Delayed-onset PTSD among war veterans: the role of life events throughout the life cycle

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Abstract

**Background** The underlying mechanisms of delayed-onset PTSD are yet to be understood. This study examines the role of stressful life events throughout the life cycle in delayed-onset PTSD following combat.

**Methods** 675 Israeli veterans from the 1982 Lebanon War, 369 with antecedent combat stress reaction (CSR) and 306 without CSR were assessed prospectively, 1, 2 and 20 years after the war. Veterans were divided into four groups, according to the time of first PTSD onset (first onset at 1983, 1984, and 2002 and no PTSD onset). They were assessed for post-, peri- and pre-traumatic life events, as well as military and socio-demographic characteristics.

**Results** Our findings indicate that shorter delays in PTSD onset were associated with a higher risk for CSR, a higher number of pre- and post-war life events, more severe subjective battle exposure, greater perceived danger during combat and a more stressful military position. CSR was found to be the most powerful predictor of PTSD onset. A recency effect was also found, with more recent life events proving to be stronger predictors of PTSD onset.

**Conclusions** First, our findings validate the existence of delayed-onset PTSD, as it was found among a substantial number of participants (16.5%). Second, post-, peri- and pre-traumatic life events are associated with the time of PTSD onset. Thus, practitioners and researchers are encouraged to examine not only the original trauma, but also the stressful experiences throughout the survivors’ life cycle. In particular, identification of antecedent CSR may help mental help professionals in targeting high-risk populations.

**Keywords** PTSD · Life events · War trauma · Epidemiology

Introduction

War is one of the most potent traumatic experiences known to man. The most common psychiatric disorder resulting from war-related trauma is post-traumatic stress disorder (PTSD). The course of PTSD over time is highly varied and complex [1]. In some instances, the onset of PTSD is delayed. According to DSM-IV-TR [2], when PTSD first appears 6 months or more after the traumatic experience, this is considered “delayed-onset PTSD” (DPTSD). This phenomenon was found among victims of various kinds of traumatic events [3–5]. The lengths of delay prior to first PTSD onset were found to be extremely varied, ranging from several months [6] to several decades [7]. Reported rates of DPTSD have also varied, ranging between 0 and 68% [8]. This wide variance may stem from differences in the type of trauma, definition of DPTSD, study design, assessment methods and length of follow-up period.

DPTSD is a highly relevant phenomenon at a time when life expectancy is gradually rising, thereby exposing more and more trauma survivors to the risk of late PTSD onset. This is particularly relevant with the aging of millions of war veterans from numerous wars around the globe (e.g. WW-II, Korea, Vietnam, Falkland and other wars). However, despite its importance and its intriguing and controversial nature, DPTSD is not fully understood. In fact, only a limited number of studies—mostly case studies...
and retrospective studies—have systematically examined this phenomenon.

Over the years, various psychodynamic [9–11], cognitive [12] and biological [13] explanations for DPTSD have been proposed. The present study aims to assess a contextual explanation, namely the implication and role of stressful life events occurring throughout the life cycle in DPTSD.

The term “life events” refers to major stressful and mostly negative or undesirable life events. The experience of stressful life events is often implicated in the onset of PTSD (e.g. [14]). Several models explaining the association between trauma and life events have been proposed. The “life change model” or “vulnerability perspective” views life events as stressors that upset homeostasis and exhaust coping resources required for readjustment, thereby rendering individuals vulnerable to psychopathology (e.g. [15]). A considerable body of empirical research supports this model (e.g. [16]). On the other hand, the “facilitator model” or “resilience perspective” contends that prior stressful experiences may lower the risk of psychological distress following subsequent exposure to stress (e.g. [17]). This may be explained by the concept of stress inoculation [18], according to which multiple stressful experiences increase familiarity with stress, lead to a decrease in the amount of perceived stress and enable more successful adaptation. This notion was also supported by several studies (e.g. [19]).

The associations between PTSD and life events occurring at different stages of life have been examined in several studies. Some studies have focused on the effect of pre-traumatic life events on the subsequent development of PTSD. For example, negative childhood experiences were implicated in subsequent PTSD among war veterans (e.g. 16, 20]). Peritraumatic experiences (i.e., stressful experiences that occur during a traumatic event) have also been consistently implicated in the severity of PTSD. In this regard, a distinction is often made between measures of objective and subjective exposure. While some studies revealed that both subjective and objective dimensions of trauma exposure are implicated in future mental distress (e.g. [21]), others have shown and concluded that survivors’ subjective assessment of the event is the critical determinant of outcome (e.g. [22]).

