Psychiatric Disorders After Pediatric Traumatic Brain Injury: A Prospective, Longitudinal, Controlled Study

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The objective was to examine the effects of traumatic brain injury (TBI), as compared with orthopedic injury (OI), relative to the risk for psychiatric disorder. There has only been one previous prospective study of this nature. Participants were age 7–17 years at the time of hospitalization for either TBI (complicated mild-to-severe) or OI. The study used a prospective, longitudinal, controlled design, with standardized psychiatric assessments conducted at baseline (reflecting pre-injury functioning) and 3 months post-injury. Assessments of pre-injury psychiatric, adaptive functioning, family adversity, and family psychiatric history status were conducted. Severity of injury was assessed by standard clinical scales. The outcome measure was the presence of a psychiatric disorder not present before the injury ("novel"), during the first 3 months after TBI. Enrolled participants (N=141) included children with TBI (N=75) and with OI (N=66). The analyses focused on 118 children (84%) (TBI: N=65; OI: N=53) who returned for follow-up assessment at 3 months. Novel psychiatric disorder (NPD) occurred significantly more frequently in the TBI (32/65; 49%) than the OI (7/53; 13%) group. This difference was not accounted for by pre-injury lifetime psychiatric status; pre-injury adaptive functioning; pre-injury family adversity, family psychiatric history, socioeconomic status, injury severity, or age at injury. Furthermore, none of these variables significantly discriminated between children with TBI who developed, versus those who did not develop, NPD. These findings suggest that children with complicated mild-to-severe TBI are at significantly higher risk than OI-controls for the development of NPD in the first 3 months after injury.

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Traumatic brain injury (TBI) in children and adolescents is a major public health problem.[1] Accumulated knowledge of psychiatric complications after pediatric TBI derives from over 20 cohorts, including 7 from our group,[2–26] studied over the past 80 years. Other pediatric TBI studies that include behavioral outcomes, but not specifically psychiatric outcomes, complement

the psychiatric literature.[27–31] However, there has only been one published study of pediatric TBI that used a standardized psychiatric interview, a prospective longitudinal design, and had an injured control group.[3] That study included 60 children with TBI (31 with severe TBI, 29 with "mild" TBI) and 28 orthopedic injury (OI) controls studied at baseline (soon after injury), at 4 months, 12 months, and 27 months after injury. New psychiatric disorder developed in 48% of children (10/21) with "severe" TBI, in 15% of children (3/20) with "mild" TBI, and 5% of children (1/22) with OI in the first 4 months after injury. The onset of psychiatric disorders within the first 4 months after injury in the severe TBI group was associated with the child's concurrent intellectual level. In children with severe TBI, pre-injury behavior predicted new psychiatric disorder at 12 months, and psychosocial adversity predicted new psychiatric disorder that persisted over two assessments. This seminal study has weaknesses, by contemporary standards, related to the use of one of the earliest versions of a standardized psychiatric interview for parents of children, direct children's psychiatric assessment by a brief mental status exam only, and in the classification of TBI severity before common use of the Glasgow Coma Scale.[32] Our attempts to estimate the reported severity of TBI in the 1981-published study by modern criteria suggest that individuals in the severe-TBI group would retain such classification, and that about ⅔ of the "mild-TBI" group (defined clinically by posttraumatic amnesia greater than 1 hour but less than 1 week) would retain such classification, whereas about ⅓ of the "mild-TBI" would be classified as complicated mild-or-moderate TBI.[33] Another limitation of the older study was that children who were judged to have a psychiatric disorder before the injury were not considered eligible for the development of a "new psychiatric disorder." In our psychiatric studies, which began in 1992, we coined the term "novel psychiatric disorder (NPD)" so that the outcome of interest would not be confused with "new psychiatric disorder" used in the landmark study. The new term was necessary to understand the development of psychopathology in children who have a pre-injury psychiatric disorder because this is very common in children with TBI.[2] Novel psychiatric disorder (NPD) is therefore diagnosed in one of two conditions. The first condition coincides with the definition of new psychiatric disorder, and this could occur in a participant with no lifetime pre-injury psychiatric disorder who then develops a psychiatric disorder after injury. The second condition could occur in the case of a participant with a lifetime psychiatric disorder who then develops a post-injury psychiatric disorder that was never before present (e.g., a subject with a lifetime history of major depressive disorder who develops oppositional defiant disorder after the injury would receive the classification, but would not if only a new episode of major depression occurred).

