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Secondary salutogenic effects in veterans whose parents were Holocaust survivors?

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ABSTRACT

Addressing the ongoing controversy over inter-generational transmission of trauma, we examined the impact of the Nazi Holocaust on PTSD course and co-morbid symptoms (e.g., depression, anxiety) among offspring of survivors following their own adversity in two longitudinal studies. Two samples of Israeli war veterans included Second Generation Holocaust (i.e., SGH) survivors and comparable veterans with no such family history (i.e., not-SGH). Study I: 1982 Lebanon War veterans ($N = 669$) were assessed 1, 3, and 20 years after the war. Study II: 1973 Yom Kippur War veterans ($N = 343$) were followed up 18, 30, and 35 years after the war. Results indicated that SGH endorsed higher PTSD and co-morbid symptoms criteria rates than not-SGH veterans in the initial post-war years but this pattern was reversed in the long-term, that is, lower rates were evident among SGH in later follow-ups. These findings suggest the development of a complex trauma reaction among offspring of trauma survivors. Possibly there is a transmission of positive trauma outcomes from one generation to the next rather than merely negative ones. Future studies are therefore warranted to re-evaluate the notion of inter-generational transmission of trauma and examine its components.

1. Introduction

Inter-generational transmission of trauma implies that the psychological effects of trauma spill over from one generation to the next. Second generation Holocaust (SGH) survivors, i.e., adult offspring of Holocaust survivors, have been the most widely-studied group in respect to trauma-transmission. While more than 500 studies have examined the psychological well-being of the SGH, they have yielded highly controversial results (e.g., see Kellermann, 2001; for review).

Initially, the manifestations of Holocaust trauma-transmission were conceived to be pathological. Early case studies and a few empirical investigations, although poorly designed (i.e., lack of control group), noted clinical symptoms (i.e., anxiety, depression) and impaired functioning among the SGH which resembled their parents condition (e.g., Barocas and Barocas, 1973; Sigal et al., 1973). In turn, extensive theoretical formulations have been put forward including, family predisposition for psychiatric illness (e.g., Yehuda et al., 2000), overly-silent parental communication style (e.g., Danieli, 1998), family enmeshment (e.g., Seifter-Abrams, 1999) etc.

In the last decade, the pathogenic inter-generational effects of the Holocaust have been questioned. A recent meta-analysis study (van IJzendoorn et al., 2003) comparing non-clinical samples of SGH and matched controls found no significant differences between the groups in lifetime prevalence of traumatic stress (e.g., Schwartz et al., 1994), psychopathological symptomatology (e.g., Sigal and Weinfeld, 1989) and general mental health (i.e., social adaptation and personality development) (e.g., Sagi-Schwartz et al., 2003). As might be expected, psychopathology was evident in clinical SGH samples, mostly representing individuals undergoing treatment. Thus, generally speaking, current empirical data underscores that a pathogenic transmission of Holocaust trauma is uncommon.

More interestingly, there is some evidence to suggest that the effects of trauma-transmission become visible only in the context of adversity. In a follow-up study of war veterans who had no indication of psychopathology prior to combat, Solomon et al. (1988) documented higher rates of PTSD and slower recovery in SGH compared to controls. Similarly, in their original study of women diagnosed with breast cancer, Baider et al. (2000) found higher posttraumatic symptoms and related distress (i.e., depression, anxiety, etc.) among SGH. Thus, SGH are likely to respond with heightened vulnerability to a life-threatening stressor. In accord, biological abnormalities have been documented in SGHs' stress

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response, indicating dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis (Yehuda et al., 2000).

Along with this vulnerability notion, is the less common view that SG would be relatively resilient in the wake of a traumatic event. To have lived through massive and prolonged trauma, the parents may have possessed or acquired resilient qualities (e.g., assertiveness, flexibility etc.) (Helmreich, 1992). This is in line with the growing evidence that individuals may flourish and experience positive psychological changes following trauma even of extreme nature (e.g., Tedeschi and Calhoun, 2004). As some studies reported, Holocaust survivors endorsed relatively positive views of the world (e.g., Cohen et al., 2001), high self-esteem (e.g., Cassel and Suedfeld, 2006), and feelings of immunity to subsequent threat (Robinson et al., 1994).

