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Posttraumatic Growth and Posttraumatic Distress: A Longitudinal Study

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This longitudinal study examined the course and bidirectional relation between posttraumatic distress and posttraumatic growth (PTG). A sample of Israeli ex-prisoners of war and matched controls were followed over 17 years. Participants' posttraumatic stress disorder (PTSD), depression, and anxiety symptoms were measured at three time-points. PTG was assessed twice. Applying an autoregressive cross-lagged modeling strategy, initial PTSD predicted subsequent PTG above and beyond PTG stability, but not vice versa. Cross-lagged relations of PTG to depression and anxiety were not significant. Moreover, analysis of PTG trajectory revealed that individuals with PTSD reported higher PTG levels across times than those without PTSD. Thus, growth is facilitated and maintained by endorsement rather than absence of PTSD. The findings are discussed in the context of the illusionary versus adaptive notion of PTG.

Keywords: posttraumatic growth, posttraumatic stress disorder, longitudinal study, positive illusion, posttraumatic distress

There are two seemingly opposing notions concerning the ramifications of trauma. The first, which is widely held, postulates that trauma has a pathogenic effect. Traumatic events jeopardize physical and psychological equilibrium giving rise to a wide range of physical and mental health complications. A considerable body of empirical research lends support to this view, documenting increased rates of posttraumatic stress disorder (PTSD), depression, anxiety, somatization, and alcoholism (e.g., Breslau, Davis, Andreski, & Peterson, 1991; Kessler, Sonnega, Bromet, & Hughes, 1995).

An alternative perspective proposes that trauma has a salutogenic effect. Individuals can develop a positive outlook and further experience positive psychological changes in the wake of traumatic events (e.g., Tedeschi & Calhoun, 2004). In line with currently prevalent positive psychology theory (Seligman & Csikszentmihalyi, 2000) and earlier salutogenic models (Antonovsky, 1979), survivors may gain psychological benefits. The commonly held term, posttraumatic growth (PTG), (Tedeschi & Calhoun, 1996) signifies that the individual has transformed in new ways that go beyond his or her pretrauma level of psychological functioning. This entails increase in personal strength, relational intimacy, sense of spirituality, appreciation of life, and life possibilities. Not undermining the pathogenic impact of trauma, recently, a growing body of studies has consistently revealed PTG reported by survivors following various physical and psychological traumas (see Calhoun & Tedeschi, 2006; Linley & Joseph, 2004, for reviews).

As both salutogenic and pathogenic trauma outcomes have been documented, an imperative issue is how they relate. While there has been a proliferation of research on the subject, as evident in a recent meta-analysis reporting 77 studies (see Helgeson et al., 2006, for a review), the relation of growth to distress is still ill-defined. The aim of this longitudinal study is to shed light on the interplay between PTG and PTSD by examining their directional (i.e., temporal) relation.

Relation of Growth to Distress

Three possible modes of association have been suggested. In the first, growth and distress are opposite ends of the same continuum. Accordingly, a negative association was found between the outcomes (e.g., Frazier et al., 2001). Alternatively, growth is thought to coexist with distress in a positive manner. Solomon and Dekel (2007) for example, found that the higher the distress, the more growth. Finally, growth is possibly an independent outcome. In line with this view, some studies documented no significant relations with distress (e.g., Joseph et al., 1993). These perplexing findings are attributed in part to methodological issues. A crucial limitation of the existing research is its overwhelming reliance on cross-sectional, rather than longitudinal designs (Park & Helgeson, 2006). Cross-sectional designs fail to detect the catalizator variable that sets into motion the outcome variable, making them imperfect in fully grasping the nature of association.

A possible path suggests that distress triggers subsequent growth. Tedeschi and Calhoun (2004) argue that PTG is the outcome of the posttrauma psychological struggle. Stress-induced cognitive rumination transforms into constructive processing, which in turn initiates the development of growth. According to this formulation, it is expected that PTSD and PTG are positively

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related. Alternatively, in the reverse path, growth proceeds distress rather than vice versa. It has been suggested that PTG is a means for offsetting pathogenic outcomes. For example, Davis, Nolen-Hoeksema, and Larson (1998) deem PTG a construal of meaning; while others (e.g., Taylor & Armor, 1996) consider PTG a self-enhancing appraisal (see Zoellner & Maercker, 2006, for a review). Viewing PTG as a coping mechanism, it is expected that PTSD and PTG are negatively related such that the more growth, the less the distress subsequently experienced.

As shown in Table 1 longitudinal investigations examining the direction of association of distress to growth are scant and their findings are mixed. While a group of studies showed that growth predicts later reduction in distress; a few studies found them unrelated.

