



# Is the war really over? A 20-year longitudinal study on trajectories of suicidal ideation and posttraumatic stress symptoms following combat



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## ABSTRACT

Combat stress reaction (CSR) has widespread long-term consequences, including profound psychopathology in the form of posttraumatic stress disorder (PTSD). Studies have established the link between combat, PTSD, and suicidality. However, little is known about the temporal course of suicidal ideation (SI) in general, specifically among war veterans. We aimed to trace the trajectories of SI in the aftermath of war and to explore the role of CSR and PTSD in SI trajectories. Israeli veterans with CSR ( $n=164$ ) and a matched control group (NCSR,  $n=111$ ) were assessed, using self-report measures at three points over the course of 20 years. Veterans with CSR reported significantly higher levels of SI, compared to the NCSR group at all measurement points. Among veterans with CSR, SI increased 2 years after the war and then decreased 20 years following the war. This pattern was particularly characteristic of veterans with chronic PTSD. The results indicate that CSR is a strong predictor of subsequent PTSD and suicidality. These findings highlight the importance of prevention and treatment efforts for these traumatized veterans, who are at risk for suicide even 20 years after the end of the war.

## 1. Introduction

The intense pressures of war have long been recognized to produce both immediate (acute) and long-term (chronic and delayed) psychopathology (e.g., Kardiner, 1947). On the battlefield, the most common acute reaction is combat stress reaction (CSR), also termed battle shock and battle fatigue, among other designations (Isserlin et al., 2008). CSR comprises various polymorphic and labile psychiatric and somatic symptoms, with its diagnosis determined by soldiers' impaired functioning, as evaluated by clinicians on the battlefield. Symptoms characterizing this condition include paralyzing fear of death, emotional and physical numbness, withdrawal, severe depression, and impaired combat functioning (Kardiner, 1947; Solomon, 1993).

At the end of a war, the debilitating effects of CSR may abate, either spontaneously or by means of professional intervention (Solomon and Mikulincer, 2006). However, in other cases, CSR may have long-term psychological and physiological consequences. CSR often crystallizes into profound and prolonged psychopathological sequelae in the form of posttraumatic stress disorder (PTSD) (e.g., Solomon and Mikulincer, 2006). Recently, a plethora of research has been conducted on the adverse psychiatric effects of military service (e.g., Riviere et al., 2011), indicating high risk for suicide ideation and behavior (e.g., Stein et al.,

2010). However, little is known about long-term trajectories of suicidality after combat and, more specifically, about the predictors of these trajectories.

### 1.1. Suicidal ideation

Suicidal behavior is an important public health problem that results in significant morbidity and mortality worldwide (WHO, 2012). Suicide ideation (SI), comprising thoughts or plans about suicide, has been identified as one of the powerful predictors of completed suicide (Suominen et al., 2004).

A considerable body of research suggests that previous traumas in general and war-related traumas specifically are associated with an increased risk of suicidal behaviors (Jakupcak et al., 2009; Kemp and Bossarte, 2013). However, veterans' suicide rates reflect some variability in suicide behavior (e.g., Pietrzak et al., 2010; Hellmuth et al., 2012). Moreover, whereas some studies suggest that exposure to stress is a risk factor for SI (e.g., Dubow et al., 1989), others claim that the adjustment to stress, and not the exposure itself, predicts suicidal ideation (e.g., Warheit et al., 1996). Both CSR and PTSD are expressions of difficulties in adjustment to combat stress and may serve as predictors for SI. Surprisingly, the cross-sectional and longitudinal

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associations between CSR and SI have yet to be explored. The current study aimed to fill this gap.

### 1.2. PTSD and SI

Studies have repeatedly documented significant relationships between war-related PTSD and suicidal behaviors (e.g., Jakupcak et al., 2009). For example, Iraq and Afghanistan war veterans reporting symptoms of PTSD were more likely to report SI (Hellmuth et al., 2012) and a desire to harm themselves (Maguen et al., 2012), compared to veterans without PTSD.

