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Injury versus non-injury factors as predictors of post-concussive symptoms following mild traumatic brain injury in children

Kelly A. McNally^{1,2}, Barbara Bangert³, Ann Dietrich^{2,4}, Kathy Nuss^{2,4}, Jerome Rusin⁵, Martha Wright^{6,7}, H. Gerry Taylor^{6,7}, and Keith Owen Yeates^{2,8}

¹Section of Pediatric Psychology and Neuropsychology, Nationwide Children's Hospital, Columbus, Ohio

²Department of Pediatrics, The Ohio State University, Columbus, Ohio

³Departments of Radiology and Neurosurgery, University Hospitals Health System, Cleveland, Ohio

⁴Department of Emergency Medicine, Nationwide Children's Hospital, Columbus, Ohio

⁵Department of Radiology, Nationwide Children's Hospital, Columbus, Ohio

⁶Department of Pediatrics, Case Western Reserve University, Cleveland, Ohio

⁷Rainbow Babies & Children's Hospital, University Hospitals Case Medical Center, Cleveland, Ohio

⁸Center for Biobehavioral Health, The Research Institute at Nationwide Children's Hospital, Columbus, Ohio

Abstract

Objective—To examine the relative contributions of injury characteristics and non-injury child and family factors as predictors of postconcussive symptoms (PCS) following mild traumatic brain injury (TBI) in children.

Methods—Participants were 8- to 15-year-old children, 186 with mild TBI and 99 with mild orthopedic injuries (OI). Parents and children rated PCS shortly after injury and at 1, 3, and 12 months post-injury. Hierarchical regression analyses were conducted to predict PCS from (1) demographic variables; (2) pre-morbid child factors (WASI IQ; WRAT-3 Reading; Child Behavior Checklist; ratings of pre-injury PCS); (3) family factors (Family Assessment Device General Functioning Scale; Brief Symptom Inventory; and Life Stressors and Social Resources Inventory); and (4) injury group (OI, mild TBI with loss of consciousness [LOC] and associated injuries [AI], mild TBI with LOC but without AI, mild TBI without LOC but with AI, and mild TBI without LOC or AI)

Results—Injury group predicted parent and child ratings of PCS but showed a decreasing contribution over time. Demographic variables consistently predicted symptom ratings across time. Premorbid child factors, especially retrospective ratings of premorbid symptoms, accounted for the most variance in symptom ratings. Family factors, particularly parent adjustment, consistently predicted parent, but not child, ratings of PCS.

Conclusions—Injury characteristics predict PCS in the first months following mild TBI but show a decreasing contribution over time. In contrast, non-injury factors are more consistently related to persistent PCS.

Keywords

Concussion; Pediatric; Outcomes; Recovery

Introduction

Mild traumatic brain injuries (TBI) are a common occurrence in children and adolescents (Bazarian et al., 2005), yet the outcomes associated with such injuries remain poorly understood. Many children experience post-concussive symptoms (PCS) after mild TBI, particularly during the acute period immediately following the injury. PCS include subjective somatic, cognitive, and emotional problems such as headache, dizziness, forgetfulness, inattention, and depressed or anxious mood. Although not specific to TBI, these symptoms are more common in children with mild TBI than in children with injuries not involving the head or demographically-matched, healthy controls (Farmer, Singer, Mellits, Hall & Charney, 1987; Fay, Jaffe, Polissar, et al., 1993; Mittenberg, Wittner, & Miller, 1997; Ponsford, Willmott, Rothwell, et al., 1999; Yeates, Luria, Bartkowski, et al., 1999). PCS tend to be most frequent and severe shortly after the injury, and in most cases resolve over time; however, symptoms can persist in at least a subset of children with mild TBI (Mittenberg, Wittner, Miller, 1997; McKinlay, Dalrymple-Alford, Horwood, & Fergusson, 2002; Nacajauskaite, Endziniene, Jureniene, & Schrader, 2006; Yeates et al., 2009, in press).

The etiology of PCS has been a highly controversial topic, particularly in the adult literature. PCS are not specific to TBI, occur with some frequency in the general population, and can occur in cases without objective signs or evidence of brain injury (e.g. loss of consciousness or neuroimaging abnormalities). This has sparked a debate regarding the determinants of PCS that has often been framed in terms of “psychogenesis” versus “physiogenesis” (Lishman, 1988; Alexander, 1997; Bigler, 2008). Proponents of “psychogenesis” argue that PCS are not related to alterations of brain function, but rather reflect premorbid differences, post-injury psychological factors, or even frank malingering (Binder, 1986). In contrast, proponents of “physiogenesis” argue that PCS are related to changes in brain function that occur following TBI, citing evidence from animal models as well as human clinical studies documenting acute neuropathology related to mild TBI (Barkhoudarian, Hovda, & Giza, 2011). Although “psychogenesis” and “physiogenesis” are often portrayed as competing explanations, they are not mutually exclusive. In fact, research in adults has shown that both injury characteristics and non-injury related factors help account for neurobehavioral outcomes in mild TBI (Kashluba, Paniak & Casey, 2008; Luis, Vanderploeg, & Curtiss, 2003; Ponsford et al., 2000).

Although previous research on mild TBI in children is limited, recent data support the assertion that PCS are associated with injury-related factors, including whether the injury involves TBI and its severity. Yeates and colleagues (Taylor et al., 2010; Yeates et al., 2009, in press) have shown that children with mild TBI are more likely than children with orthopedic injury to demonstrate high levels of acute PCS, as well as persistent elevations in PCS, particularly if their clinical presentation includes indicators of more severe injury, such as loss of consciousness and neuroimaging abnormalities.

However, Yeates et al. (2009) have also described a subgroup of children, equally comprised of children orthopedic injury and mild TBI, who demonstrate a symptom

trajectory characterized by moderate, persistent elevations in PCS. This finding suggests that, at least for some children, persistent PCS may be associated with factors unrelated to brain insult. Several authors have suggested that injury-related factors not directly associated with TBI may contribute to PCS. For example, acute or chronic pain is common following mild TBI, particularly when accompanied by associated bodily injuries, and pain is known to be associated with a variety of cognitive, emotional, and behavioral symptoms (Beaupre, De Guise, McKerral, 2012). Additionally, the experience of symptoms of post-traumatic stress disorder (PTSD) following mild TBI has also been linked to increased PCS in adults (Ponsford, Cameron, Fitzgerald, Grant, Mikocka-Walus, & Schoenberger, 2012). PTSD symptoms may be more common following certain types of injuries, such as those associated with motor vehicles (Olofsson, Bunketorp, & Andersson, 2009). Thus, other injury-related factors, such as mechanism of injury, should probably be considered as potential predictors of PCS.

