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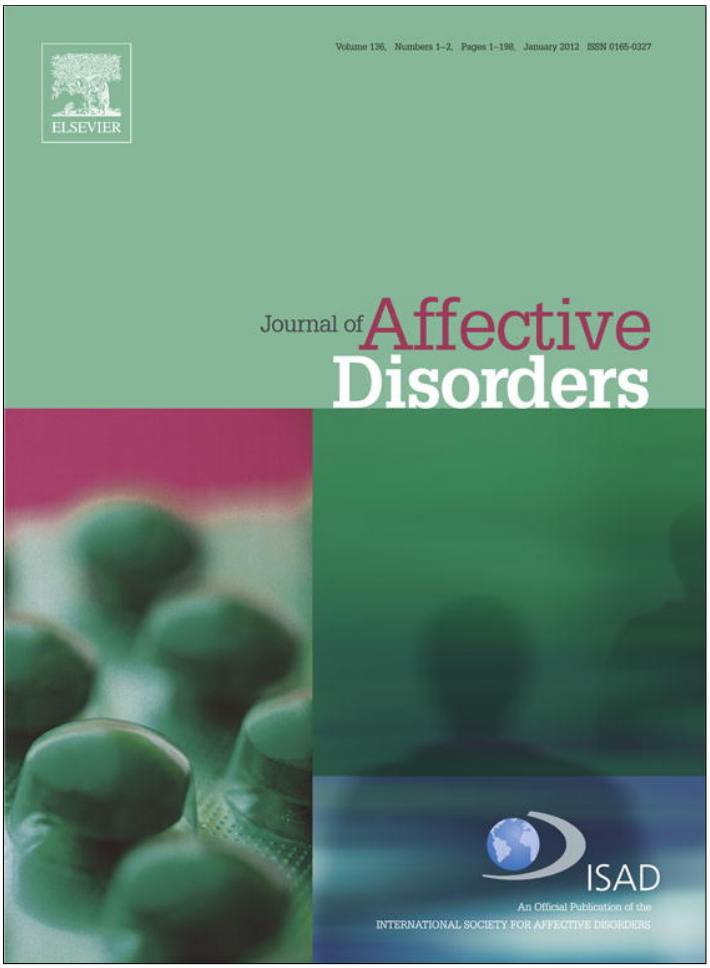

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Research report

The role of fathers' psychopathology in the intergenerational transmission of captivity trauma: A twenty three-year longitudinal study

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ABSTRACT

Background: The aversive impact of combat and parents' combat-induced posttraumatic stress disorder (PTSD) on young children has been examined in a few studies. However, the long-term toll of war captivity on the secondary traumatization (ST) of adult offspring remains unknown. This study aimed to assess the longitudinal associations between former prisoners of war (ex-POWs), PTSD, depressive symptoms and their adult offsprings ST.

Method: A sample of 134 Israeli father-child dyads (80 ex-POWs dyads and a comparison group of 44 veterans'dyads) completed self-report measures. The fathers participated in three waves of measurements following the Yom Kippur War (T1: 1991, T2: 2003, and T3: 2008), while the offspring took part in T4 (2013).

Results: Offspring of ex-POWs with PTSD at T3 reported more ST symptoms than offspring of ex-POWs without PTSD and controls. Ex-POWs' PTSD hyper-arousal symptom cluster at T3 was positively related to offsprings ST avoidance symptom cluster. Offspring of ex-POWs with chronic and delayed PTSD trajectories reported more ST symptoms than offspring of ex-POWS and controls with resilient trajectories. Ex-POWs' PTSD and depression symptoms at T1, T2 and T3 mediated the link between war captivity (groups) and offsprings ST in T4.

Limitations: The use of self-report measures that did not cover the entire span of 40 years since the war, might may bias the results.

Conclusions: The intergenerational transmission of captivity related trauma following the Yom Kippur War was exemplified. ST symptoms among ex-POWs' adult offspring are closely related to their father' PTSD and related depressive symptom comorbidity.

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1. Introduction

War captivity is one of the most severe man-made traumatic events to which an individual can be subjected. Prisoners of war (POWs) endure deliberate human cruelty through the infliction of physical and psychological torture, isolation, systematic humiliation, starvation, and the use of psychological tactics aimed at breaking their spirit and altering their psyche. As a result, ex-POWs may suffer from deteriorated physical health, long-term mental health disorders and profound personality changes, the most common of which is posttraumatic stress disorder (PTSD; e.g., Meziab et al., 2014). Indeed, high rates of PTSD, ranging from 16% to 88%, have been observed in ex-POW samples (e.g., Rintamaki et al., 2009). However, traumatic events may entail long-term consequences not only for the direct trauma survivors but also for their significant others, in the form of secondary traumatization (ST; Zerach et al., 2013). The term "ST" has been used to indicate those who have come into close contact with a traumatized person and who may experience emotional distress and PTSD-like responses similar to those exhibited by the survivor (Figley, 1995).

The intergenerational transmission of war trauma to veterans' offspring in the form of ST has recently attracted growing interest (Maršanić et al., 2013). However, most studies have focused on young children and adolescents (Rosenheck and Fontana, 1998) and based their conclusions on parents' reports (Ruscio et al., 2002). Moreover, almost no attention has been paid to adult offspring of ex-POWs in the existing literature. Bernstein (1998)

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found that offspring of World War II (WWII) ex-POWs retrospectively described their fathers as quick to suffer from outbursts of anger, emotionally distant, and generally unresponsive to their emotional needs. A cross-sectional study among Iran-Iraq War ex-POWs' adult offspring reported a higher prevalence of depression and general anxiety compared to an average adult group (Razavi et al., 2012). Recently, it was found that 1973 Yom Kippur War ex-POWs' adult offspring reported higher levels of ST compared to adult offspring of combatants who were not held captive (Zerach and Aloni, 2015). Although these studies provide insights regarding the long-term effects of captivity trauma on ex-POWs' offspring, their cross-sectional design limits the possibility to understand the mechanisms of intergenerational transition of captivity trauma. The present study sought to fill this knowledge gap by focusing on the ST of ex-POW's adult offspring longitudinally, as related to their fathers'trajectories of psychopathology.