We reported on NPD in an uncontrolled study involving 50 consecutively hospitalized children with uncomplicated mild-to-severe TBI who were recruited soon after injury and studied prospectively for 2 years.[2,34–36] NPD occurred in 45% of children (17/38) followed at 3 months post-injury, including 82% of children (9/11) with severe TBI and 30% of children (8/27) with mild-to-moderate TBI. Five of six models to account for NPD at 3 months were significantly predictive: severity of injury, lifetime psychiatric disorder, family psychiatric history, pre-injury family functioning, and socioeconomic status/pre-injury intellectual functioning.

We reported on NPD in a study of 24 consecutively hospitalized children with severe TBI, individually matched to 24 children hospitalized for mild TBI, and individually matched to 24 children hospitalized for OI.[10] The children were studied retrospectively an average of 2 years after injury. Severe TBI was associated with a significantly higher rate of NPD (15/24; 63%), compared with children with mild TBI (5/24; 21%) and orthopedic injury (1/24; 4%). These findings could not be attributed to age at injury or assessment, gender, race, social class, pre-injury psychiatric disorders, family psychiatric history, family stress, or injury-to-assessment duration.

In accordance with previous findings, we hypothesized that: 1) NPD 3 months post-injury would be significantly increased in the TBI versus the OI group; 2) pre-injury psychiatric disorder, pre-injury psychosocial

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adversity, and greater-intensity family psychiatric history would be significantly associated with NPD; and 3) severity of TBI would be related to NPD.

METHODS

Study procedures were approved by the institutional review boards of the participating organizations and complied with the NIH policies on human subjects. Participants with TBI or OI were recruited from consecutive admissions to medical centers in Dallas, Houston, and Miami. Inclusion of the OI group was intended to control for factors predisposing children to injury and for stress resulting from hospitalization. Children were age 7–17 years at the time of injury. Participants with TBI were included if they had a complicated mild-to-severe TBI. Severity of TBI classification was based on the lowest post-resuscitation score on the Glasgow Coma Scale (GCS),[32] which was recorded from clinical notes. The GCS is the standard measure of severity of acute brain injury associated with TBI. The scale measures motor, eye-opening, and verbal responsiveness. Scores range from 3 (unresponsive) to 15 (normal). Severe TBI was defined by GCS scores of 3–8, moderate TBI by GCS scores of 9–12, and complicated mild TBI by GCS scores of 13–15, with brain lesions (contusions, hematomas) indicated by computed tomographic scans. The OI patients had mild-to-moderate orthopedic injuries as defined by the Abbreviated Injury Scale.[37] The current investigation examined participants at baseline within 1 month after injury, and 3 months post-injury. All participants were English-speaking. Children were excluded if they had a previous head injury, penetrating gunshot wound to the brain, history of child abuse, preexisting neurologic disorders (e.g., mental retardation and epilepsy), pervasive developmental disorder, prematurity or low birth weight, hypoxia, or hypotension.

Psychiatric Measures

DSM-IV psychiatric diagnoses[38] were derived by utilizing a semistructured interview, the Schedule for Affective Disorders and Schizophrenia for school-aged children, Present and Lifetime Version (K-SADS-PL).[39] The K-SADS-PL is an integrated parent–child interview that generates diagnoses based on a clinician's synthesizing data collected from parent and child separately, querying symptoms that were present in the weeks before injury and pre-injury lifetime symptoms (at baseline), and symptoms present or past from injury to 3 months (at 3-month assessment). The entire interview was completed by the parent, including the attention-deficit hyperactivity disorder (ADHD) supplement, regardless of whether the ADHD screen was positive. The interview of the children was shortened to relieve burden on the children, who also completed an extensive neurocognitive battery. All children completed the depression and anxiety disorder sections, and children 13 years and older also answered the conduct disorder, drugs, and alcohol sections.

