THE JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
Journal of Child Psychology and Psychiatry 53:7 (2012), pp 767–774 doi:10.1111/j.1469-7610.2011.02520.x

The mutual prospective influence of child and parental post-traumatic stress symptoms in pediatric patients

Markus A. Landolt,[1] Eivind Ystrom,[2] Felix H. Sennhauser,[1] Hanspeter E. Gnehm,[3] and Margarete E. Vollrath[2,4]

[1]University Children's Hospital Zurich and Children's Research Center, Zurich, Switzerland; [2]Division of Mental Health, Norwegian Institute of Public Health, Oslo, Norway; [3]Children's Hospital, Aarau, Switzerland; [4]Psychological Institute, University of Oslo, Oslo, Norway

Background: Previous studies found notable rates of post-traumatic stress symptoms (PTSS) and post-traumatic stress disorder (PTSD) in pediatric patients and their parents and suggest a significant association between child and parent PTSS. However, little is known about mutual influences between child and parental PTSS over time. This study prospectively examined the presence of PTSS and PTSD and the mutual influence of child and parental PTSS in a large sample of pediatric patients with different medical conditions. **Methods:** A total of 287 children (aged 6.5–16 years) and their mothers (n = 239) and fathers (n = 221) were assessed at 5–6 weeks and 1 year after an accident or a new diagnosis of cancer or diabetes mellitus type 1 in the child. **Results:** At the first assessment 11.1% and at the second assessment 10.2% of the children had moderate to severe PTSS. At 5–6 weeks 29.3% of mothers and 18.6% of fathers met criteria for PTSD. At 1 year the rates were 14.6% for mothers and 7.9% for fathers. There were considerable differences of PTSS among different medical diagnostic groups in children and parents. Mothers were more vulnerable than fathers. Structural equation analysis revealed that initially high PTSS in mothers and fathers were longitudinally related to poorer recovery from PTSS in the child. Cross-lagged effects from the child to the parents and from one parent to the other were not significant. **Conclusions:** This study highlights the long-term influence of parental PTSS on the child's recovery after trauma and calls for a family systems approach and for early interventions in the treatment of traumatized pediatric patients. **Keywords:** Trauma, injury, chronic illness, post-traumatic stress disorder.

Introduction

It is well established that accidents and life-threatening diseases in children can cause post-traumatic stress symptoms (PTSS) and post-traumatic stress disorder (PTSD) both in the child and his or her parents (Bruce, 2006; Cabizuca, Margques-Portella, Mendlowicz, Coutinho, & Figueira, 2009; Kassam-Adams, Fleisher, & Winston, 2009; Kazak et al., 2004; Kazak, Boeving, Alderfer, Hwang, & Reilly, 2005; Landolt, Vollrath, Ribi, Gnehm, & Sennhauser, 2003; Landolt, Vollrath, Laimbacher, Gnehm, & Sennhauser, 2005a; Olofsson, Bunketorp, & Andersson, 2009). Although there is some variation between studies, the rates of PTSD and clinically relevant PTSS usually show a decline over time when assessed longitudinally (Helfricht, Latal, Fischer, Tomaske, & Landolt, 2008; Landolt et al., 2005a; Landolt, Vollrath, Timm, Gnehm, & Sennhauser, 2005b; Le Brocque, Hendrikz, & Kenardy, 2010a,b).

Pathogenetic models of pediatric medical traumatic stress (Kazak et al., 2006) acknowledge that the risk factors for PTSS are complex and include both medical and family factors, among others. Yet, medical characteristics such as severity of injury or intensity and duration of medical treatment are not strongly related to subsequent PTSS (Kazak et al., 2006). With regard to parental characteristics, most studies showed that parental psychopathology, including PTSD, is a significant risk factor for PTSS in the child (Kazak et al., 2006; Landolt et al., 2005b; Le Brocque et al., 2010b).

Scheeringa and Zeanah (2001) introduced the concept of 'relational PTSD' that suggests that parent and child symptoms mutually influence each other concurrently and sustain each other over time. Consequently, not only do parents' responses to a traumatic event contribute to the child's distress but also the child's responses contribute to the parents' distress. The latter causal direction has been neglected in research on PTSS in families of ill and injured children.

Studying the interaction of child and parental PTSS over time calls for prospective studies and large samples, but many previous studies were cross-sectional. Existing prospective studies showed that a substantial share of the children and their parents have elevated PTSS initially that tend to decline over time (Helfricht et al., 2008; Landolt et al., 2005a,b; Le Brocque et al., 2010a,b). However, these studies failed to take the mutual influence and co-development of child and parental symptoms into account.

Conflict of interest statement: No conflicts declared.

Moreover, studying the interaction of child and parental PTSS demands an independent assessment of child symptoms, as parental reports may be biased by the parents' own mental health (Valentino, Berkowitz, & Smith Stover, 2010).

