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The Relation of Mild Traumatic Brain Injury to Chronic Lapses of Attention

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We assessed the extent to which failures in sustained attention were associated with chronic mild traumatic brain injury (mTBI) deficits in cognitive control among college-age young adults with and without a history of sport-related concussion. Participants completed the ImPACT computer-based assessment and a modified flanker task. Results indicated that a history of mTBI, relative to healthy controls, was associated with inferior overall flanker task performance with a greater number of omission errors and more frequent sequentially occurring omission errors. Accordingly, these findings suggest that failures in the ability to maintain attentional vigilance may, in part, underlie mTBI-related cognition deficits.

Key words: cognitive control, concussion, sustained attention, vigilance

Of the approximately 3 million mild traumatic brain injuries (mTBI or concussions—used synonymously within the present manuscript) that occur annually in the United States (Langlois, Rutland-Brown, & Wald, 2006), attentional dysfunctions are among the most pervasive and persistent symptoms individuals experience following a concussive incident (Leclercq & Azouvi, 2002). These attentional dysfunctions typically manifest as greater difficulty concentrating and maintaining attention, and increased off-task behavior in the weeks, and even years, postinjury (Leclercq & Azouvi, 2002; Oddy, Coughlan, Tyerman, & Jenkins, 1985). However, despite the prevalence of these symptoms, rapid neuropsychological screening tools (such as the ImPACT computer-based assessment and others) have been found ineffective in detecting these attentional dysfunctions following the acute injury. The inability to maintain attentional focus over time indicates failures in sustained attention (Parasuraman, Warm, & See, 1998; Warm, Parasuraman, & Matthews, 2008). Sustained attention to task relevant details, also known as vigilance, requires substantial attentional resources such that over a prolonged time period, depletion of attentional stores manifests as vigilance decrements (i.e., a lack of a response to a required action, resulting in an omission error; Temple et al., 2000; Warm et al., 2008). Thus, these sustained attention failures allow for neural resource conservation until attention can be re-engaged (Fisk & Schneider, 1981). While a growing body of research has noted persistent neurocognitive deficits occurring well beyond the initial recovery phase from mTBI (Chen, Johnston, Petrides, & Ptito, 2008; Chen, Kareken, Fastenau, Trexler, & Hutchins, 2003; Collins et al., 1999; Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000; Ellemberg, Leclerc, Couture, & Daigle, 2007; Gaetz, Goodman, & Weinberg, 2000; Gosselin, Theriault, Leclerc, Montplaisir, & Lassonde, 2006; Guskiewicz et al., 2005; Pontifex, Hillman, Fernhall, Thompson, & Valentini, 2009), those investigations predominately focused on the ability to execute a correct response and largely ignored

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the extent to which concussion-related cognition deficits may relate to failures in sustained attention.

Cicerone (1997) provided initial evidence that examining sustained attention may provide additional insight into mTBI-related cognition deficits. Specifically, this small body of research revealed increased omission errors relative to mTBI-related deficits in participants performing a continuous task, compared to healthy control participants, with larger deficits accompanying increases in task complexity (Cicerone, 1997). These findings are consistent with previous investigations assessing sustained attention in healthy populations, suggesting a negative association between attentional vigilance and the extent that cognitive control is required (Caggiano & Parasuraman, 2004; Fisk & Schneider, 1981; Parasuraman, 1979).

Cognitive control (also referred to as executive control) describes an overarching set of higher order cognitive operations, which are involved in regulating goal-directed interactions (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Meyer & Keiras, 1997; Norman & Shallice, 1986). These processes allow for behavior optimization through selecting, scheduling, coordinating, and maintaining computational processes that underlie perception, memory, and action (Botvinick et al., 2001; Meyer & Keiras, 1997; Miyake, Friedman, Emerson, Witzki, & Howerter, 2000; Norman & Shallice, 1986). The core processes of cognitive control include inhibition, working memory, and cognitive flexibility (Diamond, 2006). Inhibition is particularly important to effective cognitive control functioning (Barkley, 1997; Brocki & Brohlin, 2004), given its relation to the ability to override a prepotent response in order to perform a less potent but correct response, block out task irrelevant information in the stimulus environment, and stop an ongoing response (Barkley, 1997; Davidson, Amso, Anderson, & Diamond, 2006). These abilities are thought to be vital in sustaining attention and maintaining control over one's actions (Diamond, 2006).

