Journal of the International Neuropsychological Society (2010), **16**, 94–105.
Copyright © INS. Published by Cambridge University Press, 2009.
doi:10.1017/S1355617709991007

Cognitive reserve as a moderator of postconcussive symptoms in children with complicated and uncomplicated mild traumatic brain injury

TARYN B. FAY,[1,2] KEITH OWEN YEATES,[2,3] H. GERRY TAYLOR,[4,5] BARBARA BANGERT,[6] ANN DIETRICH,[2,7] KATHRYN E. NUSS,[2,7] JEROME RUSIN,[8] AND MARTHA WRIGHT[4,5]

[1]Department of Psychology, Ohio State University, Columbus, Ohio
[2]Department of Pediatrics, Ohio State University College of Medicine, Columbus, Ohio
[3]The Research Institute at Nationwide Children's Hospital, Columbus, Ohio
[4]Department of Pediatrics, Case Western Reserve University, Cleveland, Ohio
[5]Rainbow Babies and Children's Hospital, Cleveland, Ohio
[6]Departments of Radiology and Neurosurgery, University Hospitals Health System, Cleveland, Ohio
[7]Department of Emergency Medicine, Nationwide Children's Hospital, Columbus, Ohio
[8]Department of Radiology, Nationwide Children's Hospital, Columbus, Ohio

(RECEIVED March 31, 2009; FINAL REVISION September 14, 2009; ACCEPTED September 14, 2009)

Abstract

The occurrence of postconcussive symptoms (PCS) following mild traumatic brain injury (TBI) in children may depend on cognitive reserve capacity. This prospective, longitudinal study examined whether the relationship between mild TBI and PCS is moderated by cognitive ability, which served as a proxy for cognitive reserve. Participants included 182 children with mild TBI and 99 children with orthopedic injuries (OI), ranging from 8 to 15 years of age when injured. Mild TBI were classified as complicated (n = 32) or uncomplicated (n = 150) depending on whether they were associated with trauma-related intracranial abnormalities on magnetic resonance imaging. PCS were assessed initially within 3 weeks of injury, and again at 1, 3, and 12 months post injury. The initial assessment also included standardized tests of children's cognitive skills and retrospective parent ratings of pre-injury symptoms. Hierarchical linear modeling indicated that ratings of PCS were moderated jointly by cognitive ability and injury severity. Children of lower cognitive ability with a complicated mild TBI were especially prone to cognitive symptoms across time according to parents and to high acute levels of PCS according to children's self-ratings. Cognitive reserve is an important moderator of the outcomes of mild TBI in children and adolescents. (*JINS*, 2010, *16*, 94–105.)

Keywords: Nervous system trauma, Pediatric, Prospective studies, Head injury, Concussion, Head trauma, Cognitive ability

INTRODUCTION

Mild traumatic brain injuries (TBI) are common in children and adolescents. As many as 500,000 youth under the age of 15 years sustain TBI that require hospital-based medical care each year in the United States, and the large majority of these injuries are mild in severity (Bazarian et al., 2005; Kraus, 1995). As hospitalization rates for children with mild TBI decrease (Thurman & Guerrero, 1999), health care professionals need guidance to make informed decisions regarding the management of mild TBI in children (American Academy of Pediatrics, 1999). Research is therefore needed to identify children who are at risk for negative outcomes following mild TBI.

The outcomes of mild TBI in children and adolescents are controversial. Both parents and children frequently report somatic, cognitive, and emotional symptoms following mild TBI (Mittenberg, Wittner, & Miller, 1997; Rivara et al., 1994; Yeates et al., 1999), yet these postconcussive symptoms (PCS) often occur in the absence of objective evidence of brain injury, such as abnormal neuroimaging findings or deficits on standardized cognitive testing (Asarnow, et al., 1995; Bijur, Haslum, & Golding, 1990; Fay et al., 1993; Satz, 2001; Satz, Zaucha, McCleary, Light, & Asarnow, 1997). These apparently contradictory findings have engendered disputes about whether the etiology of PCS reflects "psychogenesis" or "physiogenesis" (Alexander, 1997; Lishman, 1988). Psychogenesis refers to the assertion that PCS reflect

Correspondence and reprint requests to: Keith Owen Yeates, Ph.D., Department of Psychology, Nationwide Children's Hospital, 700 Children's Dr., Columbus, OH 43205. E-mail: keith.yeates@nationwidechildrens.org

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

premorbid or post-injury psychological factors rather than any alteration in brain function, whereas physiogenesis is based on the argument that mild TBI can result in significant neuropathology that engenders PCS (Alexander, 1997). However, these explanations are not mutually exclusive. Research is needed to identify both injury and noninjury related factors that predict the risk of persistent PCS (Satz, 2001; Yeates & Taylor, 2005).

Injury severity is one factor that is potentially related to the occurrence of PCS following mild TBI in children and adolescents. Williams, Levin, & Eisenberg (1990) suggested that the presence of abnormal findings on neuroimaging, also referred to as "complicated" mild TBI, may help to improve the classification of mild TBI and prediction of outcomes. In a recent study, Levin et al. (2008) compared adolescents with complicated *versus* uncomplicated mild TBI and found that neuropsychological performance during the first year post injury was poorer among children with complicated mild TBI, despite a nearly equivalent incidence of loss of consciousness in the two groups. Recently, we showed that children with more severe mild TBI display different trajectories of PCS than children with less severe mild TBI or orthopedic injuries not involving the head (Yeates et al., 2009).

One noninjury related factor that may moderate the outcomes of mild TBI is reserve capacity, which encompasses both the passive capacity of the brain (i.e., brain reserve capacity) and the active capacity in the individual (i.e., cognitive reserve capacity) to cope with neurological insults (Dennis, Yeates, Taylor, & Fletcher, 2006; Katzman, 1993; Satz, 1993; Stern, 2002). Brain reserve capacity is a hypothetical construct that refers to the neural substrates of brain function, such as brain size, differences in brain structure, or the number or density of neurons or synapses, with functional deficits occurring when the pathology burden is such that the brain substance is reduced below a critical threshold (Dennis et al., 2006; Satz, 1993). Cognitive reserve capacity refers to individual differences in recruiting the same or alternative substrates and focuses on the functionality of brain processes (Dennis et al., 2006; Stern, 2002). This concept is not measured directly, but can be estimated by proxies such as preinjury academic functioning and intelligence, post-insult cognitive ability, level of education (in adults), socioeconomic status, and family functioning. Premorbid learning problems are another proxy for cognitive reserve, and increase the risk of memory deficits following TBI in children (Farmer et al., 2002). Developmental research shows that children of higher cognitive ability consistently demonstrate better outcomes or resilience against adversity (Masten, 2001), and childhood IQ has been shown to predict the risk of adult mental disorder (Koenen et al., 2009). However, cognitive reserve has not been examined as a moderator of PCS in children with mild TBI. The current study examined a proxy of cognitive reserve capacity using measures of cognitive abilities administered within 3 weeks post injury.

