inclusion. Ethics approval was provided by the Human Research Ethics committees of St. John of God Health Care (#1628), Curtin University (HRE2019-0209), Royal Perth Hospital (#RGS0000003024), and Ramsay Health Care (#2009). Participants had received a medical diagnosis of concussion (< 24 hr posttraumatic amnesia, < 30 min loss of consciousness; Gumm et al., 2011) and were assessed within 7 days of injury (M = 4.22, SD = 1.26). Data were collected from 19 participants (10 females), aged between 21 and 65 years with an average of 13.5 years of education. Participation was voluntary, and participants were remunerated for parking costs if applicable. Written informed consent to participate in the study was obtained from all participants.

Procedure

Participants were recruited from several emergency departments, medical and allied health practitioners, as well as sporting clubs and self-referral across Perth, Western Australia. All participants were required to have a medical diagnosis of concussion. A phone interview (Phase I) was conducted with all participants, and those who met eligibility criteria were invited to attend a face-to-face assessment (Phase II) within 7 days of injury. Phase II inclusion criteria included participants being willing and able to attend the Curtin University and Perron Institute for Neurological and Translational Sciences research tenancies located at the Ralph and Patricia Sarich Neuroscience Research Institute within 7 days of injury, and Sir Charles Gardiner Hospital for MRI within 9 days of injury (a leeway of an additional 2 days was required for the fMRI component, due to the hospital-based MRI scanner having limited availability in the context of a global COVID-19 pandemic). Inclusion criteria for Phase I and exclusion criteria for Phase I and Phase II are details in Gozt et al. (2021).

The Phase II assessment was 2.5 to 3 hr in duration and included qEEG, blood analysis, neuropsychological assessment, exercise tolerance testing, as well as vestibular/oculomotor testing. Eligible participants also had a brain MRI scan within 9 days following their mTBI. This study focused only on the qEEG and components of the neuropsychological measures at Phase II; more details regarding participant recruitment and assessment procedures for the wider CREST study are available in Gozt et al. (2021). Data from other outcomes will be presented at a later date.

Materials

Connectivity within the DMN, SN, and FPN was measured using resting-state qEEG. EEG recording (resting-state, eyes-closed condition) was acquired via a 19-channel Electro-cap (Electro-cap International Inc., n.d.) and a MITSAR-EEG-BT amplifier (Mitsar, Ltd., n.d.). Quantitative analysis was performed via NeuroGuide and NeuroNavigator software (Applied Neuroscience, Inc., 2023), which have been previously validated in an mTBI population (Rapp et al., 2015; Thatcher, North, et al., 2001). Scalp recording of the EEG signals was conducted using a 19-channel Electro-cap with standardized 10–20 placement, which was fitted for size according to head circumference. Preparation for each electrode included parting of the hair and insertion of Electro-Gel electroconductive gel. A linked-ears montage was used to record activity from all 19 electrodes, using a sampling rate of 500 Hz, with impedance < 10 kΩ and a low pass filter of 50 Hz. Both eyes-open and eyes-closed data were obtained in a resting state, recording 5 min for each condition, with an eye mask used for the eyes-closed condition, to reduce eye-movement artefact. A minimum of 1 min of artifact-free data from the eyes-closed recording was selected via NeuroGuide software, which also accounted for drowsiness. Low resolution electromagnetic tomography analysis (LORETA) via the NeuroNavigator software, was used for source localization to detect altered network activity. NeuroNavigator has been described as a

> 3-Dimensional Electrical Neuroimaging tool [that] uses a real-MRI with 12,270 voxels; the Boundary Element Method (to compute the inverse solution to avoid the errors inherent in the use of a spherical head model, allowing for more accurate source localization at depths); and swLORETA (standardized weighted LORETA; adjusts for source space inhomogeneity and provides accurate estimates of source gradients from the upper to the lower cortical layers). (Applied Neuroscience Inc., 2023)

Research has supported the use of sLORETA to localize brain activity changes to fMRI (Cannon et al., 2011; Kerasidis & Simmons, 2021; Vitacco et al., 2002). Participants' activity was quantified in deviation from the normal (z score), by comparison

to a sex and age-matched normative database (*N* = 727; information about the normative database is available in Thatcher, Walker, et al., 2003). The component Brodmann areas (BA; Brodmann, 1909) for all three networks were automatically selected by the software program (DMN: bilateral BA 2, 7, 10, 11, 19, 29, 30, 31, 35, 39, 40; SN: bilateral BA 8, 9, 10, 13, 22, 23, 24, 25, 29, 30, 31, 32, 33; FPN: bilateral BA 1, 2, 3, 5, 7, 8, 9, 10, 39, 40, 45, 46). For clarification, the Brodmann areas were a topical reference of the software's source localization, the network nodes were not selected based on predetermined Brodmann areas, but rather the Brodmann area was provided as a reference point for the localization of the network.

The level of network function was denoted by *z* scores (i.e., the amount of activity in standard deviations as compared to a normative database), for a spectrum of frequencies ranging from 1 to 30 Hz. Measures included CSD for individual Brodmann areas as well as IC, LC, and PD to represent the degree of connectivity between Brodmann area pairs, within the networks of interest. CSD was measured in microamperes squared per cycle/second, while IC and LC were measured as correlation coefficients, with numbers approaching 1 representing higher similarity (coherence) between the signals. PD was estimated by calculating the instantaneous PD between time series and ranged from 0 to ±180 degrees. The measures of coherence, phase, and CSD were found to differentiate between mTBI and controls in previous research (Thatcher, North, et al., 2001); hence, a correlation with PCSS scores was expected on all four measures. Five separate measures were calculated for each network, including the peak *z* score, mean *z* score, *z* score variance, total number of *z* scores above ±1.96, and the percentage of *z* scores above ±1.96. Activity ranging between *z* scores of 0 and ±1.96 was considered normal, while *z* scores greater than ±1.96 were considered significantly altered (deviated from the norm). With regards to the five levels of each measure (peak *z*, mean *z*, *z* variance, number of *z* scores above ±1.96, percentage of *z* scores above ±1.96), peak *z* score was used as per previous literature (Ims, 2019). The remaining measures were used to capture several elements of the network dysregulation (if present). Based on the standard normal curve cut-off for significant deviation from the normal, 1.96 was selected. We intended to capture not only how many areas were dysregulated, but also what percentage of the network they equate to (as not all networks had the same number of sites). The focus of the present study was on dysregulated activity, regardless of whether it was hypoactive (negative *z* score) or hyperactive (positive *z* score). A topographical representation of network dysregulation can be seen in Figure 1, where the degree of dysregulated activity is represented by colors on a spectrum of *z* scores.

Post-Concussion Symptoms (PCS)

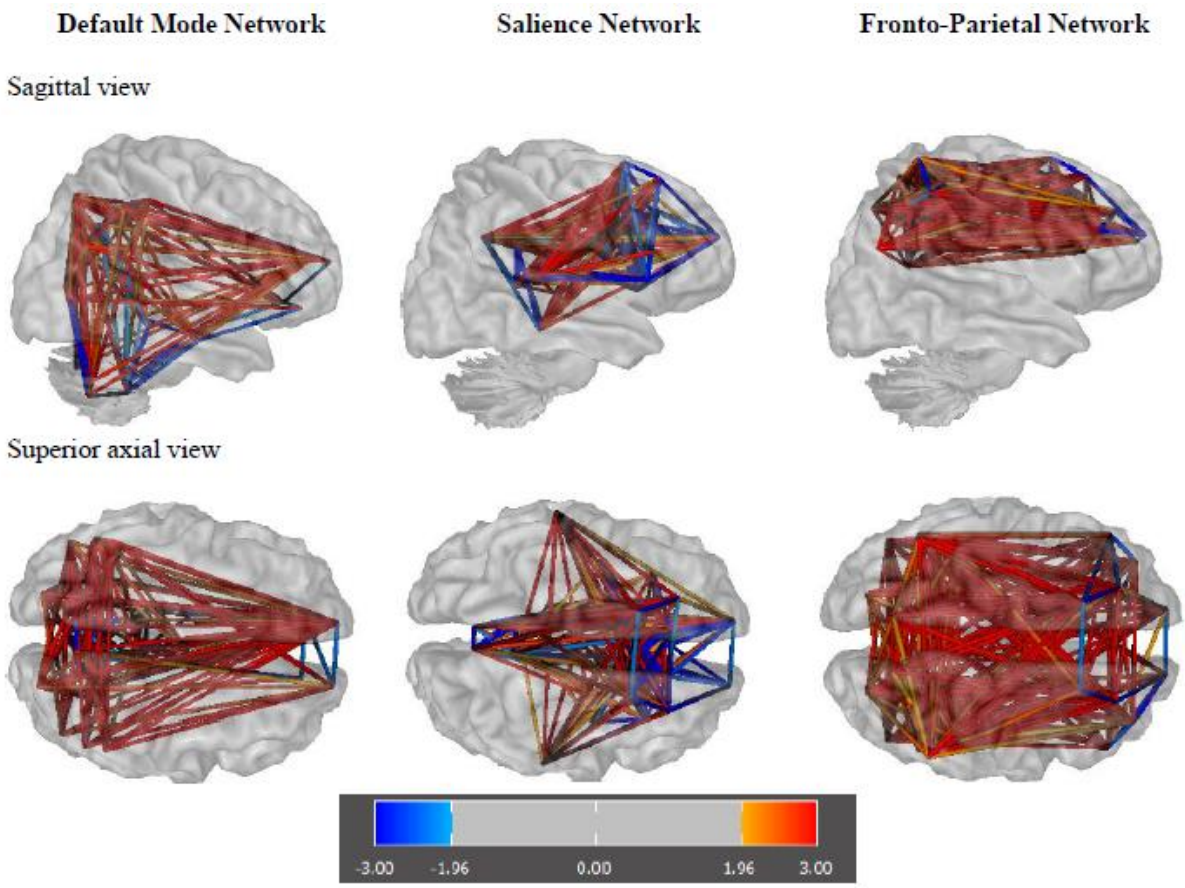
The PCSS (Lovell et al., 2006) consists of 22 self-report items used to assess symptom severity after concussion. Symptoms were rated on a Likert scale from 0 (*none*) to 6 (*severe*), where higher PCSS total symptom scores indicated an increased symptom burden. The cognitive, physical, affective, and sleep symptom domains were also analyzed using a factor structure established in a concussed adult sample (Merritt et al., 2017). The test–retest reliability of the PCSS measured as part of the ImPACT (Immediate Post-Concussion Assessment and Cognitive Testing) neuropsychological battery was 0.81 (Schatz & Ferris, 2013).

Cognitive Function

Processing Speed and Inhibition/Switching. The Trail Making Test (TMT) part A and B (Reitan & Wolfson, 1985) were used. TMT-A required participants to sequentially connect numbers on a page, while TMT-B required a similar sequential procedure while switching between numbers and letters (e.g., 1-A, 2-B, etc.). The raw scores (time to complete the task) were converted to *z* scores using the normative sample detailed in Tombaugh (Tombaugh, 2004) for data analysis, where a higher positive *z* score indicated faster task completion. Test-retest reliability was found to be adequate (0.70) for TMT-A and TMT-B (Levine et al., 2004), and predictive validity for psychosocial outcomes following head injury was also noted (Colantonio et al., 2000).

Overall Neuropsychological Function. The Repeatable Battery for the Assessment of Neuropsychological Status Update (RBANS) (Randolph, 2012) was used. It was selected due to its utility in assessing a broad range of relevant cognitive domains that have been identified to be impacted following mTBI, while remaining brief to administer.

Figure 1. *Topographical Representation of Dysregulated Network Connectivity, in NeuroNavigator.*



Note. The occipital lobe is located on the left side of all images. Individual lines represent altered connectivity within a network. The degree of deviation from the norm is indicated by the color scale on the *z* score color spectrum. These images represent partial networks only, since normal (or unaltered) connections are not depicted. Data displayed above is from a single participant with mTBI.

