

Depression and PTSD Co-Morbidity: What are We Missing?

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Abstract

Background: Posttraumatic stress disorder (PTSD) and depression comorbidity is highly common. Many hypotheses concerning this relation have been raised but the pertinent issues, including the wide clinical picture of this comorbidity, are still not clear. The current study aims to bridge these gaps.

Method: We assessed PTSD, depression and comorbid indicators including dissociation, somatization, self-destructive behavior and suicidality among Israeli Yom Kippur war veterans at three time points (N = 349, 287, 301).

Results: Dissociation, somatization, self-destructive behavior and suicidality were predicted separately by group (PTSD, depression and comorbidity) and time of measurement using ANOVA and Chi squared analyses. The 'comorbidity' group expressed significantly higher dissociation, somatization, self-destructive behavior and suicidality, revealing high vulnerability of this group. Somatization presented a curvilinear-like development, increasing between T1 and T2 and slightly declining at T3, especially among the 'comorbidity' group. Suicidality showed a constant increase along the three measurements, especially among the comorbidity group.

Conclusions: A PTSD/depression comorbidity is both highly prevalent and long lasting and is often expressed concurrently with other related symptomatology, which causes further suffering and makes it more complicated for treatment. Implications for policy makers are briefly discussed.

Keywords: Depression; PTSD; Dissociation; Somatization; Self-destructive behaviors; Suicidality; Ex-POWs

Introduction

The relationship between posttraumatic stress disorder (PTSD) and co-morbid disorders has been a focus of trauma research over the past 20 years. Co-morbid depression has been documented at exceptionally high rates among numerous populations including American and Israeli veterans with PTSD, reaching 67%-82% [1,2]. Similarly, the prevalence of co-morbid PTSD among veterans suffering from depression is also high, ranging between 36%-73% [1-3]. Although this comorbidity has been studied both empirically and theoretically, the relationship between these disorders, including its causal effects and symptomatology, is still not fully clear [4].

War captivity is a pathogenic experience as it involves extreme situations in which one's life is being threatened in a prolonged and repeated nature. Ex-POWs suffer from a wide range of psychiatric disorders, most common among them being PTSD and depression. Comorbidity of these disorders was found to be highly prevalent among ex-POWs when compared to other combatants in a previous study conducted by our research group [8].

Recent studies suggest that PTSD and depression represent a range of symptomatic expressions of the same latent traumatic response, calling into question the theoretical distinction between PTSD and mood/anxiety disorders among individuals who have experienced trauma. Neurobiological studies show that the comorbidity of PTSD

and depression differs clinically and biologically from each of these disorders, supporting the need to better explain and elucidate the PTSD-depression comorbidity [10].

Most studies of PTSD and depression are cross-sectional in nature and relatively few studies have explored the longitudinal relationship between these two disorders. Studies conducted among both U.S. Gulf War veterans [11] and Israeli ex-prisoners of war (ex-POWs), [8] showed a bi-directional connection between the disorders, with PTSD predicting depression and vice versa. Similarly, Breslau et al. [12] found that the probability for depression to precede PTSD was as strong as the probability of PTSD to precede depression among young women. Recently, Ginzburg, Solomon, & Ein-Dor [1] found that, among Israeli 1982 Lebanon War veterans, early onset of PTSD predicts later depression and later PTSD/depression comorbidity, but not the other way around. These studies' inconsistent results concerning the course of these disorders further highlight the complexity of the connection between the two disorders and call into question their distinction as separate constructs.

Research suggests that one of the major concerns that comorbidity entails is its clinical severity and subsequent impairment. The outcome and correlates of the PTSD/depression comorbidity seem to differ from individual disorders, with the comorbid disorder presenting worse psychosocial functioning [1] higher suicidality, and poorer prognosis than PTSD alone [3,13].

PTSD and depression, especially when observed after a traumatic experience, both indicate a decreased ability to regulate and modify

affect, thoughts and experiences [14-16], which can be manifested in somatization and dissociation [17,18] as well as a tendency for self-destructiveness [19]. To date, most studies connecting trauma and depression to these related outcomes were conducted with early trauma samples such as childhood maltreatment and sexual abuse. The literature on the relationship between comorbid PTSD/depression and the above outcomes and correlates is limited among adults, especially with respect to individuals who underwent severe trauma in adulthood, such as the military population.