The overall purpose of the current study was to determine whether the relationship between mild TBI and PCS is moderated by cognitive reserve. The study relied on data collected as part of a larger 5-year project focused on the neurobehavioral outcomes of mild TBI in children and adolescents (Taylor et al., in press; Yeates & Taylor, 2005; Yeates et al., 2009). Participants were recruited prospectively and included children with mild TBI and a comparison group of children with mild orthopedic injuries (OI), ranging from 8 to 15 years of age when injured. Mild TBI were classified as complicated or uncomplicated depending on whether they were associated with trauma-related intracranial abnormalities on magnetic resonance imaging (MRI). Assessments of PCS were completed within 3 weeks of injury, and again at 1, 3, and 12 months post injury. Because little evidence exists for persistent cognitive deficits following mild TBI (Asarnow et al., 1995; Bijur et al., 1990; Fay et al., 1993; Satz, 2001; Satz et al., 1997), cognitive reserve capacity was assessed using standardized measures of cognitive ability administered within 3 weeks post injury. The primary study hypothesis was that cognitive ability would moderate the occurrence of PCS following mild TBI, such that differences between the mild TBI and OI groups in the severity of PCS would be greater for children of lower cognitive ability than for those with higher cognitive ability. The secondary hypothesis was that the moderating effects of cognitive reserve would be most apparent in children with complicated mild TBI. Because research has demonstrated that most PCS that occur after mild TBI tend to resolve by about 3 months post injury (Satz et al., 1997; Yeates et al., 2005), we also expected group differences in PCS to be more pronounced earlier in time, and hence to find more evidence of moderation at earlier occasions post injury.

METHOD

Participants

All children between 8 to 15 years of age who presented to the Emergency Departments of Nationwide Children's Hospital in Columbus, Ohio and Rainbow Babies and Children's Hospital in Cleveland, Ohio with head trauma or orthopedic trauma were screened on a daily basis to determine if they met criteria for participation. Children were included in the mild TBI group if they sustained a blunt head trauma resulting in an observed loss of consciousness not exceeding 30 minutes or a Glasgow Coma Scale (Teasdale & Jennett, 1974) score of 13 or 14, or if they displayed at least two acute signs or symptoms of concussion as noted by medical personnel (i.e., transient neurological deficits, persistent post-traumatic amnesia, vomiting, nausea, headache, diplopia, dizziness, disorientation, or other mental status changes). Children were excluded from the mild TBI group if they displayed delayed neurological deterioration, but not if they required hospitalization or demonstrated intracranial lesions or skull fractures on acute computerized tomography.

Inclusion criteria for the OI group consisted of upper or lower extremity fractures that were associated with an Abbreviated Injury Scale (AIS; American Association for the

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

Advancement of Automotive Medicine, 1990) score of 3 or less. The AIS is a widely used scoring system that assesses the severity of injuries to specific anatomic regions on a scale of 1 to 6. Children were excluded from the OI group if they displayed any evidence of head injury or symptoms of concussion.

Additional exclusion criteria for children in both groups included: (a) any injury that required surgical or neurosurgical intervention; (b) any associated injury with an AIS score greater than 3; (c) any injury that interfered with neuropsychological testing (e.g., fracture of preferred upper extremity); (d) hypoxia, hypotension, or shock associated with the injury; (e) ethanol or drug ingestion involved with the injury; (f) documented history of previous head trauma requiring medical treatment; (g) premorbid neurological disorder or mental retardation; (h) any injury resulting from child abuse or assault; (i) history of severe psychiatric disorder requiring hospitalization; or (j) any medical contraindication to MRI. Children were not excluded for premorbid learning difficulties or attention problems. The mild TBI and OI groups did not differ on retrospective parent ratings of premorbid school performance or attention problems.

Among children meeting inclusion criteria, participation rates were 35% in the OI group and 48% in the mild TBI group. Participants and nonparticipants did not differ in age, sex, ethnic/racial minority status, or in census tract measures of socioeconomic status (i.e., mean family income, percentage of minority heads of household, and percentage of households below the poverty line). The final sample included 186 children with mild TBI and 99 children with OI. Recreational and sports related injuries were the most common mechanism of injury in both groups. Transportation related injuries were more common in the mild TBI group.

Among the children with mild TBI, 32 (18%) showed trauma-related intracranial abnormalities on MRI, and were classified as having complicated mild TBI, with the rest classified as having an uncomplicated mild TBI. Four children from the mild TBI group could not complete the MRI and were excluded from this study. Children with complicated mild TBI, uncomplicated mild TBI, and OI did not differ in age at injury, sex, or socioeconomic status (SES; see Table 1). As expected, the complicated mild TBI group displayed greater overall injury severity than the other two groups as measured by the Modified Injury Severity Scale (MISS), which is an overall index derived from AIS scores, calculated as the sum of the squares of the three most severely injured body areas (Mayer, Matlak, Johnson, & Walker, 1980). However, when the contribution of the head injury to MISS scores was omitted, the OI group had the highest mean score. Unexpectedly, the complicated mild TBI group included significantly fewer minority children than children in the other two groups (see Table 1). The groups did not differ on parents' retrospective ratings on two out of three measures of premorbid symptoms; they did differ on ratings of premorbid somatic symptoms, with somewhat higher ratings in the uncomplicated mild TBI group.

Procedure and Attrition

The research was approved by the appropriate institutional review boards, and informed parental consent and child assent were obtained in writing prior to participation. Children who met all inclusion/exclusion criteria and whose parents agreed to participate were scheduled for an initial assessment no later than 3 weeks post injury, with 80% completed between 1 and 2 weeks post injury (M = 11.35 days, SD = 3.42). Parents and children rated current PCS at the initial assessment and again at 1, 3, and 12 months post

Table 1. Sample characteristics

Variable	Group		
	Complicated Mild TBI (n = 32)	Uncomplicated Mild TBI (n = 150)	OI (n = 99)
Age at injury (in years), M (SD)	12.16 (1.93)	11.98 (2.27)	11.76 (2.23)
Sex (% male), n (%)	22 (69%)	108 (72%)	64 (65%)
Race (% Caucasian), n (%)*	30 (94%)	99 (66%)	64 (65%)
Socioeconomic status,[1] M (SD)	–0.01 (0.81)	0.08 (0.93)	–0.09 (1.15)
Glasgow Coma Scale score (GCS), M (SD)	14.78 (.55)	14.86 (.42)	n/a
Loss of consciousness, n (%)	17 (53%)	54 (36%)	n/a
Modified Injury Severity Scale, M (SD)*	9.84 (7.04)	3.49 (2.84)	3.25 (1.52)
Modified Injury Severity Scale without head injury component, M (SD)*	0.38 (0.49)	0.31 (0.73)	3.25 (1.52)
Premorbid PCS-I Total Symptoms, M (SD)	.84 (1.22)	1.09 (1.35)	1.13 (2.06)
Premorbid HBI Cognitive Symptoms, M (SD)	9.56 (7.89)	11.60 (7.31)	10.92 (7.59)
Premorbid HBI Somatic Symptoms, M (SD)*	2.19 (2.57)	3.54 (3.38)	2.46 (3.16)

Note. MRI = magnetic resonance imaging; PCS-I = Postconcussive Symptom Interview; HBI = Health and Behavior Inventory; TBI = traumatic brain injury; OI = orthopedic injury.