The role of life events occurring after the target traumatic event was also examined. Mehlum and Weisaeth [23], for example, have claimed that life events occurring post-trauma may augment the difficulties experienced by the survivors, thereby undermining their re-adjustment. This difficulty in re-adjustment might, in turn, facilitate the onset of PTSD. Similarly, other studies have showed that post-war life events are associated with a higher risk for PTSD (e.g. [24]). More specifically, stressful events occurring after the war may serve as reminders for the adverse experiences of combat, and therefore trigger emotional distress. Some of these stressful events are specifically related to the homecoming process. The homecoming soldier often has difficulty re-adjusting to civilian life [25], and therefore faces stressful experiences such as divorce, unemployment and physical disease. These events, in turn, may increase the risk of PTSD.

Finally, some have suggested (e.g. [26]) that recent life events are more potent pathogenic agents than remote life events. Others have suggested, however, that it is not the timing of the life event, but rather its content, which plays the most important role. According to the latter view, life events that are reminiscent of the original stressful experience might activate and unmask latent psychopathology [27].

Only a few studies, however, have examined the relative contribution of life events throughout the life cycle to DPTSD. Several researchers have concluded that stressful events following a traumatic event were implicated in DPTSD. For example, in a study conducted among New York City residents following the September 11 attacks [28], individuals with DPTSD reported a higher number of negative life events since the attacks compared to individuals with either a resilient, acute or remitted PTSD course. As for pre-traumatic events, it was found that adult patients with DPTSD were significantly more likely to report histories of childhood trauma than patients who have already reported PTSD at intake [29]. Combat experiences were also examined with regard to DPTSD. Gray et al. [30], for example, have found that the frequency in which one had experienced specific mission-related events predicted DPTSD among Somalia peacekeepers. Other studies, however, have failed to find a connection between DPTSD and life events. For example, in a study of MVA casualties [31], no difference was found between individuals with delayed and acute onsets of PTSD in the number of post-accident life events.

In contrast to the scarcity in empirical studies assessing the role of life events in DPTSD, there are many clinical reports demonstrating how stressful life events, mostly those occurring after the index trauma, may serve as “triggers” for this phenomenon (e.g. [32]). As these case studies indicate, some trauma casualties experience a long latency period during which they preserve good functioning and present little or no PTSD symptoms. However, following this period they may encounter an event (e.g. accident, death of a loved one, terror attack) that is actually or symbolically reminiscent of their traumatic event, and therefore brings it to the forefront again.

Nonetheless, there is still an ongoing debate regarding the question of whether, and to what degree, life events contribute to the onset of DPTSD. The present study aims...
to assess the implications of different types of life events and combat experiences occurring throughout the life cycle to the delayed onset of PTSD among both clinical and non-clinical groups of Israeli veterans from the 1982 Lebanon War. More specifically, we aim to answer the following questions: (a) is the number of life events (pre-, peri- and post-war life events) experienced by the veteran associated with the delay in PTSD onset? and, (b) what is the relative contribution of various types of life events (pre-, peri- and post-war events) to the delay in PTSD onset?

Method

Participants

The current study is part of a large-scale prospective study on the consequences of war among Israeli war veterans. Six hundred and seventy-five male veterans from the Lebanon War were assessed at three points in time: 1 year post-war (1983), 2 years post-war (1984) and 20 years post-war (2002) after the war. Three hundred and sixty-nine of these veterans (the clinical group) experienced psychic breakdown on the battlefield and were identified as suffering from combat stress reaction (CSR). This condition is characterized by various labile and polymorphic psychiatric and somatic symptoms, such as overwhelming anxiety or total withdrawal, and results in polarization of behaviors and seriously impaired battlefield functioning [33]. The remaining 306 veterans (non-clinical group) have participated in the same frontline combat units as the CSR group but did not show symptoms of CSR. For each CSR casualty, a matched control participant was randomly selected from among eligible soldiers who had similar socio-demographic characteristics (age, education and military rank and assignment).

