Parental Posttraumatic Stress Symptoms as a Moderator of Child's Acute Biological Response and Subsequent Posttraumatic Stress Symptoms in Pediatric Injury Patients

Nicole R. Nugent,[1] MA, Sarah Ostrowski,[1] MA, Norman C. Christopher,[2,3] MD, and Douglas L. Delahanty,[1,4] PhD

[1]Department of Psychology, Kent State University, [2]Emergency/Trauma Services, Akron Children's Hospital, [3]Department of Emergency Medicine and Pediatrics, Northeastern Ohio Universities College of Medicine (NEOUCOM), and [4]Department of Psychology in Psychiatry, Northeastern Ohio Universities College of Medicine (NEOUCOM)

Objective To examine how parental responses following pediatric injury may influence their child's posttraumatic stress symptoms (PTSS). **Methods** Heart rate (HR) from 82 pediatric injury patients was measured during emergency medical services (EMSs) transport and following hospital admission. Twelve-hour urinary cortisol levels were assessed upon admission. Child PTSS and parental PTSS and general distress were assessed 6 weeks and 6 months after trauma. **Results** Six-week parental PTSS predicted 6-month child PTSS even after controlling for demographics and general parent distress ($\Delta R^2 = .08$, $p = .03$). Parental PTSS moderated the relationship between (a) child cortisol levels and 6-month child PTSS ($\Delta R^2 = .08$, $p = .03$) and between (b) hospital HR and 6-month child PTSS ($\Delta R^2 = .09$, $p = .03$). **Conclusion** The present findings suggest that parental response to trauma may interact with child acute physiological responses to predict persistent child PTSS.

Key words acute predictors; children; parents; posttraumatic stress disorder.

Posttraumatic stress disorder (PTSD) in children is associated with numerous negative outcomes including significant social impairment (Berman, Kurtines, Silverman, & Serafini, 1996; Stallard, Velleman, & Baldwin, 1998), cognitive deficits (Bremner et al., 1995), poor academic performance (Reinherz, Gianconia, Lefkowitz, Pakiz, & Frost, 1993), and a variety of comorbid behavioral and emotional disorders (Fletcher, 1996). Despite the lower rates of PTSD observed in emergency department (ED) samples (Aaron, Zaglul, & Emery, 1999; Daviss et al., 2000; Kassam-Adams, Garcia-Espana, Fein, & Winston, 2005) as compared with interpersonal traumas such as rape and sexual abuse (Breslau et al., 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), research into early biological predictors of posttraumatic stress symptoms (PTSS) has largely been conducted in hospital settings, as they allow for the earlier assessment of biological factors (typically within a few hours posttrauma; Delahanty, Nugent, Christopher, & Walsh, 2005; Kassam-Adams et al., 2005; Nugent, Christopher, & Delahanty, in press).

Research in pediatric injury samples has produced inconsistent findings concerning the prognostic utility of many initial predictors (e.g., injury severity, child age, and gender), perhaps partly due to the moderating effects of posttrauma variables. One such variable that has received considerable research interest is parental response to trauma. However, results concerning the concordance of parent and child PTSS following a child's trauma have been equivocal, with some studies reporting that child and parent PTSS secondary to pediatric injury are significantly correlated (Daviss et al.,

All correspondence concerning this article should be addressed to Douglas L. Delahanty, PhD, Department of Psychology, Kent State University, Kent, Ohio 44242. E-mail: ddelahan@kent.edu.

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2000; De Vries et al., 1999), while others have not found significant relationships (Bryant, Mayou, Wiggs, Ehlers, & Stores, 2004; Winston et al., 2000). Possible explanations for these discrepant results have included differences between studies with respect to the child's age, the timing of assessment(s), or possibly complex interactions of unmeasured variables (e.g., Pfefferbaum & Pfefferbaum, 1998). For instance, studies examining distress levels soon after the trauma typically find no association between parent and child PTSD (Bryant et al., 2004; Winston et al., 2000), while studies looking at more chronic distress report significant correlations (Koplewicz et al., 2002; Smith, Perrin, Yule, & Rabe-Hesketh, 2001). Prospective studies suggest that initial child PTSS can impact subsequent symptoms in the parent (Koplewicz et al., 2002) and that acute symptoms of distress in the parent predict subsequent PTSD in the child (Daviss et al., 2000; McFarlane, 1987).