In general, there is comprehensive literature regarding the effects of parental psychopathology on children (e.g., Beardslee et al., 2011). Specifically, case studies (Rosenheck and Nathan, 1985), empirical studies (e.g., Ahmadzadeh and Malekian, 2004), literature reviews (Dekel and Goldblatt, 2008) and meta-analyses (Lambert et al., 2014) have exemplified the associations between parents' combat-related PTSD symptoms (PTSS) and children's psychological difficulties and behavioral problems. In the absence of studies among ex-POWs' adult offspring, a line can also be drawn from studies among Holocaust survivors' offspring showing that parental PTSD predicts offsprings psychiatric disorders, including PTSD (e.g., Yehuda et al., 2008). Nevertheless, following the severe, prolonged and inter-personal trauma of captivity, a question remains regarding the role of fathers' PTSS cluster profile, PTSD trajectories and depression comorbidity in their adult offsprings ST.

Several PTSS clusters may be especially relevant to the intergenerational transmission of combat-related trauma due to their effect on maladaptive parenting practices. Specifically, empirical literature suggests that the avoidance and emotional numbness symptoms on the one hand, and hyper-arousal symptoms on the other hand, are significantly implicated in the father's parenting (Ruscio, et al., 2002; Samper et al., 2004). Avoidance and emotional numbness symptoms entail reduced involvement of the traumatized veteran in his offsprings life that may even result in disengagement (Marshall et al., 2006). In this sense, these symptoms tend to severely undermine the father's ability to create and maintain close meaningful and supportive interactions with his children, which in turn can lead to an experience of low paternal care and higher levels of ST for ex-POWs' offspring (Zerach and Aloni, 2015). It has been found that hyper-arousal symptoms might be related to the use of verbal aggression (Solomon et al., 2008). Fathers with PTSD often have a low frustration threshold and may find it difficult to contain negative feelings towards their children, which could be attributed to their behavioral dysregulation.

Moreover, the psychiatric literature mostly treats PTSD as a monolithic entity with prototypic symptom clusters that change in the same manner and direction. However, long-term reactions to traumatic stress are highly heterogeneous and labile. The Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition (DSM-5; American Psychiatric Association, 2013) identifies two possible PTSD courses: typical (with no distinction between acute and chronic phases) and delayed expression. Blank (1993) also proposed intermittent and reactivated PTSD as additional courses, with possible changes over the years.

To date, there is conflicting empirical evidence regarding the course of captivity-induced PTSD. While most studies point to a gradual decrease in the number of posttraumatic symptoms (e.g., Zeiss and Dickman, 1989), other studies observed a fluctuating course with symptoms increasing and decreasing over time (e.g., Port et al., 2001). A prospective study among Israeli ex-POWs found that 34.7% of ex-POWs and only 2.5% of the matched veterans group met PTSD criteria 35 years after the war, with prior fluctuations. As well, an incline in PTSD rates over a 17-year period was reported with the majority of ex-POWs who experienced delayed PTSD (Solomon et al., 2012). An important question is whether the impact that the fathers' PTSD has on offspring goes beyond his current mental health status. As some studies (e.g., Lambert et al., 2012) found stronger effects from trauma that had taken place in the distant past, rather than more recently, it is relevant to look at the long-term effects of trauma in the form of PTSD trajectories.

PTSD has been recognized in the DSM-5 as a distinguished psychiatric disorder, precipitated by exposure to a traumatic event. However, the literature points to a strong comorbidity between PTSD and other psychiatric disorders, most notably with depression (e.g., Campbell et al., 2007). Numerous studies have reported high rates of individuals with PTSD who meet diagnostic criteria for depression at some point in time in both community (e.g., Breslau et al., 1997) and clinical samples (e.g., Keane and Kaloupek, 1997). Among Israeli ex-POWs it has been found that symptoms of PTSD and depression were inter-related over the years, with up to 30% comorbidity (Dekel et al., 2014). Comorbidity is also known to be a more severe disorder, with dire implications, possibly leading to further impaired parenting (Beardslee et al., 2011; Ginzburg et al., 2010). To the best of our knowledge, no study has gone beyond the current state of the fathers' PTSS to explore the link between their depressive symptom comorbidity and their adult offsprings ST.

We hypothesize that: (a) Adult offspring of ex-POWs with PTSD at T3 will report more ST symptoms than adult offspring of ex-POWs without PTSD and adult offspring of controls; (b) Adult children of ex-POWs with chronic and delayed PTSD trajectories will report more ST symptoms than adult offspring of ex-POWs and adult offspring of controls with resilient trajectories; (c) Ex-POWs' PTSS clusters at T1, T2 and T3 will be positively related to adult offsprings ST symptom clusters; (d) Ex-POWs' PTSS and depression symptoms at T1, T2 and T3 will mediate the link between war captivity (groups) and adult offsprings ST.

2. Method

2.1. Participants

This study constitutes part of a larger longitudinal study assessing the psycho-social impact of war captivity (for more details see Solomon et al. (2012)). The sample consisted of 134 Israeli father-adult offspring dyads in which the father was a veteran from the Israeli Defense Forces (IDF) from the 1973 Yom Kippur War. The sample was divided into the following two groups: (a) 80 dyads of ex-POWs and their adult offspring and (b) 44 control dyads in which the fathers fought on the same fronts as the ex-POWs, but were not held captive, and their adult offspring. Control participants were sampled from IDF computerized data banks and selected on the basis of their similarity to the ex-POWs in relevant military and personal variables. As can be seen in table 1, ex-POWs and controls did not differ at T3 in age, education, religiosity or fathers' country of birth. Furthermore, the groups did not differ in participation in previous wars, combat exposure and number of negative life events after the war. Data was collected from the fathers at three time points: 1991 (T1), 2003 (T2), and 2008 (T3), and from adult offspring at one time point (2013–2014).

