Journal of the International Neuropsychological Society (1997), **3**, 617–630.

Preinjury family environment as a determinant of recovery from traumatic brain injuries in school-age children

KEITH OWEN YEATES,[1] H. GERRY TAYLOR,[2] DENNIS DROTAR,[2] SHARI L. WADE,[2] SUSAN KLEIN,[2] TERRY STANCIN,[3] AND CHRISTOPHER SCHATSCHNEIDER[4]

[1]Department of Pediatrics, The Ohio State University, and Columbus Children's Hospital
[2]Department of Pediatrics, Case Western Reserve University, and Rainbow Babies and Children's Hospital
[3]Department of Pediatrics, Case Western Reserve University, and MetroHealth Medical Center
[4]Department of Psychology, University of Houston, Houston, TX

Abstract

Previous studies of childhood traumatic brain injury (TBI) have emphasized injury-related variables rather than environmental factors as predictors of recovery. We addressed this concern using data collected during a prospective study of children with either TBI or orthopedic injuries (OI) and their families. Participants included 53 children with severe TBI, 56 with moderate TBI, and 80 with OI, all from 6 to 12 years of age at the time of injury. Measures of the preinjury family environment were collected shortly after the injury (baseline). Child cognitive and behavioral outcomes were assessed at baseline and at 6- and 12-month follow-ups. Individual growth curve analyses showed that measures of the preinjury family environment consistently predicted both the level of cognitive and behavioral functioning at 12 months postinjury and the rate of intraindividual change during the 12-month follow-up period, even after taking into account group membership and injury severity. In some cases, the preinjury family environment was a significant moderator of the effect of TBI, buffering its impact in high-functioning families and exacerbating it in low-functioning families. Thus, preinjury environmental factors predict recovery following TBI in children, even after accounting for injury-related variables. (*JINS*, 1997, *3*, 617–630.)

Keywords: Brain injury, Children, Family environment, Recovery

INTRODUCTION

The annual incidence of traumatic brain injuries (TBI) among children is approximately 180:100,000 (Kraus, 1995). Unfortunately, between 2 and 14% of pediatric TBI are fatal. Among survivors, moreover, TBI often causes significant neurobehavioral morbidity. Chronic intellectual sequelae include deficits in nonverbal skills, attention and memory, executive functions, and speeded motor performance, with attendant problems in school performance (Fletcher & Levin, 1988). Behavioral changes include increases in psychiatric difficulties, as well as declines in social competence and adaptive functioning, which often persist following resolution of cognitive deficits (Fletcher et al., 1990, 1996; Perrot et al., 1991; Shaffer, 1995). These sequelae, however, are not characteristic of all children with TBI; previous research has documented substantial variability in long-term outcomes following TBI, even among children with more severe injuries (Levin et al., 1995).

Both injury severity and preinjury environmental factors have been hypothesized to account for variability in neurobehavioral outcomes following TBI in children. However, most previous studies of childhood TBI have emphasized injury-related variables as predictors of recovery, and have neglected to assess outcomes relative to important environmental factors, such as socioeconomic status, social stressors and resources, or parent or family functioning (Fletcher et al., 1995). Although injury severity has been a consistent predictor of neurobehavioral outcomes following pediatric TBI, it does not account for most of the variance in out-

Reprint requests to: Keith Owen Yeates, Department of Psychology, Children's Hospital, 700 Children's Dr., Columbus, OH 43205. E-mail: yeates.1@osu.edu.

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

comes, leading to the speculation that environmental factors, such as family and school influences, also may be critical determinants of injury sequelae (Fletcher et al., 1990).

The hypothesis that environmental influences are critical determinants of neurobehavioral outcomes in childhood TBI receives indirect support from two major sources. First, studies of children with other chronic childhood illnesses have consistently shown that children's behavioral adjustment is linked to environmental variables such as socioeconomic status, family stressors and resources, and parental and family adjustment (Wallander & Thompson, 1995). The other major source of support for the importance of the environment is experimental studies of nonhuman animals, which have shown that environmental influences can have a major impact on behavioral functioning following experimental brain lesions (Kolb, 1989). Thus, studies of children with chronic illness, as well as experiments involving nonhuman animals, argue for a closer examination of the relative contributions of injury characteristics and the social environment as determinants of recovery from TBI.

Studies of the role of injury characteristics and the social environment as determinants of recovery from TBI must address several important issues. One issue is whether the environment moderates the impact of traumatic brain injury, by either buffering or exacerbating its adverse consequences. Environmental factors are clearly related to cognitive and behavioral functioning in normal and high-risk samples (Sameroff & Chandler, 1975). We would not be surprised if individual differences in functioning among children with TBI were also predicted by the social environment, independent of any relationships with injury severity. In contrast, the potential moderating effects of the social environment are much less certain, but of considerably more significance for understanding the consequences of TBI. That is, the social environment can be considered a significant influence on children's recovery from TBI only if it interacts with injury-related factors during the recovery process. In contrast, if the social environment simply affects the functioning of children with TBI in the same way that it does children without TBI, then it does not play a major role in determining the consequences of TBI *per se*. More practically, if injury-related and social factors interact during recovery from TBI, then rehabilitative efforts should be directed to both levels, because social intervention may facilitate a more rapid or complete recovery.

The second issue is the manner in which recovery from TBI is conceptualized. The impact of childhood TBI is best understood in terms of its consequences for subsequent development, rather than in terms of outcomes at any particular point in time postinjury (Fletcher et al., 1995). An understanding of the developmental sequelae of TBI depends on the study of intraindividual change over time, preferably through growth curve modeling (Francis et al., 1991; Fletcher et al., 1995). Growth curve modeling permits the outcomes associated with TBI to be characterized in terms of individual growth curves that are defined both by the rate at which recovery is proceeding and by the overall level of the outcome at a particular point in time postinjury. In growth curve models, the parameters describing rate of change and level of outcome are conceptually and statistically distinct, and can have very different relationships to other predictors (Francis et al., 1991). Previous research on childhood TBI has generally focused on level of outcome, and disregarded intraindividual change. Change is nevertheless a critically importance index of development that is potentially more sensitive to the effects of TBI and relatively free from the influences of preinjury status (Thompson et al., 1994).

A final issue is the possibility that the relative importance of injury severity and social factors varies in accordance with the type of outcome under consideration. In children with TBI, injury severity often accounts for similar amounts of variance in both cognitive and behavioral outcomes. However, the two types of outcomes are not strongly correlated, suggesting that their remaining variance may not be accounted for by the same influences (Fletcher et al., 1990). Previous research involving children who suffered from meningitis during early childhood found that social factors and acute-phase medical factors varied in their relative importance as predictors, depending on the specific outcome being considered (Taylor & Schatschneider, 1992). In general, social factors were more highly related to behavioral outcomes, whereas medical variables tended to be better predictors of cognitive performance. Similarly, in a study of prematurity and low birthweight, psychomotor skills were more closely related to medical risks, whereas language skills were more closely associated with social risk (Bendersky & Lewis, 1994). Based on these findings, we might anticipate that TBI severity would predict both cognitive and behavioral outcomes, but that the family environment would be more closely related to behavioral than to cognitive sequelae.

We attempted to address these issues using data collected from a prospective study of the effects of TBI on children and their families (Taylor et al., 1995). The study involved three groups of children, one with severe TBI, one with moderate TBI, and a comparison group with orthopedic injuries (OI). Information regarding premorbid child and family characteristics was collected during a baseline assessment shortly after the children's injuries. Child neurobehavioral outcomes were assessed at baseline and approximately 6 and 12 months postinjury. The data were analyzed using growth curve models to test three hypotheses regarding the influence of injury severity and the preinjury social environment on neurobehavioral outcomes.

