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Imaging "Brain Strain" in Youth Athletes with Mild Traumatic Brain Injury during Dual-Task Performance

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Abstract

Mild traumatic brain injury (mTBI) is a common cause of injury in youth athletes. Much of what is known about the sequelae of mTBI is yielded from the adult literature, and it appears that it is mainly those with persistent post-injury symptoms who have ongoing cognitive and neural abnormalities. However, most studies have employed single-task paradigms, which may not be challenging enough to uncover subtle deficits. We sought to examine the neural correlates of dual-task performance in male athletes aged 9-15 years using a functional neuroimaging protocol. Participants included 13 youths with a history of mTBI three to six months prior to testing and 14 typically-developing controls. All participants completed a working memory task in isolation (single-task) and while completing a concurrent motor task (dual-task); neural activity during performance was then compared between groups. Although working memory performance was similar during the single-task condition, increased working memory load resulted in an altered pattern of neural activation in key working memory areas (i.e., dorsolateral prefrontal and parietal cortices) in youth with mTBI relative to controls. During the dual-task condition, accuracy was similar between groups but injured youth performed slower than typically-developing controls, suggesting a speed-accuracy tradeoff in the mTBI group only. The injured youths also exhibited abnormal recruitment of brain structures involved in both working memory and dual-tasking. These data show that the dual-task paradigm can uncover functional impairments in youth with mTBI who are not highly symptomatic and who do not exhibit neuropsychological dysfunction. Moreover, neural recruitment abnormalities were noted in both task conditions, which we argue suggests mTBI-related disruptions in achieving efficient cognitive control and allocation of processing resources.

Key words: dual-task; fMRI; mild TBI; working memory

Introduction

Mild traumatic brain injury (mTBI) causes pathophysiological changes in the absence of perceptible structural brain damage, resulting in rapid neurological disruption and a graded set of symptoms, including headache, nausea, apathy/low mood, and cognitive disturbances.[1,2] These acute symptoms coincide with abnormalities visualized via functional magnetic resonance imaging (fMRI) in resting state networks[3,4] and in circuitry subserving a variety of cognitive processes.[5–9] Symptom resolution occurs spontaneously in 85-90% of affected individuals within a month of injury,[10,11] which coincides with a ''normalization'' of both task performance and neural functioning.[12–16] Yet in those with persistent and/or severe symptoms, neurocognitive deficits—including poor working memory performance and abnormal brain activation—remain evident.[12,13,17,18] These individuals also tend to exhibit additional activations not typically seen in non-injured controls,[18] suggesting changes in the recruitment of neural processing resources.

mTBI incurred during participation in sport is a common cause of injury in children and adolescents.[19–22] Much insight into sequelae of mTBI has been elucidated from adult participants[23] despite the fact that pediatric brain injuries affect the ongoing maturation of neural circuitry and consequently, subsequent cognitive, emotional, and behavioral development.[24-26] A child's brain is especially vulnerable to insult due to a variety of physiological and biomechanical sensitivities not present in adults[27,28] yet only a handful of fMRI studies have been conducted with youth with

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mTBI. Symptomatic athletes ages 11 to 17 were shown to have reduced total cerebral blood flow within 72 h of injury that persisted for one month despite total symptom resolution.[29] Conversely, no neural activation abnormalities were observed in youth with mTBI within a month of injury while performing a working memory task[30] but the behavioral data were not reported, making it impossible to establish whether performance was dissimilar between groups. Utilizing a similar task as Krivitzky and colleagues,[30] fMRI abnormalities in male high school football athletes were discovered acutely and at the post-season assessment but there was no evidence of working memory impairment at either time point.[31] The latter data are similar to what has been shown in some studies with adults[9,12,17,32–38] and suggest that mTBI in youth can result in persistent physiological disruption in the absence of cognitive, structural, or metabolic disturbances.

Conversely, it may be that neuropsychological tests, which typically target isolated areas of functioning, are not sensitive enough to detect long-term impairments following mTBI,[39,40] and this may hold for both standardized and experimental tasks of cognition. Indeed, in the ''real-world,'' rarely are cognitive demands presented in isolation. Rather, individuals are often required to integrate processing demands in order to carry out daily activities, such as driving a car. Assessing abilities concurrently via a dual-task paradigm is a means to explore the integration of multiple cognitive processes where two tasks are presented simultaneously.[41-43] Any performance changes to either individual task as a consequence of the dual-task manipulation is termed the ''dual-task cost'' and is thought to reflect underlying limitations in working memory and/or attention.[44] In healthy adults, dual-tasking recruits additional neural activity in the dorsolateral prefrontal cortex (DLPFC),[45–49] anterior cingulate cortex,[50] left inferior frontal sulcus,[51] precuneus,[52,53] and cerebellum.[54]

In adults with mild to severe TBI, decrements in both locomotor and cognitive performance have been noted in dual-task conditions.[55–60] While much research has focused predominantly on college-aged athletes, increased dual-task cost has been shown in youth with mTBI relative to non-injured controls despite intact single-task performance.[61,62] Thus, these data suggest that the challenge of a dual-task may be able to uncover cognitive-behavioral difficulties in the absence of abnormal neuropsychological test findings and this may provide a more accurate index of functional ability following mTBI.[63,64]

To our knowledge, only one study has described the neural substrates of dual-task performance following TBI: Adults in the chronic phase of a severe TBI displayed increased activation during a dual-task condition, which the authors concluded may reflect effortful processing and neural network compensation.[65] The objective of the current study was to explore dual-task performance and fMRI correlates in youth athletes with a history of mTBI. Working memory performance was examined in both single- and dual-task conditions using an n-back paradigm so that task difficulty (load) could be increased incrementally to tax processes related to learning, language comprehension, inhibitory control, reasoning, and other higher-order cognitive functions.[66-68]

If mTBI is truly a transient ''minor'' neurological disturbance, then the injured children should perform similarly to non-injured controls with no observable group differences in brain activation. Conversely, if mTBI causes persistent neural dysfunction, then abnormalities should be measurable in behavioral performance, brain imaging, or both, with any group differences in fMRI signaling potentially reflecting differences in the recruitment of processing resources, compensatory mechanisms, or changes in cognitive effort/control required to complete the tasks[35,36,69] We hypothesized that youth athletes with mTBI would exhibit similar working memory performance as controls during the single-task condition but would show impairments in accuracy and speed and a greater dual-task cost during the dual-task condition. Group differences in neural activation were expected only during the dual-task condition, specifically in nodes mediating working memory and dual-task performance such as the DLPFC.

Methods

Participants

Study participants were males ages 9-15 years who were actively participating in competitive sports (Table 1). Exclusion criteria for all participants included pre-existing neurological conditions, history of moderate and/or severe TBI, psychiatric disorders, motor disturbances, and any MRI contraindications. The mTBI group included 13 participants, of whom four had incurred two injuries within a year. Participants were not excluded if they had previously sustained an mTBI. All mTBI participants had sustained an injury while participating in sport as diagnosed by a medical doctor three to six months prior to study participation. We chose this timeframe to minimize the potential effects of spontaneous neural and symptom recovery on performance.[70] mTBI history was confirmed both at study recruitment by probing for mTBI using criteria outlined by the 3rd International Consensus Conference on Concussion in Sport in Zurich (2008) and by the World Health Organization (2004; also, see McCrory and colleagues[1]), and at study participation through the administration of the ThinkFirst Concussion Questionnaire created by Drs. J. Scott Delaney and Karen M. Johnston on behalf of the ThinkFirst-SportSmart Concussion Education and Awareness Committee. Although the Glasgow Coma Scale (GCS) score was not available, based on parent reports, four of 13 mTBI participants experienced a brief loss of consciousness described as lasting seconds to minutes; thus, we can be reasonably confident that the lowest GCS scores of all children in the mTBI group ranged from 13-15—the range for a mild severity injury. A third questionnaire was given at study participation to probe for ongoing post-mTBI symptoms via a checklist of symptoms where participants rated on a 6-point Likert scale the intensity to which they were exhibiting typical mTBI symptoms (i.e., none to severe) such as headache, nausea, difficulty concentrating, poor memory, and irritability. Two individuals continued to exhibit very mild somatic post-mTBI symptoms (e.g., headache) but like other children in the mTBI group, they had returned to sport and school activities without further difficulty or distress. Fourteen athletes without a known history of mTBI were included in the control group. No participants were currently taking any medications.

