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Are Children of Holocaust Survivors Less Well-Adapted? A Meta-Analytic Investigation of Secondary Traumatization

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H. Keilson (1979) coined the term "sequential traumatization" for the accumulation of traumatic stresses confronting the Holocaust survivors before, during, and after the war. A central question is whether survivors were able to raise their children without transmitting the traumas of their past. Through a series of meta-analyses on 32 samples involving 4,418 participants, we tested the hypothesis of secondary traumatization in Holocaust survivor families. In the set of adequately designed nonclinical studies, no evidence for the influence of the parents' traumatic Holocaust experiences on their children was found. Secondary traumatization emerged only in studies on clinical participants, who were stressed for other reasons. A stress-diathesis model is used to interpret the absence of secondary traumatization in nonclinical offspring of Holocaust survivors.

KEY WORDS: Holocaust; trauma; stress-diathesis; secondary traumatization; second generation.

More than half a century ago, about 6 million Jews were murdered in an unprecedented genocide (Benz, 2001). About one and a half million children below 15 years of age were killed (Keren, 2001). Although Hitler and his followers almost reached the "Final Solution," many Jewish children and adults managed to survive World War II in hiding, on continuous escape with no shelter, or they went through the ordeals of ghettos, concentration camps, and death camps alive. How did the survivors adapt to postwar life, and how did they deal with the traumas they experienced during the war? After the war, many survivors were confronted with the loss of their parents and other family members, and they were not always made to feel welcome in their home countries (Bossenbroek, 2001; Gay, Fuchs, & Blittner, 1974). Keilson (1979) coined the term "sequential traumatization" for the accumulation of traumatic stresses before, during, and after the war. Nevertheless, many survivors built up new lives and families, and a central question is whether they were able to raise their children without transmitting the traumas of their past, providing the next generation with new chances to develop in a balanced way (Bar-On et al., 1998).

This issue is not only important for the study of the Holocaust. During the past century, unfortunately, genocide has not been restricted to World War II. In the *International handbook of multigenerational legacies of trauma* (Danieli, 1998), genocides in Armenia, Cambodia, former Yugoslavia, Rwanda, and Nigeria are mentioned, and these constitute only a selection. The Holocaust was a unique and barbaric genocide because of its scale, its almost industrial design, and its uselessness in political, economic, or military respects (Lacqueur, 2001). Nevertheless, we may learn from the Holocaust survivors and their children about the long-term and intergenerational effects of traumatic experiences, and develop insights into the fate of survivors of more recent and future genocidal catastrophes.

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After the Vietnam War the concept of posttraumatic stress disorder (PTSD) was formally introduced to describe some of the psychological consequences of exposure to or witnessing of atrocities such as torture and killing. According to *DSM-IV* (American Psychiatric Association, 1994) PTSD is defined as the persistent reexperiencing of a traumatic event, the persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness, and persistent symptoms of increased arousal such as hypervigilance or irritability. The symptoms are chronic, and the disturbance causes impairment in social, occupational, or other important functioning (pp. 428–429).

It is exactly this impairment in the social domain, i.e., family life, that stimulated researchers such as Danieli (1998) and others to suggest that intergenerational transmission of traumatic experiences would exist. Secondary traumatization was supposed to be caused by the disturbances of the survivors who as parents would be unable to protect their children from the aftermath of the Holocaust (Rosenheck & Nathan, 1985). Paradoxically, in their effort to separate their past from the present family life, the survivors would help create a "conspiracy of silence" (Danieli, 1998) that indirectly traumatized their offspring. Is sufficient evidence available for secondary traumatization to exist in second-generation Holocaust survivors, or is it restricted to selected clinical cases, as (important) exceptions to the rule (Suedfeld, 2000)?

Studies on the psychological consequences of the Holocaust for the first generation, the survivors, do not show unequivocal outcomes. Some researchers demonstrate that most Holocaust survivors were able to establish a productive and successful existence, as well as a happy family life. In their view Holocaust survivors do not seem to be seriously hampered by psychological problems (Krell, 1993; Leon, Butcher, Kleinman, Goldberg, & Almagor, 1981; Suedfeld, 2000). Other researchers mention profound disturbances such as chronic anxieties and depression, and discordant family functioning (Danieli, 1998; Niederland, 1981). In particular, clinically oriented researchers report many psychological disturbances in Holocaust survivors, but they sometimes rely on impressionistic case-studies or selected samples of survivors who had asked for psychotherapy or were involved in treatment.