[1]Socioeconomic status was assessed 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.

*Groups differ significantly, $p < .05$.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

injury. At the initial assessment, parents also completed a retrospective report of pre-injury symptoms, prior to reporting current symptoms. Additionally, children with mild TBI completed standardized testing of cognitive skills, as well as MRI of the brain.

Of 285 children who completed the initial assessment, 280 (98%) completed the assessment at 1-month post injury, 268 (94%) completed the 3-month assessment, and 253 (89%) completed the 12-month assessment. The groups did not differ in the proportion of missed assessments. At each assessment, children who did and did not return did not display any significant differences in demographic or clinical variables (i.e., SES, age at injury, severity of injury, or initial PCS), except for the 12-month assessment, when children who did not return had lower SES and were more likely to be of minority race.

Magnetic Resonance Imaging

The MRI pulse sequence included sagittal T1-weighted spin echo images, axial T2-weighted and proton density fast spin echo images, coronal 2-dimensional gradient echo images, coronal fluid attenuated inversion recovery (FLAIR) images, and axial diffusion-weighted echo planar images. Board-certified radiologists reviewed each MRI using a standard protocol, unaware of the results of any other assessments. Each MRI was scored dichotomously in terms of whether it contained trauma-related intracranial abnormalities.

Measures

Cognitive ability

Children were administered the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). The Wide-Range Achievement Test–Third Edition (WRAT-3; Wilkinson, 1993) was administered to assess spelling, word reading, and math calculation skills. The California Verbal Learning Test–Children's Version (CVLT-C; Delis, Kramer, Kaplan, & Ober, 1994) was administered to assess verbal memory, and the Developmental Test of Visual-Motor Integration (VMI; Beery & Beery, 2004) was administered to examine visuomotor skills. The test battery also included three subtests from the Cambridge Neuropsychological Testing Automated Battery (CANTAB; Sahakian & Owen, 1992): Pattern Recognition Memory, Stockings of Cambridge, and Spatial Working Memory. These subtests assess recognition memory for visual patterns, planning and spatial problem solving, and retention and manipulation of spatial information, respectively.

Exploratory factor analyses of age-based standard scores indicated that the cognitive tests could be represented by two highly correlated factors ($r = .50$), one constituted primarily by verbal or crystallized skills and another constituted primarily by nonverbal or fluid skills. Thus, for the sake of parsimony, cognitive ability was represented in a single overall composite score. The use of a single cognitive composite resulted in increased power and decreased likelihood of Type I error, because it resulted in fewer analyses. Composite scores for each child were computed by transforming all cognitive variables to standard scores with a mean of 100 and a standard deviation of 15, and then taking the mean score across all cognitive tests. The mild TBI and OI groups did not differ on any of the individual cognitive measures, except for small differences on one CANTAB subtest favoring the complicated mild TBI group. The mean scores for the cognitive ability composite differed significantly between the OI and mild TBI groups, with the complicated mild TBI group showing a higher mean score on the cognitive ability composite than the other two groups (see Table 2). We should note that all hierarchical linear modeling analyses reported below were repeated for the separate cognitive factors. Results were essentially the same as those found when using a single overall composite score.

Postconcussuve symptoms

Postconcussuve symptoms (PCS) were assessed using two measures, the Postconcussive Symptom Interview (PCS-I; Mittenberg et al., 1997) and the Health and Behavior Inventory (HBI; Yeates et al., 1999). The parent and child forms of both measures are worded slightly differently to be age-appropriate and to reflect first- *versus* third-person perspectives. The PCS-I is administered orally, and asks children and their parents to report the presence or absence of 15 postconcussive symptoms over the past week. The symptoms include cognitive, somatic, and emotional difficulties, similar to those listed in the research diagnostic criteria for postconcussional disorder in the *Diagnostic and Statistical Manual of Mental Disorders*, *Fourth Edition* (DSM-IV; American Psychiatric Association, 1994) and for postconcussion syndrome in the International Classification of Diseases (World Health Organization, 1993). The total number of symptoms was used as the measure of PCS, and displayed satisfactory internal consistency at each of the four assessments (Cronbach's alpha from .78 to .82 for parents' ratings and from .70 to .77 for children's ratings).

The HBI is a 50-item self-report questionnaire that includes a variety of cognitive, somatic, emotional, and behavioral symptoms. The questionnaire requires parents and children to rate the frequency of occurrence of each symptom over the past week on a four-point scale, ranging from "never" to "often." Cognitive and somatic symptom dimensions of the

Table 2. Mean scores on the Cognitive Ability Composite by group

	Cognitive Ability	
Group*	*M*	*SD*
Complicated Mild TBI ($n = 32$)	105.11	8.92
Uncomplicated Mild TBI ($n = 150$)	98.91	9.08
OI ($n = 99$)	98.53	11.08

Note. TBI = Traumatic Brain Injury; OI = Orthopedic Injury.
*Groups differ significantly, $p < .05$.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

HBI were used as measures of PCS, based on a recent factor analytic study showing substantial agreement between parents and children for those symptom dimensions as compared to emotional and behavioral symptom dimensions (Ayr, Yeates, Taylor, & Browne, 2009). Total scores for each dimension were used as measures of PCS and displayed satisfactory internal consistency across all four assessments (Cronbach's alpha from .83 to .95 for parents' ratings and from .86 to .91 for children's ratings).

Data Analysis

Hierarchical linear modeling was used to examine child and parent ratings of PCS longitudinally (Burchinal, Bailey, & Snyder, 1994; Jennrich & Schluchter, 1986). The analyses involved a group × cognitive ability × time design. Three groups (complicated mild TBI, uncomplicated mild TBI, and OI) were entered into the analyses using dummy variables contrasting each of the mild TBI groups with the OI group. Analyses were conducted for six dependent variables (PCS-I total score, HBI cognitive score, and HBI somatic score, based on parent and child ratings). Intraindividual growth curves (i.e., Level 1 models) were characterized using intercept, slope, and acceleration/deceleration terms (i.e., linear and quadratic change). For all analyses, time post injury was centered at 90 days post injury because research suggests that PCS often resolve around 3 months after mild TBI (Satz et al., 1997; Yeates & Taylor, 2005). Thus, the intercept parameter for the Level 1 models represented the expected level of PCS at 3 months post injury, and the linear change parameter represented the linear rate of change in PCS at 3 months post injury. Because data were available for four time points (i.e., initial assessment and 1, 3, and 12 months follow-ups), the model included a quadratic parameter representing the rate of deceleration/acceleration in PCS across time.