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Table 1
Socio-Demographic characteristics of ex-POWs' and controls' adult offspring.

Fathers' variables (T3)		Ex-POW dyads($n=80$)	Control dyads($n=44$)	
Age		$M=57.86$ (SD=6.25)	$M=56.58$ (SD=4.16)	$t(112)=-1.14$
Education		$M=14.04$ (SD=4.40)	$M=14.43$ (SD=3.16)	$t(112)=.49$
Participation in previous wars		$M=0.30$ (SD=0.72)	$M=0.62$ (SD=1.02)	$t(119)=1.81$
Combat exposure		$M=1.41$ (SD=0.56)	$M=1.68$ (SD=0.71)	$t(61)=1.65$
Negative life events since war		$M=7.10$ (SD=5.02)	$M=6.74$ (SD=5.12)	$t(119)=-0.37$
Father's country of origin	Israel	50 (68.5%)	33 (84.6%)	$\chi^2_{(2)}=4.02$
	America	16 (21.9%)	3 (7.7%)	
	Europe	7(9.6%)	3 (7.7%)	
Religiosity	Secular	49 (65.3%)	23 (59%)	$\chi^2_{(2)}=0.80$
	Traditional	20 (26.7%)	11 (28.2%)	
	Religious	6 (8%)	5 (12.8%)	
Offspring variables				
Age		$M=35.12$ (SD=6.49)	$M=34.84$ (SD=5.44)	$t(132)=-0.23$
Education (years)		$M=14.89$ (SD=2.81)	$M=16.42$ (SD=2.51)	$t(120)=3.00^{**}$
Gender	Female	43 (55.1%)	20 (45.5%)	$\chi^2_{(1)}=1.05$
	Male	35 (44.9%)	24 (54.5%)	
Birth order	Firstborn	42(53.8%)	28 (63.6%)	$\chi^2_{(4)}=5.25$
	Second born	19 (24.4%)	13 (29.5%)	
	Third born	9 (11.5%)	1 (2.3%)	
	Fourth born	6 (7.7%)	2 (4.5%)	
	Fifth born	2 (2.6%)	0 (0%)	
Marital status	Single	27 (34.6%)	14 (31.8%)	$\chi^2_{(3)}=3.76$
	Married	42 (53.8%)	29 (65.9%)	
	Divorced	8 (10.3%)	1 (2.3%)	
	Other	1 (1.3%)	0 (0%)	
Military service	Complete	62 (79.5%)	36 (81.8%)	$\chi^2_{(3)}=5.40$
	Partial	4 (5.1%)	4 (9.1%)	
	National	2 (2.5%)	3 (6.8%)	
	Other	10 (12.8%)	1 (2.3%)	
Religiosity	Secular	54 (70.1%)	25 (59.5%)	$\chi^2_{(4)}=3.96$
	Traditional	13 (16.9%)	13 (31%)	
	Religious	8 (10.4%)	3 (7.1%)	
	Orthodox	1 (1.3%)	0 (0%)	
	Other	1 (1.3%)	1 (2.4%)	
Place of birth	Israel	72 (93.5%)	41 (93.2%)	$\chi^2_{(2)}=0.18$
	America	1 (1.3%)	1 (2.3%)	
	Europe	4 (5.2%)	2 (4.5%)	
Employment	Not working	4 (5.3%)	0 (0%)	$\chi^2_{(2)}=2.55$
	Part time job	16 (21.1%)	8 (18.6%)	
	Full time job	56 (73.7%)	35 (81.4%)	
Income	Well below average	4 (5.2%)	6 (13.6%)	$\chi^2_{(4)}=8.77$
	Below average	14 (18.7%)	8 (18.2%)	
	Average	23 (30.7%)	67 (13.6%)	
	Above average	23 (30.7%)	12 (27.3%)	
	Well above average	11 (14.7%)	12 (27.3%)	

** $p < .01$.

2.2. POWs' adult offspring

This group consisted of 80 adult offspring, of whom 37 (47%) were male and 42 (53%) were female. Ages ranged from 22 to 53 years ($M=35.19$, $SD=6.44$). Twenty-five participants (22.8%) were born before the war while the rest were born afterwards. No differences were found among the adult offspring in relation to their birth before or after the war. We contacted 92 adult offspring, only one child from each family, of which 80 participated and 12 declined. Response rate in this group was 87%.

2.3. Controls' adult offspring

This group consisted of 44 participants, of whom 24 (54.5%) were male and 20 (45.5%) were female. Ages ranged from 21 to 47 ($M=34.84$, $SD=5.44$). Twelve participants (18.5%) were born before the war, while the rest were born afterwards. We contacted 68 adult offspring, only one child from each family, of which 14 declined to participate. Response rate in this group was 80.6%.

The two adult offspring groups did not differ in age, gender, birth order, marital status, military service, level of religiosity, place of birth, employment, or income. As can be seen in Table 1, the groups did differ in the years of education, with controls' adult offspring reporting more years of education compared to POWs' adult offspring.

2.4. Measures

2.4.1. PTSD Inventory

(PTSD-I; Solomon et al. (1994). Fathers' PTSS and adult offsprings ST as reflected in PTSS were assessed by using a self-report scale corresponding to PTSD symptom criteria listed in DSM-IV-TR (American Psychiatric Association, 2000). Subjects were asked to indicate on a 4-point scale ranging from 1 (never) to 4 (almost always) the frequency with which they experienced the described symptom in the previous month, in relation to their fathers' experience of combat or captivity. (e.g., "I have recurrent pictures or thoughts about my fathers' captivity"). The intensity of their secondary traumatization was assessed by the number of positively endorsed symptoms that was calculated by counting the items in which the respondents answered "3" or "4". The scale was found to have good psychometric properties, including high convergent validity compared with clinical interviews based on the SCID (Solomon et al., 1994). The PTSD-I was administrated to fathers in all three waves: 1991, 2003 and 2008. Reliability values for total and subscale scores were high at all assessments (Cronbach's α: 0.78–0.96). The PTSD-I reliability value for adult offsprings ST was Cronbach's $\alpha=0.86$. As offsprings ST symptoms variable was over dispersed we employed the Poisson transformation (also known as the Freeman–Tukey transformation) which is recommended for bound at the bottom count-type variables (Cohen et al., 2013). All analyses were carried out with the ST symptoms transformed values.