One possible mechanism through which parental responses to a child's trauma may influence the child is via general levels of parental distress. General distress in the parent, regardless of PTSS, may lead the parent to be less available to their child (Schwartz, Dohrenwend, & Levav, 1994). Alternatively, more recent work has supported a specific role for parental PTSS, suggesting that parental symptoms related specifically to the traumatic event as compared with general parental distress are more predictive of the development of PTSD in children (Pelcovitz et al., 1998). Examination of the relative saliency of general parental distress versus specific parental response to their child's trauma can provide information regarding mechanisms through which children develop PTSS and can lead to the development of early interventions targeted at optimizing family adjustment to pediatric trauma.

In addition, although prior research has predominantly examined the impact of parental PTSS as a whole on child overall symptomatology, distinct aspects of PTSS may influence the persistence of specific symptoms in children more than others. For instance, whereas adult trauma victims may choose to avoid reminders of a traumatic event on their own, child avoidance of traumatic reminders may be influenced by parental responses. Parents with PTSS stemming from a child's injury might have difficulty discussing the event with their child or might actively avoid activities or places associated with the event, preventing the child from habituating to these reminders. Alternatively, children may be sensitive to their parents' levels of distress and may avoid situations/discussions that could further distress their parents (Hopkins & King, 1994), thus reducing the child's exposure to, and subsequent habituation to, trauma-related stimuli. Prior research has suggested that pediatric trauma survivors who felt less able to talk (with family, friends, or a professional) about a road traffic accident were more likely to present with PTSD 30 weeks after trauma (Stallard et al., 2001). Furthermore, parental PTSS may interact with their child's initial response and risk factors, either buffering or exacerbating the impact of these risk factors.

To examine the extent to which parental responses to a child's trauma interact with the child's initial risk factors to result in more persistent PTSS, it is necessary to identify relatively strong early predictors of child PTSS. Recent research examining initial biological predictors of PTSS in pediatric injury has found that heart rate (HR) during emergency transport (Kassam-Adams et al., 2005; Nugent et al., in press) and urinary cortisol levels soon after a traumatic event (Delahanty et al., 2005) are equally as good, if not better, predictors of subsequent child PTSS than psychosocial factors. In an investigation of PTSS in injured children admitted to the ED, Delahanty et al. (2005) prospectively examined the relationship between 12-hr urinary cortisol levels measured in children immediately following trauma and subsequent child PTSS at 6 weeks. Findings revealed a significant association ($r = .31$) between urinary cortisol and 6-week child PTSS, with urinary cortisol significantly predicting 6-week PTSS even after controlling for parent income, child gender, and concurrent depression. Given these biological predictors, we were interested in the extent to which parental PTSS might moderate the impact of biological predictors on subsequent child PTSS, potentially exacerbating or buffering the effects of this initial biological vulnerability.

The present study was designed to prospectively examine the associations and interactions between child and parent PTSS stemming from the child being admitted to the ED for pediatric injuries secondary to a variety of traumatic experiences. Based on prior literature, we hypothesized that (a) child and parent PTSS with reference to the child's trauma would be significantly cross-sectionally associated at each time point and (b) parent PTSS related to their child's trauma at 6 weeks would predict child PTSS at 6 months beyond 6-week parental general distress levels. In addition, to examine the extent to which parent PTSS impacted the relationship between initial biological risk factors and subsequent PTSS in the child, we hypothesized that parental PTSS would moderate the relationship between the child's initial physiological responses and subsequent 6-month PTSS. Accordingly, we recruited

consecutive admissions from a Midwestern children's hospital following exposure to a variety of traumatic events (i.e., vehicular accident, sports accident, and assault). We assessed initial physiological markers, 6-week child PTSS, parent PTSS anchored to their child's trauma, and parent general distress; and 6-month child PTSS and parent PTSSS.

