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Adolescents' internalizing problems following traumatic brain injury are related to parents' psychiatric symptoms

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Abstract

Background—A small body of previous research has demonstrated that pediatric traumatic brain injury increases risk for internalizing problems, but findings have varied regarding their predictors and correlates.

Methods—We examined the level and correlates of internalizing symptoms in 130 teens who had sustained a complicated mild to severe TBI within the past 1 to 6 months. Internalizing problems were measured via both maternal and paternal report Child Behavior Checklist. We also measured family functioning, parent psychiatric symptoms, and post-injury teen neurocognitive function.

Results—Mean parental ratings of internalizing problems were within the normal range. Depending on informant, 22–26% of the sample demonstrated clinically elevated internalizing problems. In multiple and binary logistic regression models, only parent psychiatric symptoms consistently provided unique prediction of teen internalizing symptoms. For maternal but not paternal report, female gender was associated with greater internalizing problems.

Conclusion—Parent and teen emotional problems are associated following adolescent TBI. Possible reasons for this relationship, including the effects of TBI on the family unit, are discussed.

Keywords

Closed head injury; neurobehavioral outcomes; pediatrics; anxiety; depression

Conflicts of interest
The authors have no conflicts of interest.

Introduction

Pediatric traumatic brain injury (TBI) can lead to numerous physical, cognitive, and behavioral problems, particularly when the injury is severe.[1] Attention to the emotional sequelae of pediatric TBI has been mounting recently as well.[2–4] A small body of research has now demonstrated that pediatric TBI increases the risk for internalizing problems, but findings have varied regarding the influence of injury, individual, and environmental factors.[3, 5–8] The current study examines the level and correlates of parent-reported internalizing symptoms in teens who recently sustained a complicated mild to severe TBI.

In a seminal prospective study, Brown and colleaugues[9] followed children who had sustained TBI or orthopedic injuries for 2 years postinjury. The group with severe TBI showed elevated rates of new-onset psychiatric diagnoses, whereas children who sustained orthopedic or milder brain injuries did not. Since then, research has confirmed that TBI conveys risk for novel psychiatric disorders, with the most common diagnoses including Attention-Deficit Hyperactivity Disorder (ADHD), Oppositional Defiant Disorder (ODD), anxiety disorders, and depressive disorders.[5, 8, 10–12] Specific rates of new-onset internalizing disorders have varied by study, with prevalence ranging from 7% to 44% for anxiety disorders and 0% to 26% for depressive disorders.[3, 7, 8, 10–13]This inconsistency probably owes to several factors, including both sample characteristics (e.g., age, injury severity) and methodological factors (e.g., specific diagnostic interview, DSM-III versus DSM-IV criteria). Duration of follow-up interval is one methodological factor that could be particularly important, as some evidence suggests that a substantial portion of novel internalizing disorders remit in the months or years following pediatric TBI.[8]

In addition to studies that utilized diagnostic interviews and thus emphasized categorical disorder status, related research has measured emotional-behavioral dysfunction with broad-band parent rating scales, such as the Child Behavior Checklist (CBCL[14]). Overall, findings from these studies demonstrate that moderate to severe TBI conveys risk for emotional-behavioral problems in general[15–19] and for internalizing symptoms in particular.[5, 6, 16] Two studies directly compared the categorical and dimensional approaches to measurement of psychiatric problems following pediatric TBI[20, 21] and found limited convergence between the two methods. Diagnostic interviews were more sensitive in detecting clinical problems other than ADHD. However, studies in this population that have classified internalizing disorders via cut-points in total symptom counts have sometimes identified a larger percentage of cases than have diagnostic interviews. Rates of internalizing disorders on parent CBCL following TBI were 20% or greater in three studies.[6, 15, 16] Further, broad-band rating scales offer a number of advantages. They are designed to provide a continuous measure of children's functioning in a variety of settings and thus may offer better insight into the full range of emotional-behavioral outcomes following TBI. Rating scales are also easier and faster to administer than diagnostic interviews.

Converging evidence supports a dose-response relationship between TBI severity and emotional-behavioral problems in children and adolescents,[5, 7, 9, 11, 15, 16, 19, 22] similar to that found for other neuropsychological deficits.[1] However, not every study has reported this relationship, with null results for both categorical[3, 8] and dimensional[6, 23, 24] approaches. Although neurocognitive and emotional-behavioral deficits each correlate with severity of TBI, these sequelae consistently show little or no relation to each other in pediatric populations.[6, 11, 13, 15, 23]

Other consistent predictors of poorer emotional-behavioral outcome following pediatric TBI include premorbid psychiatric problems and lower socioeconomic status (SES).[6, 9–12, 15] SES predicts outcomes from TBI rather broadly, but the mechanism for these effects

remains unclear.[1] More specific measures of family functioning, parental distress, and the social environment also relate to post-injury emotional-behavioral problems,[15, 25, 26] with some evidence for bidirectional influences between child and family functioning over time.[27]

There are theoretically motivated reasons to predict an effect of age at injury on new-onset internalizing symptoms, though the literature suggests that these effects may occur in either of two different directions. The broader TBI literature has established that younger age at injury is a robust risk factor for poorer outcomes in multiple domains.[28] Conversely, in the typically developing population, depression shows an approximately three-fold increase in adolescence.[29] While most research has reported no relation between age at injury and emotional-behavioral problems, two studies found that anxiety problems were more likely to follow TBI in younger than older children.[3, 13] Similarly, there is limited evidence for a relationship between gender and psychiatric outcome following TBI, though at least one study with adolescents reported higher levels of internalizing symptomatology in males than females.[16] Again, this finding is in contrast to the normal epidemiology of depression.[30]

TBI may raise risk for internalizing symptoms through direct neurologic disruption, secondary effects, or both. In adult populations, research has established an association between TBI and depression.[31] Further, post-TBI depressive symptoms in adults are associated with damage to left anterior brain regions,[32] consistent with a direct effect (although there may be indirect effects as well). Evidence for a relationship between damage to specific brain regions and internalizing symptoms in pediatric populations is more limited, though a recent study reported a link between anxiety symptoms and superior frontal gyrus lesions.[3] Risk for internalizing problems following pediatric TBI is elevated relative to orthopedically-injured control groups whose injuries did not include the head.[6, 7] While this pattern is consistent with a direct neurologic effect, this line of evidence is not conclusive. TBI-exposed children often demonstrate social, academic, and adaptive deficits not typically seen following orthopedic injury, all of which could possibly lead to secondary emotional consequences. In sum, pediatric TBI most likely leads to internalizing symptoms through both direct and indirect pathways, but definitive evidence pertaining to this question is still lacking.

