

The association between mild traumatic brain injury history and cognitive control

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ABSTRACT

The influence of multiple mild traumatic brain injuries (mTBIs) on neuroelectric and task performance indices of the cognitive control of action monitoring was assessed in individuals with and without a history of concussion. Participants completed a standard clinical neurocognitive assessment and the error-related negativity of the response-locked event-related brain potential and task performance were measured during a modified flanker task. The findings suggested that those individuals with a history of mTBI demonstrate certain failures in cognitive control, and indicated that a greater number of mTBIs may relate to poorer integrity in the evaluation or signaling for control during instances of conflict. Given that these neuroelectric and behavioral differences exist in the absence of disparities in standard clinical assessment, the findings suggest that measures of cognitive control may be more sensitive to signs of chronic cognitive dysfunction resulting from mTBI.

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While an estimated 1.6–3.8 million traumatic brain injuries occur annually in the United States (Langlois, Rutland-Brown, & Wald, 2006), little is known regarding the persistent consequences of these injuries on cognitive function. Mild traumatic brain injuries (mTBIs), or concussions, result from traumatic impacts to the cranium resulting in neuropathological changes in brain tissue (Aubry et al., 2002), with functional neuronal alterations manifesting in a myriad of patterns (Giza & Hovda, 2001). During the acute stages of injury, deficits in neurocognition and postural control are clearly evident (Broglio & Puetz, 2008); however, most reports suggest that these neurological impairments return to baseline levels within five to seven days of concussion diagnosis (Delaney, Lacroix, Leclerc, & Johnston, 2002; McCrea et al., 2003).

Despite this rapid restoration of functional cognitive performance, emerging evidence suggests that deficits in cognitive function may persist beyond the initial recovery phase. However, previous research in this area has yielded seemingly inconclusive results. Within samples of young adults, findings have suggested that despite differences in the number of previous concussive incidents endured, there appears to be no relationship between injury history and neurocognitive functioning (Collie, McCrory, & Makkdissi, 2006; Guskiewicz, Marshall, Broglio, Cantu, & Kirkendall, 2002; Iverson, Brooks, Lovell, & Collins, 2006). However, in a sample of retired athletes, Guskiewicz et al. (2005) observed an

increased incidence of mild cognitive impairment and memory deficits related to previous concussive injury history. Given the apparent discrepancy between the transient nature of concussive injuries in young adults and manifestations of cognitive dysfunction later in life; it may be that previous research with younger adults utilized tests that lacked the requisite sensitivity to detect the potentially subtle neurocognitive decrements indicative of long-term neurological impairments that result from recurrent traumatic brain injury (Broglio, Ferrara, Piland, & Anderson, 2006). Further, these subtle neurocognitive decrements associated with mTBI may become more pronounced with age-related atrophy of neurocognitive processes (De Beaumont et al., 2009). Accordingly, the utilization of tasks, which may be more sensitive to these neurocognitive deficits are necessary to further elucidate these discrepancies.

Aubry et al. (2002) suggest that given the pervasive nature of concussive injury, and the wide variety of injury outcomes, the assessment of multiple aspects of cognitive functioning (such as information processing, planning, memory, and cognitive flexibility) may provide additional insight into the persistent effects of concussive injuries. These aspects of cognition that Aubry et al. (2002) describe are core cognitive processes collectively termed 'cognitive control' (also known as 'executive control'; Diamond, 2006). The term cognitive control describes a subset of goal-directed, self-regulatory operations involved in the selection, scheduling, and coordination of computational processes underlying perception, memory, and action. These cognitive control processes allow for the flexible modulation of top-down attentional control in order to optimize behavioral interactions (Botvinick, Braver, Barch, Carter, & Cohen, 2001; MacDonald, Cohen, Stenger,

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& Carter, 2000). These processes require conscious awareness and are functionally distinct from the processes they organize (Rogers & Monsell, 1995). As a result, tasks requiring cognitive control processes do not habituate or become automatic over time.

Previous investigations utilizing cognitive control tasks have been successful at further elucidating the long-term consequences of mTBI on cognition. Ellemberg, Leclerc, Couture, & Daigle (2007) observed deficits on the Stroop color-word test, which requires inhibition, as well as the Tower of London DX, which requires planning and cognitive flexibility, in a group of previously concussed soccer athletes. Similarly, Collins et al. (1999) reported that previously concussed collegiate athletes demonstrated deficits on the Trails Making Test – B. These studies provide evidence that tasks which require aspects of cognitive control may be better suited for examining the relation between mTBI history and long-term cognitive impairment. Collectively, the findings suggest that further investigation of the influence of mTBI history on specific aspects of cognitive control is warranted.