A number of investigations found cognitive control tasks are particularly sensitive to mTBI-related cognition deficits (Collins et al., 1999; Ellemberg et al., 2007; Pontifex et al., 2009). That is, across a number of tasks requiring inhibitory control modulation (Trails Making Test–B, Stroop, Flanker), a history of previous concussive incidents has been associated with cognitive control decrements even several years after injury (Collins et al., 1999; Ellemberg et al., 2007; Pontifex et al., 2009). To date, however, no investigation has examined the relationship between mTBI history and sustained attention (i.e., errors of omission) in response to tasks requiring inhibitory aspects of cognitive control.

Accordingly, the purpose of our investigation was to determine how sustained attention deficits may relate to mTBI-related deficits in cognitive control. Based on previous findings, we hypothesized that individuals with a concussion history would demonstrate cognitive control deficits with a greater number of omission errors relative to healthy controls. Further, given that failures in sustained attention are believed to allow for neural resource regeneration until attention can be re-engaged (Fisk & Schneider, 1981), the second purpose of this investigation was to examine whether mTBI history was associated with longer delays in attention re-engagement as indexed by greater frequency of error runs (i.e., two or more sequential omission errors) and the mean distance of error runs (i.e., number of sequential errors). We hypothesized that mTBI history would relate to more frequent and longer lapses in sustained attention.

Method

Participants

A total of 80 (23 women) college-age athletes were recruited from recreational ice hockey, rugby, martial arts, and soccer teams as well as the women's varsity soccer team at the University of Illinois. Participants were assigned to either control (those who reported never having an mTBI; $n = 42$; 17 women) or mTBI (those reporting a history of mTBI but symptom free at the time of testing; $n = 38$; 6 women) groups based on physician-diagnosed concussion history. We defined symptom free as not currently experiencing symptoms related to concussive injury. Participants from the control and mTBI groups reported some symptoms during the evaluation, which were attributed to factors unrelated to concussion (Piland, Ferrara, Macciocchi, Broglio, & Gouteyron, 2010). One individual in the control group and 2 in the mTBI group reported hyperactivity. No other secondary factors to cognitive performance were reported. The Institutional Review Board of the University of Illinois at Urbana-Champaign approved this study. All participants provided written informed consent and reported being free of neurological disorders, cardiovascular disease, any medications that influence central nervous system function, had a normal IQ (defined as ≥ 85) as measured by the Kaufman Brief Intelligence Test (Kaufman & Kaufman, 1990), and had (corrected to) normal vision. Table 1 summarizes the demographic and mTBI data for all participants.

Cognitive Assessments

ImPACT. The ImPACT (version 6.7; ImPACT Applications, Pittsburgh, PA) computer-based assessment was used to report general demographic information and mTBI symptoms and to provide a clinical measure of cognition through modules assessing verbal memory, visual memory, visual motor speed, reaction time, and impulse control. Symptom scores are based on the participant's

ranking of 22 symptoms using a Likert-type scale, with 0 = not experiencing the symptom and 6 = the most severe. The ImPACT has been used in a number of settings for mTBI research (Lovell et al., 2007; Pellman, Lovell, Viano, & Casson, 2006) and has been shown to provide similar sensitivity to postconcussion cognitive declines with pen and paper tests (Broglio, Macciocchi, & Ferrara, 2007). Administration of the ImPACT lasted 20–25 min and was conducted in a quiet room free from distractions.