The primary goal of the current study was to further investigate the level and correlates of internalizing symptoms in teens who had sustained a TBI. We expanded upon previous research in several ways. First, in the typically developing population, adolescence presents a vulnerable developmental period for internalizing problems, but most similar studies of TBI outcome have included younger children only or have collapsed results across a broad age range in order to obtain a sufficient sample size. We report data on internalizing problems in a larger sample of adolescents with TBI than has been described to date. Second, both maternal and paternal reports of teen internalizing problems were available for approximately half our sample. Most previous studies either relied exclusively on maternal report or have not compared results from both parents. In non-injured populations, mother-father correspondence for internalizing and externalizing problems is only moderate,[33] so explicit examination of paternal report should be informative. Third, pediatric TBI is known to increase caregiver distress[27, 34] but little previous research has investigated links between parents' psychiatric symptoms and the emotional functioning of their injured children.

We tested two primary hypotheses. Hypothesis 1: Adolescents with TBI will demonstrate an elevated level of internalizing problems on parent-report measures compared to national norms, with evidence for a dose-response relationship. Hypothesis 2: Teen internalizing problems will be associated with demographic/social variables (including SES, family function, and parent psychiatric symptoms[15, 25–27]) but not with post-injury neurocognitive

function. In addition, we conducted exploratory analyses to compare maternal and paternal report of teen functioning and to examine links between specific parent psychiatric symptom clusters and teen internalizing problems.

Method

Overview

The current study is part of a larger randomized clinical trial comparing the efficacy of two internet-based interventions: Counselor Assisted Problem Solving (CAPS), a 6-month web-based, family-centered intervention that focuses on problem solving, communication, and self regulation, and an internet resource comparison group. The parent study is being conducted at five major trauma-centers. Only data collected as part of an initial assessment at the family's home 1 to 6 months after injury and prior to the randomized intervention are presented here.

Participants

132 children (86 males) aged 12–17 years (M = 14.47; SD = 1.72), who were hospitalized overnight for a complicated mild to severe TBI and their families were initially recruited. To be eligible, participants had to display an alteration of neurological functioning as measured by a Glasgow Coma Scale (GCS) score less than 13 or evidence of neurological insult as seen on computerized tomography or magnetic resonance imaging. Additional inclusionary criteria included English as the primary language in the home, the ability for the adolescent with TBI to participate in the intervention, and residence within 3-hours drive of the hospital.

GCS scores were recorded in the medical records for 125 of the 132 participating adolescents. Fifty participants had severe TBI (GCS 3–8), 21 had moderate TBI (GCS 9–12), and 54 had complicated mild TBI (GCS 13–15 with evidence of neurological abnormality on CT or MRI). Following previous research,[6, 15, 23] we grouped individuals with the moderate and complicated mild injuries together into a single "less severe" or moderate group. For the remaining 7 participants without a documented GCS score, a clinical neuropsychologist at each site reviewed the medical record. Adolescents in this subset were considered tohave a severe TBI if the medical record indicated that the child was verbally unresponsive with no spontaneous eye opening or purposeful movementand a moderate TBI if there was an indication of impaired consciousness or abnormal imaging findings, but the child did not meet criteria for severe TBI. Two participants in the moderate group were removed from further analyses because CBCLs were missing for both parents. The final groups included 51 participants with severe TBI and 79 participants with moderate TBI.

Analysis of demographics

Of the 308 families initially identified as potentially eligible to participate in the study, 52 were found to be ineligible, 52 refused participation, 5 were unable to be contacted, and 67 were unable to be recruited during the initial 6 months post-injury. Age at injury, injury severity, and race were compared between the remaining 132 families that agreed to participate and non-participating families. Nonparticipants had less severe injuries on average and were more likely to be non-white.

Measures

Injury characteristics and demographic information—Information regarding injury severity was collected from relevant hospital records. SES information was collected from

the primary caregiver. An SES z-score was computed reflecting the combination of maternal education and census tract income.

Teen neurocognitive functioning—Teens completed several neurocognitive measures that were selected to assess constructs known to be affected by TBI (e.g.,[35, 36]). The 2-subtest version of the Wechsler Abbreviated Scale of Intelligence (WASI[37]) was administered to obtain an estimate of general intellectual ability. Processing speed was measured with the Processing Speed Index (PSI) of the Wechsler Adult Intelligence Scale-IV (WAIS-IV[38]) or Wechsler Intelligence Scale for Children-IV (WISC-IV[39]), depending on the age of the teen. The age-appropriate version of the California Verbal Learning Test,[40, 41] a repeated list-learning measure, was selected to assess verbal learning and memory. A T-score measuring total learning over 5 trials as well as z-scores indexing short- and long-delay free recall were used in analyses.

Teen emotional-behavioral functioning—Both parents were asked to complete the Child Behavior Checklist (CBCL[14]), a parental report that focuses on problematic behaviors in day-to-day family, social, and school situations. The CBCL has high test-retest reliability and criterion validity[14] and is sensitive to behavioral problems found following TBI.[15, 27] The CBCL provides Internalizing, Externalizing, and Total Behavior Problem composites, as well as a number of subscales. In the present study, we examined the Internalizing and Externalizing Problems composites, as well as subscales related to specific internalizing symptom clusters (Anxious/Depressed, Withdrawn/Depressed, and Somatic Complaints) and the subscales corresponding to related DSM diagnoses (DSM: Affective Problems, DSM: Anxiety Problems, and DSM: Somatic Problems). Composites and subscales are reported as T-scores. T-scores of 63 or greater on composites are considered clinically elevated. For subscales, T-scores of 65 to 69 are classified as borderline elevated, and T-scores of 70 and above are classified as clinically elevated. Given the many other measures administered to the adolescents as part of the larger study and to limit their respondent burden, they were not asked to complete parallel self-ratings of internalizing symptoms. To further evaluate whether emotional-behavioral difficulties caused clinically significant impairment, caregivers answered a yes/no question on the background questionnaire regarding whether the teen had been referred for counseling or behavior therapy after the injury.

Family functioning—The Family Assessment Device is a self-report measure of family functioning with established reliability and validity[42, 43]. A 12-item General Functioning scale (FAD-GF) was used as an index of family functioning. Mothers, fathers, and teens completed the FAD. Because teen internalizing symptoms were measured via parental report, common rater effects were avoided by using the teen-completed FAD in the primary analyses. Higher scores on the FAD-GF reflect greater family dysfunction, with scores of 2.16 of higher indicating clinically significant problems.