Holocaust survivors may also function normally in daily life and under regular circumstances, and perhaps even under mild stress, but at the same time they may be vulnerable in more extreme stressful conditions. Studies on the psychological consequences of recent wars in Israel illustrate this potential vulnerability. The Gulf War, for example, with its missiles attacks on Israel, including threat of missiles filled with biological or chemical weapons, provoked more intense emotional responses in Holocaust survivors compared to other groups (Solomon & Prager, 1992). These findings seem to concur with the outcome of studies on Vietnam veterans (Goldstein, Van Kammen, Shelly, Miller, & Van Kammen, 1987), Korean veterans (Thienes-Hontos, Watson, & Kucala, 1982), and prisoners of war in the Japanese army during World War II (Goldstein et al., 1987).

Clinical studies have reported disturbed family relations of Holocaust survivors, who would be overprotective of their offspring. The children would develop anxious and ambivalent bonds with their parents, and they would be hampered in their search for autonomy (Barocas & Barocas, 1973, 1979, 1980). Secondary traumatization has also been demonstrated in a large-scale study on children of Vietnam veterans (Rosenheck & Fontana, 1998). Twice as many children with serious behavior problems were found in families in which the fathers had experienced concrete and violent actions and atrocities compared to a group of Vietnam veterans without direct exposure to violence. In contrast with some studies on secondary traumatization of the Holocaust in the clinical domain (Solomon, Kotler, & Mikulincer, 1988; Zilberfein, 1996), several epidemiological investigations did not find any negative effects on the psychological health of children of Holocaust survivors (Leon et al., 1981; Sigal & Weinfeld, 1989).

Appearances may be deceiving, however. In a study on the consequences of the Lebanon war for Israeli soldiers, Solomon et al. (1988) found that adult children of Holocaust survivors were more vulnerable to develop PTSD in combat. Three years after the Lebanon war, soldiers who grew up in families with one or two Holocaust survivors as parents showed significantly more indicators of PTSD than soldiers without this family background. A similar incidence of combat-related psychological disturbances was observed in both groups, but the children of Holocaust survivors appeared to show more signs of PTSD for a longer period of time. In a small sample of second-generation Holocaust survivors, Yehuda et al. (1998) found lower levels of the stress hormone cortisol compared to a matched group of participants without a Holocaust background. Lower levels of cortisol may indicate hypersensitivity to stress as a consequence of previous trauma.

Thus, studies on intergenerational transmission of traumatic experiences show inconsistent outcomes (Bar-On et al., 1998). Secondary traumatization, therefore, still is a concept in need of further empirical validation. It refers to traumatic effects of events that did not take place in the lives of the second-generation participants themselves, but in those of their parents who may or may not have communicated their experiences—in a verbal

or nonverbal way (Schuengel, Bakermans-Kranenburg, & Van IJzendoorn, 1999). If the parents did communicate about their Holocaust experiences, these interactions may or may not have impacted their children in a traumatizing way. Therefore, several assumptions are implied in the concept of secondary traumatization that renders its existence less plausible than the well-established concept of (primary) traumatization. In a research domain with a new concept and with conflicting results, the method of narrative review may be insufficient to create a coherent picture (Cooper & Hedges, 1994). Counting studies with supporting versus falsifying evidence is an inadequate approach to reconciling diverging study outcomes, as it does not take into account the highly heterogeneous quality and size of the studies (Kellermann, 2001). A quantitative analysis and synthesis of the secondary traumatization literature is needed to come to a more definite conclusion. Through meta-analysis, it is possible to compute the average effect size across studies, and to explain differences in outcome between studies on the basis of study characteristics (Mullen, 1989; Rosenthal, 1991, 1995).

Through a series of meta-analyses on 32 samples involving 4,418 participants, we tested the following questions and hypotheses. First, we tested the hypothesis that secondary traumatization in Holocaust survivor families exist. Secondary traumatization is conceptualized in a more stringent way: display of symptomatology such as PTSD symptoms, and also in a broader sense: less mental health in terms of social adaptation or personality development, and psychopathological symptomatology. Second, we investigated whether diverging study results may be explained by the quality of the research design. In particular, we test whether the recruitment of the sample is an important factor. In several Holocaust studies, investigators rely on convenience samples and recruit their respondents through advertisements or through Holocaust survivor organizations or meetings. This practice might lead to stronger Holocaust effects as it may involve participants who already have more problems and are more aware of the Holocaust influences to be expected. Third, we tested whether Holocaust studies on clinical samples find larger intergenerational transmission of trauma than studies conducted in nonclinical samples. This may indicate whether potential vulnerability in stressful circumstances exists in second-generation Holocaust survivors. Fourth, we test whether different effects are found when both parents were Holocaust survivors or only one of the parents. Lastly, we explored the differences in secondary traumatization between groups raised in Israel versus groups growing up in Western European or North American countries, addressing the issue of possible protective mechanisms specifically present in Israeli society. The survivors may have felt supported by the emergence of Israel, by knowing that anti-Semitism was unlikely to take place within a predominantly Jewish community, and by the common goal of building up a new society almost from scratch. In Israel they were in the company of many thousands of fellow-survivors who had lived through similar traumatic experiences, and who may be the only ones to really understand their suffering (Wiesel, 1960/1986). Survivors may have felt more often heard and better understood in a country with many fellow-survivors.

