THE TEMPORAL RELATIONSHIP BETWEEN POSTTRAUMATIC STRESS
SYMPTOMS AND POSTTRAUMATIC GROWTH AMONG ISRAELI JEWS AND
ARABS: A LONGITUDINAL CROSS-LAGGED PANEL ANALYSIS

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The Temporal Relationship between Posttraumatic Growth and Posttraumatic Stress Symptoms among Israeli Jews and Arabs:
A Longitudinal Cross-Lagged Panel Analysis

Studies have addressed the link between exposure to potentially traumatic events (PTE) and the development of posttraumatic stress disorder (PTSD; Kessler, Sonnega, Bromet, & Nelson, 1995) and sought to identify factors that contribute to the development of PTSD following PTE (e.g., Brewin, Andrews, Valentine, 2000). Although these areas of inquiry are important and vitally necessary to the traumatic stress field, preoccupation with disorder following PTE may obfuscate important information about the potential for a positive legacy that trauma may leave in its wake (Linley & Joseph, 2004). This is especially important given the substantial variation with regard to the incidence of PTSD following PTE as relatively few individuals who experience PTE develop PTSD (Kessler et al., 1995).

For example, participants surveyed five to eight weeks following the September 11, 2001 terror attacks reported a 7.5% prevalence rate for probable PTSD, and participants who lived near the World Trade Center reported 20% prevalence of the disorder (Galea et al., 2002). During a heightened period of terrorism in Israel, the prevalence of probable PTSD among Jews was 6.6% and 18% among PCI (Hobfoll, Canetti-Nisim, Johnson, Palmieri, Varley, & Galea, 2008). In the tumultuous period of
the Gaza disengagement, 26% of a representative sample of Jewish settlers reported symptoms consistent with probable PTSD (Hall et al., 2008). Hence, in each sample, the majority of individuals exposed to trauma did not report symptoms consistent with probable PTSD.

Utilizing alternative models that incorporate both positive and negative reactions to PTE is a more comprehensive approach to understanding posttraumatic adaptation (Linley and Joseph, 2004). A burgeoning effort has been undertaken in the health and traumatic stress field to investigate the positive reactions that can result from exposure to PTE. Tedeschi and Calhoun’s (1995) theoretical work on the positive changes occurring as a result of PTE exposure, a construct they called posttraumatic growth (PTG), gave impetus to these recent investigations.

*Posttraumatic Growth: A Multidimensional Construct*

Sultogenic models of functioning following PTE are not entirely new (e.g., Antonovsky, 1987). However, the recent work of Tedeschi and Calhoun (1995) has led to a greater number of investigations into this phenomenon. Most of the work on PTG has occurred within the past 10 years and this nascent literature continues to develop. Although it is too early to cite the recent flourish of PTG literature as a sea-change in the traumatic stress field, it does serve as a harbinger of continued investment into understanding positive post-trauma changes.

PTG is broadly defined as a “positive psychological change experienced as a result of the struggle with highly challenging life circumstances” and trauma (Tedeschi &
Calhoun, 2004, pg. 1; Calhoun and Tedeschi, 1999). PTG does not simply involve a return to pre-trauma levels of functioning following few symptoms of disorder being present following exposure to PTE (e.g., resiliency; Bonanno, 2005), nor does it speak to the capacity to resist developing psychological disorder (Layne et al., 2007; Hobfoll, Palmieri, Johnson, Canetti-Nisim, Hall, & Galea, 2009). Rather it is defined as achieving an enhanced level of functioning, sense of meaning or spirituality, and developing closer relationships that were not present before the PTE occurred (Linley & Joseph, 2004).

PTG is a multidimensional construct involving both interpersonal and intrapersonal dimensions. Tedeschi and Calhoun (1995) conceptualized PTG as positive changes in self-perception, philosophy of life (e.g., personal meaning), and interpersonal relationships experienced following a stressful life event. PTG exemplifies Nietzsche’s famous life-affirming maxim “that which does not kill me makes me stronger” (Nietzsche, 1889/1997).

**PTG and Psychological Distress**

One of the most vexing and contentious issues involving PTG is (1) whether it is related to or unrelated to psychological distress, and further, (2) whether is serves as a protective factor or a risk factor for psychological disorder. Zoellner and Maercker (2006) astutely observed that “if posttraumatic growth is a phenomenon worthy to be studied in clinical research, it is assumed to make a difference in people’s lives by affecting levels of distress, well-being, or other areas of mental health. If it does not have any impact, PTG might just be an interesting phenomenon possibly belonging to the
areas of social, cognitive, or personality psychology” (p. 631; emphasis added). If there is a lack of evidence regarding the adaptive significance of PTG it is questionable whether it should be promoted (e.g., Calhoun & Tedeshi, 1999). The assumption that PTG is a health promoting process or protective factor should be empirically tested.

In their early theoretical contributions to the PTG literature, Tedeschi and Calhoun (1995) suggested that PTG and psychological distress are orthogonal constructs. They posited that an individual can report growth from a PTE and can also report psychological distress at the same time, and that these two dimensions are unrelated to each other. The existence of the co-occurrence of positive and negative changes following a PTE is less debated and research studies have demonstrated that people do indeed report both simultaneously (Bellizzi, Miller, Arora, Rowland, 2007; Frazier, Conlon, and Glaser, 2001). Other studies have indicated that the acknowledgement of both positive and negative reactions is related to less psychological distress (Cheng, Wong, & Tsang, 2006), whereas only acknowledging positive reactions is related to greater psychological distress.

What is particularly problematic, and not widely supported by empirical research, is a lack of relatedness between PTG and PTSD symptom severity. Some studies have demonstrated that PTG is associated positively with PTSD symptom severity such that greater PTG is related to greater PTSD symptom severity (Elder & Clipp, 1988; Hall, Hobfoll, Canetti-Nisim, Johnson, & Galea, 2010; Helgeson, Reynolds, & Tomich, 2006; Hobfoll et al., 2006; Hobfoll et al., 2008; Hobfoll et al., 2009; Lehman, Davis, DeLongis, Wortman, Bluck, & Mandel, 1993; Pargament, Smith, Koenig, & Perez, 1998; Park,
Cohen, & Murch, 1996; Tomich & Helgeson, 2004; Wild & Paivio, 2003), some have demonstrate that PTG is associated negatively with PTSD symptom severity such that more PTG is related to less PTSD symptom severity (Ai, Cascio, Santangelo, & Evans-Campbell, 2005; Frazier et al., 2001; Hall et al., 2008; McMillen, Smith, & Fisher, 1997), and few studies support the viewpoint that PTG and psychological distress is unrelated (Cordova, Giese-Davis, Golant, Kronenwetter, Chang, & Spiegel, 2007; Val & Linley, 2006; Widows, Jacobsen, Booth-Jones, Fields, 2005). The results of a recent meta-analysis of 77 cross-sectional studies (Helgeson et al., 2006) indicated that PTG was related to greater avoidance and intrusive thoughts, core features of PTSD.

PTG is generally associated with PTSD symptom severity as the majority of studies do indicate their relatedness. Meta-analytic findings also suggest that on average, PTG is related to greater reexperiencing and avoidance-related PTSD symptom severity. However, the direction of the relationship between PTG and PTSD symptom severity across studies indicates insufficient consistency to draw clear conclusions about whether PTG contributes to less PTSD symptom severity or whether it contributes to greater PTSD symptom severity. Moreover, it is yet unclear whether PTSD symptom severity leads to greater PTG. Additionally, few studies have examined the relationship between each of the PTSD symptom clusters and PTG. This would represent a more nuanced approach to understanding which dimensions of PTSD may be related or unrelated to PTG.

Longitudinal Posttraumatic Growth Research
The majority of the PTG literature has measured the association between PTG and psychological distress within cross-sectional correlational designs. In some cases, PTG is associated with PTSD symptom severity. The more important question is whether PTG leads to greater PTSD symptom severity or whether it can lead to fewer symptoms of the disorder over time, or whether the reverse is true. Questions of causality cannot be evaluated using cross-sectional designs (Kazdin, 2003).

In a sample of 258 physically injured survivors of community violence (Updegraff & Marshall, 2005), PTG was associated with greater PTSD. Baseline PTSD symptom severity was moderately associated with PTG measured 3-months later. Follow-up PTSD symptom severity and PTG were also moderately associated in the study. Although PTG was not measured at baseline, this study nevertheless indicated that PTG and PTSD symptom severity were related over time and at each measurement occasion.

Two studies reported that PTG was related to less PTSD symptom severity in longitudinal studies. McMillen, Smith, and Fisher (1997) conducted a study of 195 people who were either exposed to a tornado, a plane crash, or a mass shooting incident. Results indicated that those who reported PTG four to six weeks following their traumatic event were 72% less likely to meet criteria for PTSD three years later. Frazier, Conlon, and Glaser (2001) conducted a study of survivors of sexual assault. Positive changes in areas of self and spirituality were associated with less PTSD symptom severity. The study also indicated that people who reported PTG at 2-weeks post-trauma
and 12-months post-trauma reported significantly less distress than people who never reported PTG or who stopped reporting PTG during the course of the study.

Two studies reported no association between PTG and PTSD symptom severity over time. Ai and colleagues (Ai, Tice, Whitsett, Ishisaka, & Chim, 2007) conducted a longitudinal study of 50 Kosovar war refugees. Baseline PTG was unrelated to PTSD symptom severity assessed 10 months following initial interview. In a study of 83 adults who suffered traumatic limb amputation, Phelps and colleagues (2008) reported that baseline PTG was not associated with PTSD symptom severity at three-month follow-up interview. The study authors also noted that the PTG reported in the sample was relatively low when compared to other studies of different types of trauma.

The results of these longitudinal studies indicate that, similar to the cross-sectional literature, there exist a mixture of findings regarding the relationship between PTG and PTSD symptom severity. Clearly more longitudinal studies are warranted that measure PTSD symptom severity and its relationship to PTG. Although the timing of PTG with respect to PTSD symptom severity remains a largely unanswered question, it appears that PTG reported within several weeks (Frazier et al., 2001) or months (McMillen et al., 1997) following a traumatic event may lead to more positive outcomes.

The Association of Posttraumatic Growth and PTSD

This next section presents the four longitudinal associations between PTG and PTSD symptom severity and the theory and empirical studies that support each. PTG and PTSD symptom severity can be associated such that 1) PTG leads to less PTSD symptom
severity, 2) PTG leads to greater PTSD symptom severity, 3) PTSD symptom severity leads to greater PTG, and 4) PTG and PTSD symptom severity are unrelated. An additional potential association that has not received theoretical or empirical support is that PTSD symptom severity leads to less PTG.

*PTG was originally seen as relating to less PTSD.* PTG was thought to lead to less PTSD symptom severity via the process of true transcendence, transformation and healing following a PTE. Victor Frankl (1963) witnessed firsthand the horrors of war and the inhumanness man can inflict on man. As a Nazi Concentration Camp internee he also noted how some people were able to cope and survive under constant threat of death and annihilation. Frankl witnessed that people who maintained hope and found greater meaning and appreciation for their lives actively participated in behaviors that continued to imbue life with purpose. As an example of this mode of coping, Frankl quoted Nietzsche: “He who has a why to live for can bear with almost any how.” (Friedrich Nietzsche, quoted in 1963, p. 121).

According to Frankl having meaning in one’s life can serve a protective function against psychological disorder. It follows then, if a person lacks such meaning, distress will follow as emptiness and boredom begets despair (“Sunday neurosis”). Frankl spoke of three values that can aid one in finding meaning. Experiential values involve experiencing something or someone we value, especially through love. Creative values involve becoming active in one’s life and in one’s projects and generally becoming a progenitor of artistic products and good deeds. Frankl also stressed the importance of attitudinal values. Examples include having a good sense of humor and cultivating
compassionate understanding for others. These values generally convey the idea that through “right actions and right conduct” one can gain meaning.

Building upon Frankl’s work, Hobfoll and colleagues (Hall & Hobfoll 2006; Hobfoll et al., 2007) posited that for PTG to relate to fewer symptoms of psychological distress, actions would need to accompany the cognitive meaning making process. According to their model, it is not sufficient to think one has greater meaning and appreciation for life – a cognitive phenomenon – it is also necessary for these thoughts to become actualized through meaningful actions.