These inconsistencies are partly due to methodological limitations. When PTG was found adaptive, unstandardized measures of growth were often used, such as single open-ended questions. Thus, clear evidence that growth promotes postevent adjustment is still lacking (Zoellner & Maercker, 2006). Also of significance are two gaps in the literature. Although theoretically derived (Tedeschi & Calhoun, 2004), quite surprisingly the alternative notion that growth is an outcome of distress has rarely been tested. While the liable quality of PTSD has been repeatedly documented (e.g., Solomon, Horesh, & Ein-Dor, 2009), insight into the temporal course of PTG is scarce, particularly in the long-run.

The current study is a prospective and longitudinal investigation of a sample of Israeli veterans. First, it aims to assess the bidirectional relation between PTG and distress. We put forward two competing hypotheses: (1) growth is a response to distress, and (2) growth leads to a reduction in distress. The second aim is to examine possible changes in growth over time.

Method

Participants and Procedure

The present study uses data from a longitudinal study on the psychological implications of war (see Solomon & Dekel, 2005, for full details). A cohort of Israeli veterans who participated in the October 1973 Yom Kippur War were followed over 17 years with assessment at three time-points: 1991 (T1), 2003 (T2), and 2008 (T3). Using updated Israel Defense Forces (IDF) files, we phoned those participants and after explaining the purpose of the study, we asked them to take part in the follow-up assessment. The questionnaire packet was administered in their homes or in another location of their choice. Before filling out the questionnaire, the participants signed an informed consent form.

According to Israel's Ministry of Defense, 240 soldiers from the Israeli Army land forces were captured during the war. One hundred sixty-four participated in the first assessment, 144 participated in the second (10 could not be located/refused, 4 had died, and 6 could not participate due to mental deterioration), and 183 took part in the third (29 could not be located/refused, 20 had died, and 6 could not participate due to mental deterioration).

In addition, 280 veterans were sampled from IDF computerized data banks (Solomon et al., 1994). These individuals participated in the same war, but were not taken captive and were matched on military background and sociodemographic status. They constituted combat veterans exposed to battlefield stressors, including encounters with injury and death bodies, active fighting, and exposure to life-threatening events. Among them, 185 participated at T1, 143 participated at T2 (41 could not be located and 1 had died), and 118 took part at T3 (20 could not be located/refused and 5 had died).

For all participants mean age at T2 was 53.4 ($SD = 4.4$); their gender was male; mean years of schooling was 14.02 ($SD = 3.41$); the majority were secular (67%); and with an average income (62%). Sixteen percent could have qualified for clinical diagnosis of PTSD. No significant differences were found between those who participated in the follow-up assessments with regard to the level of PTSD in 1991, rank, age, and education.

Measures

Posttraumatic growth. PTG was measured at T2 and T3 utilizing the Post Traumatic Growth Inventory (PTGI; Tedeschi & Calhoun, 1996). This questionnaire is a 21-item self-report including five subscales: relating to others, new possibilities, personal strength, spiritual change, and appreciation of life. Participants were asked to report on a 4-point scale the extent of change that occurred in their life following a specified trauma. The PTGI has good internal consistency, construct, convergent, and discriminant validity (Tedeschi & Calhoun, 1996). In the present study, the trauma was identified as captivity (for ex-prisoners of war; POWs) or participation in the combat (for controls). Subscales scores, as well as general scores were calculated. Reliability values for total and subscales scores were high (Cronbach's alpha .94 and .93, respectively).

Posttraumatic distress. PTSD was measured at all assessments, using the PTSD Inventory[1] (Solomon et al., 1993). This is a self-report scale based on the criteria of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition Revised* (*DSM–III–R*; American Psychiatric Association (APA), 1987). The inventory consists of 17 statements corresponding to the 17 PTSD symptoms listed in the *DSM–III–R*. Participants are asked to indicate whether or not they had the symptom in the past month, on a 4-point scale ranging from 1 (not at all) to 4 (I usually did). The inventory has proven psychometric properties in terms both of high test-retest reliability and concurrent validity compared with clinical diagnosis (Solomon et al., 1993). In the study, mean number of PTSD symptoms, as well as the mean number of symptoms in each cluster (intrusion, avoidance, and hyper-arousal) were assessed. In order to conform to the *DSM–IV* (APA, 1994) symptom clusters, we analyzed the data in accordance. Reliability values for total and subscale scores were high at all assessments (Cronbach's alpha ranging from .78 to .96).