Methods

Data Collection

Pertinent studies were collected systematically, using three different search strategies (Mullen, 1989; Rosenthal, 1991). First, PsycLit, Medline, and PILOTS (a comprehensive database on posttraumatic stress) were searched with key-words "holocaust," "second-generation," and "survivors." Second, the references of the collected papers, books, and book chapters were searched for relevant Holocaust studies. Third, some recent narrative reviews were used as a source for relevant papers, in particular the exhaustive Kellermann (2001) review.

Holocaust survivors spent the war in Nazi-occupied Europe, either in concentration/labor camps, or in various hideaway shelters, being adopted by gentile families, or a combination of escape and survival strategies. More often than not, they lost parents and other family members. "Second-generation Holocaust survivors" has become an accepted term to refer to children of Holocaust survivors (Solomon, 1998). Our selection criteria were rather broad, in order to include as many Holocaust studies as possible, regardless of research design qualities. The most important criterion was that the study should contain at least one comparison group, and that it presented data to derive the pertinent meta-analytic statistics from. The idea was to test the influence of design features empirically, and not to exclude any quantitative studies on a priori grounds (Rosenthal, 1995). Case studies were excluded, as well as unpublished studies or studies that were only reported at meetings or conferences.

We collected second-generation Holocaust studies that aimed at comparing the adjustment of children of Holocaust survivors with that of a comparison group. Thus, we included only quasi-experimental investigations with a more or less careful comparison between children of Holocaust survivors and their peers with parents who were not Holocaust survivors. Purely descriptive studies of samples with only offspring of Holocaust survivors (e.g., Danieli, 1982; Levine, 1982) were excluded. Adjustment was

defined broadly, and involved indicators of posttraumatic stress, other symptomatology, and general mental health. As a result of our search, 112 papers were identified, of which 81 were eliminated because they did not meet the set of criteria for the meta-analysis. For an overview of the included studies, see Table 1.

Coding System

A coding system was used to rate every Holocaust study on design, sample, and measurement characteristics. We coded sample size (children of Holocaust survivors and comparison group in the study) and recruitment as design characteristics. Studies were coded as nonselect when participants were randomly sampled, e.g., from several neighborhoods (Brom, Kfir, & Dasberg, 2001), or a population registry (Sagi et al., 1999; Schwartz, Dohrenwend, & Levav, 1994), or when the entire Jewish population of a certain country was involved (Major, 1996). They were coded as select when samples were recruited through, e.g., Holocaust survivor meetings, personal contact, or advertisements (e.g., Felsen & Erlich, 1990; Halik, Rosenthal, & Pattison, 1990). We coded whether the sample was clinical (e.g., Baider et al., 2000; Solomon et al., 1988) or