Informed consent and assent was obtained at the time of study participation. All procedures and methods were in accordance with

Table 1. Participant Characteristics

Group	*Age at test (mean + standard deviation)*	*Handedness (right:left)*	*Cause of injury (sport)*
Control	12.59 (1.55)	13:1	N/A
mTBI	12.61 (1.55)	13:0	Hockey ($n=12$), Martial arts ($n=1$)
With 1 injury	12.09 (1.38)	9:0	Hockey ($n=7$), Martial arts ($n=1$)
With 2 injuries	13.79 (1.35)	4:0	Hockey ($n=4$)

mTBI, mild traumatic brain injury.

the Research Ethics Boards at the University of Toronto and the Hospital for Sick Children.

Imaging protocols

Structural and functional MRI data were collected with a Siemens 3 Tesla Imaging and Spectroscopy System (Siemens Healthcare Global, Munich, Germany) with a 32-bit head coil at the Hospital for Sick Children. A full set of clinical images were acquired, including: T1 and T2-weighted, fluid-attenuated inversion recovery, diffusion tensor and T2* images. Full-brain fMRI was performed to measure blood-oxygen-level-dependent (BOLD) signal changes associated with cognitive function acquired with a single-shot T2*-weighted pulse sequence with gradient echo planar imaging. Thirty 5-mm axial slices were collected orthogonal to the participant with the following parameters: TR = 2 seconds, TE = 30 milliseconds, matrix = 128 × 128, FOV = 192 mm, bandwidth = 2694 Hz/Px, and flip angle = 70°.

Clinical scans were reviewed by a staff neuroradiologist at the Hospital for Sick Children. One child in the control group returned for further imaging, but the finding was determined to be a normal structural variant. No participant in the mTBI group displayed structural abnormalities.

Experimental tasks

E-Prime 2.0 (Psychology Software Tools, Inc., Sharpsburg, PA) was used to run three experimental tasks on a notebook computer (Acer America Corporation (Canada), Mississauga, ON). A visuospatial working memory (n-back) task was administered at four levels of complexity as described previously.[71] At all levels, the stimuli were blue squares appearing in eight different loci on the computer screen. The 0-back condition served as a cognitive baseline with minimal memory demands during which participants were asked to respond to a square in a pre-specified location (e.g., ''upper right corner'') by pressing a button on a response box (Current Designs, Inc., Philadelphia, PA) with their left index finger. In the working memory conditions, difficulty (load) was manipulated by instructing participants to press a button when the blue square matched the location of a square that was presented 1, 2 or 3 screens previously (1-back, 2-back, and 3-back conditions, respectively). Similar to Jaeggi and colleagues,[71] we used a blocked periodic design (Fig. 1).

Participants also performed a motor task[65]: Whenever a green screen appeared, alternating button presses were required with the right index and middle fingers at a rhythm of about two button presses per second. The baseline condition was an equivalent period of rest (30 seconds) where no button press occurred in response to a black screen.

The n-back and motor tasks were performed in isolation (single-task condition) and simultaneously (dual-task condition). The order of presentation of each condition was counterbalanced across participants. Two versions of the n-back task were created (A and B) where the spatial location of the squares was randomized across versions, and the presentation of the 0-, 1-, 2-, and 3-back conditions was randomized across participants.

Neuropsychological testing

To control for any cognitive differences, we briefly assessed intelligence and other domains commonly impaired following mTBI (Table 2).[72–76] No participant exhibited impairment on any task and there were no significant differences between groups; thus, all participants were included in the study and these scores were not used in subsequent analyses.

Procedure

Depending on the timing of the participant's arrival, imaging was performed either before or after the neuropsychological tests.

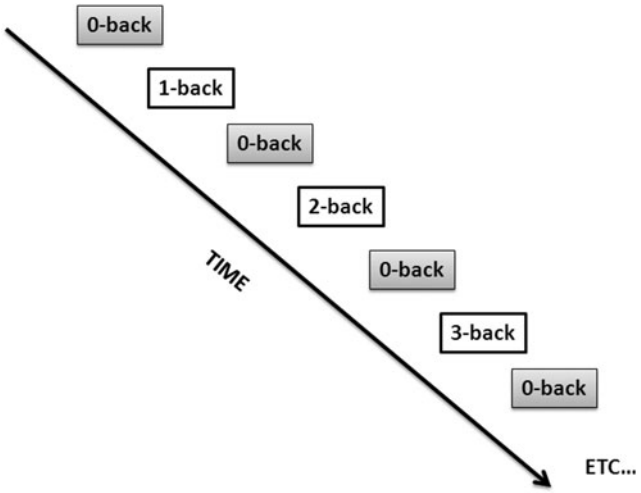

FIG. 1. n-Back working memory task. In this task, a 30-sec 0-back condition (baseline) always preceded and followed the 60-sec 1-, 2-, and 3-back working memory conditions. A 15-sec instruction screen preceded all conditions to alert participants to task demands. The order of the working memory conditions was randomized, such that each occurred twice and always preceded and followed by the baseline condition. Each working memory condition was presented twice, and such that the entire task took 12 min 45 seconds to complete.

All participants practiced each experimental task once to ensure comprehension outside the MRI, and there were no differences between groups in terms of practice performance parameters (i.e., single or dual-task hit reaction time or accuracy). Once in the scanner, goggles were placed over the participants' eyes to deliver task presentation; prescription strength goggles were provided for one individual. Anatomical images were acquired prior to presentation of the experimental tasks. Total scan time was approximately 1 h.

Following image acquisition, all participants completed a ''task difficulty'' questionnaire (Appendix A) to informally assess perceived cognitive effort. There were no group differences in the perception of task difficulty across conditions. Generally, participants reported greater difficulty with working memory at higher cognitive loads. The motor task was reportedly easier than the working memory task; and the dual-task condition was perceived as more challenging than the single-task condition, especially at higher loads.

Behavioral analysis

Both groups performed at chance during the 3-back condition; thus, these data were excluded from further analyses. Because there were significant differences in working memory performance across task versions (data not shown), ''version type'' was entered as a between-subjects variable.

Dependent variables of interest were accuracy (hits minus false alarms, expressed as a percentage) and median reaction time (RT; milliseconds) on ''hit'' or correct trials only. Dual-task cost was calculated to determine whether there was a change in performance measures while performing two tasks at once by controlling for individual differences in single-task performance: dual-task cost (%) = [(single-task performance – dual-task performance)/single-task performance] × 100.[43]

A two-way repeated measures analysis of variance (ANOVA) was computed for all behavioral measures with working memory load (0-2 back) as the within-subjects factor, and group (mTBI, controls) and version (A, B) as the between-subjects factors. Similar repeated measures ANOVAs were used to examine error

Table 2. Neuropsychological Test Performance (Mean + Standard Deviation)

Domain assessed	*Task administered**	*Control group*	*mTBI group***
General intellectual ability	Wechsler Abbreviated Scale of Intelligence (Pearson Education, Inc., San Antonio, TX)		
	Vocabulary	11.25 ± 2.527	11.17 ± 2.250
	Matrix Reasoning	10.92 ± 2.314	11.50 ± 1.732
Visuospatial Memory	Children's Memory Scale (Pearson Education, Inc., San Antonio, TX)		
	Dots Immediate Recall	11.92 ± 1.881	12.75 ± 1.603
	Dots Delayed Recall	12.92 ± 1.084	12.83 ± 0.937
Inhibitory control and behavioral flexibility	Stroop Color and Word Test: Children's Version (PAR Inc., Lutz, FL)	37.92 ± 17.537	38.50 ± 14.988
Fine motor speed and dexterity	Grooved Pegboard Test (Lafayette Instrument Company, Loughborough, Leics, UK)		
	Dominant hand	− 0.027 ± 0.783	-0.543 ± 1.156
	Non-dominant hand	0.309 ± 0.652	-0.749 ± 1.689
Processing speed	Trail Making Test		
	Trails A	0.659 ± 0.683	0.545 ± 0.724
	Trails B	0.486 ± 0.674	0.802 ± 0.505

*Tests were presented in the following order: Dots Immediate Recall, Vocabulary, Trail Making Test A and B, Stroop Colour and Word Test, Dots Delayed Recall, Grooved Pegboard, and Matrix Reasoning.