That is, the broad framework collectively known as cognitive control may be deconstructed into at least two dissociable subsystems of conflict monitoring referred to as 'evaluative' and 'regulative' (see Botvinick et al., 2001 for review). The evaluative subsystem acts to monitor for instances of conflict during information processing and initiates the activation of compensatory adjustments in top-down control (Botvinick et al., 2001). Previous neuroimaging research has suggested that the anterior cingulate cortex (ACC) is involved in the evaluative system of cognitive control as well as the signaling and detection of conflict (Botvinick et al., 2001). The regulative subsystem, which neuroimaging research suggest is likely rooted in the dorsolateral prefrontal cortex (DLPFC; MacDonald et al., 2000), acts to exert top-down control during ongoing information processing allowing for flexible online adjustments of attention (Botvinick et al., 2001). These alterations in attention serve to provide improved maintenance of task demands and stimulus representations to alter an individuals' behavior. As a result, the two subsystems of cognitive control interact to optimize behavioral interactions within the environment (MacDonald et al., 2000).

Previous investigations utilizing a variety of neuroimaging techniques (i.e., functional magnetic resonance imaging [fMRI] and positron emission tomography [PET]) have observed that concussed participants exhibited decreased activation of the DLPFC as well as decreased gray matter volume in the DLPFC and ACC despite observing no differences in task performance (Chen, Johnston, Petrides, & Ptito, 2008; Chen, Kareken, Fastenau, Trexler, & Hutchins, 2003). Thus, the evaluation of cognitive operations occurring at a covert level appears to be more informative in understanding the possible chronic influences of mTBI on cognitive control. As such, the investigation of event-related brain potentials (ERPs) together with measures of task performance provides a means to investigate evaluative (i.e., error-related negativity [ERN], error positivity [P_e]) and regulative (i.e., behavioral adjustments) aspects of the cognitive control of action monitoring. The ERN (also known as the N_e ; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991) is a negative-going response-locked ERP component following incorrect responses that exhibits a maximum amplitude over the midline fronto-central recording sites (Falkenstein et al., 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). A variety of neuroimaging methods have localized the source of the ERN to be at or very near the dorsal ACC (Carter et al., 1998; Dehaene, Posner, & Tucker, 1994; Miltner et al., 2003; van Veen & Carter, 2002). Following the ERN, the P_e , is a positive-going response-locked component that exhibits a maximum amplitude over midline centro-parietal recording sites following incorrect responses (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Previous research has localized the P_e to a distinct neuronal gen-

erator, separate from the ERN, in the rostral ACC (van Veen & Carter, 2002) such that the ERN and P_e are thought to reflect different aspects of error processing (Herrmann, Römmler, Ehrlis, Heidrich, & Fallgatter, 2004). Specifically, the P_e is believed to represent a neuroelectric index of the allocation of attention (Mathewson, Dywan, & Segalowitz, 2005) or the post-response evaluation of an error (Davies, Segalowitz, Dywan, & Pailing, 2001; Falkenstein et al., 1990). Accordingly, the ERN and P_e reflect aspects of the evaluative subsystem of cognitive control. In contrast, behavioral changes following the commission of an error are thought to reflect aspects of the regulative subsystem of cognitive control. According to Rabbitt (2002), individuals are typically aware of erroneous responses and are able to exhibit behavioral corrections to the commission of an error on the vast majority of trials. Therefore, behavioral regulations, such as post-error response slowing, serve as indices of increased levels of regulative aspects of cognitive control acting to modulate an individual's interaction with the environment (Gehring et al., 1993; Kerns et al., 2004). Taken together, the investigation of neuroelectric and behavioral indices of conflict monitoring processes may provide a more sensitive means of investigating the relationship between previous history of mTBI and cognition.