Table 1. Studies on Second-Generation Holocaust Effects

Author	*N*	Recruitment	Sample	Gender	Country of Residence	Outcome	Effect Size	95% CI
Aleksandrowicz, 1973	34	select	clinical	mixed	Israel	symptomatology	0.00	(−0.79–0.79)
Baider et al., 2000	208	nonselect	clinical	female	Israel	stress	1.28	(0.98–1.58)
						symptomatology	1.64	(1.32–1.95)
Baron, Eisman, Scuello, Veyzer, & Lieberman, 1996	201	select	normal	mixed	USA	stress	0.30	(−0.01–0.61)
Baron, Reznikoff, & Glenwick, 1993	350	select	normal	mixed	USA	stress	0.00	(−0.23–0.23)
						symptomatology	0.00	(−0.23–0.23)
						mental health	0.00	(−0.23–0.23)
Brom et al., 2001	62	nonselect	normal	female	Israel	symptomatology	−0.23	(−0.74–0.28)
De Graaf, 1975	40	select	clinical	mixed	Israel	symptomatology	0.31	(−0.34–0.97)
Eland, van der Velden, Kleber, & Steinmetz, 1990; Bar-On et al., 1998	60	nonselect	normal	mixed	Europe	symptomatology	0.41	(−0.11–0.93)
						mental health	0.22	(−0.30–0.73)
Felsen & Erlich, 1990	62	select	normal	mixed	Israel	mental health	0.17	(−0.34–0.68)
Gay et al., 1974	46	select	clinical	mixed	Israel	symptomatology	−0.01	(−0.61–0.58)
Gay & Shulman, 1978	49	nonselect	normal	mixed	Israel	mental health	0.00	(−0.58–0.58)
Halik et al., 1990	38	select	normal	female	Australia	mental health	1.13	(0.42–1.84)
Keinan et al., 1988	93	nonselect	normal	mixed	Israel	symptomatology	0.00	(−0.41–0.41)
						mental health	0.08	(−0.34–0.49)
Leon et al., 1981	57	select	normal	mixed	USA	symptomatology	0.00	(−0.59–0.59)
						mental health	0.00	(−0.59–0.59)
Lichtman, 1984	107	select	normal	mixed	Israel	symptomatology	0.00	(−0.39–0.39)
Magids, 1998	100	select	normal	mixed	USA	symptomatology	−0.07	(−0.47–0.32)
Major, 1996	56	nonselect	normal	mixed	Europ	symptomatology	0.43	(−0.14–1.01)
Nadler, Kav-Venaki, & Gleitman, 1985	38	select	normal	mixed	Israel	mental health	2.04	(1.23–2.85)
Rieck, 1994	823	nonselect	normal	mixed	Israel	symptomatology	0.02	(−0.13–0.16)
Rose & Garske, 1987	40	select	normal	mixed	USA	symptomatology	0.00	(−0.64–0.64)
Rubenstein et al., 1989	52	select	normal	mixed	USA	symptomatology	0.46	(−0.21–1.12)
Sagi-Schwartz et al., 2003	98	nonselect	normal	female	Israel	stress	0.00	(−0.40–0.40)
						mental health	0.00	(−0.40–0.40)
Schwartz et al., 1994 (I)	334	nonselect	normal	male	Israel	stress	−0.13	(−0.38–0.13)
Schwartz et al., 1994 (II)	513	nonselect	normal	mixed	Israel	stress	−0.19	(−0.39–0.02)
Sigal, Silver, Rakoff, & Ellin, 1973	67	select	clinical	mixed	USA	mental health	0.48	(−0.02–0.98)
Sigal & Weinfeld, 1989	318	nonselect	normal	mixed	USA	stress	0.00	(−0.26–0.26)
	(188)					symptomatology	−0.16	(−0.42–0.10)
						mental health	−0.00	(−0.32–0.31)
Solomon et al., 1988	96	nonselect	clinical	male	Israel	stress	0.52	(0.11–0.93)
Sorscher & Cohen, 1997	78	select	normal	mixed	USA	stress	0.68	(0.22–1.14)
Weiss et al., 1986	50	select	normal	mixed	USA	mental health	−0.18	(−0.75–0.39)
Yehuda et al., 1998	23	select	normal	mixed	USA	stress	1.15	(0.21–2.08)
Yehuda, Schmeidler, Wainberg, Binder-Brynes, & Duvdevani, 1998	108	select	normal	mixed	USA	stress	0.37	(−0.02–0.76)
						symptomatology	0.61	(0.21–1.00)
Zilberfein, 1996	76	select	clinical	mixed	USA	symptomatology	0.76	(0.28–1.23)
	(44)					mental health	0.60	(−0.02–1.22)
Zlotogorski, 1985	141	select	normal	mixed	USA	symptomatology	−0.04	(−0.37–0.30)
	(139)					mental health	0.16	(−0.18–0.49)

nonclinical; what the current country of residence was (Israel, USA, Europe, or Australia); and the percentage of participants with both parents (rather than only one of them) being Holocaust survivors. We also coded gender of the samples (male, female, or mixed). However, one of the subsets proved to be too small (only two samples were all-male), therefore, gender could not be included as a moderator in the meta-analysis. Lastly, we coded the type of outcome: general mental health or adaptation, posttraumatic stress symptoms, and psychopathological symptomatology. We defined general mental health as general social adaptation or personality development. Mental health was indexed by standard measures such as the Brief Mental Health Index (Gunderson & Arthur, 1969; e.g., in Weiss, O'Connell, & Siiter, 1986) and the Tennessee Self-Concept Scale (Fitts, 1965), measuring positive self-regard (e.g., in Felsen & Erlich, 1990). Posttraumatic stress symptoms, such as reexperiencing of trauma, numbing of responsiveness, and reduced involvement with the external world was indicated by, e.g., the Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979; e.g., in Schwartz et al., 1994), the Endler Multidimensional Anxiety Scales (EMAS; Endler, Edwards, & Vitelli, 1991, e.g., in Sagi et al., 1999), or Solomon et al.'s PTSD inventory (Solomon et al., 1988). Psychopathological symptomatology included all symptoms of malfunctioning and maladaptation, anxieties, or depression, excluding posttraumatic stress symptoms. Indicators were the State Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970; e.g., in Keinan, Mikulincer, & Rybnicki, 1988; Zilberfein, 1996), the Minnesota Multiphasic Personality Inventory (MMPI; e.g., in Leon et al., 1981; Lichtman, 1984), and Beck's Depression Inventory (BDI; Beck et al., 1961; e.g., in Rose & Garske, 1987). These were all standard and valid instruments, commonly used for the assessment of mental health, posttraumatic stress, and maladaptation. The intercoder reliability of the coding system was established on 12 studies. Reliabilities ranged from .86 to 1.00 ($M = .93$).