This study is the first to address these issues by means of a prospective study including independent assessments of mothers, fathers, and children. Moreover, this study also takes the severity of the child's medical condition into account. Our purpose was twofold: First, we aimed to assess the incidence and course of PTSS in children and their parents in the first year after the diagnosis of a life-threatening disease or the occurrence of an unintentional injury in the child. We expected a significant proportion of participants to show initially clinically relevant PTSS and PTSD with a decline over time (Cabizuca et al., 2009; Shudy et al., 2006). Second, we wanted to investigate (a) cross-sectional associations, (b) course over time, and (c) co-development in child and parental PTSS while controlling for the severity of the child's medical condition.

Figure 1 presents the conceptual model with the variances and paths denominated. Parameters o and s represent the variance in PTSS at 1 month. Parameters p and v represent the variance in change of PTSS from time 1 to time 2 (i.e., residual change; time 2 PTSS regressed on time 1 PTSS). First, we expected to find cross-sectional associations (d1 and h1) between mother, father, and child PTSS and between their rate of change in symptoms (d2 and h2). Second, we hypothesized that child, maternal, and paternal PTSS at time 1 are to some extent stable over time (within-individual stability), and therefore would influence PTSS at time 2 (e and j). Third, we expected to find longitudinal effects of maternal and paternal PTSS on child PTSS (f), longitudinal effects of child symptoms on parental PTSS (q), and longitudinal effects across the parents (g). Finally, we expected that the distress caused by the child's medical condition (measured by the length of hospital stay) would influence child and parental PTSS cross-sectionally and longitudinally (c1, c1–2, c2, m1, m1–2, m2).

Methods

Participants and procedure

The study was approved by the ethical review boards of all hospitals. Written informed consent was obtained from parents and from children age 12 years and older. Families were consecutively recruited in four children's hospitals in the German-speaking part of Switzerland. They were asked to participate in the study within the first 2 weeks after the child's admission to the hospital if the following criteria were met: (a) hospitalization of at

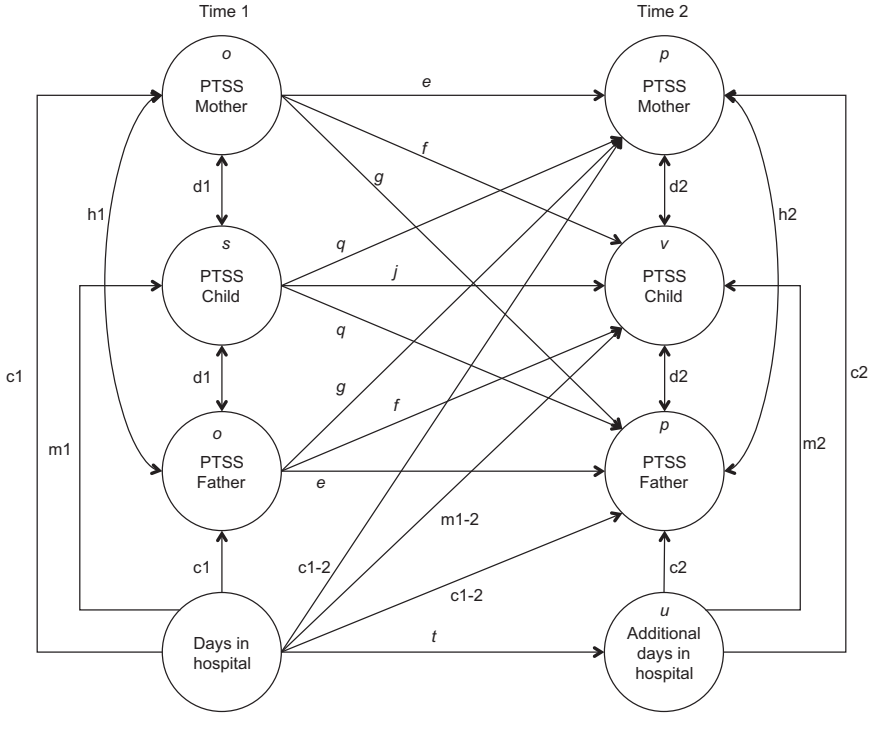

Figure 1 Conceptual model. All parameters with the same name are estimated as one parameter. Rectangles are observed variables; circles are observed variables adjusted for error of measurement; figures within the variables denote residual variance; single arrows denote linear regressions; double arrows denote residual correlations. All the variables at T2 are adjusted for initial status at T1; hence the residual variance is interpreted as change in the characteristic. The familial residual correlations at 1 year (parameters d2 and h2) are therefore a figure of similarity in recovery from PTSS. The regressions within characteristic across time (e and j) denote stability of PTSS, adjusted for days in hospital and familial similarity. The cross-lagged effects (f, g, q) denote mutual effects on change between family members (e.g., parameter f denotes the extent to which initial level of parental PTSS predicts recovery from PTSS in the child; adjusted for days in hospital)

least 24 hr, (b) new diagnosis of cancer or type 1 diabetes mellitus or occurrence of an unintentional injury (excluding severe head injury), (c) age 6.5–16 years, (d) fluency in German, and (e) no previous evidence of intellectual developmental delay. Of 357 children who met inclusion criteria, 70 (22 girls, 48 boys) did not participate, mainly because the study seemed too overwhelming (n = 30), too time consuming (n = 17) or because families had no interest in the study (n = 11). The final sample comprised 287 children (response rate 80.4%). The actual response rate in parents was lower than in children, with 239 mothers and 221 fathers participating. In most cases nonparticipation of parents was due to insufficient command of the German language. There were no significant differences between participants and nonparticipants in child age (t = 1.82, p = .07), sex (χ^2 = 1.26, p = .26), and medical diagnosis (χ^2 = 4.84, p = .09).