2.5. Depressive symptoms

were assessed using the depression subscale of the Symptom Checklist-90 (SCL-90; Derogatis and Lipman (1977). Participants were asked to indicate how frequently they experienced each symptom during the last two weeks on a 5-point distress scale. For each participant, we calculated the average frequency of experiencing depressive symptoms in each of the time points. The SCL-90 has high concurrent validity and the subscales display high empirical agreement across various samples (Derogatis et al., 1976). In this study, reliability values were high in all assessments (Cronbach's α: were 0.92, 0.94, 0.90, respectively, in T1, T2, and T3).

2.6. Life events checklist

(LEC; Gray et al., 2004). This questionnaire is comprised of 17 potential traumatic events over the lifetime of the participant that

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can lead to PTSD or psychological distress (e.g., work or car accident, physical or sexual assault, exposure to violent death). The use of this questionnaire was for the purpose of statistical control. For each item, the respondent marked whether the event happened to him/her personally (0), was witnessed by him/her (1), heard of it (2), not sure (3), or irrelevant (4). Items that were marked as happened personally (0) were encoded as "1", while the other items (1–4) were coded as "0". The sum of negative life events that participants personally experienced was used for analysis. The possible range of LEC index is 0–17, and the actual range in this study was 0–10. The LEC has shown good psychometric properties (Gray et al., 2004). The LEC inventory reliability in the current study was Cronbach's $\alpha=0.87$.

2.7. Socio-demographic measurements

were assessed using demographic characteristics of country of origin, location of residence in Israel, family status, religious orientation, age, gender, birth order, level of education, and years of living with the father.

3. Results

3.1. Differences between adult offspring of ex-POWs with PTSD, adult offspring of POWs without PTSD and adult offspring of controls in ST symptoms

The first aim of the current study was to examine whether adult offspring of ex-POWs with PTSD at T3 will report more ST symptoms than adult offspring of ex-POWs without PTSD and adult offspring of controls. To assess this we performed a MANCOVA analysis for the ST symptoms and its three clusters (intrusion, avoidance, hyper-arousal) with the offsprings number of negative life events as covariate. We separated the two groups into three groups: ex-POWs with PTSD ($n=44$), ex-POWs without PTSD ($n=31$), and control group without PTSD ($n=39$). Due to the low number of participants in the control group with PTSD ($n=3$, 5.1%), our ability to examine the interaction between research groups (ex-POWs and controls) and PTSD was limited; therefore we decided to omit the interactions from this analysis. We found a significant difference between the groups with respect to the adult offsprings ST general factor (*Pillai's Trace* F $(8,218)=2.18$, $p=0.03$, Partial Eta$^2=0.08$). As hypothesized, and can be seen in Table 2, separate ANCOVA analyses revealed that adult offspring of ex-POWs with PTSD reported a higher total number of ST, intrusion, avoidance and hyper-arousal symptoms when compared to adult offspring of ex-POWs without PTSD and adult offspring of controls.

Differences in adult offsprings ST symptoms according to their fathers' PTSD trajectories between T1 and T3.

The second aim of this study was to examine whether adult offspring of ex-POWs with chronic and delayed PTSD trajectories will report more ST symptoms than adult offspring of both ex-POWS and controls with resilient trajectories. First, the adult offspring were divided into four groups according to the ex-POW's self-reported PTSS trajectories at T1–T3 (only ex-POWs who participated in all three measurement waves were included; $n=141$): chronic PTSD, if the ex-POW met criteria for PTSD in all three waves ($n=4$; 4.5%); delayed PTSD, if the ex-POW did not endorse PTSD criteria in the first wave but did in subsequent waves ($n=63$; 70.8%); recovered PTSD, if they endorsed PTSD criteria in either of the first two waves, but not in the third ($n=1$; 1.1%); and resilient, if the ex-POW never endorsed criteria for PTSD ($n=21$; 23.6%). Second, due to the small group sizes, the recovered group was omitted from our analyses and the chronic group was added to the delayed PTSD group. An additional group was included in the comparisons: adult offspring of resilient control veterans who did not endorse PTSD criteria in any of the measurement waves ($n=55$; 85.9%).

A MANCOVA, using the offspring number of negative life events as covariate, returned no significant effect of group (adult offspring of ex-POWs with chronic and delayed PTSD trajectories, adult offspring of ex-POWs with resilient trajectory, and adult offspring of controls with resilient trajectory) on ST total score or ST symptom clusters (*Pillai's Trace* F $(8,132)=0.82$, $p=0.586$, Partial Eta$^2=0.05$). When we combined the two resilient trajectory groups and re-ran the MANCOVA with number of offspring negative events covaried, there was no significant effect of group on ST total score or ST symptom clusters (*Pillai's Trace* F $(4,66)=1.04$, $p=0.395$, Partial Eta$^2=0.06$). Separate univariate ANCOVAs of the outcome variables revealed marginally significant effects for total ST symptoms (F $(1,69)=3.44$, $p=0.068$, Partial Eta$^2=0.05$) and hyper-arousal symptoms (F $(1,69)=3.69$, $p=0.059$, Partial Eta$^2=0.05$). Due to the low number of participants in the group, we also ran this analysis without the statistical control of negative life events. Results showed the same pattern of results with significance for ST symptoms (F $(1,72)=4.54$, $p=0.036$, Partial Eta$^2=0.06$) and hyper-arousal symptoms (F $(1,72)=5.32$, $p=0.024$, Partial Eta$^2=0.07$). Thus, adult offspring of ex-POWs with chronic or delayed trajectories reported more ST symptoms, and specifically hyper-arousal symptoms, as compared to adult offspring of ex-POWs and controls with resilient trajectory.