The Neuropsychiatric Rating Schedule (NPRS)[40] is a semistructured interview designed to identify symptoms and subtypes of personality change (PC) due to TBI.[24,41,42] Both parents and children served as informants in the interview, which took place at baseline and at 3 months after injury. We specifically waived the 1-year duration of symptomatology criterion to allow us to monitor symptomatology that was clinically significant. The instrument has been shown to provide reliable and valid diagnoses of the common subtypes of PC.[40] Previous studies have emphasized that PC is not a personality disorder, and its diagnosis does not require the assessment of personality. Rather, PC[38] is diagnosed when the child presents with clinically significant affective lability, aggression, disinhibition, apathy, or paranoia.[24,40–43]

Best-estimate psychiatric diagnoses[44] were generated by the interviewer after integrating the reports of the parent and the child from the NPRS and the K-SADS interviews and, when available, from the Behavioral Assessment System for Children, 2nd Edition (BASC-2)[45] and the Behavior Rating Inventory of Executive Function (BRIEF),[46] completed by the teacher. The methodology of diagnoses generated by the clinician from interview and questionnaire data provided by multiple sources is designed to minimize the risk of over- or under-reporting that may occur with questionnaire-derived data only.[47]

Predictive Variables

Family Psychiatric History The Family History Research Diagnostic Criteria[48] interview was conducted by trained research assistants at each site. Criteria were modified to conform to DSM-IV criteria. At least one parent acted as the informant and was questioned about psychiatric disorders in each first-degree relative of the index child with TBI. Family ratings were then summarized for

first-degree relatives, on a 4-point scale[49] of increasing severity — 0: no family psychiatric disorder; 1: at least one family member met criteria for a psychiatric disorder, but no treatment was received; 2: a family member met criteria for a psychiatric disorder and has received outpatient treatment or been arrested for antisocial behavior; 3: a family member met criteria for a psychiatric disorder and has had inpatient psychiatric treatment or has been incarcerated.

Socioeconomic Status (SES)

The Socioeconomic Composite Index (SCI)[50] was based on three variables: maternal education, coded on a 7-point scale, with values representing <7 years' education to attainment of a graduate degree; the Duncan Occupational Status Index;[51] and annual family income, based on an 8-point scale, ranging from <$20,000 to >$60,000, as part of the Life Stressors and Resources Scale (LISRES).[52] These three variables were transformed into z-scores and then averaged together to yield a composite z-score, which was standardized (mean: 0; standard deviation [SD]: 1).

Psychosocial Adversity Measure

The Life Stressors and Social Resources Inventory–Adult Form (LISRES–A)[52] was completed by parents. The Family Stressors score was computed as the mean of the T scores for the Stressors scales (Work, Health, Spouse, Extended Family, and Friends). The Family Resources score was defined as the mean of the T scores for Resource scales (Work, Spouse, Extended Family, and Friends).

Adaptive Functioning Measure

Pre-injury adaptive functioning was retrospectively assessed shortly after the injury by use of the Vineland Adaptive Behavior Scale interview.[53] This assessment involved a semistructured interview with the primary caretaker, conducted by a trained research assistant. The Adaptive Behavior composite standard score was the predictive variable of interest.

Data Analysis

The analyses conducted included comparisons between the TBI versus OI groups, NPD versus no-NPD groups, and participants versus nonparticipants at 3 months. Fisher's exact test and independent-sample *t*-tests were used to compare the groups for dependent variables that were categorical or continuous, respectively. To control for potentially confounding dependent variables, logistic regression was used when needed in analyses of NPD.

RESULTS

A group of 141 subjects were recruited and participated in the "baseline" psychiatric assessment to record pre-injury psychiatric diagnoses. These included 75 children with TBI and 66 children with OI. Table 1 shows pre-injury characteristics of the participants. The TBI group was significantly older than the OI group (mean age [SD]: 13.4 [2.8] versus 12.0 [2.5]; $t[139]=2.99$; $p=0.003$). The groups differed significantly by race, with the OI having a higher representation of Black/Biracial children versus Caucasian/Asian children and Hispanic/American Indian children ($\chi^2[2]=8.68$; $p=0.013$). The groups were not significantly different in gender, socioeconomic composite index, pre-injury adaptive functioning, pre-injury family stressors, pre-injury family resources, and family psychiatric history. Table 2 shows that the groups were not significantly different in rates of the general category of pre-injury psychiatric disorder or specific pre-injury psychiatric disorders.

Occurrence

In all, 118 of the original 141 eligible children (84%) returned for the 3-month psychiatric assessment. The returning group was not significantly different from the children who did not return in age, gender, socioeconomic composite index, pre-injury adaptive functioning, and pre-injury psychiatric disorder status. However, Black/Biracial participants were less likely to return for follow-up (11/37 Black/Biracial, 3/53 Caucasian/Asian, and 9/51 Hispanic/American Indian children did not return; $\chi^2[2]=9.35$; $p=0.009$).