For the purpose of this study, the 675 participants were divided into four study groups, according to the time of their first PTSD onset: (1) the “1983 PTSD” group consisted of soldiers who already had PTSD in the first assessment ($N = 299$), (2) the “1984 DPTSD” group consisted of soldiers who did not have PTSD in the first assessment, but did suffer from PTSD in the second assessment ($N = 58$), (3) the “2002 DPTSD” group consisted of veterans who did not have PTSD in the first and second assessments, but did suffer from the disorder in the third assessment ($N = 53$), (4) the “no PTSD” group consisted of soldiers who did not suffer from PTSD at any of the three assessments ($N = 265$). All participants have served in frontline units during the war.

Missing data were handled using the multiple imputation (MI) method. According to Rubin’s [34] multiple imputation procedure, each missing value is replaced by a set of plausible values that represent the uncertainty about the right value to impute.

Measures

Post-traumatic stress disorder inventory

The PTSD inventory [35] consists of 13 statements describing the DSM-III [36] symptoms of PTSD, as adapted for combat trauma. DSM-III was the standard used when the study commenced, and was therefore applied at all three measurements to allow comparison over time. The 13 items refer to three symptom categories, corresponding to the three PTSD symptom clusters presented in DSM-III: (1) re-experiencing of the trauma (2) numbing of responsiveness to or reduced involvement with the external world, (3) additional symptoms, including hyper-alertness, sleep disturbance and memory or concentration difficulties. Participants were asked to indicate on a four-point scale ranging from “never” to “very often” the frequency in which they experienced the described symptom within the past month. The number of positively endorsed symptoms was calculated by counting the positive responses on the 13 items. Diagnosis of PTSD was done when participants reported at least one intrusion symptom, one avoidant symptom and two hyper-arousal symptoms.

The clinical validity of the PTSD inventory was assessed by concurrent clinical interviews for a sample of 114 soldiers, 1 year after the Lebanon War. Clinicians experienced in diagnosis and treatment of PTSD assessed the existence of each symptom in the inventory. Concordance percentages calculated for each symptom ranged from 68.75 to 80%, indicating considerable agreement between the self-report and the clinical diagnosis of PTSD. The PTSD inventory was administered twice within a 1-week interval to 20 soldiers. Percentage of agreement was 82.3%, indicating high test–retest reliability [37]. At time 1, the PTSD inventory was also found to correlate with the Impact of Event Scale [38], a measure designed specifically to assess the impact of traumatic experiences. Cronbach alphas for the PTSD inventory in the present study were 0.89 at 1983, 0.88 at 1984 and 0.94 at 2002.

Life events before the war were assessed by means of a short questionnaire especially devised for the present study. Experienced researchers familiar with life in Israel reviewed a number of life events inventories and compiled a list of 36 life events. The 36 events cover six areas: family, work, health, education and personal and social life. Participants were asked to indicate whether they had experienced each event during the 3 months preceding the war. For each positively endorsed event, participants were asked (a) to indicate whether the event was desirable or undesirable, and (b) to assess the impact of the event in
terms of the amount of readjustment it required (from “not at all” to “very much”). The score was the sum of reported undesirable events. In order to validate this questionnaire, it was compared with data based on clinical interviews. The comparison yielded high congruency [39].

Negative childhood life events were assessed using a nine-item self-report scale especially devised for the present study. Participants were asked to indicate if they had experienced stressful life events before the age of 10 (yes/no). Eight items in the scale refer to specific stressful life events: death of a parent; death of a sibling; parents’ divorce; prolonged illness in the family; significant financial difficulties or unemployment of a parent; severe accident, illness or injury experienced by the respondent; sibling’s severe illness or handicap; domestic violence. The ninth item is an open question, allowing participants to note any other stressful childhood life event that was not included in the previous categories. The sum of childhood life events was used for analyses.

Subjective combat exposure was assessed using the following two questions: (1) “to what degree do you believe the experiences you had during the war were dangerous and threatening?”. Responses were given on a five-point scale ranging from “not at all” to “very much”. This item is similar to that used by Green, Grace, and Gleser [40]. (2) Participants were also asked the following questions: “the battles in which I had participated were: “difficult” = 1, “average” = 2, “easy” = 3, “did not participate” = 4.