**There were no statistically significant differences between groups on any test.

type, with ''misses'' (omission errors) and ''false alarms'' (commission errors) as the dependent variables. In line with previous work,[77] age at testing correlated significantly with working memory accuracy (all, $p < 0.05$); thus, this factor was entered as a covariate in the accuracy and error analyses. Effect sizes for each ANOVA are reported as ''partial $\eta 2$'', where 0.1 represents a small effect, 0.25 represents a medium or moderate effect size, while >0.5 represents a large or strong effect size.

fMRI analysis

Images acquired during the fMRI scans were corrected for motion artifacts using 3-D prospective acquisition correction technique implemented by Siemens for real-time motion correction of BOLD data.[78] Motion-corrected images were spatially smoothed with a 6-mm full-width at half-maximum Gaussian filter to increase the signal-to-noise ratio of the data. A principal components analysis was then carried out to reveal temporal and spatial drifts. The voxel-wise statistical analysis was performed using fMRIstat[79] where the BOLD data was converted to percentage of the whole volume, and significant percent BOLD changes between experimental and baseline conditions were determined at each voxel based on a linear model with correlated errors. A design matrix of the linear model containing the onset time and duration of each event was convolved with a hemodynamic response function modeled as a difference of two gamma functions and corrected for slice-timing to coincide with the acquisition of each slice.[80] Spatial and temporal drifts identified from the above analysis were incorporated into the design matrix and removed from analysis. For each participant, contrasts comparing activity during each event were calculated using voxel-level t-statistics to identify brain regions that were differentially engaged across varying levels of task complexity relative to baseline. The autocorrelation parameter was estimated from the least squares residuals using the Yule-Walker equations, after a bias correction for correlations induced by the linear model. The autocorrelation parameter was first regularized by spatial smoothing, and then used to whiten the data and the design matrix. Next, the linear model was re-estimated using least squares on the whitened data to produce estimates of effects and their standard errors.

To obtain the average group t-map, data from first-level individual analysis were normalized through linear registration to the Montreal Neurological Institute template (ICBM152) using an in-house algorithm.[80] The resulting t statistic images were thresholded using the minimum given by a Bonferroni correction and random field theory to correct for multiple comparisons, taking into account the non-isotropic spatial correlation of the errors.[79] Normalized data were then combined according to study group in a second-level analysis using a mixed effects linear model with fixed effects standard deviations taken from the previous analysis. A random effects analysis was performed by estimating the ratio of the random effects variance to the fixed effects variance, then regularizing this ratio by spatial smoothing with a Gaussian filter. The variance of the effect was estimated by the smoothed ratio multiplied by the fixed effects variance. The amount of smoothing was chosen to achieve 100 effective degrees of freedom.

Between-group comparisons (mTBI vs. controls) were carried out in a third-level analysis using fixed effects linear model. As described in Rasmussen and colleagues,[65] a combined contrast was calculated for analysis of differences in the dual-task condition at each level of complexity (0-, 1-, and 2-back) and the combined single-task conditions such that combined contrast = dual-task (ON-OFF) – (motor task, ON-OFF + n-back task, ON-OFF). A whole-brain regression analysis also was conducted to further examine neural correlates of behavior across tasks and conditions.

Results

Single-task performance: Accuracy

Accuracy in target detection was similar between the baseline (0-back) and 1-back task; however, as working memory load increased, performance decreased (linear effect, $F[2, 44] = 3.947$, $p = 0.027$, partial $\eta 2 = 0.152$; Table 3). Specifically, participants exhibited a greater number of commission errors (linear effect, $F[2, 44] = 5.374$; $p = 0.008$; partial $\eta 2 = 0.196$) as the task complexity increased. There was no significant working memory load by group interactions, nor any significant differences between groups ($p > 0.05$). There was a significant interaction between load and version (quadratic effect, $F[2, 44] = 4.453$; $p = 0.017$; partial $\eta 2 = 0.168$) but further elaboration is unwarranted because there was no significant interaction between group and version type.

TABLE 3. PERFORMANCE ON THE N-BACK WORKING MEMORY TASK IN THE SINGLE-TASK AND DUAL-TASK CONDITIONS (MEAN + STANDARD ERROR)

	Single-task: percent accuracy			*Single-task: median reaction time (milliseconds)*			*Dual-task: percent accuracy*			*Dual-task: median reaction time (milliseconds)*		
Group	*0-Back*	*1-Back*	*2-Back*	*0-Back*	*1-Back*	*2-Back*	*0-Back*	*1-Back*	*2-Back*	*0-Back*	*1-Back*	*2-Back*
Control	87.77 (1.43)	88.29 (2.12)	66.39 (4.9)	523.04 (20.10)	493.61 (32.97)	582.36 (49.98)	92.53 (2.50)	88.31 (4.58)	72.00 (4.68)	594.39 (36.21)	645.89 (47.54)	630.18 (55.47)
mTBI	86.50 (1.49)	90.09 (2.21)	79.10 (5.13)	555.20 (20.91)	513.89 (34.32)	618.54 (52.03)	92.78 (2.60)	83.33 (4.77)	74.49 (4.87)	654.85 (37.69)	712.51 (49.48)	829.99 (57.73)

mTBI, mild traumatic brain injury.

Single-task performance: Median RT

Participants performed somewhat faster in the 1-back condition relative to the 0-back condition but slowed as the working memory load increased (i.e., to the 2-back condition; quadratic effect, $F[2, 46] = 6.720$; $p = 0.003$; partial $\eta 2 = 0.226$). There were no significant RT differences between groups nor were there any significant interactions (see Table 3).

Single-task neural activation

Table 4 reports the significant n-back task-related activation peaks and Figure 2 displays the major activation sites on a standardized brain atlas. Figure 3 reveals the results of the subtraction analyses. Although there were similar areas of activation between groups during task performance, there were some significant differences as well. Controls displayed greater activity than the mTBI group in the DLPFC during the working memory conditions relative to the baseline condition (1-0 comparison, non-significant trend for the right DLPFC; 2-0 comparison, left DLPFC). In the 2-1 subtraction, an opposite finding in the right DLPFC was found, with greater activity displayed by the mTBI group. In contrast, the control group displayed greater activity in the bilateral rostral insula.

Results from the whole-brain regression analyses (Fig. 4) revealed that when working memory demand increased from 1- to 2-back, the BOLD signal in the right DLPFC was positively correlated with accuracy performance in the mTBI group only, such that greater brain activity was associated with better performance ($r = 0.682$; $p = 0.01$). There were also positive correlations between performance and activity in the right parietal cortex ($r = 0.741$; $p = 0.004$) and supplementary motor area ($r = 0.832$; $p < 0.001$). With respect to median RT, the main differences were again in the 2-1 back comparison in the mTBI group only where slower the response, the greater the signal in the right DLPFC ($r = 0.614$; $p = 0.02$) and a non-significant trend in the right parietal lobe ($r = 0.525$; $p = 0.06$).

Dual-task performance: Accuracy

In the dual-task condition, working memory accuracy decreased as load increased from the 1-back to the 2-back condition (linear effect, $F[2, 44] = 4.141$; $p = 0.023$; partial $\eta 2 = 0.158$; Table 3) due to an increased number of commission errors (linear effect, $F[2, 44] = 4.406$; $p = 0.018$; partial $\eta 2 = 0.167$). There was a significant interaction between version and load (linear effect, $F[2, 44] = 3.777$; $p = 0.031$; partial $\eta 2 = 0.147$) but no significant main effect of version or group, or an interaction between version and group.

Dual-task performance: Median RT

Speed slowed as a function of working memory load (linear effect, $F[2, 44] = 6.779$; $p = 0.003$; partial $\eta 2 = 228$; Table 3). There was no main effect of group on median RT, yet there was a significant interaction between RT and group (linear effect, $F[2, 44] = 3.777$; $p = 0.030$; partial $\eta 2 = 0.141$) such that in the control group, speed increased from the baseline to the 1-back condition, while the mTBI group became progressively slower as the task became more challenging (i.e., from 1- to 2-back).