Hall and colleagues (Hall et al., 2008) sought to gain initial evidence to support their assertions of action focused growth in a study of 190 Gaza settlers conducted one week prior to the Gaza disengagement. It was hypothesized that settlers who reported greater terrorism-related PTG in the process of a lifestyle that demanded daily actions to face their traumatic circumstances (e.g., settlers actively resisted leaving their homes) would be less likely to develop PTSD than those who did not report PTG from their experiences. Consistent with predictions, people who reported greater PTG were less likely to have probable PTSD.

PTG, especially within the domain of positive changes in relationships to others, is associated with social support (Erbes, Eberly, Dikel, Johnsen, Harris, & Engdahl, 2005). Research has indicated that social support serves an important stress buffering function. For example, a test development study of an inventory of military deployment risk and protective factors was conducted with a sample of 357 Gulf War Veterans (King, King, Vogt, Knight, Samper, 2006). King and colleagues (2006) reported that
deployment and post-deployment social support was related to less PTSD symptom severity, depression, and anxiety. The greater the soldier’s level of received social support when he or she leaves for war and the greater the soldier’s social support when they return, the less psychological distress they endure.

Communal coping (Lyons, Michelson, Sullivan, & Coyne, 1998) is another key feature of social processes that may facilitate or attenuate positive post-trauma adaptation. There are three basic principles of this coping process: Beliefs in the advantageousness of joint effort, communication, and cooperative action-oriented problem solving. Generally, this type of coping style signifies a ‘together we can’ attitude. Lyons and colleagues (1998) hypothesized that engagement in this form of coping has the ability to expand resources, improve the capacity for dealing with stressors, assist in achieving social support and the investment in future available support, and to buffer current stress. PTG may serve as a proxy for this type of coping and could indeed prove more beneficial as communal coping has been linked to positive health outcomes (e.g., Monnier & Hobfoll, 1997).

Paradoxical Effects of Posttraumatic Growth

PTG however, can also lead to greater PTSD symptom severity. This association would indicate that the greater amount of PTG a person reports, the greater their subsequent distress. In order to find new meaning in life and grow from a PTE, a person may have to employ cognitive processes. Calhoun and colleagues (Calhoun, Cann, Tedeschi, & McMillian, 2000; Calhoun & Tedeschi, 2006) incorporated what they
termed “positively oriented rumination” in their model of PTG. According to these authors, the meaning making process invariably involves some degree of life review and this process can be facilitated by what they call constructive rumination. This type of rumination is focused on the positive changes that have come from a traumatic event. Calhoun and Tedeschi (2006) believe that this process is integral in developing new cognitive life schemas that allow for the integration of the trauma and the development of meaning in its wake.

Although conceived of as a positive process, engaging in rumination for the purpose of meaning making has been shown to relate to greater psychological distress. Davis and colleagues (Davis, Wortman, Lehman, and Silver, 2000) conducted a prospective study of 124 parents coping with the sudden loss of an infant. The majority of the parents (77%) indicated that they did not engage in a meaning making process. Baseline results indicated that parents who were engaged in searching for meaning reported greater anger, sadness, anxiety, global distress, and less well-being than parents who reported never searching for meaning or who had stopped searching for meaning during the month post-loss and before the initial assessment. Results of longitudinal analyses indicated that parents who were not searching for and did not find meaning were doing as well across all outcomes as parents who searched for and found meaning. Parents who searched in vain and did not find meaning were doing worse across all outcomes.

Nolen-Hoeksema and colleagues (Nolen-Hoeksema, McBride, & Larson, 1997) investigated the effects of rumination in a longitudinal study of bereaved men who lost
their marital partners. Results of longitudinal regression analyses controlling for baseline measurement indicated that finding meaning in the loss (e.g., thoughts about the relationship, thinking about life without partner, trying to find meaning) was related to greater depression and less well-being 12 months later.

Counterfactual thinking is another example of how rumination can lead to distress. Following traumatic events, people may focus their attention on behaviors that they “should” have done differently in order to forestall the event. They believe that they are to blame for what happened and if only they would have acted differently, a past event would not have occurred. Engaging in this type of thinking has been linked prospectively with greater emotional distress, guilt and feelings of personal responsibility for having experienced a traumatic event (Davis et al., 1996).

Reverse Causation: PTSD symptom severity could also lead to greater PTG.
Studies have begun to explore whether the growth reported actually took place or is an artifact of a self-enhancement bias (i.e., the tendency to give self-reports that are positively biased; Paulhus, 1984). In their reviews of the self-enhancement literature, Taylor and colleagues (Taylor, 1984; Taylor and Brown, 1988) reported that self-enhancement is a fairly ubiquitous phenomenon. People on average tend to hold more positive views about themselves than would actually be considered truthful or factual. They further posit self-enhancement can aid in coping with stress.

Research has demonstrated that people tend to report growth and development over time in order to cultivate more positive views of the current self (Albert, 1977; McFarland & Alvaro, 2000; Ross 1989; Wilson and Ross, 2001). Tedeschi and Calhoun
(1995) also recognized the possibility of cognitive bias stating “…we should consider whether the construal of benefits and the self-perception of growth simply represent another cognitive bias, or is real” (pg. 119, italics in original). Indeed, studies have found that people tend to report greater PTG following events that they retrospectively evaluate as more distressing (Carboon, Anderson, Pollard, Szer, & Seymour, 2005; Erbes et al, 2005; Morris, Shakespeare-Finch, Rieck, & Newbery, 2005; Wild & Pavio, 2003).

McFarland and Alvaro (2000) conducted four studies that examined how self-enhancement may contribute to PTG. The first study found that survivors of trauma were more likely than the acquaintances of survivors to report growth following a traumatic life event (versus a milder stressor). Survivors also reported improvement by derogating their pre-event attributes. People who experienced more stressful events had a tendency to recall more negative pre-event personal characteristics than people who reported experiencing less stressful life events. Study 2 and 3 served to replicate and further bolster the confidence in the results of study 1. In the final study reported, a laboratory manipulation induced feelings of threat regarding the loss of self-esteem in relation to an actual past event in participants’ lives. People who felt greater threat of loss to self-esteem reported more negative past attributes and more improvement over time than people who did not experience those feelings. These findings supported the viewpoint that people use positive illusions to cope with and bolster themselves in response to threatening life experiences.

In order to further explore the relationship between self-enhancement and PTG, Hall and colleagues conducted a study of 245 undergraduate students exposed to a variety
of PTE (Hall, Miller, Laverne, Nemes & Hobfoll, 2008). Results indicated that self-enhancement bias was associated with greater PTG. Results also indicated that the hypothesized interaction of psychological distress experienced at the time of the PTE (i.e., peri-traumatic psychological distress) and self-enhancement bias in the prediction of self-reported PTG was also significant. The interaction demonstrated that low self-enhancers reported the same amount of PTG irrespective of the degree to which they experienced peri-traumatic distress. However, as their report of peri-traumatic distress increased, high self-enhancers reported more PTG.

This finding provided further evidence that people use PTG as a way of coping with more severe stress. When people experience more psychological distress during a traumatic event, they may be motivated to bolster themselves and enhance their self-impression post-event (Ross, 1989). Moreover, these findings call into question whether the PTG reported in this study was simply a function of self-enhancement processes.

**Posttraumatic Growth and PTSD as Orthogonal Constructs**

*PTG has been argued to be unrelated to PTSD symptom severity.* If no association is observed between PTG and PTSD symptom severity, the assumption that Tedeshi and Calhoun (1995) proffered regarding the independence of these constructs would be supported. There is some empirical evidence that an orthogonal relationship exists; however, the majority of studies demonstrate that they are related. If PTG and PTSD are unrelated, they would be considered two distinct processes, but again, this would call into
question the utility of PTG as a phenomenon capable of affecting clinical outcomes (Zoellner and Maercker, 2006).

These multiple pathways between PTSD symptom severity and PTG have still not been formally tested at the same time. Much of the PTG literature is cross-sectional, and with too few participants to evaluate these relationships adequately. Only with a sufficient sample size and multiple measurement waves, can questions about the association between PTSD symptom severity and PTG be fully addressed. Furthermore, testing these pathways simultaneously in a longitudinal design would allow for the evaluation of the relative strength of each pathway, should more than one exist. The current state of the art leaves us uncertain regarding the relatedness between these constructs and this necessitates further research. It also renders investigations into this association exploratory, as sufficient evidence exists to support any of the pathways described above.

*Multicultural Considerations in Posttraumatic Growth*

Whether PTG functions as a risk or protective factor for ethnic minority group members has begun to receive attention in the literature. As a recent meta-analytic study demonstrated, minority group members in the United States reported greater rates of PTG, and minority status was a significant moderator of benefit finding and mental health (Helgeson et al., 2006). It may be possible that minority group members interpret PTG differently than majority group members, and therefore investigations into the role of
minority status as a potential moderator between PTSD symptom severity and PTG, especially in longitudinal studies, is warranted.

Given that the population in Israel is comprised of distinct ethno-cultural groups (i.e., Jews and Palestinian Citizens of Israel; PCI), each with their own unique ideology, religion, and ties to their land, it is important to assess whether PTSD symptom severity and PTG are related in the same way for Jews and PCI. Similar to minority groups in the U.S., PCI occupy an underprivileged status within Israeli society (Canetti-Nisim, Ariely, Halperin, 2008) and are more collectivistic than Israeli Jews (Sagy, Orr, Bar-on, & Awwad, 2001). These similarities to minority groups in other countries may lead PCI to respond differently than Israeli Jews on measures of PTG, as was the case in studies of ethnic minorities in the United States (Helgeson et al., 2006).

In a cross-sectional study of a nationally representative sample in Israel, Jewish Israeli citizens were at greater risk for probable PTSD as their report of PTG increased; however, an association between probable PTSD and PTG was not found for PCI (Hobfoll, Canetti-Nisim, Johnson, Palmieri, Varley, & Galea, 2008). Although this finding demonstrated differences between these two groups with regard to their posttraumatic responses, additional work is needed to determine whether there are differences in the relationship between PTSD symptom severity and PTG for these two groups within a longitudinal framework.

Measurement considerations and cultural issues. The factor structure of PTSD has recently received growing empirical attention (King, King, Leskin & Weathers,
Several alternative models of PTSD that do not follow the *DSM-IV* have been tested, and generally appear to better account for the construct. A two factor model of PTSD that splits the three *DSM-IV* factors into reexperiencing/avoidance and emotional numbing/hyperarousal, has been confirmed in at least two studies (Taylor, Kuch, Koch, Crokett, & Passey, 1998; Buckley, Blanchard, & Hickling, 1998). Alternatively, a four factor model that contains the reexperiencing and hyperarousal clusters and splits the *DSM-IV* avoidance factor into behavioral avoidance and emotional numbing has enjoyed the widest empirical support (King et al., 1998; Schinka, Brown, Borenstein, & Mortimer, 2007; DuHamel et al., 2004; Elhai, Gray, Docherty, Kashdan, & Kose, 2007; Palmieri & Fitzgerald, 2005; Andrews, Joseph, Shevlin, & Troop, 2006). No known study has examined the factor structure of PTSD within Jewish Israeli and PCI adults, and only one has examined the factor structure of PTSD within terrorism context (Palmieri, Weathers, Difede, & King, 2007). Clearly, in order to study the relationship between PTSD and PTG, a necessary precondition is to establish the factor structure of PTSD within these samples (Chan, 1998; Horn & McArdle, 1992; Tyson, 2004; Vandenberg & Lance, 2000).

The cross-cultural variation between Jews and PCI regarding how PTSD is related to PTG and how PTSD and PTG are interpreted are substantive empirical research questions (Byrne & Watkins, 2003). Given the cross-cultural variation between Jews and PCI, establishing measurement equivalence and cross-cultural generalizability are important issues to examine before other meaningful substantive questions can be asked. Indeed, the assessment of measurement invariance is essential when evaluating between-
group differences on psychological measures or theoretical models (Byrne & Watkins, 2003; Chan, 1998; Horn & McArdle, 1992; Tyson, 2004; Vandenberg & Lance, 2000). It is important to ensure that the construct under study is being measured equivalently, and with equivalent accuracy between groups. Invariance of assessment instruments ensures that measurement bias does not exist as a function of group membership (e.g., ethnicity), and that the scores on an instrument reflect the underlying construct under study, unaffected by group membership.