Moreover, it provided an embedded measure of effort which was determined to be an important factor in mTBI research (Young, 2020). The RBANS consisted of five subscales including immediate memory, visuospatial/constructional, language, attention, and delayed memory. An index score was obtained for each of the subscales, and these were summed up to give a composite total score. Higher scores indicated better cognitive function. The RBANS total index score was found to have high specificity (0.94) and sensitivity (0.82) in a traumatic brain injury sample (McKay et al., 2008).

Effort. The Rey 15-Item Test (FIT; Rey, 1964) was used to measure performance validity. Participants were asked to recall 15 items. One point was awarded for each correct item (maximum = 15 points), where scores of 9 or higher demonstrated adequate effort. The FIT has been found to have a specificity of 85% (Reznek, 2005). Additionally, the RBANS effort index was calculated as described in Silverberg et al. (2007).

Design and Statistical Analysis

An observational cross-sectional study design was used. For the first hypothesis, the dependent variable was network function with three levels: DMN, SN, and FPN. For each network the functional connectivity was characterized by five measures including peak *z* score, mean *z* score, *z* score variance, total number of *z* scores above ±1.96, and the percentage of *z* scores above ±1.96 across IC, LC, and PD. The independent variable was PCSS score with five levels, total score and four symptom domain scores: cognitive, physical, affective, and sleep. Pearson product moment correlation and Spearman's rank-order correlation were used to

measure the correlation between brain dysregulation and PCSS scores.

For the second hypothesis, the dependent variable was network function with three levels as described for the first hypothesis. The independent variables included six cognitive index scores from the RBANS (total index score, immediate memory, visuospatial/constructional, language, attention, and delayed memory), as well as the *z* scores for TMT-A and TMT-B. Spearman's rank-order correlation were used to measure the correlation between brain network function and cognitive function.

For the third hypothesis, the dependent variables included six cognitive index scores from the RBANS (total index score, immediate memory, visuospatial/constructional, language, attention, and delayed memory), as well as the *z* scores for TMT-A and TMT-B. The independent variable was PCSS score with five levels, total score and four symptom domain scores: cognitive, physical, affective, and sleep. Pearson product moment correlation and Spearman's rank-order correlation were used to measure the correlation between cognitive function and PCSS scores.

The Benjamini-Hochberg method was used to correct for multiple comparisons as described in the relevant results sections below.

Results

Network Connectivity and Post-Concussion Symptoms

To test the hypothesis that network (DMN, SN, FPN) dysregulation (as measured by qEEG) would be positively correlated with PCSS scores in the acute post-concussion phase, analyses were conducted on 19 participants. Of the 60 network function variables, 6 were normally distributed; however, the assumptions of linearity and homoscedasticity were not met, so nonparametric analysis was conducted on all measures. Eighty-four outliers were detected (> 3 standard deviations from the mean) and winsorized (Field, 2013).

Demographic Characteristics

Table 1 shows the descriptive statistics for demographic variables including concussion injury characteristics, medical, and health history. Of note, none of the participants had a history of epilepsy/seizure disorder or non-migraine-type headaches, 47.4% were taking at least one medication (asthma medication, *n* = 4; antidepressants, *n* = 2; antihypertensives, *n* = 1; hormone therapy, *n* = 2; paracetamol, *n* = 1; melatonin, *n* = 1; thyroxine, *n* = 2; hair loss medication, *n* = 1; dexamphetamines, *n* = 1), and 89.5% of participants exercised regularly (2.88 days per week on average). Only one participant had legal involvement relating to their concussion. Of the 19 participants, 63.2% had a sport-related injury and 57.9% sustained additional physical injuries at the time of their concussion. While only 21.1% reported experiencing loss of consciousness ranging from 3 s to 10 min (31.6% were unsure), 57.9% experienced posttraumatic amnesia (ranging from < 5 min to > 120 min). Just over half the participants (52.6%) had one or more previous concussions, and 26.3% reported they were under the influence of alcohol at the time of injury.

Network Connectivity and Post-Concussion Symptoms

To assess the size and direction of the linear relationship between brain network dysregulation and PCSS, a Spearman's rank-order correlation was conducted for all network connectivity measures as listed in Table 2. No significant correlations were found between network dysregulation and total PCSS score.

Network Connectivity and Post-Concussion Symptom Domains

To further explore the relationship between brain network dysregulation and PCSS symptom domains (cognitive, physical, affective, sleep), Spearman's rank-order correlation was conducted for all network connectivity measures as listed in Table 3. After one outlier was detected and winsorized (Field, 2013), only the cognitive symptom domain was normally distributed. Since the assumptions of linearity and homoscedasticity were not met, nonparametric analyses were conducted for all variables. A significant correlation was found between the SN and the PCSS affective symptom domain; however, this correlation did not remain significant after Benjamini-Hochberg correction for multiple comparisons.

Network Connectivity and Cognitive Function

To test the hypothesis that altered network connectivity would be associated with cognitive performance (measured by a neuropsychological battery) in the acute post-concussion phase, Spearman's rank order correlation was conducted. Three outliers were detected within the cognitive variables and winsorized (Field, 2013), being three or more standard deviations away from the mean. Assumption testing revealed that the variables which were normally distributed did not meet the

assumptions of linearity or homoscedasticity, so nonparametric analyses were used. Table 4 shows that DMN and FPN connectivity as well as DMN, SN and FPN CSD were significantly correlated with the RBANS immediate memory index. There was also a significant correlation between DMN and SN network connectivity and the TMT-A; however, this did not remain significant after correcting for multiple comparisons using the Benjamini-Hochberg method.

Table 1

Descriptive Statistics for Demographic Variables and Injury Characteristics

	% (n)	*M*	*SD*
Age (years)	100 (19)	34.26	12.45
Education (years)	100 (19)	13.47	2.20
Sex			
Male	47 (9)		
Female	53 (10)		
Time Since Concussion Injury (days)	100 (19)	4.22	1.26
Exercise Days Per Week	100 (19)	2.88	0.93
Number of Alcohol Drinks at Time of Injury	100 (19)	3.50	2.45
	Yes	No	Unsure
	% *(n)*	% *(n)*	% *(n)*
Mechanism of Injury (Sport-related)	63.2 (12)	36.8 (7)	
Loss of Consciousness (< 30 min)	21.1 (4)	47.7 (9)	31.6 (6)
Posttraumatic Amnesia (< 24 hr)	57.9 (11)	42.1 (8)	
Alcohol at Time of Injury	26.3 (5)	73.7 (14)	
Other Injuries at Time of Concussion	57.9 (11)	42.1 (8)	
Previous Concussions	52.6 (10)	47.4 (9)	
Epilepsy/Seizure Disorder		100 (19)	
Migraine	21.1 (4)	78.9 (15)	
Other Headaches		100 (19)	
Mental Health Disorder	31.6 (6)	68.4 (13)	
Sleep Disorder	10.5 (2)	89.5 (17)	
Learning Disorder	15.8 (3)	84.2 (16)	
Regular Exercise	89.5 (17)	10.5 (2)	
Medications	47.4 (9)	52.6 (10)	
Legal Involvement	5.3 (1)	94.7 (18)	

Note. The participants listed in the No category for Loss of Consciousness (n = 9) did not experience loss of consciousness. Overall, participants who reported > 30 min of loss of consciousness and > 24 hr of posttraumatic amnesia were excluded from the study.

Table 2

Spearman's Rank-Order Correlation Between Network Connectivity and PCSS Scores, N = 19

Network Connectivity Measures	Descriptive Statistics		Spearman's Correlation	
	M	*SD*	*r*	*p*
DMN IC Peak *Z* score	4.81	1.89	−0.060	0.808
DMN IC Mean *Z* Score	0.84	0.27	0.101	0.681
DMN IC *Z* Score Variance Within Network	0.40	0.22	0.075	0.759
DMN IC Number of *Z* scores > 1.96 *SD*	485.32	540.95	0.090	0.713
DMN IC Percentage *Z* scores > 1.96 *SD*	7.00	7.81	0.090	0.713
SN IC Peak *Z* Score	5.02	2.53	−0.185	0.449
SN IC Mean *Z* Score	0.89	0.29	−0.023	0.927
SN IC *Z* Score Variance Within Network	0.47	0.29	−0.022	0.930
SN IC Number of *Z* scores > 1.96 *SD*	846.53	923.99	−0.009	0.970
SN IC Percentage *Z* scores > 1.96 *SD*	8.68	9.48	−0.009	0.970
FPN IC Peak *Z* Score	5.28	2.36	0.044	0.857
FPN IC Mean *Z* Score	0.83	0.24	0.146	0.550
FPN IC *Z* Score Variance Within Network	0.48	0.30	0.107	0.663
FPN IC Number of *Z* scores > 1.96 *SD*	594.11	592.77	0.102	0.679
FPN IC Percentage *Z* scores > 1.96 *SD*	7.18	7.16	0.102	0.679
DMN LC Peak *Z* Score	4.22	2.21	−0.178	0.465
DMN LC Mean *Z* Score	0.73	0.19	0.166	0.497
DMN LC *Z* Score Variance Within Network	0.40	0.26	−0.032	0.898
DMN LC Number of *Z* scores > 1.96 *SD*	349.63	349.11	0.070	0.775
DMN LC Percentage *Z* scores > 1.96 *SD*	5.05	5.04	0.070	0.775
SN LC Peak *Z* score	4.23	2.30	−0.067	0.786
SN LC Mean *Z* Score	0.69	0.14	0.108	0.659
SN LC *Z* Score Variance Within Network	0.35	0.20	−0.028	0.909
SN LC Number of *Z* scores > 1.96 *SD*	361.21	319.76	0.067	0.786
SN LC Percentage *Z* scores > 1.96 *SD*	3.70	3.28	0.067	0.786
FPN LC Peak *Z* Score	3.88	1.97	−0.025	0.919
FPN LC Mean *Z* Score	0.68	0.16	0.063	0.798
FPN LC *Z* Score Variance Within Network	0.35	0.25	0.008	0.973
FPN LC Number of *Z* scores > 1.96 *SD*	293.21	291.49	0.078	0.752
FPN LC Percentage *Z* scores > 1.96 *SD*	3.54	3.52	0.078	0.752

Table 2

Spearman's Rank-Order Correlation Between Network Connectivity and PCSS Scores, N = 19

Network Connectivity Measures	Descriptive Statistics		Spearman's Correlation	
	M	*SD*	*r*	*p*
DMN PD Peak *Z* score	5.04	2.82	−0.172	0.482
DMN PD Mean *Z* score	0.84	0.38	−0.047	0.848
DMN PD *Z* Score Variance Within Network	0.30	0.11	−0.022	0.930
DMN PD Number of *Z* scores > 1.96 *SD*	574.68	829.51	−0.074	0.763
DMN PD Percentage *Z* scores > 1.96 *SD*	3.19	2.35	−0.038	0.878
SN PD Peak *Z* score	5.28	3.77	−0.345	0.148
SN PD Mean *Z* score	0.75	0.20	−0.224	0.357
SN PD *Z* Score Variance Within Network	0.30	0.12	−0.216	0.375
SN PD Number of *Z* scores > 1.96 *SD*	361.53	291.44	−0.217	0.372
SN PD Percentage *Z* scores > 1.96 *SD*	3.71	2.99	−0.217	0.372
FPN PD Peak *Z* Score	5.11	2.17	−0.088	0.721
FPN PD Mean *Z* Score	0.98	0.51	−0.083	0.736
FPN PD *Z* Score Variance Within Network	0.40	0.17	−0.071	0.771
FPN PD Number of *Z* scores > 1.96 *SD*	969.58	1187.48	−0.119	0.628
FPN PD Percentage *Z* scores > 1.96 *SD*	11.71	14.34	−0.119	0.628
DMN CSD Peak *Z* score	2.85	1.44	−0.062	0.801
DMN CSD Mean *Z* score	1.22	0.88	−0.209	0.390
DMN CSD *Z* Score Variance Within Network	0.37	0.29	−0.210	0.389
DMN CSD Number of *Z* scores > 1.96 *SD*	127.11	200.44	−0.173	0.478
DMN CSD Percentage *Z* scores > 1.96 *SD*	19.26	30.37	−0.173	0.478
SN CSD Peak *Z* score	3.10	1.79	−0.111	0.652
SN CSD Mean *Z* score	1.28	0.88	−0.164	0.504
SN CSD Mean *Z* Score Variance Within Network	0.43	0.38	−0.159	0.515
SN CSD Number of *Z* scores > 1.96 *SD*	171.95	258.94	−0.182	0.456
SN CSD Percentage *Z* scores > 1.96 *SD*	20.47	30.83	−0.182	0.456
FPN CSD Peak *Z* score	2.87	1.69	−0.169	0.489
FPN CSD Mean *Z* Score	1.36	1.06	−0.163	0.506
FPN CSD *Z* Score Variance Within Network	0.28	0.15	−0.227	0.350
FPN CSD Number of *Z* scores > 1.96 *SD*	155.95	249.25	−0.194	0.427
FPN CSD Percentage *Z* scores > 1.96 *SD*	21.66	34.62	−0.194	0.427

Note. DMN = default mode network; SN = salience network; FPN = frontal-parietal network; IC = instantaneous coherence; LC = lagged coherence; PD = phase difference; CSD = current source density.

