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Stressful Life Events and Posttraumatic Stress Symptoms in Children with Cancer

Joseph M. Currier[1,2], **Lisa E. Jobe-Shields**[1,2], and **Sean Phipps**[1]

1*St. Jude Children's Research Hospital, Memphis, Tennessee*

2*Department of Psychology, University of Memphis*

Abstract

This study examined the contribution of stressful life events (SLEs) in posttraumatic stress symptoms (PTSS) stemming from childhood cancer among 121 patients. When controlling for demographic characteristics (age, gender, ethnicity, and socioeconomic status), cancer factors (treatment status, time since diagnosis, and cancer type), and intensity of parental PTSS, history of SLEs in the child's life emerged as a salient correlate of PTSS across the different measures and reporting methods used in the study. Overall, children who had experienced more frequent and severe life stressors endorsed greater PTSS in relation to the cancer experience. Clinical work and future research on children with cancer should focus accordingly on the potential cumulative impact of SLEs on PTSS.

Keywords

posttraumatic stress; anxiety; childhood cancer; stressful life events

Childhood cancer can be a devastating experience that places patients at increased risk for disruption in psychological functioning. Accordingly, the fourth edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV) expanded the list of possible A1 stressors for posttraumatic stress disorder (PTSD) to include a "diagnosis of a life-threatening illness" (American Psychiatric Association, 1994). Since this modification in criteria, many studies have investigated the incidence of PTSD among child cancer patients (see Bruce, 2006, for review). Despite some initial suggestion about high rates of PTSD (Nir, 1985), subsequent research found that the incidence of the full PTSD syndrome was relatively low in this population (Barakat et al., 1997; Kazak et al., 1997; Stuber et al. 1994). Researchers have therefore shifted their focus onto subclinical levels of posttraumatic stress symptomatology (PTSS) as a way of gauging the severity of psychological difficulties stemming from childhood cancer. As a growing body of evidence has demonstrated that PTSD occurs at low rates among child cancer patients, it has become all the more important to identify specific factors associated with trauma responses in this population.

Research has shown that certain subsets of children are indeed at greater risk for PTSS than others. For example, female patients appear to be more vulnerable than their male counterparts (Stuber et al., 1997). Although findings are less consistent, research has also explored how the age of the child and the socioeconomic status (SES) of his or her family can influence one's ability to adapt to the cancer experience (Landolt et al., 2003). Cancer factors such as whether the child is receiving active treatment, the amount of time elapsed since diagnosis, and the type

Direct correspondence to: Sean Phipps, Ph.D. Division of Behavioral Medicine, St. Jude Children's Research Hospital, 262 Danny Thomas Place Memphis, TN 38105-2794 Email: sean.phipps@stjude.org..

of cancer may each play important roles as well. Compared to long-term survivors, there is some evidence to suggest a higher incidence of PTSS among children on active treatment (Pelcovitz et al., 1998) and those patients who recently received a diagnosis of malignancy (Phipps, Long, Hudson, & Rai, 2005). However, these two factors usually overlap with one another, which can create problems distinguishing unique associations with PTSS. Relations between cancer type and PTSS typically go unreported in studies (Bruce, 2006), thereby making it difficult to conjecture whether a particular type of malignancy places patients at increased risk of PTSS compared to other forms of cancer. In contrast, severity of parental PTSS in response to the child's cancer is a commonly studied risk factor that has emerged as a significant correlate of child PTSS across a number of studies. As one would anticipate, research has shown that children with parents who are traumatized by the cancer experience have a greater vulnerability to cancer-related PTSS themselves (Barakat et al., 1997; Kazak et al., 1997; Stuber et al., 1994, 1996).