Our first hypothesis was that differences between the TBI and OI groups would be greater at higher compared to lower levels of preinjury environmental risk. We expected that social risk would moderate the rate of change over time, as well as the level of functioning at 12 months postinjury. Thus, we predicted that children with TBI who were at higher social risk would display a slower rate of improvement cognitively and a steeper rise in behavior problems, as well as greater cognitive deficits and more severe behavior problems at 1 year postinjury, compared to children with TBI at lower social risk. Inclusion of the OI group permitted us to estimate the extent to which environmental risk was related to outcomes in children without TBI. By taking these influences into account, we were able to isolate the effects of environmental risk on recovery from TBI *per se*. Our second hypothesis was similar to the first, but pertained only to children with TBI. That is, we expected that when analyses were restricted to children with TBI, the effect of TBI severity on rates of change in cognitive and behavioral functioning over time, and on outcomes at 12 months postinjury, would be more pronounced for children at higher compared to lower preinjury environmental risk. Finally, our third hypothesis was that in both sets of analyses, preinjury environmental risk would be a better predictor of behavioral outcomes than of cognitive sequelae.

METHODS

Research Participants

The sample included a total of 189 children; 109 with TBI and 80 with OI, who were recruited from consecutive admissions to four hospitals in the midwestern United States. All children were between 6 and 12 years of age at the time of injury, and used English as their primary language at home. Children were excluded if they had a history of child abuse, previous neurological disorder, or mental retardation. Children were eligible for the TBI group if they sustained a blunt head trauma and their lowest postresuscitation Glasgow Coma Scale (GCS; Jennett & Bond, 1975) score was 12 or less, or if the GCS score was between 13 and 15 but was associated with an intracranial lesion on neuroimaging, skull fracture, neurological deficits, or sustained loss of consciousness (i.e., longer than 15 minutes). Children with TBI resulting from causes other than blunt head trauma (e.g., near-drowning, gunshot wound) were excluded. Children were eligible for the OI group if they sustained a bone fracture that required at least an overnight hospitalization but did not demonstrate any evidence of loss of consciousness or other indication of possible brain injury.

Following established conventions, the TBI group was divided into two groups based on injury severity (Fletcher & Levin, 1988). Children whose lowest postresuscitation GCS scores were 8 or less were considered to have *severe injuries*, and children with scores of 9 or more were considered to have *moderate injuries*. Many of the children in the moderate injury group had GCS scores ranging from 13 to 15, but they all demonstrated additional complications indicative of a more severe injury (e.g., intracranial lesion on neuroimaging, skull fracture, focal neurological deficits, or sustained loss of consciousness). Thus, consistent with previous research, their injuries were considered moderate rather than mild in severity (Fletcher & Levin, 1988).

Demographic features of the three groups (i.e., severe TBI, moderate TBI, OI) are summarized in Table 1. The groups did not differ in age at injury or sex. They also did not differ in maternal education, annual family income, or the Duncan occupational status index (Stevens & Featherman, 1981). The groups did differ in race, with a significantly higher proportion of non-Whites in the OI group compared to the two TBI groups [$\chi^2(2, N = 189) = 6.64, p < .05$].

As anticipated, the groups also differed in injury severity. The Injury Severity Score (ISS; Mayer et al., 1980) presented in Table 1 is based on all injuries the children sustained, whereas the partial ISS is calculated based only on injuries unrelated to TBI. As Table 1 shows, the severe TBI group suffered more severe injuries overall than either the moderate TBI group or OI group. In turn, the moderate TBI group suffered more severe injuries overall than the OI group. When injury severity was defined based only on injuries not involving the brain, the severe TBI and OI groups were comparable, and were both higher than the moderate TBI group. Thus, the severe TBI group had the most severe in-

Table 1. Demographics features of participants

Variable	Group					
	OI		Moderate TBI		Severe TBI	
n	80		56		53	
Child's sex (% male)	59		73		74	
Maternal ethnic status (% White)*	58		75		75	
Maternal education[a] (*M*, *SD*)	3.67	1.13	3.49	1.12	3.61	1.30
Family income[b] (*M*, *SD*)	3.45	2.77	4.22	2.81	3.65	2.81
Duncan occupational status index (*M*, *SD*)	32.40	19.68	32.44	18.31	32.59	20.62
Child's age at injury (years) (*M*, *SD*)	9.28	1.91	9.98	1.89	9.37	2.09
Glasgow Coma Scale score (*M*, *SD*)*	15.00	0.00	14.02	1.85	4.83	1.81
Injury severity score (*M*, *SD*)*	7.32	3.15	12.47	5.66	20.08	11.91
Partial injury severity score (*M*, *SD*)*	7.32	3.15	2.29	3.64	8.53	10.25

[a]Scale from 1 (*less than 7 years*) to 7 (*graduate degree*).
[b]Scale from 1 ($\leq$ *\$20,000*) to 8 ($\geq$ *\$60,000*).
*Groups differ significantly, $p < .05$.

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

juries overall, but did not differ from the OI group in the severity of injuries not involving the brain.

Procedures

All age-appropriate admissions to the four participating hospitals were monitored for potential eligibility. Once children meeting entry criteria were deemed medically stable, their parents were invited to participate in the study. After informed consent was obtained, baseline family interviews were conducted, during which demographic information and ratings of premorbid family characteristics were elicited. Parents also provided ratings of children's premorbid behavioral adjustment and adaptive functioning prior to or during the baseline assessment. Ratings of children's postinjury behavioral adjustment and adaptive functioning were elicited during follow-up interviews conducted approximately 6 and 12 months after the baseline assessments. In most cases, the respondent for interviews and questionnaires was the child's mother.

Baseline assessments of children's cognitive functioning were conducted as soon as possible after their injuries. In almost all cases, the baseline assessments occurred within 4 weeks of the injury. Prior to the baseline assessment, children in the TBI groups were screened for posttraumatic amnesia using the Children's Orientation of Amnesia Test (COAT; Ewing-Cobbs et al., 1990). They were not considered eligible for testing until their score on the COAT was within 2 standard deviations of the mean for their age for 2 consecutive days. The children completed follow-up assessments of their cognitive functioning approximately 6 and 12 months after the baseline assessment.

Across the entire sample, 149 children completed all three assessments, 20 completed two of the three assessments, and 18 completed only baseline assessments. The 2 remaining children did not complete any assessments, 1 because the family dropped out before the baseline assessment, and the other because the child was in a persistent vegetative state. The OI and TBI groups did not differ significantly in the proportion of children who completed at least two of the three assessments (86% *vs.* 92%, respectively), which was the minimum number required for the estimation of growth curve parameters. Eight families, 5 from the OI group and 3 from the TBI groups, were missing at least one of the measures of preinjury family status, and also were eliminated from data analyses.

Thus, 161 children had sufficient data available for growth curve analyses. They did not differ in age or sex from the 28 children without sufficient data. However, the children included in the analysis did demonstrate a higher proportion of Whites, and also scored higher on a composite measure of socioeconomic status than the children who were excluded. These differences did not vary across the OI and TBI groups. Among children with TBI, those who were included in the analyses displayed less severe injuries (i.e., higher mean GCS score) than those who were excluded. These results indicate that attrition and missing data may have reduced the generalizability of the findings in terms of race and socioeconomic status, but did not differentially affect the OI and TBI groups. The loss of more severely injured children from the TBI group may have reduced generalizability, making it less likely to detect differences in outcomes between the OI and TBI groups or as a function of TBI severity, in part because of a loss of statistical power.

