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## Obsessive Compulsive Symptoms Predict Posttraumatic Growth: A Longitudinal Study

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### ABSTRACT

Although posttraumatic growth (PTG) has received growing attention, the relationship between PTG and distress remains unclear. This longitudinal study examines the relationship between posttraumatic obsessive-compulsive (OC) symptoms and PTG. Israeli veterans were followed over 17 years using self-report questionnaires of OC symptoms, posttraumatic stress disorder (PTSD), and PTG. Hierarchical regression analyses demonstrated that OC symptoms predicted PTG, even when initial PTG levels and PTSD symptoms were controlled for in the combatants group. These preliminary findings suggest that OC symptoms may play an important role in facilitating psychological growth. Future research is warranted to explore the mechanisms responsible for this relationship.

### ARTICLE HISTORY

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### KEYWORDS

Obsessive-compulsive symptoms; posttraumatic growth; posttraumatic stress disorder

In recent years, rapidly growing attention has been directed toward investigating positive outcomes following traumatic events. Studies reveal that some individuals may experience positive psychological changes following exposure to trauma (see Zoellner & Maercker, 2006, for a review), most commonly referred to as posttraumatic growth (PTG; Tedeschi & Calhoun, 1996). Psychological growth has been observed across different aspects of survivors' lives, including perceptions of personal strength, intimate relationships, appreciation of life, new possibilities, and spirituality (Tedeschi & Calhoun, 2004).

An instrumental component in the development of PTG is the cognitive process of the traumatic event (Calhoun, Cann, Tedeschi, & McMillan, 2000). Ruminative thinking on the traumatic event may signify efforts to make sense of the trauma vis-à-vis the shattering of one's world assumptions following the trauma, which may ultimately facilitate growth.

The association between posttraumatic stress disorder (PTSD) symptoms implicated in the intrusive cognitive processing of the event and PTG received significant attention. Several studies demonstrated a positive association of

PTSD and PTG in different trauma samples, including veterans' samples exposed to combat and war captivity (Dekel, Ein-Dor, & Solomon, 2012).

There is a gap in the research about the relationship between PTG and other posttrauma psychiatric conditions manifested in cognitive processing, such as obsessive-compulsive disorder (OCD). Intrusive cognitive processing is the defining symptom of OCD. OCD is highly common following trauma and often co-occurs with PTSD (e.g., Pitman, 1993). Although OC symptoms, similar to PTSD symptoms, entail ruminative thinking and have negative effects on survivors (De Silva & Marks, 2001), there are no available studies that examined whether these symptoms may also foster subsequent growth.

Posttraumatic obsessive symptoms can manifest in trauma-specific intrusions (e.g., repeated thoughts about contamination following exposure to human flesh; Sasson et al., 2005) and therefore provide a consistent reminder of the trauma and a repeated mental revisit of the event, altogether possibly allowing for the ongoing reprocessing of the event. Moreover, individuals with OC symptoms are likely to endorse a ruminative response style (Wahl, Ertle, Bohne, Zurowski, & Korson, 2011), which in turn may amplify PTSD-related intrusive recollection of the trauma and facilitate growth. Intrusive thoughts may trigger the development of more deliberative thought and subsequently enhance growth.

In this longitudinal study of veterans of the Yom Kippur War, we examine the relationship between posttraumatic psychological growth and posttraumatic obsessive-compulsive symptoms. We focused our analysis on the prediction of whether OC symptoms predict PTG over time.