Two levels of invariance in the factor loadings of latent constructs have been identified: configural invariance (i.e., “weak factorial invariance”) and metric invariance (i.e., “strong factorial invariance”) (Horn, & McArdle, & Mason, 1992). Configural invariance – the first type of invariance that is tested, and a minimum condition for factorial invariance – requires that the configuration of the latent factor structure of a latent construct (e.g., the number of factors and the pattern of factor loadings) are equal across groups. Demonstrating configural invariance ensures that the dimensionality of a construct under study is equivalent across groups. Without this type of factorial invariance, comparisons between groups are arbitrary and therefore, cannot be made.

Following configural invariance, and providing that it is upheld, metric invariance is tested and requires that all like-item loadings are equal between groups. Metric invariance upholds assumptions that a change in a score for one group corresponds to an equal change in a score in the other. It measures the strength of the association between an item and its loading on a latent variable. This is considered to be a more stringent form of factorial invariance.
Temporal invariance evaluates whether configural or metric invariance is observed across measurement occasions of a longitudinal study. This form of invariance is crucial when changes over time are evaluated, as changes in a construct can either be attributed to *true* change, or other sources of potential measurement bias such as time, or group membership.

To date, no research has evaluated whether Jews and PCI respond similarly on self-report measures of PTSD or PTG. Furthermore, when evaluating associations between variables, it is desirable to demonstrate the degree to which findings can generalize to members of minority groups.

*The Present Study*

The purpose of the present study is to evaluate the potential relationships between PTSD symptom severity and PTG within a longitudinal framework. Confirmatory factor analysis (CFA) was used to identify the best fitting latent models for PTSD and PTG. Ethnicity was examined as a potential moderator of the relationship between these constructs after configural and metric factorial invariance was established, a necessary preliminary step in evaluating potential moderation.

The current study had 3 Aims. Aim 1 evaluated whether the PTSD and PTG constructs were similar to prevailing models of PTSD found in the published literature, and further to establish whether these constructs were invariant between ethnic minority group members (i.e., PCI) and ethnic majority members (i.e., Jews). Aim 2 evaluated whether there is a temporal relationship between PTSD symptoms and PTG. Given that no other studies have evaluated this relationship, and indeed the studies that have
evaluated the relationship between PTSD symptom severity and PTG report mixed findings based largely on cross-sectional studies, directional hypotheses were not made. Finally, Aim 3 evaluated whether ethnic minority individuals were more likely to benefit from PTG.

Method

Data Collection and Sample

A nationally representative sample of 1613 Israelis was randomly surveyed. Phone interviews were conducted on three measurement occasions (wave 1, between August 17 and September 8, 2004; wave 2, between February 22, 2005 and March 13, 2005; and, wave 3, between July 31 and October 9, 2005) by a survey institute in Israel using a structured questionnaire that was carefully pilot-tested and used in prior studies (Hobfoll et al., 2006). All scales were translated and back translated by language experts and it was completed by participants in 30-40 minutes. Initial contact was made by a Hebrew speaker; Arabic and Russian speakers were available if individuals did not speak Hebrew, and callbacks were arranged within 24 hours if a Russian or Arabic speaker was not immediately available. Fifteen attempts were made to contact an adult at each telephone number. At the onset of the interview, oral informed consent was obtained. Mental health referrals were made if interviewees requested such a referral or became upset during the interview.

The response rate for eligible participants was 57% (Hall et al., 2009; Hobfoll et al., 2008). The response rate of all potentially working numbers was 37% (i.e., not all
working numbers had adults who spoke Hebrew or Arabic). The rate compared favorably with other studies conducted on adults following the September 11, terror attacks within the United States (Galea et al., 2002). The dialing methods in Israel, unlike the United States include business phones (approximately 10%) which are treated as a failed attempt. The higher response rates in United States do not typically include non-answered phones (Galea et al., 2002; Stuber, Galea, Boscarino, & Schlesinger, 2006). Participation rates between 30% and 70% are only weakly associated with survey bias, and any potential bias in sampling is addressed by examining the representativeness of the obtained sample (Galea & Tracy, 2007). There were no statistical differences between the current sample and the 2003 Israeli Census in terms of sex, ethnicity, age, and education. Of the 1613 (1136 Jews; 477 PCI) participants in the first wave of data collection, 840 (52% overall; 54% Jew; 48% PCI) were retained for 6-month follow-up. The majority of these individuals (716 people, 85% overall; 94% Jews; 62% PCI) were retained at 12-month follow-up.

**Instruments**

The questionnaire included measures of exposure to terrorism, PTSD symptom severity, and PTG. Demographic information was obtained regarding participants’ age, gender, income, and educational attainment.

*Exposure to terrorism.* Participants were asked whether they had been exposed to various terrorism-related events ever, and during the three months prior to the phone interview. They were asked whether they experienced a death of a family member or a friend, witnessed a terrorist attack or had been present at a site where there were injuries
or fatalities, experienced an injury, or experienced a period of time when they did not know if someone close to them was killed or injured, but feared they might be, whether they had to take bus routes or go to places that had been targets of attack, or if family had to take bus routes or go to places that had been targets of attack, or whether they happened to be at a place or on a bus route within 48 hours before it was the target of a terrorist attack or an act of war. One summed score was created for each measurement occasion that represented the total number of exposures to terrorism and the total number of exposures within three months before the start of the study, or since the last measurement occasion for 6-month and 12-month follow-up. These events satisfy the A1 criteria for potentially traumatic events that could lead to a diagnosis of PTSD as outlined in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, text revision* (*DSM-IV-TR*; American Psychiatric Association, 2000).

*Posttraumatic stress disorder.* PTSD symptom severity was measured using the posttraumatic stress disorder symptom scale, interview format (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993). This scale has been used previously in non-Western, low income regions (Johnson & Thompson, 2008) and within the Israeli population (including both Palestinians and Jews; Hobfoll et al., 2006; Canetti, Galea, Hall, Johnson, Palmieri, & Hobfoll, in press; Hall et al., 2008; Palmieri et al., 2008). Participants reported on the severity of PTSD symptoms occurring for at least one month relating to experiences involving a terrorist attack. The PSS-I contains 17 items that assess PTSD symptom criteria based on the *DSM-IV-TR* (American Psychiatric Association, 2000). Items were
answered on a 4-point scale ranging from 0 not at all to 3 extremely. The measure was given at each measurement occasion.

Posttraumatic growth. Four items, rated from 0 not at all to 3 extremely from the COR-Evaluation (Hobfoll & Lilly, 1993) were used to assess posttraumatic growth. To assess changes experienced owing to Intifada-related terrorism exposure, items were all prefaced with “As a result of the Intifada, I have more…. The individual items were as follows: “intimacy with one or more family members,” “closer relationship with friends,” “feelings that my life has purpose,” “more confidence in my ability to do things.” This brief scale captures growth in the three domains of self-perception, interpersonal relationships, and philosophy of life posited by Tedeschi and Calhoun (1995) and has demonstrated adequate psychometric properties (α = .82) in studies previously conducted in Israel (Hobfoll et al., 2006). A recent reliability and validity study (Hall & Hobfoll, 2008) was conducted with 245 undergraduate students who reported experiencing a variety of traumatic and stressful life events to determine the relatedness between this brief scale and Tedeschi and Calhoun’s (1996) Posttraumatic Growth Inventory (PTGI) which was also developed in an undergraduate sample of similar composition (i.e., similar in terms of age, ethnicity, and type of traumatic event exposure). The two scales were highly correlated (r = .85) indicating that they measure highly similar constructs. Limitations of questionnaire space in the current study precluded the use of the 21-item PTGI in this investigation. The four COR-evaluation items were given at each measurement occasion.

Statistical Analyses
Sample Size Requirements for Structural Equation Modeling

Although the treatment of statistical power and the related issue of adequate sample size are thoroughly addressed for many other correlational statistical techniques (Cohen, 1992), structural equation modeling (SEM) does not have the same breadth of treatment of this topic. Kline (2005) suggested that sample sizes of fewer than 100 participants be considered “small,” 100 to 200 “medium,” and 300 and greater considered “large.” Generally, SEM is considered a “large sample” statistical technique as the complexity of a specified model can attenuate the reliability of model coefficients in smaller samples; therefore, sample sizes should increase as the number of parameter estimates increase – a ratio specified as $N:q$ – where $N$ indicates the size of the sample and $q$ represents the number of parameters in a model.

Based on this ratio, researchers have adopted a general rule-of-thumb has been adopted by researchers that specifies 10 participant cases to each parameter that is estimated in a path model. This $N:q$ ratio has not received ample statistical validation, and indeed Jackson (2001) suggested that it may not be an adequate approach. Based on data from true models (Jackson, 2001) and models based on simulated data (Jackson, 2003), sample sizes ranging from 200 to 400 participants were deemed sufficient for many models. However, special consideration should also be paid to the indicator loadings of latent variables, and when factor to variable paths average less than .60, it may be necessary to consider larger than 400 participants in order to evaluate model misspecification (Jackson, 2003).
In addition to considering the adequacy of sample size, the analysis of large samples can also be considered problematic as significance tests can be both a product of either large samples or large effect sizes (i.e., the likelihood of Type II error increases as sample size increases). Rather than evaluate a model based on the Chi-square statistic alone – a test statistic that is particularly vulnerable to Type II error when sample sizes are large – examining the various fit statistics together can aid in the interpretation of model fit.

**Missing Data**

Missing data is a relatively common occurrence in most research, but it is especially problematic in longitudinal designs. Listwise deletion, pairwise deletion, mean substitution, and last observation carried forward are all examples of common approaches to missing data in longitudinal designs (Wothke, 2000). Each of these procedures introduces bias in statistical results (e.g., biased parameter estimates) and may lead to spurious results, especially in the instance where the data is not missing completely at random (MCAR; Cook, Zeng, Yi, 2004; Little & Rubin, 1987). MCAR is a difficult standard to reach in many applied data sets (Muthén, Kaplan, & Hollis, 1987), and indicates that missing values of one variable are entirely unrelated to other variables, and to itself.

Sophisticated and “modern” techniques for handling missing data have emerged that greatly reduce bias and are preferable to the “traditional” approaches mentioned above. These approaches rely on a more probable assumption of data being missing at random (MAR), an untestable assumption that the probability of data missingness for a
variable is unrelated to itself (but can be related to other variables under study) (Rubin, 1976).

Full information maximum likelihood estimation (FIML) offers distinct advantages to the other forms of missing data handling mentioned previously (Enders & Bandalos, 2001). FIML is a non-imputation based method (although it does imply imputation via conditional expectation of the missing value) of parameter estimation for missing data. Enders and Bandalos describe it technically as involving the computation of a “casewise likelihood function using only those variables that are observed” for a certain case (Enders & Bandalos, 2001). This technique creates parameter estimates in a SEM model by utilizing all of the available data for a participate case and this information is aggregated across cases.

For example, in a longitudinal framework, if participant x only has data on variable y at T1, that information will be contributed to the parameter estimation of y and all other parameters in the model via the conditional correlation between variable y and the other variables in the model, based on observed associations. FIML is therefore an efficient missing data handling technique as it fully utilizes all available data (cf. listwise deletion). Furthermore, simulation studies have shown FIML to yield unbiased parameter estimates under the conditions of MCAR and the less restrictive MAR (Arbuckle, 1996; Graham et al., 1996; Muthén et al, 1987; Wothke, 2000). Muthen and Muthen (2007) recommend that at least 10% covariance coverage is present in analyses utilizing FIML.

Data Analyses
Confirmatory factor analyses were conducted to address Aim 1, to evaluate whether the PTSD and PTG constructs were similar to prevailing models of PTSD found in the published literature, and further to establish whether these constructs were invariant between ethnic minority group members (i.e., PCI) and ethnic majority members (i.e., Jews). In order to establish the best fitting model of the PTSD factor structure, the following confirmatory factor analyses (CFA) were conducted separately for Jews and PCI utilizing data from the first study wave: Model 1: one factor model of PTSD with 17 items, Model 2: two factor correlated model of PTSD with reexperiencing symptoms and avoidance symptoms comprising one factor (7 items), and numbing and hyperarousal comprising the other (10 items), Model 3: three factor correlated model of PTSD following the current DSM-IV diagnostic nomenclature of reexperiencing (five items), avoidance (seven items) and hyperarousal (five items), and Model 4: four factor model of PTSD with reexperiencing symptoms (five items), hyperarousal (five items), numbing (five items) and avoidance (two items).