Parent psychiatric symptoms—The Global Severity Index (GSI) of the Symptom Checklist-90-R (SCL-90-R), a 90-item self-report inventory, provided a measure of global psychiatric symptoms and parental distress. The SCL-90-R has well-documented reliability and validity. The GSI score is reported as a T score, and scores greater or equal to 63 are considered to be indicative of clinically significant levels of distress[44]. The SCL-90-R also provides nine symptom scales: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism.

The Center for Epidemiologic Studies Depression Scale (CES-D) was used to assess specific symptoms of depression. It has well-established psychometric properties and is useful for screening individuals at risk for developing clinical depression. A raw score of 16 is typically used as a cutoff to indicate clinically significant depressive symptoms.[45]

Analyses

All variables were examined for extreme outliers and distributional properties. Several variables were positively skewed (> 1), including most CBCL subscales for both parents, father CES-D total, and some SCL-90 subscales. Remaining variables, including the CBCL Internalizing and Externalizing Problems composites, showed acceptable skew (absolute value < 1) and kurtosis (absolute value < 3). Primary analyses utilized non-parametric statistics for positively skewed variables, and parametric statistics in all other cases. Results were similar for parametric and non-parametric approaches.

Hypothesis 1 was that level of internalizing problems in adolescents with TBI would be elevated on parent-report measures compared to national norms, with evidence for a dose-response relationship. We tested this hypothesis primarily by examining the Internalizing Problems composite on maternal and paternal CBCLs. Analyses of the Externalizing Problems composite were included for comparison, and follow-up analyses investigated CBCL subscales. We computed one-sample t- tests to determine whether parents of adolescents with TBI reported elevated levels of emotional-behavioral problems in comparison to national norms. Relationship to injury severity was examined both categorically, using independent samples t-test to compare moderate and severe groups, and continuously, using Pearson correlations between GCS and CBCL T-scores.

Hypothesis 2 held that internalizing problems would be associated with demographic/social variables but not with post-injury neurocognitive function. We first characterized our sample in terms of family dysfunction, parent psychiatric symptoms, and post-injury teen neurocognitive function. To test hypothesis 2, we then computed multiple regression models in which gender, age, SES, family functioning, parent psychiatric symptoms, and post-injury neurocognitive function (i.e., CVLT Total Learning T-score, Wechsler PSI, and WASI FSIQ) were examined as predictors of teen internalizing symptoms. Father psychiatric problems were not included in the initial regression models because of the large amount of missing data due to single-parent households. These primary analyses treated internalizing symptoms continuously to reflect dimensional differences in symptom ratings. A parallel series of binary logistic regressions was also conducted to examine predictors of clinically elevated internalizing symptoms.

Missing data—Maternal CBCL datawere available for 127 of the 130 participants in the current study and paternal CBCL for 72 participants. In all but one case, CBCLs were missing because the mother or father was not a participant in the study. Due to time constraints or parent refusals, participating parents also failed to complete some of the parent self-report questionnaires. Data on parent psychiatric symptoms were missing for these reasons for 7 mothers and 2 fathers. Composite measures for questionnaires with missing item ratings were prorated based on the items completed.

Results

Sample Characteristics

Table 1 displays demographic information for the moderate and severe TBI groups. The groups were similar in terms of age at injury, gender, race, socioeconomic status (SES), and family composition.

The groups with and without paternal CBCL were similar in terms of age at injury, lowest GCS, and gender. Not surprisingly, individuals with paternal CBCL were more likely to live in two-parent households (90.3% versus 37.9%; $X^2(1) = 39.76$, $p < .001$). The majority of fathers (74.2%) from two-parent homes completed the CBCL. Rates of paternal reports were higher for white than non-white families (90.3% versus 70.7%; $X^2(1) = 8.19$, $p = .004$) and for families of higher SES (0.44 versus −0.48; $t(124.4) = -6.02$, $p < .001$). For the subset of adolescents living in two-parent households, rates of paternal reports continued to be associated with SES but were only marginally related to race.

Mother and father report of both internalizing and externalizing symptoms correlated significantly, though there was substantial unique variance in both cases (internalizing: $r = 0.45$, $p < .001$; externalizing: $r = 0.61$, $p < .001$). Paired-sample t-tests failed to find a significant difference between mother and father report of internalizing problems; however, mothers tended to report significantly more externalizing problems than fathers ($t(68) = 2.68$, $p = .009$). Consistent with results for the Internalizing Problems composite, Wilcoxon Signed Ranks Sum tests detected no significant differences in mother versus father report for any of the six internalizing subscales.

Hypothesis 1: Level of internalizing problems in adolescents with TBI will be elevated on parent-report measures compared to national norms, with evidence for a dose-response relationship

Table 2 shows mean maternal and paternal CBCL T-scores for the Internalizing and Externalizing Problems composites as well as the 6 internalizing subscales. Neither maternal nor paternal report of either internalizing or externalizing symptoms differed significantly for the moderate versus severe groups. Furthermore, continuous GCS did not correlate significantly with internalizing or externalizing symptoms for either parental report. However, caregiver report indicated that more teens with severe injuries had been referred for counseling or behavior therapy after injury, compared to teens with moderate injuries (38.0% versus 18.5%; $X^2(1) = 6.12$, $p = .01$).

For the total sample, maternal report of both internalizing and externalizing problems was elevated compared to national norms (Internalizing Problems composite: $t(126) = 3.79$, $p < .001$; Externalizing Problems composite: $t(126) = 3.42$, $p = .001$), although group means were well within the normal range. Paternal report was not statistically elevated in either case.

To examine rates of high internalizing symptoms in the sample, elevated scores on the CBCL were defined as T-scores of 63 or higher on the Internalizing Problems composite, and 65 or higher on the relevant subscales, cut-offs corresponding to the top 9% and 5% of the norming sample, respectively. On the Internalizing Problems composite, mothers identified 26.0% of the full sample as having clinically elevated symptoms relative to the norming sample [relative risk (RR) = 2.9]; this elevation was statistically significant ($Z = 4.37$, $p < .001$). Similarly, fathers identified 22.2% of participants as meeting this cut-off (RR = 2.5; $Z = 2.70$, $p = .007$). Chi-square analyses did not detect different rates of clinically elevated internalizing problems for the moderate versus severe groups for either maternal or paternal report [maternal report: 25.6% (moderate) vs. 26.5% (severe); paternal report: 26.8% (moderate) vs. 16.1% (severe)].

