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Childhood trauma and risk for PTSD: Relationship to intergenerational effects of trauma, parental PTSD, and cortisol excretion

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Abstract
Among the adverse mental health consequences of childhood trauma is the risk related to the development of posttraumatic stress disorder (PTSD) in adulthood. Other risk factors for PTSD, including parental trauma exposure and parental PTSD, can also contribute to the experience of child trauma. We examined associations between childhood trauma and PTSD in 51 adult children of Holocaust survivors and 41 comparison subjects, in consideration of parental trauma exposure and parental PTSD. We also examined these variables in relation to 24-hr urinary cortisol levels. Adult offspring of Holocaust survivors showed significantly higher levels of self-reported childhood trauma, particularly emotional abuse and neglect, relative to comparison subjects. The difference was largely attributable to parental PTSD. Self-reported childhood trauma was also related to severity of PTSD in subjects, and emotional abuse was significantly associated with 24-hr mean urinary cortisol secretion. We conclude that the experience of childhood trauma may be an important factor in the transmission of PTSD from parent to child.

There has been considerable interest in examining the impact of childhood maltreatment on the mental health of the adult. To date, both retrospective and prospective studies have demonstrated that individuals exposed to childhood trauma—specifically, physical and sexual abuse and physical neglect—are more likely to show symptoms of posttraumatic stress disorder (PTSD) compared to persons who do not report such experiences (e.g., Bremner, Southwick, Johnson, Yehuda, & Charney, 1993; Elliott & Briere, 1995; Epstein, Saunders, & Kilpatrick, 1997; Follette, Polusny, & Milbeck, 1994; Widom, 1999; Zlotnick, Zakriski, Shea, Costello, Begin, Pearlstein, & Simpson, 1996). In addition to PTSD, many other psychological difficulties have been documented in adults maltreated as children. These include anxiety (e.g., Briere, Evans, Runtz, & Wall, 1988; Briere & Runtz, 1988; Bushnell, Wells, & Oakley–Browne, 1992; Greenwald, Leitenberg, Cado, & Tarran, 1990; Moeller, Bachmann, & Moeller, 1993; Sedney & Brooks, 1984), depression (e.g., Briere et al., 1988; Bushnell et al., 1992; Fromuth & Burkhart, 1989; Sedney & Brooks, 1984), suicidality (e.g., Brown & Anderson, 1991; Moeller et al., 1993; van der Kolk, Perry, & Herman, 1991), dissociation (e.g.,

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Briere, 1988; Chu & Dill, 1990; Nash, Hulsey, Sexton, Harralson, & Lambert, 1993), personality disorders (e.g., Johnson, Smailes, Cohen, Brown, & Bernstein, 2000; Luntz & Widom, 1994; Silverman, Reinherz, & Giaconia, 1996), and substance abuse (e.g., Epstein, Saunders, Kilpatrick, & Resnick, 1998; Fleming, Mullen, Sibthorpe, & Bammer, 1999; Merrill, Newell, Thomsen, Gold, Milner, Koss, & Rosswork, 1999). Other problems, particularly among those who experienced sexual abuse, include impaired sexual functioning (Briere, 1994; Brunngraber, 1986; Finkelhor, 1979; Gold, 1986; Lindberg & Distad, 1985; Meiselman, 1978; Stein, Golding, Siegel, Burman, & Sorenson, 1988; Tsai, Feldman–Summers, & Edgar, 1979), interpersonal problems (Kendall–Tackett & Simon, 1988; Wyatt & Newcomb, 1990), and physical illness (Felitti, 1991).

The number and severity of potential consequences is alarming and has served to highlight childhood maltreatment as a major social and public health problem. Ironically, however, the wide range and lack of specificity of negative outcomes associated with childhood trauma also makes it more difficult to identify trauma as a major precipitant of any of the above-mentioned consequences. Although numerous outcomes have been associated with childhood trauma, no outcome has been shown to be a *specific* indicator of early maltreatment, occurring in all, or even most, exposed individuals. Formulating the precise mechanism through which exposure to childhood maltreatment culminates in the spectrum of adverse consequences observed in the adult may require a consideration of how other risk factors interact with early trauma exposure (Cicchetti & Rizley, 1981). As part of this process, it is necessary to examine whether and how specific types and doses of early trauma may result in distinct consequences. Elucidating these relationships is a necessary prerequisite for developing a model that explains both the diversity of responses to adversity in childhood and the factors that contribute to resilience and vulnerability to trauma in adulthood.

In considering the specific outcome of increased PTSD symptoms among adults exposed to childhood maltreatment, investigators have emphasized the importance of subsequent traumatic events in adulthood that serve to revictimize the adult survivor (Bremner et al., 1993; Zlotnick et al., 1996). There is increasing evidence that childhood trauma is directly associated with risk for victimization in adulthood and that such reexposure results in PTSD in adults abused as children. The relationship between a history of child sexual abuse and vulnerability to rape was recently confirmed in a study by Nishith, Mechanic, and Resick (2000), which used path analysis to examine the relative contributions of both these experiences to current PTSD symptomatology. Although subjects with childhood sexual abuse had more adult PTSD than those who did not, the intervening adult rape was both predicted by childhood abuse and the precipitant of PTSD symptoms in the adult. Similarly, in a large, prospective study that followed children to adulthood, elevated PTSD symptomatology was observed in those adults who had experienced childhood physical or sexual abuse and physical neglect compared to those without such exposure (Widom, 1999). However, here too those with childhood maltreatment were far more likely to experience events such as rape, physical assault, or other traumatic events in adulthood. Moreover, subjects with histories of child trauma were more likely to come from families with significant problems (parents being arrested, drugs or alcohol in family, low level of education subsequent to abuse, early behavioral problems prior to abuse experiences). These risk factors alone were found to be highly contributory to PTSD, even in the absence of child abuse experiences.

The above studies suggest that there are likely to be numerous factors affecting the relationship between childhood trauma and adult PTSD, including the risk factors associated with exposure to childhood trauma. Indeed, childhood maltreatment, particularly chronic maltreatment, does not occur in a vacuum. Since childhood should be a time of adult supervision and protection, prolonged abuse or neglect during this period may be indicative of a larger breakdown in the familial, social, or cultural environment that per-

mits maltreatment, or fails to notice and prevent it from occurring (Cicchetti & Lynch, 1993). At the very least, childhood trauma can be understood as resulting from more global environmental adversity or chaos, implicating some form of neglect. The presence of these additional elements may help to explain the contrast between the low rates of PTSD observed in children who experience single traumatic events, or discrete chronic traumas such as war (e.g., Pynoos, Frederick, Nader, Arroyo, Steinberg, Eth, Nunez, & Fairbanks, 1987; Sack, Clarke, Him, Dickason, Goff, Lanham, & Kinzie, 1993; Schwarz & Kowalski, 1991; Terr, 1981), and the high rates of symptomatology associated with ongoing childhood maltreatment. The child who has an otherwise stable and secure environment can identify discrete traumatic events as being unusual. A child who need only recover from the effects of an event, and not also from the more pervasive factors that gave rise to that event, may be less vulnerable to mental health consequences in adulthood.

This paper will consider the specific outcome of PTSD in relation to both childhood trauma and to two other risk factors that have been associated with PTSD—namely, parental trauma exposure and parental PTSD. There is evidence linking trauma exposure in a parent to both subsequent maltreatment of children (e.g., Kaufman & Zigler, 1989; Ertem, Leventhal, & Dobbs, 2000; Oliver, 1993) and to an increased prevalence of PTSD in the offspring following exposure to trauma in adulthood (Yehuda, Schmeidler, Wainberg, Binder–Brynes, & Duvdevani, 1998). In addition, we have recently hypothesized that parental PTSD, rather than just parental trauma exposure, may be a more specific risk factor for the development of PTSD in offspring (Yehuda, Schmeidler, Giller, Siever, & Binder–Brynes, 1998).