Given that the instrument used to measure PTG has not previously been evaluated in terms of its dimensionality, an exploratory factor analysis was first conducted on the four items. The result of this analysis was then submitted to a CFA to establish the fit of this model for Jews and PCI separately.

Multigroup Longitudinal CFA. Before examining the structural models, the configural, metric and temporal invariance of PTSD and PTG was evaluated in multigroup longitudinal measurement models, utilizing the fully latent models identified in the previous analyses (Byrne & Stewart, 2006). This follows a widely accepted two-
step procedure wherein the measurement model is first evaluated, and after establishing a
good fitting model, the structural model is tested. This approach ensures that any source
of poor model fit is attributed to the casual model, and not from misspecification of the
measurement model (Anderson & Gerbing, 1988).

First, longitudinal configural invariance of PTG and PTSD was established by
estimating these latent constructs at each measurement occasion allowing all residual
errors of the like-items measured at each time to freely correlate (i.e., correlated
uniquenesses) for both groups. This served as the base model with which all subsequent
more constrained measurement models were compared. Next, the temporal metric
invariance of PTSD and PTG was evaluated by fixing the unstandardized factor loadings
for each of the latent variables to be equal over time. Multigroup temporal metric
invariance was next tested by fixing the item loadings for each of the latent constructs to
be equal for Jews and PCI. Establishing metric invariance between Jews and PCI is a
necessary precondition for testing multigroup structural models (Byrne & Watkins, 2003;
Chan, 1998; Horn & McArdle, 1992; Tyson, 2004; Vandenberg & Lance, 2000; Marsh,
1994). If constructs are not invariant, the structural pathways cannot be clearly
interpreted because identical constructs do not exist in each group, and therefore cannot
be compared.

*Cross-lagged panel analyses were conducted to address Aim 2, to evaluate
whether there is a temporal relationship between PTSD symptoms and PTG, and Aim 3,
to evaluate whether ethnic minority individuals were more likely to benefit from PTG.*
The relationship between PTSD symptomatology and PTG concurrently and over the
span of one year utilizing three measurement occasions was evaluated with a fully latent cross-lagged panel analysis based on the previously defined measurement models for PTSD and PTG. A cross-lagged panel design allows for the evaluation of causal linkages between constructs over time. One strength of the design is that autoregressive paths (i.e., paths that regress the later measurement of a construct on itself at the preceding measurement occasion) are included. Therefore, the effect of the construct serving as the independent variable from the previous measurement lag is isolated, controlling for the autoregressive effect. A second strength of this model is that bidirectional pathways are estimated so each construct in the model serves as an independent variable at the earlier measurement lag, so all possible relationships can be evaluated.

Structural models were tested that first examined the best-fitting fully latent PTSD construct with PTG, and then followed with examining each identified symptom cluster of PTSD with PTG separately. Covariates (age, sex, income, trauma exposure) were entered into the models to control for their influence on each latent construct, at each measurement wave.

The cross-lagged model paths were evaluated in a multistep nested models procedure. The multigroup models were tested with all autoregressive paths, all cross lagged paths, and all correlated like-item residuals free to vary between groups. This saturated model served as the base model for further between group model comparisons. Four additional models were specified, each constraining one pathway at equivalence for Jews and PCI. These models were used to evaluate whether a cross-lagged pathway was equal in terms of significance and strength between Jews and PCI.
Evaluation of model fit and missing data. All analyses were conducted using Full information Maximum Likelihood (FIML) for missing data (covariance coverage ranged from 25% to 100% in the current study), MLR estimation (i.e., robust methods) was used for non-normally distributed data, using Mplus version 5.1 (Muthen & Muthen, 2007). Six goodness-of-fit indices were used to evaluate the adequacy of the CFA and structural models: the comparative fit index (CFI; Bentler, 1990), the Tucker Lewis Index (TLI; Bentler & Bonett, 1980; Tucker & Lewis, 1973), the standardized root mean square residual (SRMR), the root mean square of approximation (RMSEA; Steiger, 1990). Values equal to, or greater than, .90 for the CFI and TLI, and values lower than .08 for the RMSEA and SRMR, were considered indicators of model goodness-of-fit (Bentler, 1990; Browne & Cudeck, 1993; Vandenberg & Lance, 2000). Nested CFA and cross-lagged models were be compared using Sattora-Bentler χ² difference test (S-B Δχ²) incorporating the scaling correction factor for non-normal data provided by Mplus (Bollen, 1989), with a p value set at .05. Changes in the CFI, TLI, RMSEA, SRMR, and the Bayesian information criterion (BIC; Muthen & Muthen, 1998) values were also evaluated (Cheung & Rensvold, 2002). Although not an indicator of model fit when evaluated alone, when evaluating nested models, lower relative BIC values indicate an improvement in model fit.

Results

Descriptive and Bivariate Analyses

Table 1 presents the demographic composition of the sample for Jews and PCI and presents the means, standard deviations, and percentages of study variables. The sample
consisted of 1136 Jewish adults and 477 Arab adults. The sex composition was 53% female for Jews and 52% for PCI. The mean age for Jews was 43.38 (SD =16.56) and 33.28 (11.75) for PCI. In terms of reported highest level of education for Jews, 3.7% finished elementary school, 55.9% finished high school, and 40% had a college degree. For PCI, 12.6% finished elementary school, 60% finished high school, and 26.9% had a college degree. Compared to being single/widowed/divorced, the majority of Jews (64%) and PCI (66%) were married. The annual household income for Jews was 34.4% below average (i.e., 8,000 New Israeli Shekel), 22.1% average, and 33.1% above average. For PCI, the majority reported being below average (65%) with 18.7% being average, and 14.3% reporting being above average. With regard to ever being exposed to terrorism, 20% of the Jewish sample reported none, 48% reported exposure to 1 event, 22.5% were exposed to 2 events, and 9.4 reported being exposed to 3 or more events. For the PCI sample, 44.2% reported no exposures, 43.6 reported 1 exposure, 8.6% 2 or more exposures, and 3.5% of the sample reported 3 or more exposures. In terms of recent exposure to terrorism, 35% of people in the Jewish sample reported no exposure, 23.6% exposure to 1 event, 21.3% exposure to 2 events, and 11% reported being exposed to 3 or more events. For the PCI sample, 49.7% reported no exposure, 11.9% exposure to 1 event, 3.6% exposure to 2 events, and 2% reported being exposed to 3 or more events.
Table 1. Means, standard deviations of study variables and demographic composition of the sample.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Jews (n = 1136)</th>
<th>Arabs (n = 477)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>M (SD)</td>
</tr>
<tr>
<td>Age</td>
<td>45.38 (16.56)</td>
<td>18 – 96</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>53.0</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elementary</td>
<td>3.7</td>
<td>12.6</td>
</tr>
<tr>
<td>High School/Post High School</td>
<td>55.9</td>
<td></td>
</tr>
<tr>
<td>College Graduate</td>
<td>40.0</td>
<td></td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single/widowed/divorced</td>
<td>35.0</td>
<td></td>
</tr>
<tr>
<td>Married/cohabitating</td>
<td>64.0</td>
<td></td>
</tr>
<tr>
<td>Yearly Household Income</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Below Average</td>
<td>34.4</td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>22.1</td>
<td></td>
</tr>
<tr>
<td>Above Average</td>
<td>33.1</td>
<td></td>
</tr>
<tr>
<td>T1 Exposure to Terrorism (Ever)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exposure reported</td>
<td>20.1</td>
<td></td>
</tr>
<tr>
<td>Exposure to 1 event</td>
<td>48.0</td>
<td></td>
</tr>
<tr>
<td>Exposure to 2 events</td>
<td>22.5</td>
<td></td>
</tr>
<tr>
<td>Exposure to 3 or more events</td>
<td>9.4</td>
<td></td>
</tr>
<tr>
<td>T1 Exposure to Terrorism (Recent)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exposure reported</td>
<td>44.1</td>
<td></td>
</tr>
<tr>
<td>Exposure to 1 event</td>
<td>23.6</td>
<td></td>
</tr>
<tr>
<td>Exposure to 2 events</td>
<td>9.8</td>
<td></td>
</tr>
<tr>
<td>Exposure to 3 or more events</td>
<td>1.3</td>
<td></td>
</tr>
<tr>
<td>T2 Exposure to Terrorism (Last six months)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exposure reported</td>
<td>24.4</td>
<td></td>
</tr>
</tbody>
</table>
Table 1 (continued). Means, standard deviations of study variables and demographic composition of the sample.

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure to 1 event</td>
<td>11.5</td>
<td>11.5</td>
<td>11.5</td>
</tr>
<tr>
<td>Exposure to 2 events</td>
<td>9.0</td>
<td>9.0</td>
<td>9.0</td>
</tr>
<tr>
<td>Exposure to 3 or more</td>
<td>5.0</td>
<td>5.0</td>
<td>5.0</td>
</tr>
<tr>
<td>PCI</td>
<td>41.0</td>
<td>41.0</td>
<td>41.0</td>
</tr>
<tr>
<td>PTG</td>
<td>5.2</td>
<td>5.2</td>
<td>5.2</td>
</tr>
<tr>
<td>T3 Exposure to Terrorism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Last six months)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exposure reported</td>
<td>1.4</td>
<td>1.4</td>
<td>1.4</td>
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<tr>
<td>Exposure to 1 event</td>
<td>1.4</td>
<td>1.4</td>
<td>1.4</td>
</tr>
<tr>
<td>Exposure to 2 events</td>
<td>1.4</td>
<td>1.4</td>
<td>1.4</td>
</tr>
<tr>
<td>Exposure to 3 or more</td>
<td>0.6</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>T1 Reexperiencing</td>
<td>3.14 (3.54)</td>
<td>2.72 (3.39)</td>
<td>2.30 (3.27)</td>
</tr>
<tr>
<td></td>
<td>0 – 15</td>
<td>0 – 15</td>
<td>0 – 15</td>
</tr>
<tr>
<td>T2 Reexperiencing</td>
<td></td>
<td>3.78 (3.93)</td>
<td>4.23 (4.07)</td>
</tr>
<tr>
<td></td>
<td>0 – 15</td>
<td>0 – 15</td>
<td>0 – 15</td>
</tr>
<tr>
<td>T1 Behavioral Avoidance</td>
<td>2.29 (2.90)</td>
<td>2.02 (2.65)</td>
<td>1.84 (2.59)</td>
</tr>
<tr>
<td></td>
<td>0 – 6</td>
<td>0 – 6</td>
<td>0 – 6</td>
</tr>
<tr>
<td>T2 Behavioral Avoidance</td>
<td></td>
<td>5.01 (3.37)</td>
<td>5.58 (3.38)</td>
</tr>
<tr>
<td></td>
<td>0 – 6</td>
<td>0 – 6</td>
<td>0 – 6</td>
</tr>
<tr>
<td>T1 Emotional Numbing</td>
<td>1.50 (1.94)</td>
<td>1.28 (1.80)</td>
<td>1.11 (1.81)</td>
</tr>
<tr>
<td></td>
<td>0 – 15</td>
<td>0 – 15</td>
<td>0 – 15</td>
</tr>
<tr>
<td>T2 Emotional Numbing</td>
<td></td>
<td>2.10 (2.13)</td>
<td>2.37 (2.24)</td>
</tr>
<tr>
<td></td>
<td>0 – 15</td>
<td>0 – 15</td>
<td>0 – 15</td>
</tr>
<tr>
<td>T1 Hyperarousal</td>
<td>3.27 (3.52)</td>
<td>3.30 (3.44)</td>
<td>3.03 (3.54)</td>
</tr>
<tr>
<td></td>
<td>0 – 15</td>
<td>0 – 15</td>
<td>0 – 15</td>
</tr>
<tr>
<td>T2 Hyperarousal</td>
<td></td>
<td>6.57 (3.92)</td>
<td>7.82 (3.77)</td>
</tr>
<tr>
<td></td>
<td>0 – 15</td>
<td>0 – 15</td>
<td>0 – 15</td>
</tr>
<tr>
<td>T1 PTG</td>
<td>3.33 (3.63)</td>
<td>3.14 (3.39)</td>
<td>3.14 (3.39)</td>
</tr>
<tr>
<td></td>
<td>0 – 12</td>
<td>0 – 12</td>
<td>0 – 12</td>
</tr>
<tr>
<td>T2 PTG</td>
<td></td>
<td>5.21 (3.97)</td>
<td>6.26 (4.00)</td>
</tr>
<tr>
<td></td>
<td>0 – 12</td>
<td>0 – 12</td>
<td>0 – 12</td>
</tr>
<tr>
<td>T3 PTG</td>
<td>2.29 (3.24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0 – 12</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Not all percentages add to 100 owing to missing data. T1 = baseline interview. T2 = 6-month follow-up. T3 = 12-month follow-up. PCI = Palestinian Citizen of Israel. PTG = posttraumatic growth.
Identifying the best fitting model for PTSD. As can be seen in Table 2, of the latent models of PTSD tested, the four factor model of PTSD with correlated reexperiencing, avoidance, numbing and hyperarousal factors fit the data best for Jews ($\chi^2(112, N = 1136) = 369.217$, CFI = .93, TLI = .91, RMSEA = .045 (.040 - .050) SRMR = .039) and PCI ($\chi^2(112, N = 477) = 238.120$, CFI = .91, TLI = .88, RMSEA = .049 (.040 - .057) SRMR = .05).