Cognitive Control Task. A modified flanker task (Eriksen & Eriksen, 1974; Pontifex & Hillman, 2007; Pontifex et al., 2009) was used to assess the inhibitory aspects of cognitive control. This paradigm is conceptually simplistic in that it requires the discrimination of a centrally presented target stimulus amid lateral flanking stimuli. In this task, participants were required to make a left hand thumb press on a Neuroscan STIM system switch response pad (Compumedics, Charlotte, NC) when the target stimulus pointed left and a right hand thumb press when the target stimulus point right. Perceptually induced response interference can be evoked by manipulating the compatibility of the target and flanking stimuli. In the congruent array (e.g., <<<<< or >>>>>), the target stimulus and the flanking stimuli are identical, resulting in faster and more accurate responses relative to the incongruent array (e.g., >><>> or <<><<), in which the target and flanking stimuli are mapped to opposing action-schemas (Eriksen & Schultz, 1979). The incongruent array results in the concurrent activation of the correct (elicited by the target) and incorrect responses (elicited by the flanking stimuli) before stimulus evaluation is complete, thus, requiring greater amounts of interference control to inhibit the flanking stimuli and execute the correct response (Spencer & Coles, 1999). Following completing 40 practice trials, participants performed two blocks of 200 trials, presented with equiprobable congruency and directionality on a computer monitor at a distance of 1 m using Neuroscan STIM2 software. The stimuli were 3-cm tall white arrows, which appeared for 80 ms on a black background with a response window of 1,000 ms and a variable intertrial interval of 1,100, 1,300, or 1,500 ms.

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

Measure	Control		mTBI	
	M	*SD*	*M*	*SD*
N	42 (17 women)		38 (6 women)	
Age (years)	19.4	1.3	20.0	1.3
K-BIT (IQ)	107.5	8.1	106.8	6.0
BMI (kg/m^2)	26.3	3.5	26.6	4.4
Education (years)	13.6	1.3	14.3	1.2
Number of concussions (range: 1–5)	—		1.5	0.7
Time since last concussion (years)	—		3.6	3.2
Symptom score	8.4	9.2	6.4	9.0
ImPACT verbal memory (% correct)	90.2	8.3	90.0	8.6
ImPACT visual memory (% correct)	83.0	9.4	81.4	8.9
ImPACT motor speed (composite score)	44.0	7.8	43.0	6.4
ImPACT reaction time (ms)	529.8	7.6	524.5	4.3
ImPACT impulse control (composite score)	9.6	19.9	13.5	23.1

Note. mTBI = mild traumatic brain injury; *M* = mean; *SD* = standard deviation; K-BIT is a composite score for IQ; BMI = body mass index; there were no significant differences between groups for any demographic variable; the number of symptoms and symptom score were obtained as a part of the ImPACT computer-based assessment; the symptom score was based on a 0–6 severity ranking of 22 possible symptoms associated with mTBI.

Statistical Analysis

Group differences in cognitive functioning were evaluated across verbal memory, visual memory, visual motor speed, reaction time, and impulse control components using a multivariate analysis of variance (MANOVA; Broglio, Ferrara, Piland, & Anderson, 2006; Pontifex et al., 2009). Symptom reports at the time of testing were assessed using a one-way analysis of variance. Group differences in flanker task performance (RT, response accuracy) were assessed using a two-factor (congruent, incongruent) repeated measures MANOVA. Group (mTBI, control) differences in the distribution of commission errors (incorrect responses) and omission errors (nonresponses), the number of error runs (two or more sequential errors) for commission and omission errors, and the mean distance (number of sequential errors) for commission and omission errors were assessed separately using multiple probability corrected Mann-Whitney tests with a family-wise alpha level of $p = .05$. We chose the Mann-Whitney tests to account for the nongaussian distribution of the error data (Motulsky, 1995). Estimates of effect size, partial eta-square (η^2), are provided for all main effects and interactions. Last, bivariate Spearman's rank correlation coefficients were calculated between the dependent variables and the number of mTBIs (zero concussions: $n = 42$; one concussion: $n = 22$; two concussions: $n = 13$; three concussions: $n = 1$; four concussions: $n = 1$; five concussions: $n = 1$).