Methods

Participants

Participants consisted of 82 children consecutively admitted to a Midwestern children's hospital following a traumatic injury and their primary caretakers (predominantly mothers with the exception of four fathers who had paternal custody). Of 85 families approached for participation, 82 families provided consent (96% acceptance rate). To permit informed consent, only children with a Glasgow Coma Scale (GCS) greater than 13 were recruited. There were no differences in demographics (age, gender, or race) between those families that consented and those who refused to participate. The average age of the children was 13.19 years (*SD* = 2.94, range 8.04–17.89; see Table I). The participants were primarily Caucasian (79.2%), 19.5% were African American, and 1.3% indicated mixed heritage. Ninety-three percent of participants completed the 6-week follow-up, with attrition exclusively due to inability to contact participants. At the 6-month follow-up, 70% of the original samples were retained; reasons for drop-out included inability to contact participants (n = 13), multiple missed appointments (n = 3), and children/parents who stated disinterest in continuing with the study (n = 3). No differences were found concerning gender, age, race, parental income or education level, type of trauma, HR levels, urinary hormone levels, or severity of injury between those who successfully completed the study and those who did not complete the 6-week or 6-month follow-ups (all p's > .37). Participants who did and did not complete the 6-month assessment did not differ on 6-week PTSS or depression scores, $t(74) = .25$, $p = .80$; $t(74) = .08$, $p = .93$.

Table I. Participant Demographics

	6 weeks	6 months
Child age (*SD*)	13.25 (2.96)	13.63 (3.02)
Child race (White/African-American/Mixed)	60/15/1	47/9/1
Type of trauma		
Vehicular/bicycle accident	41	31
Sports accident	11	8
Assault	4	3
Miscellaneous[a]	20	15
Child PTSS (CAPS-CA total) (*SD*)	22.80 (22.53)	16.74 (15.43)
Parental PTSS (IES-R) (*SD*)	18.17 (17.27)	12.12 (15.50)
Parental distress (SCL-90-R GSI *T*-score) (range)	40 (24–60)	Not collected

CAPS-CA, Clinician-Administered posttraumatic stress disorder (PTSD) Scale for Children and Adolescents; GSI, Global Severity Index; IES-R, Impact of Event Scale-Revised; SCL-90-R, Symptom Checklist-90-R.

[a]Miscellaneous injuries include accidental burns, dog bites, serious falls (e.g., out of a window/roof, off of a horse, and on a hiking trail), lawn mower accidents, accidental self-inflicted gun injury, swimming accidents, and injury resulting from being hit by a falling tree.

Participants represented consecutive admissions with no exclusionary criteria for mechanism of injury or for whether children's and/or parents' perceptions of the accident met Criterion A (see "Child PTSS" section). This protocol was adopted to increase generalizability and was supported by previous research that has not found differences in PTSS associated with mechanism of injury (Shalev et al., 1998). Participants sustained a variety of injuries, including mild head trauma, tissue lacerations/abrasions, fractures, organ damage, traumatic amputation, dental damage, suspected spinal damage burns, and gunshot wounds. No systematic differences on any of our variables of interest were found with respect to type or extent of injury. Although all the children were exposed to an event involving serious life threat, injury, or threats to physical integrity, six of the children who completed the 6-week follow-up did not report experiencing subjective fear, helplessness, or horror and, therefore, did not meet Criterion A.

Procedure

Procedures for this study have been previously described in detail (Delahanty et al., 2005) and were approved by the institutional review boards at both Kent State University and Akron Children's Hospital. Briefly, all urine excreted during the participants' first 12 hr after admission was collected, aliquotted, and frozen until cortisol assays were conducted. Although 24-hr samples would allow for assessment of hormone levels throughout the circadian rhythm, 12-hr samples were collected, as hospital protocol requires removal of catheters as soon as possible to reduce patient discomfort.

Children and their parents were approached in hospital, and parental consent and child assent or consent, depending on the child's age, were obtained. Urine samples were discarded without assaying if patients did not agree to participate. As part of the consent process, patients gave permission to have their medical charts reviewed for the collection of HR data, medications taken, chronic medical conditions, and injuries sustained.