Measures

Preinjury family environment

Four variables were selected as measures of the preinjury family environment and used as predictors of neurobehavioral outcomes. The four variables represent the major measures of the preinjury family environment collected as part of the larger prospective study (Taylor et al., 1995). They were selected to reflect both distal (i.e., socioeconomic status) and proximal (i.e., family functioning) influences on children's development, as well as more indirect risk factors (i.e., stressors and resources) that influence children even though they do not experience them directly (Bendersky & Lewis, 1994). The four variables included the Socioeconomic Composite Index (SCI), a summary measure of socioeconomic status; measures of overall social stressors and resources derived from the Life Stressors and Social Resources Inventory (LSSRI; Moos et al., 1989); and a measure of overall family functioning derived from the McMaster Family Assessment Device (FAD; Miller et al., 1985; Byles et al., 1988). Preliminary analyses revealed significant but modest correlations among the variables. The OI and TBI groups did not differ on any of the variables.

The SCI was based on three variables: maternal education, coded on a 7-point scale from *less than 7 years* to *graduate degree*; annual family income, coded on an 8-point scale from $\leq$ *$20,000* to $\geq$ *$60,000*; and the Duncan occupational status index (Stevens & Featherman, 1981). Because the three variables were moderately correlated, they were combined in a composite measure that was constructed by averaging z scores computed for each variable across the entire sample. The averaged z scores were then standardized ($M = 0$, $SD = 1$) to produce the SCI.

The LSSRI (Moos et al., 1989) is an extensive interview measure that generates standard scores for stressors and resources across a variety of domains. It has demonstrated satisfactory reliability and validity in prior research (Wade et al., 1996). For the current study, standard scores from the following subscales were averaged separately for stressors and resources: (1) personal health; (2) home and neighborhood; (3) work; (4) spouse or partner; (5) children; (6) relatives; and (7) friends and social activities. The averaged standard scores were then standardized ($M = 0$, $SD = 1$) across the entire sample to yield overall measures of preinjury stressors and resources. Subscales representing family finances and life events were omitted. The former was highly correlated with the SCI, whereas the latter, unlike

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

the other subscales, was correlated with injury severity, suggesting that parents had incorporated the experience of their child's injury into their ratings of life events.

Preinjury family functioning was assessed using the General Functioning Scale from the FAD. The FAD is a rating scale that has shown satisfactory reliability and validity in previous research (Miller et al., 1985; Byles et al., 1988). It is designed to assess family functioning across a variety of domains, and generates scores on seven subscales. It also generates an overall measure of family functioning, which is the 12-item General Functioning Scale. For this study, the scores from the General Functioning Scale were standardized across the entire sample ($M = 0$, $SD = 1$). Consistent with the scaling of the original scores, high scores reflect worse family functioning.

Neurobehavioral Outcomes

Cognitive outcomes were assessed at baseline and approximately 6 and 12 months postinjury using a comprehensive neuropsychological test battery. For the current study, we selected three measures of cognitive functioning from the larger test battery (Taylor et al., 1995) as dependent variables. The three measures were chosen because they tap domains of functioning (i.e., nonverbal skills and memory) that have been shown to be particularly sensitive to the effects of TBI in prior research (Levin et al., 1995). The first measure of cognitive functioning was a prorated Performance Scale IQ (PIQ) derived from a short form of the third edition of the Wechsler Intelligence Scale for Children (WISC–III; Wechsler, 1991). The short form included the Block Design and Object Assembly subtests, from which the prorated Performance Scale IQ was derived. Based on the formula presented by Sattler (1992), the prorated PIQ has a reliability of .85 and validity coefficient of .83. The PIQ is a measure of nonverbal skills sensitive to the acute effects of TBI in children (Fletcher & Levin, 1988). The second measure of cognitive functioning was the total raw score from the Developmental Test of Visual–Motor Integration (VMI; Beery, 1989). The VMI is a drawing task that requires visuoperceptual, constructional, and graphomotor skills. It has satisfactory reliability and validity, and has been shown to be sensitive to TBI in children (Thompson et al., 1994). The last measure of cognitive functioning was the total number of words recalled across five learning trials on a shortened, preliminary version of the children's California Verbal Learning Test (CVLT; Delis et al., 1994). The CVLT is a word-list learning task that measures verbal memory skills. Total recall on the CVLT is a reliable and valid measure of verbal memory that has been shown to discriminate between children with TBI and matched controls (Yeates et al., 1995).

Behavioral outcomes also were assessed at baseline and 6 and 12 months postinjury using the Child Behavior Checklist (CBC; Achenbach, 1991) and the Vineland Adaptive Behavior Scales (VABS; Sparrow et al., 1984). At baseline, parents were asked to respond based on children's premorbid functioning. The 6- and 12-month follow-up assessments measured postinjury functioning. The CBC is a well-known rating scale designed to assess disordered behavior. It was standardized on a large sample of community and clinic-referred children between the ages of 4 and 18, and has demonstrated satisfactory reliability and validity in previous research (Achenbach, 1991), although it has not always been sensitive to the effects of childhood TBI (Fletcher et al., 1990, 1996). For the current study, behavioral adjustment was measured using the total T score from the CBC. The VABS is a widely-used measure of adaptive functioning that has been shown to be sensitive to the effects of childhood TBI (Fletcher et al., 1990, 1996). The VABS is completed through a structured interview with a parent. For the current study, adaptive behavior was measured using the Adaptive Behavior Composite standard score from the VABS.

Statistical Analysis

Individual growth curve analyses (Francis et al., 1991) were used to examine quantitative change after injury. Analyses were performed on age-dependent raw scores when they were expressed in a meaningful metric (i.e., total words recalled on CVLT; total raw score on VMI). Age-corrected standard scores were used for the other measures (PIQ, CBC, VABC), which combine raw scores from multiple subtests or subscales.

Each outcome measure was subject to two analyses, one that involved children in both the TBI and OI groups and another that involved only children with TBI. In the first analysis, injury severity was defined based on group membership (OI, moderate TBI, severe TBI). Two dummy variables were used to define group membership. When entered together as predictors, the two dummy variables permitted comparisons between the OI and moderate TBI groups, and between the OI and severe TBI groups. For the analyses that were restricted to children with TBI, injury severity was defined in terms of the lowest post-resuscitation GCS score and treated as a continuous variable.

In all analyses, growth curves were characterized using the following model: $Y_{it} = \pi_{0i} + \pi_{li} a_{it} + R_{it}$, where the individual's (i) score at a given time (t), on the outcome measure designated Y, is modeled by the following parameters: π_{0i} = the intercept or expected performance level; π_{li} = the constant (or linear) rate of change around the intercept; a_{it} = time postinjury for person i at time t; and R_{it} = random error, the degree to which an observed score deviates from the model for person c_i at time t. For all analyses, time postinjury was centered at 1 year. Thus, the intercept parameter represents expected performance at 1 year postinjury, and the linear change parameter represents the constant rate of change per year or, by implication, the amount of change that occurred during the 1st year postinjury. Because data were available at only three time points, the model did not include a parameter representing the quadratic rate of change (i.e., nonlinearity). Prior studies of childhood TBI that have included higher-order components

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

suggest that nonlinear change accounts for relatively little variance in outcomes, particularly during the 1st year postinjury (Thompson et al., 1994).

The hierarchical linear models (HLM; Bryk & Raudenbush, 1987) method of analysis was used because it permits multivariate estimation of effects on the change parameters. That is, the HLM method evaluates the contribution of a predictor variable to one aspect of the growth curve (e.g., intercept) while controlling for the effects of that predictor on other aspects of the growth curve (e.g., linear change). The HLM method also provides improved estimation of individual growth parameters when not all participants are seen at all times (Francis et al., 1991). In the current study, some children did not complete all neuropsychological assessments. In other cases, the nature of their injuries precluded standardized administration of baseline testing (e.g., upper extremity cast, spica cast, traction). Children tested despite such circumstances displayed significantly lower baseline PIQ scores than children tested under optimal conditions. In contrast, their baseline performance on the VMI and the CVLT was not affected by the circumstances of testing. Thus, baseline PIQ data obtained under nonoptimal circumstances were excluded from analysis.