Results

ImPACT

Analysis of the ImPACT scores revealed no significant group differences in cognitive performance, $F(1, 78) <$

0.01, $p = .93$, $\eta^2 < .001$. Similarly, there were no differences between the mTBI and control groups for the number of symptoms reported at the time of testing, $F(1, 78) = 1.0$, $p = .33$, $\eta^2 = .12$.

Flanker Task Performance

Preliminary analyses tested for potential differences between the first and second block of 200 trials on the modified flanker task. Findings revealed no significant main effects or interactions involving trial blocks. Thus, all further analyses were collapsed across blocks.

Reaction Time. Analysis revealed a main effect of congruency, with longer RT latency for incongruent (M = 464.5 ms, SD = 5.7) relative to congruent (M = 395.5 ms, SD = 5.1) trials, $F(1, 78) = 877.3$, $p < .001$, $\eta^2 = .92$. There were no group differences for RT latency, $F(1, 78) = 0.23$, $p = .637$, $\eta^2 = .003$.

Response Accuracy. Analysis revealed main effects of congruency, with decreased response accuracy for incongruent (M = 81.9%, SD = 1.0) relative to congruent (M = 94.0%, SD = 0.7) trials, $F(1, 78) = 248.3$, $p < .001$, $\eta^2 = 0.76$, and group, with decreased response accuracy observed for the mTBI group (M = 85.8%, SD = 1.1) relative to the control group (M = 90.2%, SD = 1.1), $F(1, 78) = 8.1$, $p = .006$, $\eta^2 = .09$ (see Figure 1).

Error Analysis. Decomposition of observed group differences in response accuracy was conducted by examining the distribution of commission errors relative to omission errors. Findings revealed more omission errors occurred for the mTBI (M = 12.3, SD = 2.5) relative to the control (M = 4.6, SD = 0.9) group, Mann-Whitney U = 535.0, Z = 2.6, $p = .01$ (see Figure 1). There were no group differences for commission errors, Mann-Whitney U = 766.5, Z = 0.3, $p = .76$. Further decomposition of the observed group differences in response accuracy was conducted by examining the number of error runs (two or more sequential errors) for omission and commission errors. Analysis revealed more omission error runs (2 or more sequential errors) occurred in the mTBI ($M = 2.2 \pm 0.5$) relative to the control (0.7 ± 0.2) group, Mann-Whitney U = 600, Z = 2.5, $p = .025$, with no group differences for error runs, Mann-Whitney U = 789.5, Z = 0.09, $p = .9$ (see Figure 1). Last, examination of the mean distance (number of sequential errors) revealed no significant group differences for omission errors (mTBI: M = 0.9, SD = 1.3; control: M = 1.2, SD = 1.2) or commission errors (mTBI: M = 1.0, SD = 1.1; control: M = 1.0, SD = 1.0), Mann-Whitney U's ≥ 680.5, $Z \leq 1.3$, $p \geq 0.2$.

Correlations

Bivariate Spearman's rank correlation coefficients calculated between the dependent variables and number of mTBIs endured revealed the number of mTBI incidents was positively correlated with the number of omission errors, Spearman's $\rho = .29$, $p = .009$, as well as the number of omission error runs, Spearman's $\rho = .25$, $p = .027$. No other