Table 3
Spearman's Rank-Order Correlation Between Network Connectivity and PCSS Domains, N = 19

Network Connectivity Measures	Spearman's Correlation							
	PCSS Cognitive		PCSS Physical		PCSS Affective		PCSS Sleep	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*
DMN IC Peak *Z* score	−0.279	0.247	−0.050	0.839	−0.113	0.646	0.023	0.926
DMN IC Mean *Z* Score	−0.070	0.777	−0.001	0.996	−0.187	0.444	0.171	0.484
DMN IC *Z* Score Variance Within Network	−0.103	0.676	−0.059	0.812	−0.111	0.651	0.170	0.488
DMN IC Number of *Z* scores > 1.96 *SD*	−0.056	0.819	−0.016	0.949	−0.111	0.651	0.209	0.391
DMN IC Percentage *Z* scores > 1.96 *SD*	−0.056	0.819	−0.016	0.949	−0.111	0.651	0.209	0.391
SN IC Peak *Z* Score	−0.328	0.171	−0.143	0.558	−0.260	0.282	−0.108	0.658
SN IC Mean *Z* Score	−0.078	0.750	0.069	0.778	−0.272	0.260	0.068	0.783
SN IC *Z* Score Variance Within Network	−0.119	0.629	0.012	0.961	−0.230	0.343	0.140	0.566
SN IC Number of *Z* scores > 1.96 *SD*	−0.085	0.728	0.040	0.872	−0.271	0.262	0.159	0.515
SN IC Percentage *Z* scores > 1.96 *SD*	−0.085	0.728	0.040	0.872	−0.271	0.262	0.159	0.515
FPN IC Peak *Z* Score	−0.173	0.479	0.070	0.775	−0.092	0.707	0.166	0.498
FPN IC Mean *Z* Score	0.055	0.824	0.140	0.567	−0.071	0.774	0.167	0.494
FPN IC *Z* Score Variance Within Network	−0.054	0.825	−0.027	0.913	−0.044	0.857	0.159	0.516
FPN IC Number of *Z* scores > 1.96 *SD*	−0.034	0.889	0.013	0.958	−0.089	0.718	0.127	0.606
FPN IC Percentage *Z* scores > 1.96 *SD*	−0.034	0.889	0.013	0.958	−0.089	0.718	0.127	0.606
DMN LC Peak *Z* Score	−0.247	0.307	−0.150	0.541	0.020	0.936	−0.091	0.712
DMN LC Mean *Z* Score	0.036	0.883	0.074	0.764	0.254	0.294	0.245	0.312
DMN LC *Z* Score Variance Within Network	−0.170	0.485	−0.074	0.765	0.126	0.606	0.052	0.831
DMN LC Number of *Z* scores > 1.96 *SD*	−0.083	0.736	−0.083	0.736	0.161	0.509	0.136	0.579
DMN LC Percentage *Z* scores > 1.96 *SD*	−0.083	0.736	−0.083	0.736	0.161	0.509	0.136	0.579
SN LC Peak *Z* score	−0.176	0.471	−0.129	0.598	0.091	0.710	−0.032	0.897
SN LC Mean *Z* Score	−0.015	0.950	−0.005	0.983	0.103	0.673	0.152	0.535
SN LC *Z* Score Variance Within Network	−0.130	0.597	−0.147	0.548	0.094	0.703	−0.018	0.941
SN LC Number of *Z* scores > 1.96 *SD*	−0.025	0.920	−0.159	0.516	0.012	0.962	0.086	0.727
SN LC Percentage *Z* scores > 1.96 *SD*	−0.025	0.920	−0.159	0.516	0.012	0.962	0.086	0.727
FPN LC Peak *Z* Score	−0.081	0.742	−0.127	0.603	0.032	0.897	−0.123	0.615
FPN LC Mean *Z* Score	−0.036	0.885	−0.075	0.759	0.128	0.603	0.043	0.862
FPN LC *Z* Score Variance Within Network	−0.126	0.608	−0.154	0.528	0.044	0.858	−0.046	0.853
FPN LC Number of *Z* scores > 1.96 *SD*	−0.053	0.831	−0.160	0.512	0.103	0.674	−0.003	0.990
FPN LC Percentage *Z* scores > 1.96 *SD*	−0.053	0.831	−0.160	0.512	0.103	0.674	−0.003	0.990

Table 3

Spearman's Rank-Order Correlation Between Network Connectivity and PCSS Domains, N = 19

Network Connectivity Measures	Spearman's Correlation							
	PCSS Cognitive		PCSS Physical		PCSS Affective		PCSS Sleep	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*
DMN PD Peak *Z* score	−0.304	0.206	−0.255	0.292	−0.200	0.412	−0.004	0.989
DMN PD Mean *Z* score	−0.207	0.394	−0.012	0.961	−0.301	0.210	0.000	0.999
DMN PD *Z* Score Variance Within Network	−0.180	0.461	0.032	0.895	−0.263	0.276	0.069	0.780
DMN PD Number of *Z* scores > 1.96 *SD*	−0.251	0.300	−0.015	0.952	−0.304	0.206	0.029	0.907
DMN PD Percentage *Z* scores > 1.96 *SD*	−0.218	0.371	0.017	0.945	−0.271	0.261	0.063	0.799
SN PD Peak *Z* score	−0.396	0.093	−0.245	0.312	−0.378	0.111	−0.186	0.446
SN PD Mean *Z* score	−0.265	0.274	−0.092	0.709	**−0.500***	**0.029**	−0.098	0.690
SN PD *Z* Score Variance Within Network	−0.255	0.293	−0.022	0.927	**−0.465***	**0.045**	−0.094	0.702
SN PD Number of *Z* scores > 1.96 *SD*	−0.261	0.280	−0.034	0.890	−0.446	0.056	−0.081	0.742
SN PD Percentage *Z* scores > 1.96 *SD*	−0.261	0.280	−0.034	0.890	−0.446	0.056	−0.081	0.742
FPN PD Peak *Z* Score	−0.236	0.331	−0.052	0.832	−0.322	0.178	0.118	0.631
FPN PD Mean *Z* Score	−0.149	0.543	0.045	0.856	−0.402	0.088	0.044	0.858
FPN PD *Z* Score Variance Within Network	−0.166	0.497	−0.010	0.968	−0.380	0.109	0.069	0.781
FPN PD Number of *Z* scores > 1.96 *SD*	−0.212	0.384	−0.065	0.792	−0.392	0.097	0.055	0.823
FPN PD Percentage *Z* scores > 1.96 *SD*	−0.212	0.384	−0.065	0.792	−0.392	0.097	0.055	0.823
DMN CSD Peak *Z* score	−0.232	0.339	−0.072	0.769	−0.143	0.559	0.018	0.941
DMN CSD Mean *Z* score	−0.359	0.131	−0.313	0.191	−0.132	0.590	−0.110	0.653
DMN CSD *Z* Score Variance Within Network	−0.348	0.144	−0.265	0.273	−0.119	0.628	−0.172	0.481
DMN CSD Number of *Z* scores > 1.96 *SD*	−0.323	0.177	−0.226	0.353	−0.176	0.471	−0.069	0.778
DMN CSD Percentage *Z* scores > 1.96 *SD*	−0.323	0.177	−0.226	0.353	−0.176	0.471	−0.069	0.778
SN CSD Peak *Z* score	−0.287	0.233	−0.103	0.674	−0.073	0.768	−0.039	0.873
SN CSD Mean *Z* score	−0.324	0.176	−0.255	0.293	−0.064	0.796	−0.101	0.680
SN CSD Mean *Z* Score Variance Within Network	−0.304	0.205	−0.165	0.500	−0.058	0.815	−0.154	0.529
SN CSD Number of *Z* scores > 1.96 *SD*	−0.349	0.143	−0.175	0.474	−0.120	0.626	−0.091	0.712
SN CSD Percentage *Z* scores > 1.96 *SD*	−0.349	0.143	−0.175	0.474	−0.120	0.626	−0.091	0.712
FPN CSD Peak *Z* score	−0.336	0.160	−0.156	0.524	−0.168	0.492	−0.090	0.714
FPN CSD Mean *Z* Score	−0.314	0.190	−0.236	0.331	−0.073	0.767	−0.034	0.889
FPN CSD *Z* Score Variance Within Network	−0.317	0.186	−0.230	0.343	−0.097	0.691	−0.255	0.293
FPN CSD Number of *Z* scores > 1.96 *SD*	−0.342	0.152	−0.182	0.455	−0.154	0.530	−0.079	0.747
FPN CSD Percentage *Z* scores > 1.96 *SD*	−0.342	0.152	−0.182	0.455	−0.154	0.530	−0.079	0.747

Note. * = significant at $p = 0.05$; DMN = default mode network; SN = salience network; FPN = frontal-parietal network; IC = instantaneous coherence; LC = lagged coherence; PD = phase difference; CSD = current source density; PCSS = Post-Concussion Symptom Scale.

Table 4

Spearman's Rank-Order Correlation Between Network Connectivity and Cognition, N = 19