The statistical analysis consisted of two steps. In the first step, unconditional models were run on each outcome variable to examine the mean and variance of the growth curve parameters. Significance tests for both means and variances were conducted. Because of concerns about the power of the chi-square tests for variances in HLM, a parameter was allowed to remain random (i.e., to vary across participants) if the probability value of the chi-square was .10 or less or if a test of the variance-covariance components was significant when the parameter's variance was fixed to zero (cf. Thompson et al., 1994). A parameter was retained whenever its variance differed from zero. A parameter was also retained if its mean value differed from zero, even if the variance did not; in such cases, the parameter was considered to be nonrandomly varying (i.e., its residual variance was fixed to zero).

In the second step of the analysis, we tested a series of *a priori* conditional models to assess the contributions of the predictor variables to level of performance at 12 months postinjury and to linear change up to that point. Age at injury and ethnic status were included in all models. The first model added injury severity (i.e., group membership or lowest postresuscitation GCS score). The second model omitted injury severity but included the four environmental measures (i.e., SCI, LSSRI stressors, LSSRI resources, FAD General Functioning scale). The third model included both injury severity and the environmental measures. The three models are analogous to a series of hierarchical regression analyses, and permit an examination of the unique contribution of injury severity and preinjury family environment to the neurobehavioral outcomes. The fourth model, finally, added interaction terms representing the possible moderating effects of the preinjury family environment on injury severity.

We expected that the TBI groups would show poorer performance than the OI groups at 12 month postinjury in both cognitive and behavioral outcomes, and that the deficits would be more pronounced among children with more severe TBI. We also expected that the rate of change would be greater in the TBI groups than the OI group, and among children with more severe TBI. The direction of change, however, was expected to vary according to the type of outcome. Because all cognitive assessments were conducted postinjury, we expected to find greater improvement over time in the TBI groups than in the OI group, and among children with more severe compared to less severe TBI. In contrast, because the baseline assessment of behavioral outcomes was intended to assess preinjury status, we expected to find greater deterioration in the TBI groups than in the OI group, and among children with more severe TBI. Comparison of the two TBI groups (i.e., moderate and severe) to the OI group permitted us to examine the effects of TBI severity in the between-groups as well as within-group analyses.

RESULTS

Between-Groups Analyses

Unconditional models

The results of the between-groups unconditional models are summarized in Table 2. As expected, the estimated mean intercepts were significantly different from zero for all variables. In addition, four out of the five linear change parameters had an estimated mean significantly different from zero. For instance, at 12 months postinjury, the mean total raw score on the VMI for all children was estimated to be 25.37; in addition, their total raw score was estimated to increase an average of 1.89 points per year.

More critically, the estimated true parameter variance was significant for all intercepts and four out of five linear change parameters. In other words, the children displayed significant variation in their level of performance at 12 months postinjury on all variables. They also displayed significant variation in their rate of linear change across the 1st year postinjury for all measures except the CBC. For conditional models involving the CBC, the linear change parameter was retained but treated as nonrandomly varying (i.e., its residual variance was fixed to zero), because we expected the severe TBI group to show an increase in behavior problems during the 1st year postinjury when compared to the OI group. The linear change parameter for the VABC was also treated as nonrandomly varying in all conditional models. Although the parameter demonstrated significant residual variance in the unconditional model, tests of variance–covariance components in conditional models were not significant when its residual variance was fixed to zero.

Table 2 also displays the parameters' reliability, which is the ratio of estimated parameter variance (i.e., "true" variance) to the total parameter variance (i.e., "true" plus error

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

Table 2. Results of the between-group unconditional models

Outcome measure	Estimated parameter	*SE*	Estimated parameter variance	Total variance	Reliability	Parameter *r*
VMI						
Intercept	25.37**	0.88	114.91**	133.15	.86	
Linear	1.89*	0.63	18.20**	64.56	.28	.23
CVLT						
Intercept	38.83**	0.67	62.04**	77.35	.80	
Linear	5.56**	0.60	19.97**	59.62	.34	−.09
PIQ						
Intercept	103.60**	1.54	376.52**	403.56	.93	
Linear	6.22**	0.90	62.38**	130.78	.48	.42
VABC						
Intercept	94.45**	1.01	139.83**	167.06	.84	
Linear	−2.51*	0.77	26.60*	96.38	.28	.66
CBC						
Intercept	51.12**	0.92	114.50**	139.97	.82	
Linear[a]	−0.50	0.66	4.82	69.81	.07	.94

Note. The intercept represents the average performance at 12 months postinjury. The linear term represents the average amount of change across the first 12 months postinjury. VMI = Developmental Test of Visual Motor Integration total raw score. CVLT = California Verbal Learning Test total words recalled, Trials 1–5. PIQ = Prorated Performance IQ score. VABC = Vineland Adaptive Behavior Composite standard score. CBC = Child Behavior Checklist total *T* score.

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

*$p < .01$. ** $p < .001$.

variance). The reliability reflects the percentage of parameter variance that is potentially explainable by predictors in the conditional models. The reliability of the intercepts was considerably higher than that of the linear change parameters. The intercepts were estimated more reliably in part because the number of time points available for estimating linear change (i.e., three) was relatively small, but also because three of the outcome variables were expressed as standard scores, which afford less accurate estimation of change (Francis et al., 1991).

Finally, Table 2 also provides an estimate of the correlation between the intercept and linear change parameters. The correlation was low for the VMI and CVLT, moderately large for the PIQ and VABC, and very large for the CBC. The magnitude of the correlations reflects in part the scaling of the variables. Level of performance is less likely to be correlated with change when outcome measures are age-dependent raw scores (e.g., VMI, CVLT) as opposed to age-corrected standard scores (e.g., PIQ, CBC, VABC). The correlation for the CBC was particularly high, probably because of the scale's restricted floor and the skewed distribution of scores in the sample.

Conditional models

The results of the between-groups conditional models are summarized in Tables 3 and 4. For each between-groups model, Table 3 lists the individual predictors that contributed significantly to each outcome, along with the estimated coefficients for those predictors. Table 4 lists the total percentage of true parameter variance explained by each model.

Table 3 demonstrates the importance of controlling for ethnic status and age at injury when estimating level of outcome. Ethnic status was a significant predictor of the intercept parameter for all cognitive outcome measures, regardless of the specific model being tested, with scores being consistently higher for White compared to non-White children. Age at injury was a significant predictor of the intercept parameter for the VMI, CVLT, and VABC. As expected, older children performed better than younger children on the two cognitive variables that were age-dependent raw scores. An unexpected finding was that older children had a lower VABC standard score than younger children.

After controlling for ethnic status and age at injury, injury severity accounted for significant variance in the intercept parameters. When tested collectively, the two dummy variables representing group membership contributed significantly to the prediction of the CVLT, VABC, and CBC, even after controlling for the preinjury family environment (i.e., when testing Model 3). The contrast between the severe TBI and OI groups accounted for most of the differences in level of outcome (see Table 3). For instance, under Model 3 for the CVLT, the severe TBI group was estimated to recall a total of 5.93 fewer words at 12 months postinjury than the OI group, but there was not a significant difference between the moderate TBI and OI groups.