The distribution of NPD that occurred in the TBI and OI groups is shown in Table 3. NPD at any point within the first 3 months post-injury occurred significantly more in the TBI group (32/65; 49%) than the OI group (7/53, 13%; Fisher's exact test: $p<0.0005$). Because age at injury was significantly different between the TBI and OI groups, we conducted a logistic-regression analysis, with NPD as the independent variable and group (TBI versus OI) and age at injury as the dependent variables. The regression was significant (−2 log likelihood $\chi^2[2]=18.93$;$p=0.0001$), although, of the dependent variables, only Group (TBI versus OI) was significant (Wald $\chi^2[1]=15.51$; $p=0.0001$), whereas age at injury was

TABLE 1. Demographics and Injury Characteristics , Mean (standard deviation)

	TBI (N=75)	OI (N=66)	*t*	df
Age at injury, mean (SD)	13.4 (2.8)	12.0 (2.5)	2.99	139
Male (%)	50 (67)	47 (71)		
Race				
Black	11	23		
White	32	20		
Hispanic	30	20		
American Indian	1	0		
Asian	0	1		
Biracial	1	2		
Socioeconomic Composite Index	−0.0443 (0.833) N=74	0.0967 (0.834) N=63	−0.99	135
Pre-injury Adaptive Functioning	95.3 (14.4) N=63	99.0 (11.1) N=58	−1.58	119
Pre-injury Family Stressors	46.7 (9.6) N=57	48.5 (10.7) N=57	−0.93	112
Pre-injury Family Resources	53.8 (10.3) N=58	54.8 (10.7) N=57	−0.51	113
Pre-injury Family Psychiatric History	1.24 (1.0) N=50	1.27 (1.1) N=59	−0.15	107
Mechanism of injury				
Auto, truck, bus (driver/passenger)	26	2		
Motorcycle/moped	7	5		
RV/off-road	7	1		
Bicycle	5	5		
Fall	12	13		
Falling object	0	1		
Sports/play	4	32		
Hit by motor vehicle (pedestrian)	13	3		
Other	1	4		

All comparisons were nonsignificant except age at injury (p=0.003).
The OI group had a higher representation of Black/Biracial children versus Caucasian/Asian children and Hispanic/American Indian children (p=0.013)
TBI: traumatic brain injury; OI: orthopedic injury; SD: standard deviation.

not significant. Similarly, NPD that was unresolved at the 3-month assessment occurred significantly more in the TBI group (32/65; 49%) than the OI group (5/53; 9%; Fisher's exact test: $p<0.0005$). A corresponding logistic regression controlling for age at injury was significant (−2 log likelihood $\chi^2[2]=24.09$; p=0.0001), although, of the dependent variables, only Group (TBI versus OI) was significant (Wald $\chi^2[1]=17.84$; p=0.0000), whereas age at injury was not significant. The specific NPDs that occurred significantly more in the TBI versus the OI group were Personality Change disorder (22/65, 34% versus 0/53; Fisher's exact test: $p<0.0005$) and Externalizing disorder (11/60, 18% versus 2/49, 4%; Fisher's exact test: p=0.035). The subtypes of personality change occurred as follows: affective lability (N=21); disinhibited (N=15), aggressive (N=8), apathetic (N=1). Subtypes of novel ADHD in the TBI cohort included Inattentive (N=2), Combined (N=1), and Not otherwise specified (N=3). Novel ADHD in the OI group was Inattentive (N=1).

The hypothesized predictors of NPD were first examined with the combined TBI and OI cohort (Table 4). Socioeconomic composite index, lifetime pre-injury psychiatric disorder, pre-injury adaptive functioning, pre-injury family stressors, and pre-injury family psychiatric history were not significantly related to NPD in the first 3 months after injury. Pre-injury family resources were nonsignificantly higher in the NPD group (p=0.096 [NS]); neither was age at injury associated with NPD. There was a trend for girls to have a higher rate of NPD (18/40 [45%] versus 21/78 [27%]; Fisher's exact test: p=0.063 [NS]). Also, there was a trend for Blacks/Biracial versus Caucasian/Asian and Hispanic/American Indian children to have a lesser rate of NPD (Fisher's exact test: p=0.087 [NS]). This was most likely due to the over-representation of this group in the OI group, which had a significantly lower incidence of NPD.