There seems to be empirical grounds to propose that a positive inter-generational trauma transmission can occur (e.g., Dekel et al., under review), suggesting that SGH could be better equipped to cope with trauma than individuals whose parents were not exposed to the Holocaust. A well-documented moderating factor of the psychological implications of trauma is time. Posttraumatic symptoms fluctuate rather than follow a stable trajectory (e.g., Port et al., 2001). Interestingly, no study to the best of our knowledge examined SGs' response to trauma over a long time course; therefore, how they eventually adjust to trauma remains unknown.

The aim of the present study was to investigate the implications of the parents' traumatic exposure (Holocaust) in their offspring's long-term response to trauma. We examined two longitudinal studies of Israeli combat veterans: of the Lebanon War, over 20 years, and of the Yom Kippur War, over 35 years after the war. The main research questions were: (1) Do SGH report more or less posttraumatic distress than not-SGH? (2) Are the differences moderated by the time since exposure?

2. Methods

This investigation is part of two prospective studies on the psychological implications of war among Israeli male veterans.

2.1. Participants

Study I comprised a cohort of veterans who fought in the 1982 Lebanon War (see Solomon and Mikulincer, 2006; for details). In this study, 669, 389, and 422 participants, respectively, were assessed at 3 points in time: 1 (T1), 3 (T2), and 20 (T3) years after the war (i.e., 1983, 1985, and 2002).

Study II included a cohort of veterans who fought in the 1973 Yom Kippur War, among them individuals who were taken captive (see Dekel et al., 2012; for details). In this study, 343, 205, and 154 participants, respectively, were assessed at 3 points in time: 18 (T1), 30 (T2) and 35 (T3) years after the war (i.e., 1991, 2003, and 2008).

Participants from both cohorts in follow-up assessments did not differ from drop outs with regard to initial PTSD symptom level; military rank, age, and education. The study commenced after the Israel Defense Forces and Tel Aviv University committees on human research approved their design. Informed consent was obtained from all participants.

2.2. Measures

Posttraumatic symptoms were measured using the PTSD Inventory (Solomon et al., 1993). This is a self-report scale that initially included 13 items accord with the PTSD symptoms listed in DSM-III (APA, 1980) which was the standard of practice at the beginning of the study. To conform to the updated definition of PTSD, data were analyzed in accordance with the new DSM-IV (APA, 1994) symptom clusters. Participants were asked to indicate the frequency with which they experienced the described symptom anchored on the war experience within the past month on a 4-point scale ranging from 1 ("never") to 4 ("very often") with "2" indicating cutoff for symptom endorsement.

Internal consistency among the symptoms and symptom clusters (i.e., intrusion, avoidance, and arousal) was high in all measurements (total: Cronbach's α ranging between 0.87 and 0.96; subscales: Cronbach's α ranging between 0.90 and 0.93). Assessment of PTSD was conducted using DSM-IV symptoms criteria (at least 1 re-experiencing, 3 avoidance and 2 hyper-arousal symptoms) and also to conform with criterion F included a measure of disability as indicated by self-report of dysfunction at work or in social relations. The PTSD inventory has been previously used in veteran (e.g., Dekel et al., 2011) and clinical populations (e.g., Defrin et al., 2008). It was found to have good psychometric properties and high concurrent validity when compared with clinical diagnoses (Solomon et al., 1993).

Co-morbid symptoms which are noted to occur following trauma were assessed using the widely used self-report checklist-90 (SCL-90, Derogatis, 1977) targeting 90 symptoms and their symptoms cluster. Participants are asked to indicate how frequently they experienced each symptom during the last 2 weeks on a 5-point distress scale. In the present study, symptom clusters of depression, anxiety, paranoid ideation, obsessive-compulsive, somatization, and hostility were assessed. Based on SCL cutoff norms for psychiatric outpatients (Derogatis, 1977) mean scores equal to or above 0.73 at each subscale were considered as an indication for endorsement of clinical symptoms, in line with previous studies (e.g., Ginzburg et al., 2010). The subscales have high concurrent and convergent validity with similar scales in established psychiatric measurements (Dinning and Evans, 1977), such as Minnesota Multiphasic Personality Inventory (MMPI) (e.g., Derogatis et al., 1976) and Beck Anxiety Inventory (BAI) (Steer et al., 1993); and they display high empirical agreement across various samples (Derogatis et al., 1976). While not customized for diagnostic purpose, sensitivity and specificity of the subscales are quite similar to those obtained by comprehensive diagnostic interviews (e.g., Peveler and Fairburn, 1990). The SCL-90 has shown good test-retest reliability (e.g., Horowitz et al., 1988) and in this study reliability values were high at all assessments (Cronbach's α for subscales ranging between 0.83 and 0.90).