Table 2. Identification of the Best Fitting Model for PTSD for Jews and PCI

<table>
<thead>
<tr>
<th>Model</th>
<th>S-B $\chi^2$</th>
<th>Df</th>
<th>Scaling factor</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
<th>SRMR</th>
<th>BIC</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Jews</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (one factor Global PTSD)</td>
<td>559.23</td>
<td>118</td>
<td>1.508</td>
<td>.88</td>
<td>.86</td>
<td>.057 (.053 - .062)</td>
<td>.048</td>
<td>48024.84</td>
</tr>
<tr>
<td>Model 2 (two factor reexperiencing/avoidance and numbing/hyperarousal)</td>
<td>441.25</td>
<td>117</td>
<td>1.502</td>
<td>.91</td>
<td>.90</td>
<td>.049 (.045 - .054)</td>
<td>.042</td>
<td>47851.42</td>
</tr>
<tr>
<td>Model 3 (three factor DSM)</td>
<td>472.74</td>
<td>115</td>
<td>1.503</td>
<td>.90</td>
<td>.88</td>
<td>.052 (.047 - .057)</td>
<td>.044</td>
<td>47913.01</td>
</tr>
<tr>
<td><strong>Model 4 (four factor reexperiencing, avoidance, numbing, and hyperarousal)</strong></td>
<td><strong>369.22</strong></td>
<td><strong>112</strong></td>
<td><strong>1.492</strong></td>
<td><strong>.93</strong></td>
<td><strong>.91</strong></td>
<td><strong>.045 (.040 - .050)</strong></td>
<td><strong>.039</strong></td>
<td><strong>47774.65</strong></td>
</tr>
<tr>
<td><strong>PCI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (one factor Global PTSD)</td>
<td>429.58</td>
<td>118</td>
<td>1.098</td>
<td>.76</td>
<td>.73</td>
<td>.074 (.067 - .082)</td>
<td>.064</td>
<td>24330.19</td>
</tr>
<tr>
<td>Model 2 (two factor reexperiencing/avoidance and numbing/hyperarousal)</td>
<td>350.71</td>
<td>117</td>
<td>1.097</td>
<td>.82</td>
<td>.80</td>
<td>.065 (.057 - .073)</td>
<td>.058</td>
<td>24249.57</td>
</tr>
<tr>
<td>Model 3 (three factor DSM)</td>
<td>326.76</td>
<td>115</td>
<td>1.094</td>
<td>.84</td>
<td>.81</td>
<td>.062 (.054 - .070)</td>
<td>.056</td>
<td>24234.67</td>
</tr>
<tr>
<td><strong>Model 4 (four factor reexperiencing, avoidance, numbing, and hyperarousal)</strong></td>
<td><strong>238.12</strong></td>
<td><strong>112</strong></td>
<td><strong>1.089</strong></td>
<td><strong>.91</strong></td>
<td><strong>.88</strong></td>
<td><strong>.049 (.040 - .057)</strong></td>
<td><strong>.047</strong></td>
<td><strong>24155.07</strong></td>
</tr>
</tbody>
</table>

*Note. S-B $\chi^2$ Sattora-Bentler $\chi^2$ test. CFI = comparative fit index. TLI = Tucker Lewis Index. SRMR = standardized root mean square residual. RMSEA = root mean square of approximation. BIC = Bayesian information criterion. PCI = Palestinian Citizen of Israel. Model in bold is the best fitting model for each group.*
All factor loadings for each of the four latent factors were significant, and above .44 for Jews (please see Table 3).

Factor loadings for PCI were also all significant and above .44, save for numbing symptoms of feeling of detachment or estrangement from others (.37) and restricted range of affect (.29).

Table 3. Standardized Factor Loadings for Jews and PCI for the Best Fitting PTSD model

<table>
<thead>
<tr>
<th>Individual PTSD Items</th>
<th>Reexperiencing</th>
<th>Behavioral Avoidance</th>
<th>Emotional Numbing</th>
<th>Hyperarousal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. (Recurrent thoughts of trauma)</td>
<td>.61/.55</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. (Recurrent dreams of trauma)</td>
<td>.55/.54</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. (Flashbacks)</td>
<td>.63/.56</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. (Emotional reactivity to trauma cues)</td>
<td>.72/.67</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. (Physiological reactivity to trauma cues)</td>
<td>.63/.52</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. (Avoiding thoughts of trauma)</td>
<td></td>
<td>.79/.86</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. (Avoiding reminders of trauma)</td>
<td></td>
<td>.65/.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. (Inability to recall aspects of trauma)</td>
<td></td>
<td></td>
<td>.44/.53</td>
<td></td>
</tr>
<tr>
<td>9. (Diminished interest in activities)</td>
<td></td>
<td></td>
<td>.65/.50</td>
<td></td>
</tr>
<tr>
<td>10. (Detachment/estrangement)</td>
<td></td>
<td></td>
<td>.53/.37</td>
<td></td>
</tr>
<tr>
<td>11. (Restricted range of affect)</td>
<td></td>
<td></td>
<td>.47/.29</td>
<td></td>
</tr>
<tr>
<td>12. (Sense of foreshortened future)</td>
<td></td>
<td></td>
<td>.60/.45</td>
<td></td>
</tr>
<tr>
<td>13. (Sleep disturbance)</td>
<td></td>
<td></td>
<td>.62/.59</td>
<td></td>
</tr>
<tr>
<td>14. (Irritability)</td>
<td></td>
<td></td>
<td>.65/.65</td>
<td></td>
</tr>
<tr>
<td>15. (Difficulty concentrating)</td>
<td></td>
<td></td>
<td>.56/.44</td>
<td></td>
</tr>
<tr>
<td>16. (Hypervigilance)</td>
<td></td>
<td></td>
<td>.64/.49</td>
<td></td>
</tr>
<tr>
<td>17. (Exaggerated startle response)</td>
<td></td>
<td></td>
<td>.58/.52</td>
<td></td>
</tr>
</tbody>
</table>

*Note. PTSD = posttraumatic stress disorder. PCI = Palestinian Citizens of Israel. All values to the left of the diagonal line represent the standardized coefficients for the Jewish sample, values to the left are for the PCI sample. All values are significant at $p < .001$. 
For each of the models, modification indices called for the residual error for these same items (i.e., feeling of detachment or estrangement from others and restricted range of affect) to be freely estimated. This pathway was introduced in the models given the considerable conceptual overlap of these items and the boon in model fit this provided.

Correlations among the latent factors are shown in Table 4.

Table 4. Correlations among the PTSD factors for Jews and PCI

<table>
<thead>
<tr>
<th></th>
<th>Reexperiencing</th>
<th>Behavioral Avoidance</th>
<th>Emotional Numbing</th>
<th>Hyperarousal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reexperiencing</td>
<td>1</td>
<td>.56</td>
<td>.83</td>
<td>.58</td>
</tr>
<tr>
<td>Behavioral Avoidance</td>
<td>.83</td>
<td>1</td>
<td>.43</td>
<td>.29</td>
</tr>
<tr>
<td>Emotional Numbing</td>
<td>.76</td>
<td>.67</td>
<td>1</td>
<td>.73</td>
</tr>
<tr>
<td>Hyperarousal</td>
<td>.84</td>
<td>.66</td>
<td>.85</td>
<td>1</td>
</tr>
</tbody>
</table>

*Note.* PTSD = posttraumatic stress disorder. PCI = Palestinian Citizens of Israel. All values in bold represent the correlation coefficients among the factors for the Jewish sample, those not in bold are for the PCI sample. All values are significant at $p < .001$.

**Identifying the latent factor structure of PTG.**

A CFA was specified for Jews and PCI separately to test whether the factor structure of these four items best represented by a single latent factor. Results of these analyses demonstrated that a single latent factor with four items fit the data well for Jews $\chi^2(2, N = 1136) = 10.08$, CFI = .99, TLI = .96, RMSEA = .06 (.03 -.09) SRMR = .02, and for PCI $\chi^2(2, N = 477) = 10.56$, CFI = .98, TLI = .92, RMSEA = .09 (.04 -.15), SRMR = .03. Factor loadings for the four PTG items for Jews and PCI respectively were .57/.51 for Feeling that my life has meaning, .78/.72 for Intimacy with one or more family members, .75/.75 for Closer relations with my friends, and .62/.72 for More confidence in my ability to do things.
Correlation analysis results for the variables included in the models are presented in Table 5. The correlations among the study variables for the Jewish sample were all significant cross-sectionally and across the 3 measurement waves. For the Arab sample, the relationships were less consistent both cross-sectionally, and over time.

Table 5. Estimated Correlation Matrix of Latent Study Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Jews</th>
<th>PCI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T1 PTG</td>
<td>T2 PTG</td>
</tr>
<tr>
<td>1. T1 Reexperiencing</td>
<td>.43***</td>
<td>.38***</td>
</tr>
<tr>
<td>2. T2 Reexperiencing</td>
<td>.33***</td>
<td>.41***</td>
</tr>
<tr>
<td>3. T3 Reexperiencing</td>
<td>.33***</td>
<td>.34***</td>
</tr>
<tr>
<td>4. T1 Avoidance</td>
<td>.47***</td>
<td>.38***</td>
</tr>
<tr>
<td>5. T2 Avoidance</td>
<td>.29***</td>
<td>.43***</td>
</tr>
<tr>
<td>6. T3 Avoidance</td>
<td>.35***</td>
<td>.32***</td>
</tr>
<tr>
<td>7. T1 Numbing</td>
<td>.37***</td>
<td>.26***</td>
</tr>
<tr>
<td>8. T2 Numbing</td>
<td>.13**</td>
<td>.32***</td>
</tr>
<tr>
<td>9. T3 Numbing</td>
<td>.17**</td>
<td>.23**</td>
</tr>
<tr>
<td>10. T1 Hyperarousal</td>
<td>.28***</td>
<td>.26***</td>
</tr>
<tr>
<td>11. T2 Hyperarousal</td>
<td>.15**</td>
<td>.32***</td>
</tr>
<tr>
<td>12. T3 Hyperarousal</td>
<td>.19***</td>
<td>.26***</td>
</tr>
</tbody>
</table>

*Note.* T1 = baseline interview. T2 = 6-month follow-up. T3 = 12-month follow-up. PCI = Palestinian Citizen of Israel. PTG = posttraumatic growth. * p < .05. ** p < .01. *** p < .001.

Cross-Lagged Panel Analyses.