Several longitudinal studies have shown that the course of PTSD fluctuates over time (Bonanno and Mancini, 2012). While some who develop PTSD following a war may recover, others who may have initially responded adaptively to combat stress develop delayed psychopathology, following an asymptomatic latency period (e.g., Dickstein et al., 2010). In other cases, PTSD takes a chronic course. Accordingly, research has identified PTSD trajectories, most commonly resilience, recovery, and chronic (Orcutt et al., 2004).

Whereas previous studies have showed that PTSD is associated with suicidal behaviors, to our knowledge there are no studies that have examined the association between different PTSD trajectories and suicidality.

### 1.3. Trajectories of SI

Similar to PTSD, SI may fluctuate over time. However, only scant longitudinal studies prospectively explored the temporal course of suicidal ideation. Recent studies have found that some psychiatric disorders, such as depression and anxiety, increased the risk for repeated SI over time (e.g., Soloff and Chiappetta, 2012). As most studies have relied on only two measurement points interpreting their results with regard to the long-term course of SI and its predictors is problematic. Moreover, to the best of our knowledge, no studies to date have examined predictors of the persistence of suicidality over time among veterans. Such information has importance for understanding the nature of suicidal behavior and for possible clinical implications of risk assessment and monitoring.

### 1.4. The current study

In this prospective study, we investigated the temporal course of SI over the course of 20years among combat veterans, with and without CSR. Specifically, we examined whether CSR, PTSD, and PTSD trajectories are implicated in SI trajectories among veterans. Three hypotheses are posited for the current study: 1) We expect CSR veterans to report higher rates of SI compared to veterans without CSR (NCSR), at all time measurements; 2) We expect different SI trajectories between these two groups; 3) We will explore the covariation between PTSD trajectories and SI trajectories, with the expectation of mutual simultaneous variation between PTSD and SI over time. As no previous studies have addressed CSR and PTSD with regard to SI trajectories, these hypotheses are exploratory.

## 2. Method

### 2.1. Participants

All participants were IDF male war veterans assessed at three time points: 1year (T1), 2-years (T2), and 20years (T3) following the 1982 Lebanon War. The data were based on the responses of participants who participated in all three assessments to assess the longitudinal trajectory of SI. Two groups of veterans were included:

a) CSR group: 164 Israeli soldiers who fought in the 1982 Lebanon War, identified by military mental health personnel as CSR

casualties. Inclusion criteria comprised four factors: 1) participation in frontline battles, 2) a referral for psychiatric intervention made by the soldier's battalion surgeon during the war, 3) a diagnosis of CSR given on the battlefield by clinicians trained and experienced in the diagnosis of combat-related reactions, and 4) no indication in the clinician's report of serious physical injury or other psychiatric disorders. Eligibility was determined by clinicians' diagnostic records made on the battlefield.

b) Comparison group: 111 soldiers who participated in combat in the same units as the CSR group, but were not diagnosed with CSR (NCSR). This sampling procedure was chosen to ensure similar exposure to the amount and type of objective stress. All soldiers in the CSR and comparison groups had undergone stringent physical and psychiatric screening prior to commencing their military service, and no indication of diagnosable premorbid symptoms was recorded in their medical files (for more details see Solomon and Mikulincer (2006)).

The soldiers' age in the first wave of measurement ranged from 18 to 37 (Mage=25.81, SDage=4.72, Medage=26). Sixteen percent of all participants completed only eight years of schooling, 27% partial high school, 39% completed high school, and 18% studied beyond high school. The subjects in both groups did not significantly differ in age, gender, education, military rank, and military assignment. Table 1 presents main socio-demographic characteristics in the two study groups. Men participating at all three time points did not significantly differ from those declining to participate at T2 or T3, in socio-demographic and military background, pre-military adjustment, intelligence, or mental and somatic health one year after the war.