Follow-up assessments took place 6 weeks and 6 months after the child's admission date. Masters-level clinical psychology students with experience in the assessment of PTSS in children conducted the follow-up interviews. Child participants were administered the Clinician-Administered PTSD Scale for Children and Adolescents (CAPS-CA). The parent completed the Impact of Event Scale-Revised (IES-R) with regard to symptoms stemming from the child's trauma and the Symptom Checklist-90-R (SCL-90-R) as a measure of general, nontrauma-specific distress.

Measures

Child PTSS

Child PTSD symptoms were assessed using the CAPS-CA (Nader et al., 1996). This is a semistructured clinical interview that scores each PTSD symptom in terms of frequency and intensity. Individual items (including Criteria B, C, and D symptoms as well as indicators of Criterion F) are summed to yield a continuous total symptom score or may be used to provide a categorical diagnosis; total symptom scores range from 0 to 152. Symptoms were recorded as present if the child received a minimum score of 1 for frequency and 2 for intensity. Following Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria, a child received a PTSD diagnosis if they reported experiencing subjective fear, helplessness, or horror during their accident (Criterion A), endorsed at least one reexperiencing symptom (Criterion B), three avoidant symptoms (Criterion C), two arousal symptoms (Criterion D), and reported distress or impairment in important areas of functioning (Criterion F). Partial PTSD (Stein, Walker, Hazen, & Forde, 1997) was indicated when a participant did not meet full diagnostic criteria but met Criterion A, reported at least one symptom from each cluster (reexperiencing, avoidance, and hyperarousal), and indicated functional impairment. Prior research (Aaron et al., 1999; Marshall et al., 2001; Schutzwohl & Maercker, 1999; Stein et al., 1997) has found that subthreshold or partial PTSD is associated with clinically significant impairment in functioning; moreover, functional impairment, number of comorbid disorders, rates of comorbid major depressive disorder, and current suicidal ideation increase linearly and significantly with each increase in number of PTSD symptoms, supporting the importance of understanding predictors of PTSS even in populations with relatively low rates of full PTSD. Interviews were audiotaped for interrater reliability, and the intraclass correlation coefficient for total symptom scoring on a randomly selected sample of audiotapes was .98.

General Parental Distress

Parental distress, defined as global symptoms of psychological distress, was assessed with the SCL-90-R (Derogatis, 1994). The SCL-90-R is a 90-item self-report measure of psychological distress. Participants are asked to rate how much discomfort each of the problems has caused them over the past week, rating their discomfort from 1 (no discomfort) to 4 (extreme discomfort). For this study, the Global Severity Index (GSI), a composite measure of "distress," including symptoms of somatization, obsessive compulsion, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism, was used to provide an overall level of emotional distress. For the present sample, *t*-scores were calculated based on a referent nonpatient population. Scores of 63 or higher have been used to screen for further psychiatric evaluation (Derogatis, 1994). Internal consistency of the measure for the present study was .97.

Parental PTSS

Parental PTSS, defined as symptoms of PTSD secondary to their child's trauma, was measured using the IES-R (Weiss & Marmar, 1997). The IES-R is a widely used 22-item self-report measure of PTSS with respect to a specific incident. The measure assesses the frequency of intrusive (i.e., "I thought about it when I didn't want to") and avoidant thoughts and behaviors (i.e., "I tried not to think about it") and hyperarousal symptoms (i.e., "I was jumpy and easily startled") on a 5-point scale (0 = not at all, 4 = extremely) during the past week. Scores across items are summed to provide a continuous measure of parental PTSS with a possible range of scores from 0 to 88. Participants were instructed to report their own PTSS with respect to their child's accident as the index trauma. The IES-R displayed good internal consistency at 6 weeks with respect to subscales (avoidance, α = .84; reexperiencing, α = .93; and hyperarousal, α = .82, respectively) and total scores (α = .95). Similar internal consistency was found at 6 months (avoidance, α = .93; reexperiencing, α = .93; and hyperarousal, α = .89, respectively) and across the composite measure (α = .96).