Data Analysis

Because the studies included in this series of meta-analyses reported various statistics, the outcomes of all studies were recomputed with Mullen's Advanced BASIC Meta-Analysis program (Mullen, 1989), and transformed into Cohen's *d* (the standardized difference in means between the children of Holocaust survivors and comparisons). In several cases we had to compute the effect sizes on the basis of means and standard deviations provided in the study report. When more than one outcome was reported (e.g., the Intrusion and Avoidance subscales of the Impact of Event Scale, both assessing stress, in Baider et al., 2000), they were meta-analytically combined into one effect size for posttraumatic stress, that is Cohen's *d*. Moreover, an overall effect size for General Adjustment based on available indicators for mental health, stress, and/or symptomatology was computed for each study in order to avoid counting a study or participant more than once. When one of the outcomes was reported for a subsample of the study, the *n* for this outcome has been included in brackets as in Table 1. The resulting set of effect sizes were inserted into Borenstein, Rothstein, and Cohen's Comprehensive Meta-Analysis (CMA) program (Borenstein, Rothstein, & Cohen, 2000) that computed fixed as well as random effect model parameters (see Tables 2 and 3). CMA also computed confidence intervals around the point estimate of an effect size. Because all studies proposed directed hypotheses predicting that the children of Holocaust survivors would be less

Table 2. Meta-Analytic Results of Second-Generation Holocaust Effects: Outcome Measures

	k	*N*	*d*	95% CI	*Q* Homogeneity	*Q* Comparison
General adjustment	32	4418	0.28***	(0.12–0.45)	189.3***	4.50*
Select	20	1708	0.35***	(0.15–0.55)	67.9***	
Nonselect	12	2710	0.18	(−0.11–0.47)	117.0***	
Specific outcome						
Mental health	14	1333	0.30*	(0.06–0.53)	52.1***	3.50†
Select	9	845	0.46*	(0.09–0.83)	47.3***	
Nonselect	5	488	0.04	(−0.14–0.23)	0.6	
Symptomatology	19	2771	0.22†	(−0.02–0.47)	146.7***	2.06
Select	12	1151	0.16†	(−0.01–0.33)	19.2	
Nonselect	7	1620	0.30	(−0.24–0.84)	125.4***	
Stress	11	2327	0.33*	(0.03–0.62)	100.6***	0.59
Select	5	760	0.39*	(0.08–0.70)	13.4**	
Nonselect	6	1567	0.25	(−0.23–0.72)	86.6***	

Note. k = number of studies; d = effect size; CI = confidence interval of the effect size.
† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 3. Second-Generation Holocaust Effects: Moderators

	k	*N*	*d*	95% CI	Q
Sample					
Total group					63.5***[a]
Normal	25	3851	0.19**	(0.04–0.33)	85.7***
Clinical	7	567	0.53*	(0.07–0.99)	40.2***
Nonselect					94.2***[a]
Normal	10	2406	−0.04	(−0.13–0.05)	8.4
Clinical	2	304	1.00*	(0.08–1.92)	14.4***
Both parents survivors					
Total group					7.9*[a]
Unknown	9	835	0.09	(−0.09–0.27)	11.3
<75%	10	1130	0.09	(−0.03–0.21)	4.6
>75%	13	2453	0.57**	(0.22–0.92)	165.5***
Nonselect					0.4[a]
Unknown	4	536	0.13	(−0.20–0.46)	8.0*
<75%	4	296	0.09	(−0.14–0.32)	1.8
>75%	4	1878	0.28	(−0.35–0.92)	106.7***
Country of residence					
Total group					0.6[a]
Israel	15	2603	0.26†	(−0.04–0.55)	148.6***
NonIsrael	17	1815	0.29***	(0.13–0.46)	40.1***
Nonselect					0.3[a]
Israel	9	2276	0.17	(−0.19–0.54)	113.1***
NonIsrael	3	434	0.15	(−0.17–0.48)	3.6

Note. k = number of studies; d = effect size; CI = confidence interval of the effect size.
[a] Q for the comparison.
† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

well-adapted, we present here the 95% confidence boundaries (with *alpha* set at .05; see Tables 2 and 3).