3.2. The associations between ex-POWs' PTSD symptom clusters and adult offsprings' ST symptom clusters

In this section we aimed to examine the interrelations between ex-POWs' PTSS clusters at T1, T2 and T3 and adult offspring's ST symptom clusters. In order to do so we conducted Pearson correlations, with Bonnferroni correction to minimize type I error,

Table 2
Means and SD differences in secondary traumatization between adult offspring of ex-POWs with PTSD at T3, adult offspring of ex-POWs without PTSD and adult offspring of controls.

	Offspring of ex-POWs with PTSD ($n=44$)[a]		Offspring of ex-POWs without PTSD ($n=31$)[b]		Offspring of controls ($n=39$)		F	Partial Eta^2
	M	SD	M	SD	M	SD	(2,111)	
Total number of PTSS	3.80	1.76	2.68	1.57	2.49	1.59	$=7.51$ a > c; a > b	0.12
Intrusion symptoms	1.49	0.91	1.09	0.35	1.09	0.41	$=5.01^{***}$ a > c; a > b	0.08
Avoidance symptoms	2.48	1.31	1.85	1.24	1.79	1.40	$=3.76^{*}$ a > c; a > b	0.06
Hyper-arousal symptoms	2.75	1.33	2.09	1.13	1.95	1.15	$=4.96^{***}$ a > c; a > b	0.08

Note: $^{**}p=<.01$
$^{*}p=<.05$
$^{***}p=.00$

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Table 3
Pearson correlation coefficients of fathers' PTSS clusters and depressive symptoms at T1, T2, and T3 and adult offsprings ST symptoms at T4.

Fathers' variables	ST total symptoms	ST intrusion symptoms	ST avoidance symptoms	ST hyper-arousal symptoms
Total PTSS (T1)	0.04	−0.06	0.07	−0.01
Intrusion symptoms (T1)	0.11	0.01	0.15	0.05
Avoidance symptoms (T1)	−0.04	−0.05	0.01	−0.09
Hyper-arousal symptoms (T1)	0.02	−0.08	0.03	0.01
Depressive symptoms (T1)	0.18	−0.09	0.18	0.13
Total PTSS (T2)	0.32***	0.26*	0.21	0.29***
Intrusion symptoms (T2)	0.34***	0.16	0.26	0.32***
Avoidance symptoms (T2)	0.23	0.24	0.13	0.23
Hyper-arousal symptoms (T2)	0.29**	0.28**	0.17	0.21
Depressive symptoms (T2)	0.28**	0.27**	0.22	0.21
Total PTSS (T3)	0.35***	0.25**	0.25**	0.30**
Intrusion symptoms (T3)	0.35***	0.25**	0.27**	0.30**
Avoidance symptoms (T3)	0.26**	0.21	0.15	0.25**
Hyper-arousal symptoms (T3)	0.33***	0.21	0.28**	0.27**
Depressive symptoms (T3)	0.34***	0.28**	0.27**	0.27**
Mean	2.98	1.23	2.01	2.28
Standard deviation	1.72	0.64	1.23	1.25

*p=.05; **=p .01; *** = p .001. All significant correlations were corrected with Bonnferroni correction for type 1 error.

followed by hierarchical regression. As seen in Table 3, none of the fathers' PTSS clusters at T1 were related to adult offsprings ST symptom clusters. However, as expected, fathers' hyper-arousal symptom cluster at T2 was positively related to adult offsprings intrusion symptom cluster. Furthermore, results revealed significant positive relations between all three of the fathers' PTSS clusters at T2 and adult offsprings ST avoidance and hyper-arousal symptom clusters. Results revealed significant positive relations between all three of the fathers' PTSS clusters at T3 and all three of the adult offsprings ST symptom clusters. Results also show significant positive relation between depressive symptoms at T1 and adult offsprings ST avoidance. Lastly, results revealed significant positive relations between depressive symptoms at T2 and T3 and all three of the adult offsprings ST symptom clusters.

Next, we examined the unique contribution of fathers' PTSS clusters at T1, T2 and T3 to adult offsprings ST symptom clusters. Due to the lack of significant correlations between the fathers' PTSS clusters at T1 and the adult offsprings ST symptom clusters, we focused only on T2 and T3. In order to do so, three, four-step hierarchical regression analyses were conducted for each of the adult offsprings ST symptom clusters. In the first step of each regression, we entered the sum of the offsprings negative life events. In the second step, we entered the group variable (0=controls' adult offspring and 1=ex-POWs' adult offspring). In the third step, we entered the fathers' PTSS clusters at T2. In the fourth step, we entered fathers' PTSS clusters at T3. The regression model for intrusion (F (8,73)=1.40, p=0.210) was not significant, and the regression model for hyper-arousal (F(8,73)=1.45, p=0.19) was only marginally significant. None of the predictors were significantly related to the adult offsprings ST symptom clusters. Table 4 presents the regression coefficients for the prediction of the ST avoidance symptom cluster.

The total set of variables explained 17.1% of the variance of the ST avoidance symptom cluster (F(8,73)=1.88, p=0.076). As can be seen in Table 4, fathers' hyper-arousal symptom cluster in T3 predicted adult offsprings ST avoidance. The more hyper-arousal symptoms the father reported in 2008, the higher his adult child's number of ST avoidance symptoms was in 2013.