Depression, anxiety, and global distress were measured in all assessments, with the Symptom Checklist-90 (SCL-90, Derogatis, 1977). This questionnaire targets 90 psychiatric symptoms. It offers a Global Severity Index (GSI), which reflects the severity of all symptoms, and an assessment of specific symptom clusters, including depression and anxiety. Participants are asked to indicate how frequently they experienced each symptom during the last 2 weeks on a 5-point distress scale. The SCL-90 has high concurrent validity and the specific subscales display high empirical agree-

[1] When the study commenced the *DSM–III–R* was the standard for diagnosis criteria of PTSD.

Table 1
Summary of Longitudinal Studies Examining the Directional Relationship Between Growth and Distress Post-Trauma

Study	Event	*n*	Growth measure	Distress measure	Design
Category I: Initial growth predicts subsequent distress (negative relation)					
McMillen, Smith, & Fisher (1997)	Disaster	195	Perceived benefit single item	PTSD (DIS)	4–6 wk 3 yr
Davis, Nolen-Hoeksema, & Larson (1998)	Bereavement	205	Perceived benefit single item	Overall distress: depression (IDD), PTSD (11 single items), positive affect (State-Trait Anxiety Inventory)	6 wk preloss 6 mo 13 mo 18 mo
Frazier, Conlon, & Glaser (2001)	Sexual assault	171	17 items created for study	Depression (BSI) PTSD (17 items)	2 wk 12 mo
Rini et al. (2004)	Mothers of children undergoing stem cell translation	144	Benefit finding (Two items created for study)	Psychosocial (Mental Health Summary Scale)	baseline 6 mo
Ickovics et al. (2006)	Various	177	PTGI modified for study	Overall distress (BSI)	baseline 12 mo 18 mo
Lechner, Carver, Antoni, Weaver, & Phillips (2006)	Surgery following breast cancer	96	PBS modified for study	Depression (CES-D)	6 mo 5–8 yr
Linley, Joseph, & Goodfellow (2008)	Various	40	Changes in Outlook Questionnaire	PTSD (PSS), depression (HAD), anxiety (HAD)	baseline 6 mo
Category II: Initial distress predicts subsequent growth (positive relation)					
Erbes et al. (2005)*	War captivity	95	PTGI	PTSD (SCID PTSD)	91–94 02
Category III: Growth and distress are unrelated					
Phelps, Williams, Raichle, Turner, & Ehde (2008)	Amputation	83	PTGI	PTSD (PCL) Depression (PHQ-9)	6 mo 12 mo
Salsman, Segerstrom, Brechting, Carlson, & Andrykowsky (2009)	Cancer	55	PTGI	PTSD (PCL-C) Depression (MHI) Anxiety (MHI) Positive affectivity (MHI)	baseline 3 mo 13 mo
Sears, Stanton, & Danoff-Burg (2003)	Cancer	58	Benefit Finding Two items	Negative mood (POMS)	baseline 3 mo 12 mo

Note. BSI = Brief Symptom Inventory; CES-D = Center for Epidemiological Studies Depression Scale; DIS = Schedule Disaster Supplement; HAD = Hospital Anxiety and Depression; IDD = Inventory to Diagnose Depression; MHI = Mental Health Inventory; mo = month; PCL = PTSD Checklist; PCL-C = PTSD Checklist, Civilian Version; PHQ-9 = Patient Health Questionnaire-9; POMS = profile of mood states; PSS = PTSD symptom scale; SCID = Structured Clinical Interview; wk = week; yr = year.
* Results reach significance.

ment across various samples (Derogatis, Rickles, & Rock, 1976). In the study, reliability values were high at all assessments (Cronbach's alpha for depression and anxiety ranging from .83 to .90 and .86 to .93, respectively).

Results

Descriptive Statistic

Table 2 presents the means, standard deviations, and intercorrelations between our main study measures. Specifically, using a series of Pearson correlations, we assessed the magnitude of associations between PTSD, PTG, GSI, anxiety, and depression in 2003 and 2008.

The analyses yielded significant relations among PTSD, PTG, GSI, anxiety, and depression both cross-sectional and between times. The more PTSD symptoms veterans endorsed, the higher their PTG, GSI, anxiety, and depression levels. Of importance, Steiger's (1980) test for equality of dependent correlations revealed that the magnitude of the cross-sectional association between PTSD and PTG was not significantly different between 2003 and 2008, $z(168) = -.87$, $p = .19$.

Exploring the Bidirectional Associations Between PTSD and PTG

We examined the bidirectional association between PTSD and PTG over two time-points, 2003 and 2008. To this end, we employed an autoregressive cross-lagged modeling strategy (ARCL; e.g., Anderson, 1960), which allows for simultaneous assessment whether earlier measures of PTSD predict later measures of PTG, and whether earlier measures of PTG predict later measures of PTSD. Because PTSD consists of three clusters (intrusion, avoidance, and hyper-arousal), and PTG consists of five clusters (relations, new possibilities, personal strength, spiritual change, and appreciation of life), we used latent variables in a structural equation model (SEM) environment to represent the PTSD and PTG underlying phenomena.