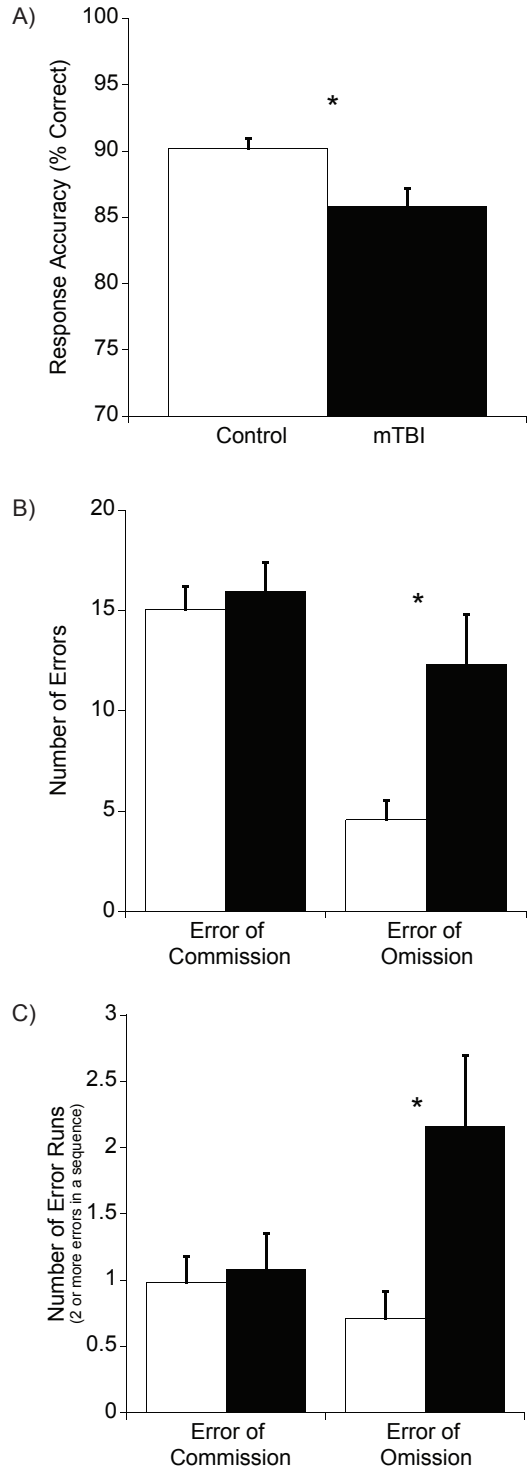

Figure 1. Mean (+1 standard error) response accuracy (A); number of errors by error type (B); and number of error runs (2 or more errors in a sequence) by error type (C); based on mTBI history (White = Control; Black = mTBI)

measures were significantly correlated with the number of mTBI incidents endured.

Discussion

Findings from this investigation indicated that a history of mTBI (3.6 years prior) was related to decreased response accuracy during a cognitive control task, despite the absence of deficits on a clinical cognitive assessment (i.e., ImPACT). Examination of behavioral performance on the modified flanker task revealed that mTBI history was associated with a greater number of omission errors and more frequent sequentially occurring omission errors, relative to healthy controls, with no differences between groups for commission errors. Further, an increased number of concussive incidents was associated with an increased overall number of omission errors and an increase in sequentially occurring omission errors. Accordingly, these findings suggest that inability to maintain attentional vigilance may, in part, underlie mTBI-related cognition deficits.

Although computer-based neurocognitive assessments, such as the ImPACT, have been found sensitive to cognition deficits during the acute phase of concussive injuries, the current findings add to a growing body of literature suggesting that these assessments may lack the requisite sensitivity to detect more subtle deficits associated with persistent cognitive dysfunction (Broglio et al., 2006; Collie, McCrory, & Makdissi, 2006; Iverson, Brooks, Lovell, & Collins, 2006). Rather, the results presented here replicate previous investigations suggesting the use of cognitive control tasks in capturing long-term cognition deficits (Collins et al., 1999; Ellemburg et al., 2007). In particular, tasks designed to tax cognitive control systems appear particularly well suited to detect attentional dysfunctions, which are among the most pervasive chronic mTBI-related symptomologies (Leclercq & Azouvi, 2002), by assessing behavioral indexes of sustained attention. That is, the current findings replicate those of Cicerone (1997), who observed an increased occurrence of omission errors for individuals with histories of mTBI, relative to healthy control populations. Accordingly, behavioral measures of attentional vigilance during cognitive control tasks may provide a low-cost assessment for persistent cognitive dysfunction associated with concussive injuries.