3.3. *Fathers' PTSD and depressive symptoms at T1, T2 and T3 mediate the link between war captivity and adult offsprings ST*

The fourth aim of this study was to examine whether fathers' PTSS and depressive symptoms at T1, T2 and T3 mediated the link between war captivity and offsprings ST symptoms. We used structural equation modeling (SEM) to examine two separate models for PTSS and depressive symptoms in which we asked whether: (1) war captivity (ex-POWs vs. controls) directly affected adult offsprings ST, controlling for PTSS or depressive symptoms at T1, T2 and T3 (path c'); (2) war captivity indirectly affected adult offsprings ST via PTSS or depressive symptoms at any of the time points (i.e., T1–T3 separately); (3) war captivity indirectly affected adult offsprings ST via a 2-step mediation process (i.e., via PTSS or depressive symptoms at T1–T2, T1–T3, and/or T2–T3), and (4) war captivity indirectly affected adult offsprings ST via a 3-step mediation process (via PTSS or depressive symptoms at T1, T2, and T3).

To estimate the model we used AMOS 21 (Arbuckle, 2012). A model has high fit to the observed data if the Comparative Fit Index (CFI) and the Tucker-Lewis Index (TLI) are greater than .95 and the Root Mean Square Error of Approximation (RMSEA) are

Table 4
Summary of hierarchical regression coefficients of ST symptoms by group and fathers'PTSS clusters at T2 and T3.

Predicting variables	ST Avoidance symptoms			
	b	SEB	β	$R^2\Delta$
Step I				0.01
Negative life events	0.08	0.09	.10	
Step II				0.01
Negative life events	0.08	.09	.010	
Group (1=ex-POWs' offspring)	0.21	.30	0.08	
Step III				0.05
Negative life events	0.3	0.10	0.03	
Group (1=ex-POWs' offspring)	−0.11	0.42	−0.04	
Intrusion symptoms (T2)	0.18	0.11	0.28	
Avoidance symptoms (T2)	−0.04	0.06	−0.07	
Hyper-arousal symptoms (T2)	0.02	0.11	0.03	
Step IV				0.11*
Negative life events	0.03	.10	.07	
Group (1=ex-POWs'offspring)	−0.11	0.42	−0.06	
Intrusion symptoms (T2)	0.13	0.11	0.20	
Avoidance symptoms (T2)	−0.04	0.10	−0.07	
Hyper-arousal symptoms (T2)	−0.12	0.12	−0.18	
Intrusion symptoms (T3)	0.13	0.12	0.19	
Avoidance symptoms (T3)	−0.10	0.10	−0.21	
Hyper-arousal symptoms (T3)	0.27	0.12	0.40*	
R^2	0.17			

**p < .01, ***p < .001.
* p < .05

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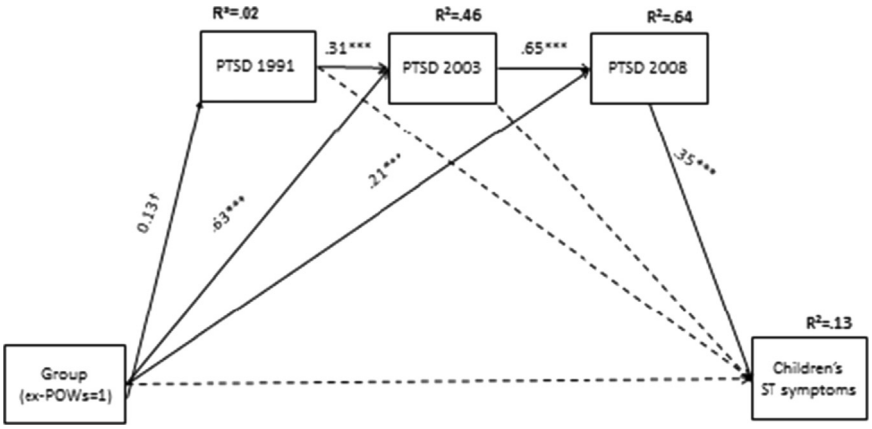

Fig. 1. Mediational model for adult offsprings ST symptoms with fathers'PTSD. Unidirectional arrows depict hypothesized directional links. Standardized maximum likelihood parameters are used. Bold estimates are statistically significant. Group is a binary-coded variable (0=controls veterans; 1=ex-POWs).†$p<.10$, ***$p<.001$.

lower than .05. A model has adequate fit to the observed data if the CFI and TLI are greater than .90 and the RMSEA are lower than .10. In order to estimate the significance of the indirect effect we employed a bootstrapped confidence interval for the *ab* indirect effect using procedures described by Preacher and Hayes (2008). In this analysis 5000 bootstrapped samples were drawn to estimate indirect effects of each of the mediators. Bias corrected and accelerated 95% confidence intervals (CIs) were computed to determine statistical significance of the *ab* paths of each mediator. A CI that does not include zero provides evidence of a significant indirect effect or significant mediation. Missing data were handled with the case-wise maximum likelihood estimation. All analyses were carried out with the number of offspring negative life events covaried. As none of the links with negative life events were significant, they are not presented in the figures for the sake of presentation clarity.

The first analysis revealed that the mediation model of PTSS had excellent fit to the observed data (χ^2 (8)=9.06, p=.33, CFI=0.99, TLI=0.99, RMSEA=0.03). As expected and can be seen in Fig. 1, war captivity had an indirect effect on the adult offsprings ST via fathers' PTSD at T1, T2, and T3. Specifically, war captivity was related to the level of adult offsprings ST through three indirect pathways: war captivity increased the level of PTSS in T3, which in turn was related to higher levels of adult offsprings ST in T4 (indirect effect b=1.24, SE=0.33, 95%I LL=0.73, UL=1.86); war captivity increased the level of PTSS in T2 and T3, which in turn was related to higher levels of adult offsprings ST (indirect effect b=1.01, SE=0.28, 95% CI LL=0.58, UL=1.54); and finally, war captivity increased the level of PTSS in T1, which in turn increased the level of PTSS in T2, which in turn increased the level of PTSS in T3, which by its own merit was related to higher levels of adult offsprings ST (indirect effect b=0.06, SE=0.04, 95% CI, LL=0.01, UL=0.15). Thus, controlling for fathers' PTSD at T1, T2, and T3, the analysis revealed that war captivity had no direct effect on adult offsprings ST.