Each analysis consisted of two steps. In the first step, unconditional Level 1 models were estimated for each dependent variable to examine the mean and variance of the growth curve parameters. In the second step, Level 2 models were tested to assess the contributions of specific predictor variables to the number of PCS at 3 months post injury, the rate of change at that time point, and the degree of curvature in the growth curve across the first year post injury. Race (white *vs.* nonwhite), SES, and age at injury were included as covariates in all models. Because ratings of premorbid symptoms were obtained from the parents at the initial assessment, analyses of parent-rated PCS also included the ratings of premorbid symptoms as covariates. Children did not complete ratings of premorbid symptoms, and so analyses of their ratings were not adjusted for premorbid functioning. All continuous predictors (i.e., SES, age at injury, premorbid PCS, cognitive ability) were centered for the analyses.

The central hypotheses were tested by including interaction terms to examine the moderating influence of cognitive ability on severity of injury (i.e., complicated mild TBI, uncomplicated mild TBI, and OI) in PCS over time. Interaction terms that were not significant were eliminated from each analysis and the models were re-estimated without them. For all significant effects involving group, cognitive ability, and their interaction, the percentage of parameter variance explained by each effect was estimated using the method described by Raudenbush and Bryk (2002).

RESULTS

Level 1 Unconditional Models

The results of the Level 1 unconditional models are summarized in Table 3. As expected, the estimated mean intercepts were significantly different from 0 for all six measures of PCS. Additionally, five of the six linear change parameters and five of the six quadratic change parameters had an estimated mean significantly different from 0. The estimated true parameter variance was significant for all intercepts and linear change parameters, and three out of six quadratic change (i.e., curvature) parameters. Thus, children displayed significant variation in the number of PCS at 3 months post injury and in the rate of linear change in PCS for all measures, as well as in the rate of acceleration/deceleration across the first year post injury for the child-rated PCS-I score, parent-rated HBI cognitive symptom score, and parent-rated HBI somatic symptom score. The linear and quadratic change parameters were consistently very highly correlated. Based on tests of the quadratic change parameter's mean and variance, it was removed from the Level 1 models involving child-rated cognitive symptoms on the HBI and was treated as nonrandomly varying for Level 1 models involving child-rated somatic symptoms on the HBI.

Level 2 Conditional Models

The results of the Level 2 models are summarized in Tables 4 (for parent ratings) and 5 (for child ratings). For each dependent variable, the tables list the individual predictors that contributed significantly to each outcome, along with the estimated coefficients for these predictors.

Parent-rated PCS-I total score

None of the interaction terms involving group and cognitive ability was significant for the parent-rated PCS-I total score. Significant main effects were obtained for group and cognitive ability, with small to medium effect sizes (see Table 4). The complicated mild TBI group demonstrated higher total scores on the PCS-I than the OI group at 3 months post injury. In addition, children with lower cognitive ability had higher total scores at 3 months. The uncomplicated mild TBI group showed a more rapid rate of decline in the total score at 3 months post injury compared to the OI group. Injury severity was also a significant predictor of the quadratic parameter, so that the uncomplicated mild TBI group showed an even more rapid decline in symptoms acutely, and eventual leveling off, compared to the OI group.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

Table 3. Results of Level 1 unconditional models

Outcome measure	Estimated parameter	*SE*	Estimated parameter variance	Reliability	Parameter *r*
Parent rated PCS-I					
Intercept	2.23**	.15	3.88**	.56	I&L = −.10
Linear	−4.62**	.59	28.34**	.27	L&Q = −.99
Quadratic	5.48**	.84	36.39	.16	I&Q = .06
Child rated PCS-I					
Intercept	3.72**	.18	5.73**	.63	I&L = .37
Linear	−2.82**	.59	17.72**	.17	L&Q = −.99
Quadratic	4.26**	.89	25.95*	.11	I&Q = −.40
Parent rated HBI Cognitive					
Intercept	11.04**	.47	44.94**	.72	I&L = .26
Linear	4.96**	1.37	107.2*	.20	L&Q = −.99
Quadratic	−8.24**	2.07	174.87*	.14	I&Q = −.28
Parent rated HBI Somatic					
Intercept	2.94**	.23	7.87**	.50	I&L = .12
Linear	−5.85**	1.04	120.98**	.38	L&Q = −.99
Quadratic	7.35**	1.50	203.56**	.29	I&Q = −.16
Child rated HBI Cognitive					
Intercept	10.98**	.38	34.67**	.82	I&L = −.27
Linear	−.04	.42	10.51**	.22	
Quadratic[a]					
Child rated HBI Somatic					
Intercept	6.06**	.33	20.25**	.82	I&L = −.35
Linear	−4.56**	.94	5.95**	.22	
Quadratic[b]	6.12**	1.41			

Note. The intercept represents the average performance at 3 months post injury. The linear terms represents that average amount of change (per year) at 3 months post injury, and the quadratic term represents the degree of curvature across 1 yea r post injury. Reliability reflects the percentage of parameter variance that is potentially explainable by predictors in the Level 2 models. Parameter *r* is the estimate of the correlations between the Intercept (I), Linear (L), and Quadratic (Q) change parameters.

[a]The parameter was deleted from the model.

[b]The parameter was retained but treated as nonrandomly varying in conditional models (i.e., residual variance was constrained to zero).

*$p < .05$, **$p < .01$.

Parent-rated HBI cognitive symptoms

Significant interactions between group and cognitive ability were found for parent-rated cognitive symptoms on the HBI, ranging from small to large in magnitude (see Table 4). To help illustrate the interactions, children were dichotomized into high and low cognitive ability groups (<105 standard score = low cognitive ability; ≥105 standard score = high cognitive ability). The dichotomization was strictly for the purposes of illustrating the interactions; cognitive ability was treated as continuous in the actual analyses. The cutoff point was selected to maximize the number of children in each of the complicated mild TBI subgroups, making the estimation of means more reliable for graphing purposes. The mean fitted values from the final model were graphed for each group relative to the number of days post injury (see Figure 1).

Figure 1 shows that children with complicated mild TBI and lower cognitive ability did not differ when compared to the other low cognitive ability groups at the initial assessment, but then demonstrated a rapid increase in cognitive symptoms across the first 3 months post injury. Higher cognitive ability appeared to protect against the development of cognitive symptoms, as all three high cognitive ability groups demonstrated relatively few symptoms across time, and did not differ from one another.