### 2.2. Procedure

This research is part of a longitudinal study comprising Israeli male combat veterans from the 1982 Lebanon War, Israel's longest and most controversial conflict. It commenced in June 1982, with Israeli soldiers remaining in Lebanon, amid periodic flare-ups, until 2000. One and two years following their participation in this war, participants were asked to report to the Surgeon General's Headquarters to take part in this study. Participants completed a battery of questionnaires in small groups. Twenty years after the war, shortly after the Israeli Defense Forces (IDF) departed Lebanon, it was decided to approach participants again. In 2002, potential participants were contacted by telephone, and the aim of the current study was explained. In the CSR group, 323 veterans were located, and 286 of them (88.5%) agreed to participate in the current study. In the control group, 258 veterans were located, and 218 of them (84.55%) agreed to participate. Approval was obtained from both the IDF and the Tel Aviv University IRB committees. The participants' informed 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. For the 2002 (20-year) measurement, qualified research students, using the instruments from

**Table 1**  
Distribution of socio-demographic variables in the CSR and NCSR groups.

	CSR		Control		
	M	SD	M	SD	
Age (1983)	29.6	8.28	30.29	9.14	t (n=669)=0.92 p=0.35
	N	%	N	%	
Education (1983)					
Less than 12 years	297	80.2	243	80.7	
12 years and more	71	19.8	60	19.3	$\chi^2$ (2, n=671)=1.85, p=0.86
Family Status (1983)					
Single	108	29.7	91	30.5	
Married	253	69.5	203	68.1	
Divorced	3	0.8	4	1.3	$\chi^2$ (3, N=662)=5.03, p=0.77

the two previous measurement times, collected data at the veterans' homes.

### 2.3. Measures

PTSD was assessed using the PTSD-Inventory, a self-report instrument based on the DSM-III, the standard used when the study commenced. The PTSD-Inventory comprises 13 statements describing symptoms according to the DSM-III PTSD symptoms as adapted for war trauma: 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 Likert-type scale ranging from 1 (never) to 4 (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. Determination of PTSD was recorded when participants reported at least one intrusion symptom, one avoidant symptom, and two hyper-arousal symptoms. These criteria were employed at all three time points to enable comparison over time. Participants were asked to indicate whether they had experienced each of the described symptoms within the past month. The inventory has proven psychometric properties in terms of both high test-retest reliability and concurrent validity, compared with clinical diagnoses (Solomon, 1993). Cronbach's  $\alpha$  ranged between 0.89 and 0.92 in our sample.

Suicidal ideation was assessed using two items derived from the Symptom Checklist-90 (SCL-90; Derogatis, 1977). Participants were asked to indicate how frequently they experienced each of two symptoms over the last two weeks on a 6-point Likert-type distress scale, ranging from 0 (not at all) to 5 (very much). The two items were thoughts about ending your life and thoughts about death. Due to the strong correlation between the items ( $r=0.56$ ), we calculated the mean score of the two items as an SI index (see Desseilles et al. (2012) and Zerach et al. (2013)). Cronbach  $\alpha$  for this measure ranged from 0.59 to 0.67 in our sample.

Depressive symptoms were assessed using the depression subscale of the Symptom Checklist-90 (SCL-90, Derogatis and Cleary, 1977), which comprises 13 items. Participants were asked to indicate how frequently they experienced each symptom over the last two weeks on a 5-point Likert-type distress scale. We excluded from this depression index the item that taps suicidal ideation (thoughts about ending your life) which was one of the components of our SI measure.

## 3. Results

### 3.1. Trajectories of SI among veterans with and without CSR

We conducted a repeated-measure ANOVA to examine the long-term trajectories of SI. Two independent variables were included: study group (CSR vs. NCSR) as a between subjects' factor, and time of measurement (T1, T2, T3) as a within subjects' factor. We included the SCL-90 index of depression (at T1) as a covariate in this analysis. Fig. 1 presents the trajectories of SI in the two study groups. This analysis yielded significant main effects for group,  $F(1,272)=4.96$ ,  $p < 0.05$ , Partial  $\eta^2 = 0.02$ , and for time  $F(2,546)=8.54$ ,  $p < 0.001$ , Partial  $\eta^2 = 0.03$ . The CSR group ( $M=0.73$ ,  $SD=0.05$ ) demonstrated significantly higher SI than the NCSR group ( $M=0.26$ ,  $SD=0.06$ ). The highest levels of SI were at T2 ( $M = 0.66$ ,  $SD = 0.97$ ), followed by lower levels at T1 ( $M = 0.59$ ,  $SD=0.91$ ), and lowest at T3 ( $M=0.36$ ,  $SD=0.72$ ). Significant differences in SI between T1-T3 and T2-T3 were revealed in  $t$ -tests using the Bonferroni correction.