3. Results

3.1. Study I – 1982 Lebanon War veterans

In 1983 (T1), 19% ($N = 127$) of the participants were SGH, i.e., at least one of their parents was a Holocaust survivor, while 81% ($N = 542$) were not. The groups did not significantly differ with respect to socio-demographic background (i.e., age, education, income) and combat exposure (i.e., battlefield stressors).

3.1.1. PTSD symptom prevalence

As noted two PTSD scores were computed: the first, corresponding to the symptom clusters criteria, and the second, including the functioning criterion. As presented in Table 1, SGH had 1.13 and 2.42, respectively, higher odds of meeting PTSD symptoms criteria than not-SGH, 1 and 3 years after the war. In contrast, 17 years later, PTSD symptoms odds were 0.69 lower among SGH than not-SGH. When F criterion was added, SGH had 1.13 and 1.77, respectively, higher odds of meeting PTSD symptoms plus F criteria at T1 and T2, yet they had as much as 0.17 lower odds than not-SGH at T3. In fact, only one (1.4%) subject among SGH in comparison to 25 subjects (7.5%) among not-SGH met symptom criteria. Thus, the pattern was reversed: in the first years after the

Table 1
PTSD and Co-Morbid symptoms criteria rates among Lebanon war veterans who were and were not offspring of Holocaust survivors.

	2nd generation		Not 2nd generation		Odds ratio	CI
	N (total)	%	*N* (total)	%		
PTSD						
1983	57 (125)	45.6	228 (534)	42.7	1.13	0.76–1.66
1985	31 (77)	40.3	68 (312)	21.8	2.42	1.43–4.1
2002	25 (76)	32.9	143 (345)	41.4	0.69	0.41–1.17
PTSD with criterion F						
1983	19 (125)	15.2	74 (529)	14	1.1	0.64–1.9
1985	2 (48)	4.2	6 (250)	2.4	1.77	0.35–9.03
2002	1 (73)	1.4	25 (332)	7.5	0.17	0.02–1.28
Depression						
1983	214 (541)	39.6	46 (127)	36.2	1.15	0.77–1.72
1985	122 (311)	39.2	32 (77)	41.5	0.9	0.54–1.50
2002	113 (345)	32.8	22 (76)	29	1.2	0.69–2.06
Anxiety						
1983	52 (127)	40.9	245 (541)	45.3	0.84	0.57–1.2
	41.6	140 (312)	44.9	0.87	0.53–1.45	
2002	22 (76)	28.9	107 (345)	31	0.91	0.53–1.56
Obsessive-compulsive						
1983	63 (127)	49.6	276 (541)	51	0.94	0.64–1.4
1985	40 (77)	51.9	149 (311)	47.9	1.18	0.71–1.94
2002	25 (76)	32.9	117 (345)	33.9	0.95	0.56–1.62
Somatization						
1983	45 (127)	35.4	214 (541)	39.6	0.84	0.56–1.25
1985	30 (77)	39	125 (312)	40.1	0.96	0.57–1.6
2002	15 (76)	19.7	107 (345)	31	0.55	0.3–1
Paranoid ideation						
1983	45 (127)	35.4	227 (541)	42	0.76	0.51–1.13
1985	33 (77)	42.9	115 (309)	37.2	1.27	0.76–2.1
2002	12 (76)	15.8	79 (345)	22.9	0.63	0.32–1.2
Hostility						
1983	52 (127)	40.9	241 (541)	44.5	0.86	0.58–1.28
1985	30 (77)	39	122 (310)	39.4	0.98	0.59–1.64
2002	19 (76)	25	101 (345)	29.3	0.81	0.46–1.42

Note. Analyses included participants with data at least at one measurement point.

Table 2
PTSD symptoms among Lebanon war veterans who were and were not offspring of Holocaust survivors.