Network Connectivity Measures	Spearman's Correlation															
	TMT-A		TMT-B		RBANS Total Index		Immediate Memory Index		Visuospatial Index		Language Index		Attention Index		Delayed Memory Index	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
DMN IC Peak *Z* score	0.280	0.246	0.089	0.718	0.018	0.940	−0.162	0.508	0.104	0.673	0.316	0.187	0.158	0.519	0.225	0.354
DMN IC Mean *Z* Score	0.286	0.234	0.198	0.417	**−0.477***	**0.039**	**−0.482***	**0.036**	−0.204	0.402	0.168	0.492	0.074	0.764	−0.352	0.139
DMN IC *Z* Score Variance Within Network	0.327	0.172	0.190	0.437	−0.399	0.090	**−0.457***	**0.049**	−0.081	0.741	0.232	0.340	0.013	0.958	−0.184	0.452
DMN IC Number of *Z* scores > 1.96 *SD*	0.336	0.159	0.238	0.327	−0.448	0.054	−0.455	0.050	−0.061	0.803	0.236	0.331	−0.084	0.731	−0.193	0.428
DMN IC Percentage *Z* scores > 1.96 *SD*	0.336	0.159	0.238	0.327	−0.448	0.054	−0.455	0.050	−0.061	0.803	0.236	0.331	−0.084	0.731	−0.193	0.428
SN IC Peak *Z* Score	0.392	0.097	0.163	0.504	−0.146	0.551	−0.298	0.216	−0.083	0.735	0.353	0.138	0.257	0.288	−0.04	0.872
SN IC Mean *Z* Score	0.332	0.165	0.338	0.157	−0.453	0.051	−0.298	0.216	−0.188	0.442	0.110	0.653	0.047	0.849	−0.181	0.457
SN IC *Z* Score Variance Within Network	0.403	0.087	0.288	0.232	−0.379	0.110	−0.237	0.329	−0.109	0.656	0.127	0.604	−0.051	0.835	−0.016	0.948
SN IC Number of *Z* scores > 1.96 *SD*	0.427	0.068	0.346	0.147	−0.443	0.058	−0.259	0.284	−0.166	0.496	0.096	0.696	−0.046	0.850	−0.118	0.631
SN IC Percentage *Z* scores > 1.96 *SD*	0.427	0.068	0.346	0.147	−0.443	0.058	−0.259	0.284	−0.166	0.496	0.096	0.696	−0.046	0.850	−0.118	0.631
FPN IC Peak *Z* Score	0.251	0.300	0.185	0.448	−0.140	0.568	−0.326	0.174	0.101	0.681	0.326	0.173	0.067	0.785	−0.003	0.991
FPN IC Mean *Z* Score	0.331	0.167	0.263	0.276	−0.422	0.072	**−0.482***	**0.037**	−0.025	0.918	0.303	0.208	0.042	0.865	−0.196	0.421
FPN IC *Z* Score Variance Within Network	0.272	0.260	0.127	0.606	−0.342	0.152	**−0.471***	**0.042**	−0.029	0.907	0.279	0.248	0.021	0.931	−0.122	0.619
FPN IC Number of *Z* scores > 1.96 *SD*	0.311	0.195	0.167	0.495	−0.369	0.120	**−0.487***	**0.035**	−0.061	0.805	0.316	0.188	0.060	0.806	−0.173	0.479
FPN IC Percentage *Z* scores > 1.96 *SD*	0.311	0.195	0.167	0.495	−0.369	0.120	**−0.487***	**0.035**	−0.061	0.805	0.316	0.188	0.060	0.806	−0.173	0.479
DMN LC Peak *Z* Score	0.443	0.057	0.370	0.119	−0.049	0.841	−0.068	0.782	0.142	0.561	0.315	0.190	−0.178	0.466	0.303	0.208
DMN LC Mean *Z* Score	0.396	0.093	0.345	0.148	−0.209	0.390	−0.173	0.478	−0.055	0.823	0.230	0.344	−0.084	0.733	0.097	0.693
DMN LC *Z* Score Variance Within Network	**0.460***	**0.048**	0.325	0.175	−0.087	0.723	−0.131	0.592	0.084	0.732	0.311	0.195	−0.093	0.706	0.274	0.257
DMN LC Number of *Z* scores > 1.96 *SD*	0.343	0.151	0.223	0.358	−0.194	0.427	−0.110	0.655	−0.008	0.974	0.214	0.378	−0.127	0.604	0.199	0.414
DMN LC Percentage *Z* scores > 1.96 *SD*	0.343	0.151	0.223	0.358	−0.194	0.427	−0.110	0.655	−0.008	0.974	0.214	0.378	−0.127	0.604	0.199	0.414
SN LC Peak *Z* score	0.346	0.147	0.267	0.270	0.029	0.906	−0.012	0.960	0.151	0.536	0.198	0.416	−0.225	0.355	0.300	0.212
SN LC Mean *Z* Score	**0.462***	**0.046**	0.422	0.072	−0.131	0.594	0.033	0.893	−0.169	0.489	0.276	0.252	−0.087	0.724	0.110	0.654
SN LC *Z* Score Variance Within Network	**0.492***	**0.033**	0.333	0.164	−0.101	0.680	−0.107	0.661	−0.050	0.840	0.320	0.181	−0.107	0.662	0.205	0.399
SN LC Number of *Z* scores > 1.96 *SD*	0.386	0.103	0.188	0.441	−0.337	0.158	−0.163	0.505	−0.140	0.567	0.152	0.535	−0.206	0.396	0.001	0.997
SN LC Percentage *Z* scores > 1.96 *SD*	0.386	0.103	0.188	0.441	−0.337	0.158	−0.163	0.505	−0.140	0.567	0.152	0.535	−0.206	0.396	0.001	0.997

Table 4

Spearman's Rank-Order Correlation Between Network Connectivity and Cognition, N = 19

Network Connectivity Measures	Spearman's Correlation															
	TMT-A		TMT-B		RBANS Total Index		Immediate Memory Index		Visuospatial Index		Language Index		Attention Index		Delayed Memory Index	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
FPN LC Peak *Z* Score	0.340	0.155	0.183	0.452	−0.065	0.791	−0.116	0.637	0.052	0.833	0.223	0.358	−0.241	0.320	0.113	0.644
FPN LC Mean *Z* Score	0.416	0.076	0.442	0.058	−0.006	0.980	0.075	0.761	−0.115	0.639	0.204	0.402	−0.156	0.523	0.146	0.552
FPN LC *Z* Score Variance Within Network	0.342	0.152	0.257	0.288	−0.043	0.861	−0.053	0.830	0.051	0.836	0.252	0.298	−0.138	0.573	0.196	0.420
FPN LC Number of *Z* scores > 1.96 *SD*	0.291	0.227	0.242	0.319	−0.112	0.649	−0.087	0.722	−0.032	0.895	0.198	0.417	−0.143	0.560	0.136	0.579
FPN LC Percentage *Z* scores > 1.96 *SD*	0.291	0.227	0.242	0.319	−0.112	0.649	−0.087	0.722	−0.032	0.895	0.198	0.417	−0.143	0.560	0.136	0.579
DMN PD Peak *Z* score	0.239	0.324	0.35	0.141	−0.303	0.207	−0.319	0.183	−0.156	0.525	0.353	0.138	0.061	0.803	−0.165	0.499
DMN PD Mean *Z* score	0.214	0.379	0.158	0.519	−0.386	0.102	**−0.469***	**0.043**	−0.218	0.369	0.140	0.567	0.253	0.296	−0.396	0.093
DMN PD *Z* Score Variance Within Network	0.296	0.219	0.207	0.395	−0.425	0.070	**−0.500***	**0.029**	−0.194	0.425	0.175	0.473	0.214	0.379	−0.403	0.087
DMN PD Number of *Z* scores > 1.96 *SD*	0.320	0.182	0.229	0.345	−0.366	0.124	**−0.478***	**0.038**	−0.165	0.499	0.213	0.380	0.208	0.392	−0.351	0.141
DMN PD Percentage *Z* scores > 1.96 *SD*	0.318	0.185	0.203	0.405	−0.368	0.121	**−0.490***	**0.033**	−0.164	0.501	0.208	0.394	0.204	0.403	−0.366	0.124
SN PD Peak *Z* score	**0.482***	**0.037**	0.424	0.071	−0.176	0.472	−0.236	0.331	−0.028	0.908	**0.481***	**0.037**	0.032	0.897	0.090	0.714
SN PD Mean *Z* score	0.447	0.055	0.298	0.215	−0.397	0.092	−0.254	0.294	−0.344	0.149	0.132	0.590	0.142	0.562	−0.276	0.252
SN PD *Z* Score Variance Within Network	**0.528***	**0.020**	0.421	0.073	−0.275	0.254	−0.178	0.467	−0.215	0.377	0.281	0.244	0.137	0.575	−0.152	0.533
SN PD Number of *Z* scores > 1.96 *SD*	**0.465***	**0.045**	0.369	0.120	−0.354	0.137	−0.221	0.363	−0.231	0.341	0.160	0.513	0.120	0.625	−0.175	0.473
SN PD Percentage *Z* scores > 1.96 *SD*	**0.465***	**0.045**	0.369	0.120	−0.354	0.137	−0.221	0.363	−0.231	0.341	0.160	0.513	0.120	0.625	−0.175	0.473
FPN PD Peak *Z* Score	0.407	0.084	0.271	0.262	−0.392	0.096	−0.433	0.064	−0.209	0.391	0.244	0.314	0.153	0.533	−0.320	0.182
FPN PD Mean *Z* Score	0.453	0.052	0.365	0.125	**−0.486***	**0.035**	−0.410	0.081	−0.307	0.201	0.219	0.368	0.184	0.450	−0.396	0.093
FPN PD *Z* Score Variance Within Network	0.447	0.055	0.315	0.189	**−0.500***	**0.029**	−0.425	0.070	−0.316	0.187	0.137	0.576	0.182	0.455	−0.426	0.069
FPN PD Number of *Z* scores > 1.96 *SD*	0.453	0.052	0.337	0.159	**−0.496***	**0.031**	−0.424	0.070	−0.310	0.196	0.178	0.465	0.133	0.587	−0.403	0.087
FPN PD Percentage *Z* scores > 1.96 *SD*	0.453	0.052	0.337	0.159	**−0.496***	**0.031**	−0.424	0.070	−0.310	0.196	0.178	0.465	0.133	0.587	−0.403	0.087

Table 4

Spearman's Rank-Order Correlation Between Network Connectivity and Cognition, N = 19

Network Connectivity Measures	Spearman's Correlation															
	TMT-A		TMT-B		RBANS Total Index		Immediate Memory Index		Visuospatial Index		Language Index		Attention Index		Delayed Memory Index	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
DMN CSD Peak *Z* score	0.215	0.376	0.146	0.551	−0.376	0.113	**−0.595*****	**0.007**	−0.004	0.988	0.198	0.417	−0.022	0.929	−0.299	0.214
DMN CSD Mean *Z* score	0.162	0.506	−0.143	0.560	−0.336	0.159	**−0.681*****	**0.001**	0.062	0.801	0.195	0.423	−0.037	0.880	−0.239	0.325
DMN CSD *Z* Score Variance Within Network	0.147	0.548	−0.042	0.865	−0.346	0.147	**−0.675*****	**0.002**	0.108	0.660	0.259	0.284	−0.053	0.829	−0.176	0.471
DMN CSD Number of *Z* scores > 1.96 *SD*	0.174	0.475	−0.058	0.815	**−0.462***	**0.047**	**−0.720*****	**0.001**	0.086	0.727	0.084	0.731	−0.100	0.682	−0.319	0.183
DMN CSD Percentage *Z* scores > 1.96 *SD*	0.174	0.475	−0.058	0.815	**−0.462***	**0.047**	**−0.720*****	**0.001**	0.086	0.727	0.084	0.731	−0.100	0.682	−0.319	0.183
SN CSD Peak *Z* score	0.122	0.619	0.048	0.846	−0.356	0.135	**−0.659*****	**0.002**	0.180	0.460	0.141	0.565	−0.037	0.879	−0.208	0.394
SN CSD Mean *Z* score	0.073	0.767	−0.172	0.481	−0.349	0.143	**−0.734*****	**0.000**	0.160	0.512	0.140	0.567	−0.005	0.984	−0.233	0.337
SN CSD Mean *Z* Score Variance Within Network	0.110	0.653	0.013	0.957	−0.332	0.165	**−0.641*****	**0.003**	0.176	0.472	0.223	0.360	−0.035	0.888	−0.137	0.577
SN CSD Number of *Z* scores > 1.96 *SD*	0.150	0.540	−0.011	0.966	−0.340	0.154	**−0.665*****	**0.002**	0.230	0.343	0.190	0.436	−0.117	0.633	−0.200	0.413
SN CSD Percentage *Z* scores > 1.96 *SD*	0.150	0.540	−0.011	0.966	−0.340	0.154	**−0.665*****	**0.002**	0.230	0.343	0.190	0.436	−0.117	0.633	−0.200	0.413
FPN CSD Peak *Z* score	0.187	0.442	0.056	0.819	−0.350	0.142	**−0.654*****	**0.002**	0.081	0.740	0.205	0.399	0.011	0.963	−0.251	0.300
FPN CSD Mean *Z* Score	0.216	0.376	−0.133	0.588	−0.327	0.172	**−0.696*****	**0.001**	0.073	0.766	0.152	0.533	0.051	0.837	−0.248	0.306
FPN CSD *Z* Score Variance Within Network	0.149	0.542	−0.037	0.881	−0.319	0.183	**−0.666*****	**0.002**	0.160	0.512	0.305	0.203	−0.077	0.754	−0.125	0.610
FPN CSD Number of *Z* scores > 1.96 *SD*	0.212	0.384	−0.041	0.869	−0.381	0.107	**−0.672*****	**0.002**	0.111	0.650	0.077	0.756	−0.070	0.777	−0.254	0.294
FPN CSD Percentage *Z* scores > 1.96 *SD*	0.212	0.384	−0.041	0.869	−0.381	0.107	**−0.672*****	**0.002**	0.111	0.650	0.077	0.756	−0.070	0.777	−0.254	0.294

Note. * = significant at $p = 0.05$; ** significant at the 0.01 level; *** = remained significant after Benjamini-Hochberg correction for multiple comparisons; DMN = default mode network; SN = salience network; FPN = frontal-parietal network; IC = instantaneous coherence; LC = lagged coherence; PD = phase difference; CSD = current source density; PCSS = Post-Concussion Symptom Scale; RBANS = The Repeatable Battery for the Assessment of Neuropsychological Status Update; TMT-A = Trail Making Test Part A; TMT-B = Trail Making Test Part B.