Dual-task cost

In terms of the influence of the dual-task on working memory accuracy, there were no within- or between-group differences, nor

Table 4. fMRI t-map Activation Peaks

		Control				*mTBI*			
Region	*BA*	*x*	*y*	*z*	*t*	*x*	*y*	*z*	*t*
Single-task (1 back - 0 back)									
Left middle frontal gyrus	10					−40	58	2	4.39
Right DLPFC	9/46	38	42	30	4.01				
Right DLPFC	9/46	42	32	42	4.03	42	30	44	4.75
Dorsal cingulate cortex	32	10	22	26	4.99	0	22	42	4.14
Right rostral insula		32	24	4	4.42	30	20	14	4.69
SMA		−6	10	54	6.02	6	8	56	4.57
Left premotor	6	−26	4	58	5.82				
Right premotor	6	32	0	56	5.44	48	6	42	5.07
Left inferior parietal lobule	40	−32	−48	42	4.88				
Right inferior parietal lobule	40	38	−60	50	5.69	42	−46	42	4.45
Right precuneus	7	2	−80	−4	5.61				
Lingual gyrus	18					−2	−76	4	5.25
Left cerebellum		−28	−58	−26	4.55	−38	−54	−34	6.03
Right cerebellum		32	−58	−28	4.06	36	−60	−46	3.84*
Single-task (2 back - 0 back)									
Left middle frontal gyrus	10	−38	52	8	4.36	−30	52	12	5.08
Right middle frontal gyrus	10	38	58	16	4.88	32	50	6	5.43
Left DLPFC	9/46	−38	28	36	4.54	−44	20	32	4.55
Right DLPFC	9/46	44	40	24	5.38				
Right DLPFC	9/46	42	34	38	5.86	48	26	32	6.61
Dorsal cingulate cortex	32	8	30	32	4.87	4	26	42	7.50
Left rostral insula		−36	20	−2	5.44	−32	18	2	5.50
Right rostral insula		34	22	4	6.92	34	18	−2	6.07
SMA		−6	8	56	5.55				
Left premotor	6	−30	0	56	6.74	26	−4	50	4.51
Right premotor	6	28	8	54	5.98	42	6	44	7.10
Left caudate nucleus		−16	−2	22	5.04				
Right caudate nucleus		14	−6	22	5.05				
Left thalamus		−12	−6	8	4.67	−16	−12	16	4.24
Right thalamus		6	−14	14	5.69	8	−18	16	6.13
Left inferior parietal lobule	40	−36	−46	42	6.37	−38	−50	42	7.96
Right inferior parietal lobule	40	40	−44	48	6.18	40	−42	48	6.40
Right inferior parietal lobule	40	34	−56	44	5.93	42	−52	44	5.87
Left precuneus	7	−8	−62	50	5.09	−4	−66	52	4.91
Right precuneus	7	6	−62	54	5.60	14	−64	50	5.11
Left cerebellum		−34	−60	−28	5.14	−30	−64	−32	5.68
Left cerebellum		−8	−82	−20	5.53	−6	−78	−18	6.63
Left cerebellum						−30	−72	−48	4.90
Right cerebellum		26	−60	−30	5.61	28	−60	−28	5.07
Right cerebellum		8	−76	−26	6.02				
Single-task (2 back - 1 back)									
Right DLPFC	9/46					48	26	32	4.38
Dorsal cingulate cortex	32	2	22	44	4.66	4	26	44	4.58
Left rostral insula		−30	20	4	5.14				
Right rostral insula		32	22	0	4.30				
Right premotor	6					42	8	40	4.27
Left inferior parietal lobule	40	−38	−46	44	4.99	−32	−48	44	4.70
Right inferior parietal lobule	40					42	−48	54	4.30
Right inferior parietal lobule	40					34	−56	52	4.68
Right inferior parietal lobule	40					38	−66	40	4.85
Left precuneus	7					−2	−66	52	5.60
Left cerebellum						−14	−72	−22	4.69
Dual-task (1 back - 0 back)									
Left middle frontal gyrus	10	−38	54	10	4.43	−28	50	0	3.93*
Left DLPFC	9	−38	28	30	5.31	−34	28	30	4.10
Right DLPFC	9/46	42	36	26	5.17	36	40	26	4.53

(*continued*)

TABLE 4. (CONTINUED)

		Control				*mTBI*			
Region	*BA*	*x*	*y*	*z*	*t*	*x*	*y*	*z*	*t*
Right DLPFC	9/46	42	28	32	5.63				
Dorsal cingulate cortex	32					2	24	44	5.12
Left rostral insula		−32	20	4	5.11	−30	20	2	4.16
Right rostral insula		26	24	6	6.02	32	22	−4	4.39
SMA		−8	12	52	4.71	6	8	54	5.47
Left premotor	6	−30	0	58	4.47	−26	4	60	3.74*
Right premotor	6	32	−4	44	4.33	38	2	54	4.10
Left caudate nucleus		−16	4	18	5.29				
Right caudate nucleus		16	6	22	4.11	18	18	10	4.07
Left thalamus		−4	−6	0	4.04				
Right middle temporal gyrus	21	58	−38	2	4.05	54	−34	−6	3.48*
Left inferior parietal lobule	40	−36	−48	44	5.62	−40	−48	52	4.99
Right inferior parietal lobule	40	44	−44	48	4.83	40	−46	44	4.50
Right inferior parietal lobule	40	36	−52	48	5.08	38	−52	56	4.35
Left cerebellum		−26	−60	−28	5.05				
Left cerebellum		−38	−68	−28	5.51				
Right cerebellum		28	−62	−26	4.03				
Right Cerebellum		36	−64	−48	4.30				
Dual-task (2 back - 0 back)									
Left middle frontal gyrus	10	−34	52	8	4.86	−30	56	−2	6.36
Right middle frontal gyrus	10	32	62	6	5.50	30	56	10	6.71
Left DLPFC	9/46	−46	26	38	4.88	−38	22	36	4.52
Right DLPFC	9/46	42	32	36	6.00	44	28	32	5.76
Dorsal cingulate cortex	32	0	24	48	6.26	2	26	42	7.89
Left rostral insula		−26	24	4	6.32	−32	22	6	5.96
Right rostral insula		34	22	−2	9.22	32	18	2	6.60
Left premotor	6	−22	2	52	6.86	−24	−4	54	6.47
Right premotor	6	32	4	62	7.19	32	2	52	7.52
Left caudate nucleus		−16	2	20	5.57				
Right caudate nucleus		18	6	22	5.47	14	−2	20	3.44*
Right thalamus		10	−8	6	6.90	10	−10	8	4.04
Left inferior parietal lobule	40	−36	−46	42	5.99	−38	−46	46	7.61
Right inferior parietal lobule	40	40	−44	44	5.25	44	−52	50	6.75
Right inferior parietal lobule	40	34	−62	44	6.00	38	−56	54	6.74
Left precuneus	7					−2	−62	54	7.09
Right precuneus	7					8	−64	50	5.53
Left cerebellum		−28	−62	−28	6.41	−26	−66	−30	4.30
Left cerebellum						−38	−66	−46	5.19
Left cerebellum						−8	−78	−24	4.92
Right cerebellum		26	−60	−30	6.71				
Dual-task (2 back - 1 back)									
Left DLPFC	9/46	−48	28	38	4.10				
Right DLPFC	9/46					46	26	42	4.80
Dorsal cingulate cortex	32	10	30	30	4.07	6	28	44	4.35
Left rostral insula						−34	22	6	4.14
Right rostral insula						32	16	2	4.24
Left premotor	6	−24	2	52	4.60	−24	0	54	5.48
Right premotor	6	18	8	60	4.64	30	0	52	5.35
Right thalamus		8	−12	8	5.01				
Left inferior parietal lobule	40					−26	−50	44	4.46
Left precuneus	7	−6	−54	44	4.26	−2	−62	52	5.39
Right precuneus	7	4	−54	44	4.46	8	−60	48	5.88
Left cerebellum		−14	−72	−26	4.09	−8	−78	−24	4.08

*non-significant trend

t threshold = 4.00

fMRI, functional magnetic resonance imaging; BA, Brodmann Area; mTBI, mild traumatic brain injury; DLPFC, dorsolateral prefrontal cortex; SMA, supplementary motor area.