PTSD symptoms		2nd generation		Not 2nd generation			*p*
		M	*SD*	*M*	*SD*	*t*	
Total	1983	4.74	3.68	4.74	3.99	−0.01	0.99
	1985	3.68	3.7	2.34	3.08	3.27	0.01
	2002	4.36	3.99	4.88	4.35	−0.96	0.34
Intrusion	1983	1.39	1.09	1.29	1.19	0.9	0.37
	1985	1.25	1.22	0.66	1.01	4.38	0.01
	2002	1.08	1.21	1.23	1.2	−1.02	0.31
Avoidance	1983	1.13	1.09	1.12	1.18	0.05	0.96
	1985	1.04	1.23	0.69	1.03	2.56	0.01
	2002	0.91	1.09	1.02	1.17	−0.77	0.43
Hyper-arousal	1983	1.95	1.6	2.04	1.69	−0.56	0.58
	1985	1.4	1.53	0.99	1.4	2.2	0.03
	2002	2.04	1.79	2.26	1.98	−0.9	0.37

Note. Analyses included participants with data at least at one measurement point.

war SGH had a heightened vulnerability but seem to be more resilient in the long-term.

To examine changes in the prevalence of PTSD symptoms criteria over time we ran a series of McNemar tests for each group separately. The variables were entered in chronological order: PTSD/no PTSD, at T1, T2 and T3. The group (SGH vs. not-SGH) was entered as the layered variable. PTSD symptom prevalence significantly decreased between T1 and T2 among both study ($p < .05$) and control group ($p < .001$): 37.2% of SGH and 59.7% of not-SGH were classified as "recovered" (i.e., endorsed PTSD at T1 but not at T2). However, while PTSD symptom prevalence continued to decrease between T2 and T3 among SGH (36.8% "recovered"), although the change was not significant ($p = .5$), the rates significantly increased ($p < .001$) among not-SGH (34.19% "delayed"). When F criterion was added, while PTSD symptom rates decreased between T1 and T2 in both groups (SGH: $p < .1$; not-SGH: $p < .001$), once again rates continued to decrease between T2 and T3 only among SGH, in fact none were classified with PTSD at T3, but not among not-SGH ($p = .51$) where 3.9% were classified as "delayed" (i.e., endorsed PTSD at T3 but not at T1 and T2).

3.1.2. Number of PTSD symptoms

Number of PTSD symptoms and symptoms cluster for the groups at the measurements are presented in Table 2. *T*-tests revealed that while SGH endorsed significantly more PTSD symptoms and symptom clusters than not-SGH at T2, no group differences were noted between the groups at T1 and T3.

3.1.3. Prevalence of co-morbid symptoms

We also compared the study groups in the prevalence of the following co-morbid symptoms which are noted to occur after trauma: depression, anxiety, obsessive-compulsive, somatization, paranoid ideation, and hostility (Table 1). While SGH had higher symptom odds for depression, obsessive-compulsive, and paranoid ideation than not-SGH three years after the war, their odds were lower 17 years later. Moreover, SGH had slightly to moderately lower symptoms odds across the measurements with respect of anxiety, somatization, and depression than not-SGH.

To examine changes in the prevalence of symptoms over time, we ran a series of McNemar tests for each group separately including only veterans who participated in all measurements (see Table 1). In both groups, symptoms prevalence was constant between T1 and T2 (*p* value ranging between 0.2–1), and tended to decrease between T2 and T3 with respect to all co-morbid symptoms (*p* value ranging between 0.06–0.14).

3.2. Study II – 1973 Yom Kippur war veterans

At T1 (1991), 22.4% ($N = 77$) of the participants were SGH while 77.6% ($N = 266$) were not-SGH. The groups did not differ significantly with respect to socio-demographic background (i.e., age, education, income) and combat exposure (i.e., battlefield stressors). Also, with respect to pre-war military experience and post-war negative life events no group differences were observed (see Dekel et al., 2012, under review; for details).

3.2.1. PTSD symptom prevalence

PTSD rates, odds ratios, and confidence intervals are presented in Table 3 presents. SGH had 0.64, 0.72, and 0.65, respectively, lower odds of meeting PTSD symptoms criteria than not-SGH at T1, T2, and T3. When criterion F was added, PTSD odds were 0.63, 0.39, and 0.48, respectively, lower among SGH than controls at the measurements. Thus, SGH veterans had a relatively low probability of endorsing PTSD across times and symptoms criteria.