The current research examines the associations between childhood trauma and PTSD in adult children of Holocaust survivors, in consideration of parental trauma exposure and parental PTSD. We also examine these variables in relation to 24-hr urinary cortisol levels in offspring. Offspring of Holocaust survivors represent an ideal population for such investigation. Relative to demographically similar comparison subjects, they show an increased prevalence of PTSD despite comparable rates of adult trauma exposure (Yehuda, Schmeidler, Giller, et al., 1998; Yehuda, Schmeidler, Wainberg, et al., 1998). Offspring of Holocaust survivors are generally stable, well-educated persons of medium to high sociodemographic status, with relatively low levels of substance abuse. Thus, the risk factors associated with the development of PTSD in offspring are likely to be largely confined to variables associated with parental trauma exposure or parental symptoms following exposure.

The Holocaust marked a watershed in modern human history, because of the systematic exposure of millions of individuals to previously unimaginable acts of human cruelty. To the extent that parental traumatic experiences are contributors to child trauma, the early experiences of children of Holocaust survivors provide a strong test of that hypothesis. Between 50 and 75% of Holocaust survivors meet the diagnostic criteria for lifetime PTSD; therefore, it is also possible to examine the contribution of parental PTSD symptoms to childhood trauma in offspring in this population. By examining interrelationships among childhood trauma, PTSD, parental PTSD, and cortisol in this unique population, we may begin to describe more complex constellations associated with individual differences in responsiveness to stress.

Method

Participants

Fifty-one offspring (20 men, 31 women) and 41 comparison participants (23 men, 18 women) gave informed consent and participated in this study, which was approved by the Institutional Review Board at Mount Sinai School of Medicine. Offspring were defined as having been born to at least one biological parent who experienced the Nazi Holocaust. For the purposes of the current study, Holocaust survivors were individuals who were in a ghetto, a labor or concentration camp, or had to hide in or flee Nazi-occupied territory after 1939.

The comparison participants were Jewish individuals in the same age range (24–60 years) who did not have a parent who was a Holocaust survivor.

The mean age of the offspring group was 40.9 years (SD = 7.6), and that of the comparison participants was 38.3 years (SD = 8.8). The two groups were also comparable in terms of years of education (offspring: M = 17.0, SD = 3.0; comparison: M = 18.2, SD = 2.4) and gender distribution, χ^2 (1) = 2.60, *ns*.

Recruitment for the study was as previously described by Yehuda, Bierer, Schmeidler, Aferiat, Breslau, and Dolan (2000). Primarily, participants were solicited from lists obtained from the Jewish community or responded to community group announcements and newspaper advertisements (n = 79). Others volunteered after taking part in short-term group psychotherapy at the Mount Sinai Specialized Treatment Program for Holocaust Survivors and their Families (n = 13). Since we could study only those who willingly approached us, it is possible that symptomatic individuals are overrepresented in this sample. The fact that one quarter of the offspring were seeking treatment in our clinic suggests a bias towards offspring who freely endorse mental health symptoms. However, the main analyses in the current research are based on divisions of offspring by the presence or absence of parental PTSD, and examinations of the effects of childhood trauma on symptomatology and biology across the sample as a whole. The conclusions drawn from these analyses should be valid even in the presence of a sampling bias among the offspring.

Other than the conditions outlined above, there were no other specific exclusion criteria for the nonbiological (i.e., clinical interview) part of the study, since we were initially interested in examining prevalence of current and lifetime psychiatric disorder (Yehuda, Schmeidler, Wainberg, 1998). However, none of the participants presenting for this study met criteria for a psychotic disorder or had any medical or psychiatric condition associated with significant cognitive impairment. Of the 51 offspring recruited for study, 41 met criteria for at least one past or current psychiatric disorder. Of the comparison participants, 13 of 41 had at least one past or current psychiatric disorder. Diagnoses included PTSD, major depressive disorder, dysthymia, depressive disorder not otherwise specified, generalized anxiety disorder, panic disorder, social phobia, specific phobia, body dysmorphic disorder, anxiety disorder not otherwise specified, substance abuse or dependence, anorexia, bulimia, and adjustment disorder. In terms of the number of current psychiatric diagnoses, 13 of the 51 offspring had a single current diagnosis, 8 had two diagnoses, 5 had three current diagnoses, and 1 met criteria for four current Axis I disorders. Of the comparison participants, 4 had a single current diagnosis, 1 had two current diagnoses, and 1 had three current diagnoses.

A subset of the participants undergoing clinical interviews were eligible or willing to provide biologic specimens (n = 42; 28 offspring, 14 comparison subjects). Participants from the above sample were excluded from urine collection if they had current alcohol or substance abuse problems, an active major medical condition (e.g., cancer, diabetes, nonstabalized endocrinopathies, chronic fatigue syndrome), or had taken beta-blockers, lithium, or other psychotropic medications within 2 months of the study. Participants were not withdrawn from medications to participate in this protocol.

Twenty-three of the participants (19 offspring, 4 comparison participants) who provided urine samples were taking medications at the time of participation. Of these, 10 were taking psychotropic medications, including buproprion, amitriptyline, fluoxetine, buspirone, nefazodone, clonazepan, alprazolam, valproic acid, venlafaxine, gabapentin, and St.-John's-wort, and 18 were taking nonpsychotropic medications, including treatments for allergies, hypertension, hypercholesterolemia, and hormone replacement, pain medication, and oral contraceptives. Evaluation of the effects of medication used by type has not revealed a significant effect on 24-hr urinary cortisol levels (Yehuda, Kahana, Binder–Brynes, Southwick, Mason, & Giller, 1995; Yehuda et al., 2000) and was not considered in statistical analyses.

Sixty-two of the participants in the clinical interview portion of the current study also

took part in a prior study examining the prevalence of trauma and PTSD in Holocaust survivor offspring (Yehuda, Schmeidler, Wainberg, et al., 1998). The current study adds 30 new participants (17 offspring and 13 comparison participants) for whom diagnostic data have not been previously published.

Similarly, cortisol data from 31 of the participants have been reported (Yehuda et al., 2000). These 31 participants represent the total number of participants from the 50 reported previously for whom data about both childhood trauma and parental PTSD were available (i.e., the Childhood Trauma Questionnaire [CTQ] and Parental PTSD Scale). These instruments were added to our assessment battery subsequent to initiating cortisol studies in offspring. Additionally, the current sample includes 11 new participants recruited since the prior publication.

Clinical assessments

All participants in the current study were screened for Axis I disorders using the mental health screening questions from the Structured Clinical Interview for DSM-IV (SCID; Spitzer, Williams, & Gibbon, 1995). Participants who endorsed a positive response on one or more of these questions were then given the complete diagnostic interview using the SCID by a trained clinical rater. Exposure to trauma was assessed using the Trauma History Questionnaire (Green, 1996), which lists a range of traumatic events in three areas (crime, disaster, interpersonal). Participants endorsed any events from this list that they had experienced, and also listed any "other" personal experiences that might have been particularly distressing or frightening. The details of these events were then discussed in order to ascertain which (if any) events met the diagnostic criterion "A" for a traumatic event (i.e., involved life threat, injury, or threat to personal integrity, and elicited the subjective response of fear, helplessness, or horror). Following this exploration, the Clinician Administered PTSD Scale (CAPS; Blake, Weathers, Nagy, Kaloupek, Gusman, Charney, & Keane, 1995) was administered to all participants who reported a traumatic event. Participants who identified more than one trauma were asked to identify their most distressing event, and this was used as the basis for the evaluation of current and lifetime PTSD.

In addition to the clinical interview, participants completed several questionnaires. Described in this paper are the results from two of these measures: the CTQ and the Parental PTSD Scale.