A variety of non-injury related factors also have been shown to be related to PCS following mild TBI, including children's pre-injury symptoms (Taylor et al., 2010), pre-injury learning or psychiatric problems (Ponsford et al., 1999), pre-injury cognitive ability (Fay et al., 2010), and coping strategies (Woodrome et al., 2011). Furthermore, factors such as family functioning, parental psychological adjustment, and availability of resources and stressors may also be related to outcome in children following mild TBI (Ponsford et al., 2000; Yeates et al., 2012).

Although both injury and non-injury related factors appear to contribute to PCS following mild TBI in children, the relative importance of these factors remains unclear. In the present study, using data from the study by Yeates and colleagues (Yeates et al., 2009, in press; Taylor et al., 2010), we examined the relative contributions of injury and non-injury related factors to the prediction of PCS as a function of time since injury. We hypothesized that injury factors would be predictive of PCS occurring shortly after an injury, but that the relative contribution of injury factors would decline over time. We also hypothesized that premorbid child and family factors would predict PCS shortly after injury, and that the relative contribution of those factors would increase over time post-injury, as the contribution of injury-related factors declined.

Method

Participants

Participants were recruited from consecutive admissions to the emergency departments at Nationwide Children's Hospital in Columbus, Ohio and Rainbow Babies & Children's Hospital in Cleveland, Ohio. All 8 to 15 year-old children presenting with closed-head trauma or orthopedic injuries (OI) were screened for eligibility.

For the mild TBI group, inclusion criteria included sustaining a blunt head trauma associated with one of the following three features: loss of consciousness (no longer than 30 minutes); Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) score of 13 or 14; or at least two acute signs or symptoms of concussion as noted by emergency department medical personnel. Acute signs and symptoms included posttraumatic amnesia, vomiting, nausea, headache, diplopia, dizziness, disorientation, or other mental status changes. Exclusion criteria included any GCS score below 13, delayed neurologic deterioration, or medical contraindication to MRI. Children who were hospitalized, or those with intracranial lesions or skull fractures, were not excluded from the study.

Children were included in the OI group if they sustained a fracture associated with an Abbreviated Injury Scale (AIS; American Association for Automotive Medicine, 1990) score

of 3 or less (i.e., injuries classified as mild, moderate or serious). Children with any evidence of head injury, including external trauma or concussive symptoms, were not eligible for the OI group.

Additional exclusion criteria applied to both groups included: neurosurgical or surgical intervention; any associated injury with AIS score > 3 (i.e., injuries classified as severe or critical); any associated injury that would interfere with neuropsychological testing (i.e. dominant upper extremity injury); hypoxia, hypotension, or shock; drug or alcohol ingestion associated with the injury; or premorbid severe psychiatric disorder requiring hospitalization. We also excluded children who sustained any injury resulting from child abuse or assault, as these injuries may be associated with different underlying pathology and may be associated with distinct psychosocial factors. Children with premorbid learning or attention problems were not excluded.

Among children meeting all inclusion/exclusion criteria, the participation rate was 48% for the mild TBI group and 35% for the OI group. Participants and non-participants did not significantly differ in age, sex, race, ethnicity, or census tract measures of socioeconomic status (SES).

The final sample included 74 children who sustained a mild TBI associated with loss of consciousness (LOC), 112 children who sustained a mild TBI with no LOC, and 99 children with an OI. The groups did not differ with respect to age, sex, minority status, or IQ as assessed shortly after injury (see Table 1). They also did not differ in premorbid child or family functioning. Children with mild TBI and LOC displayed the most severe injuries, were more likely to have associated injuries than children with mild TBI without LOC, and were more likely to have sustained injuries involving motor vehicles than children with mild TBI without LOC or those with OI.

Procedure

The research was approved by the institutional review boards of Nationwide Children's Hospital and Rainbow Babies & Children's Hospital, and informed parental consent and child assent were obtained in writing before participation. Those who agreed to participate completed an initial assessment within 3 weeks of the injury; 80% of the initial assessments were conducted between 1 and 2 weeks post-injury. Of the 285 children who completed the initial assessment, 89% remained in the study at the 12 month time point. Attrition rates did not differ by group. The children who did not complete the study were more likely to be of minority status and lower SES but did not differ in other demographic or clinical characteristics.

Measures

Post-concussive symptoms—PCS were assessed using the Heath and Behavior Inventory (HBI; Yeates et al., 1999), as well as the Post-Concussive Symptom Interview (PCS-I; Mittenberg, Wittner, & Miller, 1997). At the initial assessment, parents were asked to provide retrospective ratings of premorbid symptoms. We did not obtain child ratings of premorbid symptoms, because we were not confident that children could provide valid ratings of pre-injury symptoms; however, this may be an important issue to consider in future studies. At all post-injury assessments, both parents and children provided ratings of current symptoms.

On the HBI, parents or children are asked to rate somatic, cognitive, affective, and behavioral symptoms on a 4-point scale ranging from 'never' to 'often.' In a previous analysis, the 50 items were subjected to factor analysis, resulting in two underlying

dimensions representing cognitive symptoms and somatic symptoms (Ayr, Yeates, Taylor, Browne, 2009). Both parent and child ratings demonstrated acceptable internal consistency across time points (Cronbach's alpha from .83 to .95 for parent ratings and from .86 to .91 for children's ratings).

The PCS-I is an orally administered measure in which parents or children are asked to report the presence or absence of 15 cognitive, somatic, and emotional symptoms. The total score is the number of PCS endorsed. Both parent and child ratings demonstrated acceptable internal consistency across time points (Cronbach's alpha from .78 to .82 for parent ratings and from .70 to .77 for children's ratings).

Demographics—Demographic information was collected during the initial assessment. A socioeconomic composite index was computed by averaging sample z scores for years of maternal education, median family income for census tract, and the Duncan Socioeconomic Index, which is a measure of occupational prestige (Stevens & Cho, 1985).