The analyses were repeated with the TBI group alone because of the relatively low frequency of NPD in the OI group. A very similar pattern was evident, in that socioeconomic composite index, lifetime pre-injury psychiatric disorder, pre-injury adaptive functioning, pre-injury family stressors, pre-injury family resources, pre-injury family psychiatric history, and age were not significantly related to NPD in the first 3 months after injury. In these analyses, race was not associated with

TABLE 2. Pre-Injury Psychiatric Disorders in Children With TBI and OI, N (%)

	TBI (N=75)	OI (N=66)
Pre-injury psychiatric disorder (current)	35 (47%)	28 (42%)
Lifetime psychiatric disorder (current + resolved)	39 (52%)	28 (42%)
Specific psychiatric disorders		
Pre-injury ADHD (current)	24 (32%)	22 (33%)
Lifetime pre-injury ADHD	25 (33%)	22 (33%)
Pre-injury oppositional-defiant disorder (current)	7 (9%)	4 (6%)
Lifetime pre-injury oppositional-defiant disorder	8 (11%)	5 (8%)
Pre-injury externalizing disorder (current)	28 (37%)	22 (33%)
Lifetime pre-injury externalizing disorder	29 (39%)	22 (33%)
Pre-injury depressive disorder (current)	5 (7%)	3 (5%)
Lifetime pre-injury depressive disorder	8 (11%)	3 (5%)
Pre-injury anxiety disorder (current)	13 (17%)	12 (18%)
Lifetime pre-injury anxiety disorder	16 (21%)	13 (20%)
Pre-injury internalizing disorder (current)	17 (23%)	13 (15%)
Lifetime pre-injury internalizing disorder	22 (29%)	14 (21%)
Pre-injury drug abuse (current)	2 (3%)	0 (0%)
Lifetime pre-injury drug abuse	2 (3%)	1 (2%)

All comparisons (Fisher's exact test) were nonsignificant.

TBI: traumatic brain injury; OI: orthopedic injury; ADHD: attention-deficit/hyperactivity disorder.

Externalizing disorder consists of attention-deficit/hyperactivity disorder, oppositional-defiant disorder, or conduct disorder; internalizing disorder consists of any depressive disorder (e.g., major depression; dysthymic disorder; depressive disorder, not otherwise specified), or any anxiety disorder.

TABLE 3. Psychiatric Disorders in the First 3 Months After TBI and OI, N (%)

	TBI (N=65)	OI (N=53)	p
Novel psychiatric disorder (current)	32 (49%)	5 (9%)	0.000
Novel psychiatric disorder (current + resolved)	32 (49%)	7 (13%)	0.000
Novel psychiatric disorders			
Personality change disorder (resolved)	22/65 (0)	0 (0)	0.000
ADHD (resolved)	6/46 (0)	1/32 (0)	NS
Oppositional-defiant disorder (resolved)	5/57 (0)	1/49 (0)	NS
Externalizing disorder (resolved)	11/60 (0)	2/49 (0)	0.035
Anxiety disorder (resolved)	10/65 (0)	4/53 (1)	NS
Depressive disorder (resolved)	5/59 (1)	1/51 (1)	NS
Internalizing disorder (resolved)	12/65 (0)	5/53 (2)	NS
Drug abuse (resolved)	1/64 (0)	1/52 (0)	NS

TBI: traumatic brain injury; OI: orthopedic injury; ADHD: attention-deficit/hyperactivity disorder.

The denominators fluctuate depending on eligibility to develop a specific NPD (e.g., a child with only pre-injury ADHD can develop oppositional-defiant disorder and therefore count as a novel externalizing disorder, but a child with pre-injury ADHD and ODD would not be eligible to develop a novel externalizing disorder. The drug abuse in the child with TBI consisted of both alcohol and cannabis abuse, and cannabis abuse alone in the child with OI.

NPD, but girls again showed a trend toward a higher rate of NPD (15/23 [65%] versus 17/42 [40%]; Fisher's exact test: p=0.072 [NS]).

Severity of injury, as measured by the Glasgow Coma Scale, did not predict NPD (Table 4). The incidence of NPD by severity of injury categories was as follows: severe TBI 22/39 (56%), moderate TBI 2/11 (18%), and complicated mild TBI 8/15 (53%).