Injury Severity

Injury severity was measured with the Injury Severity Scale (ISS; Association for the Advancement of Automotive Medicine, 1990), which has been found to be a reliable and valid measure of injury severity in children (Bull, 1975). To compute the ISS, the body is divided into six regions (head, face, chest, abdomen, extremity, and external), and the extent of injury to each region is

scored and summed. The scores assigned to the three most severely injured body regions are squared and then added to calculate the ISS score. ISS scores range from 0 to 75, with high ISS scores associated with increased risk of mortality (Baker, O'Neill, Haddon, & Long, 1974; Stoner, Barton, Little, & Yates, 1977). ED personnel routinely calculates ISS scores, and scores were collected following the review of participants' charts.

Biological Measures

Based on previous findings in the present sample, we focused on examining the extent to which parental response moderated our strongest biological predictors, specifically averaged HR and urinary cortisol (Delahanty et al., 2005; Nugent et al., in press). Urinary cortisol levels were computed for 12-hr urine samples initiated upon admission to the ED. Cortisol assays were performed by the Cleveland Clinic (Cleveland, OH) and were measured with fluorescent polarization immunoassay (Abbott TDx Diagnostics, Abbott Laboratories, Abbot Park, IL). Urinary cortisol levels were calculated as amount per 12-hr sample (ug/12 hr). Urine volume was not significantly correlated with cortisol levels (all p's > .18).

Patient HR data were obtained from emergency medical services (EMSs) reports and from ED records. The first 20 min of HR data recorded during EMS transport and following admission to the ED were averaged to provide mean EMS and ED HR measures. Twenty minutes of HR records typically reflected four to five readings.

Results

Statistical Analyses

Data were analyzed for normality of variable distributions using the Shapiro–Wilks W statistic. Urinary cortisol levels and general parental distress were nonnormal and were log transformed. Initial chi-square analyses and one-way analyses of variance (ANOVAs) were conducted to examine possible relationships between demographics and PTSS at 6 weeks or 6 months. Additional potential covariates were identified with Pearson product moment correlations. Zero-order bivariate correlations were also used to examine associations between child and parent PTSS at 6 weeks and 6 months. Next, hierarchical linear regression analyses were conducted to examine the hypothesis that 6-week parental PTSS would predict child PTSS at 6 months after controlling for covariates and 6-week parental general distress levels. To examine the extent to which 6-week parental distress predicted 6-month child PTSS beyond specific parental PTSS, a similar regression was conducted with covariates in the first step, 6-week parental PTSS in the second step, and 6-week parental general distress in the second step.

To examine the hypothesis that 6-week parental PTSS would moderate the relationship between the child's initial physiological reactivity and 6-month child PTSS, we conducted parallel hierarchical linear regression analyses. Specifically, in each regression analysis, covariates were entered first, followed by respective biological measures (either cortisol or HR) in the second block, the potential moderator (parental PTSS) in the third block, and the interaction of acute biological response and the moderator in the final step (Baron & Kenney, 1986; Cohen & Cohen, 1983). Moderation was identified when the interaction term accounted for a significant percentage of the variance beyond the main effects.

Parent and Child Symptoms

Table I provides a summary of child and parent PTSS and parent general distress. Most parents evidenced low-to-moderate PTSS at 6 weeks (M = 18.17, SD = 17.27; range = 0–61) and fewer symptoms at 6 months (M = 12.12, SD = 15.50; range = 0–55). Relative to nonpatient norms, parents' 6-week distress T-scores were relatively low (M = 40; range = 24–60), with only eight parents endorsing clinically relevant (T > 63) levels of distress. At the 6-week follow-up, 5 of 76 of the children (7%) met full criteria for PTSD and 13 (17%) met criteria for partial PTSD; at 6-month follow-up, 1 of 57 children (2%) met full criteria for PTSD and 3 (5%) exhibited partial PTSD. Participant age, race, type of trauma, injury severity score, and number of years of parent education were not related to child PTSS at 6 weeks (p's = .18, .10, .66, .34, and .39, respectively) or at 6 months (p's = .68, .55, .83, .44, and .10, respectively). However, at the 6-week follow-up, girls reported significantly more PTSS than boys [$F(1, 75) = 4.94$, $p = .03$], and parent income was negatively associated with child PTSS ($r = -.25$, $p < .03$); gender and parent income were no longer related to PTSS at the 6-month follow-up (p's = .26 and .43, respectively). We conservatively covaried for gender and parent income in all regression analyses.