## Method

### Participants

The present study uses data from a larger study on the psychological implications of war (Dekel et al., 2012), following  $n$  veterans of the 1973 Yom Kippur War over three measurements: 18 (Time 1: 1991), 30 (Time 2: 2003), and 35 (Time 3: 2008) years after the war. Upon Israel Defense Forces (IDF) and Tel Aviv University Review Board's approval, 472 veterans were approached; among them, 346 voluntarily agreed to participate (T1), lending a 73.3% response rate (and 60.8% and 63.8% rates in T2 and T3, respectively). Out of those participating in T1, 287 took part in T2 and 301 in T3, lending response rates of 82% and 87%, respectively. All participants provided written consent forms. In T2, 53.30% ( $n = 121$ ) of the sample was classified as ex-prisoners of war (ex-POWs). These veterans were subjected to intense isolation, physical torture, and psychological stress. Ex-POWs did not differ from the veterans with regard to military rank, age, and education. The follow-up samples did not differ from the initial sample in demographic variables. All participants

were men, their mean age was 53.4 ( $SD = 4.40$ ) and they had 14 years on average ( $SD = 3.41$ ) of education.

### Measures

Posttraumatic growth was measured at T2 and T3 using the Posttraumatic Growth Inventory (PTGI; Tedeschi & Calhoun, 1996), listing 21 items anchored on the Yom Kippur War. Participants were asked to indicate on a 4-point scale the extent of change that occurred in their lives following the war. PTG total scores were calculated for each time point (Cronbach's alpha at .94, at both assessments).

Posttraumatic stress symptoms were measured at T1 and T2 using the PTSD Inventory (PTSDI; Dekel et al., 2012), listing 17 items anchored on the Yom Kippur War on a 4-point scale (1–4), according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev., DSM-IV-TR; American Psychiatric Association, 2000). The inventory is used with veterans and clinical populations and has good psychometric properties, including high convergent and concurrent validity. PTSD total symptom scores were calculated to assess symptom severity level for each time point (Cronbach's alpha ranged from .78 to .96 between assessments). In addition, individuals were classified according to their PTSD symptom status, that is, with or without "Probable PTSD" (endorsement of at least 1 intrusion, 3 avoidance, and 2 hyperarousal symptoms).

Obsessive-compulsive symptoms (OC) were measured at T1 and T2 using the commonly used Symptom Checklist-90 (SCL-90; Derogatis, 1977). Participants were asked to indicate how frequently they experienced each symptom during the last two weeks on a 5-point distress scale. The OC symptoms were not anchored on the war experience. Based on norms for psychiatric outpatients (Derogatis, 1977), scores equal or above 0.73 were considered as an indication for endorsement of OC symptoms (Dekel, Solomon, & Rozenstreich, 2013; Ginzburg, Ein-Dor, & Solomon, 2010). For each participant we calculated the average frequency of experiencing OC symptoms for each time point (Cronbach's alpha ranged from .88 to .93 between assessments).

### Data analysis

The magnitudes of associations between PTG and the severity of PTSD and OC symptoms were examined using a series of Pearson correlations. The contribution of OC symptoms to the prediction of PTG was examined using two separate hierarchical regression analyses. The first included predictors measured at T1 (1991) and PTG at T2 (2003), and in the second, predictors were measured at T2 and PTG at T3 (2008). In each regression, PTSD symptoms were entered in step 1 and OC symptoms were entered at step 2 to examine

OC prediction of PTG above and beyond PTSD levels. Next, we ran a similar regression analysis for the prediction of PTG in T3 but we added PTG (T2) in step 1 to control for initial PTG levels. Finally, we conducted the same analysis per study group (POWs versus combatants).

## Results

In 1991, 7.8% ( $n = 27$ ) of the sample met criteria for Probable PTSD, while 33.8% ( $n = 117$ ) of the sample reported OC symptoms. Of those who met Probable PTSD, 96.3% ( $n = 26$ ) also endorsed OC symptoms. In 2003, 36.1% ( $n = 82$ ) of the sample ( $n = 227$ ) met Probable PTSD, while 54.6% ( $n = 124$ ) reported OC symptoms. Of those who met Probable PTSD, 92.6% ( $n = 76$ ) also endorsed OC symptoms.

As can be seen in Table 1, strong and positive associations were found between the main study variables. Importantly, there were positive and significant relations between PTSD and OC symptoms severity and PTG levels, both cross-sectional and between assessments. The more PTG veterans reported, the higher were their symptom levels for both PTSD and OC.