Although one of the five proposed crossed-lagged analysis models was to include all of the latent constructs included in the correlated four-factor model of PTSD, this analysis was not able to proceed owing to a non-positive definite covariance matrix. This issue, common with some SEM models, may be due to researcher error (e.g., model misspecification) or may result from issues inherent in the data, and therefore outside of
the control of the researcher. Typically this issue is related to a correlation of above one between latent variables, or a negative residual variance (i.e., Heywood cases; Rindskopf, 1984).

After ruling out researcher error, attempts were made to ease the models’ convergence. The commonly supported method for doing so is to fix the variance of the latent factor to one, and free the factor loadings of each item. This sets the scale of the latent variable to allow for specification, but frees the constraint imposed on the factor loading (L. Muthen, personal communication, March 19th, 2010). This did not fix the problem, so this model was abandoned. It appears that significant multicollinearity is present between the factors. Given that the correlated four-factor structure indicates that each of the latent factors in the model can stand as their own construct, the substantive research questions of this study were addressed by four cross-lagged models, specified by the factors that comprise PTSD.

Reexperiencing symptoms and PTG. The initial multiple group measurement model tested configural invariance over the three study waves. The factor loadings for PTSD and PTG and the residuals for like-items were allowed to covary freely across Jews and PCI, and over time. Measurement Model 1 fit the data well, $\chi^2(564, N = 1613) = 1001.96$, CFI = .94, TLI = .92, RMSEA = .031 (.028 - .034), SRMR = .05, BIC 76183.98. Results for all reexperiencing and PTG models are displayed in Table 6.
Table 6. Multigroup Cross-lagged Analysis of Reexperiencing Symptoms

<table>
<thead>
<tr>
<th>Model</th>
<th>S-B $\chi^2$</th>
<th>df</th>
<th>Scaling factor</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
<th>SRMR</th>
<th>BIC</th>
<th>S-B $\Delta\chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measurement Models</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (configural)</td>
<td>1001.96</td>
<td>564</td>
<td>1.222</td>
<td>.94</td>
<td>.92</td>
<td>.031 (.028 - .034)</td>
<td>.05</td>
<td>76183.98</td>
<td>--</td>
</tr>
<tr>
<td>Model 2 (temporal invariance)</td>
<td>1029.93</td>
<td>592</td>
<td>1.226</td>
<td>.94</td>
<td>.93</td>
<td>.030 (.027 - .033)</td>
<td>.05</td>
<td>76015.52</td>
<td>28, 21.41</td>
</tr>
<tr>
<td>Model 3 (metric invariance)</td>
<td>1056.56</td>
<td>599</td>
<td>1.226</td>
<td>.94</td>
<td>.93</td>
<td>.031 (.028 - .034)</td>
<td>.05</td>
<td>75996.50</td>
<td>35, 42.32</td>
</tr>
<tr>
<td>Structural models</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (all cross-lagged parameters freed for Jews and PCI)</td>
<td>1103.81</td>
<td>607</td>
<td>1.224</td>
<td>.93</td>
<td>.92</td>
<td>.03 (.03 - 0.04)</td>
<td>.06</td>
<td>75992.81</td>
<td>--</td>
</tr>
<tr>
<td>Model 2 (path from T1 PTG to T2 reexperiencing fixed at equality)</td>
<td>1103.86</td>
<td>608</td>
<td>1.224</td>
<td>.93</td>
<td>.92</td>
<td>.03 (.03 - 0.04)</td>
<td>.06</td>
<td>75985.54</td>
<td>1, .03</td>
</tr>
<tr>
<td>Model 3 (path from T1 reexperiencing to T2 PTG fixed at equality)</td>
<td>1104.39</td>
<td>608</td>
<td>1.224</td>
<td>.93</td>
<td>.92</td>
<td>.03 (.03 - 0.04)</td>
<td>.06</td>
<td>75985.69</td>
<td>1, .47</td>
</tr>
<tr>
<td>Model 4 (path from T2 PTG to T3 reexperiencing fixed at equality)</td>
<td>1104.67</td>
<td>608</td>
<td>1.224</td>
<td>.93</td>
<td>.92</td>
<td>.03 (.03 - 0.04)</td>
<td>.06</td>
<td>75986.42</td>
<td>1, .70</td>
</tr>
<tr>
<td>Model 5 (path from T2 reexperiencing to T3 PTG fixed at equality)</td>
<td>1105.21</td>
<td>608</td>
<td>1.224</td>
<td>.93</td>
<td>.92</td>
<td>.03 (.03 - 0.04)</td>
<td>.06</td>
<td>75987.08</td>
<td>1, 1.14</td>
</tr>
</tbody>
</table>

Note. S-B $\chi^2$ = Sattora-Bentler $\chi^2$. CFI = comparative fit index. TLI = Tucker Lewis Index. RMSEA = root mean square of approximation. SRMR = standardized root mean square residual. BIC = Bayesian information criterion. S-B $\Delta\chi^2$ = Sattora-Bentler $\chi^2$ difference test. PCI = Palestinian Citizen of Israel.

In Measurement Model 2, the factor loadings for PTSD and PTG were constrained to be equivalent across each measurement occasion, but freely estimated between groups, $\chi^2(592, N = 1613) = 1029.93$, CFI = .94, TLI = .93, RMSEA = .030 (.027 - .033), SRMR = .05, BIC 76015.52. The difference between these models was non-significant S-B $\Delta\chi^2(28, N =$
1613) = 21.41, \( p > .05 \), which indicated that constraining these loadings did not damage model fit. Therefore, this model was retained.

In the third and final measurement model, the additional constraint of fixing at equality the factor loadings for each latent factor in the model across groups was imposed. This model fit the data well, \( \chi^2(599, N = 1613) = 1056.56 \), CFI = .94, TLI = .93, RMSEA = .031 (.028 - .034), SRMR = .05, BIC 75996.50. The difference between these models was non-significant S-B \( \Delta \chi^2(35, N = 1613) = 42.32 \), \( p > .05 \), therefore the multigroup cross-lagged model could be estimated. This model was used as the foundation for the cross-lagged models.

The first and most saturated multigroup cross-lagged model allowed all cross-lagged structural paths to be freely estimated for each group. Covariances between latent variables at each measurement occasion were also estimated. This initial model fit the data well, \( \chi^2(607, N = 1613) = 1103.81 \), CFI = .93, TLI = .92, RMSEA = .032 (.029 - .035), SRMR = .06, BIC 75992.81. This fully saturated model is depicted by Figure 1.
Figure 1. Standardized structural equation modeling results of the saturated fully cross-lagged model demonstrating the temporal relationship between reexperiencing symptoms and posttraumatic growth.

Note. All bold cross-lagged model paths are significant for one or both groups in the multigroup models. Dashed lines indicate non-significant path coefficients for both groups. *p < .05. **p < .01. Values to the left of the diagonal line represent standardized path coefficients for Jews, and values to the right represent standardized path coefficients for Arabs. PTG = posttraumatic growth. PTSD – B = posttraumatic stress disorder reexperiencing symptoms.

In order to test whether the cross-lagged relationships among study variables differed for Jews and PCI, structural pathways were fixed at equality between groups, starting with the T2 variables regressed on T1 variables, and then for the T3 variables regressed on T2 variables. When potential covariates were added to the model, the model became unstable and failed to converge. Therefore covariates were omitted from further model specification. Effect sizes for the cross-lagged structural pathways were interpreted with the conventions for regression coefficients (1 - .29 = small, .30 - .49 = medium, and .50 or higher = large, Cohen, 1988).
First, in Model 2, fixing the path from T1 PTG to T2 reexperiencing to equality yielded a non-significant result, S-B $\Delta \chi^2(1, N = 1613) = 0.03, p > .05$. This indicated that the two paths were equivalent for Jews and PCI. The non-significant model paths from the fully saturated model for Jews was ($\beta = -0.01$) and ($\beta = -0.04$) for PCI. These results indicated that T1 PTG did not predict T2 reexperiencing symptoms for Jews or PCI.

In model 3, the path from T1 reexperiencing to T2 PTG was fixed to be equal across groups. Results revealed no significant differences between the two groups for that pathway, S-B $\Delta \chi^2(1, N = 1613) = 0.47, p > .05$. The model paths from the fully saturated model for Jews was ($\beta = .15, p = .004$) and ($\beta = .17, p = .04$) for PCI indicating a small effect size relationship between T1 reexperiencing symptoms predicting T2 PTG existed for both groups.

The path from T2 PTG to T3 reexperiencing symptoms was fixed to equality in Model 4. No significant difference in model fit was noted after fixing this path, S-B $\Delta \chi^2(1, N = 1613) = 0.70, p > .05$, indicating that the two groups did not differ with regard to this path. The paths from the fully saturated model for Jews was ($\beta = -0.04, p > .05$) and ($\beta = 0.07, p > .05$) for PCI indicating that T2 PTG did not predict T3 reexperiencing symptoms for either group.

In the final model, the path from T2 reexperiencing to T3 PTG was fixed at equality. The two groups did not significantly differ with regard to the strength and direction of this path, S-B $\Delta \chi^2(1, N = 1613) = 1.14, p > .05$. However, when the paths from the fully saturated model were examined, it appears that T2 reexperiencing symptoms predicted T3 PTG for Jews ($\beta = 0.26, p < .001$) but not for PCI ($\beta = 0.08, p >$
.05). The apparent discrepancy between the S-B Δχ² test and the difference between the two parameters indicates that although this path for Jews was statistically significantly different from the null hypotheses that the path was equal to 0, the path coefficients for Jews and PCI were not statistically different from each other.

Avoidance symptoms and PTG. The initial multiple group measurement model tested configural invariance over the three study waves. The factor loadings for avoidance symptoms and PTG were estimated freely across Jews and PCI and the residuals for like-items were allowed to covary freely over time. Measurement Model 1 fit the data well, χ²(204, N = 1613) = 330.74, CFI = .97, TLI = .96, RMSEA = .028 (.022 - .033), SRMR = .04, BIC 54686.98. Results for all avoidance and PTG models are displayed in Table 7.
Table 7. Multigroup Cross-lagged Analysis of Behavioral Avoidance Symptoms

<table>
<thead>
<tr>
<th>Model</th>
<th>S-B $\chi^2$</th>
<th>df</th>
<th>Scaling factor</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
<th>SRMR</th>
<th>BIC</th>
<th>S-B $\Delta \chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measurement Models</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (configural)</td>
<td>330.74</td>
<td>204</td>
<td>1.189</td>
<td>.97</td>
<td>.96</td>
<td>.028 (.022 - .033)</td>
<td>.04</td>
<td>54686.91</td>
<td>--</td>
</tr>
<tr>
<td>Model 2 (temporal invariance)</td>
<td>341.67</td>
<td>220</td>
<td>1.188</td>
<td>.97</td>
<td>.92</td>
<td>.026 (.021 - .031)</td>
<td>.04</td>
<td>54581.44</td>
<td>16, 9.30</td>
</tr>
<tr>
<td>Model 3 (metric invariance)</td>
<td>364.46</td>
<td>224</td>
<td>1.187</td>
<td>.97</td>
<td>.96</td>
<td>.028 (.023 - .033)</td>
<td>.04</td>
<td>54578.77</td>
<td>20, 28.90</td>
</tr>
<tr>
<td>Structural models</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (all cross-lagged parameters freed for Jews and PCI)</td>
<td>400.64</td>
<td>232</td>
<td>1.183</td>
<td>.96</td>
<td>.95</td>
<td>.030 (.025 - .035)</td>
<td>.05</td>
<td>54561.11</td>
<td>--</td>
</tr>
<tr>
<td>Model 2 (path from T1 PTG to T2 avoidance fixed at equality)</td>
<td>400.86</td>
<td>233</td>
<td>1.183</td>
<td>.96</td>
<td>.95</td>
<td>.030 (.025 - .035)</td>
<td>.05</td>
<td>54553.94</td>
<td>1, .19</td>
</tr>
<tr>
<td>Model 3 (path from T1 avoidance to T2 PTG fixed at equality)</td>
<td>401.80</td>
<td>233</td>
<td>1.183</td>
<td>.96</td>
<td>.95</td>
<td>.030 (.025 - .035)</td>
<td>.05</td>
<td>54554.83</td>
<td>1, .98</td>
</tr>
<tr>
<td>Model 4 (path from T2 PTG to T3 avoidance fixed at equality)</td>
<td>400.91</td>
<td>233</td>
<td>1.183</td>
<td>.96</td>
<td>.95</td>
<td>.030 (.025 - .035)</td>
<td>.05</td>
<td>54553.97</td>
<td>1, .23</td>
</tr>
<tr>
<td>Model 5 (path from T2 avoidance to T3 PTG fixed at equality)</td>
<td>400.52</td>
<td>233</td>
<td>1.184</td>
<td>.96</td>
<td>.95</td>
<td>.030 (.025 - .035)</td>
<td>.05</td>
<td>54553.91</td>
<td>1, .08</td>
</tr>
</tbody>
</table>

Note. S-B $\chi^2$ = Sattora-Bentler $\chi^2$. CFI = comparative fit index. TLI = Tucker Lewis Index. RMSEA = root mean square of approximation. SRMR = standardized root mean square residual. BIC = Bayesian information criterion. S-B $\Delta \chi^2$ = Sattora-Bentler $\chi^2$ difference test. PCI = Palestinian Citizen of Israel.