Pre-morbid child factors—Parental ratings of children's premorbid behavioral adjustment were obtained at the initial assessment using the *Child Behavior Checklist* (CBC; Achenbach, 1991). T-scores for total problems as well as total competence were used in analyses. In addition, assessments of children's reading skills, and cognitive ability (i.e., IQ) were completed at this time. The measures of reading skills and cognitive ability were considered to represent premorbid functioning, because neither is likely to be sensitive to the effects of mild TBI (Ponsford et al., 1999). Reading skills were assessed using the standard score from the *Word Reading* subtest of the Wide Range Achievement Test-Third Edition (WRAT-3; Wilkinson, 1993) and general intellectual functioning was assessed using the Full Scale IQ from the Wechsler Abbreviated Scale of Intelligence (WASI; The Psychological Corporation, 1999).

Premorbid family factors—At the initial assessment, parents were asked to retrospectively rate family functioning, family stressors and resources, and parental psychological adjustment. The 12-item General Functioning scale from the *McMaster Family Assessment Device* (FAD; Miller, Bishop, Epstein & Keitner) was used to assess family adjustment. Premorbid family status was also assessed using the *Life Stressors and Social Resources Inventory* (LSSRI; Moos & Moos, 1988). This is an interview about a variety of stressors (e.g. physical health stressors, work stressors, spouse/partner stressors, negative life events) and resources (e.g. financial resources, friend resources, positive life events). Total standard scores for resources and stressors were included in the current analyses. Parental psychosocial adjustment was assessed using the General Severity Index from the *Brief Symptom Inventory* (BSI; Derogatis & Melisaratos, 1983).

Injury characteristics—Participants were grouped based on injury status, with the classification of children with mild TBI taking into account the presence/absence of both loss of consciousness (LOC) and associated injuries (AI). Thus, five injury groups were created: OI; mild TBI with LOC and AI; mild TBI with LOC but without AI; mild TBI without LOC but with AI; mild TBI without LOC or AI. Among children with LOC, the duration was typically brief (median = 1 minute; range = 1–15 minutes). Injury severity was also assessed using the Modified Injury Severity Scale (MISS; Mayer, Matlak, Johnson & Walker, 1980). Finally, mechanism of injury was also considered, by classifying children according to whether their injuries involved a motor vehicle (i.e., were sustained as an occupant in a motor vehicle collision or when struck by a motor vehicle as pedestrians or while bicycling).

Data Analysis

A series of hierarchical multiple regression analyses were conducted to predict parent and child report of number of symptoms on the PCS-I, as well as somatic and cognitive symptom ratings on the HBI. Separate analyses were conducted for the initial, post-acute assessment and the assessments at 1, 3, and 12 months post-injury. Thus, a total of 24 separate regression analyses were conducted (two raters; three dependent variables; four occasions). In each analysis predictors were entered hierarchically; step 1 included demographic factors (child age, child sex, child minority status, and socioeconomic status); step 2 included premorbid child factors (WASI Full Scale IQ, WRAT-3 Reading standard score, CBC Total Problems & Competence T scores, & parent premorbid symptom rating); step 3 included premorbid family factors (BSI Total T score, total Stressors and Resources scores from the LSSRI, & FAD General Functioning score); and step 4 included injury factors (dummy variables for group membership and mechanism of injury, as well as MISS). Multicollinearity diagnostics were examined, with a Variance Inflation Factor (VIF) greater than 2.5 and a tolerance under .4 considered problematic (O'Brien, 2007). Based on these criteria, the MISS and mechanism of injury variables were identified as problematic, because of their substantial correlation with group membership. Thus, they were removed from the regression analyses. Multicollinearity diagnostics were acceptable for all other variables.

The initial hierarchical regression analyses were followed by additional analyses to determine if the presence of LOC or the presence of AI explained unique variance in PCS. For each dependent variable, two additional regression models were tested. Steps 1–3 included the same variables as above (step 1, demographic variables; step 2, child factors; step 3, family factors), and steps 4 and 5 included dummy variables coded such that that one analysis assessed the unique variance in PCS explained by presence of LOC and the s analysis assessed the unique variance explained by presence of AI.

To determine if the relative contributions of individual predictors changed significantly over time, mixed model analyses were also conducted. Six mixed models were examined (2 raters; 3 dependent variables), with each predicting symptom ratings from the total set of predictors over time.

Data analyses were completed using all available data (i.e. including all children available at each occasion) and also using only participants for whom complete data were available. The results of the two sets of analyses did not differ substantially. Thus, analyses that used all available data are presented.

Results

PCS-I Total Score

Parent Ratings—As shown in Table 2, demographic variables and premorbid child and family factors consistently predicted parent ratings of post-concussive symptom counts at each assessment. In contrast, injury group predicted parent-reported symptom counts post-acute and at 1 month post-injury, but not at later times.

Table 3 shows the regression models at the final step in the hierarchical regression analysis for each time point. Child age (younger children with more symptoms), sex (females with more symptoms), and race (non-white participants with more symptoms) predicted parent reported post-concussive symptom counts at different time points. Premorbid child adjustment on the *CBC* also predicted parent-reported PCS; children with more baseline adjustment difficulties and lower competency scores were rated as having more symptoms. Ratings of pre-injury symptoms were the strongest predictor of post-injury symptoms across

time points. Parent distress on the *BSI* was a positive predictor of parent-reported symptom counts at most time periods. At the post-acute time point, children in all TBI groups, with the exception those without LOC but positive for AI, had higher parent-reported symptom counts than children with OI. Only children with mild TBI that involved both LOC and AI had higher parent-rated symptom counts at 1 month and 3 months post-injury. By the 12 month time point, none of the TBI groups differed from the OI group. Additional regression analyses revealed that the presence of LOC as well as the presence of AI accounted for unique variance in explaining parent-reported PCS post-acutely and at 1 month, but not at later time points.

The mixed model analysis revealed significant main effects for age ($t = -2.84, p < .01$), sex ($t = 3.26, p < .01$), *CBC* Total Problems ($t = 2.48, p = .01$), *CBC* Competency ($t = -2.81, p < .001$), pre-morbid symptom ratings ($t = 4.47, p < .001$), *BSI* Total ($t = 4.09, p < .001$), and injury group (mild TBI with -LOC and -AI vs. OI: $t = 3.13, p < .01$; mild TBI with -LOC and +AI vs. OI: $t = 2.10, p < .05$; mild TBI with +LOC and -AI vs. OI: $t = 3.17, p < .01$; mild TBI with +LOC and +AI vs. OI: $t = 3.98, p < .001$). In most cases, the relationship between injury group and parent-reported PCS symptom counts significantly declined over time (time by group interactions, mild TBI with -LOC and -AI vs. OI: $t = -2.01, p < .05$; mild TBI with -LOC and +AI vs. OI: $t = -1.2, p = .23$; mild TBI with +LOC and -AI vs. OI: $t = -2.0, p < .05$; mild TBI with +LOC and +AI vs. OI: $t = -1.84, p = .06$). No other variables showed a significant interaction with time, reflecting stable relationships with parent-rated post-concussive symptoms over time.