The CTQ (Bernstein, Fink, Handelsman, Foote, Lovejoy, Wenzel, Sarapeto, & Ruggiero, 1994) is a self-administered inventory that assesses experiences of abuse and neglect during childhood. The short form of the questionnaire was used in the current study. Participants were asked to respond to 25 statements on a 5-point Likert scale, rating each experience with respect to the frequency with which it occurred during their childhood (i.e., "when I was growing up"). Statements relate to five dimensions, representing different types of trauma: emotional and physical neglect, and emotional, physical, and sexual abuse. Prior research has demonstrated scores on the CTQ to be stable over time and to be consistent with reports from corroborative sources (Bernstein et al., 1994; Fink, Bernstein, Handelsman, Foote, & Lovejoy, 1995; Walker, Unutzer, Rutter, Gelfand, Saunders, VonKorff, Koss, & Katon, 1999). Factor analysis has further established the content validity of the five subscales (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997; Fink et al., 1995). Dimensional data obtained from the CTQ can be highly skewed and kurtotic (Walker et al., 1999), and this was true in the current study. Therefore, we also employed the method of dichotomizing each scale to reflect the absence or presence of maltreatment in each domain (Bernstein & Fink, 1998; Walker et al., 1999). Thus, both raw scores and dichotomized variables were analyzed and are presented.

The Parental PTSD Scale, developed by our group for use in offspring, was given to participants in order to assess parental Holocaust-related PTSD (Yehuda et al., 2000). The scale requires participants to detail their parents' Holocaust experiences, if any (e.g., ghetto, concentration camp, in hiding). For

each parent who experienced the Holocaust, participants then completed a checklist based on the 17 symptoms of PTSD listed in the fourth edition of the *Diagnostic and Statistical Manual* (DSM-IV; American Psychiatric Association, 1994), rating severity on a 4-point Likert scale. Preliminary evidence suggests that this scale correlates well with independent diagnostic evaluations. In the nine cases where both the Parental Stress History Scale and face-to-face diagnostic assessments of the parents by experienced clinicians were available, there was complete agreement regarding the presence or absence of PTSD (Yehuda et al., 2000).

Biological assessment

Urine was collected for 24 hr after the first voided urine following awakening, including the first voided urine on the following day. Two-liter polyethylene collection bottles were used and kept in freezers in the participants' residences in order to ensure stability of cortisol. Collections were scheduled to occur on days that were anticipated not to be particularly stressful, in order to obtain samples that reflect typical basal secretion. Most participants stayed at home for the 24-hr period in order to facilitate collection. Urinary-free cortisol levels were determined by using an extraction procedure and radioimmunoassay kit from Clinical Assays, Inc. (Cambridge, MA; interassay coefficient of variation was 4.0%).

Results

Research questions and analyses

Four sets of analyses were performed. First, we examined whether parental trauma exposure is related to higher levels of self-reported childhood trauma by comparing the offspring group as a whole with the comparison participants. This question was addressed by comparing scores on each of the CTQ dimensions for offspring versus comparison participants. Two-way analysis of variance (ANOVA; Group × Gender) was used to compare mean scores, and chi-square tests were used to examine the relative frequencies of clinically significant abuse, as indicated by the CTQ dichotomies.

Second, we examined whether parental PTSD specifically (rather than parental trauma exposure) is related to self-reported trauma in offspring by carrying out analyses of CTQ scores (means and dichotomies) within the offspring group, comparing offspring with versus without parental PTSD. We also carried out exploratory analyses looking more specifically at parental PTSD. ANOVA was used to compare CTQ scores in comparison subjects, offspring without parental PTSD, offspring with a single parent with PTSD, and offspring with both parents with PTSD. Pearson's correlations were used to assess dose–response effects, examining associations between parental PTSD symptoms and offspring CTQ scores. The correlational analyses were repeated separately for maternal and paternal symptoms.

Third, we examined the extent to which childhood trauma represents a risk factor for the development of PTSD. This was accomplished by performing chi-square tests comparing the frequency of PTSD in individuals with and without self-reported childhood trauma. In addition, we examined whether there are differences in self-reported childhood trauma in participants reporting PTSD to a focal trauma occurring in adulthood compared to those who experienced a trauma in adulthood but did not develop PTSD. Correlations between mean CTQ scores and PTSD symptoms were also performed in the subset of participants reporting an adult trauma. This allowed us to assess whether childhood trauma is a risk factor for PTSD to focal traumas that occur independently of childhood events. Finally, logistic regression was used to assess the extent to which self-reported childhood trauma predicted the development of PTSD, over and above the contribution of parental trauma exposure and parental PTSD.

The fourth area of inquiry concerned the relationship of early trauma to 24-hr urinary cortisol secretion. In the first analysis, total 24-hr urinary cortisol was compared using analysis of covariance (ANCOVA) in individuals with and without significant trauma, covarying for age and gender. In the second

Table 1. *Mean scores on CTQ dimensions in offspring of Holocaust survivors and comparison participants by gender*

	Offspring[a]		Comparison[b]		Group Effect		Gender Effect	
CTQ Subscale	*M*	*SD*	*M*	*SD*	*F*	*p*	*F*	*p*
Emotional abuse	12.62	5.85	7.49	3.82	20.67	<.0001	1.60	.210
Women	13.26	6.23	8.11	4.80				
Men	11.65	5.20	7.00	2.75				
Emotional neglect	12.94	5.22	8.42	3.96	20.08	<.0001	0.02	.886
Women	13.00	5.35	8.16	4.70				
Men	12.85	5.15	8.60	3.35				
Physical abuse	7.22	3.22	6.34	3.87	1.37	.24	0.04	.84
Women	7.25	3.50	6.11	3.98				
Men	7.15	2.60	6.52	3.85				
Physical neglect	7.31	2.28	5.54	1.21	19.31	<.0001	0.009	.925
Women	7.39	2.30	5.39	0.97				
Men	7.20	2.28	5.65	1.37				
Sexual abuse	6.43	2.83	5.29	0.90	4.22	.043	5.75	.019
Women	7.10	3.40	5.56	1.29				
Men	5.40	0.75	5.08	0.28				

[a]Women, $n = 31$; men, $n = 20$.
[b]Women, $n = 18$; men, $n = 23$.

analysis, three-way ANCOVAs were used to examine the extent to which associations between cortisol secretion and CTQ dimensions are mediated by the presence of PTSD or depression. Finally, we performed an exploratory ANCOVA in which we accounted for presence or absence of abuse, presence or absence of participant's PTSD, presence or absence of depressive disorder, and parental exposure and PTSD, covarying for age and gender. This analysis was performed to illustrate one type of approach that ultimately might be useful for unpacking the relative contribution of these interrelated variables to 24-hr cortisol secretion. This analysis further permitted an assessment of the effects of childhood adversity on cortisol, taking into consideration the known contributions of the effects of trauma exposure and PTSD.

Childhood adversity as a function of parental trauma exposure

Two-way ANOVA (Group × Gender) comparing total scores on the CTQ (i.e., the sum of the five subscales) indicated that offspring reported more childhood adversity than comparison participants (offspring: $M = 46.53$, $SD = 14.06$; comparison: $M = 33.07$, $SD = 11.08$), $t\ (90) = 5.57$, $p < .0005$. Table 1 presents mean scores on CTQ dimensions in offspring of Holocaust survivors and comparison participants by gender. Offspring reported significantly higher levels of emotional abuse, emotional neglect, physical neglect, and sexual abuse than comparison participants, but not physical abuse. No significant gender differences were observed, except with respect to sexual abuse. When data were analyzed using the dichotomous measures of presence or absence of trauma, chi-square tests indicated that a higher proportion of offspring reported clinically significant abuse on each of the five dimensions of the CTQ (Figure 1).