Some items contributing to the CBCL Internalizing Problems composite may reflect medical sequelae of TBI (e.g., “Underactive,” “Overtired without good reason,” or “Doesn’t eat well”). This is particularly true for items that are grouped in the Somatic Complaints and DSM: Somatic Problems subscales, and to a lesser extent, the Withdrawn/Depressed and DSM: Affective Problems subscales. It is much less characteristic of items contributing to

the Anxious/Depressed and DSM: Anxiety Problems subscales. We therefore examined rates of elevated problems on these two subscales. For maternal report, rates were significantly higher than the 5% population rate for both subscales (Anxious/Depressed: 15.0%, RR = 3.0, Z = 3.16, p = .002; DSM: Anxiety Problems: 16.5%, RR = 3.3, Z = 3.49, p < .001). For paternal report, rates were significantly elevated on the Anxious/Depressed subscale (16.7%, RR = 3.3, Z = 2.66, p = .008) and reflected a trend on the DSM: Anxiety Problems subscale (12.5%, RR = 2.5, Z = 1.92, p = .06).

Thus, both mothers and fathers identified their children with clinically significant internalizing problems at approximately 2–3 times the population rate. However, as would be expected based on the continuous correlation between maternal and paternal report, overlap of mother- and father-identified cases on the Internalizing Problems composite was far from complete. Of the 69 participants with both maternal and paternal CBCL, 6 individuals were identified as cases by both parents, 9 were identified by mother only, 9 were identified by father only, and 45 were identified by neither parent.

Hypothesis 2: Internalizing problems will be associated with demographic/social variables (including SES, family function, and parent psychiatric symptoms) but not with post-injury neurocognitive function

Parent psychiatric symptoms and family function—Both mother and father self-report of psychiatric symptomatology were significantly elevated on the SCL-90 GSI compared to national norms (mother mean T-score = 55.50; t (116) = 5.43, p < .001; father mean T-score = 54.64; t (66) = 2.91, p = .005). 26.5% of mothers and 37.3% of fathers endorsed clinically elevated symptoms on this index. Similarly, 33.1% of mothers and 27.1% of fathers endorsed clinically significant depressive symptoms on the CES-D. The SCL-90 GSI and CES-D total variables were highly correlated for each parent (mother: ρ = 0.69, p < .001; father: ρ = 0.69, p < .001). Thus, we created composite variables (mother psychiatric problems, father psychiatric problems) by averaging CES-D and SCL-90 GSI standardized z-scores for each parent. These composite variables had acceptable skew and kurtosis.

A substantial minority of the sample endorsed family dysfunction, whether assessed through mother, father, or teen report. Responses of 23.3% of mothers, 21.7% of fathers, and 35.4% of teens indicated clinically significant family dysfunction.

Post-Injury neurocognitive function—Consistent with the effects of TBI on cognitive function, the sample scored below normative means on tests of memory and processing speed. For all CVLT measures as well as the Wechsler PSI, the overall sample mean was significantly lower than the population mean. Sample means (SDs) and t-statistics were as follows: CVLT Total Learning T-score = 44.22 (12.65), t (119) = −5.21, p < .001; CVLT Short Delay Free Recall z-score = −0.57 (1.22), t (129) = −5.27, p < .001; CVLT Long Delay Free Recall z-score = −0.46 (1.23), t (129) = −4.26, p < .001; Wechsler PSI = 90.13 (16.57), t (129) = −6.79, p < .001. Average WASI FSIQ in our sample (98.79) did not differ significantly from the population mean.

Regression analyses of mother-reported internalizing problems—In regressions examining predictors of mother-reported Internalizing Problems T-scores, only female gender and greater mother psychiatric symptoms were associated with higher teen internalizing symptoms. The pattern of results was similar in a secondary multiple regression analysis conducted on the subset of the sample with both mother and father self reports of psychiatric distress, including father psychiatric problems as an additional independent variable, with only gender and mother psychiatric problems again contributing

to mother-reported internalizing symptoms. There was no indication of multicollinearity in either of the models (all VIF < 2). Multiple regression results are summarized in Table 3. In a binary logistic regression predicting mother-reported internalizing cases for the total sample, higher likelihood of caseness was significantly associated with female gender and greater mother psychiatric problems. In the subset of the sample with both mother and father self reports of psychiatric distress, higher likelihood of caseness was associated only with mother psychiatric problems. Binary logistic regression results are summarized in Table 4.

Regression analyses of father-reported internalizing problems—In parallel regressions of father-reported Internalizing Problems T-scores, only father psychiatric symptoms contributed significant unique variance, with greater father psychiatric problems being associated with greater teen internalizing symptoms (Table 3). Inclusion of mother psychiatric problems as an additional predictor variable did not change this result. There was again no indication of multicollinearity in either of these models (all VIF < 2). In the binary logistic regression predicting father-reported internalizing cases, higher likelihood of caseness was significantly associated with only father psychiatric symptoms (Table 4). Results were similar when mother psychiatric problems were included as an additional predictor.

Further exploration of relationship between parent and teen psychiatric symptoms—To investigate the potential effects of rater bias, we examined the bivariate correlations among mother psychiatric problems, father psychiatric problems, maternally-reported teen internalizing symptoms, and paternally-reported teen internalizing symptoms. Mothers' reports of their teens' internalizing problems correlated positively with both mothers' and fathers' symptoms, with similar (moderate) effect sizes ($r = .33$, $p < .001$ and $r = .34$, $p = .004$, respectively). Fathers' reports of their teens' internalizing problems also correlated positively with both fathers' and mothers' symptoms, but with a larger effect for their own symptoms ($r = .61$, $p < .001$) than for mothers' symptoms ($r = .27$, $p = .028$).

Finally, we explored whether there was specificity in the type of parent psychiatric problems that related to teen internalizing symptoms. We examined the Spearman correlations among maternal and paternal CBCL Internalizing Problems T-scores, mother SCL-90 subscales, and father SCL-90 subscales. Maternally reported internalizing symptoms correlated significantly and positively with most mother SCL-90 subscales, including Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, and Paranoid Ideation (ρ-values ranged from .19 to .35). However, across the two raters, maternally reported internalizing symptoms correlated significantly with only the father's SCL-90 Anxiety subscale ($\rho = .34$, $p = .006$). The pattern was similar for paternally reported internalizing symptoms, which correlated significantly with nearly all father SCL-90 subscales (Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Paranoid Ideation, and Psychoticism). Spearman ρ-values were larger than for mothers and ranged from .38 to .57. In a cross-rater analysis, paternally-reported internalizing symptoms correlated significantly only with mother's SCL-90 Somatization ($\rho = .28$, $p = .026$) and Anxiety subscales ($\rho = .25$, $p = .046$).