Previous investigations have demonstrated the efficacy of neuroelectric measures in detecting cognitive decrements associated with a history of mTBI (Broglia, Pontifex, O'Connor, & Hillman, 2009; Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000; Gaetz, Goodman, & Weinberg, 2000; Gosselin, Theriault, Leclerc, Montplaisir, & Lassonde, 2006). Specifically, Broglia et al. (2009) and Dupuis et al. (2000) observed deficits in the allocation of attentional resources associated with concussion history, as indexed by decrements in P3 amplitude (Polich, 1987; Polich & Heine, 1996). Others have reported deficits in stimulus processing and evaluation speed, as indexed by P3 latency, as a function of the number of previous concussive incidents endured (Gaetz et al., 2000; Gosselin et al., 2006); despite a failure to observe concomitant group differences using a standard clinical neurocognitive evaluation (Gosselin et al., 2006). Collectively, these investigations provide support for persistent neurocognitive deficits related to mTBI history. However, to date no research has investigated the extent to which previous concussive events may relate to deficits in cognitive control and action monitoring processes.

The goal of the present study was to determine the relationship between a history of mTBI and evaluative and regulative cognitive control. It was hypothesized that those with a history of mTBI would demonstrate deficits in cognitive control as measured using ERPs and task performance, while standard clinical measures of neurocognitive performance would not be sensitive to the subtle group differences. Specifically, those individuals with a history of mTBI are predicted to demonstrate behavioral deficits in response to the task as well as smaller ERN and P_e amplitude, indicative of long-term decrements in the underlying evaluative processes involved in cognitive control. Similarly, those individuals with a history of mTBI are predicted to exhibit deficient regulation of cognitive control as demonstrated by deficits in post-error task performance. Further, given the intended use of standard clinical assessments of neurocognition following a concussive incident, no differences in performance are predicted as a function of mTBI history.

1. Method

1.1. Participants

Table 1 summarizes the demographic and mTBI data for all participants. A total of 77 (25 female) college-aged athletes were recruited from recreational ice hockey, rugby, martial arts, and

Table 1
Summary of the demographic and mTBI data for all participants.

Measure	Control	mTBI
N	36 (15 females)	30 (7 females)
Age (years)	19.4 ± 1.4	19.9 ± 1.2
K-BIT (IQ)	108 ± 7.8	106.9 ± 6
BMI (kg/m ²)	26 ± 3.5	27.1 ± 4.6
Education (years)	13.6 ± 1.4	14.2 ± 1.1
Socioeconomic status	7.9 ± 2.7	8.9 ± 1.6
Mean number of concussions	–	1.7 ± 1.1 (range: 1–5)
Time since last concussion (years)	–	2.9 ± 2.9
Number of symptoms reported	9.1 ± 9.5	6.2 ± 9.0
ImPACT verbal memory (% correct)	89.7 ± 1.5	90.9 ± 1.4
ImPACT visual memory (% correct)	82.6 ± 1.6	79.5 ± 1.6
ImPACT motor speed (composite score)	44.7 ± 1.3	42.7 ± 1.2
ImPACT reaction time (ms)	527.5 ± 12.7	533.7 ± 7.1
ImPACT impulse control (composite score)	10.47 ± 3.6	14.9 ± 4.7

Note: K-BIT is a composite score for IQ.

soccer teams as well as the women's varsity soccer team at the University of Illinois. Participants were bifurcated based on mTBI history to either control ($n=44$; 18 female) or mTBI ($n=33$; 7 female) groups based on the self-report incidence of physician diagnosed concussion history using the American Academy of Neurology injury definition (Practice Parameter, 1997). Further detail of the injuries was elucidated with loss of consciousness reported in 16 of the concussed participants (LOC time was not reported) and amnesia in 15 participants. All participants in the mTBI group reported being injury free at the time of testing. The control group consisted of participants who had never incurred an mTBI incident. All participants provided written informed consent that was approved by the Institutional Review Board of the University of Illinois at Urbana-Champaign and reported being free of neurological disorders, cardiovascular disease, any medications that influence central nervous system function, and had (corrected to) normal vision based on the minimal 20/20 standard. Participants with fewer than six errors were discarded from the analyses ($n=11$; 3 from the mTBI group) leaving a total of 66 participants separated based on mTBI history into either control ($n=36$) or mTBI ($n=30$) groups.

1.2. Procedure

Participants completed an informed consent, the Edinburgh handedness inventory (Oldfield, 1971), a health history and demographics questionnaire, and had their height and weight measured using a stadiometer and a Tanita BWB-600 digital scale, respectively. Prior to neuroelectric testing, participants completed the Kaufman Brief Intelligence Test (K-BIT; Kaufman & Kaufman, 1990) and the ImPACT (ImPACT Applications, Pittsburgh, PA) computer-based neurocognitive assessment. Participants were then fitted with a 64-channel Quik-cap (Compumedics Neuroscan, El Paso, TX), provided task instructions and given 20 practice trials before the task began. Upon completion of the task condition, all electrodes were removed and participants were briefed on the purpose of the experiment.