### Do OC symptoms predict PTG?

As hypothesized, PTSD symptoms at T1 significantly predicted PTG at T2 ( $B = .05$ ,  $\beta = .23$ ,  $p = .00$ ,  $R^2 = .05$ ). Importantly, controlling for PTSD, OC symptoms at T1 predicted PTG at T2 ( $B = .20$ ,  $\beta = .17$ ,  $p = .04$ ,  $R^2 = .07$ ,  $\Delta R^2 = .02$ ), indicating their contribution above and beyond PTSD symptoms. Similar results were obtained for the prediction of PTG at T3 by measures at T2. PTSD symptoms at T2 predicted PTG at T3 ( $B = .04$ ,  $\beta = .40$ ,  $p = .00$ ,  $R^2 = .15$ ), and again OC symptoms at T2 added to the variance and made a significant contribution ( $B = .16$ ,  $\beta = .25$ ,  $p = .04$ ,  $R^2 = .17$ ,  $\Delta R^2 = .02$ ). The same pattern of results was found when conducting separate analyses per type of trauma.

**Table 1.** Means, standard deviations, and intercorrelations between main study variables.

Measure	<i>M</i>	<i>SD</i>	1	2	3	4	5	6
1. PTSD, T1	2.42	3.26	–	.49**	.56**	.44**	.29**	.19**
2. PTSD, T2	6.88	5.80	.49**	–	.37**	.80**	.47**	.40**
3. OC, T1	.65	.66	.56**	.37**	–	.51**	.27**	.19**
4. OC, T2	1.27	1.06	.43**	.80**	.51**	–	.49**	.42**
5. PTG, T1	2.23	.72	.29**	.47**	.27**	.49**	–	.55**
6. PTG, T2	2.13	.69	.19**	.40**	.19**	.42**	.55**	–

Note. PTSD = posttraumatic stress disorder symptoms. OC = obsessive-compulsive symptoms; PTG = posttraumatic growth; T1, T2, T3 = assessments at 1991, 2003, and 2008, respectively. Pearson correlations are computed as list-wise relationships.

\*\* $p < .01$ .

### ***Do OC symptoms predict PTG above and beyond initial levels of PTG?***

For the whole sample, controlling for initial PTG and PTSD levels, OC (T2) did not predict ( $B = .06$ ,  $\beta = .10$ ,  $p = .37$ ,  $R^2 = .32$ ) PTG (T3). Therefore, separate regression analyses were conducted per type of trauma (POWs versus combatants). The analysis revealed that PTG levels at T2 significantly predicted PTG levels at T3, for both combatants ( $B = .46$ ,  $\beta = .48$ ,  $p = .00$ ,  $R^2 = .23$ ) and POWs ( $B = .45$ ,  $\beta = .49$ ,  $p = .00$ ,  $R^2 = .24$ ). PTSD symptoms did not add to the variance in both groups (Combatants:  $B = .03$ ,  $\beta = .14$ ,  $p = .23$ ,  $R^2 = .25$ ; POWs:  $B = .00$ ,  $\beta = -.00$ ,  $p = .96$ ,  $R^2 = .24$ ).

Importantly, OC symptoms at T2 significantly predicted PTG at T3 in the combatants ( $B = .48$ ,  $\beta = .40$ ,  $p = .01$ ,  $R^2 = .33$ ,  $\Delta R^2 = .08$ ) and explained an additional 8% of the variance, indicating their modest contribution above and beyond initial levels of PTG. OC symptoms did not add a significant contribution in the POWs ( $B = -.05$ ,  $\beta = -.08$ ,  $p = .50$ ,  $R^2 = .25$ ).

## **Discussion**

This longitudinal study examines the relationships between OC symptoms and PTG over time in a sample of Israeli combatants and POWs who are Yom Kippur War veterans. We repeatedly demonstrate that individuals endorsing OC symptoms subsequently report psychological growth. OC symptoms in 1991 and 2003 predicted PTG in 2003 and 2008, respectively, above and beyond initial PTSD symptoms. Further, OC symptoms surpassed the contribution of initial levels of PTG in the combatants. These preliminary results suggest that OC symptoms may have an adaptive value in facilitating psychological growth.