The high levels of PTSD/depression joint expression and related outcomes and correlates over time merit further attention. The goal of the present study is to cast light on the characteristics of PTSD/depression and their comorbidity, 35 years after exposure to a war experience. Specifically, the focus will be on the relationship between these disorders over time with affect regulation and self-destructive manifestations (i.e., dissociation, somatization, self-destructive behaviors and suicidality) in two different veterans groups; ex-POWs and controls. This will be employed using a prospective design which will allow for assessments of the temporal changes in clinical manifestations.

Method

Participants and procedure

A cohort of Israeli male veterans who participated in the 1973 Yom Kippur War were followed over 17 years with assessment at three time points: 1991 (T1), 2001 (T2), and 2008 (T3). Following approval from both the Israel Defense Forces (IDF) and Tel Aviv University review boards, lists of potential participants from the IDF computerized data bank were composed. Half of the samples were ex-POWs and the other half comprised of combatants who were not ex-POW but were selected on the basis of their similarity to the ex-POWs on relevant military and personal variables such as age, combat exposure, deployment region and rank. We phoned those participants and, after explaining the purpose of the study, asked them to take part in the assessments. The questionnaire packet was administered in their homes or in another location of their choice. Informed consent was obtained from all participants. In T1 349 veterans participated, in T2 287 participated (51 could not be located/refused, 5 had died, and 6 could not participate due to mental deterioration), and 301 took part in T3 (22 could not be located/refused, 20 had died, and 6 could not participate due to mental deterioration). No significant differences were found between those who participated in the follow-up assessments in regards to initial military rank, age, and education.

Measurements

PTSD was assessed using the PTSD *Inventory* [20], a self-report Likert-type [0-4] questionnaire containing 17 PTSD symptoms listed in the DSM-IV-TR [21]. Participants were asked to rate how often they suffered from each symptom in the previous month. The number of positively endorsed symptoms was calculated by counting the items in which the respondents answered '3' or '4'. This symptom count was used to operationalize PTSD as a binary (yes/no) measure of PTSD DSM-IV-TR symptom criteria - participants were classified as having posttraumatic symptoms (PTSS) if they endorsed at least one intrusive symptom, three avoidant symptoms, and two hyper arousal symptoms. The PTSD Inventory has high reliability and convergent validity [22]. In this study the reliability of the PTSD total score was high ($\alpha=.9$) as well as of its subscales (.84 to .92).

Depression and somatization were assessed using the Symptom Checklist-90's (SCL-90, [23]) depression and somatization subscales. Based on norms for psychiatric outpatients [23] scores above .73 on the depression subscale were considered an indication of depressive symptoms, creating a dichotomous variable. In this study the reliability of the depression total subscale was high ($\alpha=.91$). Somatization was operationalized a continuous measurement and its reliability value was high ($\alpha=.92$).

Suicidal ideation was assessed using one item from the SCL-90 ('thoughts about ending your life').

Self-destructive behavior was assessed using the Revised Structured Interview for Disorders of Extreme Stress-NOS (SIDES-R; for full details regarding psychometric properties of this instrument see [24]). The questionnaire is based on a translation and adaptation of the structured interview into a self-report questionnaire [25]. Participants were asked to note if they had undergone the mentioned experience in the previous month (yes vs. no). The three items were: a) did you have any accidents or near accidents, such as kitchen accidents or car accidents?; b) did you do anything dangerous? Or, did you not protect yourself when you could have been hurt?; c) did you try to hurt yourself on purpose because you were upset?. We created a binary variable in which 'yes' represented engagement in at least one of the described behaviors and 'no' represented not doing so.