The second analysis revealed that the multiple step mediation model of depressive symptoms had excellent fit to the observed data (χ^2 (6)=2.18, p=.90, CFI=1, TLI=1, RMSEA=0.00). As can be seen in Fig. 2, controlling for fathers' depressive symptoms at T1, T2, and T3, the analysis revealed that war captivity had no direct effect on adult offsprings ST. However, as expected, war captivity had an indirect effect on the adult offsprings ST via the fathers'depressive symptoms at T1, T2, and T3. Specifically, war captivity related to the level of adult offsprings ST through three indirect (and independent) pathways: war captivity increased the level of depressive symptoms in T3, which in turn was related to higher levels of adult offsprings ST (indirect effect b=0.98, SE=0.28; 95% CI LL=0.48, UL=1.58); war captivity increased the level of depressive symptoms in T2 as well as in T3, which in turn was related to higher levels of adult offsprings ST in T4 (indirect effect b=0.72, SE=0.22; 95% CI LL=0.33, UL=1.21); and finally, war captivity increased the level of depressive symptoms in T1, which in turn increased the level of depressive symptoms in T2, which resulted in increased levels of depressive symptoms in T3, which by its own merit was related to higher levels of adult offsprings ST (indirect effect b=1.55, SE=0.09, 95% CI, LL=0.02, UL=0.39).

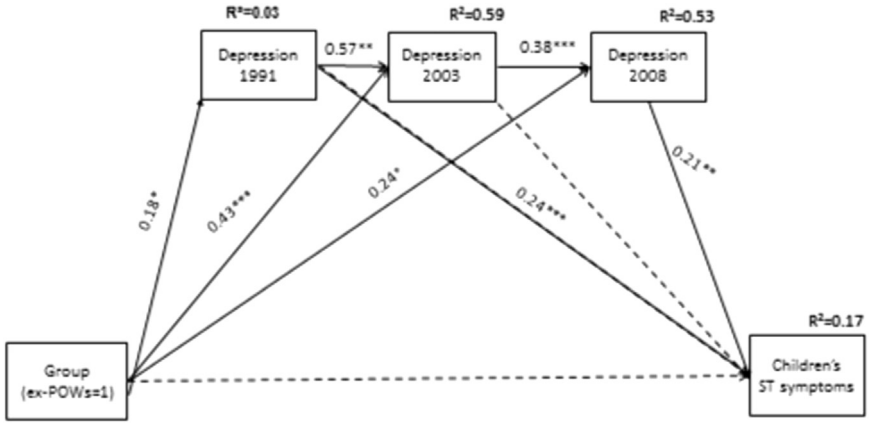

Fig. 2. Mediational model for adult offsprings ST symptoms with fathers'depressive symptoms. Unidirectional arrows depict hypothesized directional links. Standardized maximum likelihood parameters are used. Bold estimates are statistically significant. Group is a binary-coded variable (0=controls veterans; 1=ex-POWs).*$p<.05$, **$p<.01$, ***$p<.001$.

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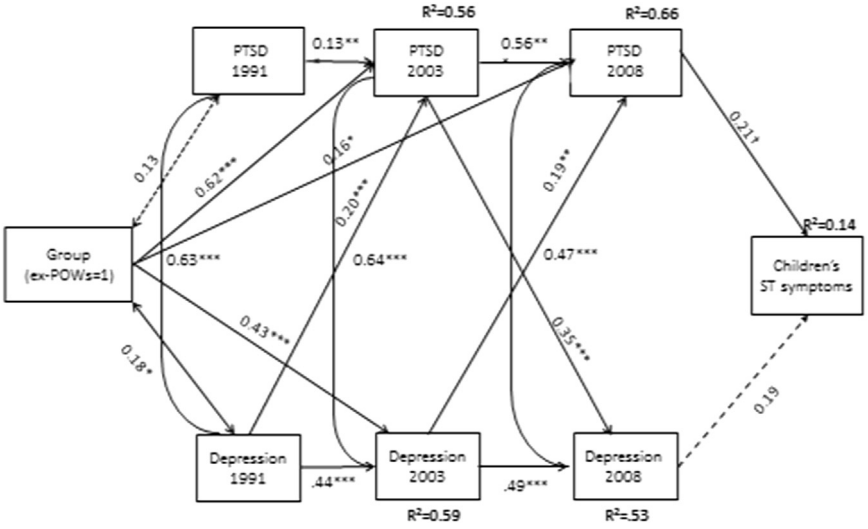

Fig. 3. Mediational model for adult offsprings ST symptoms with fathers' PTSD and depressive symptoms. Unidirectional arrows depict hypothesized directional links and bidirectional arrows depict hypothesized bidirectional links. Standardized maximum likelihood parameters are used. Bold estimates are statistically significant. Group is a binary-coded variable (0=controls veterans; 1=ex-POWs).†$p < .10$,*$p < .05$, **$p < .01$, ***$p < .001$.

Our last aim was to examine a general path model in which both fathers' PTSS and depressive symptoms at T1, T2 and T3 mediated the link between war captivity and adult offsprings ST symptoms. As noted, the literature reports on the high co-morbidity between PTSD and depression among war veterans (e.g., Ginzburg et al., 2010). Furthermore, among ex-POWs, a positive bi-directional relationship was found where PTSS mediated the effect of captivity on depressive symptoms and depressive symptoms mediated the effect of captivity on PTSS over time (Dekel et al., 2014). Hence, in the final model we allowed the residual error terms of PTSS and depressive symptoms at T1, T2 and T3 to correlate. In addition, we allowed six cross-lagged paths from: PTSD at T1 to depression at T2 and T3; PTSD at T2 to depression at T3; depression at T1 to PTSD at T2 and T3; and depression at T2 to PTSD at T3.

The general path model of PTSS and depressive symptoms had an acceptable fit to the observed data (χ^2 (18)=30.79, p =.03, CFI=0.98, TLI=0.95, RMSEA=0.07). As can be seen in Fig. 3, above and beyond the mediational process of PTSS and depressive symptoms that was described earlier, we found that PTSS at T2 predicted depression at T3. Yet, depression at T1 also predicted PTSS at T2 and depression at T2 also predicted PTSS at T3. Importantly, inclusion of all variables in the model demonstrated that the path from PTSS symptoms in T3 to adult offsprings ST, although only marginally significant, was implicated in offsprings ST.