In order to assess the appropriateness of the ARCL, we used the EQS 6.1 SEM software (Bentler & Wu, 1995). We estimated the model's fit by using the comparative fit index (CFI), Bentler-Bonett non-normed fit index (NNFI), and the root mean square error of approximation (RMSEA). A model is judged as reasonably fitting the data when CFI, NNFI, and 1-RMSEA are larger than .95 (Bollen & Curran, 2006). Missing data were handled with the case-wise maximum likelihood estimation for possible non-normality when running EQS models.

Figure 1 presents the bidirectional relations between PTSD and PTG over time. The model fit the data fairly well, $\chi^2(110) = 299.23$, $p < .01$, CFI = .97, NNFI = .97, 1-RMSEA = .96, 90% 1-RMSEA CI [.97, .95]. The analysis revealed that the stability of PTSD over time, as well as the stability of PTG, was noticeably high. Veterans with high levels of PTSD or PTG in 2003, tended to have high levels of PTSD or PTG, respectively, in 2008. More importantly, the analysis revealed that the initial level of PTSD in 2003 predicted subsequent level of PTG in 2008, above and beyond PTG stability, but not vice versa.

In order to generalize the latter results to additional time-points, we ran a second ARCL model. In this model, we added PTSD level in 1991 as a predictor of subsequent level of PTG in 2003[2] (see Figure 2). The other model parameters remained the same. The model fit the data fairly well, $\chi^2(158) = 359.56$, $p < .01$, CFI = 1, NNFI = 1, 1-RMSEA = 1, 90% 1-RMSEA CI [1, .98]. Consistent with the previous finding, the analysis revealed that the initial level of PTSD in 1991 predicted subsequent level of PTG in 2003. The other effects remained the same.

Next, we assessed whether all PTSD clusters (intrusion, avoidance, and hyper-arousal) accounted for the significant cross-lagged association between PTSD and PTG, or only specific clusters of PTSD. To this end, we ran another ARCL model. In this model, PTSD clusters served as predictors of PTG. The model fit the data fairly well, $\chi^2(100) = 433.39$, $p < .01$, CFI = .97, NNFI = .96, 1-RMSEA = .95, 90% 1-RMSEA CI [.96, .94]. The analysis revealed that whereas the number of PTSD hyper-arousal symptoms veterans endorsed in 2003 predicted their PTG level in 2008, $\beta = .40$, $t = 2.87$, $p < .01$; the number of PTSD intrusion or avoidance symptoms veterans endorsed in 2003 did not, $\beta = -.17$, $t = -1.63$, $p = .11$, and $\beta = -.04$, $t = -.32$, $p = .75$, respectively.

Is the Relation Between PTG and PTSD Specific, or Generalized to Other Psychopathologies?

We found that the number of PTSD symptoms veterans endorsed predicted their subsequent PTG level, but not vice versa. Is this link between PTSD and PTG specific, or do other psychopathologies also predict PTG level? Anxiety and depression are often comorbid with PTSD (e.g., Ginzburg, Ein-Dor, & Solomon, 2010). Therefore, to answer this question, we assessed the bidirectional relations between anxiety, depression, and PTG across time using an ARCL model. The model fit the data fairly well, $\chi^2(81) = 231.27$, $p < .01$, CFI = .97, NNFI = .96, 1-RMSEA = .95, 90% 1-RMSEA CI [.96, .94]. The analysis revealed that the stability of anxiety and depression across time was noticeably high. Veterans with high levels of anxiety or depression in 2003 tended to have high levels of anxiety or depression, respectively, in 2008, $\beta = .68$, $t = 6.19$, $p < .001$. More importantly, the analysis revealed that neither the initial levels of anxiety and depression in 2003, $\beta = .11$, $t = 1.36$, $p = .18$, nor the initial level of PTG in 2003, $\beta = .02$, $t = .27$, $p = .79$, showed cross-lagged influences, above and beyond the measures' stability.

Is the Relation Between PTG and PTSD Different for Ex-POWs and Controls?

We assessed whether the bidirectional association between PTSD and PTG over time was different for ex-POWs and controls. To this end, we ran multigroup ARCL models that estimated the relation between PTSD and PTG separately for ex-POWs and controls. The multigroup model fit the data well, $\chi^2(220; n = 349) = 429.18$, $p < .01$, CFI = .98, NNFI = .98, 1-RMSEA = .97, 90% 1-RMSEA CI [1, .96], as well as the separate models: $\chi^2(110; n = 164) = 235.33$, $p < .01$, CFI = .96, NNFI = .96, 1-RMSEA = .96, 90% 1-RMSEA CI [.98, .94] for ex-POWs, and

[2] Participants have not reported on their PTG level in 1991. Hence, we could not examine whether initial level of PTG in 1991 predicted subsequent level of PTSD in 2003.