Perhaps a more underemphasized but possibly critical factor regarding adaptation to childhood cancer pertains to a history of stressful life events (SLEs) in the child's life. SLEs have been linked with vulnerability to a range of problematic consequences in children, including behavior issues (MacLean, Perrin, Gortmaker, & Pierre, 1992), worsened physical health (Heisel et al., 1973), and maladjustment at home and school (Hodges, London, & Colwell 1990). With respect to posttraumatic stress, epidemiologic research has demonstrated that individuals who meet criteria for PTSD tend to report a history of multiple potentially traumatic events (PTEs) rather than an isolated experience (Helzer, Robins, & McEvoy, 1987). In this vein, recent empirical work on adaptation to childhood cancer has shown a similar pattern: a minority of long-term young adult cancer survivors reported experiencing a PTE (29%), and the majority of this subgroup (69%) identified an experience other than cancer as the most stressful (Gerhardt et al., 2007). From a clinical standpoint, Gerhardt et al.'s (2007) work highlights the challenging task often facing clinicians of pinpointing the primary versus secondary stressors for the small but significant subset of cancer-affected children who display problematic levels of PTSS. Importantly, these results may also support a "multiple hit hypothesis" for traumatized child patients for whom cancer may either serve as the hit that precipitates a posttraumatic stress reaction or represent a first or second of several hits that together increase the risk of poor adaptation to a subsequent PTE or SLE that may not satisfy the A1 criterion for PTSD.

There is already some suggestion that the accumulation of SLEs in general increases vulnerability to poor adaptation to childhood cancer, which itself involves a number of distinct challenges (e.g., diagnosis and treatment) that frequently persist over time (e.g., follow-up visits, late effects, threat of recurrence). Evidence suggests that long-term survivors and their parents report a greater prevalence of SLEs than non-affected controls (Brown, Madan-Swain, & Lambert, 2003; Manne, Duhamel, & Redd, 2000). Also, it appears that the association between SLEs and PTSS is strongest when assessing lifetime prevalence rather than focusing on concurrent stressors or recent events (Pelcovitz et al., 1996; Stuber et al., 1997). The relative contribution of SLEs in PTSS has only been investigated in a few studies that focused strictly on long-term survivors (Barakat et al., 2000; Stuber et al., 1997) or their mothers (Manne, Duhamel, & Redd, 2000; Pelcovitz et al., 1996). Of the available research on long-term survivors, results converged in that the accumulation of stressors added to the severity of PTSS up to several years following the completion of treatment (Barakat et al., 2000; Stuber et al., 1997). However, none of these studies included children on active therapy or incorporated parent report of life events or PTSS for the child, which suggests the need for more research on this topic.

Further examination of the association between the accumulation of SLEs and PTSS will increase our understanding of the nature of children's trauma responses to cancer. In addition

to examining the influence of demographic characteristics (age, gender, ethnicity, and SES), cancer factors (treatment status, time since diagnosis, and cancer type) and parental PTSS, this study assessed the relative contribution of other SLEs in the child's life in explaining levels of cancer-related PTSS. The sample included both children on active cancer treatment and survivors who had completed therapy. We also relied on a combination of self- and parent-report of SLEs and PTSS for the children. In keeping with prior research, we hypothesized that cancer factors and severity of parental PTSS would each account for differences in PTSS among the children. However, we also hypothesized that the accumulation of SLEs in the child's life would uniquely account for levels of PTSS endorsed in relation to the cancer experience as well.

Method

Participants

Following institutional review and approval of the study, 121 patients with a diagnosis of a malignant disease were recruited from outpatient clinics of a major children's cancer center. This data set represents three of four groups from a sample that was surveyed to also examine adaptive style (Phipps, Larson, Long, & Rai, 2006) and other factors (e.g., time since diagnosis, effects of informant; Phipps et al., 2005). The group of young adult survivors included in earlier studies was excluded due to a lack of parent data. Therefore, for each child included in this investigation, one parent completed assessments of SLEs and PTSS. Of the patients who were approached to participate, 91% agreed to do so. Please refer to Table 1 for information characterizing demographics and cancer variables among the children in the sample.

Procedure

After completing informed consent procedures, patients and parents were administered standardized measures of SLEs and PTSS. Patients and parents were asked to complete the measures separately and not to consult each other. Research assistants were available to help with the completion of measures as needed.