There were two assessments: T1 was carried out at 5–6 weeks, and T2 at 1 year after the initial admission to the hospital. Children were assessed by an interview, and parents were assessed by questionnaires that mother and father had to complete separately. Demographic and medical variables were retrieved from the patients' records. Between T1 and T2 24 children, 26 mothers, and 30 fathers dropped out.

Measures

Child PTSS were assessed by the Child PTSD Reaction Index (RI) (Frederick, Pynoos, & Nader, 1992), administered in an interview format. The RI contains 20 items with a 5-point Likert scale ranging from *none of the time* (0) to *most of the time* (4). A total PTSS score is obtained by summing across all items. Although the RI does not provide a PTSD diagnosis according to DSM-IV, there is a scoring system that establishes levels of PTSS severity as follows: score 12–24, mild; 25–39, moderate; 40–59, severe; >60, very severe. Scores higher than 24 are deemed clinically relevant. Reliability and validity of the RI in school-aged children have proven to be good (Nader, Pynoos, Fairbanks, & Frederick, 1990). This study used a German version of the RI. The translation procedure followed internationally accepted guidelines (Brislin, Lonner, & Thorndike, 1973), including independent back-translation and approval of the final version by the original authors. The RI achieved good internal consistency in the current sample (α = .78 at T1, α = .75 at T2).

Parental PTSS were assessed by the German version of the Post-traumatic Diagnostic Scale (PDS) (Foa, Cashman, Jaycox, & Perry, 1997; Griesel, Wessa, & Flor, 2006), a self-report measure that provides a diagnosis of PTSD according to DSM-IV and a PTSS severity score. Parents were asked to rate the presence of the symptoms of PTSD on a 4-point Likert severity scale ranging from *not at all (0)* to *very much* (3). The English and German versions of the PDS demonstrated excellent psychometric properties. In this study, internal consistency of the PDS was found to be good in mothers (T1: α = .87; T2: α = .90) and fathers (T1: α = .87; T2: α = .88).

To operationalize the *severity of medical stressors* we used a proxy variable, represented by the number of days of the child's hospital stay. Two variables were computed: 'days in hospital until T1' and 'additional days in hospital between T1 and T2'. These two variables are good proxies for injury/illness severity, as they correlate highly with severity of condition, number of medical complications during treatment, and child's functional impairment as rated by physician (see Table S1).

Socioeconomic status (SES) was calculated using a 6-point scale of both paternal occupation and maternal education, resulting in an SES-score ranging from 2 to 12. The three social classes were defined as follows: scores 2–5, lower class; 6–8, middle class; and 9–12, upper class. This measure has been used sucessfully in previous studies (Landolt, Nuoffer, Steinmann, & Superti-Furga, 2002).

Statistical analyses

Chi-squared analyses were used to compare categorical variables, t-tests and repeated measurement ANOVAs for comparison of continuous variables across time and among mothers and fathers. One-way ANOVAs were used to compare continuous variables across diagnostic groups. Analyses were performed with two-sided tests; an alpha of .05 was considered significant.

Our model (Figure 1) was analyzed by structural equation modeling. To conform to the assumption of normality we first transformed all PTSS scores to the 10-base logarithm, and then used the Sattora-Bentler Chi-squared correction for non-normality (Satorra & Bentler, 1994) to estimate the structural equation models. All families, including those with missing data for family members and time points, were retained for analysis (n = 270), and missing data was estimated using full-information maximum likelihood (FIML) in Mplus 6.0 (Muthén & Muthén, 2010). Error of measurement was included into the structural model by setting the association between a latent variable and the observed PTSS score to unity and the error term for the observed PTSS score to 1 minus the alpha reliability.

The structural model in Figure 1 was tested under the following assumptions: First, we assumed that mothers and fathers would report different levels of PTSS, so mothers and fathers had separate intercepts. Second, we assumed equal variance in PTSS across mothers and fathers, freeing up those variances that were significantly different. Third, we started out with an equal covariance structure for mothers and fathers (model 0), freeing up paths significantly different (model 1–2). p-values for difference parameter estimate magnitude were estimated applying the 'model test' option in Mplus using parameter constraints. To assess model fit, we used the comparative fit index (CFI) and root mean square error of approximation (RMSEA). A CFI > .95 and a RMSEA < .06 indicates a good model fit (Hu & Bentler, 1998). Importantly, because none of the model variables was significantly associated with the child's age, a confounding effect of the latter variable could be excluded (Table S2).