The analysis also revealed a significant two-way interaction between time and group,  $F(2,544) = 3.52$ ,  $p < 0.05$ , Partial  $\eta^2 = 0.013$ . In the CSR group, the mean of SI was highest at T2 ( $M=0.92$ ,  $SD=1.06$ ), followed by T1 ( $M=0.79$ ,  $SD=1.02$ ) and lowest at T3 ( $M=0.48$ ,

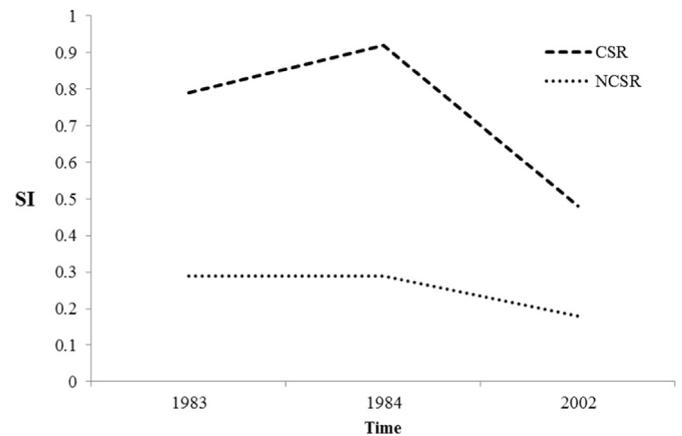


Fig. 1. Trajectories of SI in the two study groups (CSR vs. NCSR) (N=275).

$SD=0.85$ ). In the NCSR group, the SI mean did not differ between T1 ( $M=0.29$ ,  $SD=0.62$ ) and T2 ( $M=0.29$ ,  $SD=0.68$ ). Lower levels of SI were found at T3 when compared with T1 and T2 ( $M=0.018$ ,  $SD=0.46$ ). The difference between T1 and T2 was greater in the CSR group (Cohen's  $d = 0.13$ ) than in the NCSR group (Cohen's  $d=0$ ). Similarly, the difference between T2 and T3 was greater in the CSR group (Cohen's  $d = 0.46$ ,  $0.19$ , respectively).

### 3.2. Trajectories of SI and PTSD

We further divided the participants into four groups, according to the PTSD trajectories over time. We used latent-growth mixture modeling to identify long-term PTSD trajectories and their prevalence in veterans with and without CSR (for more details, see Karstoft et al. (2013)). Four PTSD trajectories emerged: resilient, recovery, delayed-onset, and chronic. Trajectories were similar across groups (CSR vs. NCSR), with differences in prevalence and intercepts (i.e., initial symptom level). We then assessed the course of SI among participants with different PTSD trajectories. We conducted repeated-measures ANOVA analyses with SI as the dependent variable and PTSD trajectory groups (i.e., the four sub-groups) as the independent variable. Here again, we included the SCL-90 index of depression (at T1) as a covariate in the analysis.

Due to the small sample size of the NCSR group, having sufficient data regarding PTSD and SI at all three time points ( $n=52$ ), we initially analysed the data only in the CSR group, then for both groups together. As the pattern of results was very similar, we based the main analyses on the full sample (i.e., The CSR and the NCSR groups) to maximize group sizes for the post-hoc analysis.

Fig. 2 presents the trajectories of SI in the four PTSD groups. This analysis yielded a significant main effect for PTSD group,  $F(3,169)$

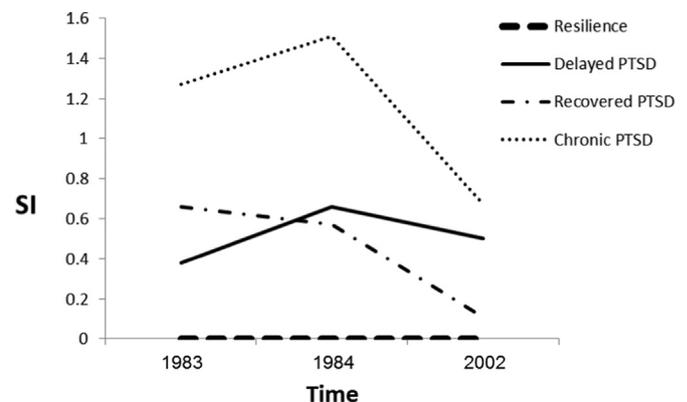


Fig. 2. Trajectories of SI in the four PTSD groups (chronic, delayed, recovered, resilience) ( $n=174$ ).