Table 2
Means, Standard Deviations, and Intercorrelations Between Main Study Measures

Measure	1	2	3	4	5	6	7	8	9	10
1. PTG, 2003	1									
2. PTG, 2008	.55**	1								
3. PTSD, 2003	.40**	.40**	1							
4. PTSD, 2008	.40**	.46**	.80**	1						
5. GSI, 2003	.42**	.36**	.82**	.74**	1					
6. GSI, 2008	.33**	.38**	.71**	.83**	.76**	1				
7. Depression, 2003	.38**	.31**	.80**	.70**	.94**	.74**	1			
8. Depression, 2008	.26*	.29**	.66**	.75**	.68**	.93**	.72**	1		
9. Anxiety, 2003	.42**	.32**	.80**	.71**	.97**	.74**	.92**	.66**	1	
10. Anxiety, 2008	.33**	.37**	.68**	.80**	.76**	.93**	.72**	.83**	.76**	1
M (*SD*)	2.27 (.74)	2.10 (.72)	6.37 (5.74)	6.62 (5.79)	.92 (.86)	.98 (.85)	1.00 (.94)	1.00 (.99)	.89 (.96)	1.21 (1.08)

* $p < .01$. ** $p < .001$.

$\chi^2(110; n = 185) = 249.30$, $p < .01$, CFI = .99, NNFI = .99, 1-RMSEA = .97, 90% 1-RMSEA CI [1, .95] for controls. The analyses revealed that for controls, the initial level of PTSD in 2003 predicted subsequent level of PTG in 2008, $\beta = .24$, $t = 2.13$, $p < .05$, above and beyond PTG stability, but not vice versa, $\beta = -.05$, $t = -.52$, $p = .60$. Conversely, there were no cross-lagged associations between PTSD and PTG for the ex-POWs ($\beta = -.03$, $t = -.26$, $p = .80$ for predicting PTG by PTSD, and $\beta = .10$, $t = 1.07$, $p = .29$ for predicting PTSD by PTG). The analyses also revealed that the link between PTSD in 2003 and PTG in 2008 was significantly stronger for controls than for ex-POWs, $\Delta\chi^2(1) = 4.04$, $p < .05$; yet, the groups did not differ in the magnitude of the link between PTG in 2003 and PTSD in 2008, $\Delta\chi^2(1) = 1.06$, $p = .30$.

Does PTSD Status in 1991 Predict PTG Trajectory of Change?

We examined whether PTSD status in 1991 (no PTSD, subclinical PTSD, clinical PTSD) predicted PTG trajectory of change