Results

Sample characteristics

Table 1 shows characteristics of the sample. Of the 287 children, 138 had suffered an unintentional

Table 1 Characteristics of the sample

	All	Injuries	Diabetes	Cancer	χ^2	*F*	*p*
N	287	138	72	77			
Sex							
Female	111	49	30	32	1.13		.57
Male	176	89	42	45			
Age (years)							
Mean	10.36	9.84[a]	10.67	11.03[b]		6.35	.002
SD	2.52	2.34	2.50	2.66			
Socioeconomic status							
Lower	25	15	7	3	9.90		.13
Middle	166	68	47	51			
Upper	78	44	15	19			
Unknown	18	11	3	4			
Living with both biological parents	221	108	56	57	0.53		.77
Length of hospital stay T1 (days)							
Mean	12.33	8.44[a]	14.69[b]	17.10[b]		31.29	.000
SD	9.06	8.34	6.83	9.14			
Length of hospital stay T2 (days)							
Mean	23.97	10.09[a]	15.21[a]	58.94[b]		82.24	.000
SD	32.67	13.34	7.00	46.14			

Subgroups with different superscripts are significantly different ($p < .05$ with Sheffe post hoc tests).

injury, 72 were newly diagnosed with type 1 diabetes, and 77 with cancer. Girls made up 38.7% of the sample, with no significant differences in gender distribution between the diagnostic subgroups. Children with cancer were older compared to children with unintentional injuries. The sample represents mainly middle to upper socioeconomic class. Length of hospital stay differed significantly between the diagnostic groups at both assessments.

Presence of post-traumatic stress symptoms and post-traumatic stress disorder

Table 2 shows child and parental post-traumatic stress symptomatology. At T1 11.1% and at T2 10.2% of the children had scores in the clinical range. Comparison of RI scores across diagnostic groups indicated significant differences at both assessment points. Children with unintentional injuries and cancer had significantly more PTSS than children with diabetes. The same result was found with regard to the percentages of children who scored above the RI cutoff score.

Among mothers, 29.3% met full criteria for PTSD at T1, and 14.6% at T2. When comparing mean levels of PTSS and rates of PTSD across diagnostic groups, significant differences were found, with mothers of children with cancer being affected most. In fathers, rates of PTSD were 18.6% at T1, and 7.9% at T2. As in mothers, fathers of children with cancer were the most affected at both time points.

In terms of effect size, parents had a large decrease in mean level PTSS from T1 to T2 (Cohen's d mothers 0.97, $p < .001$; fathers 0.84, $p < .001$). In children, however, the fall in PTSS symptoms was small and nonsignificant (d = 0.09, $p = 0.27$). Mothers had significantly higher average PTSS scores than fathers at both time points (Cohen's d T1 = 0.45, $p < .001$; T2 = 0.27, $p < .001$).

Structural equation model

Model fitting. First, the base model, model 0, with equal covariance structure across mothers and fathers but different means and intercepts, fit the data adequately (*S-B* $\chi^2 = 22.75$; $df = 14$; $p = .06$; *CFI* = 0.975; *RMSEA* = 0.047 (*95% CI* 0.000–0.080). Second, the residual variance of PTSS at T2 (parameter p) proved significantly higher ($p = .04$) for mothers than fathers; parameter p was therefore freed across parent gender (model 1). Third, the stability of PTSS (parameter e) was significantly higher ($p = .04$) in mothers than in fathers. The final model, model 2, with the two aforementioned parameters freed across parent gender, displayed an excellent fit to the data: *S-B* $\chi^2 = 12.88$; $df = 12$; $p = .38$; CF = .998; RMSEA = 0.016 (95% CI 0.000 – 0.063).

Severity of the child's medical condition. As Figure 2 shows, initial days in hospital significantly predicted high PTSS in the parents (parameter c1) but not in the child (m1). Initial days in hospital did not predict change in PTSS across time (c1–2; m1–2). Additional days in hospital were associated with more PTSS in parents at T2 (c2) but not in the child (m2). Overall, medical stress had a stronger impact on parents than on the child at both T1 ($p < .01$) and T2 ($p < .01$).

Familial associations in post-traumatic stress symptoms. The initial familial cross-sectional associations in PTSS were large and significant between the parents (h1), but they were small and insignificant between parent and child (d1).

Table 2 Numbers and percentages of individuals with significant PTSS (child) or DSM-IV PTSD (parents), and means and *SD*'s of PTSS scales