=5.86,  $p < 0.001$ , Partial  $\eta^2 = 0.09$ , and for time,  $F(2,338)=6.61$ ,  $p < 0.01$ , Partial  $\eta^2 = 0.04$ . The chronic group ( $M=1.15$ ,  $SD=0.07$ ) demonstrated a significantly higher SI than the three other groups: resilient ( $M=0.00$ ,  $SD=0.17$ ), delayed-onset ( $M=0.51$ ,  $SD=0.09$ ), and recovery ( $M=0.45$ ,  $SD=0.09$ ). The highest levels of SI appeared at T2 ( $M = 0.68$ ,  $SD = 0.08$ ), with lower levels appearing at T1 ( $M = 0.58$ ,  $SD = 0.08$ ), and the lowest levels appearing at T3 ( $M=0.33$ ,  $SD = 0.07$ ).

Finally, the analysis revealed a significant two-way interaction between time and PTSD group,  $F(6, 338) = 2.41$ ,  $p < 0.01$ , Partial  $\eta^2 = 0.04$ , indicating that changes in SI over time differed between PTSD trajectories. In the resilient group, no differences were found between the mean levels of SI at T1, T2, and T3. In the delayed-onset group, the mean levels of SI were lowest at T1 ( $M=0.39$ ,  $SD=0.76$ ), followed by T3 ( $M=0.50$ ,  $SD=0.91$ ), and highest at T2 ( $M=0.65$ ,  $SD=0.90$ ). In the recovery group, the mean levels of SI were highest at T1 ( $M=0.66$ ,  $SD=0.87$ ), followed by T2 ( $M=0.57$ ,  $SD=0.78$ ; Cohen's  $d = 0.11$ ), and lowest at T3 ( $M=0.11$ ,  $SD=0.36$ ). In the chronic group, the mean levels of SI were highest at T2 ( $M=1.51$ ,  $SD=1.10$ ), followed by T1 ( $M=1.27$ ,  $SD=1.06$ ), and lowest at T3 ( $M=0.67$ ,  $SD=0.87$ ).

These findings suggest that the difference between T1 and T2 was highest in the delayed-onset group (Cohen's  $d = 0.31$ ), followed by the chronic group (Cohen's  $d = 0.22$ ), the recovery group (Cohen's  $d = 0.11$ ), and lowest in the resilient group (Cohen's  $d = 0.00$ ). The difference between T2 and T3 was highest in the recovery group (Cohen's  $d = 0.75$ ), followed by chronic (Cohen's  $d = 0.53$ ), delayed-onset (Cohen's  $d = 0.16$ ), and lowest in the resilient group (Cohen's  $d = 0.00$ ). Fig. 2 presents the trajectories of SI and PTSD.

#### 4. Discussion

A large body of research has documented the short- and long-term impact of combat stress reaction on mental and physical health, and family, work, social, and military functioning (e.g., Solomon, 1993; Wagner and Jakupcak, 2012) and has confirmed a high risk of developing PTSD following CSR. Meta-analyses studies have shown that in general, PTSD increases the risk for suicidal ideation, suicide attempts, and completed suicides among war veterans (e.g., Pompili et al., 2013). However, little is known about the temporal course of SI in general, specifically among veterans. This study examined differences in SI between soldiers, with and without antecedent CSR, tracing the trajectories of SI in the aftermath of a war, and explored the implication of CSR and PTSD on SI trajectories.