DISCUSSION

This study is only the second prospective, controlled psychiatric study of pediatric TBI in which a standardized psychiatric interview assessment was conducted. The first study of its kind was published three decades ago, when assessment and treatment protocols were very different. Despite the passage of time, the findings of the two studies are remarkably similar. The major finding in the current study is that the development of NPD within the first 3 months after TBI (49%) occurs significantly more commonly than after OI (13%). As in most studies of pediatric TBI, NPD consisted of a heterogeneous set of specific disorders.[3,35] The most frequent NPD was personality change due to a general-medical condition,[24,41–43] characterized most commonly by affective lability, then disinhibition, aggression, rarely apathy, and no paranoia subtypes. The next most frequent disorders were internalizing disorder, externalizing disorder, anxiety disorder, ADHD, oppositional-defiant disorder, and depressive disorder. Only personality change and externalizing disorders occurred significantly more commonly in the TBI group. The TBI and OI groups were well matched and were not significantly different in socioeconomic status, gender, pre-injury adaptive functioning, pre-injury family stressors, pre-injury family resources, pre-injury family psychiatric history, a general category of pre-injury psychiatric disorder, and specific pre-injury psychiatric disorders. The OI group was significantly younger, but when age was controlled in regression analyses, Group (TBI versus

TABLE 4. Predictors of Novel Psychiatric Disorder 3 Months After TBI and OI

All Participants	Novel Psychiatric Disorder (N=39)	No Novel Psychiatric Disorder (N=79)	*t*	df	p
Age at injury	12.9 (3.2)	12.7 (2.6)	−0.35	116	NS
Gender					0.063
Male (%)	21/78 (27%)	57/78 (73%)			
Female (%)	18/40 (45%)	22/40 (55%)			
Race				2	0.087
Black	4 (15%)	22 (85%)			
White/Asian	20 (40%)	30 (60%)			
Hispanic/American Indian	15 (36%)	27 (64%)			
Socioeconomic Composite Index	0.023 (0.86)	0.092 (0.86)	0.41	116	NS
Lifetime pre-injury psychiatric disorder	21/39 (54%)	38/79 (48%)			NS
Pre-injury Adaptive Functioning	96.9 (11.5) N=35	95.9 (11.6) N=68	−0.38	101	NS
Pre-injury Family Stressors	47.6 (10.7) N=29	47.7 (9.9) N=70	0.06	97	NS
Pre-injury Family Resources	56.7 (9.4) N=30	52.8 (11.0) N=70	−1.68	98	0.096
Pre-injury Family Psychiatric History	1.4 (1.1) N=26	1.3 (1.0) N=65		89	NS
TBI Participants Only	**Novel Psychiatric Disorder (N=32)**	**No Novel Psychiatric Disorder (N=33)**	***t***	**df**	**p**
Glasgow Coma Scale score, mean (SD)	7.2 (4.9)	8.3 (4.2)	1.02	63	NS
Age at injury, mean (SD)	13.1 (3.2)	13.7 (2.6)	0.92	63	NS
Gender (%)					0.072
Male	17/42 (40%)	25/42 (60%)			
Female	15/23 (65%)	8/23 (35%)			
Race				2	NS
Black	4 (50%)	4 (50%)			
White/Asian	16 (53%)	14 (47%)			
Hispanic/American Indian	12 (44%)	15 (56%)			
Socioeconomic Composite Index	0.003 (0.87)	−0.090 (0.87)	−0.43	63	NS
Lifetime pre-injury psychiatric disorder	18/32 (56%)	15/33 (45%)			NS
Pre-injury Adaptive Functioning	95.9 (16.9) N=28	92.1 (10.3) N=27	−1.02	53	NS
Pre-injury Family Stressors	48.8 (11.2) N=22	44.9 (8.0) N=28	−1.42	48	NS
Pre-injury Family Resources	55.8 (10.3) N=23	52.9 (11.0) N=28	−0.98	49	NS
Pre-injury Family Psychiatric History	1.53 (1.02) N=19	1.04 (0.98) N=25	−1.60	42	NS

Values are mean (standard deviation), unless otherwise indicated.
TBI: traumatic brain injury; OI: orthopedic injury; SD: standard deviation.

OI) and not age at injury was significantly related to NPD. Race was significantly different between the TBI and OI groups, with an overrepresentation of Black/Biracial children in the OI group. Furthermore, Black/Biracial children had a significantly lower rate of follow-up at 3 months, such that participants at 3 months were not significantly different with regard to race.