Cognitive Function and Post-Concussion Symptoms

To test the hypothesis that higher PCSS scores are associated with reduced cognitive performance (measured by a neuropsychological battery) in the acute post-concussion phase, Pearson product-moment correlation analyses were conducted. Three outliers were detected and managed using winsorization, being three or more standard deviations away from the mean. Assumption testing revealed that the PCSS total score was not normally distributed according to the Shapiro-Wilk statistic (p = 0.018); however, since skew and kurtosis were within ±1 (0.757 and −0.236, respectively), a z-test was used to assess for normality as described in Mishra et al. (2019). The values for zskew (1.44) and zkurtosis (−0.23) were within ±1.96, so normality was assumed. All other assumptions for Pearson's correlation were met.

Table 5 shows the descriptive statistics for the cognitive variables. All participants demonstrated adequate effort on cognitive testing as measured by their FIT scores ranging from 12 to 15, and their RBANS effort index scores being 3 or below. To assess the size and direction of the linear relationship between post-concussion symptoms and cognitive function, a Pearson's product-moment correlation was conducted (see Table 5 for results). No significant correlations were found between acute symptoms and cognitive function.

To further explore the relationship between cognitive function and PCSS domains, Spearman's rank-order correlation was conducted as listed in Table 6. While the PCSS cognitive domain was normally distributed, the assumptions of linearity and homoscedasticity were not met, so nonparametric analyses were conducted for all variables. No significant correlations were found between cognitive function and PCSS symptom domains.

Discussion

To our knowledge, this study is the first to investigate the utility of measuring network function using qEEG in the acute stages post-mTBI and, as such, serves as an initial pilot study of this novel approach in an mTBI sample. The present study aimed to measure the association between brain network connectivity, acute PCSS scores, and cognition (processing speed, inhibition/switching, immediate memory, visuospatial/constructional, language, attention, and delayed memory) in Australian adults within 7 days after mTBI.

The hypotheses that network dysregulation would be positively correlated with PCSS total score and PCSS symptom domains were not supported. This finding was not in keeping with previous literature suggesting an association between altered brain network connectivity and post-concussion symptoms (D'Souza et al., 2020; Mortaheb et al., 2021; Ramage et al., 2022). DMN integrity is essential for cognition, and cingulum damage or decreased DMN connectivity as detected by diffusion tensor imaging (DTI) have been associated with sustained attention deficits following mTBI (Bonnelle, Leech, et al., 2011). Furthermore, reduced DMN connectivity, as measured by neurite orientation dispersion and density imaging (NODDI) and DTI, has been linked with increased symptoms 6 months post-mTBI (Palacios et al., 2020). Altered connections between the DMN and other networks such as the SN and FPN have also been seen on resting-state fMRI post-mTBI. The SN has a central role in shifting attention externally (Hayes et al., 2016), as well as modulating DMN activity (Sharp, Scott, et al., 2014). Impaired connectivity between the DMN and SN has been associated with reduced DMN inhibition during a task requiring external focus on fMRI, and this was linked with cognitive impairments post-TBI (Bonnelle, Ham, et al., 2012; Jilka et al., 2014). Altered connectivity between the DMN and SN may also be due to structural pathology within the SN as seen on fMRI (Hayes et al., 2016), and heightened connectivity within both networks has been associated with cognitive deficits in a DTI and NODDI study (Palacios et al., 2020). The FPN is central to various cognitive tasks including reasoning, working memory, set-shifting, attention, (Martínez et al., 2013; Niendam et al., 2012), and novel complex tasks (Chenot et al., 2021). Altered connectivity seen on fMRI within the FPN has been linked to heightened cognitive fatigue and reduced sustained effort during cognitive activities, 3 to 24 months post-mTBI (Ramage et al., 2022). Furthermore, a review of various functional neuroimaging techniques including fMRI and magnetoencephalography, found a reduced negative association between the FPN and DMN is commonly linked to PPCS (Mortaheb et al., 2021).

Table 5

Pearson Product-Moment Correlations Between PCSS Score and Cognitive Function, N = 19

Cognitive Measure	Descriptives		Pearson Correlation	
	M	*SD*	*r*	*p*
PCSS Total Score	26.89	21.57		
TMT-A *Z* score	−0.20	1.07	−0.139	0.285
TMT-B *Z* score	−0.78	1.34	−0.160	0.256
RBANS total Index Score	91.47	11.40	−0.058	0.407
Immediate Memory	93.05	14.94	0.143	0.279
Visuospatial/constructional	95	17.11	−0.110	0.328
Language	103.21	12.06	−0.371	0.059
Attention	92.47	15.92	−0.051	0.418
Delayed Memory	94.26	9.71	0.020	0.468

Note. TMT = Trail Making Test; RBANS = Repeatable Battery for the Assessment of Neuropsychological Status.

Table 6

Spearman's Rank-Order Correlation Between Cognitive Function and PCSS Domains, N = 19

Cognitive Measures	Spearman's Correlation							
	PCSS Cognitive		PCSS Physical		PCSS Affective		PCSS Sleep	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*
TMT-A	−0.279	0.248	−0.118	0.632	−0.356	0.134	−0.131	0.594
TMT-B	−0.114	0.643	0.056	0.820	−0.138	0.572	0.100	0.685
RBANS Total Index	−0.224	0.357	−0.034	0.889	0.109	0.658	−0.133	0.589
Immediate Memory Index	−0.020	0.937	−0.004	0.985	0.017	0.945	0.125	0.611
Visuospatial Index	−0.217	0.372	0.214	0.379	0.088	0.721	0.078	0.752
Language Index	−0.287	0.233	−0.173	0.478	−0.207	0.396	−0.336	0.160
Attention Index	0.027	0.914	0.180	0.461	0.070	0.776	−0.108	0.661
Delayed Memory Index	−0.189	0.439	0.069	0.779	0.263	0.278	0.082	0.738

Note. PCSS = Post-Concussion Symptom Scale; RBANS = The Repeatable Battery for the Assessment of Neuropsychological Status Update; TMT-A = Trail Making Test Part A; TMT-B = Trail Making Test Part B.

One possible reason for these findings is that while qEEG has been previously used to measure brain function in mTBI (Rapp et al., 2015), the current study used a novel approach measuring whole networks rather than individual brain areas. The whole network approach may have "averaged out" the dysfunctional areas resulting in reduced detection of dysfunction. Another reason may be that 89% of participants exercised on average 3 days per week, and physical activity has been shown to modulate brain health (Dorsman et al., 2020; McFadden et al., 2013), so it is possible that their exercise level was protective against network dysregulation and cognitive deficits. Additionally, 47% of participants were taking medication, so it is possible that their brain activity and cognitive function was modulated by the medications, concealing the true effects of mTBI on their brain's connectivity. It is therefore recommended that

exercise and medication are accounted for in future research as potential confounding factors.

The hypothesis that network dysregulation would be negatively correlated with cognitive performance was partially supported. Network connectivity (DMN, FPN) and CSD (DMN, SN, FPN) were significantly correlated with immediate memory measures. This supports the findings in previous literature that brain dysfunction is correlated with cognitive dysfunction post-mTBI (Bonnelle, Ham, et al., 2012; Jilka et al., 2014; Thatcher, North, et al., 2001). While acknowledging the limitations of the current study, this finding may have significant implications for the clinical assessment of mTBI, considering that neuropsychological measures of immediate memory are relatively quick and easy to administer in a clinical setting, and may be an indicator of brain network dysfunction in the acute post-mTBI phase. Testing this hypothesis in a larger sample, with a more comprehensive cognitive assessment battery would improve its generalizability and may add to its utility in the clinical setting.

The hypotheses that cognitive performance would be negatively correlated with PCSS total score and PCSS symptom domains were not supported. This was not consistent with previous literature suggesting that individuals with higher symptom scores had inferior cognitive function compared to those with lower symptom scores (Custer et al., 2016); however, participants in the present study were older (minimum 21 years) than those who participated in the previous study (minimum 13 years) and this may be one reason for the different findings. It is also possible that our neuropsychological measures were not sensitive enough to detect mild dysfunction as is the case for most individuals in mTBI, hence, a more comprehensive cognitive battery is recommended for future research. Finally, the PCSS may be too sensitive to non-mTBI specific symptoms, highlighting the need to better understand the underlying factor structure of this commonly used measure.

Limitations of the current study include a small sample size and limited power. The age range was also large, and in conjunction with the small sample size, did not allow for stratification of age categories, to explore whether age-related effects were also impacting the findings. Specifically, the literature suggests that with advancing age, there is a reduction in within-network resting-state functional connectivity and an increase in functional connectivity across networks (Ferreira & Busatto, 2013; Kong et al., 2020). This is especially the case for the DMN and networks involved in higher order cognition (Andrews-Hanna et al., 2007; Chan et al., 2014; Damoiseaux et al., 2008) which may be considered to encompass all three networks analyzed in this study. Additionally, it is acknowledged that age-related cognitive decline may also have an impact on neuropsychological test performance, limiting the interpretation of cognitive deficits as purely mTBI-related in older age groups. While the nature of neuropsychological testing is somewhat protective against this effect due to the comparison of raw scores with age-matched normative databases, it remains possible that subclinical changes may already be occurring, regardless of the observed scores. Given the limited sample size, the heterogenous mechanisms of injury within the sample may be seen as a limitation, particularly since over half the participants had sports-related injuries. Nonetheless, considering mTBI is a heterogeneous condition, the fact that this sample encompasses that heterogeneity makes it more applicable to the general population than a single injury mechanism sample. There was also a lack of control group; however, this was countered by the fact that the brain network and cognitive measures for each participant were compared to a normative sample of typically developing adults. It is acknowledged that normative databases used in this study may not be completely representative of the current cohort given the anticipated impact of the COVID-19 pandemic on individuals and the community, resulting in an increased prevalence of stress, mental health challenges, and a changing society. Additionally, individuals with comorbid conditions were not separated for analysis purposes, which may have impacted the results due to overlapping or amplified effects. While the impact of multiple mTBI remains controversial (Young, 2020), it has been suggested that repeated mTBI may result in poorer outcomes (Mez et al., 2017). Considering 52.6% of participants in this study had one or more previous mTBI, it is worth noting that these participants may differ from participants who had a single injury, and hence, this factor may limit generalizability of the results. Having a larger sample size may provide a more convincing demonstration of the impact of multiple mTBI on functioning. Moreover, six participants stated they were unsure whether they experienced loss of consciousness which may result in misclassification of mTBI. However, participants received a medical diagnosis of mTBI prior to being accepted into the study, so that reduced the likelihood of misclassification. Additionally, it was anticipated that if participants experienced loss of consciousness

> 30 min, their LOC would have been observed by a third party or referrer and they would not have been referred to the study. Nonetheless, removing the ambiguity in future studies would be optimal.