Objective battle exposure was assessed using the following question: “what role did you serve during combats: an active fighting role, an assisting role or a service role?”. Life events after the war were measured using Solomon and Flum’s [39] Life Events Questionnaire, which comprised 23 life events tapping four domains: family (e.g. divorce), work (e.g. dismissal), health (e.g. major disease), and personal events (e.g. accident). Participants were asked whether they had experienced any of the events since the war and whether they perceived the experienced events as positive or negative. The sum of negative life events after the war was used for analysis.

Socio-demographic characteristics

The veteran’s age, level of education, family status, income, father’s place of birth and military rank were assessed.

The variables examined in this study were assessed at different points in time. CSR was diagnosed during the 1982 Lebanon War; sociodemographics, life events before the war, and both subjective and objective risks during battle were assessed in 1983; negative postwar life events were measured in 1984 and 2002; negative childhood life events were assessed in 2002; PTSD symptoms were assessed at all three points in time.

Procedure

One and 2 years following their participation in the 1982 Lebanon War, participants were asked to report to the Headquarters of the Surgeon General to take part in this study. Participants filled out a battery of questionnaires in small groups. Data in the third wave (2002) were collected at the veterans’ homes. The participants’ consent was obtained and they were informed that the data would remain confidential and in no way influence their status in military or civilian life.

Results

Difference in socio-economical status (SES)

In this section we examined whether the four study groups differed in their SES. Specifically, we investigated whether the groups varied in their family status, military rank, economic status before the war, level of education, age and number of children. A series of Chi-square analysis of independence (using exact significance estimation to account for possible assumption violation when calculating \( \chi^2 \) analyses) revealed that the groups were not significantly different in their family status, \( \chi^2(3) = 3.51, p = 0.32 \), military rank, \( \chi^2(3) = 11.79, p = 0.07 \), economic status before the war, \( \chi^2(15) = 22.96, p = 0.09 \), or level of education, \( \chi^2(15) = 24.55, p = 0.06 \). Moreover, using multivariate analysis of variance (MANOVA) with group classification as the independent measure and age and number of children as the dependent measures, we found no significant differences among the groups in either age, \( F(3,671) = 2.15, p = 0.09, \eta^2 = 0.01 \), or number of children, \( F(3,671) = 1.27, p = 0.30, \eta^2 = 0.01 \). Means and standard deviations are presented in Table 1.

Differences in negative life events before the war

In this section we explored whether (a) the 1983 PTSD, 1984 DPTSD, 2002 DPTSD and no-PTSD groups differed in number of childhood negative events, and (b) whether they differed in the number of negative events in adulthood before the war. To this end, we conducted a MANOVA with study group as the independent variable, and number of negative events in childhood and number of negative events in adulthood before the war as the dependent variables. Due to heterogeneity of variance we used the Welch correction followed by the Tamhane post-hoc analysis. Means and standard deviations are presented in Table 1.
The analysis revealed significant differences in the number of negative events in childhood, $F(3,143.30) = 3.16$, $p < 0.05$. Results show that participants of the no-PTSD group reported less negative events in childhood than participants of the 1983 PTSD and 1984 DPTSD groups. All other effects were non-significant.

Likewise, the analysis revealed significant differences among study groups in the number of negative events in adulthood before the war, $F(3,149.77) = 3.77$, $p < 0.05$. Participants of the no-PTSD group endorsed less negative events prior to the war than participants of the 1983 PTSD group. All other effects were non-significant.

Differences in war measures

In this section we explored whether the 1983 PTSD, 1984 DPTSD, 2002 DPTSD and no-PTSD groups differed in (a) their perceived exposure to danger, (b) their subjective appraisal of battle severity, (c) their military role during the war (warriors, support and services); and (d) the probability of having CSR. To this end, we conducted a series of $\chi^2$ analysis of independence (using exact significance estimation to account for possible assumptions violation when calculating $\chi^2$). The results revealed that 64.5% of the participants in the 1983 PTSD group reported extreme exposure to danger, compared to 24.5% of the no-PTSD group, 35.8% of the 2002 DPTSD group and 41.4% of the 1984 DPTSD group, $\chi^2(12) = 120.06$, $p < 0.001$. Likewise, the analysis revealed that whereas 27.9% of the participants in the no-PTSD group reported extreme battles severity, 48.2% of the participants in the 1983 PTSD group reported extreme battles severity, $\chi^2(9) = 44.63$, $p < 0.001$. In comparison, 39.6% of the participants in the 2002 DPTSD group and 34.5% of the participants in the 1984 DPTSD group reported extreme battles severity.