In Measurement Model 2, the factor loadings for avoidance symptoms and PTG were constrained to be equivalent across each measurement occasion, but freely estimated between groups, $\chi^2(220, N = 1613) = 341.67$, CFI = .97, TLI = .96, RMSEA = .026 (.021-.031), SRMR = .04, BIC 54581.44. Constraining these loadings did not damage model fit, S-B $\Delta \chi^2(16, N = 1613) = 9.30$. Therefore, this model was retained.
In the third and final measurement model, the additional constraint of fixing at equality the factor loadings for each latent factor in the model across groups was imposed. This model fit the data well, $\chi^2(224, N = 1613) = 364.46$, CFI = .97, TLI = .96, RMSEA = .028 (.023 -.033), SRMR = .04, BIC 54578.77. No significant change in model fit was evidenced, S-B $\Delta \chi^2(20, N = 1613) = 28.90$, therefore the multigroup cross-lagged model could be estimated.

The first and most saturated multigroup cross-lagged model allowed all cross-lagged structural paths to be freely estimated for each group. Covariances between latent variables at each measurement occasion were also freely estimated. This initial model fit the data well, $\chi^2(232, N = 1613) = 400.64$, CFI = .96, TLI = .95, RMSEA = .030 (.025 -.035), SRMR = .05, BIC 54561.11. This fully saturated model is depicted by Figure 2.
Figure 2. Standardized structural equation modeling results of the saturated fully cross-lagged model demonstrating the temporal relationship between avoidance symptoms and posttraumatic growth.

Note. All bold cross-lagged model paths are significant for one or both groups in the multigroup models. Dashed lines indicate non-significant path coefficients for both groups. * $p < .05$. ** $p < .01$. Values to the left of the diagonal line represent standardized coefficients for Jews, and values to the right represent standardized path coefficients for Arabs. PTG = posttraumatic growth. PTSD – A = posttraumatic stress disorder avoidance symptoms.

As above, equality of cross-lagged relationships were evaluated. First, in Model 2, fixing the path from T1 PTG to T2 avoidance symptoms to equality yielded a non-significant result, S-B $\Delta \chi^2(1, N = 1613) = 0.19, p > .05$. This indicated that the two paths were equivalent for Jews and PCI. The non-significant model paths from the fully saturated model for Jews was ($\beta = 0.05$) and ($\beta = 0.10$) for PCI. T1 PTG did not predict T2 avoidance symptoms for Jews or PCI.

In model 3, the paths from T1 avoidance to T2 PTG were fixed to be equal. The path was not significantly different between the two groups, S-B $\Delta \chi^2(1, N = 1613) = 0.98$, **
\( p > .05 \). The model paths from the fully saturated model for Jews was (\( \beta = .13, p = .04 \)) and (\( \beta = .02, p = .79 \)) for PCI indicating a small effect size relationship between T1 avoidance symptoms predicting T2 PTG existed for Jews but not PCI.

The path from T2 PTG to T3 avoidance symptoms was fixed to equality in Model 4. No significant difference in model fit was noted after fixing this path, S-B \( \Delta \chi^2 (1, N = 1613) = 0.23, p > .05 \), indicating that the two groups did not differ with regard to this path. The paths from the fully saturated model for Jews was (\( \beta = -0.01, p > .05 \)) and (\( \beta = .06, p > .05 \)) for PCI; T2 PTG did not predict T3 avoidance symptoms for either group.

In the final model, the path from T2 avoidance symptoms to T3 PTG was fixed at equality. The two groups did not significantly differ with regard to the strength and direction of this path, S-B \( \Delta \chi^2 (1, N = 1613) = .08, p > .05 \). However, when the paths from the fully saturated model were examined, it appears that T2 avoidance symptoms predicted T3 PTG for Jews (\( \beta = 0.23, p = .002 \)) but not for PCI (\( \beta = .16, p > .05 \)).

Emotional numbing symptoms and PTG. The initial multiple group measurement model tested configural invariance over the three study waves. The factor loadings for numbing symptoms and PTG were estimated freely across Jews and PCI and the residuals for like-items were allowed to covary freely over time. Measurement Model 1 fit the data well, \( \chi^2 (558, N = 1613) = 839.17, \) CFI = .95, TLI = .94, RMSEA = .025 (.021 - .028), SRMR = .05, BIC 75694.18. Results for all emotional numbing and PTG models are displayed in Table 8.
Table 8. Multigroup Cross-lagged Analysis of Emotional Numbing Symptoms

<table>
<thead>
<tr>
<th>Model</th>
<th>S-B $\chi^2$</th>
<th>df</th>
<th>Scaling factor</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
<th>SRMR</th>
<th>BIC</th>
<th>S-B $\Delta \chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Measurement Models</strong></td>
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<tr>
<td>Model 1 (configural)</td>
<td>839.17</td>
<td>558</td>
<td>1.130</td>
<td>.95</td>
<td>.94</td>
<td>.025 (.021 -.028)</td>
<td>.05</td>
<td>75694.18</td>
<td>--</td>
</tr>
<tr>
<td>Model 2 (temporal invariance)</td>
<td>863.44</td>
<td>586</td>
<td>1.139</td>
<td>.95</td>
<td>.94</td>
<td>.025 (.021 -.028)</td>
<td>.05</td>
<td>75522.56</td>
<td>28, 18.41</td>
</tr>
<tr>
<td>Model 3 (metric invariance)</td>
<td>888.92</td>
<td>593</td>
<td>1.141</td>
<td>.95</td>
<td>.94</td>
<td>.025 (.021 -.028)</td>
<td>.05</td>
<td>75500.86</td>
<td>35, 37.80</td>
</tr>
<tr>
<td><strong>Structural models</strong></td>
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<tr>
<td>Model 1 (all cross-lagged parameters freed for Jews and PCI)</td>
<td>923.37</td>
<td>601</td>
<td>1.140</td>
<td>.94</td>
<td>.93</td>
<td>.026 (.022 -.029)</td>
<td>.06</td>
<td>75480.69</td>
<td>--</td>
</tr>
<tr>
<td>Model 2 (path from T1 PTG to T2 numbing fixed at equality)</td>
<td>923.54</td>
<td>602</td>
<td>1.140</td>
<td>.94</td>
<td>.93</td>
<td>.026 (.022 -.029)</td>
<td>.06</td>
<td>75473.52</td>
<td>1, .15</td>
</tr>
<tr>
<td>Model 3 (path from T1 numbing to T2 PTG fixed at equality)</td>
<td>928.57</td>
<td>602</td>
<td>1.140</td>
<td>.94</td>
<td>.93</td>
<td>.026 (.023 -.029)</td>
<td>.06</td>
<td>75478.74</td>
<td>1, 4.56*</td>
</tr>
<tr>
<td>Model 4 (path from T2 PTG to T3 numbing fixed at equality)</td>
<td>924.85</td>
<td>602</td>
<td>1.140</td>
<td>.94</td>
<td>.93</td>
<td>.026 (.023 -.029)</td>
<td>.06</td>
<td>75475.05</td>
<td>1, 1.30</td>
</tr>
<tr>
<td>Model 5 (path from T2 numbing to T3 PTG fixed at equality)</td>
<td>924.07</td>
<td>602</td>
<td>1.140</td>
<td>.94</td>
<td>.93</td>
<td>.026 (.022 -.029)</td>
<td>.06</td>
<td>75474.18</td>
<td>1, .61</td>
</tr>
</tbody>
</table>

*Note.* S-B $\chi^2$ = Sattora-Bentler $\chi^2$. CFI = comparative fit index. TLI = Tucker Lewis Index. RMSEA = root mean square of approximation. SRMR = standardized root mean square residual. BIC = Bayesian information criterion. S-B $\Delta \chi^2$ = Sattora-Bentler $\chi^2$ difference test. PCI = Palestinian Citizen of Israel.

In Measurement Model 2, the factor loadings for numbing symptoms and PTG were constrained to be equivalent across each measurement occasion, but freely estimated between groups, $\chi^2(220, N = 1613) = 863.44$, CFI = .95, TLI = .94, RMSEA = .025 (.021 - .028), SRMR = .05, BIC 75522.56. The difference between these models was non-significant S-B
Δχ^2(28, N = 1613) = 18.41, which indicated that constraining these loadings did not damage model fit. Therefore, this model was retained.

In the third and final measurement model, the additional constraint of fixing at equality the factor loadings for each latent factor in the model across groups. This model fit the data well, χ^2(593, N = 1613) = 888.92, CFI = .95, TLI = .94, RMSEA = .025 (.021 -.028), SRMR = .05, BIC 75500.86. No significant change in model fit was evidenced, S-B Δχ^2(35, N = 1613) = 37.80, therefore the multigroup cross-lagged model could be estimated.

The first and most saturated multigroup cross-lagged model allowed all cross-lagged structural paths to be freely estimated for each group. Covariances between latent variables at each measurement occasion were also freely estimated. This initial model fit the data well, χ^2(601, N = 1613) = 923.37, CFI = .94, TLI = .93, RMSEA = .026 (.022 -.029), SRMR = .06, BIC 75480.69. This fully saturated model is depicted by Figure 3.
Figure 3. Standardized structural equation modeling results of the saturated fully cross-lagged model demonstrating the temporal relationship between numbing symptoms and posttraumatic growth.

Baseline  | 6-month Follow-up  | 12-month Follow-up
---|---|---
![Diagram of structural equation model]

Note. All bold cross-lagged model paths are significant for one or both groups in the multigroup models. Dashed lines indicate non-significant path coefficients for both groups. * p < .05. ** p < .01. Values to the left of the diagonal line represent standardized coefficients for Jews, and values to the right represent standardized path coefficients for Arabs. PTG = posttraumatic growth. PTSD – N = posttraumatic stress disorder numbing symptoms.

As above, equality of cross-lagged relationships was evaluated. First, in Model 2, fixing the path from T1 PTG to T2 numbing symptoms to equality yielded a non-significant result, S-B $\Delta \chi^2(1, N = 1613) = 0.15$, $p > .05$. This indicated that the two paths were equivalent for Jews and PCI. The non-significant model paths from the fully saturated model for Jews was ($\beta = -0.10, p = .07$) and ($\beta = -0.04, p = .67$) for PCI. T1 PTG did not predict T2 numbing symptoms for Jews or PCI.

In model 3, the paths from T1 numbing to T2 PTG were fixed to be equal. The path was significantly different between the two groups, S-B $\Delta \chi^2(1, N = 1613) = 4.56$, $p$
< .05. The model paths from the fully saturated model for Jews was (β = .07, p = .25) and (β = .29, p = .001) for PCI indicating a small effect size relationship between T1 numbing symptoms predicting T2 PTG existed for PCI but not Jews.

The path from T2 PTG to T3 numbing symptoms was fixed to equality in Model 4. No significant difference in model fit was noted after fixing this path, S-B Δχ²(1, N = 1613) = 1.30, p > .05, indicating that the two groups did not differ with regard to this path. The paths from the fully saturated model for Jews was (β = -.04, p = .59) and (β = .15, p = .27) for PCI; T2 PTG did not predict T3 numbing symptoms for either group.