1.2.1. ImPACT

The ImPACT (ImPACT Applications, Pittsburgh, PA) computer-based assessment was used to report general demographic information, mTBI symptoms, and to provide a standard clinical measure of cognition using a variety of neurocognitive tests. The ImPACT, which is commonly employed in mTBI research (Lovell et al., 2007; Pellman, Lovell, Viano, & Casson, 2006), indexes neurocognitive function through six modules assessing verbal memory,

visual memory, visual motor speed, reaction time, and impulse control. Administration of the ImPACT lasted 20–25 min and was conducted in a quiet room free from distractions.

1.2.2. Cognitive control task

A modified flanker task (Hillman et al., 2006; Pontifex & Hillman, 2007) was used in which participants' were asked to respond as quickly and accurately as possible to the direction of a centrally presented arrow amid either congruous (e.g. <<<<<< or >>>>>>) or incongruous (e.g. <<<<<< or >>>>>>) flanking arrows. The incongruent, relative to the congruent, condition necessitates the concurrent activation of both the correct response (elicited by the target) and the incorrect response (elicited by the flanking stimuli) before stimulus evaluation is complete, and thus requires greater amounts of interference control to inhibit the flanking stimuli and execute the correct response (Spencer & Coles, 1999). Two blocks of 200 trials, randomized across congruency conditions, were presented with equiprobable congruency and directionality. The stimuli were 7.62 cm tall white arrows, which were presented focally for 80 ms on a black background with a response window of 1000 ms and a variable inter-stimulus interval of either 1100, 1300, or 1500 ms.

1.3. ERP recording

Electroencephalographic (EEG) activity was recorded from 64 electrode sites (FPz, Fz, FCz, Cz, CPz, Pz, POz, Oz, FP1/2, F7/5/3/1/2/4/6/8, FT7/8, FC3/1/2/4, T7/8, C5/3/1/2/4/6, M1/2, TP7/8, CB1/2, P7/5/3/1/2/4/6/8, PO7/5/3/4/6/8, O1/2) of the International 10–20 system (Jasper, 1958), using a Neuroscan Quik-cap, referenced to averaged mastoids (M1, M2), with AFz serving as the ground electrode, and impedance less than 10 k Ω . Additional electrodes were placed above and below the left orbit and on the outer canthus of each eye to monitor electro-oculographic (EOG) activity with a bipolar recording. Continuous data were digitized at a sampling rate of 500 Hz, amplified 500 times with a DC to 70 Hz filter, and a 60 Hz notch filter using a Neuroscan Synamps 2 amplifier. Continuous data were corrected offline for EOG activity using a spatial filter (Compumedics Neuroscan, 2003). Epochs were created from –600 to 1000 ms around the response and baseline corrected using the 100 ms pre-response period. Data were filtered using a zero phase shift 12 Hz (24 dB/octave) low-pass filter. Trials with an error of omission or artifact exceeding $\pm 75 \mu\text{V}$ were rejected. Average ERP waveforms were created for error of commission trials and correct trials, which were matched based on response time and the number of error trials (Coles, Scheffers, & Holroyd, 2001) in order to account for potential artifacts that may exist due to differences in response latency between correct and incorrect trials (Falkenstein, Hoormann, & Hohnsbein, 2001; Mathewson et al., 2005). Correct trials were individually matched, without replacement, to an error trial with the closest possible RT latency. ERN was quantified as the largest negative-going peak occurring within 0–200 ms period post-response (Gehring et al., 1993; Themanson, Hillman, & Curtin, 2006) and the P_e was quantified as the largest positive-going peak occurring within 200–500 ms period post-response (Falkenstein et al., 2000) in each of the two average waveforms (error and matched-correct). Amplitude was measured as the difference between the mean pre-response baseline and maximum peak amplitude.

1.4. Statistical analysis

Differences in group performance on the ImPACT were evaluated using a multivariate analysis of variance (ANOVA) with verbal memory, visual memory, visual motor speed, reaction time, and impulse control collectively used to represent cognitive function-

ing (Broglio et al., 2006). A separate one-way ANOVA evaluated for differences in symptom reports at the time of testing.