In addition, the analysis revealed that 79.9% of the participants in the 1983 PTSD group were either warriors or supporters, compared to 63.8, 71.7 and 75.1% of the participants in the 1984 DPTSD, 2002 DPTSD and no PTSD groups, respectively, $\chi^2(3) = 7.93$, $p < 0.05$. Finally, the analysis revealed that whereas only 27.9% of the participants in the no-PTSD group were classified as CSRs, 79.3% of the participants in the 1983 PTSD group were classified as suffering from CSR, $\chi^2(3) = 151.72$, $p < 0.001$. In comparison, 58.6% of the participants in the 1984 DPTSD group, and 45.3% of the 2002 DPTSD group were classified as suffering from CSR.

Differences in negative life events after the war

In this section we explored whether the 1983 PTSD, 1984 DPTSD, 2002 DPTSD and no-PTSD groups differed in the number of negative events in adulthood, 2 and 20 years after the war. To this end, we conducted a MANOVA with group classification as the independent variable and number of negative events in adulthood 2 and 20 years after the war as the dependent variables. Due to heterogeneity of variance we used the Welch correction followed by the Tamhane post-hoc analysis. Means and standard deviations are presented in Table 1. The analysis revealed significant differences among the groups in the number of negative events both 2 and 20 years after the war, $F(3,146.31) = 26.92$, $p < 0.001$, and $F(3,140.28) = 23.73$, $p < 0.001$, respectively. Results demonstrated that both the 1983 PTSD and the 1984 DPTSD groups endorsed more negative events 2 years after the war than the 2002 DPTSD and no-PTSD groups. In addition, the no-PTSD group reported less negative events in adulthood 20 years after the war than the other three groups. No significant differences were found between the 1984 DPTSD, 2002 PTSD and 1983 PTSD groups.

Discriminant Function Analysis (DA)

To examine the relative contribution of each independent variable to PTSD status, we used DA. Specifically, we explored the relative contribution of the number of negative events in childhood, the number of negative events in childhood, the number of negative events in adulthood before the war, the number of negative events in adulthood 2 years after the war, and the number of negative events in adulthood 20 years after the war to the classification of the participants into the 1983 PTSD, 1984 DPTSD, 2002 DPTSD and no-PTSD groups.

Table 1 Means and standard deviations for SES and negative life events for the PTSD, delayed to 1984, delayed to 2002, and no-PTSD groups

<table>
<thead>
<tr>
<th></th>
<th>PTSD At 1984</th>
<th>PTSD At 2002</th>
<th>No-PTSD</th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Number of children (1983)</td>
<td>1.48</td>
<td>1.35</td>
<td>1.11</td>
<td>1.28</td>
<td>1.28</td>
<td>1.07</td>
<td>1.55</td>
</tr>
<tr>
<td>Age (1983)</td>
<td>29.65</td>
<td>5.90</td>
<td>27.88</td>
<td>4.78</td>
<td>28.42</td>
<td>5.87</td>
<td>29.68</td>
</tr>
<tr>
<td>NLE in childhood (2002)</td>
<td>0.50</td>
<td>0.65</td>
<td>0.58</td>
<td>0.73</td>
<td>0.49</td>
<td>0.67</td>
<td>0.38</td>
</tr>
<tr>
<td>NLE prior to the war (1983)</td>
<td>0.04</td>
<td>0.07</td>
<td>0.04</td>
<td>0.07</td>
<td>0.03</td>
<td>0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>NLE 2 years following the war (1984)</td>
<td>0.07</td>
<td>0.09</td>
<td>0.07</td>
<td>0.07</td>
<td>0.03</td>
<td>0.06</td>
<td>0.02</td>
</tr>
<tr>
<td>NLE 20 years following the war (2002)</td>
<td>0.09</td>
<td>0.13</td>
<td>0.08</td>
<td>0.12</td>
<td>0.09</td>
<td>0.09</td>
<td>0.03</td>
</tr>
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</table>