Measures

Life Events Questionnaire (LEQ)—A modified version of Coddington's (1972) questionnaire was used to assess SLEs. Considering the focus on the cumulative effects of stressors, items that referred to desirable life events were not included (e.g., "outstanding personal achievement"). Also, because each child was receiving or had completed treatment for cancer, it seemed redundant to include an item on serious illness. As presented in Table 2, the LEQ consisted of 22 items, each of which required a "yes" or a "no" response according to whether the event had occurred in the child's life. Abuse and death of a parent were the only PTEs assessed on the LEQ; other events would likely not meet the A1 criterion for PTSD. When the participant endorsed an item, they also rated whether the event occurred in the past year. Because items in Coddington's (1972) measure are not expected to covary, its psychometric properties usually go unreported. A prior study using a different sample found that ratings of SLEs by child cancer patients and their parents converged at a greater degree than healthy children and their parents (Johnston, Steele, Herrera, & Phipps, 2003). Outside of two events that occurred with low frequency in the present sample, child and parent ratings both converged and correlated highly, $r(111) = .80$, $p < .001$. Therefore, rather than conducting two sets of analyses per child and parent report of SLEs, we used the mean of child and parent report on the LEQ to create a single composite measure for SLEs.

UCLA PTSD Index for DSM-IV (PTSDI; Pynoos et al., 1998)—This is a revised version of a measure formerly known as the PTSD Reaction Index (RI; Pynoos et al., 1987). The RI was designed to assess DSM-III-R PTSD criteria, and the PTSDI has been revised for the DSM-

IV. Similar versions are available for self-report by children and by parent report. Excellent internal reliability and test-retest reliability have been reported, and considerable data is available regarding the instrument's validity (Steinberg, Brymer, Decker, & Pynoos, 2004). We implemented a 22-item version designed for childhood cancer. Patients were instructed to complete the PTSDI with specific reference to their own symptoms stemming from cancer, and parents completed an identical version in reference to their child's cancer-related symptoms as well. Earlier work has demonstrated that scores of 38 and higher on the PTSDI are severe in nature (Steinberg et al., 2004). Coefficient alphas were .89 by child report and .88 by parent report, and scores on the PTSDI for children and their parents were also correlated on this measure, $r(113) = .7$, $p < .001$.

Impact of Events Scale — Revised (IES-R; Horowitz, Wilner, & Alvarez, 1979; Weiss & Marmar, 1997)—The 22-item IES-R measures PTSS in response to a specific traumatic event, (Weiss & Marmar, 1997), which again focused on the child's cancer. The IES and IES-R have been used in several studies of childhood cancer (e.g., Barakat et al., 1997; Kazak et al., 1997). Identical versions have been used by both parent and child, with just minor rewording of some items in the child version. Internal reliability (α) for the scale was .91 by child report and .95 by parent report. Both patients and parents completed the IES-R as a self-report of PTSS in relation to childhood cancer. Thus, children completed the PTSDI and IES-R as self-reports, while parents completed the PTSDI referring to their child's symptoms and the IES-R referring to their own PTSS.

Rating the Severity of Life Events

The LEQ we used included a range of SLEs for children. Coddington (1972) created "Life Change Units" to gauge the severity of the events, which are summed to generate an estimate of the child's presumed level of stress. In the present study, a Q-sort procedure was used to provide an index of stressfulness for each of the 22 items selected for this study. Ratings were performed by seven researchers with interests in trauma and childhood cancer. Two of these raters had Ph.D's, two had Master's degrees, and three had B.A.'s in psychology or a related field. Each researcher rated items on a 9-point Likert-type scale with anchor points of 1 = *Least Stressful* and 9 = *Most Stressful*. Using the intraclass correlation coefficient (ICC), interrater reliability was .64 when comparing differences between raters and .93 for differences based on the average rating for each item. The average rating was used as an index to weight the level of stressfulness for each item (see Table 2). Raters consistently identified death of a parent and abuse (the two PTEs) as the most stressful, followed by parental divorce, incarceration of a parent, parental separation, and learning that one was adopted.

Data Analysis

We used the weighted life events scores throughout the analyses. However, we also performed the analyses using a simple frequency count of SLEs for the children and the pattern of results was the same as reported in this paper. Following frequency analyses of SLEs, we performed two sets of statistical analyses. These included (1) univariate analyses for SLEs and PTSS to determine whether these factors were significantly related and (2) three multivariate hierarchical regressions to explore whether the number of severity of SLEs in the child's life accounted for unique variance in cancer-related PTSS beyond demographics (age, gender, ethnicity, and SES), cancer factors (treatment status, time since diagnosis, and cancer type), and severity of parental PTSS (assessed by the IES-R).