Preliminary analysis for ERN and P_e amplitudes were conducted using a 2 (accuracy: error, correct) \times 4 (site: Fz, FCz, Cz, Pz) multivariate repeated measures ANOVA to verify that these data conformed to the expected topography and accuracy effects. ERN amplitude was assessed using a 2 (group: mTBI, control) \times 2 (accuracy: error, correct) multivariate repeated measures ANOVA at the FCz electrode site due to evidence that localizes the ERN at or near the dorsal ACC (Carter et al., 1998; Dehaene et al., 1994; Miltner et al., 2003). P_e amplitude was similarly assessed using a 2 (group: mTBI, control) \times 2 (accuracy: error, correct) multivariate repeated measures ANOVA at the Pz electrode site because this component typically has its topographic maximum amplitude at this site.

Task performance (RT, response accuracy) was assessed separately using a 2 (group: mTBI, control) \times 2 (congruency: congruent, incongruent) multivariate repeated measures ANOVA and t -tests of group (mTBI, control) for flanker interference effects (incongruent–congruent). Post-error task performance was assessed using multiple probability corrected t -tests. A family-wise alpha level of $p = 0.05$ was adopted, and analyses with three or more within-subjects levels used the Wilks' Lambda statistic. Post hoc univariate ANOVAs with Bonferroni corrected t -tests were used to decompose significant effects when appropriate. Lastly, bivariate Pearson product moment correlations were calculated between the dependent variables and the number of mTBIs endured (zero concussions: $n = 36$; one concussion: $n = 17$; two concussions: $n = 9$; three concussions: $n = 1$; four concussions: $n = 1$; five concussions: $n = 2$).

2. Results

Preliminary analyses were performed to test whether Sex was related to any behavioral or neuroelectric variables. Findings revealed no significant main effects or interactions, F 's (1, 62) ≤ 2.1 , $p \geq 0.15$, $\eta^2 \leq 0.03$. Given that Sex effects were not observed, all further analyses were collapsed across Sex.

2.1. ImPACT

Analyses of the ImPACT scores revealed no significant group differences in cognitive performance, $F(5, 60) = 0.8$, $p = 0.54$, $\eta^2 = 0.06$. Similarly, there was no difference between the concussion history and non-concussed groups for the number of symptoms reported at the time of testing, $F(1, 64) = 1.6$, $p = 0.22$, $\eta^2 = 0.23$.

2.2. Task performance

2.2.1. Reaction time

Analyses revealed a significant Congruency effect with longer RT latency for incongruent (464.7 ± 5.8 ms) relative to congruent (398.1 ± 5.8 ms) trials, $F(1, 64) = 466.0$, $p < 0.001$, $\eta^2 = 0.89$, and a marginal interaction of Group \times Congruency, $F(1, 64) = 3.8$, $p = 0.056$, $\eta^2 = 0.06$. Decomposition of the Group \times Congruency interaction revealed no significant differences between groups for congruent, $t(64) = 0.6$, $p = 0.56$, or incongruent, $t(64) = 1.6$, $p = 0.11$, trials. However, analysis of the interference effect (incongruent RT–congruent RT) revealed that the mTBI group (72.7 ± 19.2 ms) exhibited increased flanker interference relative to the control group (60.7 ± 28.9 ms), $t(64) = 2.0$, $p < 0.05$ (see Fig. 1).

2.2.2. Response accuracy

The omnibus analyses revealed a main effect of Group, $F(1, 64) = 7.3$, $p = 0.009$, $\eta^2 = 0.10$, with decreased response accuracy

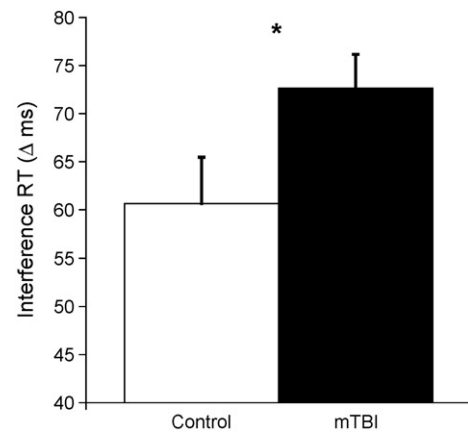


Fig. 1. Mean (+1 SE) interference score (incongruent RT–congruent RT) based on mTBI history.

observed for the mTBI group ($84.4 \pm 1.3\%$) relative to the control group ($89.0 \pm 1.1\%$) (see Fig. 2).