Novel in the current investigation was our examination of how mTBI history was associated with delays in attention re-engagement. That is, failures in sustained attention occur as attentional resources diminish, which is thought to allow for the regeneration of neural resources until attention can be re-engaged (Fisk & Schneider, 1981). Accordingly, a behavioral index of the ability to regenerate neural resources can be obtained by assessing the frequency of omission error runs and the mean distance of omission error runs. Our findings suggest that while a history of mTBI was not associated with a longer delay in attention re-engagement, concussion history was associated with an increased frequency of omission error runs with no group differences for the frequency or distance of commission error runs. Accordingly, these findings suggest that mTBI history may relate to an increased occurrence of sustained attention failures in response to a cognitive control task with more frequent runs of vigilance decrements. Further, the relationship between the number of concussive incidents and sustained attention failures suggest that mTBI incidence may result in cumulative attention impairments.

Although speculative, the mechanisms underlying these mTBI-related deficits in sustained attention may relate to impairments in brain structure or function. For instance, a growing body of research has noted neuroelectric deficits in the allocation of attentional resources (Broglio, Pontifex, O'Connor, & Hillman, 2009; Dupuis et al., 2000; Pontifex et al., 2009), delays in stimulus classification and processing speed (Gaetz et al., 2000; Gosselin et al., 2006), and deficits in evaluating and signaling for modulations in top-down control during action monitoring processes (Pontifex et al., 2009) in individuals with a history of mTBI. Thus, these deficient processes may result in inefficient neural resource allocation, which may contribute to increased failures in sustained attention. Further, neuroimaging investigations have suggested that concussion-related neuropathologies may stem from cellular death within hippocampal structures and decreased gray matter volume within both the dorsolateral prefrontal cortex and the anterior cingulate cortex, two structures critical for regulating attention and cognitive control (Chen et al., 2003, 2008; Maxwell et al., 2003; Tashlykov et al., 2007). Others have speculated that changes in dopamine production and metabolism may play a role in long-term effects (Bales, Wagner, Kline, & Dixon, 2009). It is important to note, however, that individual differences or some other factor may account for the sustained attention failures, given the cross-sectional nature of this investigation.

While our results align with previous investigations, this study is not without limitations that warrant prudence when interpreting results. We relied on our participants' self-report of previous concussion instances. While similar methodologies have been reported (Broglio et al., 2006; Broglio et al., 2009; Collie et al., 2006; Gaetz et al., 2000), it is possible some athletes may not be aware they had previously sustained a concussion (McCrea, Hammeke, Olsen, Leo, & Guskiewicz, 2004). Thus, it is feasible that previously concussed athletes may have been included in the control group. However, this would strengthen our findings, as the performance of such athletes would suppress overall control group performance toward that of the previously concussed group. Similarly, given that athletes were screened for previous concussions, it is unlikely that

nonconcussed athletes were included in the concussed group. However, despite the inherent limitations of this retrospective design, the findings provide evidence to warrant prospective longitudinal designs with physician-confirmed concussion diagnosis to better examine these findings. It is also feasible that some of the concussed participants sustained injuries outside the context of sport. Regardless, the clinical presentation of nonsport mTBI would be no different from those resulting from sport participation. More severe brain injuries resulting from auto or other accidents with a Glasgow Coma Scale rating of < 13, or positive imaging, would be classified as moderate or severe TBI, not mTBI or concussion.

Collectively, these findings add to a growing body of research demonstrating concussion-related deficits, which persist beyond the acute stage of injury (Chen et al., 2003, 2008; Collins et al., 1999; Dupuis et al., 2000; Ellemberg et al., 2007; Gaetz et al., 2000; Gosselin et al., 2006; Guskiewicz et al., 2005; Pontifex et al., 2009). In particular, mTBI history was associated with cumulative impairments in the ability to sustain attention, mirroring the most reported mTBI-related symptom of persistent difficulty concentrating and maintaining attention (Leclercq & Azouvi, 2002; Oddy et al., 1985). Although additional research is necessary to elucidate the relationship between mTBI, sustained attention, and underlying anatomical and neurophysiologic pathologies, these findings suggest that examining aspects of sustained attention may provide a more sensitive index of chronic cognitive dysfunction than present neurocognitive evaluations.

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