Results

Frequency of Life Events

Children experienced a mean of five SLEs over their lifetime according to self- (M = 4.74, SD = 5.11, range = 0 to 14) and parent-report (M = 5.11, SD = 3.16, range = 0 to 14). On average, patients (M = 1.98, SD = 1.96, range = 0 to 11) and parents (M = 2.02, SD = 1.98, range = 0 to 9) each indicated that two events had occurred in the past year. These results are consistent with those of Johnston and colleagues (2003) who found that both children with cancer and healthy children experienced around five SLEs over the lifetime with two stressors occurring in the past year. In the present sample, only 4% of the children had not experienced a SLE. Nevertheless, only 8% of the children and parents endorsed 10 or more events, none of whom reported above 14. The majority (59%) had experienced between three to seven lifetime stressors. Using the stressfulness ratings shown in Table 2, the mean weighted SLE score for the children was 26.45 (SD = 16.84).

We also investigated the impact of SLEs from the past year. As with prior work (Pelcovitz et al., 1996; Stuber et al., 1997), events that occurred in the past year did not have the same influence on PTSS as the total accumulation of stressors in the child's life. Because the past-year variable failed to achieve statistical significance and other results stayed the same across the analyses, we focused on lifetime prevalence of SLEs rather than simply examining recent or concurrent stressors.

Levels of Child PTSS

In terms of severity of PTSS among the children, 13% and 7% had scores that exceeded the clinical cutoff on the PTSDI (i.e., scores above 38; Steinberg et al., 2004) according to self- and parent-report, respectively. The mean levels of child PTSS were 19.46 (SD = 13.5) and 18.68 (SD = 11.8) per self- and parent-report on the PTSDI, respectively. Children had a mean score of 14.25 (SD = 13.6) on the IES-R, which was significantly lower than the average level of parental PTSS (M = 20.78; SD = 18.23), paired $t(114) = 4.18$, $p < .001$. Results of univariate analyses showed that children with a greater number and severity of SLEs had more cancer-related PTSS (PTSDI child self-report $r(109) = .21$, $p = .03$; PTSDI parent report $r(109) = .31$, $p = .001$; and IES-R child self-report $r(109) = .22$, $p = .02$).

Predicting Child PTSS

Child Self-Report of PTSS on the PTSDI—In an effort to evaluate whether the significant correlations between SLEs and child PTSS would explain differences in child PTSS beyond other risk factors, we conducted three multivariate regression analyses predicting scores on PTSS measures (see Table 3). Using child self-report on the PTSDI as the dependent variable in the first analysis, the overall regression model was statistically significant, $R^2 = .31$, F (12, 91) = 3.34, $p < .001$. Notwithstanding a marginal trend for more parental PTSS to associate with higher child PTSS, $p = .1$, SLEs for the child was the only factor to explain unique variance in child PTSS, $p = .02$.

Parent Report of Child PTSS on the PTSDI—The second regression analysis focused on parent report of child PTSS on the PTSDI as the outcome variable. The overall model achieved statistical significance, $R^2 = .43$, F (12, 91) = 5.68, $p < .001$. Among the individual predictors, severity of parental PTSS, $p < .001$, and the accumulation of SLEs, $p = .01$, accounted for unique variance in child PTSS. Results showed that children with more traumatized parents and those who had experienced more SLEs had significantly worse PTSS.

Child Self-Report of PTSS on the IES-R—Using child self-report on the IES-R as the outcome, the overall model was again statistically significant, $R^2 = .40$, F (12, 91) = 5.07, $p <$.

001. However, contrary to the initial analyses, cancer factors explained a significant portion of the variance in this analysis, $p = .01$. Of the three cancer factors, several of the contrasts between cancer types were significant. Specifically, children without ALL endorsed more PTSS than children with ALL, $p = .05$, and children with other types of leukemia, $p < .001$, and HD/NHL, $p = .05$, each reported more PTSS compared to children with other forms of malignancy. As with other results, intensity of parental PTSS, $p < .001$, and the number and severity of SLEs, $p = .01$, accounted for unique variance in child PTSS in this analysis.