Years in parentheses refer to the time in which the variable was measured
adulthood prior to the war, CSR, subjective battle severity, exposure to danger, position during the war, and number of negative events in adulthood 2 and 20 years after the war. The analysis revealed one significant canonical discriminant function, which explained 95.2% of the variance among the study groups, Wilks’ $\lambda = 0.62$, $\chi^2(24) = 316.20, p < 0.001$. As expected, the 1983 PTSD, 1984 DPTSD, 2002 DPTSD and no-PTSD groups could be placed on one continuum, with the 1983 PTSD group on one end, followed by the 1984 DPTSD and 2002 DPTSD groups (in that order), and the no-PTSD group on the other end. Standardized canonical discriminant function coefficients are presented in Table 2. As can be seen in Table 2, CSR and exposure to danger were the most powerful predictors of PTSD status, with CSR making the highest contribution and the number of negative events in childhood making the lowest contribution to the explained variance among groups.

Discussion

The present study set out to explore the contribution of various types of stressful life events throughout the life cycle to DPTSD following combat. The contribution of negative life events before and after the war, combat exposure, military role and CSR was assessed. Overall, our findings indicate that veterans with a shorter delay in PTSD onset reported a higher number of pre- and post-war life events, more severe subjective battle exposure, encountering greater danger during combat, occupying a more stressful military position and higher rates of CSR. Finally, examination of the relative contribution of these stressful experiences showed CSR to be the most powerful predictor of PTSD onset, followed by subjective combat severity. The number of negative events in childhood was the weakest predictor of PTSD onset.

The findings of this study indicate that stressful experiences throughout the life cycle are associated with the delay in PTSD onset. In general, the results of this study show that the length of delay in PTSD onset is negatively associated with the number of stressful experiences one encountered in the course of one’s life. These results seem to support the “life change model”, which postulates that stressful experiences weaken one’s coping abilities and therefore increase psychological vulnerability. Our findings are also in line with previous studies (e.g. [28]), showing a positive connection between stressful life events and DPTSD.

An interesting finding of this study reveals a recency effect in the association between life events and DPTSD. When the predictive power of various life events throughout the life cycle was compared, it was found that more recent life events (i.e., 20 years after the war) made the strongest contribution to the variance in PTSD onset, followed by events occurring 2 years after the war, events that took place several months before the war, and finally childhood life events, which made the weakest contribution. Thus, it appears that the negative impact of stressful experiences decreases as time goes by. This finding is in line with previous studies (e.g. [26]). It also seems to support the general notion, mostly derived from case reports [32], that DPTSD is associated with recent external triggers. This finding may have several possible explanations. One explanation seems to be that a latent post-traumatic disorder often remains inactive until one encounters a stressful event which brings traumatic memories into the forefront again. This, in turn, may either exacerbate already existing PTSD symptoms or trigger the onset of a disorder that is previously inexistential. However, an opposite causal mechanism is also possible, in which individuals with recently diagnosed DPTSD experience more events as a result of their mental state. Alternatively, these individuals may retrospectively endorse events in light of their current state, in an effort to explain their predicament and gain a sense of meaning from their traumatic experience.

The findings of this study also indicated that peritraumatic experiences (i.e., CSR and subjective battle severity) made the largest relative contribution to DPTSD. CSR was previously found to be a marker for future PTSD [41]. It thus seems that when a soldier experiences psychic breakdown on the battlefield, he faces an increased risk for developing a more chronic post-traumatic disorder in the future [33]. The present study adds to these findings, as it shows that CSR may leave vulnerability to DPTSD and predicts not only the mere onset of PTSD, but also the time

### Table 2 Standardized canonical discriminant function coefficients for relative differences between PTSD, delayed to 1984, delayed to 2002, and no-PTSD groups