Even after controlling for group membership, the four family variables together accounted for significant variance in

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

Table 3. Significant predictors in between-groups models

Outcome measure	Conditional model: Model 1 (Age, Ethnic Status, Injury Severity)		Model 2 (Age, Ethnic Status, Environment)		Model 3 (Age, Ethnic Status, Injury Severity, Environment)		Model 4 (Model 3 + Interaction terms)	
				Parameter = Intercept				
VMI	Ethnic status	(7.42)	Ethnic status	(5.38)	Ethnic status	(5.70)	Ethnic status	(6.35)
	Age at injury	(3.56)	Age at injury	(3.52)	Age at injury	(3.52)	Age at injury	(3.54)
	Severe TBI	(−3.35)	SCI	(1.77)	SCI	(1.77)	Stress	(−2.17)
			Stress	(−1.57)	Stress	(−1.50)		
CVLT	Ethnic status	(5.34)	Ethnic status	(2.95)	Ethnic status	(3.96)	Ethnic status	(4.32)
	Age at injury	(1.98)	Age at injury	(1.95)	Age at injury	(1.99)	Age at injury	(2.11)
	Severe TBI	(−5.88)	SCI	(2.35)	Severe TBI	(−5.93)	Severe TBI	(−5.96)
					SCI	(2.30)	Severe TBI × FAD	(−3.27)
PIQ	Ethnic status	(18.94)	Ethnic status	(13.65)	Ethnic status	(14.12)	Ethnic status	(14.84)
			SCI	(7.53)	SCI	(7.55)		
VABC	Age at injury	(−1.80)	Age at injury	(−1.94)	Age at injury	(−1.83)	Age at injury	(−1.64)
	Severe TBI	(−6.72)	SCI	(5.02)	Severe TBI	(−6.29)	Severe TBI	(−6.46)
			Resources	(2.33)	SCI	(4.99)	Resources	(3.40)
					Resources	(2.34)	Severe TBI × FAD	(−5.83)
CBC	Moderate TBI	(7.40)	SCI	(−4.09)	Moderate TBI	(6.62)	Moderate TBI	(6.54)
	Severe TBI	(8.37)	Stress	(3.02)	Severe TBI	(7.34)	Severe TBI	(7.52)
					SCI	(−4.10)	Stress	(3.86)
					Stress	(2.36)		
				Parameter = Linear change				
VMI	Severe TBI	(3.15)	Stress	(−2.02)	Severe TBI	(3.90)	Severe TBI	(3.59)
					Stress	(−2.16)	Stress	(−2.26)
CVLT	None		Age at injury	(−0.63)	None		Moderate TBI × FAD	(−3.23)
PIQ	Moderate TBI	(4.83)	Stress	(2.42)	Moderate TBI	(4.24)	Moderate TBI	(4.36)
	Severe TBI	(8.01)			Severe TBI	(7.50)	Severe TBI	(7.56)
VABC	Age at injury	(−1.75)	Age at injury	(−1.86)	Age at injury	(−1.73)	Age at injury	(−1.64)
	Severe TBI	(−5.31)			Severe TBI	(−5.70)	Moderate TBI	(−3.20)
							Severe TBI	(−5.70)
							Severe TBI × FAD	(−4.30)
CBC	Moderate TBI	(3.09)	SCI	(−2.36)	Severe TBI	(6.34)	Moderate TBI	(3.23)
	Severe TBI	(6.75)			SCI	(−2.32)	Severe TBI	(6.66)

Note. Significant predictors are paired with estimated coefficients. VMI = Developmental Test of Visual Motor Integration total raw score. CVLT = California Verbal Learning Test total words, Trials 1–5. PIQ = Prorated Performance IQ score. VABC = Vineland Adaptive Behavior Composite standard score. CBC = Child Behavior Checklist total *T* score. SCI = Socioeconomic composite index standardized score. Stress = LSSRI average stressors standardized score. Resources = LSSRI average resources standardized score. FAD = Family Assessment Device general functioning scale standardized score.

the level of all of the outcome measures. For example, under Model 3 for the CVLT, children whose family SCI was 1 standard deviation above the sample mean recalled approximately 2.30 words more than average, whereas those whose family SCI was 1 standard deviation below the sample mean recalled 2.30 words less than average. Notably, Table 4 shows that preinjury family environment generally accounted for a larger percentage of variance in the level of outcome at 12 months postinjury than did group membership, and was an especially potent predictor of behavioral outcomes.

A different pattern emerged when modeling linear change parameters, with injury severity explaining more variance in change than the preinjury family environment. Collectively, group membership accounted for significant variance in change for all outcomes except the CVLT. In general, the TBI groups displayed more improvement in cognitive outcomes and more deterioration in behavioral outcomes than the OI group, and the amount of change was larger for more severe TBI. For instance, under Model 3 for the PIQ, the severe TBI group gained 7.5 more points across the first year postinjury than did the OI group, whereas the moder-

Table 4. Percentage of parameter variance explained by between-groups models

Outcome measure	Conditional model				
	Age, Ethnic Status	Model 1 (add Injury Severity)	Model 2 (add Environment)	Model 3 (add Injury Severity + Environment)	Model 4 (Model 3 + Interaction terms)
			Intercept		
VMI	51	52	57	57	57
CVLT	30	39	33	43	48
PIQ	19	19	28	29	27
VABC	2	5	28	30	31
CBC	0	6	26	31	30
			Linear change[a]		
VMI	1	8	4	13	0
CVLT	9	4	8	3	0
PIQ	5	19	11	23	16

Note. VMI = Developmental Test of Visual Motor Integration total raw score. CVLT = California Verbal Learning Test total words recalled, Trials 1–5. PIQ = Prorated Performance IQ score. VABC = Vineland Adaptive Behavior Composite standard score. CBC = Child Behavior Checklist total *T* score.

[a]The percentage of variance accounted for in the linear change parameter could not be computed for the VABC or CBC, because both parameters were considered to be nonrandomly varying in conditional models. Although the linear change parameter for the VABC was significant in the unconditional model, its residual variance was not significant in conditional models. Tests of variance–covariance components also were not significant, indicating that the linear change parameter for the VABC should be treated as nonrandomly varying in conditional models (i.e., its residual variance was constrained to zero). For both the CBC and VABC, the percentage of variance accounted for in the intercept was computed based on an unconditional model in which the linear change parameter was considered to be nonrandomly varying.

ate TBI group gained 4.24 more points. Similarly, for the VABC, the severe TBI group declined 5.70 more points than the OI group, but the moderate TBI and OI groups did not differ in their rate of change.

The four measures of the preinjury family environment, collectively, were significant predictors of change for only two of the outcome measures, the VMI and CBC. Several individual predictors were also significantly related to these outcomes. For instance, under Model 3, children showed more improvement over time on the VMI when their family's reported level of stress was lower than the sample mean, and less improvement when family stress was above average. Similarly, children displayed a decline in behavior problems as reported on the CBC when their family's SCI was higher than the sample mean, but did not do so when the SCI was below average.

Table 2 also shows four significant interactions between group membership and the preinjury family environment. The interactions involved both the intercept and linear change parameters. In all cases, family functioning as measured by the FAD moderated the effect of TBI, such that above-average functioning was associated with a more rapid and complete recovery following a TBI, whereas below-average functioning was associated with a slower and less complete recovery (i.e., less cognitive improvement and more behavioral deterioration during the 1st year postinjury, and worse outcomes at 12 months postinjury).

For illustrative purposes, Figure 1 portrays the interaction between the FAD and group membership for the CVLT intercept parameter. The figure shows that at 12 months postinjury, the difference in total recall between the severe TBI and OI groups was directly proportional to family functioning. The group difference was only 2.69 words for families whose FAD scores reflected above-average family function-

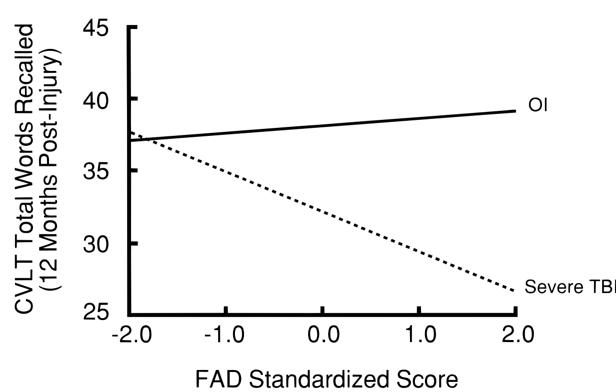

Fig. 1. Relationship between the preinjury standardized score on the Family Assessment Device (FAD) General Functioning scale and total words recalled on the California Verbal Learning Test (CVLT) at 12 months postinjury as a function of group membership.