Associations between Child and Parental PTSS

To explore the associations between child and parent PTSS, Pearson product moment correlations were conducted (see Table II). Findings revealed a significant association between child and parent PTSS at 6 weeks, $r = .45$, $p < .001$. Moreover, parent PTSS at 6 weeks were

Table II. Correlations Among Child and Parental Symptom Scores

	1	2	3	4
6-Week child measure				
1. CAPS-CA total				
6-Week parent measures				
2. IES-R total	.45***			
3. SCL-90-R GSI	.50***	.60***		
6-Month child measure				
4. CAPS-CA total	.75***	.45***	.39**	
6-Month parent measures				
5. IES-R total	.25	.70***	.76***	.23

CAPS-CA, Clinician-Administered posttraumatic stress disorder (PTSD) Scale for Children and Adolescents; GSI, Global Severity Index; IES-R, Impact of Event Scale-Revised; SCL-90-R, Symptom Checklist-90-R.
$^{**}p < .01$, $^{***}p < .001$, two-tailed.

significantly related to child PTSS at 6 months, $r = .45$, $p < .001$. Parent PTSS at 6 months, however, were not significantly correlated with child PTSS at 6 weeks or at 6 months, p's = .12 and .15, respectively.

To test whether 6-week parent PTSS would be a better predictor of 6-month child PTSS than 6-week general parent distress, hierarchical linear regression analyses were conducted. Findings revealed that parental 6-week PTSS significantly predicted 6-month child PTSS above covariates (gender and parent income) and 6-week parental distress, $\Delta R^2 = .08$, $p = .03$. By contrast, parent general distress did not predict child PTSS after controlling for gender, parental income, and 6-week parental PTSS, $\Delta R^2 = .03$, $p = .22$.

Parental PTSS as a Moderator of the Physiological Reactivity PTSS Relationship

To examine whether parental PTSS would moderate the relationship between the child's initial physiological reactivity to the trauma and 6-month child PTSS, we conducted parallel hierarchical regressions examining the interaction of each biological variable with parental PTSS. Although structural equation modeling would be a preferable analytic technique for investigating the interactions between indicators of initial biological response and parental PTSS, given our small sample and concerns with model underidentification, parallel regressions were a more appropriate analysis. Results revealed that the cortisol × parental PTSS interaction term accounted for a significant percentage of the variance above the control variables and the main effects of initial cortisol and parental PTSS ($\Delta R^2 = .08$, $p = .03$; see Table III). Decomposition analyses were conducted to determine the significance of the slope of child PTSS on initial cortisol at low parental PTSS (1 *SD* below the *M*) and high parental PTSS (1 *SD* above the *M*; see Fig. 1).

Table III. 6-Week Parental Posttraumatic Stress Symptoms (PTSS) as a Moderator of the Relationship between Initial Pediatric Cortisol and 6-Month Child PTSS

Step	Variables	R^2	β	ΔR^2	F of ΔR^2	Significant $F\Delta$
1	Parent income, gender	.02	.15, –.04	.02	.34	.714
2	Cortisol	.05	–.24	.03	1.32	.258
3	Parental PTSS	.29	.52	.24	14.11	.001
4	Cortisol × parental PTSS	.37	–.29	.08	5.05	.030

Significance of the regression model, $F(5, 40) = 4.688$, $p = .002$.

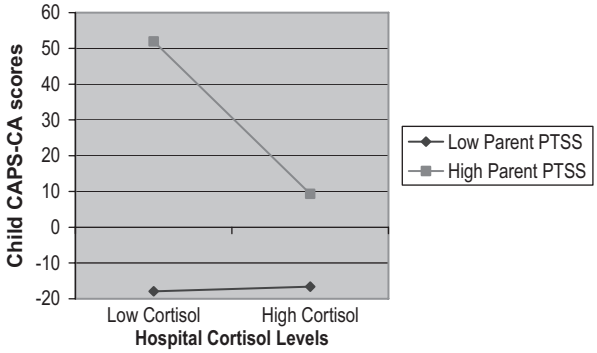

Figure 1. Moderation of relationship between cortisol and 6-month child PTSS by 6-week parent PTSS.