Discussion

The current study investigated the level and correlates of internalizing problems in 130 adolescents who had sustained a complicated mild to severe TBI in the previous 1 to 6 months. We replicated previous findings that internalizing symptoms are elevated in adolescents following a TBI, and that these symptoms are associated with the social environment. A number of novel results emerged. Mother and father report identified comparable proportions of teens with internalizing problems. However, consistent with

research in non-injured populations, there were substantial rater discrepancies, highlighting the importance of gathering information from multiple informants. Parent psychiatric symptoms consistently provided unique prediction of teen internalizing problems in regression analyses.

To our knowledge, this is the first study to compare maternal and paternal reports of their children's internalizing symptoms following a TBI. Discrepancies among different raters of children's emotional-behavioral functioning are consistently reported in the child psychopathology literature.[46, 47] In our study, there were some differences for the groups with and without paternal CBCL available. While analyses of paternal reports are likely to be informative, particularly since so little previous research has investigated such data, it is important to bear in mind that current results may not generalize to non-white or lower SES samples. Mother and father report of their teens' internalizing problems correlated significantly in our sample ($r = 0.45$), at a magnitude similar to that reported in non-injured populations.[46] Both parents reported comparable levels of internalizing symptoms. Further, reports of both parents yielded similar rates of cases with internalizing problems. However, agreement between mother- and father-identified cases was modest. Although the present focus was on internalizing symptoms, an important finding was that fathers reported lower levels of externalizing problems than mothers. This finding is surprising in light of the well established link between pediatric TBI and later externalizing problems,[48–51] to which maternal CBCL is sensitive.[15, 20, 21] Fathers may underestimate externalizing problems in this population, but further research is needed to confirm this result.

Consistent with our hypothesis, we obtained evidence for elevated risk of internalizing problems in adolescents following a TBI. The level of symptoms reported by mothers in our sample was significantly higher than in the norming sample, though mean ratings were well within the normal range. Both parents identified a statistically elevated proportion of the sample as having clinically elevated symptoms compared to the norming population. Depending on rater, cases occurred at rates of 22–26%, similar to previous studies. Although several items contributing to the CBCL Internalizing Problems composite could reflect direct medical sequelae, the pattern of results was similar for the anxiety subscales, which minimize this confound.

Elevated levels of internalizing symptoms compared to the norming sample could relate to differences in background characteristics, rather than to the effects of TBI. This explanation is made somewhat less likely by comparison to two other pediatric TBI studies that also included orthopedically injured controls.[6, 15] Both studies defined clinically elevated internalizing problems in an identical manner to the current study, and obtained rates that were very similar to ours. Kirkwood and colleagues[6] reported that 6 months post-injury, 20% of the TBI sample had elevated internalizing problems, compared to 7.3% of orthopedic controls, and group differences were statistically significant. Similarly, at an extended 4-year follow-up, Schwartz and colleagues[15] reported that 22% of the TBI sample had elevated internalizing problems. This figure was not directly compared to orthopedic controls. However, rates of total emotional-behavioral problems were consistently higher in children with TBI than in those with orthopedic injuries, and rates of internalizing and externalizing problems in the TBI group were similar.

Contrary to our prediction, little evidence for a link between injury severity and emotional outcome was found. There were no differences for injury severity groups on maternal or paternal CBCL. While a dose-response relationship between TBI severity and emotional-behavioral outcome is typically reported, several null results for both categorical and dimensional approaches have also been noted. Despite the similarity in parent-reported symptoms for adolescents with moderate or severe TBI, those with severe TBI were

significantly more likely to have been referred for counseling or behavior therapy following their injuries. Several possible explanations could help to account for the discrepancy in results. Parents of children with more severe injuries may be more likely to seek or to be offered help, or the same level of emotional-behavioral symptomatology may cause more significant functional impairment in the context of a severe TBI. Alternatively, our instrumentation might not have been sufficiently sensitive to detect true symptom differences associated with the need for intervention.

Our findings were consistent with previous research and demonstrated a link from teen internalizing symptoms to the social environment but not to post-injury neurocognitive function. Parent psychiatric symptoms consistently provided unique prediction of teen internalizing symptoms in regression models, and cross-rater analyses suggested that maternal and paternal anxiety in particular related to the presence of internalizing problems in teens with TBI. Mothers but not fathers reported more internalizing symptoms in girls than boys. Gender differences have rarely been reported in the pediatric TBI literature. Mothers may be more attuned than fathers to their daughters' internalizing problems. Alternatively, distressed mothers may over-report their children's problems (i.e., the depression-distortion hypothesis[52]), particularly for girls. When controlling for all other variables in the model, we did not find evidence for an association between age at injury, SES, or family functioning and teen internalizing symptoms. Previous research has documenteda relationshipbetween SES or family functioning and teen emotional functioning following TBI.[6, 11, 15]However, none of these studies included measures of parent psychiatric symptoms in addition toSES and family functioning in modeling child behavior outcomes. To the extent that these measures are correlated, including all three factors as predictors would diminish the unique effects of each in the model and thus may have made it more difficult to detect effects of SES and family functioning.

Serious TBI represents a major stressor for the whole family.[34] One longitudinal study reported that parent distress and child emotional-behavioral problems showed bidirectional influences in the year following children's TBI. The child's brain injury had a direct effect on both parent and child functioning 6 months later, which then showed reciprocal relations over the following 6 months.[27] Parent psychopathology and family dysfunction may be synergistic with the effects of brain injury, such that an adolescent's TBI could increase the negative effects of parent and family adjustment problems on the emotional functioning of the adolescent. This hypothesis offers one explanation for the current findings, and makes specific predictions that can be tested with a future longitudinal study.

Several other explanations exist for the association between parental psychiatric symptoms and teen internalizing problems following TBI. Parent psychopathology in general, and anxiety in particular, is known to influence parents' ratings of their children's behavior.[47] In the current sample, this apparent bias was more evident for fathers than mothers. Fathers' ratings of their own distress correlated strongly with their ratings of teens' internalizing problems, but moderately to mothers' ratings of teens' internalizing problems. Mothers' self-reported distress related similarly to both mother and father ratings of teens' internalizing problems. For both parents, regression analyses demonstrated that self-reported psychiatric symptoms uniquely predicted that parent's rating of the teen's internalizing problems but not the other parent's rating of teen internalizing problems. Rater bias may have contributed to the current findings, but appears unlikely to fully account for the key results. A third possible explanation is that parental distress or anxiety serves as a proxy for adolescents' pre-injury risk for developing internalizing problems in response to a major stressor. Supporting this idea is the fact that family history has sometimes been found to be a significant predictor of novel psychiatric disorders following TBI.[11] Finally, overall family stress may also produce internalizing problems in both parents and children when no direct

link between the two exists or when parents are worried about their adolescent's internalizing problems.