Child Ratings—Similar to the parent ratings of symptom counts, demographic factors and premorbid child factors significantly predicted symptoms across time points (Table 2). However, family factors were not associated with child-reported symptom counts. Injury group predicted children's ratings of symptoms post-acutely and at 1 month post-injury, but not at later times.

When looking at the individual variables at the final step in the regression models (Table 4), sex was the strongest demographic predictor across time points, with females reporting more symptoms. Retrospective parent ratings of pre-injury symptoms significantly predicted child-reported post-injury symptoms at most time points. At the post-acute time point, children in all mild TBI groups, with the exception of those without LOC or AI, had higher self-reported symptom counts than children with OI. In contrast, at 3 and 12 months post-injury, none of the injury groups differed significantly from the OI group. Additional regression analyses revealed that presence of LOC, but not AI, explained unique variance in self-reported PCS symptom counts post-acutely and at 1 month post-injury, but not at later time points.

The mixed model analysis revealed significant main effects for *CBC* Total Problems ($t = 2.30, p = .02$), premorbid symptom ratings ($t = 2.66, p < .01$), and injury group, with the exception of children with mild TBI and AI but no LOC (mild TBI with -LOC and -AI vs. OI: $t = 2.74, p < .01$; mild TBI with -LOC and +AI vs. OI: $t = 0.99, p = .32$; mild TBI with +LOC and -AI vs. OI: $t = 2.86, p < .01$; mild TBI with +LOC and +AI vs. OI: $t = 2.17, p < .05$). No variables showed significant interactions with time.

HBI Somatic Symptoms

Parent Ratings—As shown in Table 2, demographic variables and family factors contributed to a small but significant proportion of variance in parent ratings of somatic symptoms at most, but not all, time points. Premorbid child factors consistently predicted parent ratings of somatic symptoms across times. Injury group predicted parent-ratings of somatic symptoms post-acutely and at 1 month post injury, but not at later times.

Table 5 shows the individual variables at the final step of the regression analyses across time points. For demographic variables, non-white race and female sex were positively related to somatic symptom ratings at different time points. WASI IQ score was related to somatic symptom ratings at some time points, with higher IQ associated with fewer somatic symptoms, post-acutely and at 1 month post-injury. Retrospective ratings of pre-injury somatic complaints significantly predicted post-injury somatic complaints at most time points. Family variables, including parental distress on the *BSI* and presences of stressors on the *LSSRI*, were positively related to parent-reported somatic symptoms at different time points. Children in all mild TBI groups had higher parent ratings of somatic symptoms than children with OI at the post-acute time point. Children with mild TBI who had AI also differed from the OI group at the 1 month time point, whether or not they also had LOC. By 3 months post injury, only the mild TBI group without LOC but with AI differed from the OI group. Additional regression analyses revealed that at the post-acute time point, both LOC and AI explained unique variance in parent-reported somatic symptoms. At 1 and 3 months post-injury, the presence of AI, but not LOC, explained unique variance.

The mixed model analysis revealed significant main effects of sex ($t = 2.32, p = .02$), WASI IQ ($t = -2.53, p < .02$), pre-morbid somatic symptom ratings ($t = 4.01, p < .001$), *BSI* Total score ($t = 4.17, p < .001$), and injury group (mild TBI with -LOC and -AI vs. OI: $t = 4.05, p < .001$; mild TBI with -LOC and +AI vs. OI: $t = 4.01, p < .001$; mild TBI with +LOC and -AI vs. OI: $t = 3.56, p < .001$; mild TBI with +LOC and +AI vs. OI: $t = 3.51, p < .001$). For most groups, the relationship between injury group and parent-reported somatic symptoms declined significantly over time (time by group interactions for mild TBI with -LOC and -AI vs. OI: $t = -4.36, p < .001$; mild TBI with -LOC and +AI vs. OI: $t = -1.62, p = .10$; mild TBI with +LOC and -AI vs. OI: $t = -2.28, p = .02$; mild TBI with +LOC and +AI vs. OI: $t = -2.20, p = .03$). The relationship between premorbid somatic symptoms and post-injury somatic symptoms also varied as a function of time, with the relationship strengthening (time by pre-morbid symptom interaction, $t = 2.69, p < .01$). No other variables showed a significant interaction with time.

Child Ratings—Demographic and pre-injury child factors consistently predicted child-reported somatic symptoms across time points (Table 2). Family factors did not predict child-ratings of somatic symptoms at any time. Injury group predicted child ratings post-acutely, but not at later time points.

Table 6 shows the individual variables at the final step of the regression analyses predicting child ratings of somatic symptoms across time points. Sex was the strongest demographic predictor, with girls reporting more somatic symptoms across time points. WASI IQ was also a significant negative predictor of child-reported somatic symptoms at some time points. Retrospective ratings of pre-injury somatic complaints significantly predicted post-injury somatic complaints at each time point. At the post-acute time point, children in all mild TBI groups, with the exception of those without LOC or AI, had higher self-reported somatic symptoms than children with OI. In contrast, at later time points, none of the mild TBI groups differed from the OI group. Additional analyses revealed that at the post-acute time point, but not at later time points, the presence of both LOC and AI accounted for unique variance in self-reported somatic symptoms.

The mixed model analysis revealed a significant main effect of sex ($t = 2.36, p = .02$) premorbid somatic symptom ratings ($t = 2.82, p < .01$) and injury group, but only when comparing those with LOC to the OI group (mild TBI with -LOC and -AI vs. OI: $t = 1.83, p = .07$; mild TBI with -LOC and +AI vs. OI: $t = 0.54, p = .54$; mild TBI with +LOC and -AI vs. OI: $t = 3.02, p < .01$; mild TBI with +LOC and +AI vs. OI: $t = 1.96, p = .05$). The relationship between injury group and child-reported somatic symptoms significantly

declined as a function of time but only when comparing those without LOC or AI to the OI group (time by group interactions for mild TBI with –LOC and –AI vs. OI: $t = -2.21$, $p < .05$; mild TBI with –LOC and +AI vs. OI: $t = -.063$, $p = .53$; mild TBI with +LOC and –AI vs. OI: $t = -1.45$, $p = .15$; mild TBI with +LOC and +AI vs. OI: $t = -1.78$, $p = .08$). No other variables showed a significant interaction with time, indicating stable relationships with child-rated somatic symptoms over time.