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

ing (i.e., 1 *SD* below the sample mean). In contrast, the difference between the severe TBI and OI groups was 9.23 words for children whose families had FAD scores that reflected below-average family functioning (i.e., 1 *SD* above the sample mean). In other words, given a standard deviation of 5.68 for the CVLT intercept estimate, a difference on the FAD of 2 standard deviations resulted in a greater than 1 standard deviation increase in the discrepancy between the OI and severe TBI groups in total recall on the CVLT.

Within-Group Analyses

Unconditional models

The results of the within-group unconditional models, which involved only the children with TBI, were generally similar to those for the between-group models.[1] The mean intercepts were significantly different from zero for all outcomes, and the mean estimates of linear change were significantly different from zero for all measures except the CBC. Similarly, the estimated parameter variance was significant for all five intercepts and for all linear change parameters except that for the CBC, and the reliability of the parameter variance was again higher for the intercepts than for linear change.

The correlations between the intercept and linear change parameters were also similar, although the correlation was higher for the VABC in the within-group than the between-groups models. Nonetheless, the linear change parameter for the VABC was treated as random in the within-group models, because tests of residual parameter variance were generally significant, as were tests of variance-covariance components when the parameter was treated as nonrandomly varying.

Conditional models

For the intercept parameter, the results of the within-group conditional models were similar to those of the between-groups models. The results confirmed the importance of controlling for ethnic status and age at injury, especially when modeling the intercept. They also confirmed the importance of injury severity as a predictor of level of outcomes after TBI. Under Model 3, which controlled for the preinjury family environment, injury severity was a significant predictor of the level of outcome at 12 months postinjury for the CVLT and VABC. Finally, the results also confirmed the contribution of the preinjury family environment. Collectively, the four measures of the preinjury family environment were a significant predictor of all outcomes except for the VMI.

The results for the linear change parameter were also similar for the two sets of models, although injury severity was a less consistent predictor of change when only the children with TBI were considered. For instance, under Model 3, injury severity was a significant predictor of change for only the CBC. The lack of consistent relationships between injury severity and the rate of change may reflect the presence of more rapid change for all children with TBI, as compared to those in the OI group. In the between-group analyses, both the moderate and severe TBI groups showed more change than the OI group on three of the outcome measures (see Table 3). The relationship between injury severity and rate of change within the TBI group might have been stronger if the sample had included children with mild TBI.

The within-group models also provided less evidence for interactions between injury severity and the preinjury family environment. Nevertheless, there was one significant interaction, which occurred between the GCS and the LSSRI Resources standardized score when modeling the level of outcome at 12 months postinjury on the CVLT. The interaction was similar to those obtained in the between-group analyses. Specifically, the effect of TBI severity was reduced in families whose preinjury resources were above average and increased in families whose preinjury resources fell below average.

DISCUSSION

The findings indicate that preinjury environmental factors account for significant variability in neurobehavioral outcomes following TBI in children, over and above that explained by injury-related variables. After controlling for the family environment, age at injury, and ethnic status, TBI severity (i.e., group membership in between-group models; GCS score in within-group models) accounted for up to 20% of the variance in the level of outcome, and as much as 15% of the variance in the rate of change, following childhood TBI. In comparison, after controlling for injury severity and demographics, the four measures of the family environment accounted for up to 25% of the variance in level of outcome, and as much as 5% of the variance in rate of change. The results provide convincing support for the notion that environmental factors, including family influences, are critical determinants of the children's functioning after TBI, and must be considered along with injury-related variables in predicting neurobehavioral outcomes.

Even more critically, the findings are consistent with our hypothesis that the family environment is a significant moderator of the impact of TBI. More specifically, the deficits in memory and adaptive functioning associated with severe TBI were buffered by above-average family functioning and exacerbated by below-average family functioning. Children with severe TBI whose families were functioning poorly displayed less rapid recovery over time and lower functioning at 12 months postinjury than children whose families were functioning well. Given the low probability of detecting interactions in nonexperimental research designs (McClelland & Judd, 1993), the presence of multiple significant interactions, all of which were of a similar nature, argues

[1]Tables summarizing the results of the within-group unconditional and conditional models are available from the senior author.

strongly for the existence of a complex interplay between the damaged brain and its environmental context during recovery from TBI. More concretely, the findings provide clear evidence that the consequences of TBI in children are moderated by environmental factors.

These results are consistent with previous research on children with meningitis, which found that environmental risk exacerbated the impact of meningitis on a test of verbal skills (Taylor et al., 1993). Family risk factors also have been found to exacerbate the psychiatric sequelae associated with prematurity and very low birth weight (Breslau, 1995), although family factors were less predictive of developmental outcomes among children with the highest medical risk (Hack et al., 1992; Bendersky & Lewis, 1994). These findings suggest that the nature of the interaction between biological and social risk is likely to depend on the timing and nature of the biological insults that children sustain. Future research will be needed to determine how the role of the family environment following a biological insult varies according to age at injury, time since insult, age at testing, and the type of insult incurred (Taylor & Alden, 1997).

The results also confirm our hypothesis that the relative importance of injury characteristics and the preinjury family environment as predictors of recovery will vary across different types of outcomes. Injury severity accounted for a similar amount of variance in cognitive and behavioral outcomes. The preinjury family environment, on the other hand, was more closely related to behavioral outcomes than to cognitive outcomes. In the between-group growth curve analyses, the measures of the family environment accounted for approximately 25% of the variance in the VABC and CBC intercept parameters, but no more than 10% of the variance in the intercept parameters for the CVLT, VMI, or PIQ. A possible explanation for this pattern of findings is that cognitive functioning depends primarily upon the integrity of the central nervous system following a TBI, and hence is less affected by the family environment. In contrast, behavioral adjustment and adaptive functioning are likely to depend not only on the integrity of the central nervous system, but also on the many environmental contingencies that shape behavior.

The role of the environment may vary according to the specific type of risk factor being considered. In the current study, proximal measures of family functioning derived from the FAD were more likely to moderate the outcomes associated with severe TBI than were distal measures, such as the SCI, or measures of indirect influences, such as social stressors and resources. In contrast, distal measures and measures of indirect influences were more likely than proximal measures to be related to outcomes independent of TBI. These findings confirm the importance of considering the effects of distal and proximal variables separately. Proximal variables may be more likely than distal variables to change over time, and hence may exert more powerful influences on development (Aylward, 1992; Bendersky & Lewis, 1994). One implication of this notion is that the moderating influence of preinjury family functioning on both the rate of change and level of outcome following severe TBI might be a function of changes that occur in family functioning following the injury.

The influence of different social risk factors on children's functioning also may vary according to the type of developmental outcome that is assessed. In the current study, measures of social resources were related to adaptive functioning (i.e., VABC), whereas measures of social stressors were related to maladaptive functioning (i.e., CBC). The latter finding is consistent with research indicating that positive social support bolsters psychological well-being, but that negative support and social stress tends to result in negative psychosocial outcomes (Rook, 1984; Pagel et al., 1987). More generally, the finding also highlights the importance of what Bronfenbrenner and Crouter (1983) called *person–process–context* models of environmental effects, which postulate in part that specific aspects of the environment affect particular developmental functions.

Our findings also indicate that the preinjury family environment is related in distinct ways to different aspects of recovery. In the growth curve analyses, the measures of the preinjury family environment were more strongly related to the level of outcome than to the rate of change postinjury. Indeed, the environmental measures generally accounted for as much or more variance in the level of outcome than did indices of injury severity. In contrast, injury severity was as likely to be related to rate of change as to level of outcome, and consistently accounted for more variance in rate of change than did the preinjury family environment. These findings suggest that injury-related variables are important influences on the rate of recovery during the 1st year after a TBI, but that the eventual level of cognitive and behavioral functioning attained by the child depends as much if not more on environmental factors.