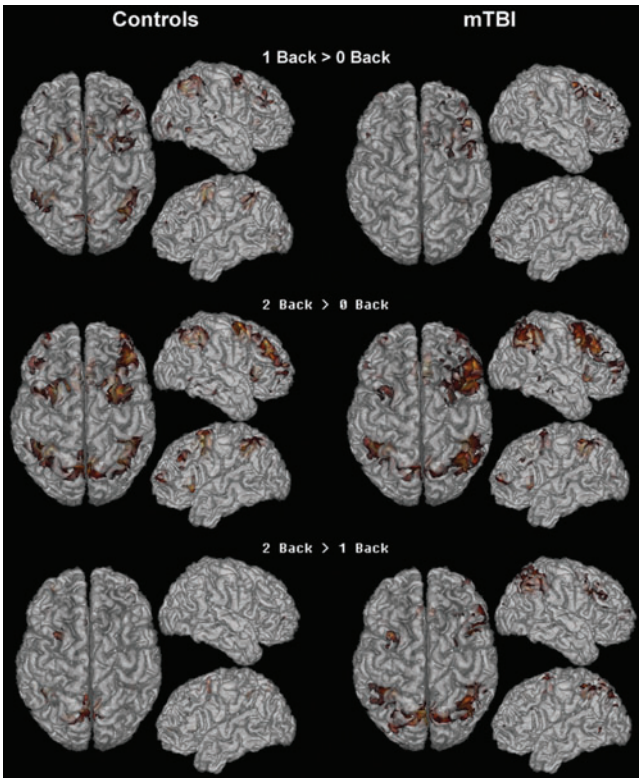

FIG. 2. Major working memory activation sites during the single-task condition. Color image is available online at www.liebertpub.com/neu

were there any significant interactions with group membership. There was a significant interaction between cost and version (linear effect, $F[2, 44] = 6.074$; $p = 0.005$; partial $\eta 2 = 0.216$) but there was no significant main effect of version or interaction between group and version. There was a significant main effect of working memory load on median RT (quadratic effect, $F[2, 44] = 5.282$; $p = 0.009$; partial $\eta 2 = 0.187$), with both groups performing slower during the dual-task relative to the single-task condition. There were no significant effects of group on dual-cost RT nor were there any interactions.

Dual-task neural activations

Table 4 reports the significant dual-task-related activation peaks, Figure 5 displays the major activation sites on a standardized brain atlas, and Figure 6 displays the between-group subtraction results.

Similarities in activation patterns were found between groups, but differences were also noted. The control group displayed stronger activation in the right DLPFC, compared with the mTBI group in the 1-0 back and 2-0 back comparisons, and they also displayed greater activation in the 1-0 back subtraction in the right rostral insula. Although the control group exhibited greater activity in the left DLPFC in the 2-1 comparison, the mTBI group displayed greater activity in the same structure on the right.

The regression analysis revealed that in the high load dual-task condition (2-back), accuracy was positively correlated with BOLD signal in the right DLPFC ($r = 0.881$; $p < 0.01$), right rostral insula ($r = 0.906$; $p < 0.01$), and bilateral parietal cortex (left: $r = 0.785$; $p < 0.01$; right: $r = 0.781$; $p < 0.01$) in the control group only (Fig. 7). For median RT, the main findings are again in the 2-back relative to

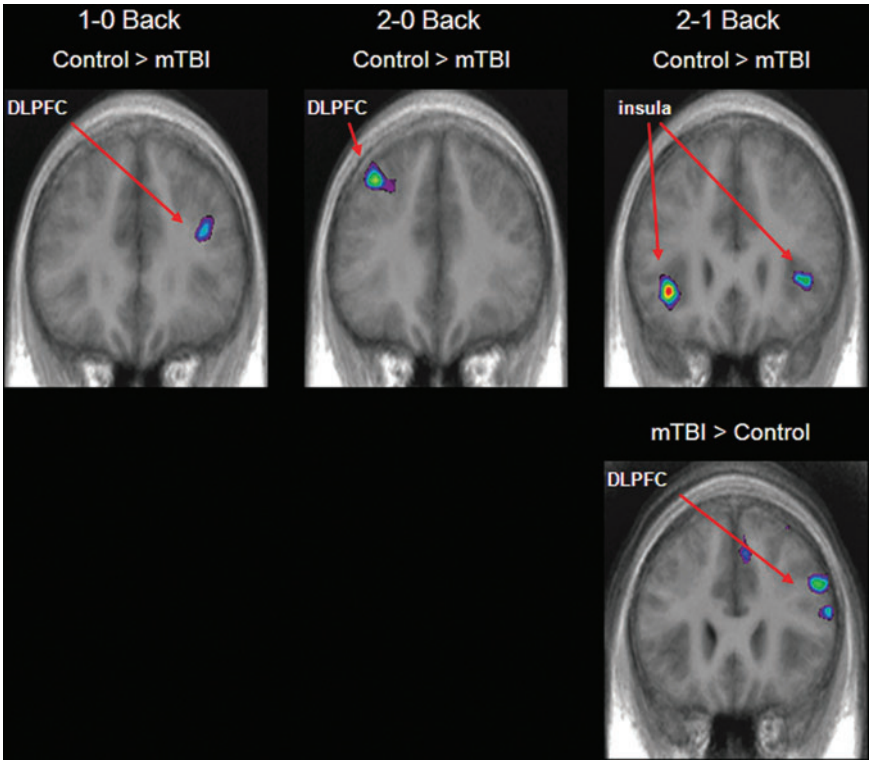

FIG. 3. Between-group comparisons, single-task condition. Controls displayed greater activity than the mTBI group in the right dorsolateral prefrontal cortex (DLPFC) in the 1-0 subtraction and left DLPFC in the 2-0 subtraction. In the 2-1 comparison, there was greater activity displayed by the mild traumatic brain injury in the right DLPFC, with the control group displaying greater activity in the bilateral rostral insula. Color image is available online at www.liebertpub.com/neu

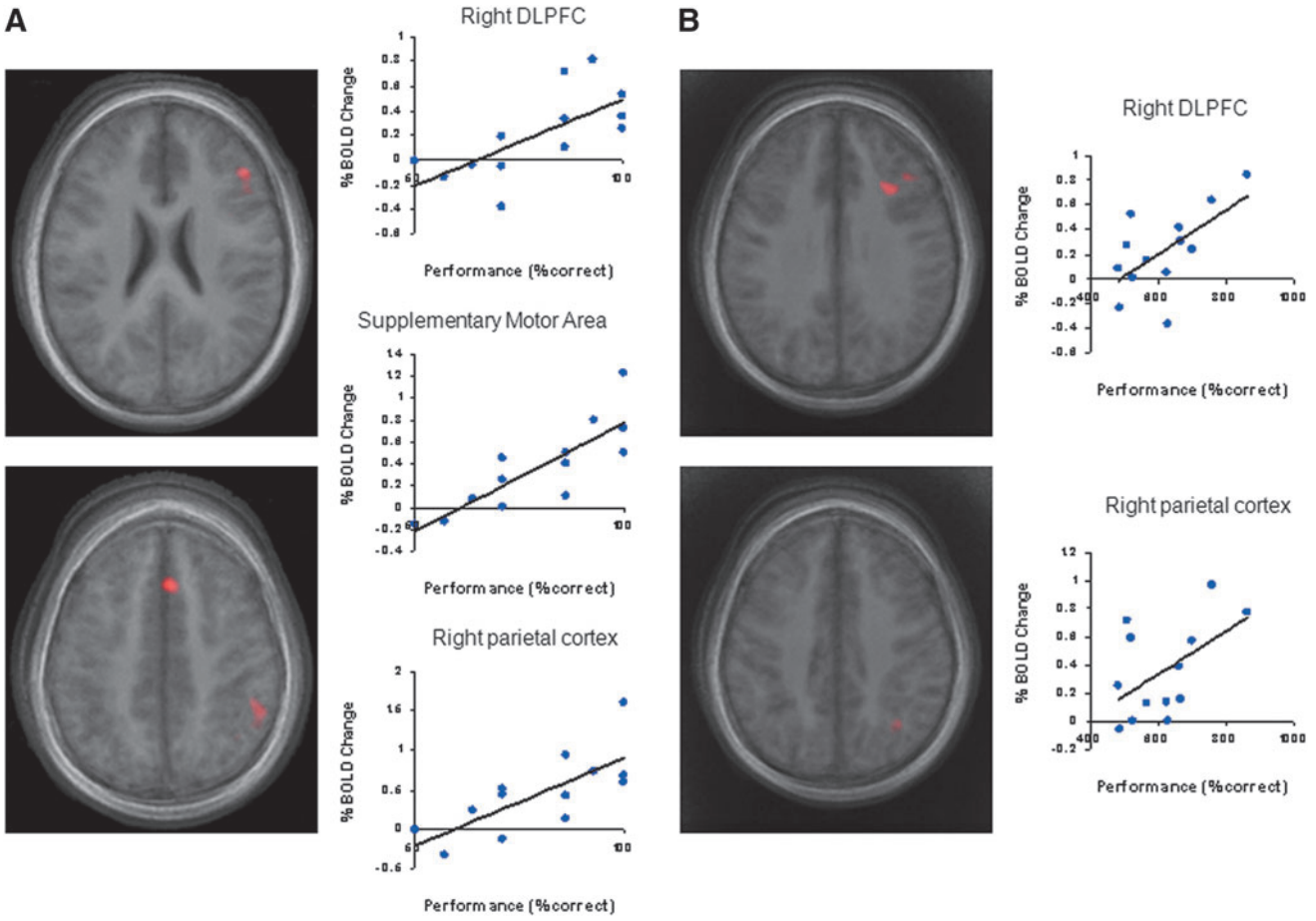

FIG. 4. Single-task significant blood-oxygen-level-dependent regressions. **(A)** Mild traumatic brain injury (mTBI) group accuracy performance, 2-1 back subtraction. **(B)** mTBI group reaction time performance, 2-1 back subtraction. Color image is available online at www.liebertpub.com/neu