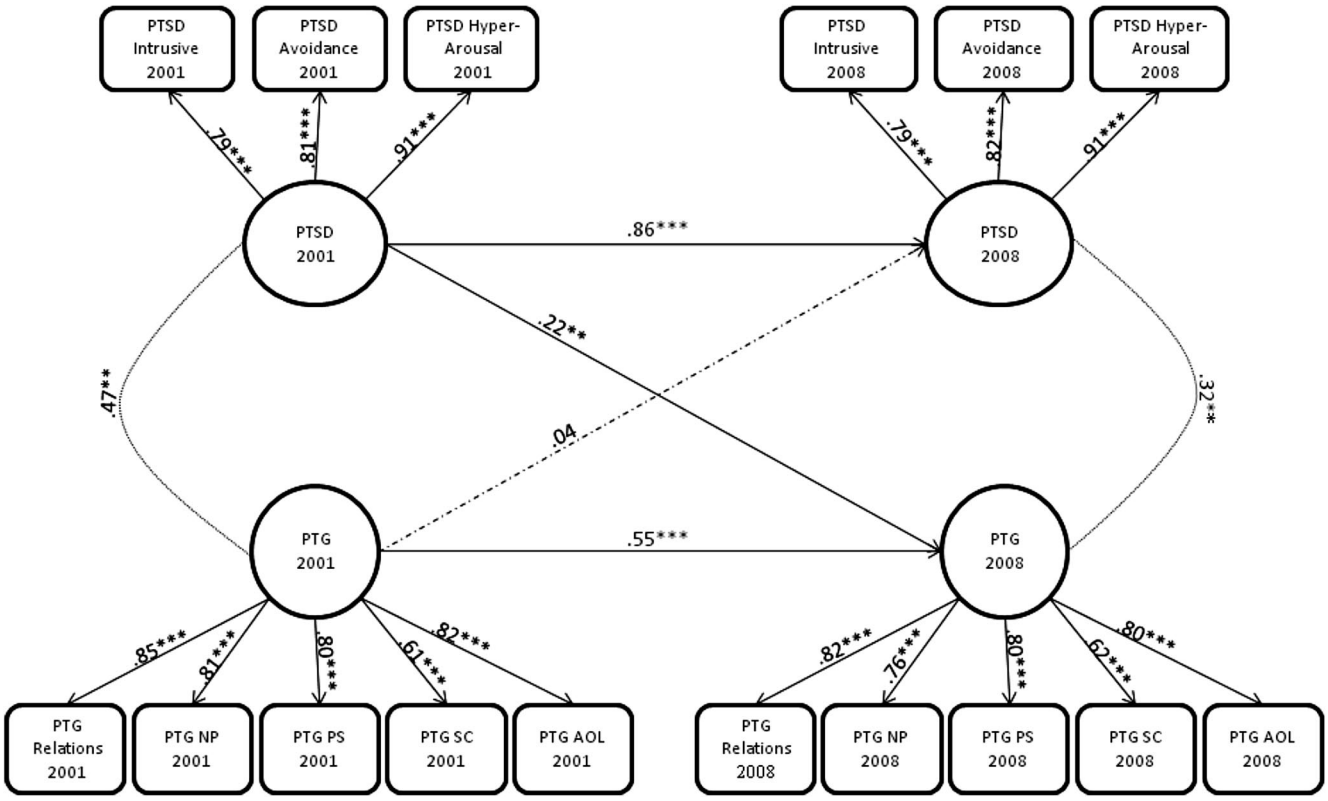

Figure 1. ARCL model for assessing the bidirectional association between PTSD and PTG across two time-points, 2003 and 2008.