Of course, the relationship between injury severity and level of functioning is likely to vary according to the amount of time that has elapsed since the injury, and might have been stronger in the current study if we had chosen to center the intercept in the growth curve models at an earlier time (e.g., 1 month postinjury). Growth curve analyses require the specification of underlying models of intraindividual change and their associated parameters. Model specification depends in part on the research questions of interest. Our decision to center at 12 months postinjury reflected our interest in long-term outcomes. More generally, however, we chose to conduct growth curve analyses because of our interest in the changes that lead to long-term outcomes. We wanted to highlight the importance of conceptualizing recovery from TBI as a developmental process that involves intraindividual change over time. We believe our results illustrate the potential contributions afforded by studying rate of change in addition to level of outcome using growth curve models (Francis et al., 1991; Fletcher et al., 1995).

In our study, injury severity and the preinjury family environment accounted for considerably more of the variance in level of outcome than in the rate of change. In the between-

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

groups models, for instance, Model 3 accounted for as much as 60% of the variance in the intercept parameter, but no more than 25% of the linear change parameter. The comparatively small amount of variance accounted for in the rate of change may reflect the lower reliability of the linear change parameters as compared to the intercepts, a difference attributable both to the limited number of time points at which outcome data were collected and to the standard score format of several of the outcome measures. These weaknesses bolster the call for studies of TBI that involve prospective data collection at multiple time points, to increase the reliability with which linear change is estimated as well as to permit modeling of nonlinear change. They also highlight the need for outcome measures derived from interval-scaled test instruments sensitive to developmental variation across the ages under study (Thompson et al., 1994).

Another methodological limitation of the current study is that the sample may not be representative of the general population of children with TBI (Kraus, 1995). The selective loss of non-White children and of families of lower socioeconomic status may restrict the generalizability of the findings. The differential attrition of more severely injured children from the TBI group is a related concern. Perhaps a more problematic shortcoming is that the TBI and OI groups were not equivalent demographically at baseline, because they differed in race. Although ethnic status was controlled for in all statistical analyses, the nonequivalence of the groups on this important variable complicates between-group comparisons.

Measurement issues are also a concern in the current study. For instance, we had only one measure of proximal environmental influences (i.e., the FAD). The hypothesized role of proximal environmental factors as moderators of the impact of severe TBI is difficult to assess without measures of other variables, such as parental responsivity and the availability of daily stimulation, which have been found to be related to children's functioning independently of distal variables such as socioeconomic status (Gottfried & Gottfried, 1984). More refined measures of injury severity also would have been desirable. For instance, the relationship betwen injury severity and outcomes might have been stronger if we had obtained measurements of lesion volume (Levin et al., 1993, 1994). Finally, the scope of outcome measures was relatively limited. Future studies should include measures of other cognitive functions known to be affected by TBI, such as discourse skills and executive functions (Dennis & Barnes, 1990; Chapman et al., 1992; Dennis et al., 1996; Levin et al., 1997). Studies of more specific behavioral outcomes, particularly those linked to frontal lobe damage (Levin et al., 1991), also would be informative.

Despite these weaknesses, the results of the current study have important clinical implications. They suggest that rehabilitation programs must devote resources, not only to the child who has suffered a TBI, but also to the child's family. To the extent that the family environment is related to neurobehavioral outcomes, and actually moderates the effects of severe TBI, rehabilitative interventions must involve efforts to assess family functioning, identify families that are at risk, and foster better family functioning. The best means for promoting family functioning, unfortunately, are unclear. Although the current findings confirm the importance of the preinjury family environment as a predictor of neurobehavioral outcomes following childhood TBI, they afford little insight into the process by which the environment actually affects outcomes. Indeed, the factors that mediate the relationship between the preinjury family environment and neurobehavioral outcomes after childhood TBI remain to be explicated.

An understanding of the mechanisms by which the environment affects outcomes is likely to require study of postinjury family functioning. The current study considered only preinjury environmental risk factors, in part to simplify the causal assumptions reflected in the growth curve models. That is, by restricting ourselves to preinjury risk factors, we can safely conclude that the family environment affects children's postinjury functioning, rather than *vice versa*. At the same time, we must acknowledge that child outcomes are likely to be determined most directly by the postinjury family environment, and that the preinjury environment may only provide a rough gauge of family status postinjury. Examination of family circumstances after injury, including the family's perceived burden and the ways in which a family copes with changes in a child as a consequence of TBI, will be needed to better understand the processes by which the family environment influences child outcomes, as well as the reciprocal impact of child outcomes on the family environment. We plan to examine several possible models of this process using additional postinjury family data collected during this prospective study.

ACKNOWLEDGMENTS

The research presented here was supported by Grant MCJ-390611 from the Maternal and Child Health Research Bureau (Title V, Social Security Act), Health Resources and Services Administration, Department of Health and Human Services awarded to H. Gerry Taylor. The authors wish to acknowledge the contributions of Nori Mercuri Minich, Elizabeth Shaver, Madeline Polonia, Barbara Shapero, and Matt Diamond. We also wish to acknowledge the participation of the Children's Hospital Medical Center of Akron and the collaboration of Duane Bishop, Timothy Mapstone, Scott Maxwell, George Thompson, G. Dean Timmons, and Dennis Weiner. A preliminary version of this paper was presented at the meeting of the International Neuropsychological Society, Orlando, FL, February 1997.

REFERENCES

Achenbach, T.M. (1991). *Manual for the Child Behavior Checklist/ 4–18 and 1991 profile*. Burlington, VT: Department of Psychiatry, University of Vermont.

Aylward, G.P. (1992). The relationship between environmental risk and developmental outcome. *Journal of Developmental and Behavioral Pediatrics*, *13*, 222–229.

Beery, K.E. (1989) *Revised Administration, Scoring, and Teach-*

ing Manual for the Developmental Test of Visual–Motor Integration. Cleveland, OH: Modern Curriculum Press.

Bendersky, M. & Lewis, M. (1994). Environmental risk, biological risk, and developmental outcome. *Developmental Psychology*, *30*, 184–194.

Breslau, N. (1995). Psychiatric sequelae of low birth weight. *Epidemiologic Reviews*, *17*, 96–106.

Bronfenbrenner, U. & Crouter, A. (1983). The evolution of environmental models in developmental research. In W. Kessen (Ed.), *Handbook of child psychology: Vol. 1. History, theory, and methods* (pp. 357–414). New York: Wiley.

Bryk, A.S. & Raudenbush, S. W. (1987). Application of hierarchical linear models to assessing change. *Psychological Bulletin*, *101*, 147–158.

Byles, J., Byrne, C., Boyle, M.H., & Oxford, O.R. (1988). Ontario Child Health Study: Reliability and validity of the General Functioning Scale of the McMaster Family Assessment Device. *Family Process*, *27*, 97–104.

Chapman, S.B., Culhane, K.A., Levin, H S., Harward, H., Mendelsohn, D., Ewing-Cobbs, L., Fletcher, J.M., & Bruce, D. (1992). Narrative discourse after closed head injury in children and adolescents. *Brain and Language*, *43*, 42–65.

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

Dennis, M. & Barnes, M. (1990). Knowing the meaning, getting the point, bridging the gap, and carrying the message: Aspects of discourse following closed head injury in childhood and adolescence. *Brain and Language*, *39*, 428–446.

Dennis, M., Barnes, M.A., Donnelly, R.E., Wilkinson, M., & Humphreys, R.P. (1996). Appraising and managing knowledge: Metacognitive skills after childhood head injury. *Developmental Neuropsychology*, *12*, 77–103.

Ewing-Cobbs, L., Levin, H.S., Fletcher, J.M., Miner, M.E., & Eisenberg, H.M. (1990). The Children's Orientation and Amnesia Test: Relationship to severity of acute head injury and to recovery of memory. *Neurosurgery*, *27*, 683–691.