	All	Injuries	Diabetes	Cancer	χ^2	*F*	*p*
T1							
Child RI > 24 (*n* = 270)	30 (11.1%)	22 (16.5%)	3 (4.3%)	5 (7.4%)	8.14		.02
Mean (*SD*) child RI	13.10 (9.81)	14.92[a] (11.10)	9.05[b] (7.35)	13.65[a] (8.06)		8.75	.000
PTSD mother (*n* = 239)	70 (29.3%)	21 (18.6%)	14 (24.1%)	35 (51.5%)	23.15		.000
Mean (*SD*) PDS mother	12.67 (8.44)	11.14[a] (8.34)	12.07[a] (8.49)	15.70[b] (7.87)		6.69	.001
PTSD father (*n* = 221)	41 (18.6%)	11 (10.5%)	7 (11.9%)	23 (40.4%)	24.20		.000
Mean (*SD*) PDS father	9.11 (7.50)	7.23[a] (6.67)	8.71[a] (6.38)	12.98[b] (8.62)		12.05	.000
T2							
Child RI > 24 (*n* = 246)	25 (10.2%)	21 (17.4%)	1 (1.6%)	3 (4.7%)	13.81		.001
Mean (*SD*) child RI	12.22 (8.71)	14.18[a] (9.93)	8.42[b] (6.21)	12.16[a] (7.03)		9.49	.000
PTSD mother (*n* = 213)	31 (14.6%)	5 (5.1%)	11 (19.6%)	15 (25.4%)	13.81		.001
Mean (*SD*) PDS mother	5.43 (6.42)	3.18[a] (4.51)	6.47[b] (7.33)	8.18[b] (6.97)		13.62	.000
PTSD father (*n* = 191)	15 (7.9%)	2 (2.2%)	4 (8.0%)	9 (18.4%)	11.59		.003
Mean (*SD*) PDS father	3.80 (4.84)	2.27[a] (3.94)	3.79[a] (4.02)	6.68[b] (5.82)		15.26	.000

Subgroups with different superscripts are significantly different ($p < .05$ with Sheffe post hoc tests).
PDS, post-traumatic diagnostic scale; RI, child PTSD Reaction Index.

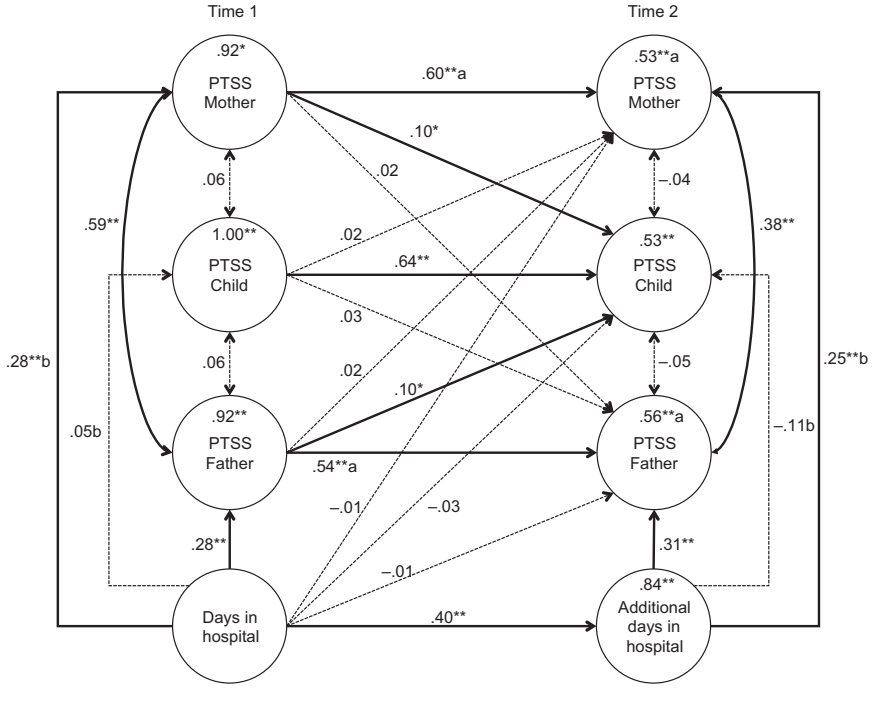

Figure 2 Structural equation model (figures are standardized) *$p < .05$; **$p < .01$. (a) Parameter is significantly different between mothers and fathers ($p < .05$). (b) Significant difference between parent and child ($p < .01$)

Within-individual stability in post-traumatic stress symptoms. Within-individual stability in PTSS, adjusted for days in hospital and familial associations in PTSS, was substantial in both parents (e) and children (j). Mothers had a significantly higher stability than fathers.

Familial effects over time in post-traumatic stress symptoms. High initial levels of PTSS in the parents had an unfavorable effect on the change of PTSS in the child, significantly predicting a higher level of child PTSS at T2 (f). However, there was neither a direct child-parent effect (q) nor a direct parent-parent effect (g) from initial PTSS level to change in PTSS at 1 year after hospitalization.

Co-development in post-traumatic stress symptoms. Intrafamilial co-development in PTSS is indexed by the residual correlations at T2 (parameters d2 and h2). Parents had a substantial degree of co-development across time, but there was no significant co-development between the parents and the child (i.e., change in parental PTSS from T1 to T2 was not related to the child's change in PTSS).

Discussion

The first aim of this study was to assess the incidence and course of PTSS in injured and seriously ill children and their parents. Our results are in line with previous findings that show that a significant

proportion of children and their parents develop clinically relevant PTSS (Cabizuca et al., 2009; Kassam-Adams et al., 2009; Kazak et al., 2004; Landolt et al., 2003, 2005a; Olofsson et al., 2009). The incidence of moderate to severe PTSS in children was between 4.3% and 16.5% at 5–6 weeks, and between 1.6% and 17.4% at 1 year. At both times, children with diabetes showed the lowest rates, and children with injuries had the highest rates. Because PTSS have not been studied in children with diabetes, comparison of our findings with other studies is not possible. The low rates, however, suggest that the diagnosis and treatment of this well manageable disease is only minimally traumatizing for children and that the current model of care in Switzerland works well for these patients. In addition, however the findings could mean that children do not completely understand the implications of their disease.