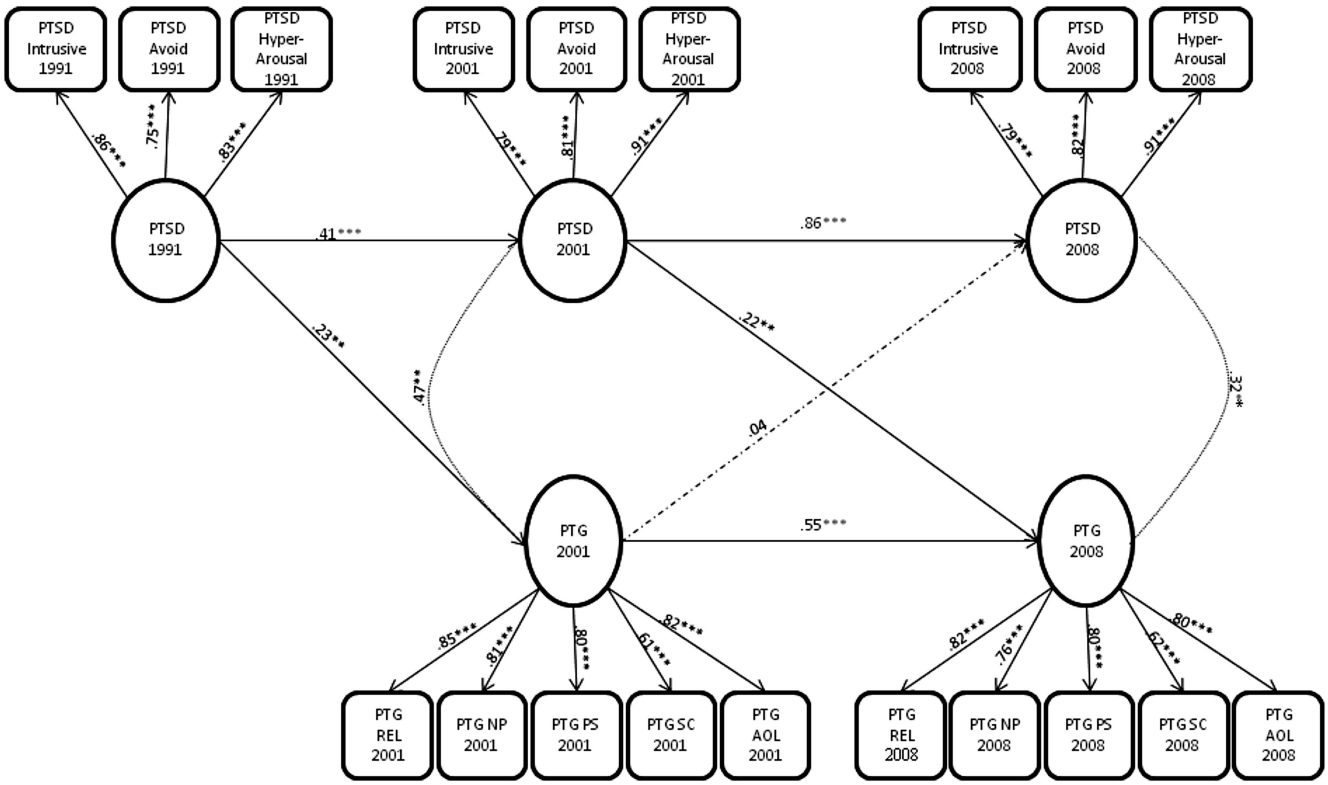

Figure 2. ARCL model for assessing the bidirectional association between PTSD and PTG across two time-points, 2003 and 2008, with PTSD level at 1991 as another predictor.

between 2003 and 2008. In 1991, 7.8% ($n = 27$) of the veterans were classified as suffering from PTSD symptoms, 12.1% ($n = 42$) were classified as suffering from subclinical PTSD (had at least 2 of the 3 symptom criteria for PTSD), and 80.2% ($n = 279$) did not have PTSD. In order to analyze whether PTSD predicted PTG trajectory of change, we used mixed, repeated-measures design with PTSD status in 1991 as the between-subjects variable, time of measurement (2003 vs. 2008) as the within-subjects variable, and PTG level as the dependent variable. The analysis revealed a main effect for time of measurement, $F(1, 144) = 7.49$, $p < .01$, $\eta_p^2 = .05$. Veterans' PTG level at 2003 ($M = 2.53$, $SE = .11$) was significantly higher than their PTG level at 2008 ($M = 2.25$, $SE = .10$). Moreover, the analysis revealed a main effect for PTSD status in 1991, $F(2, 144) = 5.38$, $p < .01$, $\eta_p^2 = .07$. Post hoc analyses revealed that the subclinical ($M = 2.55$, $SE = .14$; $p < .01$) and clinical ($M = 2.50$, $SE = .23$; marginally significant, $p < .1$) PTSD groups had higher levels of PTG than the no-PTSD group ($M = 2.12$, $SE = .06$). The interaction between PTSD status in 1991 and time of measurement was not significant, $F(2, 144) = 1.70$, $p = .19$, $\eta_p^2 = .02$.

Discussion

This longitudinal study is original in examining the bidirectional association between PTG and distress and its course over time. The main finding is that growth is a response to distress and not vice versa. Initial elevated PTSD reported in 2003 predicted subsequent elevated PTG symptoms reported in 2008 above and beyond PTG. More so, individuals with PTSD had higher PTG levels across times than resilient individuals who did not endorse PTSD symptoms. Taken as a whole, the present study contributes to the literature by suggesting that endorsement of posttraumatic distress is crucial for facilitating and maintaining psychological growth following a traumatic event.

Growth being an outcome of distress is partly in line with Tedeschi and Calhoun's model (2004). Accordingly, it is the emotional struggle in the wake of trauma, which pushes forward PTG rather than mere exposure to traumatic events. The ensuing cognitive rumination transforms into constructive processing of the traumatic event and becomes the drive for positive change. Similarly, distress triggers meaning making efforts targeted at making sense of the event and its philosophical and existential consequences (Janoff-Bulman & Frantz, 1997), allowing for subsequent growth (Joseph & Linley, 2006). Overall, in both models, PTG is a marker of "significant beneficial change" (Tedeschi et al., 1998, p. 3).

An alternative explanation for the finding that growth is an outcome of distress has to do with its proposed self-deceptive quality (Maercker & Zoellner, 2004). Rather than viewing PTG as the end result of successful coping, PTG itself is regarded a coping process. Taylor et al. (e.g., Taylor et al., 2000) use the term "positive illusions" to describe this phenomenon. Accordingly, perceptions of growth are self-enhancing illusions that help the survivor counterbalance emotional distress. In line with this perspective, PTG can be viewed as a defense against pathogenic

trauma sequelae. Endorsement of elevated PTG over time, as seen in our study in veterans with PTSD, would then signify that these individuals have failed to restore well-being and still need to deploy self-deceptive strategies in an attempt to sedate their felt distress.