2.2.3. Post-response RT

The omnibus analyses revealed a main effect of Accuracy, $F(1, 64) = 29.7$, $p < 0.001$, $\eta^2 = 0.32$, with longer RT following error trials (443.0 ± 0.1 ms) relative to following match-correct trials (409.0 ± 0.1 ms). No significant group differences were observed, t 's (64) ≤ 0.3 , $p \geq 0.75$.

2.2.4. Post-response accuracy

The omnibus analyses revealed a main effect of Accuracy, $F(1, 64) = 3.7$, $p = 0.05$, $\eta^2 = 0.06$, with increased response accuracy following error trials ($87.6 \pm 1.6\%$) relative to following match-correct trials ($83.7 \pm 1.5\%$). The analyses also revealed a main effect of Group, $F(1, 64) = 8.8$, $p = 0.004$, $\eta^2 = 0.12$, with decreased post-response accuracy for the mTBI group ($82.1 \pm 1.8\%$) relative to the control group ($89.2 \pm 1.6\%$).

2.3. Neuroelectric measures

Preliminary analyses were conducted on the number of included error/correct trials to ensure that between-groups differences in ERP components were not the result of different number of trials included in the ERP averages. Analyses revealed no significant effects for number of trials, $t(64) = 0.2$, $p = 0.81$.

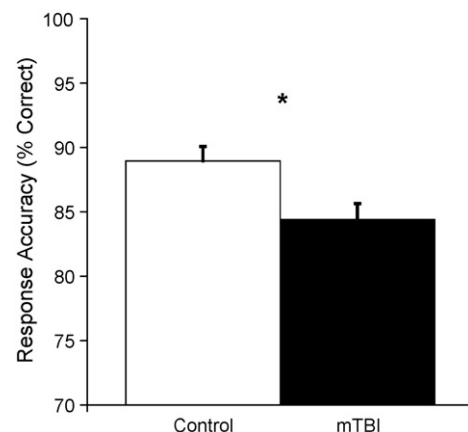


Fig. 2. Mean (+1 SE) response accuracy based on mTBI history.

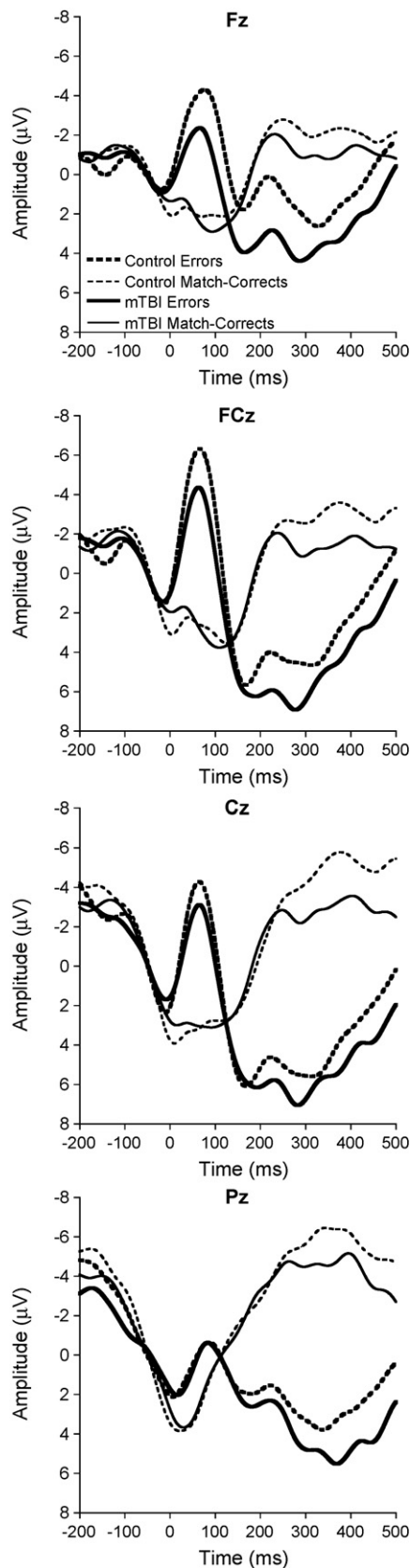


Fig. 3. Grand average ERP waveforms based on mTBI history for error and match-correct conditions.