Parent-rated HBI somatic symptoms

None of the interactions between group and cognitive ability were significant for parent-rated somatic symptoms on the HBI. Group differences accounted for a significant variance in the intercept parameters, with a medium effect size (see Table 4). The complicated mild TBI group demonstrated more somatic symptoms than the OI group at 3 months post injury. A significant positive curvature of medium to large magnitude indicated a rapid rate of decline in parent-rated somatic symptoms acutely for the uncomplicated mild TBI group, with the rate of decline slowing after 90 days post injury. Cognitive ability also accounted for significant variance in the intercept parameter, again of medium to large magnitude, with children with higher cognitive ability demonstrating less parent-rated somatic symptoms at 3 months post injury.

Child-rated PCS-I total score

Significant interactions involving group and cognitive ability were found for the child-rated PCS-I total score,

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

Table 4. Significant parameter estimates (and variance explained) in Level 2 conditional models for parent-rated PCS

	Measure								
	PCS-I total symptoms			HBI cognitive symptoms			HBI somatic symptoms		
Predictors	Intercept	Linear change	Quadratic change	Intercept	Linear change	Quadratic change	Intercept	Linear change	Quadratic change
Complicated Mild TBI *vs*. OI	.86* (1.5%)			5.00** (6.0%)	17.86** (12.1%)	−29.26** (18.5%)	1.99* (5.2%)		
Uncomplicated Mild TBI *vs*. OI		−3.31** (7.9%)	3.95* (8.5%)					−7.53** (8.6%)	8.18** (5.3%)
Race									
SES									
Age at Injury	−.20**								
Premorbid PCS-I symptoms	.58**								
Premorbid HBI Cognitive symptoms				.66**					
Premorbid HBI Somatic symptoms							.48**	.68*	
Cognitive Ability	−.04** (3.4%)						−.08** (7.9%)		
Complicated Mild TBI *vs*. OI × Cognitive Ability				−.28** (0.2%)	−1.29** (6.0%)	2.28** (11.6%)			
Uncomplicated Mild TBI *vs*. OI × Cognitive Ability									

Note. PCS-I = Postconcussive Symptom Interview; HBI = Health and Behavior Inventory; TBI = traumatic brain injury; OI = orthopedic injury.
**$p < .01$, *$p < .05$.

ranging from small to large in magnitude. The interaction involving the contrast between the complicated mild TBI and OI groups and cognitive ability was a significant predictor of the intercept, linear, and quadratic parameters (see Table 5). Figure 2 shows that children with complicated mild TBI and lower cognitive ability had higher child-rated PCS-I total scores at the initial assessment, and then showed a rapid decrease in symptoms, so that by 3 months post injury, they did not differ from the children with complicated mild TBI and higher cognitive ability. The uncomplicated mild TBI group with low cognitive ability also showed high total scores at the initial and 1-month assessment, and then at 3 months post injury began to follow the same trend as the OI group with low cognitive ability. Higher cognitive ability appeared to help protect against the development of PCS, as children with high cognitive

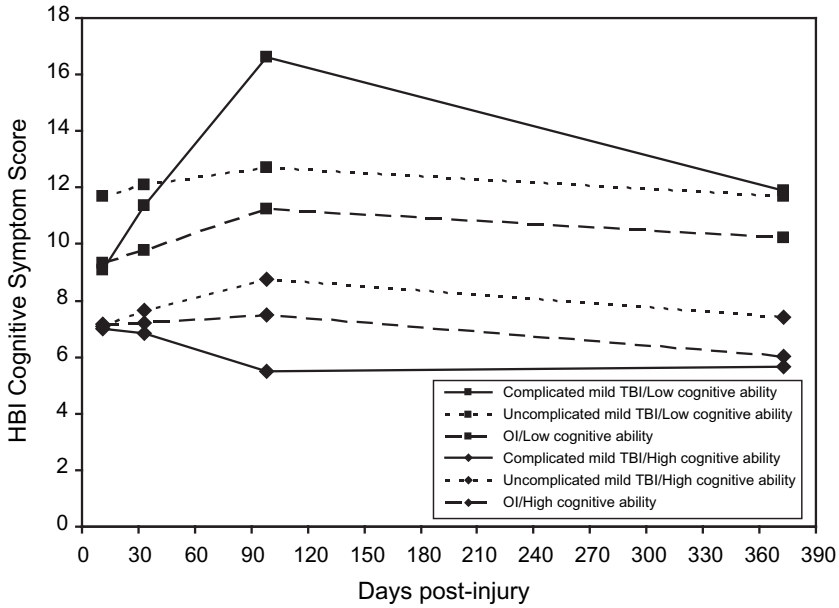

Fig. 1. Parent-rated HBI cognitive symptoms by cognitive ability and group.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

Table 5. Significant parameter estimates (and variance explained) in Level 2 conditional models for child-rated PCS

	Measure								
	PCS-I symptoms			HBI Cognitive symptoms			HBI Somatic symptoms		
Predictors	Intercept	Linear change	Quadratic change	Intercept	Linear change	Quadratic change	Intercept	Linear change	Quadratic change
Complicated Mild TBI *vs*. OI		−7.28** (23.9%)	9.33** (26.1%)					−7.65* (2.3%)	
Uncomplicated Mild TBI *vs*. OI								−4.31* (−1.8%)	
Race				2.08*					
SES					1.08*				
Age at Injury	−.29**	−.76**	1.15**						
Cognitive Ability	−.07* (3.7%)			−.19** (6.6%)					
Complicated Mild TBI *vs*. OI x Cognitive Ability	.11* (2.2%)	.58** (16.4%)	−.70* (16.6%)						
Uncomplicated Mild TBI *vs*. OI x Cognitive Ability									

Note. PCS-I = Postconcussive Symptom Interview; HBI = Health and Behavior Inventory; TBI = traumatic brain injury; OI = orthopedic injury.
**$p < .01$, *$p < .05$.

ability in all three groups demonstrated relatively few PCS across time.

Child-rated HBI cognitive symptoms

No interactions involving group and cognitive ability were significant for child-rated cognitive symptoms on the HBI. Group differences also did not account for significant variance in the intercept or linear change parameters (see Table 5). Cognitive ability was a significant predictor of the intercept parameter, with a medium effect size. Children with lower cognitive ability reported more cognitive symptoms at 3 months than children of higher cognitive ability.