Discussion

Childhood cancer can be a highly stressful experience, and there are several factors that may increase the risk of poor adaptation for children. This study examined the contribution of several of these factors in PTSS, including demographic considerations, factors pertaining to the cancer experience itself, severity of parental PTSS, and the cumulative impact of SLEs in the child's life. Consistent with other research, the majority of children did not report clinical levels of PTSS. Nonetheless, the intensity of PTSS was shown to vary on the basis of several risk factors, which suggests that certain subsets of the children were more vulnerable than others. There was some suggestion that the type of cancer increased risk for PTSS; however, these results are difficult to interpret given that they were isolated to a single outcome measure. Treatment status and time since diagnosis both failed to significantly correlate with PTSS when entered in the same model. However, when analyzed separately, each of these cancer factors have yielded significant associations with PTSS (Pelcovitz et al., 1998; Phipps et al., 2005), and the current pattern of results likely highlights the overlap between them rather than a lack of clinical utility. As with prior research on childhood cancer (Barakat et al., 1997; Kazak et al., 1997; Stuber et al., 1994, 1996), we also found that levels of parental PTSS associated with child PTSS when controlling for other risk factors. In terms of identifying at-risk patients, children with parents who endorsed more severe PTSS had a greater vulnerability to trauma responses themselves.

Beyond these risk factors, the accumulation of SLEs in the child's life emerged as a salient predictor of adjustment to childhood cancer. After controlling for demographics, cancer factors, and levels of parental PTSS, the lifetime prevalence of SLEs uniquely contributed to the intensity of trauma responses across the different measures and reporting methods used in the study. In view of the frequencies of SLEs among the patients, the majority had experienced multiple challenges besides dealing with cancer. On average, children and their parents each reported the occurrence of five difficult experiences. Events endorsed with the greatest frequency included the death of a grandparent, close friend or other relative followed by financial problems, the birth of a sibling, and parental separation. Although only a small percentage of the patients experienced a PTE that may meet the A1 criterion for PTSD, we found that as the number and magnitude of these stressors increased, children's vulnerability to trauma responses to cancer worsened as well. These results converge with other work (Gold, Marx, Soler-Baillo, & Sloan, 2005) to suggest that non-A1 stressors that are not life-threatening and do not precipitate intense feelings of fear can influence the intensity of PTSS and even engender clinically significant difficulties for some children.

A related interpretation pertains to the overall cumulative impact of SLEs. The majority of patients in the sample had been "hit" multiple times by varying degrees of life stressors. For patients at the beginning stages of treatment, these experiences probably occurred before the diagnosis of malignancy was made such that difficulties associated with cancer may have directly precipitated PTSS. However, we also included long-term survivors who presumably faced challenges in the period after the successful completion of therapy. In these instances, the cancer experience may have served as one of several hits that increased vulnerability to future difficulties rather than directly giving rise to PTSS. Following Gerhardt and colleagues'

findings (2007), many of these survivors may have even viewed a non-cancer event as being the most difficult. Another possibility is the occurrence of SLEs after cancer may have retriggered a traumatic reaction to cancer among a subset of survivors. We cannot offer definitive statements as to whether the accumulation of prior SLEs catalyzed the tendency toward PTSS in response to cancer, if the cancer experience may have served as a diathesis that moderated the onset of PTSS following a subsequent PTE, or even whether post-cancer SLEs had a traumatic retriggering impact among survivors in the sample. Instead, this study simply explored the cumulative impact of SLEs among child cancer patients and did not focus on these finer relations. Although this decision conforms to other approaches (Barakat et al., 2000; Stuber et al., 1997), SLEs from different time points may have had a different type of impact on adaptation to cancer. Future research would do well to expand on these findings by exploring other details of a multiple hit hypothesis for children with cancer.