Dissociation was assessed using the Dissociative Experiences Scale [DES, 26]. This 28-item self-report measure taps the frequency of dissociative experiences (e.g., loss of awareness of one's surroundings, amnesia, depersonalization, de-realization, absorption, imaginative involvement in daily life). Respondents were asked to indicate the degree to which they experienced each symptom on a scale ranging from 0 to 100. In accordance with Carlson [27], scores above 30 were deemed to suggest a dissociative disorder. Internal consistency in the current study was high ($\alpha=.87$).

Data analyses

In order to assess the course of affect regulation and self-destructive symptoms among the two groups of veterans (ex-POWs and controls) and their relationship to depression/PTSD/comorbid symptomatology, we conducted several analyses. First, we formed four 'clinical groups' based on PTSS and depressive symptoms at T3: the 'comorbidity' group included 90 participants whose scores exceeded the cutoff point for both depressive symptoms and PTSS; the 'PTSS' group included 16 participants who scored higher than the diagnostic cut-off for apparent PTSD but not for depressive symptoms; and the 'depressive symptoms' group included 46 participants whose scores exceeded the diagnostic cut-off for apparent depression but not for PTSS. The 'no PTSS and no depressive symptoms' group included 135 participants who scored lower than the diagnostic cut-off for both PTSS and depressive symptoms. In the next step, we conducted two repeated measures ANOVA analyses with suicidality and somatization as the outcome variables and with 'clinical' groups, study groups (ex-POWs vs. controls) and time of measurement as factors. Since, dissociation was measured only at T3 we conducted Univariate analysis with the two groups' variables as factors. In the last step we conducted two chi square analyses with self-destructive dichotomous variable in one row and the groups (psychopathology/veterans group) in the second row.

Results

In **Table 1** we present distribution of participants in each type of 'clinical' group (comorbidity, PTSD, depressive symptoms, no PTSD and no depressive symptoms) at T3, separated for ex-POWs and controls. As it can be seen in the table, the majority of the ex-POWs

(50.6%) exceeded the cutoff point for comorbid symptomatology and constituted 95% of this group. Means and standard deviation of the study variables in each of the four 'clinical' groups is presented in **Table 2**.

Comorbidity		PTSD		Depression		None	
Ex-POW	Controls	Ex-POW	Controls	Ex-POW	Controls	Ex-POW	Controls
90 (31.4%)		16 (5.6%)		46 (16%)		135 (47%)	
86 (50.6%)	4 (3.4%)	15 (8.8%)	1(9%)	28 (16.5%)	18 (15.4%)	41 (24.1%)	94 (80.3%)

Table 1: Distribution of participants in the four 'clinical' groups, separated for ex-POWs and controls, in Time 3.

Variables	Comorbidity (N=90)		PTSD (N=16)		Depression (N=46)		No symptomatology (N=135)	
	M	Sd	M	Sd	M	Sd	M	Sd
Somatization								
T1	.59	.73	.63	.37	.37	.47	.39	.04
T2	1.8	1.02	1.13	1.19	.97	.71	.95	.08
T3	1.63	1	.48	.55	.68	.56	.78	.07
Dissociation	3.64	1.66	1.73	.39	2.34	1.06	1.66	.1
Suicidality								
T1	.06	.31	.00	.00	.14	.35	.84	.09
T2	.71	1.17	.33	.70	.30	.63	1.3	.11
T3	1.53	1.39	.46	1.12	.67	.94	1.65	.11
	Frequencies		Frequencies		Frequencies		Frequencies	
Self-destructive Behavior	22 (24%)		0		6 (13%)		4 (5.2%)	