Of significance, this study shows the unique role of PTSD in promoting PTG. Neither initial depression nor anxiety levels predicted subsequent levels of PTG. The idiosyncratic relation of PTSD to PTG may have to do with the prominent role of hyperarousal. An intriguing finding of this study is that of the PTSD symptom clusters, the hyper-arousal was the single predictor of subsequent growth, rather than the re-experiencing as the theory suggests (Tedeschi et al., 1998). This seems perplexing as hyperarousal manifested in irritation, outbursts, and exaggerated startle response (APA, 1994) has been recently documented as the strongest symptom cluster predictor of impaired functioning and overall distress (e.g., Solomon et al., 2009). Possibly, the hyper-arousal vigilance state facilitates that the individual participate in the world rather than withdraw socially and engage in avoidant behaviors. In turn, the individual may re-evaluate his life, relationships, and so forth in the process of promoting growth. Moreover, due to its early onset, hyper-arousal may be regarded as the engine of PTSD that fuels subsequent trauma outcomes, including PTG. Previous studies documenting growth associated with intrusive thinking (i.e., re-experiencing) were cross-sectional and assessed trauma outcomes in the relatively short phase following the event (see Helgeson et al., 2006, for a review). In contrast, this study relied on longitudinal methods for assessing in the very long-phase the relationship between PTG and distress.

An interesting finding relates to the role of trauma exposure in growth. Our findings show that PTSD predicted later growth only for the matched controls and not the ex-POWs, suggesting that there are different pathways that set in motion growth and possibly different types of growth. The psychological damage of captivity is even more enduring than that of combat. In a previous study with the same sample, we found that ex-POWs were 10 times more likely to experience deterioration in their mental health status from 1991 to 2003, and their PTSD symptom severity rate in 2003 was 7 times higher than the controls' (Solomon & Dekel, 2005). Possibly moderate distress is optimal for triggering the experience of posttraumatic growth. In accord, previous studies, although cross-sectional, documented a curvilinear relationship between growth and both level of exposure and distress (see Helgeson et al., 2006, for a review).

Inconsistent with previous longitudinal studies, we were unable to show the adaptive value of PTG in reducing distress. Initial PTG levels reported in 2003 did not predict subsequent PTSD levels reported in 2008. It could be argued that growth improves some aspects of well-being and not others. Our findings, however, suggest that PTG does not facilitate reduction in depression and anxiety levels. It could also be hypothesized that PTG has an adaptive value that is moderated by trauma exposure; although, neither for the ex-POWs nor the controls did PTG ease subsequent distress. Possibly the discrepancy in the findings are in part attributed to the measurement. While this study used the PTGI (Tedeschi & Calhoun, 1996), a validated and widely-used questionnaire, previous studies often used nonstandardized tools (see Table 1). Taken as a whole, it might be that PTG does not have a significant value in lessening distress after all, but is nevertheless an illusion of value.

The current study assessed the temporal course of PTG, documenting a decrease in PTG with the passage of time. Previous studies, however, showed that PTG intensifies over time in the short-run (e.g., Frazier et al., 2001). Altogether, the findings suggest that within PTG's relatively stable nature, it has a liable quality of increase and decrease, as previously seen in PTSD (e.g., Port, Engdahl, Frazier, 2001). The decline in PTG might have to do with PTSD trajectory of change. If one considers PTG as opposite to distress, then a decrease in the former should accompany an increase in the latter. In the current study, however, levels of PTSD remained steady between assessments (T2–T3). Processes due to aging may also explain the decrease. In midlife, there is a natural decline in physical health and stressors related to retirement and loss may surface, possibly impinging on PTG. Although, in the current study participants were in midlife years at both the assessment points.

It has been argued that the reduction of PTG over time indicates its illusionary quality. Constructive growth implicated in symptom reduction should follow an increase trajectory; while growth in the form of a merely positive illusion that does not promote mental health in the long-run, as we found, is expected to diminish over time (Zoellner & Maercker, 2006). It may be that two decades after the Yom Kippur War, when symptoms do not remit, it is much more difficult to sustain a self-enhancing appraisal and a perception of positive psychological change.

The present study has several methodological shortcomings. First, it offers predictions and therefore does not allow clear-cut inferences regarding causational relations. Second, there are limitations inherent in longitudinal designs. Our results show that distress pushes forward growth and not vice versa when examined at the study's assessment points and for participants taking part in the follow-up assessments. Although the cross-lagged association was revealed at two different occasions and the sample attrition rate was not high, this cannot fully negate the contribution of the time and sample nature to the finding. Future longitudinal studies are warranted to examine whether distress predicts growth in other trauma populations and time-points. Also, it would be relevant to examine mental health outcomes, such as positive well-being, in addition to mere distress and their relationship with PTG.

These limitations notwithstanding, this longitudinal study contributes to our knowledge concerning the relation of pathogenic and salutogenic trauma outcomes and their trajectories. We show that distress is crucial to set growth in motion and keep it going. Hence, individuals experiencing PTSD, particularly when it is enduring, have the potential for positive psychological change. This study is likely to have promising implications for treatment: rather than targeting resistant symptoms, it might be important to strengthen the patient's capacity for growth, be it illusionary or real.

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