Table IV. 6-Week Parental Posttraumatic Stress Symptoms (PTSS) as a Moderator of the Relationship between In-Hospital Pediatric Heart Rate (HR) and 6-Month Child PTSS

Step	Variables	R^2	β	ΔR^2	F of ΔR^2	Significant $F\Delta$
1	Parent income, gender	.02	.29, .10	.03	.53	.595
2	Hospital HR	.03	.06	.00	.00	.963
3	Parental PTSS	.28	.61	.25	12.04	.001
4	Parental PTSS × HR	.37	–.33	.09	4.89	.034

Significance of the regression model, $F(5, 33) = 3.96$, $p = .006$.

The slope of child PTSS on initial cortisol levels was significantly different from 0 at high parental PTSS ($b = -10.55$, $SE = 3.88$, $p = .01$) but not at low parental PTSS ($b = .85$, $SE = 3.37$, *ns*). Thus, children with low initial urinary cortisol and high 6-week parental PTSS showed higher levels of 6-month child PTSS than did children with high initial urinary cortisol and high 6-week parental PTSS.

Parental PTSS also moderated the relationship between in-hospital HR and 6-month child PTSS ($\Delta R^2 = .09$, $p = .03$; see Table IV). Decomposition of the HR × parent PTSS interaction did not identify significant slopes for high or low parental PTSS (see Fig. 2). Although high parental PTSS were associated with greater child PTSS for children with both low and high initial HR, children with low initial HR appeared to

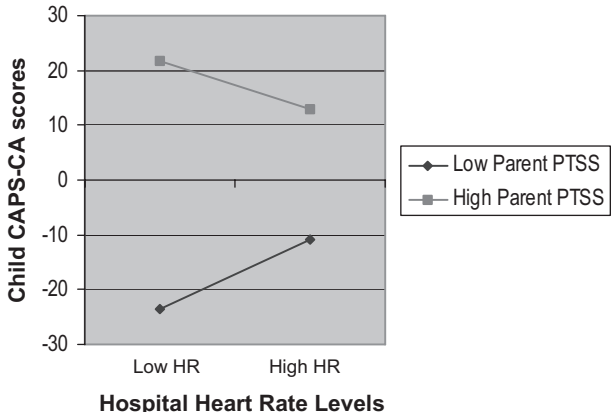

Figure 2. Moderation of relationship between heart rate and 6-month child PTSS by parental PTSS.

be most influenced by parental PTSS (exhibiting greater PTSS at high levels of parental PTSS and lower PTSS at low levels of parental PTSS). The interaction of parental PTSS and EMS HR did not account for a significant percentage of the variance in 6-month child PTSS.

To determine whether observed relationships with parent PTSS were driven by any particular PTSD symptom cluster (e.g., avoidance, reexperiencing, or hyperarousal), we also conducted additional analyses substituting the individual symptom cluster scores for parent PTSS. Analyses revealed similar patterns of associations across parent symptom clusters.

Discussion

The findings of this study confirm the importance of addressing parental PTSS to facilitate child adjustment to pediatric injury. Specifically, we found that parental PTSS at 6 weeks predicted 6-month child PTSS after controlling for demographics and general parent distress. By contrast, general distress reported by parents did not predict subsequent child PTSS after controlling for 6-week parent PTSS, suggesting that specific parental PTSS may be of greater importance than general levels of distress in contributing to child maladaptation following trauma. The present results supported an association between concurrent child and parent reports of PTSS at 6 weeks but not at 6 months. Initial parental response may be particularly salient to children, influencing both the development and the maintenance of their PTSS, whereas long-term parental adjustment may be related to other factors beyond their child's PTSS.