HBI Cognitive Symptoms

Parent Ratings—As shown in Table 2, demographic variables consistently predicted parent report of cognitive symptoms at each time point. Premorbid child factors accounted for a significant and large proportion of the variance across times. Family factors and injury factors significantly predicted parent reported cognitive symptoms post-acutely and at 3 months post-injury, but not at other time points.

As shown in Table 7, retrospective ratings of pre-injury cognitive symptoms were the strongest predictor of parent reported cognitive symptoms across time points. *WASI*Q and child adjustment on the *CBC* were significant predictors at some, but not all, time points. Parent distress on the *BSI* significantly predicted parent report of cognitive symptoms at most time points. Children with mild TBI and LOC but without AI had higher parent ratings of cognitive symptoms than children with OI at all occasions. The mild TBI group with both LOC and AI had higher parent ratings of cognitive symptoms post-acutely and at 3 months. In contrast, children with mild TBI but without LOC did not differ from the OI group in parent-rated cognitive symptoms at any occasion, regardless of whether they had AI. Additional analyses revealed that LOC accounted for unique variance in parent-rated cognitive symptoms at most time points (post-acute, 1 month, and 12 months). The presence of AI explained unique variance post-acutely and at 3 months.

The mixed model analysis revealed a significant main effect of *CBC* Competency ($t = -3.74$, $p < .001$), pre-morbid cognitive symptom ratings ($t = 13.46$, $p < .001$), *BSI* Total ($t = 4.33$, $p < .001$), and injury group, but only when comparing those with LOC to the OI group (mild TBI with –LOC and –AI vs. OI: $t = 1.46$, $p = .14$; mild TBI with –LOC and +AI vs. OI: $t = -0.15$, $p = .88$; mild TBI with +LOC and –AI vs. OI: $t = 3.93$, $p < .001$; mild TBI with +LOC and +AI vs. OI: $t = 3.81$, $p < .001$). The relationship between premorbid cognitive symptoms and post-injury cognitive symptoms varied as a function of time, with the strength of the relationship declining (time by pre-morbid cognitive symptom interaction $t = -2.70$, $p < .01$). No other variables showed a significant interaction with time.

Child Ratings—As shown in Table 2, demographic variables accounted for a significant proportion of variance in child ratings of cognitive symptoms at most time points. Premorbid child factors also accounted for a significant proportion of the variance in child-reported cognitive symptoms at each time point. Family factors and injury factors did not significantly predict cognitive symptoms at any time.

Table 8 shows the individual variables at the final step of the regression analyses predicting child ratings of cognitive symptoms at each time point. Female gender and minority status were significantly associated with child-reported cognitive symptoms across time points. Premorbid child factors, including competency on the *CBC* and *WASI*Q, were negatively associated with symptoms at some time points. Retrospective ratings of pre-injury cognitive symptoms significantly predicted post-injury cognitive symptoms post-acutely and at 1 month post-injury. Regardless of LOC or AI status, children with mild TBI did not differ from the OI group in self-report of cognitive symptoms at any occasion. Similarly, additional regression analyses showed that neither LOC nor AI accounted for unique variance in self-reported cognitive symptoms at any time point.

The mixed model analysis revealed significant main effects for age ($t = 2.20$, $p = .02$), race ($t = -2.10$, $p < .05$), *CBC* competency ($t = -2.73$, $p < .01$) and premorbid cognitive symptom ratings ($t = 2.31$, $p = .02$). No variables showed a significant interaction with time.

Discussion

Consistent with our primary hypothesis, injury characteristics predicted parent and child ratings of PCS, but tended to make a decreasing contribution over time. Non-injury related child and family factors were also consistently related to PCS ratings; contrary to our predictions, however, their contribution generally remained stable rather than increasing over time. Demographic variables consistently predicted symptom ratings across time. Premorbid child factors, especially retrospective ratings of premorbid symptoms, accounted for the most variance in symptom ratings. Family factors, particularly parent adjustment, consistently predicted parent, but not child, ratings of PCS.

This study provides further evidence that injury characteristics are important in predicting PCS, particularly in the first month after mild TBI. This finding is consistent with recent studies asserting that injury severity is a significant factor in predicting acute outcomes following mild TBI, and contrasts with assertions that mild TBI is not associated with demonstrable increases in PCS (e.g. Carroll et al, 2004). The latter conclusion may be attributable to methodological factors, most notably related to the definition of mild TBI, which has varied across studies (Yeates, 2010). Specifically, many studies have excluded children with more severe injuries, or those that may be considered to have a “complicated” mild TBI (i.e., children with intracranial lesions or skull fractures). Studies that have included children with more severe forms of mild TBI tend to report more pronounced and persistent differences in PCS as compared to controls (Mittenberg, Wittner, & Miller, 1997; Taylor et al., 2010; Yeates et al., 1999, 2009, in press).

As expected, the relative contribution of injury factors in predicting somatic PCS and total symptom counts declined over time. The relationship was less consistent when predicting ratings of cognitive PCS, for which mild TBI associated with LOC was consistently associated with more symptoms at all occasions according to parent report. At 3 months post-injury and beyond, however, demographic variables and premorbid child and family factors made a greater contribution to predicting PCS than did injury-related factors. Thus, when evaluating and treating children who demonstrate persistent PCS, clinicians should pay close attention to factors that may elicit symptoms for reasons other than an underlying brain injury (Kirkwood et al., 2008).

Although underlying brain injury may be one explanation for PCS, particularly in the first weeks following the injury, other injury-related factors associated may also play an important role. Indeed, the present results showed that, in many cases and particularly for reports of somatic symptoms, the presence of an associated bodily injury in addition to mild TBI accounted for unique variance in PCS, above and beyond LOC. Although pain ratings were not obtained in the present study, the experience of acute or chronic pain associated with associated injuries may have contributed to increased PCS (Beaupre, De Guise, McKerral, 2012). Mechanism of injury may also be an important factor to consider, as motor-vehicle related trauma may place children at higher risk for PTSD symptoms; however, disentangling these influences was not possible in the current study because mechanism of injury was too highly correlated with injury group.