Child-rated HBI somatic symptoms

Although the interactions of group and cognitive ability were not significant for child-rated somatic symptoms on the HBI, group differences accounted for a small but significant amount of variance in the linear and quadratic change parameters. Children with complicated mild TBI showed the most rapid rate of decline in child-rated somatic symptoms

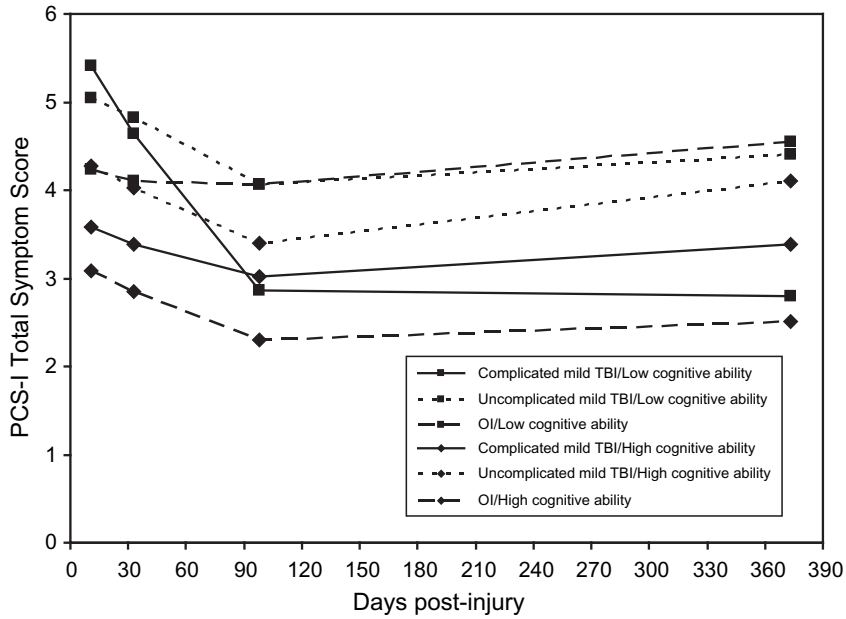

Fig. 2. Child-rated total symptoms on the PCS-I by cognitive ability and group.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

at 3 months post injury and different curvature over time compared to the OI group (see Table 5).

Covariates

Parent-rated premorbid PCS was consistently related to parent-rated post injury PCS symptoms, with more premorbid PCS predicting higher PCS at 3 months post injury (see Table 4). Age at injury was a significant predictor of the intercept parameter for both the parent- and child-rated total PCS-I score (see Tables 4 and 5). Younger children consistently were rated as showing more PCS at 3 months post injury than older children. Race was a significant predictor of the intercept parameter for child-rated cognitive symptoms on the HBI, with white children reporting fewer symptoms relative to nonwhite children at 3 months (see Table 5). SES was a significant predictor of the linear change parameter for child-rated cognitive symptoms on the HBI (see Table 5). Children from lower SES households reported less rapid change in cognitive symptoms at 3 months post injury relative to children from higher SES backgrounds.

DISCUSSION

The primary goal of the current study was to determine whether the relationship between mild TBI and PCS is moderated by cognitive ability. The findings are consistent with the hypothesis that mild TBI are more likely to result in PCS, relative to OI, among children of lower cognitive ability than among children of higher cognitive ability. This was especially true for children with complicated mild TBI. Thus, cognitive ability moderated PCS in the complicated mild TBI group on both the child-rated PCS-I total score and parent-rated HBI cognitive symptom score.

On the PCS-I measure, which includes a mixture of cognitive, somatic, and emotional symptoms, children with low cognitive ability and a complicated mild TBI endorsed the most symptoms at baseline relative to all other groups, but then showed a rapid linear decrease in symptoms to 3 months post injury. In contrast, children with complicated mild TBI but high cognitive ability endorsed relatively few symptoms across time. Parent ratings of cognitive symptoms on the HBI demonstrated a more pronounced pattern of increased vulnerability to mild TBI among children with low cognitive ability. More specifically, children with low cognitive ability and complicated mild TBI showed significant increases in cognitive symptoms to 3 months post injury, and the largest group differences relative to children with OI were apparent for children of low cognitive ability with complicated mild TBI.

At first glance, the nature of these two interactions may appear somewhat contradictory. Children with low cognitive ability and complicated mild TBI reported more PCS initially, followed by a rapid decline, whereas their parents reported an increase specifically in cognitive symptoms. However, the findings probably reflect differences between the outcome measures in terms of the types of symptoms they assess, as well as their relative sensitivity for parents *versus* children. The PCS-I consists of a mixture of different types of PCS, so it is difficult to know whether its moderation by cognitive reserve reflects specific types of symptoms (i.e., cognitive, somatic, emotional). In contrast, the HBI assesses specific dimensions of PCS, and evidence of moderation was apparent only for cognitive symptoms. However, the PCS-I may be a more sensitive measure for children than the HBI, and *vice versa* for parents, because previous analyses have found larger group differences for parents on the HBI scales, whereas for children group differences are largest on the PCS-I (Taylor et al., in press). The format of the PCS-I, which involves oral administration and simple yes/no responses, may be easier for children to complete than that of the HBI, which involves written administration and a four-point rating scale. In contrast, parents may provide more refined estimates of specific symptom types using the HBI.

Thus, a possible explanation for the current pattern of findings is that children who are vulnerable because of low cognitive ability report a general increase in PCS immediately after complicated mild TBI that they perceive resolves fairly quickly, but their parents become more aware of persisting cognitive symptoms over time, perhaps as the effects of inattention, forgetfulness, and distractibility begin to impact the child's school work or abilities to function at home. Notably, no significant interactions were found for the HBI somatic symptom rating, for either parent or child ratings. This may be because somatic symptoms are not particularly susceptible to moderation by cognitive reserve. Cognitive symptoms may be more susceptible to moderation because they are more closely linked to cognitive reserve, and parents may be better able to distinguish these symptoms than the children themselves. Indeed, group contrasts were significant for children's ratings of somatic, but not cognitive symptoms, but were significant for parent ratings of both types of symptoms.

In summary, we believe the presence of two significant interactions argues strongly for the existence of a complex interplay between cognitive ability and injury severity during recovery from mild TBI, especially given the low probability of detecting interactions in nonexperimental research designs (McClelland & Judd, 1993). Finding moderation effects for both child and parent ratings of PCS also helps to bolster confidence in the results. Thus, the findings provide support for reserve capacity theory (Dennis et al., 2006; Satz, 1993; Stern, 2002). Children with high cognitive ability and thus higher reserve may be somewhat protected from the effects of complicated mild TBI, while those with less reserve (i.e., low cognitive ability) demonstrate greater vulnerability to the development of PCS immediately after and up to 3 months post injury. These findings are consistent with previous research in children with moderate to severe TBI (Farmer et al., 2002), as well as in children at risk for reasons other than TBI (Masten, 2001). However, cognitive ability has not been investigated previously as a moderator of PCS after mild TBI. The mechanism by which cognitive ability acts as a moderator of PCS is unclear. Higher cognitive ability could be reflected in the individual's active capacity to cope with neurological insults. However, higher cognitive

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

ability could also be linked to the passive capacity of the brain to adapt to brain injury, given the correlations between cognitive ability and brain structure and function (Choi et al., 2008; McDaniel, 2005).