The present results indicate that severity of cancer-related PTSS for child patients may in part reflect the occurrence of SLEs both related and unrelated to the cancer experience, which raises several implications for clinical practice. Beyond assessing the impact of cancer factors and parental functioning, these findings suggest that clinicians should gather information pertaining to other difficult experiences in the child's life. For those children who experienced multiple stressors, close monitoring of functioning following diagnosis of malignancy and during active treatment could be warranted. In instances of elevated PTSS or other types of symptomatology (e.g., depression, behavior problems), intervention targeting the child and his or her family may prove helpful. The present results also indicate the importance of focusing on the lifetime prevalence of SLEs as opposed to only assessing the influence of recent or concurrent stressors on the child. Additionally, among selected patients who manifest sufficient symptomatology to require psychological treatment, the present results raise the possibility that intervention need not focus exclusively on cancer-related issues, but should also attempt to help the child and his or her family deal with prior or ongoing stressors that may not appear directly cancer-related.

This study had several limitations that may have affected the present conclusions. We have already noted the lack of assessment of the timing of SLEs in relation to the onset of cancer. We also did not include a group of healthy children and their parents. In view of research suggesting rates of PTSD are not higher for children who survive cancer than controls (e.g., Barakat et al., 1997), it is possible that patients would not have manifested greater PTSS than non-affected peers. In turn, analyses may have failed to demonstrate that cancer status accounted for significant differences in PTSS, which would have complicated the present results. Another limitation pertains to our reliance on survey methods to gauge PTSS. Based on low levels of PTSD demonstrated in most studies (Bruce, 2006), we approached PTSS as a dimensional construct, focusing on the full spectrum of symptom severity. However, diagnostic interviews are considered the standard for clinical assessment of trauma responses, and it is possible that we missed certain indications of distress by relying solely on questionnaire data.

With respect to grading the stressfulness of the life events, we relied on objective ratings from cancer researchers rather than on the participants themselves. Although we viewed this procedure as a viable alternative to Coddington's (1972) original method, we may have done better to include questions on the LEQ that allowed participants to rate the subjective level of stress for items they endorsed. For example, SLEs in the LEQ could have been more or less distressing depending on a variety of developmental and contextual factors for the child, and we were not able to account for these possibilities with the present measurement strategy. A final limitation to note is that we directed the children and parents to rate PTSS specifically in relation to cancer. As Gerhardt and colleagues (2007) found, many of the patients may not have viewed cancer as their most traumatic experience. Particularly for long-term survivors

who had recovered and were no longer facing as strong a possibility for death, this approach possibly limited the ability to understand the impact of other SLEs.

Despite these limitations, this study provided important information on the apparent interplay between SLEs and PTSS stemming from the experience of childhood cancer. Clinical work and research with child cancer patients would do well to focus accordingly on the potential cumulative impact of SLEs over the child's lifetime.

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Table 1

Demographic and Cancer Variables

Child age [*M (SD)*]	12.9 (3.0)
Years since diagnosis	4.8 (5.3)
Gender [*N* (%)]	
Male	62 (51.2)
Female	59 (48.8)
Ethnicity	
Caucasian	93 (76.9)
African American	25 (20.7)
Other	3 (2.5)
SES[a]	
I & II	34 (28.2)
III	38 (31.6)
IV & V	49 (40.0)
Treatment status	
Receiving active therapy	74 (61.2)
Completed therapy	47 (38.8)
Type of cancer	
ALL[b]	42 (34.7)
Other leukemia	14 (11.6)
HD/NHL[c]	14 (11.6)
Solid tumor	40 (33.1)
Brain tumor	11 (9.1)
Parent respondent	
Mother	99 (81.8)
Father	18 (14.9)
Other[d]	4 (3.3)

[a] *Note*. Socioeconomic status per Hollingshead four-factor index (Hollingshead, 1975)

[b] Acute lymphocytic leukemia

[c] Hodgkins disease/non-Hodgkins lymphoma

[d] Stepparent or grandparent as custodial guardian

Table 2

Negative Life Events: Prevalence, Parent/Child Agreement, and Mean Rating of Stressfulness