Significance tests and moderator analyses in fixed effects models are based on the assumption that differences between studies leading to differences in effects are not random, and that, in principle, the set of study effect sizes is homogeneous at the population level. Significance testing is based on the total number of participants, but generalization is restricted to other participants that might have been included in the same studies of the meta-analysis (Rosenthal, 1995). Statistical inferences may be regarded as applying only to the specific set of studies at hand (Hedges, 1994). In random effects models, this assumption is not made (Hedges & Olkin, 1985), and they allow for the possibility that each separate study has its own population parameter. In random effects models, significance testing is based only on the total number of studies and generalization is to the population of studies from which the current set of studies was drawn (Rosenthal, 1995).

It has been argued that random effects models more adequately mirror the heterogeneity in behavioral studies, and use noninflated alpha levels when the requirement of homogeneity has not been met (Hunter & Schmidt, 2000). We decided to present the combined effect sizes and their confidence intervals in the context of random effects models (see Tables 2 and 3). The Q-statistics are presented to test the homogeneity of the specific set of effect sizes (a significant Q for homogeneity indicates that the set of outcomes is heterogeneous), and to test the significance of moderators (a significant Q for the comparison indicates that the difference between the combined effect sizes of the subsets of studies is significant; Borenstein et al., 2000; Mullen, 1989; Rosenthal, 1995). In our series of meta-analyses several data sets were heterogeneous. In those cases, the random effects model parameters (significance, confidence intervals) are somewhat more conservative than the fixed effects parameters, and the moderator tests (based on the fixed effects parameters) should be considered to be descriptive of the specific set of studies at hand (Rosenthal, 1995).

The current analyses included 31 papers. One report (Schwartz et al., 1994) presented more than one sample, and separate effect sizes were computed for each of the samples. In the 32 samples, data on 4,418 participants (children of Holocaust survivors and comparisons) were included. More than 2,000 (adult) children of surviving parents were included. Thirty-eight percent of the studies were nonselect, 22% of the studies involved clinical samples, and in 47% the respondents' current country of residence was Israel. The sample sizes ranged from 23 (Yehuda et al., 1998) to 823 (Rieck, 1994). For each of the 32 samples, the standardized difference between the Holocaust and comparison group was computed (Cohen's d; Mullen, 1989). For each sample, Fisher Z was computed as an equivalent to the correlation coefficient r (see Mullen, 1989). No outlying effect sizes were identified in the set of Holocaust studies on the basis of standardized z-values larger than 3.29 or smaller than −3.29 ($p < .001$; Tabachnick & Fidell, 2001).

Results

Does Secondary Traumatization Exist?

In the total set of 32 samples on 4,418 families, we found a significant difference in psychological well-being and adaptation between the second-generation Holocaust survivors and their comparisons. The size of the combined effect was a Cohen's d of .28 ($p < .001$), with a rather broad confidence interval: $.12 < d < .45$ (see Table 2). Based on the overall effect size for general adjustment in each sample, the second-generation Holocaust survivors did not adapt as well as the comparisons. This finding was replicated in the set of studies with assessments of general mental health ($k = 14$, $d = .30$, $p < .05$), with assessments of posttraumatic stress ($k = 11, d = .33, p < .05$), and to a lesser extent also in studies with measures of psychopathological symptomatology ($k = 19, d = .22, p < .10$).

Are Study Results Associated With the Type of Recruitment of Participants?

When the set of studies was divided in a subset with convenience samples and a subset with nonselect samples, we found diverging outcomes. The 20 convenience samples, including 1,708 participants, showed a rather large combined effect size of $d = .35$ ($p < .001$) for general adjustment. The 12 nonselect studies with a more adequate recruitment of participants showed no significant effect ($d = .18$, *ns*, $N = 2{,}710$). The difference between the two subsets was significant: $Q(1) = 4.50$, $p < .05$. Thus, studies with nonselected samples did not show less well-being or adaptation in second-generation Holocaust survivors than did the comparisons in these studies. Only studies with convenience samples showed the secondary traumatization effect. This finding was replicated for the measures of general mental health and posttraumatic stress symptoms, with significant effect sizes for the studies with select samples and nonsignificant effect sizes in the nonselect studies (see Table 2). For the other symptomatology index, the pattern was comparable, but at the $p < .10$ significance level (see Table 2). Thus, taken as a whole, secondary traumatization as indicated by measures of posttraumatic stress symptoms, general mental health, and psychopathological symptomatology may be an artifact of the design of Holocaust studies.