<table>
<thead>
<tr>
<th>Measure</th>
<th>$\beta$</th>
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<tbody>
<tr>
<td>NLE in childhood (2002)</td>
<td>-0.06</td>
</tr>
<tr>
<td>NLE prior to the war (1983)</td>
<td>-0.17</td>
</tr>
<tr>
<td>CSR (1982)</td>
<td>0.61</td>
</tr>
<tr>
<td>Exposure to danger (1983)</td>
<td>0.17</td>
</tr>
<tr>
<td>Subjective battle severity (1983)</td>
<td>0.48</td>
</tr>
<tr>
<td>Position during the war (1983)</td>
<td>-0.38</td>
</tr>
<tr>
<td>NLE 2 years following the war (1984)</td>
<td>-0.26</td>
</tr>
<tr>
<td>NLE 20 years following the war (2002)</td>
<td>-0.28</td>
</tr>
</tbody>
</table>

Higher values refer to heightened differences between classification groups. Positive values refer to higher values for the PTSD group than the no-PTSD group. Negative values refer to lower values for the PTSD group than the no-PTSD group. Years in parentheses refer to the time in which the variable was measured.
of onset. The time factor, added here perhaps for the first time in CSR research, seems to suggest a broader and more complex definition of vulnerability to trauma.

As noted, our findings also reveal a negative association between the level of combat exposure and the delay in PTSD onset. This finding is in line with other trauma studies showing a direct connection between the severity of trauma and the level of psychological distress experienced by the victim (e.g. [21]). According to the “dose–response” perspective, one’s distress as response to stressful experiences will increase as a function of one’s level of exposure to the stressor [42]. This perspective was confirmed in numerous studies of war-related PTSD (e.g. [43]). Our findings are also in line with studies of DPTSD. Several studies have shown that trauma casualties with delayed PTSD differed in trauma exposure from both casualties with chronic PTSD (e.g. [44]) and those with no PTSD [30]. This finding also relates to the heated debate regarding the A criterion for PTSD, included in the DSM. In recent years, some have raised doubts regarding the role of stressor characteristics as predictors of clinical outcomes (e.g. [45]). However, our study suggests that trauma severity may be a particularly powerful predictor of PTSD onset. Finally, in the present study, subjective battle exposure was a more powerful predictor of the time of PTSD onset than objective battle exposure. Thus, it seems that the soldier’s perception of battle severity is a more critical determinant of future PTSD onset than the specific role one had filled during the war. This finding is in line with other studies showing subjective exposure to trauma to be a more powerful predictor of PTSD than objective exposure (e.g. [46]).

The present study is methodologically unique, as it employs a relativistic approach to the study of DPTSD. Instead of defining the delay in onset according to its rigid DSM criteria, we chose to see the four study groups as representing a continuum of PTSD onsets over time. The study seems to indicate that while the 1983 PTSD group is clearly distinguished from the other groups, the two delayed onset groups (1984 and 2002) are much less easily distinguished from one another. Thus, it seems that the first year in delay is the crucial year, with further delays playing a less important role. This may point to the existence of a “critical period” for PTSD onset, after which changes in the timing of PTSD onset are less significant.

This study has several methodological limitations. First, it is based on self-report measures that although very common in trauma studies, still entail the risk of biased reporting and/or biased memory. Special consideration should be given to our definition and measurement of DPTSD. First, our first assessment occurred 1 year after the war, thereby exceeding the 6-month mark determined by the DSM. Second, because of the time lag between assessments, we have no way of knowing if veterans suffered from PTSD in between our examinations.

Despite these limitations, however, the present study has theoretical, clinical and methodological importance. First and foremost, our findings provide strong support to the existence of DPTSD. This phenomenon has been the subject of ongoing debate, and the results of this study seem to validate its existence among a considerable number of war veterans (16.5%). Second, the original methodology employed here enables us to conceptualize and assess PTSD onset at various points across time. The delay in PTSD onset does not seem to be an all-or-none phenomenon, but rather one that has differential lengths (e.g. delays of 1 year, 20 years etc.). To the best of our knowledge, this study also presents the most comprehensive examination of life events throughout the life cycle vis-à-vis DPTSD. This issue has special importance, as life events have long been known to serve as triggers for DPTSD. In the lack of an immediate temporal relationship between stressor and stress disorder, it is crucial to understand what experiences, other than the index trauma, may contribute to a delayed PTSD onset. This information may be of particular importance to practitioners, in their attempt to both predict PTSD and treat existing PTSD among their patients. Finally, this study reveals a strong connection between antecedent CSR and DPTSD. This finding may have important clinical implications, as it identifies a possible marker for a faster PTSD onset.

References