Item	Lifetime			Past Year			
	Child Prevalence	Parent Prevalence	κ	Child Prevalence	Parent Prevalence	κ	Stressfulness (1 to 9)
Birth of a sibling	39.1%	50.0%	.75	4.1%	6.6%	.60	3.43
Parents separate	34.2%	29.3%	.80	6.6%	6.6%	.60	7.00
Parents divorce	25.4%	21.5%	.90	2.5%	2.5%	.66	7.71
Parent remarried	19.1%	19.8%	.74	2.1%	5.0%	.43	6.41
Serious illness of parent	22.6%	22.4%	.47	9.1%	10.7%	.63	7.71
Serious illness of sibling	16.5%	12.9%	.52	7.4%	5.8%	.60	6.43
Parent died	4.3%	5.2%	.91	0	.8%		9.00
Grandparent died	53.9%	47.4%	.69	17.4%	15.7%	.82	6.43
Relative or close friend died	50.4%	50.0%	.53	24.8%	24.8%	.65	5.86
Learn of being adopted	4.3%	1.7%	.27	.8%	.8%	-.01	7.00
Parent lost job and out of work for awhile	22.8%	25.9%	.64	14.9%	16.5%	.56	5.57
Parent started a new job	19.5%	18.3%	.77	10.7%	9.9%	.69	3.43
Parent spent less time at home with the child	25.4%	27.6%	.52	15.7%	17.4%	.52	4.86
Family member moved in to the home	9.6%	10.5%	.47	1.7%	5.0%	.23	3.71
Sibling left home	28.1%	18.1%	.68	11.6%	9.9%	.66	3.43
Child was abused	2.6%	1.7%	-.02	.8%	.8%	-.01	8.86
Parents fought more	20.0%	17.2%	.59	11.6%	10.7%	.63	5.57
Parent went to jail	6.1%	2.6%	.42	3.3%	1.7%	.66	7.71
Family moved school districts in same city	14.8%	12.9%	.66	7.4%	7.4%	.64	3.43
Family moved to new city	21.7%	18.1%	.64	7.4%	4.1%	.40	5.00
Parents began to worry about money	43.5%	50.0%	.56	31.4%	34.5%	.51	4.00
Drug or alcohol problems with a family member	11.3%	8.6%	.40	5.8%	4.1%	.48	6.71

Note.κ = measure of inter-rater agreement for which values from .49 to .74 are typically considered acceptable and values of .75 or greater are viewed as excellent agreement (Fleiss, 1981)

Table 4

Demographics, Cancer Factors, Parental PTSS, and Stressful Life Events Predicting Child PTSS

	PTSDI (Self-Report)				PTSDI (Parent Report)				IES-R (Self-Report)			
Predictor	***B***	***SE B***	**β**	***R*²**	***B***	***SE B***	**β**	**R²**	***B***	***SE B***	**β**	***R*²**
Demographics				.04				.03				.04
Age	0.47	0.46	.11		0.14	0.37	.04		0.35	0.42	.08	
Gender	2.79	2.52	.11		2.97	2.02	.13		1.72	2.28	.07	
Ethnicity (Caucasian vs. other groups)	2.62	2.97	.08		0.67	2.38	.02		4.88†	2.69	.16†	
SES	0.13	0.09	.13		0.11	0.08	.13		0.14	0.09	.14	
Cancer Factors				09†				.07				.11*
Active tx (1 = yes or 0 = no)	4.01	3.54	.15		3.73	2.84	.16		3.76	3.21	.14	
Time since diagnosis	0.00	0.00	-.04		0.00	0.00	-.03		0.00	0.00	-.05	
Type of diagnosis												
ALL[a](1 = yes or 0 = no)	3.72	4.62	.13		3.09	3.70	.13		8.30*	4.18	.31*	
Other leukemia (1 = yes or 0 = no)	9.70	5.29	.24		3.73	4.24	.11		17.74*	4.79	.45*	
HD/NHL[b](1 = yes or 0 = no)	5.56	5.64	.14		6.20	4.52	.18		10.02*	5.10	.26*	
Solid tumor (1 = yes or 0 = no)	1.10	4.80	.04		1.93	3.85	.08		6.19	4.35	.22	
Parental PTSS	0.13†	0.08	.17†	.02†	0.27*	0.06	.41*	.12*	0.26*	0.07	.35*	.09*
Stressful life events	0.10*	0.04	.24*	.05*	0.08*	0.03	.23*	.04*	0.09*	0.04	.24*	.05*

[a] *Note*.Acute lymphocytic leukemia

[b] Hodgkins disease/non-Hodgkins lymphoma.

* $p < .05$

† < .1