Is Secondary Traumatization Stronger in Clinical Samples?

In the total set of 32 studies we found a strong effect for second-generation Holocaust survivors and their comparisons from the clinical groups: $d = .53$ ($p < .05$; $k = 7$, $N = 567$), whereas in the nonclinical groups, the combined effect size was considerably smaller ($d = .19$, $k = 25$, $N = 3,851$; see Table 3). The difference was significant, $Q(1) = 63.46$, $p < .001$. This finding was replicated in the subset of 12 nonselect studies, $Q(1) = 94.18$, $p < .001$ (see Table 3). Second-generation Holocaust survivors were less well-adapted than comparisons only in clinical groups, that is, in groups that were also stressed by the occurrence of serious psychological or physical illnesses such as combat disorder or breast cancer.

Is Secondary Traumatization Stronger When Both Parents Are Holocaust Survivors?

Nine studies did not report explicitly the number of participants who were the offspring of two Holocaust survivors. In the 13 samples with more than 75% of the participants being the offspring of two Holocaust survivors, the combined effect size was rather large, $d = .57$ ($p < .01$; $N = 2{,}453$). In studies with less than 75% participants having two Holocaust survivors as parents, the combined effect size was not significant, $d = .09$ ($k = 10$, $N = 1{,}130$). The difference was significant, $Q(2) = 7.91$, $p = .02$. This finding was not replicated in the subset of 12 nonselect studies, $Q(2) = 0.38$, *ns* (see Table 3). In sum, the occurrence of secondary traumatization was more apparent in offspring of two (rather than only one) Holocaust survivors in the total set of studies, but this result could not be replicated in the subset of nonselect studies.

Is the Second-Generation Holocaust Survivors Better Adjusted When Raised in Israel?

In the total set of studies we did not find a significant difference in adjustment between offspring of Holocaust survivors growing up in Israel ($d = .26$, $k = 15$, $N = 2{,}603$) and second-generation survivors raised in other countries ($d = .29$, $k = 17$, $N = 1{,}815$; see Table 3). In the nonselect samples, we did not find a significant difference in adjustment either, $Q(1) = 0.25$, *ns* (Table 3). In the Israeli-samples, the combined effect size was $d = .17$, whereas for non-Israeli samples, the effect size was $d = .15$. Thus, living in Israel did not make a difference for the occurrence of secondary traumatization.

Discussion

In the total set of 32 samples, involving thousands of participants, some evidence for secondary traumatization was found. However, in the subset of adequately designed studies with nonselect samples, this Holocaust effect was absent. Only in a few studies on second-generation *clinical* participants, who were also stressed by psychological or physical adversities unrelated to the Holocaust experiences of their parents, such as combat stress reaction or breast cancer, we found more posttraumatic stress symptoms than in non-Holocaust comparisons with similar adversities. For normal samples of second-generation Holocaust survivors, we were not able to document the existence of secondary traumatization in our comprehensive set of meta-analyses. It is possible that the Holocaust survivors never communicated about their traumatic experiences. However, in light of empirical evidence documenting "secondary traumatization" transmitted in nonverbal ways to children by mothers who suffered unresolved losses of close family members (Schuengel et al., 1999), we are inclined to interpret our meta-analytic result also as a sign of resilience on the part of the survivors who as parents seem to have managed to protect their children from being affected by the Holocaust.

What might be a plausible explanation for our meta-analytic findings? In a biopsychological stress-diathesis model of PTSD, Paris (2000) elaborated three important protective or risk factors determining the intensity and duration of posttraumatic stress. The first factor concerns the repeated exposure to traumatic events. The accumulation of stressors, both recent traumatic events as well as earlier adversities would determine whether the threshold for the appearance of overt posttraumatic stress symptoms would be lowered. The second factor is the presence of a genetic predisposition for PTSD. As Paris (2000) argues, although the risk of "blaming the victims" may be present, it is in line with a stress-diathesis model that heritable predispositions are necessary conditions for PTSD. Genetic predispositions such as high levels of neuroticism may elevate the sensitivity as well as the chance of exposure to stresses. The third factor is lack of social support in coping with the traumatic experiences. This is a social predisposition that lowers the threshold at which symptoms develop, and it may also determine the specific form of the symptoms (Paris, 2000).