Fletcher, J.M., Ewing-Cobbs, L., Francis, D.J., & Levin, H.S. (1995). Variability in outcomes after traumatic brain injury in children: A developmental perspective. In S.H. Broman & M.E. Michel (Eds.), *Traumatic head injury in children* (pp. 3–21). New York: Oxford University Press.

Fletcher, J.M., Ewing-Cobbs, L., Miner, M., Levin, H., & Eisenberg, H. (1990). Behavioral changes after closed head injury in children. *Journal of Consulting and Clinical Psychology*, *58*, 93–98.

Fletcher, J.M. & Levin, H.S. (1988). Neurobehavioral effects of brain injury in children. In D.K. Routh (Ed.), *Handbook of pediatric psychology* (pp. 258–295). New York: Guilford.

Fletcher, J.M., Levin, H.S., Lachar, D., Kusnerik, L., Harward, H., Mendelsohn, D., & Lilly, M.A. (1996). Behavioral outcomes after pediatric closed head injury: Relationships with age, severity, and lesion size. *Journal of Child Neurology*, *11*, 283–290.

Francis, D.J., Fletcher, J.M., Stuebing, K.K., Davidson, K.C., & Thompson, N.M. (1991). Analysis of change: Modeling individual growth. *Journal of Consulting and Clinical Psychology*, *59*, 27–37.

Gottfried, A W. & Gottfried, A.E. (1984). Home environment and cognitive development in young children of middle socioeconomic status families. In A.W. Gottfried (Ed.), *Home environment and early cognitive development: Longitudinal research* (pp. 57–112). New York: Academic Press.

Hack, M., Breslau, N., Aram, B., Weissman, B., Klein, N., & Borawski-Clark, E. (1992). The effect of very low birth weight and social risk on neurocognitive abilities at school age. *Developmental and Behavioral Pediatrics*, *13*, 412–420.

Jennett, B. & Bond, M. (1975). Assessment of outcome after severe brain damage. *Lancet*, *1(7905)*, 480–487.

Kolb, B. (1989). Brain development, plasticity, and behavior. *American Psychologist*, *44*, 1203–1212.

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., Culhane, K.A., Mendelsohn, E., Lilly, M.A., Bruce, D., Fletcher, J.M., Chapman, S.B., Harward, H., & Eisenberg, H.M. (1993). Cognition in relation to magnetic resonance imaging in head-injured children and adolescents. *Archives of Neurology*, *50*, 897–905.

Levin, H.S., Ewing-Cobbs, L., & Eisenberg, H.M. (1995). Neurobehavioral outcome of pediatric closed-head injury. In S.H. Broman & M.E. Michel (Eds.), *Traumatic head injury in children* (pp. 70–94). New York: Oxford University Press.

Levin, H.S., Goldstein, F.C., Williams, D.H., & Eisenberg, H.M. (1991). The relationship of frontal lobe lesions to the neurobehavioral outcome of closed head injury. In H.S. Levin, H.M. Eisenberg, & A.L. Benton (Eds.), *Frontal lobe function and dysfunction* (pp. 318–338). New York: Oxford University Press.

Levin, H.S., Mendelsohn, D., Lilly, M., Fletcher, J.M., Culhane, K.A., Chapman, S.B., Harward, H., Kusnerik, L., Bruce, D., & Eisenberg, H.M. (1994). Tower of London performance in relation to magnetic resonance imaging following closed head injury in children. *Neuropsychology*, *8*, 171–179.

Levin, H.S., Song, J., Scheibel, R.S., Fletcher, J.M., Harward, H., Lilly, M., & Goldstein, F. (1997). Concept formation and problem solving following closed head injury in children. *Journal of the International Neuropsychological Society*, *3* (this issue).

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

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

Miller, I.W., Bishop, D.S., Epstein, N.B., & Keitner, G.I. (1985). The McMaster Family Assessment Device: Reliability and validity. *Journal of Marital and Family Therapy*, *11*, 345–356.

Moos, R.H., Fenn, C.B., Billings, A.G., & Moos, B.I. (1989). Assessing life stressors and social resources: Applications to alcoholic patients. *Journal of Substance Abuse*, *1*, 135–152.

Pagel, M.D., Erdly, W.W., & Becker, J. (1987). Social networks: We get by with (and in spite of) a little help from our friends. *Journal of Personality and Social Psychology*, *54*, 793–804.

Perrot, S., Taylor, H.G., & Montes, J. (1991). Neuropsychological sequelae, family stress, and environmental adaptation following pediatric head injury. *Developmental Neuropsychology*, *7*, 69–86.

Rook, K.S. (1984). The negative side of social interaction: Impact on psychological well-being. *Journal of Personality and Social Psychology*, *46*, 1097–1108.

Sameroff, A. & Chandler, M. (1975). Reproductive risk and the continuum of caretaking casualty. In F. D. Horowitz (Ed.), *Review of child development research* (Vol. 4, pp. 187–244). Chicago: University of Chicago Press.

https://doi.org/10.1017/S1355617797006176 Published online by Cambridge University Press

Sattler, J.M. (1992). *Assessment of children* (3rd ed.). San Diego, CA: Jerome M. Sattler.

Shaffer, D. (1995). Behavioral sequelae of serious head injury in children and adolescents: The British studies. In S.H. Broman & M.E. Michel (Eds.), *Traumatic head injury in children* (pp. 55–69). New York: Oxford University Press.

Sparrow, S.S., Balla, D.A., & Cicchetti, D. (1984). *Vineland Adaptive Behavior Scales.* Circle Pines, MN: American Guidance Service.

Stevens, G. & Featherman, D.L. (1981). A revised socioeconomic index of occupational status. *Social Science Research*, *10*, 364–395.

Taylor, H.G. & Alden, J. (1997). Age-related differences in outcomes following childhood brain insults: An introduction and overview. *Journal of the International Neuropsychological Society, 3* (this issue).

Taylor, H.G., Barry, C.T., & Schatschneider, C. (1993). School-age consequences of *Haemophilus influenzae* Type b meningitis. *Journal of Clinical Child Psychology*, *22*, 196–206.

Taylor, H.G., Drotar, D., Wade, S., Yeates, K.O., Stancin, T., & Klein, S. (1995). Recovery from traumatic brain injury in children: The importance of the family. In S. Broman & M. Michel (Eds.), *Traumatic head injury in children* (pp. 188–218). New York: Oxford University Press.

Taylor, H.G. & Schatschneider, C. (1992). Child neuropsychological assessment: A test of basic assumptions. *Clinical Neuropsychologist*, *6*, 259–275.

Thompson, N.M., Francis, D.J., Stuebing, K.K., Fletcher, J.M., Ewing-Cobbs, L., Miner, M.E., Levin, H.S., & Eisenberg, H. (1994). Motor, visual-spatial, and somatosensory skills after closed-head injury in children and adolescents: A study of change. *Neuropsychology*, *8*, 333–342.

Wade, S.L., Taylor, H.G., Drotar, D., Stancin, T., & Yeates, K.O. (1996). Childhood traumatic brain injury: Initial impact on the family. *Journal of Learning Disabilities*, *29*, 652–661.

Wallander, J.L., & Thompson, R.J., Jr. (1995). Psychosocial adjustment of children with chronic physical conditions. In M.C. Roberts (Ed.), *Handbook of pediatric psychology* (2nd ed., pp. 124–141). New York: Guilford.

Wechsler, D. (1991). *Manual for the Wechsler Intelligence Scale for Children–Third Edition*. New York: The Psychological Corporation.

Yeates, K.O., Blumenstein, E., Patterson, C.M., & Delis, D.C. (1995). Verbal learning and memory following pediatric closed-head injury. *Journal of the International Neuropsychological Society*, *1*, 78–87.