2.3.1. ERN amplitude

The omnibus analyses revealed significant effects for Accuracy, $F(1, 65) = 27.2$, $p < 0.001$, $\eta^2 = 0.30$, and Site, $F(3, 63) = 15.6$, $p < 0.001$, $\eta^2 = 0.43$, which were superseded by an interaction of Accuracy \times Site, $F(3, 63) = 20.7$, $p < 0.001$, $\eta^2 = 0.50$. Decomposition of the Accuracy \times Site interaction revealed the expected significant and largest Accuracy effect at FCz, with larger ERN amplitude for error ($-7.1 \pm 0.6 \mu\text{V}$) relative to match-correct ($-2.2 \pm 0.5 \mu\text{V}$) trials, $t(65) = 7.1$, $p < 0.001$. Thus, subsequent ERN analyses used amplitude scores from the FCz electrode site (Falkenstein et al., 2001).

Fig. 3 illustrates the grand average ERP waveform for each group. The omnibus Group \times Accuracy analyses revealed a significant effect for Accuracy, $F(1, 64) = 50.7$, $p < 0.001$, $\eta^2 = 0.44$, which was superseded by an interaction of Group \times Accuracy, $F(1, 64) = 5.4$, $p = 0.02$, $\eta^2 = .08$. Decomposition of this interaction indicated no differences between groups for correct trials, $t(64) = 0.01$, $p = 0.99$. However, ERN amplitude on error trials was significantly smaller for the mTBI group ($-5.4 \pm 0.6 \mu\text{V}$) relative to the control group ($-8.5 \pm 1.0 \mu\text{V}$), $t(64) = 2.7$, $p = 0.01$.

2.3.2. P_e amplitude

The omnibus analyses revealed significant effects for Accuracy, $F(1, 65) = 51.1$, $p < 0.001$, $\eta^2 = 0.44$, and Site, $F(3, 63) = 19.8$, $p < 0.001$, $\eta^2 = 0.49$, which were superseded by an Accuracy \times Site interaction, $F(3, 63) = 6.4$, $p = 0.001$, $\eta^2 = 0.23$. Decomposition of this interaction revealed the expected significant and largest Accuracy effect at Pz, with larger P_e amplitude for error ($7.6 \pm 0.7 \mu\text{V}$) relative to match-correct ($2.3 \pm 0.5 \mu\text{V}$) trials, $t(65) = 8.0$, $p < 0.001$. Thus, subsequent P_e analyses used amplitude scores from the Pz electrode site (Falkenstein et al., 2000; Themanson et al., 2006). The omnibus Group \times Accuracy analyses revealed a significant effect for Accuracy, $F(1, 64) = 67.6$, $p < 0.001$, $\eta^2 = 0.51$, with larger P_e amplitude on error trials relative to match-correct trials. No significant group effects were observed (see Fig. 3).

2.4. Correlations

Bivariate Pearson product moment correlations calculated between the dependent variables and the number of mTBIs endured revealed that the number of mTBI incidents was negatively correlated with ERN amplitude, $r = -0.325$, $p = 0.004$, such that an increase in the number of mTBI incidents was related to a decrease in ERN amplitude. No other measures were significantly correlated with the number of mTBI incidents.

3. Discussion

The current findings indicated decreased response accuracy and increased interference RT for the mTBI group during performance on a flanker task. Similar group differences were not observed on the ImpACT neurocognitive assessment, indicating that specific measures of cognitive control may be more sensitive to long-term neurocognitive impairments than standard clinical assessments. Neuroelectric indices of cognitive control revealed smaller ERN amplitude for the mTBI group relative to the control group, and the nature of this relationship was further indicated via an association between a linear reduction in ERN amplitude with an increased number of concussive incidents. These findings suggest that mild traumatic brain injuries may result in cumulative long-term impairments to the evaluative subsystem of cognitive control.

The current findings corroborate previous investigations suggesting a lack of long-term cognitive impairments following mTBI using the computer-based ImpACT neurocognitive assessment (Broglia et al., 2006; Collie et al., 2006; Iverson et al., 2006). Thus,

it appears that the ImPACT does not have the requisite sensitivity to detect the subtle deficits indicative of long-term cognitive impairment following mTBI. This finding is not surprising however, as these tests were designed to detect dramatic neurocognitive deficits observed immediately following mTBI, rather than the subtle chronic changes that remain. Thus, there is a need to identify additional measures to capture these long-term indices of cognitive dysfunction.