This is consistent with a more direct transmission of parental psychopathology and/or adjustment to the child, which may be translated through parental modeling or implicit learning of parental cognitive styles, coping mechanisms, and reactions to stress (Schwartz et al., 1994). Children may use parental PTSS as a gauge for the seriousness of the trauma and their initial appraisal or later reappraisal of the threat of the traumatic experience. Alternately, parents may directly impact their children's exposure to traumatic reminders, thereby increasing or decreasing opportunities for avoidance or habituation. Subsequent research should further examine possible mechanisms through which parental PTSS may impact the development and maintenance of child PTSS.

Our findings further supported an interaction between the child's initial physiological reactivity and parental symptoms of PTSS. Specifically, high levels of parental PTSS were particularly deleterious for children who excreted low levels of cortisol soon after their accident, whereas parental PTSS were less relevant for children with high levels of initial cortisol. Similarly, high parental PTSS were associated with greater child PTSS in children with low in-hospital HR. We had expected that parental PTSS might be particularly salient for children at increased risk for PTSS (with elevated initial physiological levels) because parental symptoms of PTSS would exacerbate biological vulnerability and prevent habituation to the initial biological response to the trauma. The present pattern of results, however, suggests that children who are not at increased risk for the development of PTSS on the basis of initial biological response may still develop PTSS partly due to their parent's response to the trauma.

A qualifying limitation of the this investigation is the relatively small sample size, which increases the likelihood that the stability of identified models may be sample specific. Sample size was particularly relevant given our interest in parental PTSS as a moderator; our regressions were underpowered for the identification of small effect sizes, as are more frequently found in tests of moderation. As such, our ability to detect significant interactions points to the potentially robust nature of our findings. Concerns related to sample size are further exacerbated by the relatively low incidence of diagnostic levels of PTSS in our sample, which limits the extent to which we may generalize these findings to more seriously traumatized children. This sample of pediatric injury patients was exposed to a variety of types of trauma, ranging from vehicular accidents to physical assault. Prior adult investigations of peritraumatic physiological predictors of PTSD have used similarly heterogeneous groups of ED patients exposed to motor vehicle accidents, physical assault, terrorism, and combat (Shalev

et al., 1998), presuming that the results may generalize across trauma types. Although the heterogeneity of this sample supports the generalizability of these findings across types of trauma, and our analyses did not identify differences in PTSS associated with trauma type, it is possible that a larger sample may have identified significant differences between responses across trauma types. Future investigations should examine the extent to which different types of pediatric trauma may differentially influence early child physiological indices and parental responses as well as PTSS in children. Additionally, given the statistical limitations inherent in investigations with small rates of PTSD and possible limitations in the generalizability of these findings to samples with greater rates of PTSD, future research should replicate these findings, with samples showing higher rates of PTSS such as those found secondary to interpersonal traumas.

Future investigations may improve upon the present examination by more comprehensively assessing and exploring parental response to trauma. Specifically, whereas we used a self-reported continuous measure of parent PTSS, clinical interview of parental PTSS may provide a more sensitive and an accurate indicator of parental PTSS. Furthermore, future investigations should also incorporate an assessment of parent and child perceptions of life threat/threat to physical integrity to better assess the extent to which victims meet PTSD Criterion A. On a similar note, a clinical interview format may have impacted the reporting of symptom development in children. Prior research has found that children who felt able to talk with others about their traumatic experience were less likely to exhibit PTSD at later follow-ups (Stallard et al., 2001).

Elucidation of models to aid in conceptual understanding and to guide the development of treatment for pediatric trauma depends upon the thorough investigation of the reciprocal influences of parental and child adjustment. Accordingly, future research should examine the trajectory of the development of child PTSS as impacted by both biological and psychosocial variables. Although the present findings are suggestive, a more systematic examination of parent and child response to pediatric injury is necessary to clarify the salient aspects of parental response that may impact a child's responses and risk for PTSS following a traumatic event. More frequent assessments in the weeks following the trauma might allow for more sensitive assessment of the effects of early parental responding and subsequent child adjustment.

Despite these limitations, the present investigation represents an early attempt to prospectively track child and parent adjustment, with particular attention to the interactions of parental response and peritraumatic child physiological reactivity. Our findings underscore the importance of assessing parental responses to a child's trauma and lend some early support for the development of acute interventions that are targeted toward promoting healthy parental response to their child's trauma.

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