4. Discussion

In the current study we aimed to investigate the role of ex-POWs' PTSD status, trajectories, symptoms profile, and depression symptoms comorbidity in their adult offsprings ST. Our main results indicated that adult offspring of ex-POWs with PTSD at T3 reported more ST symptoms than adult offspring of ex-POWs without PTSD and adult offspring of controls. Ex-POWs' PTSD hyper-arousal symptom cluster at T3 predicted adult offsprings ST avoidance symptom cluster. Adult offspring of ex-POWs with chronic and delayed PTSD trajectories reported more ST symptoms than adult offspring of both the ex-POWs and the control group with resilient trajectories. Ex-POWs' PTSD and depression symptoms at T1, T2 and T3 mediated the link between war captivity (groups) and adult offsprings ST in T4.

Our results clearly show that forty years after the Yom Kippur War, ex-POWs' adult offspring are at risk for more ST symptoms in comparison to the control veterans' adult offspring. Furthermore, ex-POWs' adult offspring whose fathers suffer from PTSD are at a greater risk for ST symptoms. These results are in line with case studies (Rosenheck and Nathan, 1985), empirical studies (e.g., Ahmadzadeh and Malekian, 2004), literature reviews (Dekel and Goldblatt, 2008) and meta-analyses (Lambert et al., 2014) that exemplified the associations between parents' combat-related PTSS and children's psychological difficulties and behavioral problems. However, this is the first study to document this effect among adult offspring of ex-POWs, who reported on their own symptoms and were compared to a matched group of control veterans' adult offspring.

Importantly, our results show that it is not only the ex-POWs' current status of PTSD that affects their adult offsprings mental health, but also the PTSD status and trauma characteristics in the forms of chronic and delayed trajectory over the years. Taken together, over the years the fathers' PTSD serves both as a linear mediator between captivity and their adult offsprings ST and as a non-linear risk factor of chronic and delayed courses of the disorder. We would like to suggest that the severe, prolonged and interpersonal trauma of captivity ignites two parallel and interrelated processes. The first is intra-psychic in its nature and is reflected in the posttraumatic changes that ex-POWs undergo, and the second is interpersonal in its nature and is reflected in the deteriorated family relations and parenting practices. Both of these processes might put offspring at risk for their own ST symptomatology.

A possible example for this argument is our finding that, above and beyond the relation between fathers' PTSS clusters and adult offsprings ST symptom clusters, ex-POWs' PTSD hyper-arousal symptom cluster in T3 predicted adult offsprings ST avoidance symptom cluster in T4. At the intra-psychic level, hyper-arousal symptoms were found to be a platform that initiated the development of further PTSS, probably due to strong links to various neurological, neuro-chemical and hormonal roots (Solomon et al., 2009). However, at the inter-personal level, hyper-arousal symptoms were also positively related to anger and aggression among traumatized veterans (e.g., Taft et al., 2008) and lower parenting adjustment among ex-POWs (Zerach et al., 2012). Thus, it is possible that adult offsprings avoidance is both a symptom of the fathers'captivity and PTSD, and also an adaptive reaction to the

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behaviors of nervousness and aggression directed toward them and other family members by the ex-POW.

Our findings also show that depressive symptoms, on their own and co-morbid with PTSS, also predicted adult offsprings ST symptoms. Other studies, mainly among non-combat samples, revealed mixed findings. For example, among individuals who were exposed to the 9/11 terror attack, parental PTSD status, but not depression, was associated with the severity of the offsprings ST (Fairbrother et al., 2003). However, among 97 injury survivors, parental depression (but not PTSD) predicted offsprings ST (Zatzick, et al., 2006). It is possible that PTSD shares common variance with "negative" depressive symptoms. Hence, symptoms of active avoidance and emotional numbing that are known to be related to negative parenting practices and satisfaction (Ruscio et al., 2002) might explain the mediation of depression on the link between the parent's captivity and the adult offsprings ST. Moreover, a previous study among Israeli ex-POWs found that PTSD and depressive symptoms might be part of a common general traumatic stress construct (Ginsburg et al., 2012). Thus, it is possible that the mutual links between PTSD and depressive symptoms are actually a reflection of one factor that is responsible for the intergenerational transition of captivity trauma.

This study has several limitations. First, due to the attrition of participants between measurements, the sample may be somewhat selective. Second, the use of self-report measures, although very common in trauma studies, entails the risk of reporting bias. Future studies should make use of objective measures, such as observation of the fathers'actual functioning. Third, the lack of pre-combat assessment of fathers' PTSD limits our ability to infer causality. Fourth, our measurements did not cover the entire span of the 40 years since the war. Therefore, we were unable to monitor changes in the course of posttraumatic symptoms during the gap between the war and later measurement periods. Fifth, the low number of participants in our study might have hindered the possibility for path significance (e.g., Fig. 3), despite the actual association. Future studies should replicate the proposed model with larger samples. Finally, it is possible that adult offsprings personality and mental health also affects their fathers' PTSD, and not only vice versa. Future studies should examine this assumption in prospective design studies.

To conclude, the findings of this study suggest that 40 years after the end of the war, ex-POW's adult offspring are at risk for ST symptoms. Furthermore, fathers' psychopathologies of PTSD and depressive symptoms were found to be a possible mechanism of the intergenerational transmission of captivity trauma. The current study capitalized on a long-term prospective study that allowed us to track not only current PTSD status, but also the changes of PTSD phenomena and its relation to adult offsprings ST symptoms. Our findings highlight the necessity to screen and treat ex-POWs'children who might be troubled by the negative impact of the captivity experiences and PTSD of their fathers, which may also be implicated in problematic family relations and maladaptive parenting practices.

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