To examine changes in the prevalence of PTSD over time, we ran a series of McNemar tests for each group separately (including only veterans who participated in all measurements). PTSD prevalence significantly increased between T1 and T2 among SGH ($p < .01$) and not-SGH ($p < .001$): 25.6% of SGH and 31.1% of not-SGH were classified as "delayed" (i.e., endorsed PTSD at T2 but not at T1). Also, PTSD rates between T2 and T3 were constant in both groups (SGH: $p = .99$; not-SGH: $p = .9$). When criterion F was added, PTSD prevalence among SGH did not change significantly between T1 and T2 and T2 and T3 ($p = .5$ and $p = .62$, respectively). Yet, among not-SGH, PTSD rates significantly increased ($p < .001$) between T1 and T2 (13.3% were classified as "delayed") and the rates tended to increase between T2 and T3 ($p = .1$).

Table 3
PTSD and Co-Morbid symptoms criteria rates among Yom-Kippur war veterans who were and were not offspring of Holocaust survivors.

	2nd generation		Not 2nd generation		Odds ratio	CI
	N (total)	%	*N* (total)	%		
PTSD						
1991	4 (77)	5.2	21 (266)	7.9	0.64	0.21–1.92
2003	12 (44)	27.3	55 (160)	34.4	0.72	0.34–1.5
2008	11 (34)	32.4	51 (120)	42.5	0.65	0.29–1.5
PTSD with criterion F						
1991	3 (77)	3.99	16 (256)	6	0.63	0.18–2.2
2003	3 (44)	6.82	25 (157)	15.9	0.39	0.11–1.35
2008	5 (33)	15.2	31 (114)	27.2	0.48	0.17–1.35
Depression						
1991	14 (77)	18.2	51 (264)	19.3	0.93	0.48–1.79
2003	15 (44)	34.1	75 (161)	46.6	0.59	0.29–1.12
2008	14 (34)	41.2	61 (119)	51.3	0.66	0.31–1.44
Anxiety						
1991	17 (77)	22.1	58 (264)	22	1	0.55–1.86
2003	11 (43)	25.6	67 (161)	41.6	0.48	0.23–1.02
2008	17 (34)	50	69 (119)	58	0.73	0.34–1.56
Obsessive compulsive						
1991	21 (77)	27.3	94 (264)	35.6	0.68	0.39–1.19
2003	15 (44)	34.1	90 (161)	55.9	0.41	0.2–0.82
2008	20 (34)	58.8	70 (119)	58.8	1	0.46–2.17
Somatization						
1991	12 (77)	15.6	51 (264)	19.3	0.77	0.39–1.53
2003	15 (44)	34.1	70 (161)	43.5	0.67	0.34–1.35
2008	11 (34)	32.4	47 (119)	39.5	0.73	0.33–1.64
Paranoid ideation						
1991	12 (77)	15.6	67 (264)	25.4	0.54	0.28–1.07
2003	13 (43)	30.2	70 (161)	43.5	0.56	0.27–1.16
2008	16 (34)	47.1	65 (119)	54.6	0.74	0.34–1.59
Hostility						
1991	12 (77)	15.6	51 (264)	19.3	0.77	0.39–1.53
2003	12 (44)	27.3	59 (161)	36.6	0.65	0.31–1.36
2008	17 (34)	50	57 (119)	47.9	1.09	0.51–2.33

Note. Analyses included participants with data at least at one measurement point.

3.2.2. Number of PTSD symptoms

Table 4 presents the average number of PTSD symptoms and symptom clusters for the study groups at the measurements. While *t*-tests revealed no group differences in PTSD symptoms count, there were significant differences in the number of symptoms according to the three PTSD clusters: SGH significantly endorsed fewer intrusion symptoms than those not-SGH at T1 and T2.

3.2.3. PTSD rates, Holocaust status, and type of exposure

As previously noted, the Yom-Kippur sample included a subgroup of individuals who were taken captive during the war, i.e., ex-prisoners of war (POWs). Consequently, this group of veterans was exposed to severe and prolonged trauma which bears a resemblance to the trauma of the Holocaust (i.e., starvation, torture).

Surprisingly, SGH ex-POWs also had lower odds of meeting PTSD criteria than not-SGH ex-POWs. The SG group had 0.84, 0.90 and 0.87, respectively, lower odds at T1, T2, and T3. When criterion F was added, PTSD odds were 0.93, 0.39 and 0.55, respectively, lower among SGH ex-POWs than controls. While 15% of SGH ex-POWs met PTSD criteria, as many as 31.1% of not-SGH ex-POWs met PTSD criteria.