The current study included a number of limitations. Our methods are similar to those used by several previous studies,[16, 24, 27, 53] but they excluded ratings by the adolescents themselves or by observers outside the family (e.g., teachers). The latter ratings could have allowed more sensitive assessment of internalizing, as adolescents may not share their perceptions of internalizing symptoms with parents. A second limitation is that, as part of a larger randomized clinical trial for TBI intervention, we did not include a control group without brain injuries and comparisons were thus made relative to national norms instead. We did not measure premorbid emotional functioning, which limited our ability to draw firm conclusions about the temporal relationship between TBI and internalizing problems. Previous research has documented that pre-injury psychopathology conveys higher risk for developing novel post-injury emotional problems.[12, 13] In the current study, teen internalizing symptoms and social environment variables were all assessed at a single time point within 6 months of injury. Thus, our results cannot shed light on the causal relations among these variables nor on the natural history of internalizing symptomatology following TBI.

Despite these limitations, the current study makes a valuable contribution to the small body of research investigating emotional problems following pediatric TBI. Our results indicate that attention to internalizing symptoms is likely to be an important part of the overall treatment plan for a substantial portion of adolescents during the initial months following TBI. Moreover, our findings are relevant to understanding the impact of TBI on the family unit. Current results suggest the possibility of complex inter-relationships between parent and teen psychopathology that should be investigated further in future research. Clinically, these results suggest that evaluating and addressing parental anxiety may provide an avenue for ameliorating adolescents' internalizing symptoms following TBI. Family-centered interventions focused on the reciprocal influences between parents and teens may also promote more successful adaptation over time.

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References

1. Yeates, KO. Traumatic brain injury. In: Yeates, KO.; Ris, MD.; Taylor, HG.; Pennington, BF., editors. Pediatric neuropsychology: Research, theory, and practice. 2. New York, NY US: Guilford Press; 2010. p. 112-46.
2. Grados MA, Vasa RA, Riddle MA, Slomine BS, Salorio C, Christensen J, et al. New onset obsessive-compulsive symptoms in children and adolescents with severe traumatic brain injury. Depression and Anxiety. 2008; 25(5):398–407. [PubMed: 17957806]
3. Max JE, Keatley E, Wilde EA, Bigler ED, Levin HS, Schachar RJ, et al. Anxiety disorders in children and adolescents in the first six months after traumatic brain injury. The Journal of Neuropsychiatry and Clinical Neurosciences. 2011; 23(1):29–39. [PubMed: 21304136]
4. Noggle CA, Pierson EE. Psychosocial and behavioral functioning following pediatric TBI: Presentation, assessment, and intervention. Applied Neuropsychology. 2010; 17(2):110–5. [PubMed: 20467951]

5. Max JE, Koele SL, Smith WL, Sato Y, Lindgren SD, Robin DA, et al. Psychiatric disorders in children and adolescents after severe traumatic brain injury: A controlled study. Journal of the American Academy of Child and Adolescent Psychiatry. 1998; 37(8):832–40. [PubMed: 9695445]
6. Kirkwood M, Janusz J, Yeates KO, Taylor HG, Wade SL, Stancin T, et al. Prevalence and correlates of depressive symptoms following traumatic brain injuries in children. Child Neuropsychology. 2000; 6(3):195–208. [PubMed: 11402397]
7. Luis CA, Mittenberg W. Mood and anxiety disorders following pediatric traumatic brain injury: A prospective study. Journal of Clinical and Experimental Neuropsychology. 2002; 24(3):270–9. [PubMed: 11992209]
8. Bloom DR, Levin HS, Ewing-Cobbs L, Saunders AE, Song J, Fletcher JM, et al. Lifetime and novel psychiatric disorders after pediatric traumatic brain injury. Journal of the American Academy of Child and Adolescent Psychiatry. 2001; 40(5):572–9. [PubMed: 11349702]
9. Brown G. A prospective study of children with head injuries: III. Psychiatric sequelae. Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences. 1981; 11(1): 63–78.
10. Max JE, Lindgren SD, Robin DA, Smith WL. Traumatic brain injury in children and adolescents: Psychiatric disorders in the second three months. Journal of Nervous and Mental Disease. 1997; 185(6):394–401. [PubMed: 9205426]
11. Max JE, Robin DA, Lindgren SD, Smith WL Jr, Sato Y, Mattheis PJ, et al. Traumatic brain injury in children and adolescents: Psychiatric disorders at two years. Journal of the American Academy of Child & Adolescent Psychiatry. 1997; 36(9):1278–85. [PubMed: 9291730]
12. Max JE, Smith WL Jr, Sato Y, Mattheis PJ. Traumatic brain injury in children and adolescents: Psychiatric disorders in the first three months. Journal of the American Academy of Child & Adolescent Psychiatry. 1997; 36(1):94–102. [PubMed: 9000786]
13. Vasa RA, Gerring JP, Grados M, Slomine B, Christensen JR, Rising W, et al. Anxiety after severe pediatric closed head injury. Journal of the American Academy of Child and Adolescent Psychiatry. 2002; 41(2):148–56. [PubMed: 11837404]
14. Achenbach, TM.; Rescorla, LA. Manual for the ASEBA School-Age Forms & Profiles. Burlington VT: University of Vermont, Research Center for Children, Youth, & Family; 2001.
15. Schwartz L, Taylor HG, Drotar D, Yeates KO, Wade SL, Stancin T. Long-term behavior problems following pediatric traumatic brain injury: Prevalence, predictors, and correlates. Journal of Pediatric Psychology. 2003; 28(4):251–63. [PubMed: 12730282]
16. Geraldina P, Mariarosaria L, Annarita A, Susanna G, Michela S, Alessandro D, et al. Neuropsychiatric sequelae in TBI: A comparison across different age groups. Brain Injury. 2003; 17(10):835–46. [PubMed: 12963550]
17. Taylor HG, Yeates KO, Wade SL, Drotar D, Stancin T, Burant C. Bidirectional child, Äìfamily influences on outcomes of traumatic brain injury in children. Journal of the International Neuropsychological Society. 2001; 7(6):755–67. [PubMed: 11575597]
18. Yeates KO, Taylor HG, Barry CT, Drotar D, Wade SL, Stancin T. Neurobehavioral symptoms in childhood closed-head injuries: Changes in prevalence and correlates during the first year postinjury. Journal of Pediatric Psychology. 2001; 26(2):79–91. [PubMed: 11181884]
19. Barry CT, Taylor HG, Klein S, Yeates KO. Validity of neurobehavioral symptoms reported in children with traumatic brain injury. Child Neuropsychology. 1996; 2(3):213–26.
20. Wassenberg R, Max JE, Koele SL, Firme K. Classifying psychiatric disorders after traumatic brain injury and orthopaedic injury in children: Adequacy of K-SADS versus CBCL. Brain Injury. 2004; 18(4):377–90. [PubMed: 14742151]
21. Green ML, Foster MA, Morris MK, Muir JJ, Morris RD. Parental assessment of psychological and behavioral functioning following pediatric acquired brain injury. Journal of Pediatric Psychology. 1998; 23(5):289–99. [PubMed: 9782677]
22. Donders J, Ballard E. Psychological adjustment characteristics of children before and after moderate to severe traumatic brain injury. The Journal of Head Trauma Rehabilitation. 1996; 11(3):67–73.