The neuropsychological battery used was limited to a screening and repeatable battery which may not have been sensitive enough to detect mild cognitive deficits and it may not have covered all the relevant cognitive domains for post-mTBI. For example, executive functioning measures may be more sensitive to long tract changes since execution functioning requires higher order integrated functioning. It is recommended that future research considers a more comprehensive measure of executive function. It is possible that individuals who sought medical attention required to receive a diagnosis were more motivated for recovery and this may have influenced their symptom ratings and/or their engagement with early intervention or proactive recovery. Finally, confounding variables such as medical history and medication were identified but not accounted for as a variable in this analysis. Overall, there were a number of confounding variables in this study, and it is suggested that further research will be required to confirm the findings of this study relating to the relationships between network connectivity, PCSS scores, and cognition using a more focused methodology.

Future studies could replicate the study in a larger sample with multiple follow up time-points postinjury, to determine the natural history of these findings and their implications for the long term. Observing participant recovery in a longitudinal setting and identifying predictors of long-term outcomes (e.g., symptoms, quality of life) may improve our understanding of the clinical implications of network function and cognitive deficits, better informing decision-making in the acute setting and providing guidance for rehabilitation in a clinical context. Additionally, with a larger sample size it would be of interest to explore sex differences across variables such as PCSS scores, network function, alcohol use at time of injury, exercise levels (i.e., mild versus moderate and vigorous activity and exercise duration), education and whether being under the influence of alcohol at time of injury is associated with increased or worse posttraumatic amnesia. It would also be important to analyze participants with comorbid conditions separately to improve the generalizability of the findings. Lastly, the qEEG analysis techniques utilized in this study were somewhat novel. While previous literature established that coherence, phase, and CSD of specific 10–20 electrode sites differentiated between mTBI cases and controls (Thatcher, North, et al., 2001), to our knowledge these measures had not been explored previously in the context of network connectivity in the mTBI population. The measures selected (peak *z*, mean *z*, *z* variance, numbers of *z* scores above ±1.96, percentage of *z* scores above ±1.96) were also exploratory in nature, and were intended to represent the extent of network dysregulation from several perspectives, that is, not simply looking at the highest *z* score (or the extent of deviation at a single site) but also how many sites within the network were outside the expected range based on a normal curve interpretation using standard deviations. The study requires replication in a larger sample where further stratification of the sample to account for various potential confounding variables may be accounted for. Moreover, the dysregulation was not qualitatively assessed in this study (i.e., it was not determined whether the dysregulation was more prevalent in the slow or fast frequency bands). Adding this information in future research may improve the depth of interpretation of the findings and may also provide an opportunity to differentiate subtypes of PPCS-related dysfunction. For example, those who have comorbid conditions such as attention-deficit/hyperactivity disorder or depression may display a different pattern of dysregulation (e.g., more dysregulation is slow versus fast wave activity) compared to those without comorbid conditions. Further investigation into the utility of qEEG as a measure of network function in the mTBI population, particularly by exploring the association with resting-state fMRI findings, may add to the validity of this neurophysiological measure in the acute setting, enabling a more comprehensive assessment and hence a better understanding of mTBI.

In conclusion, this study demonstrated that in an adult sample with acute concussion, PCSS scores were not a sensitive indicator of neuropsychological status (as indicated by cognitive measures) and did not reflect the status of underlying brain network regulation. The current study also provides preliminary evidence for immediate memory task performance as an indicator of underlying altered network connectivity in acute mTBI. The results of this study bring into question the common practice of using self-report symptom ratings as an indicator of recovery (or injury severity). While this practice may be a useful method to track symptom change over time, and initial symptom burden may inform the recovery trajectory, the findings highlight the need for better development of more screening measures sensitive to mTBI, as well as further evaluating neurophysiological and cognitive impacts of

concussion in the acute post-concussion period. Improving our understanding may assist with selecting targeted interventions and facilitating a more comprehensive recovery post-mTBI including the considering of appropriate guidelines for returning to daily occupations such as sports, work, and school.

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References

Allen, M. (2022). *Exercise after a concussion: When is it OK and what if it makes you feel worse?* Cognitive FX. https://www.cognitivefxusa.com/blog/exercise-after-a-concussion

Andrews-Hanna, J. R., Snyder, A. Z., Vincent, J. L., Lustig, C., Head, D., Raichle, M. E., & Buckner, R. L. (2007). Disruption of large-scale brain systems in advanced aging. *Neuron, 56*(5), 924–935. https://doi.org/10.1016/j.neuron.2007.10.038

Applied Neuroscience Inc. (2023). NeuroGuide with NeuroNavigator add-on tools. [Software]. https://appliedneuroscience.com/product/neuronavigator/

Bai, L., Yin, B., Lei, S., Li, T., Wang, S., Pan, Y., Gan, S., Jia, X., Li, X., Xiong, F., Yan, Z., & Bai, G. (2022). Reorganized hubs of brain functional networks following acute mild traumatic brain injury. *Journal of Neurotrauma, 40*(1–2), 63–73. https://doi.org/10.1089/neu.2021.0450

Barr, W. B., Prichep, L. S., Chabot, R., Powell, M. R., & McCrea, M. (2012). Measuring brain electrical activity to track recovery from sport-related concussion. *Brain Injury, 26*(1), 58–66. https://doi.org/10.3109/02699052.2011.608216

Bedard, M., Steffener, J., & Taler, V. (2020). Long-term cognitive impairment following single mild traumatic brain injury with loss of consciousness: Findings from the Canadian Longitudinal Study on Aging. *Journal of Clinical and Experimental Neuropsychology, 42*(4), 344–351. https://doi.org/10.1080/13803395.2020.1714552

Bonnelle, V., Ham, T., Leech, R., Kinnunen, K. M., Mehta, M. A., Greenwood, R. J., & Sharp, D. J. (2012). Salience network integrity predicts default mode network function after traumatic brain injury. *Proceedings of the National Academy of Sciences of the United States of America, 109*(12), 4690–4695. https://doi.org/10.1073/pnas.1113455109

Bonnelle, V., Leech, R., Kinnunen, K. M., Ham, T. E., Beckmann, C. F., De Boissezon, X., Greenwood, R. J., & Sharp, D. J. (2011). Default mode network connectivity predicts sustained attention deficits after traumatic brain injury. *The Journal of Neuroscience, 31*(38), 13442–13451. https://doi.org/10.1523/JNEUROSCI.1163-11.2011

Brodmann, K. (1909). Vergleichende lokalisationslehre der grosshirnrinde in ihren prinzipien dargestellt auf grund des zellenbaues. Leipzig, Germany: Johann Ambrosius Barth.

Cannon, R. L., Kerson, C., & Hampshire, A. (2011). sLORETA and fMRI detection of medial prefrontal default network anomalies in adult ADHD. *Journal of Neurotherapy, 15*(4), 358–373. https://doi.org/10.1080/10874208.2011.623093

Carroll, E. L., Outtrim, J. G., Forsyth, F., Manktelow, A. E., Hutchinson, P. J. A., Tenovuo, O., Posti, J. P., Wilson, L., Sahakian, B. J., Menon, D. K., & Newcombe, V. F. J. (2020). Mild traumatic brain injury recovery: A growth curve modelling analysis over 2 years. *Journal of Neurology, 267*(11), 3223–3234. https://doi.org/10.1007/s00415-020-09979-x

Carroll, L. J., Cassidy, J. D., Cancelliere, C., Côté, P., Hincapié, C. A., Kristman, V. L., Holm, L. W., Borg, J., Nygren-de Boussard, C., & Hartvigsen, J. (2014). Systematic review of the prognosis after mild traumatic brain injury in adults: Cognitive, psychiatric, and mortality outcomes: Results of the international collaboration on mild traumatic brain injury prognosis. *Archives of Physical Medicine and Rehabilitation, 95*(3 Suppl.), S152–S173. https://doi.org/10.1016/j.apmr.2013.08.300

Chan, M. Y., Park, D. C., Savalia, N. K., Petersen, S. E., & Wig, G. S. (2014). Decreased segregation of brain systems across the healthy adult lifespan. *Proceedings of the National Academy of Sciences of the United States of America, 111*(46), E4997–E5006. https://doi.org/10.1073/pnas.1415122111

Chenot, Q., Lepron, E., De Boissezon, X., & Scannella, S. (2021). Functional connectivity within the fronto-parietal network predicts complex task performance: A fNIRS study. *Frontiers in Neuroergonomics, 2*, 718176. https://doi.org/10.3389/fnrgo.2021.718176

Colantonio, A., Ratcliff, G., Chase, S., & Escobar, M. (2000). Is cognitive performance related to level of community integration many years after traumatic brain injury? *Brain and Cognition, 44*(1), 19–20. https://doi.org/10.1006/brcg.1999.1207

Custer, A., Sufrinko, A., Elbin, R. J., Covassin, T., Collins, M., & Kontos, A. (2016). High baseline postconcussion symptom scores and concussion outcomes in athletes. *Journal of Athletic Training, 51*(2), 136–141. https://doi.org/10.4085/1062-6050-51.2.12

D'Souza, M. M., Kumar, M., Choudhary, A., Kaur, P., Kumar, P., Rana, P., Trivedi, R., Sekhri, T., & Singh, A. K. (2020). Alterations of connectivity patterns in functional brain networks in patients with mild traumatic brain injury: A longitudinal resting-state functional magnetic resonance imaging study. *The Neuroradiology Journal, 33*(2), 186–197. https://doi.org/10.1177/1971400920901706

Damoiseaux, J. S., Beckmann, C. F., Arigita, E. J. S., Barkhof, F., Scheltens, P., Stam, C. J., Smith, S. M., & Rombouts, S. A. R. B. (2008). Reduced resting-state brain activity in the "default network" in normal aging. *Cerebral Cortex, 18*(8), 1856–1864. https://doi.org/10.1093/cercor/bhm207

de Freitas Cardoso, M. G., Faleiro, R. M., de Paula, J. J., Kummer, A., Caramelli, P., Teixeira, A. L., de Souza, L. C., & Miranda, A. S. (2019). Cognitive impairment following acute

mild traumatic brain injury. *Frontiers in Neurology, 10*, 198. https://doi.org/10.3389/fneur.2019.00198

Dorsman, K. A., Weiner-Light, S., Staffaroni, A. M., Brown, J. A., Wolf, A., Cobigo, Y., Walters, S., Kramer, J. H., & Casaletto, K. B. (2020). Get moving! Increases in physical activity are associated with increasing functional connectivity trajectories in typically aging adults. *Frontiers in Aging Neuroscience, 12*, 104. https://doi.org/10.3389/fnagi.2020.00104

Electro-Cap International, Inc. (n.d.). Electro-Cap 19-channel system with Electro-Gel electroconductive gel [Apparatus].

Fell, J., & Axmacher, N. (2011). The role of phase synchronization in memory processes. *Nature Reviews Neuroscience, 12*(2), 105–118. https://doi.org/10.1038/nrn2979

Ferreira, L. K., & Busatto, G. F. (2013). Resting-state functional connectivity in normal brain aging. *Neuroscience & Biobehavioral Reviews, 37*(3), 384–400. https://doi.org/10.1016/j.neubiorev.2013.01.017

Field, A. (2013). Discovering statistics using IBM SPSS statistics (4th ed.). London, UK: SAGE Publications Ltd.

Gardner, R. C., & Yaffe, K. (2015). Epidemiology of mild traumatic brain injury and neurodegenerative disease. *Molecular and Cellular Neurosciences, 66*(Part B), 75–80. https://doi.org/10.1016/j.mcn.2015.03.001

Gennarelli, T. (1971). Comparison of translational and rotational motions in experimental cerebral concussion. Paper presented at the *15th Stapp Car Crash Conference*.