Table 4
PTSD symptoms among Yom-Kippur war veterans who were and were not offspring of Holocaust survivors.

PTSD symptoms		2nd generation		Not 2nd generation			*p*
		M	*SD*	*M*	*SD*	*t*	
Total	1991	2.08	3.34	2.7	3.87	−1.29	0.2
	2003	4.82	5.34	6.18	5.76	−1.4	0.16
	2008	5.56	5.37	7.03	5.9	−1.29	0.19
Intrusion	1991	0.47	1.02	0.95	1.44	−2.75	0.01
	2003	1.05	1.67	1.68	1.93	−1.97	0.05
	2008	1.5	1.93	1.88	2.02	−0.99	0.33
Avoidance	1991	0.55	1.14	0.79	1.45	−1.34	0.18
	2003	1.68	2.22	2.26	2.43	−1.41	0.16
	2008	1.94	2.16	2.61	2.44	−1.44	0.15
Hyper-arousal	1991	1.06	1.73	0.97	1.54	0.48	0.63
	2003	2.09	2.03	2.25	2.04	−0.46	0.65
	2008	2.12	1.99	2.53	1.99	−1.08	0.28

Note. Analyses included participants with data at least at one measurement point.

3.2.4. Prevalence of co-morbid symptoms

The SGH and not-SGH groups were also compared with respect to the co-morbid symptoms. Rates, odds ratios, and confidence intervals are presented in Table 3. As can be seen, SGH had slightly to moderately lower odds across the measurements than not-SGH with respect to all symptom groups.

To examine changes in symptom rates over time, we conducted a series of McNemar tests for each group separately (including only veterans who participated in all three measurements). Among SGH, results showed that symptoms prevalence between T1 and T2 was constant with respect to anxiety ($p = .34$) and obsessive-compulsive ($p = .22$) while increasing for depression, somatization, paranoid ideation ($p < .05$), and hostility ($p < .1$) Also, symptoms prevalence between T2 and T3 was constant for depression ($p = 1$), somatization ($p = .22$), and paranoia ($p = .3$), while tending to increase, although not significantly ($p = .18$), for anxiety, obsessive-compulsive, and hostility. Among not-SGH, symptom prevalence significantly increased ($p < .001$) between T1 and T2 with respect to all groups of symptoms and continued to significantly increase between T2 and T3 for hostility ($p < .05$) and anxiety ($p < .001$) while remaining constant for depression ($p = .83$), obsessive-compulsive ($p = .51$), somatization ($p = .2$), and paranoid ideation ($p = .36$).

4. Discussion

Positive adaption to trauma, reflecting some gains implicated in trauma, has been fundamentally overlooked with respect to the Nazi Holocaust and the crime of Genocide. The passing of all moral bounds can make it unbearable and even atrocious to consider the deliberate annihilation of the existence of a group by serious bodily and mental harm in somewhat positive terms. The present investigation suggests that the effects of the Holocaust on offspring of survivors may however not be solely pathogenic. Findings derived from our two studies showed that these individuals adjust relatively well in the long-term following exposure to war-induced trauma despite exhibiting increased initial psychological vulnerability. In study I, SGH veterans endorsed higher PTSD and co-morbid symptom rates (of depressive, obsessive-compulsive, paranoid symptoms) 1 and 3 years after the war than not-SGH veterans, 17 years later however, this pattern was reversed. PTSD and related symptom rates were relatively low among SGH. Similarly, in study II, SGH veterans endorsed lower PTSD and co-morbid symptom rates than not-SGH veterans 18, 30, and 35 years after the war.

Offspring of Holocaust survivors seem to develop a complex psychological response in the wake of subsequent trauma which is in line with both vulnerability and strength inoculation perspectives. According to the vulnerability perspective, repeated exposure to stressful events increases individuals' vulnerability and reduces their ability to cope with later events (e.g., Selye, 1976). At the same time, in line with the inoculation perspective, recurrent exposure to stress contributes to the development of useful coping strategies resulting in enhanced resilience (e.g., Epstein, 1983) and potential growth in the event of future traumas (Dekel et al., 2012, under review). One may then speculate that through secondary

exposure to Holocaust, and some processing and resolution of their past, the SGH can subsequently mitigate the pathogenic effects and cope with traumatic events they experience in their own lives.