the 0-back condition, where the control group shows negative correlation between BOLD signal and response speed in the right DLPFC ($r = -0.786$; $p < 0.01$), right premotor ($r = -0.821$, $p < 0.01$), supplementary motor area ($r = -0.786$, $p < 0.01$), left parietal ($r = -0.807$; $p < 0.01$) and right parietal ($r = -0.780$; $p < 0.01$). These correlations were not found in the mTBI group.

Discussion

The current study sought to examine whether simultaneously challenging youth athletes with mTBI on both a cognitive and motor task would reveal underlying behavioral and neural abnormalities not previously detected in single-task paradigms. In line with previous work,[74,81–84] none of our mTBI participants displayed significant impairment on neuropsychological testing and their single-task working memory performance was similar to that of the control group. However, the demands of the dual-task condition resulted in group differences in the speed of responding and neural activation differences emerged not only during dual-task performance but also in the single-task condition too. The implications of these findings are discussed within a neurocognitive framework that posits that mTBI in youth leads to decreased cognitive efficiency, especially with increased task demands, which in turn may result in a need for greater allocation of cognitive resources at the brain level in order to maintain optimal performance.

Single task

Working memory has been proposed by Postle[68] to be the result of ''coordinated recruitment, via attention, of brain systems that have evolved to accomplish sensory-, representation-, and action-related functions.'' In line with this sentiment and with previous data,[85–89] we found that once participants engaged in a working memory task at a low cognitive load (1-back), activation was recorded in the supplementary motor area, premotor cortex, dorsal cingulate cortex, rostral insula, cerebellum, and inferior parietal cortex. Likewise, results from the 2-0 back subtraction suggest a cerebral recognition of task difficulty, as greater activation was observed in many areas noted above, as well as in the bilateral thalamus, mid frontal gyrus, bilateral precuneus, and left DLPFC.

Although we did not explicitly test the nature of these activations, we speculate that at the neural level, the demands of the working memory task were distinguished from those associated with the simple visual-spatial detection task via activation of areas important for vigilance/visuospatial attention, saliency detection, cognitive control, goal specification, and motor planning and preparation. When task complexity increased from the 1- to the 2-back condition, additional recruitment of the dorsal cingulate cortex and the left inferior parietal cortex was found, the latter being an area important for the storage or maintenance of spatial information.[90] Clearly, further research is needed that directly tests these activations before firm conclusions can be drawn regarding the nature of the activations.

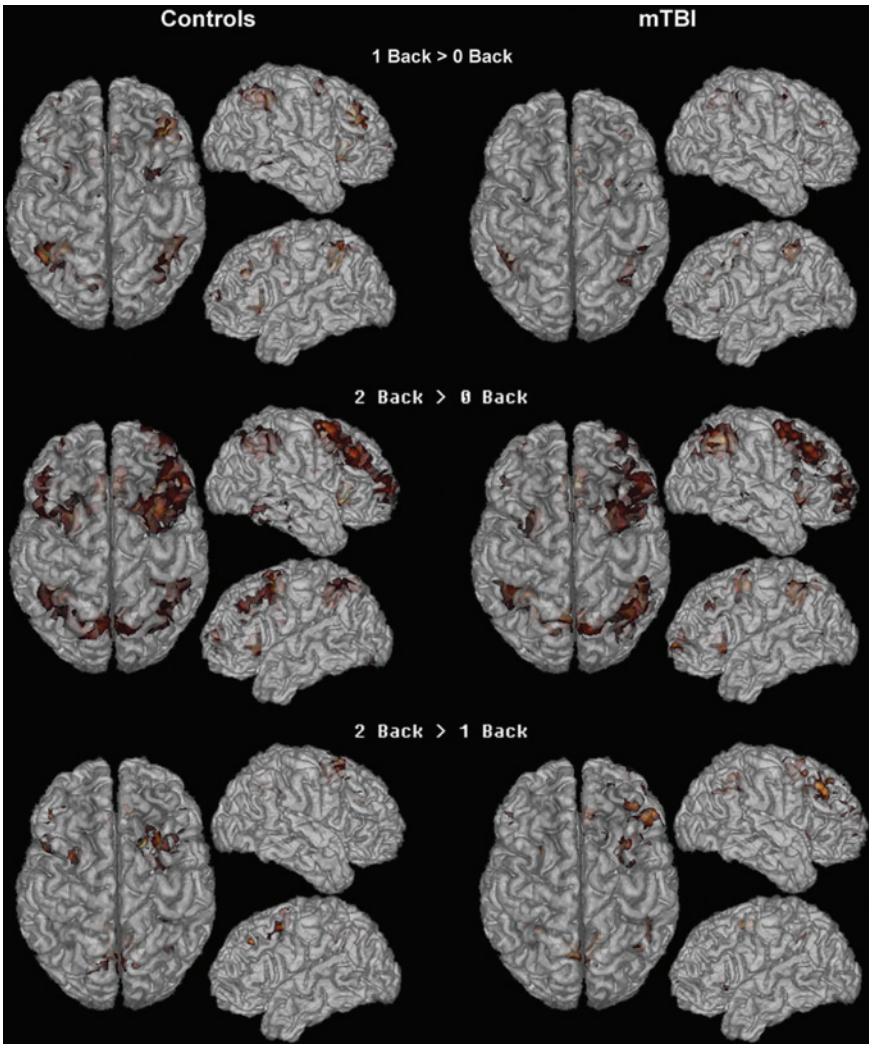

FIG. 5. Major working memory activation sites during the dual-task condition. Color image is available online at www.liebertpub.com/neu

Importantly, the control group displayed activation of expected nodes of the spatial working memory circuit during n-back performance.[88,90,91] When faced with a relatively easy level of complexity (1-back), participants activated the right DLPFC and posterior parietal cortex (precuneus and bilateral inferior parietal cortices). There was no additional recruitment of the right DLPFC with increased working memory load, but the 2-back condition did cause increased activity in the bilateral insula, supplementary motor area, and bilateral caudate. It appears then that in typically developing youth, key components of the working memory network come ''online'' once there is a need for such processes, and little or negligible increases in activation are required to maintain performance in response to increased task demands.[35] Rather, it may be that areas involved in goal-directed motor behavior are more important when faced with increased load, consistent with the idea that 2-back performance relies on higher-order executive processes, such as interference control and attentional shifting.[92,93]

We also found key activation differences between groups despite similarities in performance. In general, injured youth displayed a more unilateral activation pattern. More specifically, the mTBI group did not activate the working memory network during the 1-back task but instead displayed activity in the left mid frontal and lingual gyrus—areas known to be involved in strategic planning and encoding of complex visual information. It seems that no differentiation was made in terms of the allocation of processing resources between the levels of working memory load, as similar activations were found in the 2-0 back comparison. When the 1-back condition was specifically compared with the 2-back condition, the mTBI participants finally displayed recruitment of working memory components, including the right DLPFC and additional right inferior parietal, right premotor, and left precuneus activation. Moreover, regression data suggest that the mTBI group required greater activation of the working memory nodes in order to sustain optimal or equivalent single-task performance.