Although the key findings from this study indicate that cognitive ability moderates PCS following mild TBI, the results also indicate that cognitive ability predicts PCS generally after minor injuries. That is, lower cognitive ability predicted more PCS at 3 months post injury for all groups. We believe this is the first time that a general effect of cognitive ability on PCS in children with minor injuries has been demonstrated. Injury severity was also a significant independent predictor, such that complicated mild TBI predicted higher levels of PCS at 3 months post injury. This finding is consistent with previous studies showing an increased risk of poor outcomes after a complicated mild TBI (Levin et al., 2008). Despite an increased risk of PCS, children with complicated mild TBI also were more likely to display a rapid decrease in symptoms following high acute levels. This is encouraging, as many of these children are likely to show a long-term course of recovery similar to children with uncomplicated mild TBI (Ponsford et al., 1999; Yeates et al., 2009).

Retrospective ratings of premorbid symptoms by parents also were positively related to reports of PCS at 3 months on all parent-rated measures. Past research has noted the effects of premorbid behavior problems on outcome after mild TBI (Asarnow et al., 1995; Levin et al., 2008; Mittenberg et al., 1997; Ponsford et al., 1999). The severity of PCS clearly is related to the severity of premorbid symptoms, which should be taken into consideration when evaluating the effects of mild TBI.

Thus, the current results indicate that the occurrence of PCS after mild TBI is multidetermined. Research with children and adults indicates that both injury characteristics and noninjury related variables help explain outcomes after mild TBI, offering tentative support for both physiogenic and psychogenic explanations of PCS (Luis, Vanderploeg, & Curtiss, 2003; Ponsford et al., 1999). The current study adds to this literature by showing that PCS following mild TBI are predicted by premorbid symptoms, cognitive ability, and severity of injury, as well as the joint interaction of the latter two risk factors.

The current study has several limitations. The complicated mild TBI group had a higher mean score than the uncomplicated mild TBI and OI groups on the cognitive ability composite. A possible explanation for this finding is that the complicated mild TBI group consisted of fewer children from minority backgrounds who may have been at greater sociodemographic disadvantage compared to the other two groups. Although race was covaried in all analyses and the groups did not differ in SES, the differences in cognitive ability may have affected the results unexpectedly.

Another potential limitation was the use of a single composite measure based on cognitive tests administered post injury to represent premorbid cognitive ability. However, the groups did not differ on any post injury cognitive measure, and analyses using separate cognitive factors resulted in identical findings. Nevertheless, procuring a measure of premorbid functioning based on ratings of school performance or results of achievement tests prior to injury may be desirable in future studies. Future studies also would benefit from the inclusion of more detailed information regarding premorbid conditions such as attention-deficit/hyperactivity disorder and specific learning disabilities, both of which may serve as proxies of cognitive reserve (Farmer et al., 2002; Yeates et al., 2005).

Another shortcoming is that parents' ratings of premorbid symptoms were obtained retrospectively and may have been biased by post injury functioning. Studies that include pre-injury assessments of symptoms would provide greater insight into the relationships between premorbid functioning and PCS after mild TBI. However, the contemporaneous measurement of pre-injury symptoms is not likely to be feasible. A further limitation is that the recruitment rates for the mild TBI and OI groups were both below 50%. However, nonparticipants did not differ from participants demographically. Attrition may also have introduced bias, although the analyses included all children with at least one assessment of PCS. Children who did not return for the 1-year follow-up had significantly lower SES than those who did return. Although SES was covaried in the analyses, the findings may underestimate PCS in both groups, because children of lower SES were at higher risk for symptoms.

Despite these limitations, the current study provides support for the hypothesis that the severity of PCS after a complicated mild TBI is moderated by cognitive ability. Given these results, physicians and other health care professionals working with children who sustain mild TBI may be better able to identify children at risk of developing significant PCS after mild TBI, and focus prevention and intervention efforts on children with abnormal neuroimaging results and lower cognitive ability. Further research is needed to identify the entire range of factors that place children with mild TBI at risk for persistent PCS and to thereby improve their clinical care.

ACKNOWLEDGMENTS

The research reported here served as the basis for a dissertation submitted by the first author to The Ohio State University in partial fulfillment of the requirements for a doctoral degree. Portions of the research were presented at the annual meeting of the International Neuropsychological Society, Waikoloa, Hawaii, February, 2008. The larger study on which the research was based was supported by grants R01 HD39834 and K02 HD44099 from the National Institute of Child Health and Human Development and the National Center for Medical Rehabilitation Research to Keith Owen Yeates. The authors wish to acknowledge the contributions of Lauren Ayr, Anne Birnbaum, Amy Clemens, Kalaichelvi Ganesalingham, Christine Hajek, Amanda Lininger, Melissa Ginn, Katie Pestro, Elizabeth Roth, Elizabeth Shaver, and Heidi Walker in conducting the study.

REFERENCES

Alexander, M.P. (1997). Minor traumatic brain injury: A review of physiogenesis and psychogenesis. *Seminars in Clinical Neuropsychiatry*, *2*, 177–187.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

American Academy of Pediatrics. (1999). The management of minor closed head injury in children. *Pediatrics*, *104*, 1407–1415.

American Association for the Advancement of Automotive Medicine. (1990). *The Abbreviated Injury Scale, 1990 revision*. Des Plaines, IL: American Association for the Advancement of Automotive Medicine.

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: American Psychiatric Association Press.

Asarnow, R.F., Satz, P., Light, R., Zaucha, K., Lewis, R., & McCleary, C. (1995). The UCLA study of mild head injury in children and adolescents. In M.E. Michel & S. Broman (Eds.), *Traumatic head injury in children* (pp.117–146). New York: Oxford University Press.

Ayr, L.K., Yeates, K.O., Taylor, H.G., & Browne, M. (2009). Dimensions of post-concussive symptoms in children with mild traumatic brain injuries. *Journal of the International Neuropsychological Society*, *15*(1), 19–30.

Bazarian, J.J., McClung, J., Shah, M.N., Cheng, Y.T., Flesher, W., & Kraus, J. (2005). Mild traumatic brain injury in the United States, 1998–2000. *Brain Injury*, *19*, 85–91.

Beery, K.E., & Beery, N.A. (2004). *The Beery-Buktenica Developmental Test of Visual-Motor Integration (5th ed.): Administration, scoring, and teaching manual with supplemental developmental tests of Visual Perception and Motor Coordination*. Minneapolis, MN: NCS Pearson.

Bijur, P.E., Haslum, M., & Golding, J. (1990). Cognitive and behavioral sequelae of mild head injury in children. *Pediatrics*, *86*(3), 337–344.

Burchinal, M.R., Bailey, D.B., & Snyder, P. (1994). Using growth curve analysis to evaluate child change in longitudinal investigations. *Journal of Early Intervention*, *18*, 422–442.