In keeping with past studies, non-injury related child characteristics predicted PCS across time. Retrospective ratings of premorbid symptoms were generally the strongest predictor of post-injury symptoms. This finding highlights the importance of obtaining retrospective

ratings of pre-injury symptoms as soon as possible after an injury and carefully considering this information when interpreting current symptom reports (Kirkwood et al., 2008). Pre-injury symptom ratings that are collected during the acute evaluation of a child with a mild TBI may contribute to understanding premorbid factors that may lead to longer recovery and also be used by clinicians to understand when a child has returned to his or her typical level of functioning. Other non-injury background characteristics that should be considered when evaluating children with PCS include gender, race, and age. Girls tended to report more PCS, particularly related to somatic complaints, and younger children tended to have higher symptom counts. In some cases, ethnic minorities also reported more PCS. Children with lower IQs also tended to report more PCS, particularly more somatic complaints. This is consistent with previous reports that children with lower IQs are particularly prone to increased PCS and may be related to differences in cognitive reserve capacity (Fay, Yeates, Taylor, Bangert, Dietrich, Nuss, Russ & Wright, 2010). In other words, children with lower cognitive ability may have a reduced capacity to cope with neurological insults, as they may have a diminished capacity to recruit alternative resources following injury.

Family factors, specifically parental psychological adjustment, were related to parent, but not child, report of PCS. High levels of parental distress could lead to an exaggeration, or over reporting, of their child's symptoms. Alternatively, this finding may be consistent with previous research suggesting that the family environment is a critical determinant of children's functioning following childhood TBI (Taylor, Barry, Schatschneider, 1993; Yeates et al., 1997). However, in this sample, other family factors, including general family functioning, life stressors, and social resources, were generally not related to PCS.

This study has several methodological strengths, including clear criteria for defining mild TBI, use of a control group with injuries not involving the head, and a prospective, longitudinal design. However, the study is not without limitations. Although we sought to determine the role of premorbid child and family factors in predicting PCS, retrospective ratings were used to estimate premorbid functioning. To reduce bias, however, ratings were collected as soon as feasible following the injury. An additional limitation is that the current study was only focused on premorbid child and family factors, rather than considering how these factors may vary post-injury. Child and family factors such as parent and child adjustment, family functioning, and stressors and resources can change over time, and this change may be moderated by the injury itself. For example, significant acute PCS may lead to increases in distress and impaired family functioning, which in turn may exacerbate PCS. Thus, future research should focus on the reciprocal interactions between mild TBI, PCS, and child and family adjustment over time.

In conclusion, the current study adds to the existing literature and our previous reports by showing that injury characteristics play a role in predicting post-concussive symptoms, but that the relative contribution associated with injury severity tends to decline over time. The study also shows that non-injury child and family factors play a major role in predicting PCS, and that much of the variance in PCS is accounted for by premorbid symptoms. Future research is needed to further characterize the injury as well as non-injury related factors that influence outcome following mild TBI, and particularly those factors that contribute to persistent PCS. Such studies will help to foster an integrative model of risk and resiliency following mild TBI that will enable clinicians to target interventions to children at risk of poor outcomes.

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Table 1

Demographic and baseline clinical characteristics

	Mild TBI with LOC	Mild TBI no LOC	OI	<i>p</i>
	(N = 74)	(N = 112)	(N = 99)	
Demographics				
Age	12.15 (2.20)	11.83 (2.23)	11.76 (2.23)	.49
% Male	77.02	66.96	64.64	.19
% White	74.43	72.32	64.64	.31
SES (Z-score)	.15 (.97)	-.02(.87)	-.9 (1.1)	.27
Premorbid Child Factors				
WASI Full Scale IQ	100.09 (14.16)	99.38 (13.66)	98.90 (15.01)	.86
WRAT-3 Reading	101.46 (13.11)	99.80 (15.42)	98.39 (16.87)	.43
CBC Total Problems	48.73 (9.99)	49.97 (9.82)	50.45 (12.67)	.58
CBC Competence	49.58 (10.09)	47.76 (10.50)	45.88 (9.21)	.06
Total premorbid PCS, CSI	1.19 (1.67)	1.04 (1.65)	1.13 (2.06)	.85
Premorbid cognitive PCS	11.23 (7.59)	11.73 (7.78)	10.92 (7.59)	.74
Premorbid somatic PCS	3.14 (3.23)	3.53 (3.43)	2.46 (3.16)	.06
Premorbid Family Factors				
BSI	49.05 (10.18)	49.25 (9.52)	49.43 (11.57)	.97
LSSRI Stressors	50.18 (6.76)	50.17 (6.76)	49.85 (6.90)	.93
LSSRI Resources	52.52 (6.09)	52.56 (6.34)	50.73 (7.09)	.09
FAD General functioning	1.61 (.44)	1.61 (.35)	1.71 (.42)	.10
Injury Factors				
% With Associated Injuries	33.78	19.64	--	.00
% Motor Vehicle Related	20.17	10.71	2.02	.00
MISS	6.27 (5.19)	3.36 (3.50)	3.17 (1.50)	.00

Data are percentages or mean (standard deviation); TBI = traumatic brain injury; SES = socioeconomic status; WASI = Wechsler Abbreviated Scale of Intelligence; WRAT = Wide Range Achievement Test; CBC = Child Behavior Checklist; PCS = Post-concussive symptoms; CSI = Concussion Symptom Inventory; BSI = Brief Symptom Inventory; LSSRI = Life Stressors and Social Resources Inventory; FAD = Family Assessment Device; MISS = Modified Injury Severity Scale