Our findings revealed that veterans with CSR reported significantly higher levels of SI in comparison to the NCSR group at all three time points, reflecting a long-term effect of CSR on SI, even 20 years following the war. These findings are consistent with other studies identifying CSR as a powerful predictor of pathological outcomes (e.g., Solomon and Mikulincer, 2006; Solomon et al., 2009). Given that one of the acknowledged powerful predictors of suicide is SI (Suominen et al., 2004), it would appear that CSR could also be recognized as a risk factor for suicidality among military veterans, even many years after combat. The current findings may be explained by the negative emotions and cognitions tied to CSR. Theories and research have indicated that CSR reflects a breakdown during combat, in which the fear of death becomes salient and unbearable (Solomon, 1993; Wagner and Jakupcak, 2012). Lifton (1982) coined this experience of vivid memories and images of death and destruction as death imprint, which is difficult to dispel. The memories are associated with extreme death anxiety, in which the combatants' sense of invulnerability is destroyed, they anticipate further traumatic events, and begin to experience the world as unpredictable and lethal. These feelings are intensified by the death guilt of survival (Lifton, 1983). Thus, enduring harsh thoughts and feelings about death, dying, and guilt following the CSR may be a central reason for the differences between the CSR and NCSR groups in distress, PTSD, and SI.

More importantly, our results revealed a significant interaction

between time and group in predicting SI trajectories. The CSR group reflected changes in SI over time, with the highest levels at T2, whereas the NCSR group revealed only a small, non-significant decrease in SI from T2-T3. A closer look at the changes in SI, as it related to PTSD trajectories, revealed a noteworthy picture: A U curve in SI over time was observed among combat veterans having chronic and delayed-onset PTSD. In contrast, resilient veterans presented no SI in any of the study's three time points, and veterans who recovered from PTSD showed a decrease in SI over time. These findings are consistent with studies showing that PTSS are related to SI among traumatized veterans who were prisoners of war (Zerach et al., 2013; Levi-Belz et al., 2015).

The question arises as to why SI exacerbates at T2—two years postwar—among CSR combat veterans. Specifically, why is this pattern more prevalent among veterans with chronic and delayed PTSD?

Several possible explanations can be posited: First, as Horowitz and Solomon (1975) have suggested, once combat has ended, soldiers can let their guard down. They gradually abandon the denial and numbing mechanisms that facilitated their coping during combat. Thus, the overt expression of distress would occur only after time has passed since combat, which in turn may facilitate SI. This pattern may be more powerful for those encountering shocking experiences in battle, such as CSR. CSR in battle facilitates higher levels of distress and mental pain (e.g., Stander et al., 2014). Thus, weakening defense mechanisms generate more profound mental difficulties, thus contributing to higher SI.

Second, the soldiers' homecoming experiences, particularly the support levels that veterans receive from their social networks, may have a crucial role in determining their level of coping with post-war emotional difficulties. Several studies have shown that social resources are important protective factors in the face of traumatic experiences (Holeva et al., 2001; Horesh et al., 2013). Such support may serve important emotional, cognitive, and practical functions, often attenuating feelings of helplessness and loneliness. For example, the family environment established upon the veteran's return has often been found to be associated with his post-war adjustment and mental health (e.g., O'Donnell et al., 2006). We speculate that in the immediate time period after the war, veterans are likely to receive vast support and care from family members. However, with time the veterans' family, particularly their wives, returned to their daily life routine, and the support weakened (Horesh et al., 2013). This is especially true for veterans with chronic PTSD, who continually struggle with emotional and physical difficulties and require special attention and care. Indeed, upon examining the same sample, Solomon et al. (1991) found that two years after the war, veterans with chronic PTSD reported greater feelings of social alienation and disregard at their homecoming and less satisfaction with their social networks than did non-chronic PTSD veterans. These feelings of low support, even alienation, together with the continuation of symptoms, may explain the increase in SI in this group. Moreover, CSR often contributes to the secondary traumatization of their wives in the years following the war (e.g., Solomon et al., 1992). The consequence of this secondary traumatization is that some wives may struggle with their own emotional difficulties, with fewer resources available to them for supporting their partners. Furthermore, secondary traumatization may evoke marital difficulties and social alienation. These changes may provide an explanation for the increase in SI found in the CSR group.