Gozt, A. K., Hellewell, S. C., Thorne, J., Thomas, E., Buhagiar, F., Markovic, S., Van Houselt, A., Ring, A., Arendts, G., Smedley, B., Van Schalkwyk, S., Brooks, P., Iliff, J., Celenza, A., Mukherjee, A., Xu, D., Robinson, S., Honeybul, S., Cowen, G., Licari, M., … Fitzgerald, M. (2021). Predicting outcome following mild traumatic brain injury: Protocol for the longitudinal, prospective, observational Concussion Recovery (CREST) cohort study. *BMJ Open, 11*(5), Article e046460. https://doi.org/10.1136/bmjopen-2020-046460

Gumm, K., Taylor, T., Orbons, K., Carey, L., & PTA Working Party (2011). *Post traumatic amnesia screening and management guideline*. The Royal Melbourne Hospital. https://www.connectivity.org.au/wp-content/uploads/2022/08/TRM01.01-Post-Traumatic-Amnesia-Screening-and-Management-V5.1_06.20.docx.converted.pdf

Ham, T. E., Bonnelle, V., Hellyer, P., Jilka, S., Robertson, I. H., Leech, R., & Sharp, D. J. (2014). The neural basis of impaired self-awareness after traumatic brain injury. *Brain, 137*(2), 586–597. https://doi.org/10.1093/brain/awt350

Han, K., Chapman, S. B., & Krawczyk, D. C. (2016). Disrupted intrinsic connectivity among default, dorsal attention, and frontoparietal control networks in individuals with chronic traumatic brain injury. *Journal of the International Neuropsychological Society, 22*(Suppl. 2), 263–279. https://doi.org/10.1017/s1355617715001393

Haneef, Z., Levin, H. S., Frost, J. D. Jr., & Mizrahi, E. M. (2013). Electroencephalography and quantitative electroencephalography in mild traumatic brain injury. *Journal of Neurotrauma, 30*(8), 653–656. https://doi.org/10.1089/neu.2012.2585

Harmon, K. G., Clugston, J. R., Dec, K., Hainline, B., Herring, S., Kane, S. F., Kontos, A. P., Leddy, J. J., McCrea, M., Poddar, S. K., Putukian, M., Wilson, J. C., & Roberts, W. O. (2019). American Medical Society for Sports Medicine position statement on concussion in sport. *British Journal of Sports Medicine, 53*(4), 213–225. https://doi.org/10.1136/bjsports-2018-100338

Hayes, J. P., Bigler, E. D., & Verfaellie, M. (2016). Traumatic brain injury as a disorder of brain connectivity. *Journal of the International Neuropsychological Society, 22*(2), 120–137. https://doi.org/10.1017/S1355617715000740

Ims, P. D. (2019). Re-training the injured brain: A case series in sLORETA neurofeedback as an acute concussion intervention in youth. Towson University.

Iverson, G. L. (2019). Network analysis and precision rehabilitation for the post-concussion syndrome. *Frontiers in Neurology, 10*, 489. https://doi.org/10.3389/fneur.2019.00489

Jagnoor, J., & Cameron, I. (2014). Traumatic brain injury – support for injured people and their careers. *Australian Family Physician, 43*(11), 758–763. https://www.racgp.org.au/afp

James, S. L., Theadom, A., Ellenbogen, R. G., Bannick, M. S., Montjoy-Venning, W., Lucchesi, L. R., Abbasi, N., Abdulkader, R., Abraha, H. N., Adsuar, J. C., Afarideh, M., Agrawal, S., Ahmadi, A., Ahmed, M. B., Aichour, A. N., Aichour, I., Aichour, M. T. E., Akinyemi, R. O., Akseer, N., Alahdab, F., ... Murray, C. J. L. (2019). Global, regional, and national burden of traumatic brain injury and spinal cord injury, 1990–2016: A systematic analysis for the Global Burden of Disease Study 2016. *The Lancet Neurology, 18*(1), 56–87. https://doi.org/10.1016/S1474-4422(18)30415-0

Jilka, S. R., Scott, G., Ham, T., Pickering, A., Bonnelle, V., Braga, R. M., Leech, R., & Sharp, D. J. (2014). Damage to the salience network and interactions with the default mode network. *The Journal of Neuroscience, 34*(33), 10798–10807. https://doi.org/10.1523/JNEUROSCI.0518-14.2014

Jungfer, P. (2017). Psychiatric aspects of mild traumatic brain injury: The obsolete diagnosis of post-concussion syndrome. Precedent (Sydney, N.S.W.)(138), 37–40. https://www.austlii.edu.au/cgi-bin/viewdoc/au/journals/PrecedentAULA/2017/10.html

Kerasidis, H., & Simmons, J. (2021). Quantitative EEG analysis in clinical practice: Concussion injury. *Clinical EEG and Neuroscience, 52*(2), 114–118. https://doi.org/10.1177/1550059421989112

Kong, T. S., Gratton, C., Low, K. A., Tan, C. H., Chiarelli, A. M., Fletcher, M. A., Zimmerman, B., Maclin, E. L., Sutton, B. P., Gratton, G., & Fabiani, M. (2020). Age-related differences in functional brain network segregation are consistent with a cascade of cerebrovascular, structural, and cognitive effects. *Network Neuroscience, 4*(1), 89–114. https://doi.org/10.1162/netn_a_00110

Langer, L., Levy, C., & Bayley, M. (2020). Increasing incidence of concussion: True epidemic or better recognition? *The Journal of Head Trauma Rehabilitation, 35*(1), E60–E66. https://doi.org/10.1097/HTR.0000000000000503

Levine, A. J., Miller, E. N., Becker, J. T., Selnes, O. A., & Cohen, B. A. (2004). Normative data for determining significance of test–retest differences on eight common neuropsychological instruments. *The Clinical Neuropsychologist, 18*(3), 373–384. https://doi.org/10.1080/1385404049052420

Liotta, M. (2021). *Rest or exercise following concussion?* NewsGP. https://www1.racgp.org.au/newsgp/clinical/rest-or-exercise-following-concussion#:~:text=A%20graded%20return%20to%20daily,the%20likelihood%20of%20prolonged%20symptoms

Lovell, M. R., Iverson, G. L., Collins, M. W., Podell, K., Johnston, K. M., Pardini, D., Pardini, J., Norwig, J., & Maroon, J. C. (2006). Measurement of symptoms following sports-related concussion: Reliability and normative data for the post-concussion scale. *Applied Neuropsychology, 13*(3), 166–174. https://doi.org/10.1207/s15324826an1303_4

Luria, A. (1973). *The working brain*. Basic Books.

Martínez, K., Solana, A. B., Burgaleta, M., Hernández-Tamames, J. A., Alvarez-Linera, J., Román, F. J., Alfayate, E., Privado, J., Escorial, S., Quiroga, M. A., Karama, S., Bellec, P. & Colom, R. (2013). Changes in resting-state functionally connected parietofrontal networks after videogame practice. *Human Brain Mapping, 34*(12), 3143–3157. https://doi.org/10.1002/hbm.22129

Masel, B. E., & DeWitt, D. S. (2010). Traumatic brain injury: A disease process, not an event. *Journal of Neurotrauma, 27*(8), 1529–1540. https://doi.org/10.1089/neu.2010.1358

Mayer, A. R., Mannell, M. V., Ling, J., Gasparovic, C., & Yeo, R. A. (2011). Functional connectivity in mild traumatic brain injury. *Human Brain Mapping, 32*(11), 1825–1835. https://doi.org/10.1002/hbm.21151

Mayer, A. R., Quinn, D. K., & Master, C. L. (2017). The spectrum of mild traumatic brain injury. A review. *Neurology, 89*(6), 623–632. https://doi.org/10.1212/wnl.0000000000004214

McCrea, M., Prichep, L., Powell, M. R., Chabot, R., & Barr, W. B. (2010). Acute effects and recovery after sport-related concussion: A neurocognitive and quantitative brain electrical activity study. *The Journal of Head Trauma Rehabilitation, 25*(4), 283–292. https://doi.org/10.1097 /HTR.0b013e3181e67923

McFadden, K. L., Cornier, M.-A., Melanson, E. L., Bechtell, J. L., & Tregellas, J. R. (2013). Effects of exercise on resting-state default mode and salience network activity in overweight/obese adults. *NeuroReport, 24*(15), 866–871. https://doi.org/10.1097/wnr.0000000000000013

McInnes, K., Friesen, C. L., MacKenzie, D. E., Westwood, D. A., & Boe, S. G. (2017). Mild traumatic brain injury (mTBI) and chronic cognitive impairment: A scoping review. *PLoS ONE, 14*(6), Article e0174847. https://doi.org/10.1371 /journal.pone.0174847

McKay, C., Wertheimer, J. C., Fichtenberg, N. L., & Casey, J. E. (2008). The Repeatable Battery for The Assessment of Neuropsychological Status (RBANS): Clinical utility in a traumatic brain injury sample. *The Clinical Neuropsychologist, 22*(2), 228–241. https://doi.org/10.1080/13854040701260370

McKee, A. C., & Daneshvar, D. H. (2015). Chapter 4 - The neuropathology of traumatic brain injury. *Handbook of Clinical Neurology, 127*, 45–66. https://doi.org/10.1016/b978-0-444-52892-6.00004-0

McLeod, T. C. V., & Leach, C. (2012). Psychometric properties of self-report concussion scales and checklists. *Journal of Athletic Training, 47*(2), 221–223. https://doi.org/10.4085 /1062-6050-47.2.221

Merritt, V. C., Bradson, M. L., Meyer, J. E., & Arnett, P. A. (2017). Evaluating the test-retest reliability of symptom indices associated with the ImPACT post-concussion symptom scale (PCSS). *Journal of Clinical and Experimental Neuropsychology, 40*(4), 377–388. https://doi.org/10.1080 /13803395.2017.1353590

Messé, A., Caplain, S., Pélégrini-Issac, M., Blancho, S., Lévy, R., Aghakhani, N., Montreuil, M., Benali, H., & Lehéricy, S. (2013). Specific and evolving resting-state network alterations in post-concussion syndrome following mild traumatic brain injury. *PLoS ONE, 8*(10). https://doi.org/10.1371 /journal.pone.0065470

Mesulam, M. M. (2000). *Principles of behavioral and cognitive neurology* (2nd ed.). Oxford University Press.

Mez, J., Daneshvar, D. H., Kiernan, P. T., Abdolmohammadi, B., Alvarez, V. E., Huber, B. R., Alosco, M. L., Solomon, T. M., Nowinski, C. J., McHale, L., Cormier, K. A., Kubilus, C. A., Martin, B. M., Murphy, L., Baugh, C. M., Montenigro, P. H., Chaisson, C. E., Tripodis, Y., Kowall, N. W., Weuve, J., … McKee, A. C. (2017). Clinicopathological evaluation of chronic traumatic encephalopathy in players of American football. *JAMA, 318*(4), 360–370. https://doi.org/10.1001 /jama.2017.8334

Milz, P., Faber, P. L., Lehmann, D., Kochi, K., & Pascual-Marqui, R. D. (2014). sLORETA intracortical lagged coherence during breath counting in meditation-naïve participants. *Frontiers in Human Neuroscience, 8*, 303. https://doi.org/10.3389 /fnhum.2014.00303

Mishra, P., Pandey, C. M., Singh, U., Gupta, A., Sahu, C., & Keshri, A. (2019). Descriptive statistics and normality tests for statistical data. *Annals of Cardiac Anaesthesia, 22*(1), 67–72. https://doi.org/10.4103/aca.ACA_157_18

Mistar, Ltd. (n.d.). MITSAR-EEG-BT 21 EEG 4 Poly channel and amplifier [Apparatus]. https://mitsar-eeg.com/mitsar-eeg-systems-specification/

Mito, R., Parker, D. M., Abbott, D. F., Makdissi, M., Pedersen, M., & Jackson, G. D. (2022). White matter abnormalities characterize the acute stage of sports-related mild traumatic brain injury. *Brain Communications, 4*(4), Article fcac208. https://doi.org/10.1093/braincomms/fcac208

Mortaheb, S., Filippini, M. M., Kaux, J.-F., Annen, J., Lejeune, N., Martens, G., Calderón, M. A. F., Laureys, S., & Thibaut, A. (2021). Neurophysiological biomarkers of persistent post-concussive symptoms: A scoping review. *Frontiers in Neurology, 12*, 687197. https://doi.org/10.3389 /fneur.2021.687197

Narayana, P. A. (2017). White matter changes in patients with mild traumatic brain injury: MRI perspective. *Concussion, 2*(2), Cnc35. https://doi.org/10.2217/cnc-2016-0028