23. Fletcher JM, Ewing-Cobbs L, Miner ME, Levin HS, Eisenberg HM. Behavioral changes after closed head injury in children. Journal of Consulting and Clinical Psychology. 1990; 58(1):93–8. [PubMed: 2319050]
24. Fletcher JM, Levin HS, Lachar D, Kusnerik L. Behavioral outcomes after pediatric closed head injury: Relationships with age, severity, and lesion size. Journal of Child Neurology. 1996; 11(4): 283–90. [PubMed: 8807417]
25. Yeates KO, Swift E, Taylor HG, Wade SL, Drotar D, Stancin T, et al. Short- and long-term social outcomes following pediatric traumatic brain injury. Journal of the International Neuropsychological Society. 2004; 10(3):412–26. [PubMed: 15147599]
26. Taylor HG, Yeates KO, Wade SL, Drotar D, Klein SK, Stancin T. Influences on first-year recovery from traumatic brain injury in children. Neuropsychology. 1999; 13(1):76–89. [PubMed: 10067779]
27. Taylor HG, Yeates KO, Wade SL, Drotar D, Stancin T, Burant C. Bidirectional child-family influences on outcomes of traumatic brain injury in children. Journal of the International Neuropsychological Society. 2001; 7(6):755–67. [PubMed: 11575597]
28. Taylor HG, Alden J. Age-related differences in outcomes following childhood brain insults: An introduction and overview. Journal of the International Neuropsychological Society. 1997; 3(6): 555–67. [PubMed: 9448369]
29. Hammen, C.; Rudolph, KD. Childhood depression. In: Mash, EJ.; Barkley, RA., editors. Child psychopathology. New York, NY US: Guilford Press; 1996. p. 153-95.
30. Angold A, Rutter M. Effects of age and pubertal status on depression in a large clinical sample. Development and Psychopathology. 1992; 4(1):5–28.
31. Bombardier CH, Fann JR, Temkin NR, Esselman PC, Barber J, Dikmen SS. Rates of major depressive disorder and clinical outcomes following traumatic brain injury. JAMA: Journal of the American Medical Association. 2010; 303(19):1938–45.
32. Jorge RE, Robinson RG, Arndt SV, Starkstein SE. Depression following traumatic brain injury: A 1 year longitudinal study. Journal of Affective Disorders. 1993; 27(4):233–43. [PubMed: 8509524]
33. Duhig AM, Renk K, Epstein MK, Phares V. Interparental agreement on internalizing, externalizing, and total behavior problems: A meta-analysis. Clinical Psychology: Science and Practice. 2000; 7(4):435–53.
34. Wade SL, Taylor HG, Drotar D, Stancin T, Yeates KO. Family burden and adaptation during the initial year after traumatic brain injury in children. Pediatrics. 1998; 102(1):110–6. [PubMed: 9651422]
35. Donders J, Janke K. Criterion validity of the Wechsler Intelligence Scale for Children–Fourth Edition after pediatric traumatic brain injury. Journal of the International Neuropsychological Society. 2008; 14(4):651–5. [PubMed: 18577295]
36. Mottram L, Donders J. Construct Validity of the California Verbal Learning Test–Children's Version (CVLT-C) After Pediatric Traumatic Brain Injury. Psychological Assessment. 2005; 17(2):212–7. [PubMed: 16029108]
37. Wechsler Abreviated Scale of Intelligence. Austin, TX: Psychological Corporation; 1999.
38. Wechsler Adult Intelligence Scale. Fourth. Austin, TX: Psychological Corporation; 2008.
39. Wechsler Intelligence Scale for Children. Fourth. Austin, TX: Psychological Corporation; 2003.
40. Delis, DC.; Kramer, JH.; Kaplan, E.; Ober, BA. CVLT-C: California Verbal Learning Test–Children's Version. San Antonio, TX: The Psychological Corporation; 1994.
41. Delis, DC.; Kramer, JH.; Kaplan, E.; Ober, BA. CVLT-II California Verbal Learning Test. Second. San Antonio, TX: The Psychological Corporation; 2000.
42. Byles J, Byrne C, Boyle MH, Offord DR. Ontario Child Health Study: Reliability and validity of the General Functioning subscale of the McMaster Family Assessment Device. Family Process. 1988; 27(1):97–104. [PubMed: 3360100]
43. Miller IW, Epstein NB, Bishop DS, Keitner GI. The McMaster Family Assessment Device: Reliability and validity. Journal of Marital and Family Therapy. 1985; 11(4):345–56.