Altered neural activity despite similar performance has been shown in children with moderate-severe TBIs.[94] In adults, McAllister and colleagues[35,36] revealed that symptomatic subjects with mTBI displayed abnormal recruitment of the right DLPFC and parietal lobes during an n-back task and less activation of the working memory network overall. We have now shown that despite intact neuropsychological test performance, the lack of significant post-injury symptoms, and the absence of structural brain damage detected by conventional neuroimaging, differences exist in neural recruitment following mTBI in youth during working memory performance. Overall, these data support the hypothesis of a predominantly

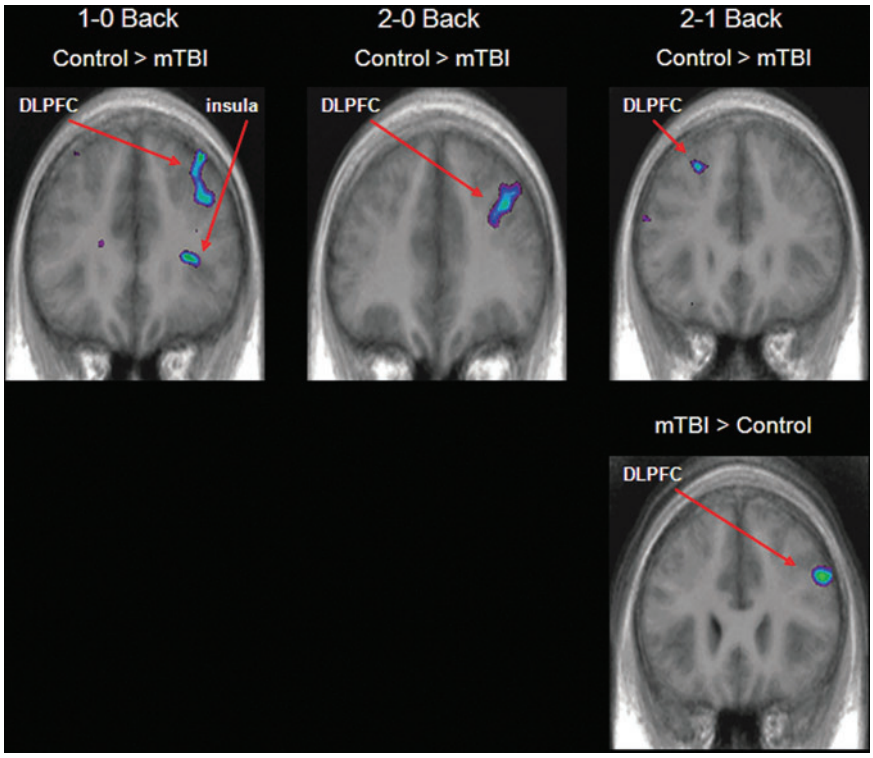

FIG. 6. Between-group comparisons, dual-task condition. The control group displayed stronger activation than the mild traumatic brain injury (mTBI) group in the right dorsolateral prefrontal cortex (DLPFC) in the 1-0 back and 2-0 back subtractions, in addition to greater activation in the 1-0 back subtraction in the right rostral insula. The control group also exhibited greater activity in the left DLPFC in the 2-1 comparison, while the mTBI group displayed greater activity in the same area on the right. Color image is available online at www.liebertpub.com/neu

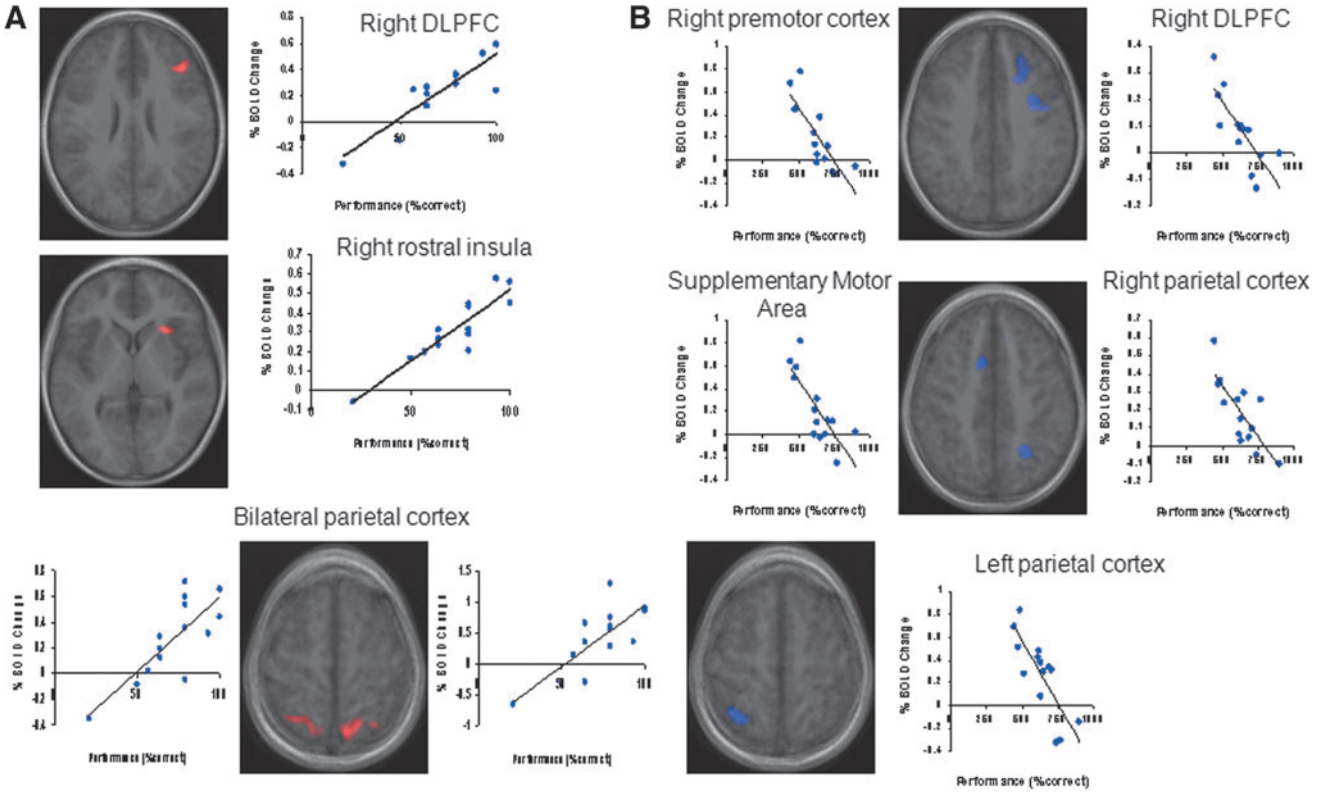

FIG. 7. Dual-task significant blood-oxygen-level-dependent regressions. **(A)** Control group accuracy performance, 2-0 back subtraction. **(B)** Control group reaction time performance, 2-0 back subtraction. Color image is available online at www.liebertpub.com/neu

functional consequence following mTBI[18,95,96] and provides evidence for neural changes persisting well beyond the acute phase of injury.

Dual-task

Consistent with a number of studies,[45,97–99] we found that dual-task performance activated a broad neural network including the left DLPFC and other aspects of the prefrontal cortex, posterior parietal cortex, caudate, and cerebellum. The left DLPFC is strongly implicated in mediating dual-task performance by allocating attention resources to both tasks[45] and for processing of sequential dependencies, especially with increased cognitive load.[71,100,101] Prefrontal regions may act to bias signals towards the parietal cortex to activate stimulus-driven, bottom-up shifts of attention that ultimately cause the coordination and implementation of a response.[98,102–106] Thus, to be efficient, response selection may entail a resolution of dual-task conflict by engaging the anterior cingulate, inferior frontal gyrus, and inferior parietal gyrus in order to re-orient attention towards task stimuli while inhibiting irrelevant, non-task specific activity and maintaining and monitoring immediate and future behavioral goals.[107–109]

Important to our central objectives, group differences in BOLD activation were found. Similar to our single-task imaging results, the control group activated the right DLPFC as soon as the task increased in complexity from the baseline to the working memory condition, with no further activation of this area noted during the 2-back condition. These participants also showed activation in the caudate, thalamus, cerebellum (i.e., areas thought to be involved in goal-directed motor planning and response control), with greater recruitment of bilateral structures in the 2-0-back subtraction suggestive of an increased reliance on these structures with increased load. Consistent with Rasmussen and colleagues,[65] we saw additional activation of the left DLPFC when the task increased in complexity from the 1-back to the 2-back dual-task condition, further supporting the idea that this region may be involved in the sequential allocation of processing resources with increased load.[101] In contrast, the mTBI group exhibited a less distributed network of activation with areas important for motor control/coordination activated to a lesser extent. Similar to the single-task findings, key working memory nodes (right DLPFC, posterior parietal areas) were recruited by the mTBI group only as cognitive load increased to the 2-back dual-task condition, and they did not exhibit any further activation of the left DLPFC beyond the 1-back dual-task condition.