Surprisingly, none of the other hypothesized predictive variables, including lifetime pre-injury psychiatric disorder, SES, pre-injury adaptive functioning, pre-injury family stressors, pre-injury family resources, pre-injury family psychiatric history, and severity of TBI were significantly related to NPD. Neither were age, gender, or race related to NPD. Larger studies may shed light on whether the trend for higher rates of NPD among girls holds true, and, if so, additional research on a possible hormonal influence on NPD may prove fruitful.

The principal data that guided our hypotheses regarding predictors of NPD were the 3-month follow-up results from our first prospective, uncontrolled psychiatric interview study of pediatric TBI.[2] These results demonstrated a significant relationship of NPD with severity of injury, lifetime psychiatric disorder, family psychiatric history, pre-injury family functioning, and socioeconomic status/pre-injury intellectual functioning. The main difference in the current study and our earlier study was that patients with uncomplicated mild TBI were excluded from the current study, but constituted 50% of the sample in the earlier study. The expansion of the range of injury severity would have the tendency to elicit a relationship of severity of injury to NPD because of expected lower rates in children with uncomplicated mild TBI. It is unclear why psychosocial variables were not significant predictors of NPD. One possible explanation is that over two-thirds of the cases classified as NPD were accounted for by personality change, which is significantly related to injury variables, rather than psychosocial variables, especially in the first year after TBI.[24,41,43] Different sets of predictors of NPD are significant, depending on time elapsed since injury.[2,34–36] It has been postulated that injury-related variables have a greater influence on outcome relative to psychosocial variables

the shorter the elapsed time since injury.[3] The opposite was found related to the specific outcome of oppositional-defiant symptoms, where the symptoms proved to be transient in less-severely injured participants and persistent in more-severely injured participants.[54]

There is the presumption that the reason that NPD was more frequent in the TBI versus the OI group is because brain damage combined with the emotional trauma inherent in children who suffer TBI is more potent in generating new-onset psychopathology than emotional trauma without brain damage in children with OI. Yet the relatively crude measure of severity of brain injury, the GCS, did not predict which children with TBI would develop NPD. A follow-up manuscript focuses on the association of NPD and structural neuroimaging modalities, including diffusion tensor imaging, volumetric analysis of regions of interest and lesions, and cortical thickness.[55]

The findings of this study must be considered within its limitations. Interrater reliability assessments for the diagnosis of NPD were not directly tested based on videotaped interviews. However, the child psychologists at each site closely supervised the assessments; and, furthermore, fidelity in diagnosis was maintained across sites by frequent telephone conferences and transmission of written summaries of psychiatric assessments that were critiqued by the first author and other interviewers, resulting in a consensus diagnosis. The psychiatric interviewers were not blind to the group status of participants (TBI or OI). However, in previous studies of pediatric TBI by our own group[10] and others,[3] unblinded and blinded ratings were very similar and did not influence the findings. As in many long-term follow-up studies, attrition was an issue, at 16%. However, participants versus nonparticipants at 3 months were not significantly different in multiple demographic and psychosocial variables, except that Black/Biracial children had a higher nonparticipation rate.

The study had a number of strengths. Three decades after the first, this was only the second, and now, largest, prospective, longitudinal, controlled psychiatric study of pediatric TBI using standardized interviews. The study used updated and in-depth psychosocial and injury-related variables to document pre-injury, injury, and post-injury status. The depth and breadth of measures were extensive and included interview measures of psychopathology, family psychiatric history, and adaptive functioning, as well as rating scales of injury and other psychosocial risk factors for NPD.

CONCLUSIONS AND IMPLICATIONS

Psychiatric disorders frequently follow brain injury in children and occur at significantly higher rates than after orthopedic injury without brain injury. TBI is common and is a major public health problem. Dealing with this problem requires intervention at the level of prevention of injury, early recognition, and treatment of post-injury psychiatric disorders. Understanding the biological basis of NPD and the functional impairments they induce will be important in the development and delivery of effective biopsychosocial treatments. Future manuscripts will examine the association of NPD with neurocognitive variables and also examine injury, neuroimaging, and psychosocial correlates of specific NPDs, such as personality change. The understanding of brain–behavior relationships will also be enhanced with the examination of the post-injury course of pre-injury psychiatric disorders, which are quite common.

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