It also may be that prior vulnerabilities of veterans who experience CSR become more salient in the aftermath of the war. Solomon et al. (1986) found that the CSR group was characterized by lower levels of social support and higher levels of loneliness before their combat experience. This characteristic leaves the veterans more vulnerable to further depletion of social resources. Therefore, we suggest that the gradual decrease in post-war support, possibly common to all war veterans, might evoke exacerbated distress and loneliness among

veterans with CSR, which, in turn, may induce SI.

Support and cohesion are also important factors in the broader national and social environment for the combatant's return. As the post-war period advances, there is typically a weakening of socially sanctioned legitimacy for intensive involvement with the war and memories of its battles. Society's unconscious voice prefers to leave these memories behind. In other words, it becomes less and less appropriate to remain concerned with war memories. However, these are precisely the concerns with which veterans with chronic and delayed PTSD struggled two years after the war. Thus, this may intensify feelings of guilt, which has been recognized as a facilitating factor for suicidality (Pompili et al., 2013) and result in feelings of abandonment and loneliness. The latter feelings reflect the thwarted belongingness experiences (Joiner et al., 2005), one of the two fundamental factors suggested by the interpersonal theory of suicide that generates suicide ideation (Van Orden et al., 2010).

In summary, the findings point to mutual simultaneous variations of PTSD and SI over time. This is not surprising, as both PTSD and SI are indicators of mental distress, and the literature has identified a strong link between PTSD and suicidality. However, it should be noted that in all groups (excepting the resilient group, which presented no SI in any of the assessment points), we found a decrease in SI from T2 to T3, 20 years after the war. It may be that shortly after the war, the pains and memories of the trauma were still a daily occurrence for the veterans and thus may have evoked SI, along with a desire to put an end to their suffering. However, many years after the war, it may be that the traces of the original trauma have slightly faded, thus reducing the distress through habituation or by adjustment to the symptoms.

This study has several methodological limitations. First, the use of self-report measures, although very common in trauma studies, may entail reporting bias. Second, the lack of pre-combat assessment of SI clearly undermines our ability to infer causality. Moreover, the long gap between T2 and T3 denotes an inability to know what occurred during the interim 16 years. Since we did not obtain detailed information on psychiatric interventions that participants may have received throughout the 20-year course of the study, we cannot account for the effect treatment could have had on the course of symptoms, especially at T3. Additionally, the measurement of SI was taken from larger measures (SCL-90, DESNOS), and not from a targeted questionnaire designed to directly address suicide ideation (Beck et al., 1979). Although the short measures consisting of few items are commonly used and have demonstrated moderate reliability, future studies should utilize structured clinical interviews to assess SI (e.g. C-SSRS, Posner et al., 2011). It is also worth noting that our findings refer to SI levels and thus cannot be generalized to suicidality-related phenomena, such as suicide attempts. Finally, the current study explored the links between SI, CSR and PTSD, but did not include other important correlates of suicidal ideation and suicidality, such as substance use and social support. These should be further explored in future studies to better understand suicidality in this unique population.

Despite these limitations, our findings have significant theoretical and clinical implications. First and foremost, our findings reveal that a psychological breakdown in battle such as CSR, may lead not only to mental distress but also to persistent experiences of SI, both in the short and long terms after the war. Thus, it is important that clinicians and researchers assess CSR among veterans, given its character as a risk factor for suicidality, a recognized marker for suicide attempts (Wilkinson et al., 2011). To the best of our knowledge, no other study has systematically examined the changes in SI over an extended time. Thus, our study revealed that, contrary to conventional wisdom, the risk for SI increased 2 years after the war, beyond the level presented immediately after the experience of battle. This highlights the importance of continuing emotional and behavioral support during the years after the war, as during these years veterans are more susceptible to suicidal thoughts. Interventions focused on veterans' social environment may help to deal with subsequent thoughts of suicide. As shown

here, veterans with chronic or delayed PTSD were more vulnerable to SI at T2. Thus, therapists who treat veterans with chronic or delayed PTSD symptoms should attend carefully to the subjective experience of distress and depression, as these veterans are more inclined to suicidality.

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