In this study, children with injuries showed similar rates of PTSS compared to previous studies (Olofsson et al., 2009). However, rates of PTSS in children with cancer were at the lower end compared to previous research (Bruce, 2006). This may be due to heterogeneity of samples across studies, different assessment measures, and differences in timing of follow-up. Also, in Switzerland all children with cancer are supported by psychologists, which might have reduced the rates of PTSS.

As in other studies (Bruce, 2006; Kazak et al., 2005), mothers and fathers in our sample had considerable rates of PTSD across all diagnostic groups and at both times. At 5–6 weeks, overall rates among parents were higher than in children. Specifically, parents of children with cancer and diabetes were affected significantly more than their children. Although parental symptoms decreased over time, PTSD rates in the cancer group were still remarkably high at 1 year (mothers 25.4%; fathers 18.4%). In contrast, only 4.7% of the children with cancer had clinically relevant PTSS. Kazak et al. (2004) found a similar parent-child discrepancy.

Taken together, children and parents seem to be affected differently by accidental injuries and the onset of severe diseases in the child. As suggested by Landolt et al. (2003), these differences might be explained by different pathogenic pathways. Although it is unclear what exactly constitutes the traumatic stressor in severe diseases, the life threat associated with the diagnosis may play an important role for parents. Children, however, may not realize the life threat because of their cognitive immaturity. Conversely, accidents are more immediate events (crash) going along with pain and emergency measures that may be experienced as frightening and distressing more directly by the child, independent of cognitive development.

The second aim of this study was to examine cross-sectional associations, course over time, cross-lagged effects, and co-development in child and parental PTSS while controlling for the influence of the severity of illness/injury as measured by the time in hospital. We found that at both time points the concurrent levels of PTSS were substantially correlated between mothers and fathers but not between either parent and the child. This shows that mothers and fathers react similarly to the stressors of the disease or injury, whereas the child's response is independent of the parents'. The similarity of the parents' PTSS has been reported previously in the literature, but the earlier findings on the cross-sectional associations between child and parental PTSS are inconsistent. Our results are in line with the findings of some studies (Kazak et al., 2004; Landolt et al., 2005b; Valentino et al., 2010) but contradict others (Barakat et al., 1997; Kazak et al., 1997). Methodological differences between studies may account for some of these inconsistencies. Therefore, it remains difficult to draw firm conclusions. The strength of the association between child and parental PTSS might depend on other factors, such as the child's age, preexisting mental health and quality of post-trauma family relationships (Kazak et al., 2006).

Post-traumatic stress symptoms were found to be quite stable over time in both children and parents, i.e., levels of PTSS at 5–6 weeks were found to be highly predictive of levels at 1 year. This finding is well known from the literature and suggests that there is a considerable risk of chronification of PTSS (Bruce, 2006; Olofsson et al., 2009). As reported by Le Brocque et al. (2010a,b) the rates of chronic PTSS trajectories in children and their parents may be around 10–14% after unintentional injuries. In our sample, PTSS were more persistent over time in mothers compared to fathers. This supports epidemiological findings that PTSD is a more chronic condition for women than men (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995).

The structural equation model also included cross-lagged effects of child and parental PTSS. Only one form of significant cross-lagged effect was observed, i.e., that initially high levels of PTSS in mothers and fathers were longitudinally related to poorer recovery of PTSS in the child. Importantly, while we did not find any cross-sectional associations between child and parental PTSS, we found a clear longitudinal effect. This finding is supported by previous studies that assessed parental and child PTSS by means of self-report and that found a longitudinal influence of parental PTSS on child PTSS in injured children (Landolt et al., 2005b; Le Brocque et al., 2010b). A reason for this negative effect might be that the parents' own symptoms prevent them from adequately address the child's needs after the trauma. Thus, the child does not receive the protection of an optimal caretaking relationship (Appleyard & Osofsky, 2003). Parents with PTSS might also avoid speaking openly about the trauma, which in turn has negative impacts on child adaptation (Cabizuca et al., 2009). However, our data do not allow any conclusions about such mechanisms.

In our model, the two other types of cross-lagged effects, from the child to the parent and from the parent to the parent, were not significant. This means that maternal and paternal PTSS were not influenced by child symptoms. This suggests that parents are more important for the course of the child's PTSS than *vice versa*. To our knowledge, this has not been reported previously. The lack of a significant cross-lagged longitudinal effect between parents suggests that the symptomatology in mothers and fathers develops quite independently of each other and may be affected by variables not assessed in this study.

Finally, our model also examined the role of illness/injury-related stress as measured by the length of hospitalization. The effect on parental PTSS at 1 year was mediated through parental PTSS at 5–6 weeks and through the number of additional days in hospitals. In children, however, medical stress had no influence on PTSS, thus confirming previous findings (Bruce, 2006; Kazak et al., 2006; Olofsson et al., 2009). Our results suggest that the distress associated with the medical condition has a greater impact on the parents than on the child. However, since distress was only measured by days in hospital, this conclusion needs to be confirmed by other studies.