Choi, Y.Y., Shamosh, N.A., Cho, S.H., DeYoung, C.G., Lee, M.J., Lee, J.-M., et al. (2008). Multiple bases of human intelligence revealed by cortical thickness and neural activation. *Journal of Neuroscience*, *28*, 10323–10329.

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (1994). *Manual for the California Verbal Learning Test for Children*. New York: The Psychological Corporation.

Dennis, M., Yeates, K.O., Taylor, H.G., & Fletcher, J.M. (2006). Brain reserve capacity, cognitive reserve capacity, and age-based functional plasticity after congenital and acquired brain injury in children. In Y. Stern (Ed.), *Cognitive reserve: Theory and applications* (pp. 53–83). New York: Psychology Press.

Farmer, J.E., Kanne, S.M., Haut, J.S., Williams, J., Johnstone, B., & Kirk, K. (2002). Memory functioning following traumatic brain injury in children with premorbid learning problems. *Developmental Neuropsychology*, *22*(2), 455–469.

Fay, G.C., Jaffe, K.M., Polissar, N.L., Liao, S., Martin, K., Shurtleff, H., et al. (1993). Mild pediatric traumatic brain injury: A cohort study. *Archives of Physical Medicine and Rehabilitation*, *74*(9), 895–901.

Jennrich, R.I., & Schluchter, M.D. (1986). Unbalanced repeated measures models with structured covariance matrices. *Biometrics*, *42*, 805–820.

Katzman, R. (1993). Education and the prevalence of dementia and Alzheimer's disease. *Neurology*, *43*, 13–20.

Koenen, K.C., Moffitt, T.E., Roberts, A.L., Martin, L.T., Kubzansky, L., Harrington, H., et al. (2009). Childhood IQ and adult mental disorders: A test of the cognitive reserve hypothesis. *American Journal of Psychiatry*, *166*, 50–57.

Kraus, J.F. (1995). Epidemiological features of brain injury in children: Occurrence, children at risk, causes and manner of injury, severity, and outcomes. In S.H. Broman & M.E. Michel (Eds.), *Traumatic head injury in children* (pp. 22–39). New York: Oxford University Press.

Levin, H.S., Hanten, G., Roberson, G., Xiaoqi, L., Ewing-Cobbs, L., Dennis, M., et al. (2008). Prediction of cognitive sequelae based on abnormal computed tomography findings in children following mild traumatic brain injury. *Journal of Neurosurgery in Pediatrics*, *1*, 461–470.

Lishman, W.A. (1998). Physiogenesis and psychogenesis in the postconcussional syndrome. *British Journal of Psychiatry*, *153*, 460–469.

Luis, C.A., Vanderploeg, R.D., & Curtiss, G. (2003). Predictors of postconcussive symptom complex in community dwelling male veterans. *Journal of the International Neuropsychological Society*, *9*, 1001–1015.

Mayer, T., Matlak, M., Johnson, D., & Walker, M. (1980). The Modified Injury Severity Scale in pediatric multiple trauma patients. *Journal of Pediatric Surgery*, *15*, 719–726.

Masten, A.S. (2001). Ordinary magic: Resilience processes in development. *American Psychologist*, *56*(3), 227–238.

McClelland, G.H., & Judd, C.M. (1993). Statistical difficulties of detecting interactions and moderator effects. *Psychological Bulletin*, *114*, 376–390.

McDaniel, M.A. (2005). Big-brained people are smarter: A meta-analysis of the relationship between *in vivo* brain volume and intelligence. *Intelligence*, *33*, 337–346.

Mittenberg, W., Wittner, M.S., & Miller, L.J. (1997). Postconcussion syndrome occurs in children. *Neuropsychology*, *11*(3), 447–452.

Ponsford, J., Wilmott, C., Rothwell, A., Cameron, P., Ayton, G., Nelms, R., et al. (1999). Cognitive and behavioral outcome following mild traumatic head injury in children. *Journal of Head Trauma Rehabilitation*, *14*(4), 360–372.

Raudenbush, S.W., & Bryk, A.S. (2002). *Hierarchical linear models: Applications and data anlaysis methods* (2nd ed.). Thousand Oaks, CA: Sage Publications.

Rivara, J.B., Jaffe, J.M., Polissar, N.L., Fay, G.C., Martin, K.M., Shurtleff, H.A., & Liao, S. (1994). Family functioning and children's academic performance and behavior problems in the year following traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *75*, 369–379.

Sahakian, B.J., & Owen, A.M. (1992). Computerised assessment in neuropsychiatry using CANTAB: Discussion paper. *Journal of the Royal Society of Medicine*, *85*, 399–402.

Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for threshold theory. *Neuropsychology*, *7*, 273–295.

Satz, P. (2001). Mild head injury in children and adolescents. *Current Directions in Psychological Science*, *10*(3), 106–109.

Satz, P., Zaucha, K., McCleary, C., Light, R., & Asarnow, R.F. (1997). Mild head injury in children and adolescents: A review of studies (1970–1995). *Psychological Bulletin*, *122*(2), 107–131.

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society*, *8*, 448–460.

Taylor, H.G., Dietrich, A., Nuss, K., Wright, M., Rusin, J., Bangert, B., Minich, N., & Yeates, K.O. (in press). Post-concussive symptoms in children with mild traumatic brain injury. *Neuropsychology*.

Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet*, *2*, 81–84.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press

Thurman, D., & Guerrero, J. (1999). Trends in hospitalization associated with traumatic brain injury. *Journal of the American Medical Association*, *282*(10), 954–957.

Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence*. San Antonio, TX: The Psychological Corporation.

Williams, D.H., Levin, H.S., & Eisenberg, H.M. (1990). Mild head injury classification. *Neurosurgery*, *27*, 422–428.

Wilkinson, G. (1993). *Wide Range Achievement Test (3rd ed.): Administration manual*. Wilmington, DE: Wide Range.

World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization.

Yeates, K.O., Armstrong, K., Janusz, J., Taylor, H.G., Wade, S., Stancin, T., & Drotar, D. (2005). Long-term attention problems in children with traumatic brain injury. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 574–584.

Yeates, K.O., Luria, J., Bartkowski, H., Rusin, J., Martin, L., & Bigler, E.D. (1999). Postconcussive symptoms in children with mild closed head injuries. *Journal of Head Trauma Rehabilitation*, *14*(4), 337–350.

Yeates, K.O., & Taylor, H.G. (2005). Neurobehavioral outcomes of mild head injury in children and adolescents. *Pediatric Rehabilitation*, *8*(1), 5–16.

Yeates, K.O., Taylor, H.G., Rusin, J., Bangert, B., Dietrich, A., Nuss, K., et al. (2009). Longitudinal trajectories of postconcussive symptoms in children with mild traumatic brain injuries and their relationship to acute medical status. *Pediatrics*, *123*, 735–743.

https://doi.org/10.1017/S1355617709991007 Published online by Cambridge University Press