Childhood trauma in relation to parental PTSD

Of the 51 Holocaust offspring, 19 had parents who did not have lifetime PTSD and 32 had one or both parents with PTSD. Table 2 shows the mean scores and statistical analyses for the five CTQ dimensions in the offspring group. Group comparisons of means indicated that offspring with parental PTSD reported

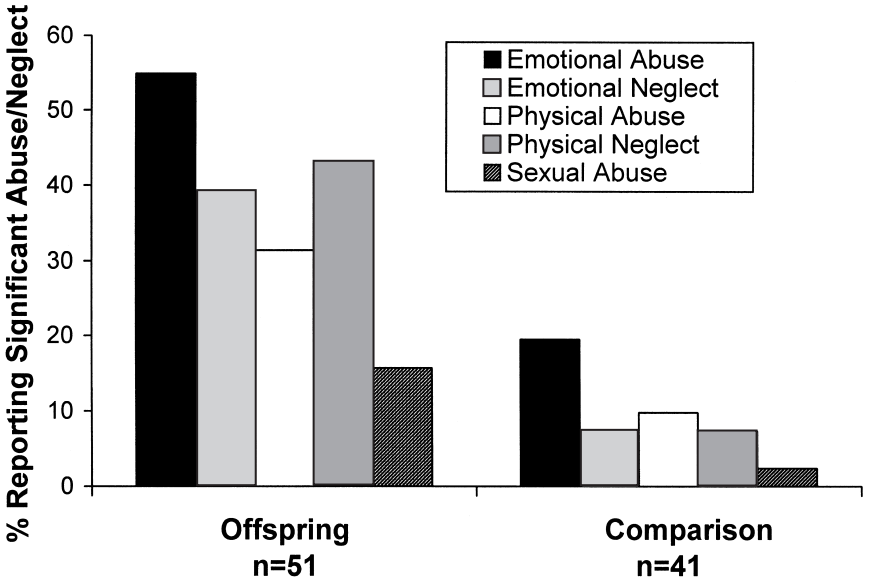

Figure 1. The prevalence of reported abuse and neglect on five CTQ dimensions in Holocaust offspring and comparison participants. The figure shows the percentage of participants reporting clinically significant childhood trauma in each of the five dimensions on the CTQ. The results of chi-square tests are as follows: emotional abuse, χ^2 (1) = 11.95, p = .001; emotional neglect, χ^2 (1) = 12.33, $p < .0005$; physical abuse, χ^2 (1) = 6.24, p = .012; physical neglect, χ^2 (1) = 14.74, $p < .0005$. For sexual abuse, the Fisher's exact test was used because of small expected frequencies in the comparison group, p = .04.

Table 2. *Scores on the childhood trauma questionnaire for offspring of Holocaust survivors grouped by parental PTSD*

	Offspring With Parental PTSD (n = 32)		Offspring Without Parental PTSD (n = 19)		Student's t Test	
CTQ Dimension	*M*	*SD*	*M*	*SD*	t (df = 49)	p
Emotional abuse	14.22	5.68	9.95	5.24	–2.67	.010
Emotional neglect	14.13	5.27	10.95	4.62	–2.18	.034
Physical abuse	7.50	3.04	6.74	3.54	–0.82	*ns*
Physical neglect	7.69	2.10	6.68	2.47	–1.54	*ns*
Sexual abuse	6.16	1.74	6.89	4.08	0.90	*ns*

more emotional abuse and emotional neglect than offspring without parental PTSD. When the differences between offspring with and without parental PTSD were analyzed according to the dichotomous CTQ variables, parental PTSD was associated with a higher incidence of emotional abuse (reported by 66% of offspring with parental PTSD vs. 37% of offspring without parental PTSD), χ^2 (1) = 3.99, p = .046, and physical neglect (reported by 56% of offspring with parental PTSD vs. 21% of offspring without parental PTSD), χ^2 (1) = 6.02, p = .014, but not emotional neglect (reported by 47% of offspring with parental PTSD vs. 26% of offspring without parental PTSD), χ^2 (1) = 2.11, *ns,* physical abuse (reported by 38% of offspring with parental PTSD vs. 21% of offspring without parental

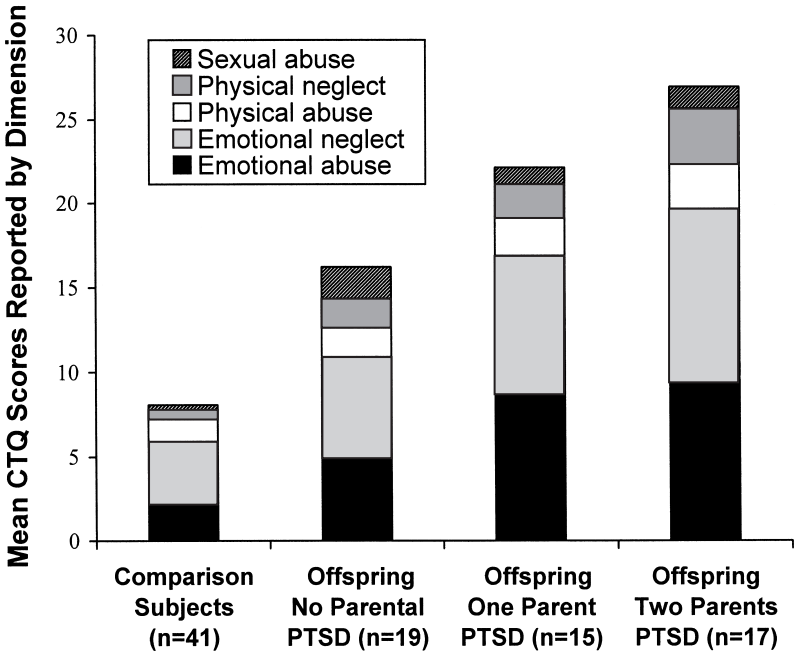

Figure 2. The CTQ scores in offspring with and without parental PTSD and comparison participants. The mean scores for each of the five CTQ dimensions in the four subject groups are shown. The results of the ANOVA are as follows: emotional abuse, F (3, 92) = 11.61, $p < .0001$; emotional neglect, F (3, 92) = 9.93, $p < .0001$; physical abuse, F (3, 92) = .684; *ns*; physical neglect, F (3, 92) = 9.763, $p < .0001$; sexual abuse, F (3, 92) = 2.53, $p < .06$. The results of the post hoc tests using Tukey's HSD are as follows: for emotional abuse, comparison participants were significantly different from offspring with one or both parents with PTSD; offspring with no parental PTSD were significantly different from offspring with both parents with PTSD. For emotional neglect, comparison participants were significantly different from offspring with one or both parents with PTSD; offspring with no parental PTSD were significantly different from those with both parents with PTSD. For physical neglect, comparison participants were significantly different from offspring with one or both parents with PTSD; offspring with no parental PTSD were significantly different from offspring with both parents with PTSD.

PTSD), χ^2 (1) = 1.50, *ns,* or sexual abuse (reported by 13% of offspring with parental PTSD vs. 21% of offspring without parental PTSD), χ^2 (1) = .66, *ns*.

In further considering the impact of parental pathology on offspring experiences, of the 32 offspring with parental PTSD, 15 offspring had one parent with PTSD and 17 offspring had two parents with PTSD. Figure 2 illustrates the mean score for each CTQ dimension in comparison participants and in offspring subdivided into three groups according to parental PTSD status (i.e., neither, one, or both parents with PTSD). ANOVAs revealed main effects of group on mean CTQ scores for emotional abuse and for emotional and physical neglect but not for physical or sexual abuse. However, post hoc tests did not reveal significant differences between offspring with one versus two parents with PTSD. (See Figure 2 legend for F values and results of post hoc testing.)