44. Derogatis, LR.; Lazarus, L. SCL-90-R, Brief Symptom Inventory, and matching clinical rating scales. In: Maruish, ME., editor. The use of psychological testing for treatment planning and outcome assessment. Hillsdale, NJ England: Lawrence Erlbaum Associates, Inc.; 1994. p. 217-48.
45. Radloff LS. The CES-D Scale: A self-report depression scale for research in the general population. Applied Psychological Measurement. 1977; 1(3):385–401.
46. Achenbach TM, McConaughy SH, Howell CT. Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. Psychological Bulletin. 1987; 101(2):213–32. [PubMed: 3562706]
47. De Los Reyes A, Kazdin AE. Informant Discrepancies in the Assessment of Childhood Psychopathology: A Critical Review, Theoretical Framework, and Recommendations for Further Study. Psychological Bulletin. 2005; 131(4):483–509. [PubMed: 16060799]
48. Max JE, Arndt S, Castillo CS, Bokura H, Robin DA, Lindgren SA, et al. Attention-deficit hyperactivity symptomatology after traumatic brain injury: A prospective study. Journal of the American Academy of Child & Adolescent Psychiatry. 1998; 37(8):841–7. [PubMed: 9695446]
49. Max JE, Castillo CS, Bokura H, Robin DA, Lindgren SD, Smith WL, et al. Oppositional defiant disorder symptomatology after traumatic brain injury: A prospective study. Journal of Nervous and Mental Disease. 1998; 186(6):325–32. [PubMed: 9653415]
50. Gerring JP, Brady KD, Chen A, Vasa R, Grados M, Bandeen-Roche KJ, et al. Premorbid prevalence of ADHD and development of secondary ADHD after closed head injury. Journal of the American Academy of Child & Adolescent Psychiatry. 1998; 37(6):647–54. [PubMed: 9628085]
51. Gerring JP, Grados MA, Slomine B, Christensen JR, Salorio CF, Cole WR, et al. Disruptive behaviour disorders and disruptive symptoms after severe paediatric traumatic brain injury. Brain Injury. 2009; 23(12):944–55. [PubMed: 19831491]
52. Richters JE. Depressed mothers as informants about their children: A critical review of the evidence for distortion. Psychological Bulletin. 1992; 112(3):485–99. [PubMed: 1438639]
53. Hayman-Abello SE, Rourke BP, Fuerst DR. Psychosocial status after pediatric traumatic brain injury: A subtype analysis using the Child Behavior Checklist. Journal of the International Neuropsychological Society. 2003; 9(6):887–98. [PubMed: 14632248]

Table 1

Demographic information for the moderate and severe TBI groups.

	Moderate TBI	Severe TBI
n	79	51
Mean (SD) age in years at injury	14.49 (1.74)	14.91 (1.71)
Mean (SD) Glasgow Coma Scale score ***	13.51 (1.78)	4.96 (1.93)
Sex (% male)	67.1	62.7
Race (% Caucasian)	77.2	88.2
Biological mother: Education (%)		
Did not complete high school	6.0	7.1
High school diploma or GED	37.3	40.5
At least 2 years college	34.3	19.0
Bachelor's or advanced degree	22.4	33.3
Family income (%)		
< $20,000	11.0	18.8
$20,000–$39,999	30.1	22.9
$40,000–$69,999	23.3	25.0
≥70,000	35.6	33.3
SES (z-score)	−0.02 (1.06)	0.11 (0.93)
Family composition (% two-parent households)	64.6	70.6

*** $p < .001$

Table 2

CBCL mean T-scores (SDs) for the moderate and severe TBI groups.

	Moderate TBI	Severe TBI
Maternal CBCL		
Internalizing Problems composite	54.05 (11.13)	53.33 (11.49)
Externalizing Problems composite	52.90 (10.72)	53.88 (11.01)
Anxious/Depressed subscale	54.92 (6.44)	55.55 (7.28)
Withdrawn/Depressed subscale	56.10 (7.08)	56.47 (8.25)
Somatic Complaints subscale	59.29 (8.82)	57.94 (7.01)
DSM: Anxiety Problems subscale	56.12 (6.75)	55.76 (6.76)
DSM: Affective Problems subscale	57.18 (7.78)	57.94 (7.57)
DSM: Somatic Problems subscale	58.19 (8.91)	56.35 (7.15)
Paternal CBCL		
Internalizing Problems composite	52.51 (11.71)	50.71 (10.67)
Externalizing Problems composite	49.51 (9.08)	49.10 (9.35)
Anxious/Depressed subscale	54.66 (6.53)	54.61 (6.88)
Withdrawn/Depressed subscale	55.63 (6.73)	52.97 (3.92)
Somatic Complaints subscale	58.29 (8.44)	56.90 (8.41)
DSM: Anxiety Problems subscale	55.29 (6.57)	55.03 (6.76)
DSM: Affective Problems subscale	55.63 (6.95)	55.74 (7.41)
DSM: Somatic Problems subscale	57.63 (7.87)	56.87 (9.80)

Table 3

Multiple regression analyses predicting continuous internalizing symptoms in teens

			β-weight								
Dependent Variable	**n**	**R^2**	**Age**	**Sex**	**SES**	**FSIQ**	**PSI**	**CVLT Total**	**FAD-GF**	**M Psych Prob**	**F Psych Prob**
CBCL Internalizing Problems T-score (mother report)	120	.30***	.00	.41***	−.07	.03	−.10	−.15	.16	.30***	–
CBCL Internalizing Problems T-score (father report)	68	.42***	−.04	.07	−.13	.08	−.07	.04	.10	–	.57***

*** p < .001

CBCL: Child Behavior Checklist

SES: Socioeconomic status z-score

FSIQ: WASI Full Scale IQ

PSI: Wechsler Processing Speed Index

CVLT Total: California Verbal Learning Test Total Learning T-Score

FAD-GF: Teen-reported family functioning

M Psych Prob: Mother psychiatric problems composite

F Psych Prob: Father psychiatric problems composite

Table 4

Binary logistic regression analyses predicting presence of clinically elevated internalizing problems in teens

		OR (95% CI)								
Dependent Variable	**n**	**Age**	**Sex**	**SES**	**FSIQ**	**PSI**	**CVLT Total**	**FAD-GF**	**M Psych Prob**	**F Psych Prob**
Internalizing caseness (mother report)	120	1.01 (.77, 1.32)	.20** (.07, .57)	.80 (.45, 1.44)	1.01 (.96, 1.05)	.99 (.96, 1.02)	.99 (.95, 1.04)	2.93 (.97, 8.87)	1.77* (1.07, 2.95)	–
Internalizing caseness (father report)	68	1.09 (.69, 1.72)	.95 (.19, 4.83)	.81 (.34, 1.90)	1.02 (.94, 1.09)	1.01 (.95, 1.07)	1.03 (.96, 1.11)	5.58 (.63, 49.23)	–	7.04** (2.23, 22.19)

* p < .05;

** p < .01;

CBCL: Child Behavior Checklist

SES: Socioeconomic status z-score

FSIQ: WASI Full Scale IQ

PSI: Wechsler Processing Speed Index

CVLT Total: California Verbal Learning Test Total Learning T-Score

FAD-GF: Teen-reported family functioning

M Psych Prob: Mother psychiatric problems composite score

F Psych Prob: Father psychiatric problems composite

OR: Odds ratio

CI: Confidence interval