Several factors are likely to account for SGH's complex response to trauma, among them is the element of time. Generally speaking, our findings showed that SGH endorsed less PTSD over a period of 20 years after the war. Thus, while they are prone to posttraumatic stress in the wake of trauma (Baider et al., 2000; Solomon et al., 1988), some may in fact adjust better later in time, in accord with the notion that time is a healer. More importantly, the prevalence of PTSD remained relatively unchanged as SGH grew older and reached mid-life years (on average 56 years old). That is, some seem protected against a delayed PTSD response, contrary to the notion that in aging symptoms intensify with the natural decline in physical and mental health (e.g., Port et al., 2001).

The basic finding of relatively good long-term adjustment among SGH raises the question of what could have been transmitted from the first generation to the next. Similar to their parents, minimization tends to characterize the coping style of SGH (Rim, 1992). Thus, they may come to downgrade the impact of their personal trauma not to reactivate the trauma of their parents (Dekel et al., under review). Baider et al. (2000) note that the conspiracy of silence (Danieli, 1981) carries its effect onto both generations. They found that SGH cancer survivors avoid thinking about their cancer experience. While the tendency to downplay the impact of the trauma hinders emotional processing of the event and trigger symptom formation (e.g., Steil and Ehlers, 2000), appraising the trauma as relatively benign in the long-term may facilitate integration of the memory for the trauma with other experiences and in turn, promote adjustment (Dekel and Bonanno, 2011).

It is also possible that a sense of meaning and purpose in life may have been a powerful legacy transmitted from the Holocaust survivors to their offspring (Kellermann, 2008). These survivors were forced to find meaning in the rebuilding of a new society after the war (Frankl, 1984). With the foundation of the State of Israel in 1948 many of them experienced a sense of psychological rebirth, the regeneration of a new identity on both a personal and communal level (e.g., Solomon et al., 1988). The children themselves may have introjected the rebirth motif when encountering related traumatic experiences. SGH veterans may come to find positive meaning in their war experiences, possibly viewing themselves heroic defenders of their homeland. Finding meaning promotes long-term adjustment by reducing fear of future traumas (Updegraff et al., 2008).

Lastly, SG's relatively good adjustment in the long-term may have to do with their social support. Within heterogenic family environments (Danieli, 1981), many were raised in highly closed family systems with overly committed and protective parents (e.g., Klein-Parker, 1988). While enmeshed relationships can undermine psychological development (e.g., Barocas and Barocas, 1980), proximity to the caregiver builds a child's sense of safety (Bowlby, 1982). Some studies documented that SGH endorsed secure attachment representations, similar to not-SGH (Sagi-Schwartz et al., 2003), and felt supported by their families when dealing with traumatic experiences (Baider et al., 2008). Possibly then the family bonds of SGH may help alleviate pathogenic effects and facilitate long-term adjustment.

This study has several shortcomings worth noting. First, it does not allow clear-cut inferences regarding causal relations between SGH status and psychological adjustment following trauma particularly since a detailed account of exposure to trauma following the war is missing. A related issue concerns absence of data on the parents' psychological well-being; one cannot make definite conclusions in respect to what has been transmitted to the children. Another important limitation, inherent in longitudinal designs, is that not all participants took part in all assessments, although the samples did not differ with respect to personal background and exposure level. Also, this study included only SGH who were raised in Israel. While no difference in general adjustment was found between Israeli SGH and those growing up in other countries (van Ijzendoorn et al., 2003), there might be differences in adjustment following trauma. Finally, we measured PTSD and comorbid symptoms rather than psychiatric diagnosis and therefore clinical implications of the study should be made with caution.

Within the context of these limitations, the present study provides preliminary evidence and encourages a new field of investigation into the complexity of secondary human response to trauma with respect to psychological adjustment, underscoring the importance of long-term follow-up. As in reality a significant number of individuals may be subject to repeated traumas, future research should unmask additional moderating factors of this kind of adjustment. Our findings clearly warrant more longitudinal research to better untangle the issue of transmission of trauma from Holocaust survivors to their children. In the course of treatment and assessment it is important that clinicians take into consideration the possible variability in SGH's response to trauma.

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Contributors

Dr. Sharon Dekel – wrote the manuscript and generated the study paradigm, conducted the statistical analysis, and prepared the manuscript.

Prof. Zahava Solomon – supervised the research project.

Dr. Eyal Rozen – contributed to the statistical analysis.

Conflict of interest

No.

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