Limitations

This study provides several new findings, but some limitations need to be mentioned. First, generalization of our results to other types of traumas is unclear. Second, we do not know if parents completed their questionnaires separately. A possible reason for the high association between maternal and paternal PTSS could be that the parents filled out the questionnaires together. Third, parental PTSD was not assessed by clinical interview but by questionniare. Although the PDS has shown high agreement with diagnoses obtained from clinical interviews, the PTSD rates in our study have to be interpreted with caution. Fourth, families with lower socioeconomic status were underrepresented in our sample. This may have influenced our findings by reducing variations in the stressors the families experienced. Fifth, whereas the FIML estimation of missing data increases the generalizability of the findings, standard errors, and therefore *p*-values, for family members with fewer observations are larger. This means increased risk for underpowering and type II errors in fathers when comparing them with mothers who had a higher response rate. Finally, although the child's age was not bivariately associated with the variables of our model, it is still possible that there are moderating effects of age.

Implications

Our findings have several clinical implications. Most importantly, this study underlines the importance of parental PTSS for the ill or injured child's long-term adjustment. Clinicians need to evaluate parent and child functioning independently at an early time after trauma. Children whose mothers and/or fathers show high levels of PTSS should be considered at greater risk for post-traumatic maladjustment. As highlighted by Kassam-Adams et al. (2009), clinicians treating trauma-exposed children have to address the needs of parents to reduce parents' own distress and also to promote parents' optimal assistance to their children. Therefore, interventions that are tailored to prevent or treat PTSS in pediatric patients should include parents (Kazak et al., 2006). Moreover, we found PTSS in children to be quite stable over time. Therefore, early identification and treatment of symptomatic children is crucial to prevent a chronic course. Finally, although maternal and paternal PTSS were significantly associated cross-sectionally, they did not influence each other over time. This implies that clinicians need to address the spouses' needs separately.

This study also suggests some issues for future research. Importantly, our findings need to be replicated in samples of nonmedically traumatized children. Also, it is still unclear in what ways parental PTSS influence the child. Previous studies have highlighted the role of impaired parenting behavior, but this has to be studied in more detail. In addition, the age of the child may be important with regard to reciprocal associations of child and parental PTSS. The possibility of moderating and confounding effects of child age should be examined more thoroughly in future studies.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Table S1 Pearson correlations between length of hospital stay and variables of injury/illness severity.

Table S2 Pearson correlations between child age at diagnosis and model variables.

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting materials supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.

Acknowledgements

This study was funded by grants from the Swiss Research Foundation Child and Cancer, the Gebert Ruef Foundation, the Hugo and Elsa Isler Foundation, the Anna Mueller Grocholski Foundation, and Bayer Diagnostics. We thank the families who participated in this project.

Correspondence to

Markus A. Landolt, PhD, University Children's Hospital Zurich, Steinwiesstrasse 75, CH-8032 Zurich, Switzerland. E-mail: markus.landolt@kispi.uzh.ch

Key points

- Accidents and life-threatening diseases in children can cause post-traumatic stress in both the child and the parents. Little is known about the mutual influence of child and parental symptoms over time.
- There are considerable differences regarding symptoms of post-traumatic stress between medical conditions and between child, mother, and father.
- Initially high levels of post-traumatic stress in mothers and fathers were longitudinally related to poorer recovery of the traumatized child.
- Cross-lagged effects from the child's symptoms to the parents and from one parent to the other could not be found.
- There is a need for a family systems approach and for early interventions in injured and severely ill children.

References

Appleyard, K., & Osofsky, J.D. (2003). Parenting after trauma: Supporting parents and caregivers in the treatment of children impacted by violence. *Infant Mental Health Journal*, *24*, 111–125.

Barakat, L.P., Kazak, A.E., Meadows, A.T., Casey, R., Meeske, K., & Stuber, M.L. (1997). Families surviving childhood cancer: A comparison of posttraumatic stress symptoms with families of healthy children. *Journal of Pediatric Psychology*, *22*, 843–859.

Brislin, R.W., Lonner, W.J., & Thorndike, R.M. (1973). *Cross-cultural research methods*. New York: John Wiley & Sons.

Bruce, M. (2006). A systematic and conceptual review of posttraumatic stress in childhood cancer survivors and their parents. *Clinical Psychology Review*, *26*, 233–256.

Cabizuca, M., Margques-Portella, C., Mendlowicz, M.V., Coutinho, E.S., & Figueira, I. (2009). Posttraumatic stress disorder in parents of children with chronic illnesses: A meta-analysis. *Health Psychology*, *28*, 379–388.

Foa, E.B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, *9*, 445–451.

Frederick, C., Pynoos, R., & Nader, K. (1992). *Childhood posttraumatic stress reaction index – A copyrighted instrument*. Los Angeles: University of California.