Indeed, other behavioral measures appear to be more sensitive to long-term cognitive dysfunction relating to mTBI history. Specifically, athletes with a history of previous concussive injury exhibited decreased response accuracy to the flanker task, replicating previous investigations observing mTBI-related deficits on tasks requiring extensive amounts of cognitive control (Collins et al., 1999; Ellemberg et al., 2007). Further, larger interference RT during the flanker task was observed for athletes with a history of concussive injury, indicating greater attentional cost associated with increased task demands. Taken together, these findings demonstrate the utility of cognitive control tasks in detecting long-term mTBI-related cognitive deficits and further suggest that previous concussive injury relates to overall failures in cognitive control (as demonstrated by poorer response accuracy) as well as an inability to upregulate cognitive control to meet the increased attentional demands necessitated by greater task difficulty (as demonstrated by larger interference RT). Collectively, these findings suggest that tasks which require aspects of cognitive control may be more sensitive to long-term cognitive dysfunction related to concussive injury than the computer-based ImPACT neurocognitive assessment.

The present findings are also consonant with previous investigations that observed deficits in the neuroelectric system, which were associated with previous mTBI history. Specifically, prior neuroelectric investigations have observed decreased P3b amplitude (Broglio et al., 2009; Dupuis et al., 2000) and increased P3b latency (Gaetz et al., 2000; Gosselin et al., 2006) for athletes with a history of mTBI relative to athletes without a history of mTBI. These findings in association with the findings reported herein provide further support for the view that the investigation of the neuroelectric system may provide the requisite sensitivity to detect subtle signs of chronic cognitive dysfunction resulting from mTBI. The present findings of reduced ERN amplitude for athletes with a history of mTBI also suggests that these chronic cognitive deficits may result from decrements in the evaluation and signaling for modulations in top-down control (Botvinick et al., 2001). Interestingly however, the lack of any group differences in post-error reaction time or accuracy suggests that previous mTBI may not influence the regulation of top-down control.

Although the underlying mechanisms have not been fully elucidated at this time, mTBI appears within this context to be more than an acute injury with potentially serious implications for long-term cognitive health. That is, the negative association between ERN amplitude and the number of previous concussive injuries suggests that concussive incidents may result in cumulative impairments to the evaluative subsystem of cognitive control. Although speculative, the decrements in both task performance and neuroelectric measures may relate to cellular death within crucial cortical structures. Specifically, animal models have demonstrated cellular death within the hippocampal structures following mTBI (Maxwell et al., 2003; Tashlykov et al., 2007). These findings coupled with decreased gray matter volume in previously concussed athletes observed within the DLPFC and ACC (Chen et al., 2003, 2008) provide support for the view that mTBI may result in the collective cellular death within these cortical structures, and may underlie the observed neuropathological manifestations (Omalu et al., 2005, 2006). Future research should continue to assess the relation of mTBI history on cognitive control to fully understand the breadth of long-term cognitive dysfunction.

Several limitations in the current study should be noted. In spite of the observed cognitive differences between previously concussed and control athletes, the use of participant-reported concussion diagnoses limits the strength of this investigation. Further, it should be noted that given the cross-sectional nature of this investigation, it is possible that individual differences or some other factor may account for the observed reductions in ERN amplitude and the likelihood of sustaining an mTBI. Future research utilizing longitudinal designs are necessary to fully elucidate these effects.

In summary, mTBI history was associated with deficits in task performance and neuroelectric measures of cognitive control, which may underlie an increased relative risk of recurrent traumatic brain injury. These findings demonstrate that concussive history is not only related to long-term decrements in cognition, but that these cognitive deficits related to mTBI appear to be cumulative. In spite of these changes, it is unclear how these deficits may impact the everyday life of these individuals. Each of the participants enrolled in this investigation were high functioning college students with no outwardly noticeable deficits. Being the first investigation to show deficits of this nature, further research is needed to elucidate the relationship between mTBI, the underlying anatomical changes, and assorted aspects of neurophysiology. Collectively, these findings support and extend a growing body of literature suggesting that cerebral concussion can no longer be considered a transient injury without long-term threats to cognitive health.

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Conflict of interest statement

No conflicting financial interests exist.

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