Table 2

Step-wise Multiple Regression Models at Each Time: Change in R squared

	Post-Acute	1 Month	3 Months	12 Months
Post-concussive symptom counts: Parent rating				
Step 1: Demographics	.03*	.05*	.07*	.04*
Step 2: Child Factors	.10*	.24*	.19*	.12*
Step 3: Family Factors	.06*	.03*	.05*	.03*
Step 4: Injury Factors	.07*	.02*	.01	.01
Total R square	.27	.34	.32	.20
Post-concussive symptom counts: Child rating				
Step 1: Demographics	.04*	.07*	.09*	.08*
Step 2: Child Factors	.10*	.11*	.07*	.10*
Step 3: Family Factors	.01	.01	.00	.02
Step 4: Injury Factors	.04*	.02*	.01	.00
Total R square	.18	.22	.17	.19
HBI somatic symptoms: Parent rating				
Step 1: Demographics	.03*	.02	.04*	.02
Step 2: Child Factors	.07*	.19*	.22*	.30*
Step 3: Family Factors	.06*	.05*	.06*	.02
Step 4: Injury Factors	.11*	.02*	.01	.01
Total R square	.27	.28	.33	.35
HBI somatic symptoms: Child rating				
Step 1: Demographics	.04*	.08*	.06*	.05*
Step 2: Child Factors	.10*	.10*	.06*	.13*
Step 3: Family Factors	.02	.01	.01	.03
Step 4: Injury Factors	.06*	.01	.00	.01
Total R square	.21	.20	.13	.21
HBI cognitive symptoms: Parent rating				
Step 1: Demographics	.04*	.06*	.10*	.06*
Step 2: Child Factors	.44*	.47*	.43*	.33*
Step 3: Family Factors	.03*	.01	.02*	.01
Step 4: Injury Factors	.03*	.01	.03*	.01
Total R square	.54	.55	.58	.41
HBI cognitive symptoms: Child rating				
Step 1: Demographics	.07*	.07*	.05*	.03
Step 2: Child Factors	.13*	.18*	.10*	.10*
Step 3: Family Factors	.01	.01	.00	.02
Step 4: Injury Factors	.01	.00	.00	.02
Total R square	.22	.26	.15	.17

Table 3

Parent Ratings: Post-Concussive Symptom Counts

		Post-acute		1 month		3 months		12 month	
Predictor		Beta		Beta		Beta		Beta	
Step 1: Demographics	Age	-.03		-.14*		-.13*		-.11	
	Sex	-.11*		-.13*		-.15*		-.10	
	Race	.18*		.02		.03		-.03	
	SES	.03		.09		.02		-.04	
Step 2: Child Factors	WASI Full Scale IQ	-.15		-.12		-.05		-.14	
	WRAT-R	.09		.07		-.07		.02	
	CBC Total Problems	.08		.13		.19*		.17*	
	CBC Competence	-.14*		-.16*		-.05		.09	
Step 3: Family Factors	# of premorbid PCS	.14*		.30*		.17*		.20*	
	BSI	.25*		.20*		.12		.15*	
	LSSRI Stressors	.05		-.02		.14*		-.03	
	LSSRI Resources	.01		-.02		.14*		.08	
Step 4: Injury Factors	FAD General Functioning	-.09		.01		.08		.10	
	OI vs. mild TBI + LOC and +AI	.23*		.14*		.11*		.08	
	OI vs. mild TBI + LOC and -AI	.23*		.11		.10		.04	
	OI vs. mild TBI - LOC and +AI	.08		.07		.07		.02	
	OI vs. mild TBI - LOC and -AI	.23*		.06		.11		.00	

SES = socioeconomic status; WASI = Wechsler Abbreviated Scale of Intelligence; WRAT = Wide Range Achievement Test; CBC = Child Behavior Checklist; PCS = Post-concussive symptoms; CSI = Concussion Symptom Inventory; BSI = Brief Symptom Inventory; LSSRI = Life Stressors and Social Resources Inventory; FAD = Family Assessment Device; OI = orthopedic impairment; TBI = traumatic brain injury; LOC = loss of consciousness; AI = associated injury.

Table 4

Child Ratings- Post-Concussive Symptom Counts

		Post-acute			1 month			3 months			12 months		
Predictor		Beta			Beta			Beta			Beta		
Step 1: Demographics	Age	.01			-.17*			-.18*			-.11		
	Sex	-.14*			-.19*			-.17*			-.27*		
	Race	.04			.06			.03			.04		
	SES	-.01			.03			-.01			.11		
Step 2: Child Factors	WASI Full Scale IQ	-.11			-.06			-.05			-.12		
	WRAT-R	.02			.06			-.04			-.06		
Step 3: Family Factors	CBC Total Problems	.17*			.14			.13			.15		
	CBC Competence	-.02			-.13			-.13			.02		
	# of premorbid PCS	.15*			.19*			.10			.16*		
	BSI	.05			.10			.01			.10		
Step 4: Injury Factors	LSSRI Stressors	.06			-.03			-.01			.01		
	LSSRI Resources	.02			-.01			.06			.10		
	FAD General Functioning	-.07			-.05			.04			-.01		
	OI vs. mild TBI + LOC and +AI	.15*			.11			.00			.02		
	OI vs. mild TBI + LOC and -AI	.17*			.16*			.06			.05		
	OI vs. mild TBI -LOC and +AI	.05			.03			.05			.03		
	OI vs. mild TBI -LOC and -AI	.18*			.07			.10			.02		

SES = socioeconomic status; WASI = Wechsler Abbreviated Scale of Intelligence; WRAT = Wide Range Achievement Test; CBC = Child Behavior Checklist; PCS = Post-concussive symptoms; CSI = Concussion Symptom Inventory; BSI = Brief Symptom Inventory; LSSRI = Life Stressors and Social Resources Inventory; FAD = Family Assessment Device; OI = orthopedic impairment; TBI = traumatic brain injury; LOC = loss of consciousness; AI = associated injury.

Table 5

HBI Somatic Symptoms: Parent Ratings

Predictor	Post-acute			12 months		
	Beta	1 month	Beta	Beta	3 months	Beta
Step 1: Demographics						
Age	.03	-.02			-.09	.01
Sex	-.13*	-.08			-.08	-.08
Race	.16*	.11			.05	.12*
SES	-.05	.06			.01	-.01
Step 2: Child Factors						
WASI Full Scale IQ	-.16*	-.19*			-.09	-.08
WRAT-R	.08	.04			-.08	-.11
CBC Total Problems	.07	-.01			.16*	.15*
CBC Competence	-.01	-.08			.03	.10
Premorbid Somatic Symptoms	.07	.31*			.24*	.42*
Step 3: Family Factors						
BSI	.19*	.24*			.10	.13*
LSSRI Stressors	.12*	-.02			.21*	-.04
LSSRI Resources	.04	-.07			.12	.08
FAD General Functioning	-.03	.04			.04	.10
Step 4: Injury Factors						
OI vs. mild TBI + LOC and +AI	.26*	.12*			.05	.05
OI vs. mild TBI + LOC and -AI	.30*	.07			.11	.05
OI vs. mild TBI - LOC and +AI	.15*	.16*			.15*	.09
OI vs. mild TBI - LOC and -AI	.29*	.12			.09	-.10

HBI = Health and Behavior Inventory; SES = socioeconomic status; WASI = Wechsler Abbreviated Scale of Intelligence; WRAT = Wide Range Achievement Test; CBC = Child Behavior Checklist; PCS = Post-concussive symptoms; CSI = Concussion Symptom Inventory; BSI = Brief Symptom Inventory; LSSRI = Life Stressors and Social Resources Inventory; FAD = Family Assessment Device; OI = orthopedic impairment; TBI = traumatic brain injury; LOC = loss of consciousness; AI = associated injury.