Table 2: Means and Standard Deviation by the 'Clinical' Groups

Somatization

In the first ANOVA mixed-model analysis we compared somatization prospectively among the four 'clinical' groups and captivity (ex-POWs and controls) between subjects' factor, and time of measurement (T1, T2, T3) - within subject's factor. In this analysis we found a significant main effect for 'clinical' group ($F(3, 143) = 16.53, p < .001, \text{Partial } \eta^2 = .26$), with the 'comorbidity' group ($M=1.45, SE=0.15$), presenting a significantly higher somatization level than the 'PTSD' group ($M=0.48, SE=0.27$) and the 'no PTSD and no depressive symptoms' group ($M=0.3, SE=0.07$). Moreover, the 'depressive symptoms' group reported significantly higher somatization compared to the 'no PTSD and no depressive symptoms' group. Ex-POWs did not significantly differ from controls ($F(1,143)=.32, p=.5, \text{Partial } \eta^2 = .00$) and no interaction was observed between captivity and 'clinical' variables ($F(3,143)=1.32, p=.27, \text{Partial } \eta^2 = .03$). In addition, the analysis revealed a main effect for time ($F(2, 286) = 14.41, p < .001, \text{Partial } \eta^2 = .09$). T-tests using the Bonferroni correction revealed statistically significant differences in somatization level among each of the three waves: the mean level of somatization was lowest at T1 ($M =$

$.53, SE = .10$), followed by T3 ($M = .94, SE = .12$), and highest at T2 ($M = 1.29, SE = .15$). Finally, the analysis revealed a significant two-way interaction between time and 'Clinical' group ($F(6, 286) = 7.09, p < .001, \text{Partial } \eta^2 = .13$). These findings suggest that the difference between T1 and T2 was significantly higher in the 'comorbidity' group (Cohen's $d = 1.14$) than in the 'PTSD' (Cohen's $d = .58$) and 'depressive symptoms' (Cohen's $d = .57$) and the 'no PTSD and no depressive symptoms' (Cohen's $d = .14$) groups. The difference between T2 and T3 was higher in the 'PTSD' group (Cohen's $d = .69$) than in the 'comorbidity' group (Cohen's $d = .22$) and higher in both groups than in the 'depressive symptoms' (Cohen's $d = .16$) and 'no PTSD and no depressive symptoms' (Cohen's $d = .09$) groups. Notably, the analyses revealed three-way interaction between time, captivity and 'clinical' group in predicting somatization, ($F(6,286)=2.9, p=.009, \text{Partial } \eta^2 = .06$). Post Hoc analyses revealed that among the 'comorbidity', 'PTSD' and 'depressive symptoms' groups, no two-way interaction between time and captivity was revealed. This interaction was found only among the 'no PTSD and no depressive symptoms' group, with the ex-POWs reporting higher somatization level at T2 ($M=.38, SE=.09$) and

T3 (M=.43, SE=.09) compared to T1 (M=.15, SE=.06), while the control group demonstrated no differences between times of measurement (T1: M=.24, SE=.03; T2: M=.35, SE=.06; T3: M=.21, SE=.05).

Suicidality

In the second ANOVA analysis, we compared suicidality prospectively amongst the four groups with time of measurement (T1, T2 and T3) as factors. This analysis yielded a significant main effect for 'clinical' group ($F(3, 141) = 5.7, p=.001, \text{Partial } \eta^2 = .11$). The 'comorbidity' group (M=0.54, SE=.12) demonstrated a significantly higher suicidality level than the 'depressive symptoms' (M=.22, SE=0.09), 'PTSS' (M=0.06, SE=0.22) and 'no PTSS and no depressive symptoms' (M=.02, SE=0.5) groups. No main effect for captivity ($F(1,141)=.01, p=.93, \text{Partial } \eta^2 = .00$) and no interaction effect between symptomatology group and captivity ($F(3,141)=.04, p=.99, \text{Partial } \eta^2 = .00$) were revealed. Additionally, a non-significant trend was found for time ($F(2, 282) = 2.64, p=.07, \text{Partial } \eta^2 = .02$). At T1 (M=.05, SE=.24) the lowest suicidality level was measured, followed by T2 (M=.27, SE=.74) and T3 (M=.33, SE=.84). Moreover, the analysis revealed a significant interaction between time measurement and symptomatology group ($F(6, 282) = 2.6, p=.02, \text{Partial } \eta^2 = .05$). Post Hoc tests suggest that in the 'comorbidity' group, T1 (M=1.16, SE=.18) was significantly lower than T2 (M=2.33, SE=.17) and T3 (M=2.73, SE=.14). Among the 'depressive symptoms' group, it was also revealed that T1 (M=.73, SE=.2) was significantly lower than T3 (M=1.59, SE=.22). Among the 'PTSS' and the 'no PTSS and no depressive symptoms' groups, no significant differences were found between times of measurement. No interactions were found between time and captivity ($F(2,282)=1.24, p=.29, \text{Partial } \eta^2 = .01$) and there was no three-way interaction ($F(6,282)=1.1, p=.36, \text{Partial } \eta^2 = .02$).