Several explanations may account for our findings. The notion that emotional distress, related to the psychological struggle with the trauma, can function as the trigger of growth is well documented (e.g., Tedeschi & Calhoun, 2004). It seems likely that heightened emotional distress is not sufficient for growth, because posttraumatic anxiety and depression have not been found to trigger PTG (Dekel et al., 2012; Dekel, Solomon, & Ein-Dor, 2016). However, it is possible that OC symptoms play a unique role in elevating general distress and facilitating a cognitive process that promotes growth.

Posttraumatic obsessive thoughts, similar to intrusive memories of PTSD, are anchored in the traumatic event (Sasson et al., 2005) and could result in an ongoing ruminative processing of the event. Cognitive rumination of the trauma is suggested to be the engine for growth by initiating a more deliberate search for understanding and finding meaning in the trauma, which subsequently supports growth (Calhoun, Cann, Tedeschi, & McMillan, 2010). Taku, Calhoun, Cann, and Tedeschi (2008) report that a variety of

intrusive thoughts about the event in the aftermath of the trauma lead to growth.

It is also possible that OC symptoms drive PTG due to a cognitive bias toward exaggerated personal control. Individuals with OC symptoms use compulsive rituals as a way to compensate and instill an illusory sense of control (Moulding, Kyrios, & Doron, 2007; Reuven-Magril, Dar, & Liberman, 2008). PTG may signify an attempt to regain mastery over one's life and provide reassurance that the outcome of the trauma is not catastrophic after all. These positive illusions might serve as a protection against current perceived dangers and anticipated threats (Taylor & Armor, 1996), promote self-enhancement, and counterbalance feelings of distress.

There are several limitations to this study that should be noted. The study offers predictions and does not allow for causal relationship inferences. There was significant time lapse between the war and the first assessment. Ideally, we would have conducted assessments immediately after the war and examined the relationship between OC symptoms and PTG over time. As is inevitable to a longitudinal study, not all participants took part in the follow-up assessments with an attrition rate up to 30%. Although OCD symptoms were assessed with the commonly used SCL-90 checklist, we may have assessed general OC rather than trauma-specific OC and we did not obtain a clinical diagnosis assessment of OCD or PTSD. Finally, we utilized the PTGI, a widely accepted self-report measure; however, as with any self-report, it is possible we assessed perceptions of growth instead of growth itself.

In summary, this preliminary study shows that OC symptoms lead to psychological growth in veterans of the Yom Kippur war, as long as 35 years after the war. Importantly, OC symptoms should be monitored in treatment as potentially indicative of growth and not only pathology. Our findings underscore the complexity of psychological growth following trauma and psychological distress pertaining to PTSD and comorbid symptoms, and clearly warrant more research to examine how growth is implicated in distress.

### Notes on contributors

**Daria Mamon**, MA, is a fifth-year doctoral candidate from Williams James College department of clinical psychology (PsyD) in Massachusetts. Her dissertation examined moderators to the relationship between PTG and different psychiatric conditions.

**Zahava Solomon**, PhD, is a professor of psychiatric epidemiology at the School of Social Work, Tel Aviv University, Israel. She is also the head of the I-Core Research Center for Mass Trauma in Israel. In 2009, Dr. Solomon was awarded the Prize of Israel for research in social work.

**Sharon Dekel**, PhD, is a faculty member at the Department of Psychiatry of Harvard Medical School and an investigator at the PTSD Research Program at Massachusetts General Hospital (MGH). She has been studying the implications of psychological trauma using a multitime, multimethod approach targeted at neurohormonal factors and higher levels. Her work on PTSD, PTG, trauma memory, and other variables has resulted in papers published in leading

scientific journals in the field. Dr. Dekel has received continued support for her research from the Brain and Behavior Research Foundation (NARSAD). She is also a recipient of MGH's Claflin Distinguished Scholar award.

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