Table 6

HBI Somatic Symptoms: Child Ratings

Predictor	Post-acute			12 months		
	Beta	1 month	Beta	3 months	Beta	12 months
Step 1: Demographics						
Age	-.08	-.12*	-.07	-.14		
Sex	-.12*	-.12*	-.14*	-.18*		
Race	.08	-.02	-.04	.03		
SES	.00	-.07	-.04	-.01		
Step 2: Child Factors						
WASI Full Scale IQ	-.23*	-.02	-.08	-.22*		
WRAT-R	.00	-.12	.02	.04		
CBC Total Problems	.10	.01	.09	.14		
CBC Competence	-.03	-.11	-.04	.11		
Premorbid Somatic Symptoms	.14*	.23*	.20*	.27*		
Step 3: Family Factors						
BSI	.11	.14	.02	.13		
LSSRI Stressors	-.01	-.04	-.02	-.08		
LSSRI Resources	-.04	-.00	.06	.11		
FAD General Functioning	-.10	-.02	-.01	-.06		
Step 4: Injury Factors						
OI vs. mild TBI + LOC and +AI	.17*	.07	-.03	-.01		
OI vs. mild TBI + LOC and -AI	.25*	.08	.06	.07		
OI vs. mild TBI - LOC and +AI	.06	.01	-.04	.00		
OI vs. mild TBI - LOC and -AI	.18*	-.03	.06	-.05		

HBI = Health and Behavior Inventory; SES = socioeconomic status; WASI = Wechsler Abbreviated Scale of Intelligence; WRAT = Wide Range Achievement Test; CBC = Child Behavior Checklist; PCS = Post-concussive symptoms; CSI = Concussion Symptom Inventory; BSI = Brief Symptom Inventory; LSSRI = Life Stressors and Social Resources Inventory; FAD = Family Assessment Device; OI = orthopedic impairment; TBI = traumatic brain injury; LOC = loss of consciousness; AI = associated injury.

Table 7

HBI Cognitive Symptoms: Parent Ratings

		Post-acute			12 months		
		1 month			3 months		
Predictor		Beta	Beta	Beta	Beta	Beta	Beta
Step 1: Demographics	Age	-.03	-.04	-.08	-.08	-.08	-.08
	Sex	-.07	.00	-.02	-.02	-.08	-.08
	Race	.03	.07	-.02	-.02	.03	.03
	SES	.04	.04	.00	.00	.00	.00
Step 2: Child Factors	WASI Full Scale IQ	-.02	-.04	-.07	-.07	-.19*	-.19*
	WRAT-R	-.02	.02	.01	.01	.04	.04
	CBC Total Problems	.01	.04	.10	.10	.20*	.20*
	CBC Competence	-.08	-.21*	-.13*	-.13*	.00	.00
Step 3: Family Factors	Premorbid Cognitive Symptoms	.59*	.55*	.48*	.48*	.40*	.40*
	BSI	.19*	.13*	.12*	.12*	.09	.09
	LSSRI Stressors	.04	.01	.03	.03	.00	.00
	LSSRI Resources	.01	-.02	.04	.04	.05	.05
	FAD General Functioning	-.03	-.02	.06	.06	.03	.03
Step 4: Injury Factors	OI vs. mild TBI + LOC and +AI	.15*	.07	.17*	.17*	.06	.06
	OI vs. mild TBI + LOC and -AI	.16*	.10*	.13*	.13*	.13*	.13*
	OI vs. mild TBI -LOC and +AI	-.02	.01	.04	.04	.09	.09
	OI vs. mild TBI -LOC and -AI	.07	.02	.09	.09	.05	.05

HBI = Health and Behavior Inventory; SES = socioeconomic status; WASI = Wechsler Abbreviated Scale of Intelligence; WRAT = Wide Range Achievement Test; CBC = Child Behavior Checklist; PCS = Post-concussive symptoms; CSI = Concussion Symptom Inventory; BSI = Brief Symptom Inventory; LSSRI = Life Stressors and Social Resources Inventory; FAD = Family Assessment Device; OI = orthopedic impairment; TBI = traumatic brain injury; LOC = loss of consciousness; AI = associated injury.

Table 8

HBI Cognitive Symptoms: Child Ratings

Predictor	Post-acute			12 months		
	Beta	Beta	Beta	Beta	Beta	Beta
Step 1: Demographics						
Age	.13*	.01	.01	.10		
Sex	-.11*	-.07	-.13*	-.16*		
Race	.16*	.19*	.07	.09		
SES	-.04	-.02	-.03	.09		
Step 2: Child Factors						
WASI Full Scale IQ	-.10	-.06	-.09	-.19*		
WRAT-R	-.07	-.13	.00	-.01		
CBC Total Problems	.11	.10	.08	.09		
CBC Competence	-.12	-.20*	-.20*	-.15		
Premorbid Cognitive Symptoms	.17*	.17*	.14	.11		
Step 3: Family Factors						
BSI	-.01	.11	.02	.12		
LSSRI Stressors	.06	-.06	.00	-.05		
LSSRI Resources	.00	-.01	.05	.12		
FAD General Functioning	-.05	.06	.00	-.02		
Step 4: Injury Factors						
OI vs. mild TBI + LOC and +AI	.05	.03	.02	.12		
OI vs. mild TBI + LOC and -AI	.08	.04	.02	.10		
OI vs. mild TBI -LOC and +AI	-.05	-.02	-.03	.01		
OI vs. mild TBI -LOC and -AI	.08	.05	.03	.00		

HBI = Health and Behavior Inventory; SES = socioeconomic status; WASI = Wechsler Abbreviated Scale of Intelligence; WRAT = Wide Range Achievement Test; CBC = Child Behavior Checklist; PCS = Post-concussive symptoms; CSI = Concussion Symptom Inventory; BSI = Brief Symptom Inventory; LSSRI = Life Stressors and Social Resources Inventory; FAD = Family Assessment Device; OI = orthopedic impairment; TBI = traumatic brain injury; LOC = loss of consciousness; AI = associated injury.