The correlation between severity of total parental PTSD in Holocaust survivor parents (i.e., the sum of mother and father's PTSD symptoms) and total CTQ scores was significant when the offspring group was considered separately, r (51) = .411, $p = .003$ (Figure 3). Among the offspring, there was a similar relationship between offspring total scores on the CTQ and maternal ($r = .45$, $n = 42$, $p = .003$) and paternal ($r = .39$, $n = 47$, $p = .006$) PTSD

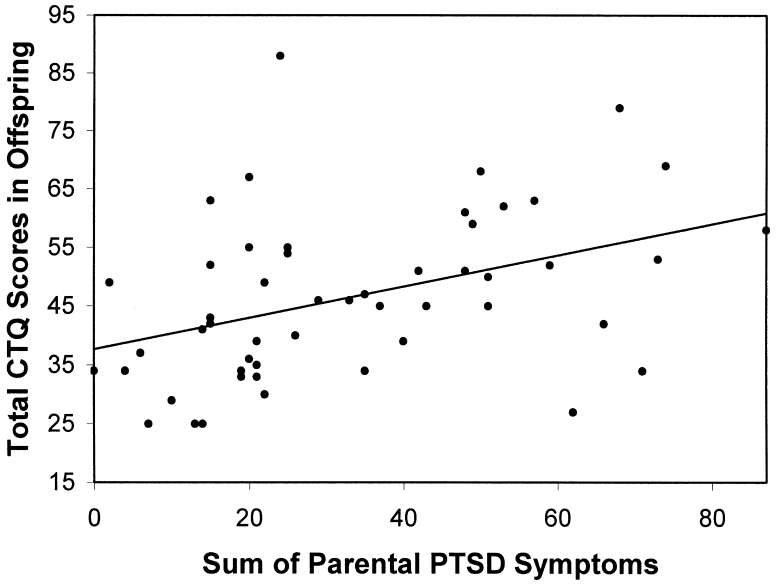

Figure 3. The relationship between total CTQ score and parental PTSD symptoms in offspring of Holocaust survivors. Pearson's correlation coefficient r (51) = .411, p = .003.

symptoms. (Differences in subject number reflect the fact that some offspring only had one Holocaust survivor parent.)

Childhood trauma in relation to psychopathology

There was no difference in the proportion of offspring versus comparison participants who reported a traumatic event of sufficient magnitude to meet the diagnostic criterion "A" for PTSD in the CAPS interview (74.5 and 65.9%, respectively), χ^2 (1) = 0.82, *ns*. Similarly, there were no group differences in the number of lifetime traumatic events reported on the Trauma History Questionnaire (offspring: M = 6.2, SD = 3.6; comparison: M = 5.4, SD = 4.1), t (83) = 1.74, *ns*. These results are consistent with those previously reported from a larger data set that included 38 offspring and 24 comparison participants from the current sample (Yehuda, Schmeidler, Wainberg, et al., 1998). Despite the comparable rates of trauma exposure, significantly more offspring developed PTSD in response to their trauma (N = 17; 33.3%) relative to comparison participants (N = 5; 12.2%), χ^2 (1) = 5.58, p = .018, consistent with the prior report.

Examination of the extent to which childhood trauma represents a risk factor for the development of PTSD indicated that a higher proportion of individuals with PTSD reported significant trauma in terms of emotional abuse and neglect, physical neglect, and sexual abuse, but not physical abuse (Table 3).

The relationship between childhood trauma and PTSD was also considered specifically in participants endorsing a criterion "A" event (n = 65; 38 offspring, 27 comparison participants). Correlations between scores on the five CTQ dimensions and severity of lifetime PTSD as assessed by the CAPS total score within this subgroup of participants were examined. Pearson's correlation coefficients were as follows: emotional abuse, r (65) = .45, $p < .0005$; emotional neglect, r (65) = .37, p = .003; physical abuse, r (65) = .23, p = .065; physical neglect, r (65) = .45, $p < .0005$; sexual abuse, r (65) = .34, p = .006. Offspring were found to be at greater risk for PTSD and scored higher on each of the CTQ dimensions than comparison participants; therefore, we repeated this analysis controlling for group

Table 3. *Incidence of clinically significant childhood trauma (CTQ) in individuals with and without PTSD*

	Lifetime PTSD (n = 22)		No PTSD (n = 70)		Chi-Square Test	
CTQ Subscale	n	%	n	%	χ^2 (df = 1)	p
Emotional abuse	15	68.2	21	30.0	10.25	.001
Emotional neglect	10	45.5	13	18.6	6.45	.011
Physical abuse	8	36.4	12	17.1	—[a]	.076
Physical neglect	12	54.5	13	18.6	10.95	.001
Sexual abuse	5	22.7	4	5.7	—[a]	.033

[a]Fisher's exact test used instead of chi-square because of small expected frequencies.

(offspring vs. comparison participants). The partial correlations were as follows: emotional abuse, r (62) = .24, p = .059; emotional neglect, r (62) = .34, p = .006; physical abuse, r (62) = .18, p = .158; physical neglect, r (62) = .36, p < .005; sexual abuse, r (62) = .27, p = .029. Thus, controlling for group only substantially altered the relationship between emotional abuse and PTSD.

To analyze the relationship between childhood trauma and the development of PTSD to a focal trauma experienced in adulthood, chi-square tests were carried out in the subset of 37 participants (16 offspring, 21 comparison participants) who reported a trauma in adulthood. Comparing individuals who developed PTSD in response to their adult trauma (n = 10) to those who did not develop PTSD (n = 27), a higher proportion of the PTSD group reported sexual abuse according to the dichotomous CTQ variables (30 vs. 4%; Fisher's exact test p = .052). Comparisons of other subscales did not reveal significant associations between risk for PTSD in adulthood and the occurrence of significant childhood trauma. When the data were analyzed using the continuous variable of CTQ scores and PTSD symptoms as assessed by the CAPS, significant correlations were present in these 37 participants. Pearson correlation coefficients were as follows: emotional abuse, r (37) = .46, p = .004; emotional neglect, r (37) = .13, *ns*; physical abuse, r (37) = .32, p = .055; physical neglect, r (37) = .40, p = .014; and sexual abuse, r (37) = .54, p = .001. When this analysis was repeated controlling for group, the results were similar but somewhat less robust for physical abuse. The partial correlations were as follows: emotional abuse, r (34) = .45, p = .006; emotional neglect, r (34) = .11, *ns*; physical abuse, r (34) = .28, p = .10; physical neglect, r (34) = .36, p = .032; sexual abuse, r (34) = .51, p = .001.

Independent contributions of parental trauma exposure and PTSD and of childhood trauma to the development of PTSD in the offspring

Logistic regression, with offspring lifetime PTSD (present or absent) as the dependent variable, and parental exposure–PTSD (i.e., no Holocaust exposure, no PTSD, PTSD) and CTQ scores as predictors, was carried out separately for each of the dimensions of the CTQ. A blocked enter procedure was used, with the parental PTSD trichotomous variable being forced into the regression equation first and mean CTQ dimension score being entered as a second step. Of the five CTQ dimensions, emotional abuse, χ^2 (1) = 4.55, p = .033, and sexual abuse, χ^2 (1) = 5.96, p = .015, were significant predictors of offspring PTSD over and above parental status, χ^2 (2) = 13.79, p = .001. Emotional neglect, physical abuse, and physical neglect did not predict offspring PTSD over and above parental status, although in the case of physical neglect there

Table 4. *Mean 24-hr urinary cortisol levels for individuals with and without clinically significant self-reported abuse on five CTQ dimensions*

	Mean Urinary Cortisol (μg/day)					
	CTQ Score Above Cutoff		CTQ Score Below Cutoff		Analysis	
CTQ Dimension	*M* (*n*)	*SD*	*M* (*n*)	*SD*	*F* (*df* = 1, 38)	*p*
Emotional abuse	41.56 (21)	17.51	63.32 (21)	26.89	9.84	.003
Emotional neglect	45.38 (11)	19.14	54.95 (31)	26.56	0.53	*ns*
Physical abuse	52.45 (12)	22.57	52.43 (30)	26.23	0.02	*ns*
Physical neglect	44.62 (15)	23.54	56.79 (27)	25.10	1.92	*ns*
Sexual abuse	62.38 (5)	45.44	51.10 (37)	21.57	1.92	*ns*

There was a nonsignificant trend for an effect of gender in the analysis of cortisol by sexual abuse ($F = 3.64$, $p = .06$). Gender was not significantly different in any of the other analyses. Age did significantly contribute to the association between any of the CTQ subscales and cortisol.

was a nonsignificant trend, χ^2 (1) = 3.41, p = .065.