Niendam, T. A., Laird, A. R., Ray, K. L., Dean, Y. M., Glahn, D. C., & Carter, C. S. (2012). Meta-analytic evidence for a superordinate cognitive control network subserving diverse executive functions. *Cognitive, Affective, & Behavioral Neuroscience, 12*(2), 241–268. https://doi.org/10.3758 /s13415-011-0083-5

Palacios, E., Owen, J. P., Yuh, E. L., Wang, M. B., Vassar, M. J., Ferguson, A. R., Diaz-Arrastia, R., Giacino, J. T., Okonkwo, D. O., Robertson, C. S., Stein, M. B., Temkin, N., Jain, S., McCrea, M., MacDonald, C. L., Levin, H. S., Manley, G. T., Mukherjee, P., & TRACK-TBI Investigators (2020). The evolution of white matter microstructural changes after mild traumatic brain injury: A longitudinal DTI and NODDI study. *Science Advances, 6*(32), Article eaaz6892. https://doi.org/10.1126 /sciadv.aaz6892

Patricios, J. S., Schneider, K. J., Dvorak, J., Ahmed, O. H., Blauwet, C., Cantu, R. C., Davis, G. A., Echemendia, R. J., Makdissi, M., McNamee, M., Broglio, S., Emery, C. A., Feddermann-Demont, N., Fuller, G. W., Giza, C. C., Guskiewicz, K. M., Hainline, B., Iverson, G. L., Kutcher, J. S., Leddy, J. J., ... Meeuwisse, W. (2023). Consensus statement on concussion in sport: The 6th International Conference on Concussion in Sport–Amsterdam, October 2022. *British Journal of Sports Medicine, 57*(11), 695–711. https://doi.org /10.1136/bjsports-2023-106898

Pavlovic, D., Pekic, S., Stojanovic, M., & Popovic, V. (2019). Traumatic brain injury: Neuropathological, neurocognitive and neurobehavioral sequelae. *Pituitary, 22*, 270–282. https://doi.org/10.1007/s11102-019-00957-9

Pozzato, I., Tate, R. L., Rosenkoetter, U., & Cameron, I. D. (2019). Epidemiology of hospitalised traumatic brain injury in the state of New South Wales, Australia: A population-based study. *Australian and New Zealand Journal of Public Health, 43*(4), 382–388. https://doi.org/10.1111/1753-6405.12878

Prichep, L. S., McCrea, M., Barr, W., Powell, M., & Chabot, R. J. (2013). Time course of clinical and electrophysiological recovery after sport-related concussion. *The Journal of Head Trauma Rehabilitation, 28*(4), 266–273. https://doi.org /10.1097/HTR.0b013e318247b54e

Rabinovich, M. I., Afraimovich, V. S., Bick, C., & Varona, P. (2012). Information flow dynamics in the brain. *Physics of Life Reviews, 9*(1), 51–73. https://doi.org/10.1016 /j.plrev.2011.11.002

Ramage, A. E., Ray, K. L., Franz, H. M., Tate, D. F., Lewis, J. D., & Robin, D. A. (2022). Cingulo-opercular and frontoparietal network control of effort and fatigue in mild traumatic brain injury. *Frontiers in Human Neuroscience, 15*, 788091. https://doi.org/10.3389/fnhum.2021.788091

Randolph, C. (2012). *Repeatable battery for the assessment of neuropsychological status: Update*. Bloomington, USA: PsychCorp.

Rapp, P. E., Keyser, D. O., Albano, A., Hernandez, R., Gibson, D. B., Zambon, R. A., Hairston, W. D., Hughes, J. D., Krystal, A., & Nichols, A. S. (2015). Traumatic brain injury detection using electrophysiological methods. *Frontiers In Human Neuroscience, 9*, 11. https://doi.org/10.3389/fnhum.2015.00011

Reitan, R. M., & Wolfson, D. (1985). *The Halstead-Reitan neuropsychological test battery: Theory and clinical interpretation*. Tucson, AZ: Neuropsychology Press.

Rey, A. (1964). *The clinical examination in psychology*. Paris: University Press of France.

Reznek, L. (2005). The Rey 15-item memory test for malingering: A meta-analysis. *Brain Injury, 19*(7), 539–543. https://doi.org/10.1080/02699050400005242

Rivara, F. P., & Graham, R. (2014). Sports-related concussions in youth: Report from the Institute of Medicine and National Research Council. *JAMA, 311*(3), 239–240. https://doi.org/10.1001/jama.2013.282985

Rowson, S., Bland, M. L., Campolettano, E. T., Press, J. N., Rowson, B., Smith, J. A., Sproule, D. W., Tyson, A. M., & Duma, S. M. (2016). Biomechanical perspectives on concussion in sport. *Sports Medicine and Arthroscopy Review, 24*(3), 100–107. https://doi.org/10.1097/jsa.0000000000000121

Schatz, P., & Ferris, C. S. (2013). One-month test-retest reliability of the ImPACT test battery. *Archives of Clinical Neuropsychology, 28*(5), 499–504. https://doi.org/10.1093/arclin/act034

Schmitt, A., Upadhyay, N., Martin, J. A., Rojas, S., Strüder, H. K., & Boecker, H. (2019). Modulation of distinct intrinsic resting state brain networks by acute exercise bouts of differing intensity. *Brain Plasticity, 5*(1), 39–55. https://doi.org/10.3233/BPL-190081

Sharp, D. J., Beckmann, C., Greenwood, R., Kinnunen, K., Bonnelle, V., De Boissezon, X., Powell, J. H., Counsell, S. J., Patel, M. C., & Leech, R. (2011). Default mode network functional and structural connectivity after traumatic brain injury. *Brain, 134*(8), 2233–2247. https://doi.org/10.1093/brain/awr175

Sharp, D. J., Scott, G., & Leech, R. (2014). Network dysfunction after traumatic brain injury. *Nature Reviews Neurology, 10*(3), 156–166. https://doi.org/10.1038/nrneurol.2014.15

Shumskaya, E., Andriessen, T., Norris, D. G., & Vos, P. (2012). Abnormal whole-brain functional networks in homogeneous acute mild traumatic brain injury. *Neurology, 79*(2), 175–182. https://doi.org/10.1212/WNL.0b013e31825f04fb

Silverberg, N., Wertheimer, J. C., & Fichtenberg, N. L. (2007). An effort index for the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS). *The Clinical Neuropsychologist, 21*(5), 841–854. https://doi.org/10.1080/13854040600850958

Skandsen, T., Nilsen, T. L., Einarsen, C., Normann, I., McDonagh, D., Haberg, A. K., & Vik, A. (2019). Incidence of mild traumatic brain injury: A prospective hospital, emergency room and general practitioner-based study. *Frontiers in Neurology, 10*, 638. https://doi.org/10.3389/fneur.2019.00638

Sours, C., Zhuo, J., Roys, S., Shanmuganathan, K., & Gullapalli, R. P. (2015). Disruptions in resting state functional connectivity and cerebral blood flow in mild traumatic brain injury patients. *PLoS ONE, 10*(8), Article e0134019. https://doi.org/10.1371/journal.pone.0134019

Sponheim, S. R., McGuire, K. A., Kang, S. S., Davenport, N. D., Aviyente, S., Bernat, E. M., & Lim, K. O. (2011). Evidence of disrupted functional connectivity in the brain after combat-related blast injury. *NeuroImage, 54*(Suppl. 1), S21–S29. https://doi.org/10.1016/j.neuroimage.2010.09.007

Stevens, M., Lovejoy, D., Kim, J., Oakes, H., Kureshi, I., & Witt, S. (2012). Multiple resting state network functional connectivity abnormalities in mild traumatic brain injury. *Brain Imaging and Behavior, 6*(2), 293–318. https://doi.org/10.1007/s11682-012-9157-4

Tang, L., Ge, Y., Sodickson, D. K., Miles, L., Zhou, Y., Reaume, J., & Grossman, R. I. (2011). Thalamic resting-state functional networks: Disruption in patients with mild traumatic brain injury. *Radiology, 260*(3), 831. https://doi.org/10.1148/radiol.11110014

Thatcher, R. W., Biver, C., Salazar, A. M., & McAlaster, R. (1998a). Biophysical linkage between MRI and EEG amplitude in traumatic brain injury. *NeuroImage, 7*(4, Part 2), S659. https://doi.org/10.1016/S1053-8119(18)31492-7

Thatcher, R. W., Biver, C., Salazar, A. M., & McAlaster, R. (1998b). Biophysical linkage between MRI and EEG coherence in traumatic brain injury. *NeuroImage, 7*(4, Part 2), S648. https://doi.org/10.1016/S1053-8119(18)31481-2

Thatcher, R. W., North, D. M., Curtin, R. T., Walker, R. A., Biver, C. J., Gomez, J. F., & Salazar, A. M. (2001). An EEG severity index of traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences, 13*(1), 77–87. https://doi.org/10.1176/jnp.13.1.77

Thatcher, R. W., Walker, R. A., Biver, C. J., North, D. N., & Curtin, R. (2003). Quantitative EEG normative databases: Validation and clinical correlation. *Journal of Neurotherapy, 7*(3–4), 87–121. https://doi.org/10.1300/J184v07n03_05

Theadom, A., Parag, V., Dowell, T., McPherson, K., Starkey, N., Barker-Collo, S., Jones, K., Ameratunga, S., & Feigin, V. L. (2016). Persistent problems 1 year after mild traumatic brain injury: A longitudinal population study in New Zealand. *British Journal of General Practice, 66*(642), e16–e23. https://doi.org/10.3399/bjgp16X683161

Tombaugh, T. N. (2004). Trail making test A and B: Normative data stratified by age and education. *Archives of Clinical Neuropsychology, 19*(2), 203–214. https://doi.org/10.1016/S0887-6177(03)00039-8

van Eijck, M. M., Schoonman, G. G., van der Naalt, J., de Vries, J., & Roks, G. (2018). Diffuse axonal injury after traumatic brain injury is a prognostic factor for functional outcome: A systematic review and meta-analysis. *Brain Injury, 32*(4), 395–402. https://doi.org/10.1080/02699052.2018.1429018

Vitacco, D., Brandeis, D., Pascual-Marqui, R., & Martin, E. (2002). Correspondence of event-related potential tomography and functional magnetic resonance imaging during language processing. *Human Brain Mapping, 17*(1), 4–12. https://doi.org/10.1002/hbm.10038

Voormolen, D. C., Cnossen, M. C., Polinder, S., Gravesteijn, B. Y., Von Steinbuechel, N., Real, R. G. L., & Haagsma, J. A. (2019). Prevalence of post-concussion-like symptoms in the general population in Italy, The Netherlands and the United Kingdom. *Brain Injury, 33*(8), 1078–1086. https://doi.org/10.1080/02699052.2019.1607557

Young, G. (2020). Thirty complexities and controversies in mild traumatic brain injury and persistent post-concussion syndrome: A roadmap for research and practice. *Psychological Injury and Law, 13*(4), 427–451. https://doi.org/10.1007/s12207-020-09395-6

Zhang, J., Yoganandan, N., Pintar, F. A., & Gennarelli, T. A. (2006). Role of translational and rotational accelerations on brain strain in lateral head impact. *Biomedical Sciences Instrumentation, 42*, 501–506. Retrieved from https://iaexpress.ca/journals/biomedical-sciences-instrumentation-publishes/

Zhou, Y., Lui, Y. W., Zuo, X.-N., Milham, M. P., Reaume, J., Grossman, R. I., & Ge, Y. (2014). Characterization of thalamo-cortical association using amplitude and connectivity of functional MRI in mild traumatic brain injury. *Journal of Magnetic Resonance Imaging, 39*(6). https://doi.org/10.1002/jmri.24705

Zhou, Y., Milham, M. P., Lui, Y. W., Miles, L., Reaume, J., Sodickson, D. K., Grossman, R. I., & Ge, Y. (2012). Default-mode network disruption in mild traumatic brain injury. *Radiology, 265*(3), 882–892. https://doi.org/10.1148/radiol.12120748

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