Griesel, D., Wessa, M., & Flor, H. (2006). Psychometric qualities of the German version of the posttraumatic diagnostic scale (PTDS). *Psychological Assessment*, *18*, 262–268.

Helfricht, S., Latal, B., Fischer, J.E., Tomaske, M., & Landolt, M.A. (2008). Surgery-related posttraumatic stress disorder in parents of children undergoing cardiopulmonary bypass surgery: A prospective cohort study. *Pediatric Critical Care Medicine*, *9*, 217–223.

Hu, L.-T., & Bentler, P.M. (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods*, *3*, 424–453.

Kassam-Adams, N., Fleisher, C.L., & Winston, F.K. (2009). Acute stress disorder and posttraumatic stress disorder in parents of injured children. *Journal of Traumatic Stress*, *22*, 294–302.

Kazak, A.E., Alderfer, M., Rourke, M.T., Simms, S., Streisand, R., & Grossman, J.R. (2004). Posttraumatic stress disorder (PTSD) and posttraumatic stress symptoms (PTSS) in families of adolescent childhood cancer survivors. *Journal of Pediatric Psychology*, *29*, 211–219.

Kazak, A.E., Barakat, L.P., Meeske, K., Christakis, D., Meadows, A.T., Casey, R., & Stuber, M.L. (1997). Posttraumatic stress, family functioning, and social support in survivors of childhood leukemia and their mothers and fathers. *Journal of Consulting and Clinical Psychology*, *65*, 120–129.

Kazak, A.E., Boeving, C.A., Alderfer, M.A., Hwang, W.T., & Reilly, A. (2005). Posttraumatic stress symptoms during treatment in parents of children with cancer. *Journal of Clinical Oncology*, *23*, 7405–7410.

Kazak, A.E., Kassam-Adams, N., Schneider, S., Zelikovsky, N., Alderfer, M., & Rourke, M.T. (2006). An integrative model of pediatric medical traumatic stress. *Journal of Pediatric Psychology*, *31*, 343–355.

Kessler, R.C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C.B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, *52*, 1048–1060.

Landolt, M.A., Nuoffer, J.M., Steinmann, B., & Superti-Furga, A. (2002). Quality of life and psychologic adjustment in children and adolescents with early treated phenylketonuria can be normal. *Journal of Pediatrics*, *140*, 516–521.

Landolt, M.A., Vollrath, M., Laimbacher, J., Gnehm, H.E., & Sennhauser, F.H. (2005a). Prospective study of posttraumatic stress disorder in parents of children with newly diagnosed type 1 diabetes. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 682–689.

Landolt, M.A., Vollrath, M., Ribi, K., Gnehm, H.E., & Sennhauser, F.H. (2003). Incidence and associations of parental and child posttraumatic stress symptoms in pediatric patients. *Journal of Child Psychology and Psychiatry*, *44*, 1199–1207.

Landolt, M.A., Vollrath, M., Timm, K., Gnehm, H.E., & Sennhauser, F.H. (2005b). Predicting posttraumatic stress symptoms in children after road traffic accidents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 1276–1283.

Le Brocque, R.M., Hendrikz, J., & Kenardy, J.A. (2010a). Course of posttraumatic stress in children: Examination of recovery trajectories following traumatic injury. *Journal of Pediatric Psychology*, *35*, 637–645.

Le Brocque, R.M., Hendrikz, J., & Kenardy, J.A. (2010b). Parental response to child injury: Examination of parental posttraumatic stress symptom trajectories following child accidental injury. *Journal of Pediatric Psychology*, *35*, 646–655.

Muthén, L.K., & Muthén, B.O. (2010). *Mplus user's guide*, (6th edn). Los Angeles: Muthén & Muthén.

Nader, K., Pynoos, R., Fairbanks, L., & Frederick, C. (1990). Children's PTSD reactions one year after a sniper attack at their school. *American Journal of Psychiatry*, *147*, 1526–1530.

Olofsson, E., Bunketorp, O., & Andersson, A.L. (2009). Children and adolescents injured in traffic – Associated psychological consequences: A literature review. *Acta Paediatrica*, *98*, 17–22.

Satorra, A., & Bentler, P.M. (1994). Corrections to test statistics and standard errors in covariance structure analysis. In A. von Eye, & C.C. Clogg, (Eds), *Latent variable analysis: Applications for developmental research*. Thousand Oaks, CA: Sage.

Scheeringa, M.S., & Zeanah, C. (2001). A relational perspective on PTSD in early childhood. *Journal of Traumatic Stress*, *14*, 799–815.

Shudy, M., De Almeida, M.L., Ly, S., Landon, C., Groft, S., Jenkins, T.L., & Nicholson, C.E. (2006). Impact of pediatric critical illness and injury on families: A systematic review. *Pediatrics*, *118*(Suppl. S), S203–S218.

Valentino, K., Berkowitz, S., & Smith Stover, C. (2010). Parenting behaviors and posttraumatic symptoms in relation to children's symptomatology following a traumatic event. *Journal of Traumatic Stress*, *24*, 403–407.

Accepted for publication: 28 November 2011
Published online: 29 December 2011