Although dual-task working memory accuracy was similar between groups, participants with mTBI slowed their speed of responding as a function of working memory load (i.e., a speed-accuracy trade-off), a finding not observed in the control group. We argue that this may reflect reduced cognitive efficiency with increased task demands.[61,62] The exact mechanism underlying this performance is unclear, but self-report measures did not reveal group differences in the perceived difficulty or effort needed to execute each task, and neuropsychological test performance did not reveal any injury-related weaknesses in motor or information processing speed. It may be that mTBI causes subtle selective attention deficits such that participants had difficulty actively maintaining information within spatial working memory stores in the dual-task condition.[110] However, if this were the case, then one would then expect group differences in task accuracy as well.

During the dual-task, although initial perceptual processing of task demands is conducted in parallel, response selection is sequential; thus, a bottleneck of attentional resources occurs when two tasks are presented at once.[107,112] Thus, with increased working memory demands, as in the 2-back dual-task condition, both task rules cannot be optimally activated simultaneously (i.e., working memory is needed to maintain both task instructions in mind at once), resulting a decrement in single-task performance also known as dual-task cost.[111] Although more research is clearly needed, it may be that mTBI decreases one's capacity for efficient response selection, leading to more effortful processing during a dual-task due to the excessive working memory demands inherent to such tasks.[45,71,113] Indeed, our neuroimaging results seem to support this idea given the ''late'' activation of the working memory circuit during the 2-back condition and the lack of progressive recruitment of key dual-task structures (left DLPFC) only observed in the mTBI group. Overall, the net effect of greater effortful processing may result in functional disruptions leading to a greater need for cognitive control[93] and/or difficulties matching or allocating processing resources to task demands, the later resulting in an over-commitment of resources and/or difficulties anticipating future processing needs.[35,36,94]

Conclusion

A number of hypotheses have been proposed to account for post-injury changes in functional neural activation. The ''reorganization hypothesis'' assumes that the injured brain permanently re-wires itself to maintain optimal operations. Given our relatively small sample size, lack of pre-morbid levels of functioning, and the fact that we did not employ a longitudinal design, a claim of a permanent change is impossible to ascertain in the current study.[8,93] The ''compensation hypothesis'' states that the injured brain recruits additional resources to facilitate performance during more challenging tasks, and importantly, these changes would be transitory.[8] However, we observed alterations in BOLD signaling during relatively easy tasks, suggesting that this hypothesis is not entirely applicable to our data either. Alternatively, we have suggested that mTBI may result in poorer cognitive efficiency, resulting in a need for greater effortful processing and ultimately, a need for increased cognitive control and/or abnormalities in the allocation of processing resources during more difficult task demands.

A limitation to the current study was that the mTBI group was not as homogenous as hoped with the final group consisting of four children with multiple TBIs. However, we did ensure that the inclusions of the multiple mTBI participants did not significantly alter group differences (data not shown). Moreover, it seems that a history of three or more, but not two, mTBIs is associated with greater consequences for neuropsychological test performance, subjective symptoms, and neurophysiology.[115,116]

Future studies will further delineate group differences by examining the behavioral and functional neuroimaging correlates of the motor (finger-tapping) task during the single and dual-task conditions. We are currently examining behavioral correlates of cortical thickness as well as structural connectivity using diffusion tensor imaging. White matter abnormalities have been observed in children and adolescents with mTBI in the acute phase of injury.[117,118] and there is some evidence to suggest that these changes persist into the chronic phase of injury and are related to poorer parent ratings of behavior.[119]

Although these specific findings need replication, the potential clinical application of these data is great given that the annual incidence of sports-related mTBI in youth is 185 cases per 100,000.[114] We have further highlighted the limitations of conventional neuropsychological tests in delineating the long-term consequences of mTBI which points to a need for more comprehensive assessment of dual-task performance in this population.

The reduced dual-task speed points to decreased efficiency that may be potentially affecting all performance on some level, including school work and exams, participation in sport, and performance of other complex motor behavior, such as driving a car. Moreover, we have shown that even when there are similarities in cognitive performance, long-lasting neural changes can occur in the absence of ongoing symptoms and daily life disturbances. This is an important point of future study for longitudinal designs to address.

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Author Disclosure Statement

No competing financial interests exist.

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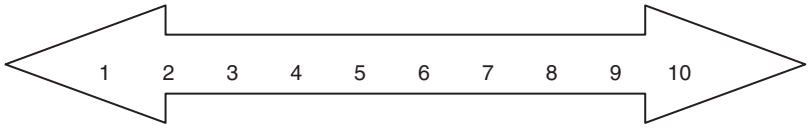
(*Appendix follows* →)

Appendix A

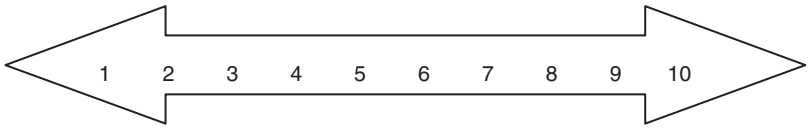
''Effort'' Questionnaire. Participants were instructed to circle their answers to each question.

How difficult did you find the motor (finger-tapping) task?



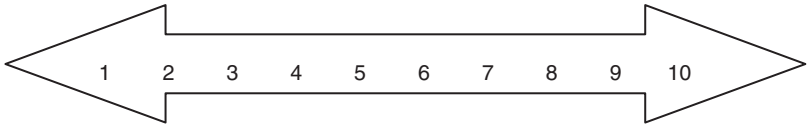
Extremely Difficult (not able to do it) — Very Easy (not difficult)

How difficult did you find the "blue squares task"?



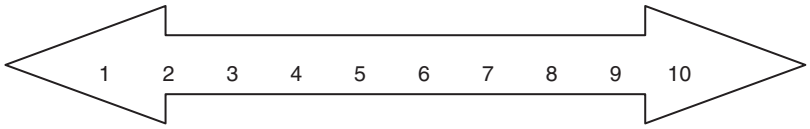
Extremely Difficult (not able to do it) — Very Easy (not difficult)

How difficult did you find responding to the location of squares when you were doing both the motor and the "blue squares task" at the same time?



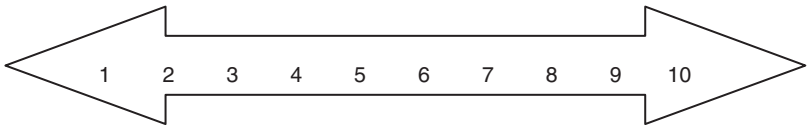
Extremely Difficult (not able to do it) — Very Easy (not difficult)

How difficult did you find the 1-back task when you were doing both the motor and the "blue squares task" at the same time?



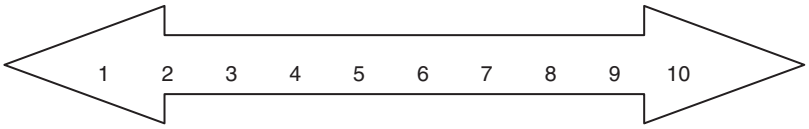
Extremely Difficult (not able to do it) — Very Easy (not difficult)

How difficult did you find the 2-back task when you were doing both the motor and the "blue squares task" at the same time?



Extremely Difficult (not able to do it) — Very Easy (not difficult)

How difficult did you find the 3-back task when you were doing both the motor and the "blue squares task" at the same time?

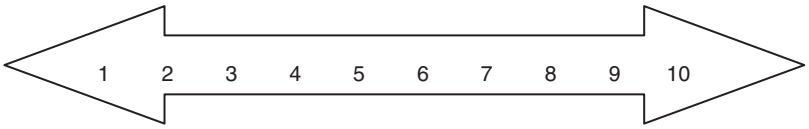


Extremely Difficult (not able to do it) — Very Easy (not difficult)

Did you find doing both the motor and "blue squares task" at the same time more difficult than doing the motor or "blue squares task" alone?

YES NO (circle one)

If YES, how much more difficult did you find doing both tasks at once?



A little harder — Extremely harder