Dissociation

In order to assess the relationship between the four 'clinical' groups, study groups and dissociation, we used a one-way ANOVA analysis (dissociation was measured only at T3). Results revealed a significant main effect for 'clinical' group ($F(3, 271) = 23.11, p<.001, \text{Partial } \eta^2 = .2$). A post hoc T-test using Bonferroni correction revealed a significantly higher dissociation level in the 'comorbidity' group (M =3.64, SE =1.66) than the 'PTSS' (M =1.74, SE =.39), 'depressive symptoms' (M =2.34, SE =1.07) and 'no PTSS and no depressive symptoms' (M =1.62, SE =.53) groups. The 'depressive symptoms' group reported a higher dissociation level than the 'no PTSS and no depressive symptoms' group. No main effect for captivity ($F(1,271)=.00, p=.98, \text{Partial } \eta^2 = .00$) and no interaction between 'clinical' group and captivity ($F(3,271)=1.29, p=.28, \text{Partial } \eta^2 = .01$) were revealed.

Self-destructive behavior

The association between self-destructive behavior (measured only at T3) and the four clinical groups using a Chi square analysis revealed a significant between groups difference (Chi square [3] = 21.06, $p<.001$), with the 'comorbidity' group presenting the highest frequencies, followed by the 'depressive symptoms' and the 'no PTSS and no depressive symptoms' groups. The 'PTSS' group reported non self-destructive behavior.

The second Chi square analysis, between self-destructive and study group (ex-POWs vs. controls) revealed a significant between groups

difference (Chi square [1] = 6.46, $p=.01$), with the ex-POWs group presenting higher frequencies.

Discussion

The relation between PTSD and depression has been the subject of significant empirical and theoretical research over the years. The present study aimed to expand the current knowledge on the long term effect of PTSD and depression among both ex-POWs and control combatants through the depiction of a clear picture of PTSD and depression-related symptoms, specifically those linked to emotional regulation and self-destructiveness (i.e., dissociation, somatization, self-destructive behaviors and suicidality).

Consistent with current literature, we found a high prevalence of comorbidity between PTSS and depressive symptoms, alongside other symptomatology that induces suffering, decades after the war. These results call into question current nosology that treats the disorders as independent. This study adds further evidence that long-term psychiatric sequelae to trauma may be expressed in a variety of forms. In doing so, our study results also contradict the conventional psychiatric (i.e., DSM) conceptualization that the posttraumatic response is restricted solely to the set of symptoms constituting a PTSD diagnosis. Current findings suggest that the present psychiatric definition is limited, ignoring central parts of the human response to trauma-related experience and thus missing its purpose.

Moreover, not only did most individuals in this sample endorse comorbidity, and not a single diagnosis, but this comorbidity showed greater complexity than depicted by PTSD or depression alone. The high prevalence of dissociation, somatization, self-destructive behavior and suicidality among the 'comorbidity' group seems to represent a change in the affect regulation mechanism and a tendency to engage in self-destructive behaviors that resemble complex PTSD [28].

The ex-POWs group, who constitute almost all the 'comorbidity' group, shows not only more severe disorder but also a complicated clinical picture. This seems to show that the type of traumatic experience a person goes through has a formative effect and supports the existent of complex PTSD that as known, is still controversial.

Complex PTSD refers to a range of symptoms, including disturbance in interpersonal and affective self-regulatory capacities that evolve after prolonged and repeated traumatic events. These symptoms and their connection to trauma related experiences have been studied mostly among individuals who were traumatized during childhood and are understood as evolving from over-activation and deactivation of interpersonal and affective states during the crucial period of development [28].

The current study's sample included Israeli combatants who had been selected to serve in a combat unit via psychological evaluation and psychometric tests. This specification does not counter-indicate possible early trauma or psychopathology, but these individuals constitute a selective and healthy group. This point further emphasizes the wide and vicarious synergistic effect of trauma, especially among those who develop the comorbid disorder.