Mean urinary 24-hr cortisol concentration and childhood trauma

For each of the CTQ dimensions, mean cortisol levels were compared in individuals with and without significant abuse according to each of the CTQ dichotomies (ANCOVA, with age and gender as covariates). There were no significant effects of age or gender, and uncorrected means are therefore reported in Table 4. As can be seen from the table, mean cortisol was lower in individuals reporting emotional abuse compared to those without emotional abuse. There were no other significant differences in 24-hr urinary cortisol in individuals divided by the presence or absence of trauma on each of the different CTQ dimensions.

Three-way ANCOVAs examining whether each of the five dichotomous CTQ dimensions were related to urinary cortisol, once the presence or absence of PTSD and of depressive disorder (i.e., major depressive disorder or dysthymia) were included as additional factors (covarying for age and gender), were also performed. Table 5 shows each main effect by CTQ dimension to illustrate the different patterns of findings that emerged when the effect of each type of trauma on urinary cortisol was considered in conjunction with both PTSD and depressive disorder.

As can be seen in Table 5, the only CTQ dimensions that demonstrated a significant relationship to cortisol when the effects of PTSD and depressive disorder were taken into account were emotional abuse and sexual abuse. There was a significant main effect of emotional abuse on cortisol, reflecting a lower mean 24-hr cortisol excretion in those with versus those without clinically significant exposure (Table 5). There were also significant main effects of PTSD, this disorder being associated with decreased 24-hr mean cortisol excretion, and of current depressive disorder, depression being associated with increased 24-hr mean cortisol excretion. Analysis of interaction effects yielded extremely small subject numbers in some subgroups. Nonetheless, both PTSD and depressive disorder showed significant interactions with emotional abuse (illustrated in Figure 4). In individuals without emotional abuse, PTSD (vs. no PTSD) appeared to be associated with slightly higher cortisol levels. In individuals with emotional abuse, PTSD was associated with significantly lower cortisol levels. Thus, the combination of PTSD and emotional abuse appeared to be associated with particularly low cortisol. Regarding the interaction between emotional abuse and depressive disorder, participants with depression but without emotional abuse

Table 5. *Results of 3-way ANCOVAs examining the relationship of each of the CTQ dimensions on urinary cortisol levels in relation to PTSD, and depressive disorder*

	F Values for Effects on 24-hr Urinary Cortisol (3-Way ANOVA With Age and Gender As Covariates)					
	Main Effects (*df* = 1, 32)			Interactions (*df* = 1, 32)		
	CTQ Dimension	PTSD	Depressive Disorder	CTQ × PTSD	CTQ × Depression	PTSD × Depression
Emotional abuse	19.48****	4.68**	9.18***	4.17**	16.32****	0.93
Emotional neglect	0.21	3.57*	0.05	0.15	0.01	0.00
Physical abuse	0.20	4.00*	0.34	0.30	0.00	0.08
Physical neglect	3.84*	0.99	0.02	3.02*	0.59	0.00
Sexual abuse	8.30***	9.72***	1.51	8.34***	2.84	0.52

*$p < .10$. **$p < .05$. ***$p < .01$. ****$p < .001$.

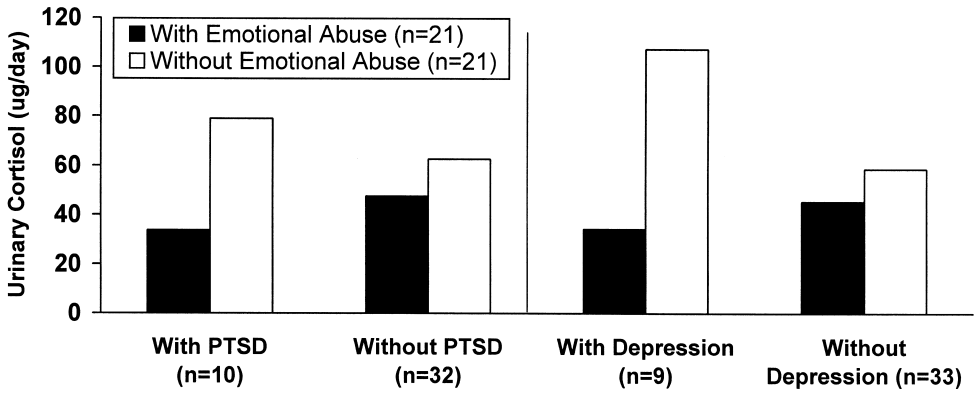

Figure 4. The interaction between emotional abuse and PTSD and depressive disorder in relation to urinary cortisol levels.

had the highest cortisol levels, whereas participants with both depression and emotional abuse tended to have low cortisol levels. In the absence of depressive disorder, the difference in 24-hr urinary cortisol between individuals with and without emotional abuse was much smaller. In the aggregate, the results of this three-way ANCOVA indicate that emotional abuse is associated with lower cortisol levels, regardless of comorbid PTSD or depressive disorder.

With regard to analyses of sexual abuse and cortisol, there was a main effect of sexual abuse on cortisol levels. However, in contrast to the effects of emotional abuse, sexual abuse was associated with higher cortisol levels (Figure 5). There were also main effects of PTSD and depression on 24-hr urinary cortisol, as above. There was a significant interaction between sexual abuse and PTSD. Sexual abuse in the absence of PTSD was associated with very high cortisol levels, whereas when sexual abuse occurred in persons with PTSD cortisol levels were as low as in persons with PTSD but without sexual abuse. Thus, the sexual abuse is generally associated with increased cortisol levels unless it co-occurs with PTSD.

We have previously described in a larger sample (of which this is a subset) lower cortisol levels in association with parental PTSD (Yehuda et al., 2000). We therefore performed an ANOVA to determine whether parental PTSD is associated with cortisol levels

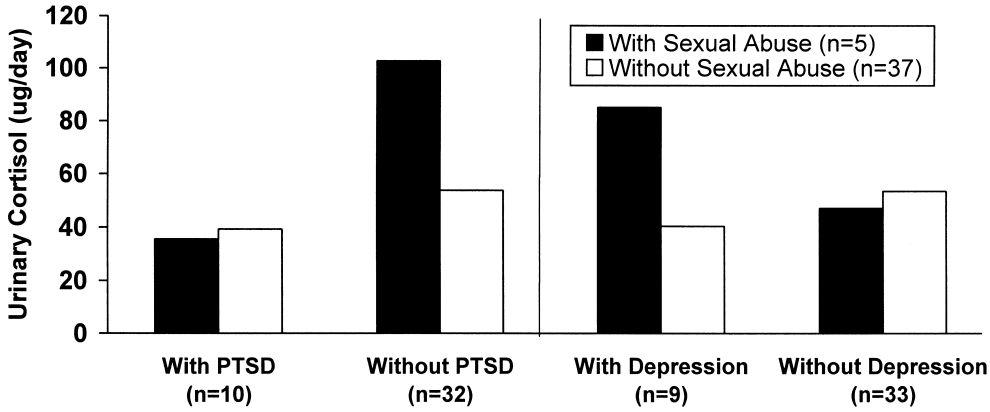

Figure 5. The interaction between sexual abuse and PTSD and depressive disorder in relation to urinary cortisol levels.

in this smaller sample. Dividing participants into three groups (comparison participants, $n = 14$; offspring without parental PTSD, $n = 8$; offspring with parental PTSD, $n = 20$), there was a main effect of group, $F(2, 39) = 4.25$, $p = .021$, on 24-hr urinary cortisol. Post hoc testing confirmed that 24-hr urinary cortisol secretion was significantly lower in the offspring with parental PTSD ($M = 42.06$, $SD = 21.87$) compared to offspring without parental PTSD ($M = 67.90$, $SD = 29.82$) and comparison participants ($M = 58.44$, $SD = 20.90$). Thus, both emotional abuse and parental PTSD appear to be associated with low cortisol and risk for PTSD.