How can we interpret the absence of secondary traumatization in the nonclinical offspring of Holocaust survivors from this stress-diathesis perspective? First, we should note that the traumatic experiences of the survivors were not inflicted by their own parents or other attachment figures (Sagi-Schwartz et al., 2003; Steele, 1999). These experiences, instead, emerged from an almost anonymous, destructive process with bureaucratic characteristics (Bauman, 1989). The Holocaust, therefore, did not undermine the feelings of basic trust in their attachment figures, maybe enabling them to adequately fulfill their own role as trusted parents for their children. It should be noted that infant attachment disorganization has been shown to predict dissociative symptoms in adolescence (Carlson, 1998; Main & Morgan, 1996). Furthermore, many Holocaust survivors in hiding did not personally experience or directly witness atrocities, which might have alleviated their own traumatic distress (Rosenheck & Fontana, 1998). Lastly, most survivors had experienced several prewar years of quiet and satisfying family life, and were thus able to establish secure attachment relationships with their parents, spouses, or other attachment figures. The survivors might have had adequate models of parenting available when they became parents themselves. Their own children might, therefore, not have experienced indirectly or in a secondary way the kind of traumatization that would lead to dissociative symptoms or other psychiatric symptomatology (Carlson, 1998; Van IJzendoorn et al., 1999).

The second stress-diathesis factor is genetic predisposition. In a twin study on more than 4,000 mono- and dizygotic twins who fought in Vietnam, True et al. (1993) showed that almost 30% of the variance in PTSD may be genetically determined. We speculate that Holocaust survivors were not genetically biased to develop intense posttraumatic stress reactions, as these responses would have left them vulnerable in their struggle for survival (Schwartz et al., 1994). Several personal witnesses of the concentration- and death-camps document the necessary condition for survival of trying to keep thoughts and feelings coherent and meaningful despite the extremely distressing conditions (e.g., Frankl, 1984; Levi, 1947/1987; Vrba, 1963). Sufferers from PTSD may have had a smaller chance of surviving the extremely stressful circumstances of hiding or in camps. Survivors may have been protected against PTSD through their genes (Goldberg, True, Eisen, & Henderson, 1990) as well as through myriads of other personality, social, and chance factors. This genetic protection against PTSD might have been transmitted to the next generation. Children of Holocaust survivors, therefore, may not have been especially sensitive to potentially traumatic events or reports arising from their parents' efforts to deal with the Holocaust atrocities.

The third factor in the stress-diathesis model concerns the presence or absence of social support to cope with trauma afterwards. After World War II, the survivors were forced to find meaning (Frankl, 1984) in helping to build up a new society, not only in Europe and Northern America, but also—and maybe especially—in Israel. The newly founded State of Israel, to which many survivors emigrated after the war, served as a symbol of the ultimate failure of the "Final Solution" (Solomon, 1998). Ever since the establishment of Israel, various memorials have been erected to commemorate the victims of the Holocaust and to support survivors and their families in working through the traumatic memories of the past.

Evidence for the impact of social support on secondary traumatization would have been derived from better adaptation in second-generation Holocaust survivors who grew up in Israel. This is not the case, however, considering the absence of differences between the Israeli and the nonIsraeli samples. One of the reasons may be indicated by Segev's notion (Segev, 1992) that in Israel after World War II the myth of the heroic "Sabra" was predominant, and Holocaust survivors were often considered to have been the passive victims of the Nazi regime. So even in Israel, Holocaust survivors were expected to be silent about their sufferings. Another explanation for this outcome may be found in the strains and stresses of living in Israel over the past 50 years. Israeli Holocaust survivors were confronted with several wars, uprisings, and acts of terrorism. The ongoing conflicts as well as the absence of a secure peace in this region certainly contribute to a

continuous exposure of Israeli Holocaust survivors and their offspring to potentially traumatic stresses. This may have aggravated the original traumatic experiences. Israel as a symbol of survival may not sufficiently have materialized its potentially protective role. This speculative interpretation is supported by the finding that the two nonselect second-generation clinical samples going through considerable stresses (life-threatening illnesses or combat experiences), who showed secondary traumatization, were both Israeli. As Paris (2000) argues, the accumulation of stressful events may tip the balance and lead to posttraumatic stress symptoms in otherwise normally functioning persons.

Our series of meta-analyses show that children of Holocaust survivors were, in general, well-adapted. It might be the case that the intergenerational transmission of the trauma skips a generation, as is the case with some biologically inherited diseases—although in one study that included the third generation (Sagi-Schwartz et al., 2003), the youngest generation appeared to develop in a normal way. Additionally, secondary traumatization that was not observed at the time of the study, i.e., in early or middle adulthood of the second-generation, may only become visible later in life. Nevertheless, this study documents the absence of secondary traumatization in nonclinical samples of second-generation Holocaust survivors. Protective factors in the children or in their environment may have lessened the impact of their parents' trauma. But under conditions of extreme stress, latent vulnerability to maladaptive and prolonged posttraumatic responses may come to the surface, even in the children of Holocaust survivors.

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