The course of symptoms related to affect regulation and self-destructiveness was tested in this study through the somatization and suicidality variables. Results showed a consistent increase in suicidality over time, especially among the 'comorbidity' group. As far as we know, this is one of the sole studies that assessed the longitudinal effect of war related trauma on suicidality, pointing to the high risk of these

individuals and the need for suicidal risk assessment when presenting with trauma history. These results may also explain in part the elevated levels of suicidality in the U.S. army during the conflict in Afghanistan and Iraq [29].

Somatization increased in time up until a point where it slightly declined, especially among the groups that reported having PTSS. It is important to take into consideration that the second measurement took place during the Palestinian Intifada [2001], which may have influenced the severity of somatization symptoms during this measurement wave and could explain the curvilinear-like pattern in somatization. This is especially salient as somatization is often viewed as an immediate response to acute stress. The longitudinal effect of trauma symptoms on somatization has also been documented in other studies [30-32], and points to an aggravated tendency of bodily expression of emotional stress among these individuals.

Moreover, we found a three-way interaction between 'clinical' group, study group (ex-POWs vs. controls) and time when predicting somatization, such that only among the individuals in the ex-POWs group who did not reach our cut off point for neither PTSS nor depressive symptoms, somatization increased with time. It may be that the physical and emotional torture that the ex-POWs went through is expressed through the body, as the traumatic experience leaves traces on the body through different biological mechanisms, especially in a prolonged and repetitive trauma, as in the case of ex-POWs [33]. This interesting result further emphasizes our point, suggesting that the effect of trauma comes in many different manifestations. We may assume that this result is specifically applicable to men, as the expression of an explicit emotionality is less legitimate than the bodily expression.

These results are also in line with other studies, including findings in a sample of veterans treated in Veterans Affairs (VA) clinics pointing to a worse prognosis for the 'comorbidity' group [3] and findings, using VA administrative data, that patients who present the comorbid disorder use more mental health treatments and antidepressant medications and have higher mental health care costs than depressed patients without PTSD [34].

Study limitations

Several limitations of this study should be noted. First, the psychopathology groups were comprised based on self-report questionnaire and not on clinical interviews. This may have biased the results. Second, self-destructive behavior was assessed using three questions from the SIDE-R questionnaire and suicidality was based on one question from the SCL-90 and not on a targeted questionnaire designed to directly address self-destructive behavior or suicidality. Nevertheless, the use of three questions of the SIDE-R' self-destructive behavior [35] as well as one item as an indicator of suicidality have been previously used (e.g., [36]) and the latter was recently found to be a valid approach [37].

Conclusions and Implications

Co-occurrence of PTSD and depression is highly prevalent, expressing rapidly after the traumatic event. According to the present study's results, the comorbidity seems to have a worrisome chronic course continuing decades after the war. Related affect regulation and self-destructive symptoms (i.e., dissociation, somatization, self-destructive behavior and suicidality) are significantly higher among patients with a comorbidity diagnosis, yet these important sequelae

may be lost in the current nosology. Depression is not explicitly anchored to a traumatic event, therefore symptom checklists and other diagnostic tools are not anchored in this manner, which may affect a patient's response [38]. Classification systems should consider the growing evidence in the literature, including the current study, concerning the wider picture of human experience and symptoms after exposure to trauma.

Moreover, the complex time dynamics and high rate of comorbidity between PTSD, depression and other psychopathologies shown in this study suggests that the current emphasis in policy and clinical practice that considers PTSD as the main traumatic response may be short-sighted. Clinicians must regularly revisit all possible symptomatic and temporal expressions and dynamically update patients' diagnoses. Therapy for these individuals has to take into consideration the experience unique to each patient, which may be comprised of a larger range of symptoms than expected. These co-occurring symptoms may have concrete implications on the therapy plan (e.g., [39]). Current diagnostic methods and tools may easily miss the comorbidity, especially given findings that it tends to present years after the initial PTSD diagnosis.

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