Accordingly, to further examine the relationship of reported childhood emotional and sexual abuse to cortisol, we performed an ANCOVA that would additionally account for parental PTSD, as well as the offspring's own psychopathology (as described above). This analysis was not performed for the three other CTQ dimensions, because these were not associated with cortisol. The first analysis examining cortisol in relation to emotional abuse demonstrated a significant main effect of parental PTSD, $F(2, 29) = 3.32$, $p = .05$. More importantly, even after accounting for presence or absence of parental trauma exposure and parental PTSD, the participants own PTSD, and depressive disorder, and covarying for age and gender, the main effect of emotional abuse was still quite salient, $F(2, 29) = 7.95$, $p = .009$. When parental PTSD was added as a main effect to the analysis examining the effect of sexual abuse on cortisol, none of the previously observed effects remained significant.

Discussion

The major finding of the current study is that adult offspring of Holocaust survivors show significantly higher levels of self-reported child trauma compared to demographically similar comparison subjects. This result was largely attributed to the presence of PTSD in Holocaust survivor parents of offspring. Scores on the CTQ were also related to severity of lifetime PTSD symptoms in subjects. One of the five CTQ dimensions, emotional abuse, was significantly associated with 24-hr mean urinary cortisol excretion. The relationship between parental trauma and childhood trauma in offspring has not been previously examined in Holocaust survivors and their children. Rather, studies have focused on examining the intergenerational transmission of symptoms in response to the Holocaust. This study adds to the field by providing evidence that childhood trauma may be an important agent of such symptom transmission from parent to child.

In interpreting the above findings, the scores obtained in this group of subjects should be considered in relation to those of other populations that have been evaluated with the CTQ. For the present comparison

group, mean scores on each of the five CTQ dimensions were lower than any previously reported for either nonpsychiatric or psychiatric groups (Bernstein et al., 1994; Bernstein, Ahluvalia, et al., 1997; Bernstein, Stein, Walker, Pogge, Ahluvalia, Stokes, & Handelsman, 1997; Bernstein & Fink, 1998; Bernstein, Jelley, & Handelsman, 1997; Walker et al., 1999). Men in the present comparison group showed scores that were 18% lower for emotional abuse, 18% lower for emotional neglect, 5.6% lower for physical abuse, 17% lower for physical neglect, and 2.3% lower for sexual abuse than previously reported scores for male college undergraduates (Bernstein, Jelley, & Handelsman, 1997). When compared to female college undergraduates (Bernstein, Jelley, & Handelsman, 1997), women in the current sample showed scores that were 23% percent lower for emotional abuse, 11% lower for emotional neglect, 3.0% lower for physical abuse, 12% lower for physical neglect, and 0.71% lower for sexual abuse. The lower severity of child trauma in our comparison group may have served to inflate differences between the two study groups.

For offspring, the mean scores were lower than those observed in the literature in medical or psychiatric samples for physical neglect, physical abuse, and sexual abuse (Bernstein, Ahluvalia, et al., 1997; Bernstein & Fink, 1998; Bernstein et al., 1994; Walker, Keegan, Gardner, Sullivan, Bernstein, & Katon, 1997). Emotional abuse and neglect scores offspring were in the same range as those reported for adult psychiatric outpatients, for both men and women (Bernstein & Fink, 1998). Thus, although offspring of Holocaust survivors and comparison subjects differed on all CTQ dimensions except physical abuse, only the severity of emotional abuse and neglect was similar to that observed in a comparable psychiatric population (adult outpatients). Regarding the "clinical significance" of the scores reported, according to the classification of CTQ scale scores (Bernstein & Fink, 1998), the mean for comparison subjects was in the "none or minimal" range for each dimension. For the offspring group as a whole, physical abuse and physical neglect were also in the "none or minimal range." Sexual abuse and emotional neglect were in the "low to moderate" range for the offspring. Emotional abuse was at the low end of the "moderate to severe" range. It will be interesting to compare these scores with those of other populations "at risk" for PTSD.

Correlational analysis revealed a strong relationship between severity of parental PTSD and total CTQ scores. When the offspring group was subdivided on the basis of presence or absence of parental PTSD, it became clear that parental PTSD was associated with a significantly higher level of emotional abuse and neglect but not other CTQ dimensions. Importantly, in terms of presence or absence of trauma on each of the five CTQ dimensions, offspring without parental PTSD were not significantly different from comparison subjects. Having one versus two parents with PTSD also did not significantly relate to the presence or absence of any CTQ dimension. However, as illustrated in Figure 2, there appeared to be a graded effect of having two versus one parents with PTSD, which may be significant in a larger sample.

The finding that parental PTSD, rather than parental trauma per se, is a more consequential variable in relation to self-reported childhood trauma may help to resolve some of the current inconsistencies regarding theories relating to the "cycle of violence." This theory, first articulated by Steele and Pollock (1968), suggested that parents who were themselves abused were far more likely to abuse their own children, who in turn would engage in abusive behavioral patterns with their children. However, a review of the literature of the intergenerational effects of physical abuse indicates that although the base rate of child abuse is substantially higher among parents who have been abused (Kaufman & Zigler, 1989), not all parents who have been abused perpetuate this behavior (Cicchetti & Aber, 1980; Cicchetti & Rizley, 1981). A recent review of studies focusing on transmission of childhood physical abuse concluded that only a minority of studies found evidence for significant transmission of physical abuse from parent to child (Ertem et al., 2000). Similarly, in a critical review of 60 studies of intergenerational transmission of child abuse,

the authors found that about one third of victims grew up to continue a pattern of abusive child rearing (Oliver, 1993). Both reviews noted that most studies that have evaluated the relationship between abuse in parent and child did not control sufficiently for sociodemographic characteristics and other relevant variables.

The relationship between type of parental trauma exposure and the consequent type of child trauma has not been studied systematically. The majority of studies have restricted the types of outcomes evaluated in relation to parental trauma exposure. This implicitly suggests that there have been assumptions that physical abuse in parents promotes physical abuse of children and that sexual abuse in parents leads to sexual abuse in children. Such views are consistent with the idea of a cycle of abuse but take a narrow view of both the risk factors leading to abuse and the widespread nature of the consequences. In the absence of more rigorous study, the conclusion that victims of a certain type of trauma are more likely to victimize others in a similar way may be premature.

The current study of the child trauma in Holocaust offspring provides data to support a more complex model, but this may be due to the uniqueness of the Holocaust survivor sample. In considering the population of Holocaust survivors, a critical distinction can be made from other populations that have been studied in relation to intergenerational effects. In Holocaust survivors, the traumatic event was not inflicted by the parents of survivors but rather by an outside force. Thus, the risk factors for intergenerational transmission of trauma may be different from those that apply to the abused parent. This would be particularly true in survivors who were older during World War II and who had the opportunity to form secure familial attachments. Given the magnitude of the trauma experienced, emotional abuse or neglect in children of Holocaust survivors may relate particularly to parental preoccupation with the event and the symptoms with which survivors had to cope. It is noteworthy that although many Holocaust survivors were physically tortured, there were no significant differences in physical abuse between offspring of Holocaust survivors and comparison subjects. Although there was a significantly higher rate of sexual abuse in offspring of Holocaust survivors, the majority of cases in this sample were not related to sexual abuse by the parent. The higher rate of sexual abuse by others may be an adverse effect of emotional abuse and emotional neglect, which could make the child vulnerable to more active forms of abuse by others.

Emotional abuse and neglect reported by Holocaust survivor offspring may be related to the minimization of their experiences in relation to those of their parents. Emotional abuse was particularly related to PTSD in the parent. A parent recovering from the pain of the Holocaust may not have been in a position to respond with appropriate emotional connectivity as a result of avoidance and numbing symptoms. By minimizing the significance of their child's concerns, a traumatized parent may have intended to console the child by placing his or her perceived crises in perspective. However, it is likely that a young child may have experienced this behavior as invalidating. Furthermore, the extreme suffering and ongoing distress experienced by the Holocaust survivor parent may have induced guilt in their children, both for requiring validation of experiences which are inconsequential relative to the Holocaust and for placing demands on their symptomatic parents. Such experiences in the aggregate would prove to be a formidable challenge to the self-worth of an offspring with symptomatic parent.

Thus, the offspring of a symptomatic Holocaust survivor is in a position to respond to his or her own adverse events in a manner consistent with PTSD. First, as a consequence of the experiences inflicted on his or her parents, the offspring believes that the world is extremely dangerous. Second, as a result of the ongoing minimization of their experiences, the offspring believes himself or herself to be incompetent and unable to cope with more substantial problems. Third, because survivors unwittingly place the burden on their offspring of compensating them for losses sustained during the Holocaust, the offspring often blame themselves for parental symptoms for which they are not responsible.

These three types of maladaptive cognitions have been proposed as risk factors for PTSD and are associated with the development of PTSD symptoms following trauma exposure (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999).

Indeed, in the current study the development of PTSD was associated with a greater degree of self-reported childhood trauma (except for physical abuse) in the entire sample. When analyses were performed only on the subset of subjects reporting a criterion "A" trauma in *adulthood,* strong associations between CTQ dimensions and severity of lifetime PTSD symptoms were observed. When both CTQ dimensions and PTSD were assessed as dichotomous variables, childhood sexual abuse was the only dimension that was specifically associated with the development of PTSD in response to an adult traumatic event.

Childhood trauma and parental PTSD were associated in the current study, and both are putative risk factors for the development of PTSD. We therefore examined whether the relationship between childhood trauma and PTSD was a reflection of the association of each of these variables with parental PTSD. In a relatively conservative analysis, logistic regression found that both emotional abuse and sexual abuse predicted risk for PTSD over and above parental PTSD. Thus, at least some childhood trauma dimensions appear to be independently significant factors in explaining risk for PTSD. Different types of childhood trauma may interact differently with other risk factors. For example, emotional abuse is related to parental PTSD and is an independent risk factor for PTSD in its own right, whereas early sexual abuse appears to be an independent risk factor for PTSD that is not associated with the risk factor of parental PTSD.

In the present study, we also performed very preliminary analyses of 24-hr urinary cortisol excretion in relation to CTQ dimensions. When a simple analysis was conducted, only the dimension of emotional abuse was associated with cortisol. This analysis clarified that emotional abuse was associated with low cortisol levels in the presence of PTSD, depressive disorder, or no disorder. In fact, subjects in this study only had low cortisol if they had emotional abuse. Particularly informative is the observation that PTSD without emotional abuse was not related to low cortisol in this sample. Furthermore, subjects with depressive disorder tended to show elevated cortisol levels, unless such subjects had emotional abuse, in which case cortisol levels were also low. These data suggest that emotional abuse is associated with cortisol independent of that associated with PTSD or depression.

When more complex analyses were performed for the other CTQ dimensions, there was a significant main effect of sexual abuse on cortisol. Interestingly, however, the relationship observed with sexual abuse was in the opposite direction to that observed with emotional abuse and only emerged once both presence and absence of PTSD and depressive disorder were considered. Although subjects with both PTSD and sexual abuse tended to have lower cortisol levels, cortisol levels were elevated in subjects with sexual abuse only. Subjects with sexual abuse and depression tended to have elevated cortisol levels, whereas subjects with depressive disorder alone were not appreciably different from any other group. Thus, although in the current sample there was some evidence that sexual abuse was associated with elevations in cortisol, this was only in the context of interactions with concurrent psychiatric diagnoses.

We have recently speculated that cortisol levels may be related to risk for PTSD, rather than to the pathophysiologic expression of this disorder per se (Yehuda, 1999). There are several lines of evidence supporting this contention. Cortisol levels, as reflected by 24-hr urinary cortisol, appear to be low in PTSD regardless of the severity and chronicity of the disorder (Mason, Giller, Kosten, Ostroff, & Podd, 1986; Yehuda, Boisoneau, Mason, & Giller, 1993; Yehuda, Southwick, Nussbaum, Wahby, Giller, & Mason, 1990). Furthermore, a series of studies have observed lower cortisol levels in the immediate aftermath of trauma in individuals at increased risk for the development of PTSD. Low cortisol levels immediately following a motor vehicle accident predicted the development of PTSD in accident victims presenting to an emergency room (Yehuda, McFarlane, & Shalev, 1998).

Low plasma cortisol levels obtained within hours of a rape were also found to be related to the risk factor of prior assault in a sample of consecutively admitted rape victims (Resnick, Yehuda, Pitman, & Foy, 1995). However, perhaps the most compelling evidence comes from the observation that low mean 24-hr urinary cortisol in adult children of Holocaust survivors is specifically associated with the risk factor of parental PTSD (Yehuda et al., 2000). The current study replicated this finding in an overlapping cohort. These studies raise the possibility that low ambient cortisol levels represent an index of risk and may contribute to the secondary biological alterations that ultimately lead to the development of PTSD.

Several other lines of evidence also support the idea that basal levels of cortisol represent more stable characteristics related to trait characteristics (e.g., "risk"). First, elevated cortisol levels have been found in first-degree relatives of depressed patients and are thought to be a trait measure related to risk for this disorder (Holsboer, Lauer, Schreiber, & Krieg, 1995). High cortisol levels predicted the occurrence of major depressive disorder in populations at increased risk for major depressive disorder based on stress exposure (Ockenfels, Porter, Smyth, Kirschbaum, Hellhammer, & Stone, 1995) and family history (Holsboer et al., 1995; Lupien, King, Meaney, & McEwen, 2000). Cortisol level increases were found to be stable when assessed in high-risk probands more than 4 years later (Modell, Lauer, Schreiber, Huber, Krieg, & Holsboer, 1998). Cortisol levels were also found to be relatively stable in normal participants who collected urine samples for 21 consecutive days (Cummins & Gevirtz, 1993). Salivary cortisol levels were stable in 30 monozygotic twin pairs tested on two separate occasions (Young, Aggen, Prescott, & Kendler, 2000). This study concluded that around 40–45% of the total variance in basal cortisol was shared by monozygotic twins. Finally, and of direct relevance to the current finding, is the observation of a correlation between cortisol levels in young offspring and mother's socioeconomic status and depressive state (Lupien et al., 2000). These findings provide support from the literature for our conclusion that cortisol levels may be related to more enduring characteristics, such as the effects of emotional abuse. In the current study, four-way ANOVA demonstrated a main effect of emotional abuse on cortisol even when parental trauma exposure and parental PTSD was taken into account, in addition to PTSD and depression in subjects. Thus, although preliminary, the biological results support the clinical observation that emotional abuse is an independent risk factor for PTSD.

The present study considers the relationship among different risk factors for PTSD. The identification of such risk factors and their interactions remains one of the major frontiers in the study of traumatology. When risk factors for PTSD are studied in isolation, important interactions among such variables cannot be evaluated. The simultaneous examination of multiple risk factors allows us to develop hypotheses about interrelationships among correlates of PTSD with respect to vulnerability, exposure, and illness. As such, it is critical to understanding the pathophysiology of PTSD. The addition of biologic measures to such studies provides a further opportunity for developing a coherent model for PTSD, in which preexisting alterations are considered in conjunction with both responses to trauma exposure and the pathophysiology of the disorder. The current study presents evidence that early developmental experiences constitute important risk factors for PTSD and may set the stage for biologic alterations that are compatible with the subsequent development of PTSD.

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