In the final model, the path from T2 numbing symptoms to T3 PTG was fixed at equality. The two groups did not significantly differ with regard to the strength and direction of this path, S-B Δχ²(1, N = 1613) = .61, p > .05. The model paths from the fully saturated model for Jews was (β = 0.04, p = .50) and (β = .17, p = .19), indicating that T2 numbing symptoms did not predict T3 PTG for either Jews or PCI.

Hyperarousal symptoms and PTG. The initial multiple group measurement model tested configural invariance over the three study waves. The factor loadings for numbing symptoms and PTG were estimated freely across Jews and PCI and the residuals for like-items were allowed to covary freely over time. Measurement Model 1 fit the data well, χ²(564, N = 1613) = 922.85, CFI = .95, TLI = .94, RMSEA = .028 (.025 - .031), SRMR = .05, BIC 79288.34.

In Measurement Model 2, the factor loadings for numbing symptoms and PTG were constrained to be equivalent across each measurement occasion, but freely estimated between groups, χ²(592, N = 1613) = 947.40, CFI = .95, TLI = .94, RMSEA =
.027 (.024 - .030), SRMR = .05, BIC 79114.27. Differences between these models was non-significant, S-B Δχ²(28, N = 1613) = 19.55, which indicated that constraining these loadings did not damage model fit. Therefore, this model was retained.

In the third and final measurement model, the additional constraint of fixing at equality the factor loadings for each latent factor in the model across groups. This model fit the data well, χ²(599, N = 1613) = 1014.77, CFI = .94, TLI = .93, RMSEA = .09 (.026 -.032), SRMR = .06, BIC 79140.53. There was a significant difference between these models, S-B Δχ²(35, N = 1613) = 73.61, p < .001, therefore, as above, the multigroup cross-lagged model could not be estimated. Without constraining these factor loadings between groups, it is not possible to be sure that the constructs are the same for both groups, and therefore multigroup comparisons are not meaningful (Cheung & Rensvold, 2002; Bollen, 1989; Drasgow & Kanfer, 1985; Horn & McArdle, 1992; Meredith, 1993; McArdle & Cattell, 1994; Steenkamp & Baumgartner, 1998). In order to evaluate the structural relationship between hyperarousal and PTG, cross-lagged models were specified separately for Jews and PCI.

**Hyperarousal and PTG: Jews.** The first and most saturated cross-lagged model allowed all cross-lagged structural paths to be freely estimated. Covariances between latent variables at each measurement occasion were also freely estimated. This initial model fit the data well, χ²(297, N = 1136) = 528.20, CFI = .93, TLI = .95, RMSEA = .026 (.022 - .029), SRMR = .05, BIC 55219.40. Results for the models appear in Table 9. This fully saturated model is depicted by Figure 4.
Table 9. Multigroup results for cross-lagged analysis of Hyperarousal Symptoms

<table>
<thead>
<tr>
<th>Model</th>
<th>S-B $\chi^2$</th>
<th>df</th>
<th>Scaling factor</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
<th>SRMR</th>
<th>BIC</th>
<th>S-B $\Delta\chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Measurement Models</strong></td>
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</tr>
<tr>
<td>Model 1 (configural)</td>
<td>922.85</td>
<td>564</td>
<td>1.129</td>
<td>.95</td>
<td>.94</td>
<td>.028 (.025 - .031)</td>
<td>.05</td>
<td>79288.34</td>
<td>--</td>
</tr>
<tr>
<td>Model 2 (temporal invariance)</td>
<td>947.40</td>
<td>592</td>
<td>1.135</td>
<td>.95</td>
<td>.94</td>
<td>.027 (.024 - .030)</td>
<td>.05</td>
<td>79114.27</td>
<td>28, 19.55</td>
</tr>
<tr>
<td>Model 3 (metric invariance)</td>
<td>1014.77</td>
<td>599</td>
<td>1.136</td>
<td>.94</td>
<td>.93</td>
<td>.026 (.026 - .032)</td>
<td>.06</td>
<td>79140.53</td>
<td>35, 73.61**</td>
</tr>
<tr>
<td><strong>Structural models: Jews</strong></td>
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<tr>
<td>Model 1 (all cross-lagged paths free)</td>
<td>528.22</td>
<td>300</td>
<td>1.279</td>
<td>.95</td>
<td>.95</td>
<td>.026 (.022 - .029)</td>
<td>.05</td>
<td>55219.40</td>
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<tr>
<td>Model 2 (T1 hyperarousal to T2 PTG fixed to 0)</td>
<td>529.49</td>
<td>301</td>
<td>1.279</td>
<td>.95</td>
<td>.95</td>
<td>.026 (.022 - .029)</td>
<td>.05</td>
<td>55213.98</td>
<td>1, .99</td>
</tr>
<tr>
<td>Model 3 (T1 PTG to T2 hyperarousal fixed to 0)</td>
<td>535.28</td>
<td>301</td>
<td>1.278</td>
<td>.95</td>
<td>.94</td>
<td>.026 (.023 - .030)</td>
<td>.05</td>
<td>55220.73</td>
<td>1, 7.22*</td>
</tr>
<tr>
<td>Model 4 (T2 hyperarousal to T3 PTG fixed to 0)</td>
<td>528.02</td>
<td>301</td>
<td>1.281</td>
<td>.95</td>
<td>.95</td>
<td>.026 (.022 - .029)</td>
<td>.05</td>
<td>55212.95</td>
<td>1, -.11</td>
</tr>
<tr>
<td>Model 5 (T2 PTG to T3 hyperarousal fixed to 0)</td>
<td>541.24</td>
<td>301</td>
<td>1.279</td>
<td>.95</td>
<td>.94</td>
<td>.027 (.023 - .030)</td>
<td>.06</td>
<td>55228.77</td>
<td>1, 10.18**</td>
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<td><strong>Structural models: PCI</strong></td>
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<tr>
<td>Model 1 (all cross-lagged paths free)</td>
<td>471.51</td>
<td>300</td>
<td>.986</td>
<td>.90</td>
<td>.89</td>
<td>.029 (.029 - .040)</td>
<td>.08</td>
<td>23736.90</td>
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<tr>
<td>Model 2 (T1 hyperarousal to T2 PTG fixed to 0)</td>
<td>473.64</td>
<td>301</td>
<td>.987</td>
<td>.90</td>
<td>.89</td>
<td>.035 (.029 - .041)</td>
<td>.08</td>
<td>23733.20</td>
<td>1, 1.65</td>
</tr>
<tr>
<td>Model 3 (T1 PTG to T2 hyperarousal fixed to 0)</td>
<td>474.85</td>
<td>301</td>
<td>.986</td>
<td>.90</td>
<td>.89</td>
<td>.035 (.029 - .041)</td>
<td>.08</td>
<td>23733.96</td>
<td>1, 3.39</td>
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<tr>
<td>Model 4 (T2 hyperarousal to T3 PTG fixed to 0)</td>
<td>471.49</td>
<td>301</td>
<td>.986</td>
<td>.90</td>
<td>.89</td>
<td>.034 (.028 - .040)</td>
<td>.08</td>
<td>23730.78</td>
<td>1, -.03</td>
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<tr>
<td>Model 5 (T2 PTG to T3 hyperarousal fixed to 0)</td>
<td>474.91</td>
<td>301</td>
<td>.987</td>
<td>.90</td>
<td>.89</td>
<td>.035 (.029 - .041)</td>
<td>.08</td>
<td>23734.48</td>
<td>1, 2.64</td>
</tr>
</tbody>
</table>

*Note.* S-B $\chi^2$ = Sattora-Bentler $\chi^2$. CFI = comparative fit index. TLI = Tucker Lewis Index. RMSEA = root mean square of approximation. SRMR = standardized root mean square residual. BIC = Bayesian information criterion. S-B $\Delta\chi^2$ = Sattora-Bentler $\chi^2$ difference test. PCI = Palestinian Citizen of Israel.
Figure 4. Standardized structural equation modeling results of the saturated fully cross-lagged model demonstrating the temporal relationship between hyperarousal symptoms and posttraumatic growth for the Jewish Sample.

Note. All bold cross-lagged model paths are significant for the models. Dashed lines indicate non-significant path coefficients. * $p < .05$, ** $p < .01$. PTG = posttraumatic growth. PTSD – H = posttraumatic stress disorder hyperarousal symptoms.

In order to test whether the individual cross-lagged paths meaningfully contributed to the model, structural pathways were deleted starting with the T2 variables regressed on T1 variables, and then for the T3 variables regressed on T2 variables.

First, in Model 2, fixing the path from T1 PTG to T2 hyperarousal symptoms to zero yielded a non-significant result, S-B $\Delta \chi^2(1, N = 1136) = 0.99, p > .05$. This indicated that this path did not contribute to the model, and that PTG did not predict later hyperarousal symptoms for Jews. The non-significant model path from the fully saturated model was ($\beta = -0.05, p = .26$).
In model 3, the paths from T1 hyperarousal to T2 PTG were fixed at zero. There was a significant difference between the saturated and constrained models, S-B $\Delta \chi^2(1, N = 1136) = 7.22, p < .001$. This indicated that this path contributed to the model. The significant model path from the fully saturated model was ($\beta = 0.13, p = .004$), which demonstrated a small effect size relationship between T1 hyperarousal predicting T2 PTG for Jews.

In model 4, the paths from T2 PTG to T3 hyperarousal was fixed at zero. There were no significant differences between the saturated and constrained models, S-B $\Delta \chi^2(1, N = 1136) = -0.11, p > .05$. This indicated that this path did not contribute to the model, and that T2 PTG did not predict T3 hyperarousal for Jews. The non-significant model path from the fully saturated model was ($\beta = 0.04, p = .57$).

In model 5, the path from T2 hyperarousal to T3 PTG was fixed at zero. Results revealed a significant difference between the saturated and constrained models, S-B $\Delta \chi^2(1, N = 1136) = 10.18, p < .001$. This indicated that this path contributed to the model. The significant model path from the fully saturated model was ($\beta = 0.14, p = .03$), which indicated that T2 hyperarousal predicted T3 PTG for Jews.

Hyperarousal and PTG: PCI. The first and most saturated cross-lagged model allowed all cross-lagged structural paths to be freely estimated. Covariances between latent variables at each measurement occasion were also freely estimated. This initial model fit the data well, $\chi^2(297, N = 477) = 471.51$, CFI = .90, TLI = .89, RMSEA = .029 (.029 - .040), SRMR = .08, BIC 23736.90. This fully saturated model is depicted by Figure 5.
Figure 5. Standardized structural equation modeling results of the saturated fully cross-lagged model demonstrating the temporal relationship between hyperarousal symptoms and posttraumatic growth for the Arab Sample.

Note. All bold cross-lagged model paths are significant for the models. Dashed lines indicate non-significant path coefficients. Asterisks indicate significance values for covariances and autoregressive paths. * p < .05. ** p < .01. PTG = posttraumatic growth. PTSD – H = posttraumatic stress disorder hyperarousal symptoms.

In order to test whether the individual cross-lagged paths meaningfully contributed to the model, structural pathways were deleted starting with the T2 variables regressed on T1 variables, and then for the T3 variables regressed on T2 variables. First, in Model 2, fixing the path from T1 PTG to T2 hyperarousal symptoms to zero yielded a non-significant result, S-B Δχ²(1, N = 477) = 1.65, p > .05. This indicated that this path did not contribute to the model, and that PTG did not predict later hyperarousal symptoms for PCI. The non-significant model path from the fully saturated model was (β = 0.12, p = .15).
In model 3, the paths from T1 numbing to T2 PTG were fixed at zero. Results failed to reveal a significant difference between the saturated and constrained models, S-B $\Delta \chi^2(1, N = 477) = 3.39, p > .05$. This indicated that this path did not contribute to the model, and that T1 hyperarousal did not predict T2 PTG for PCI. The non-significant model path from the fully saturated model was ($\beta = 0.15, p = .05$).

In model 4, the paths from T2 PTG to T3 hyperarousal was fixed at zero. Results revealed no significant differences between the saturated and constrained models, S-B $\Delta \chi^2(1, N = 477) = -0.03, p > .05$. This indicated that this path did not contribute to the model, and that T2 PTG did not predict T3 numbing for PCI. The non-significant model path from the fully saturated model was ($\beta = 0.02, p = .83$).

In model 5, the path from T2 numbing to T3 PTG was fixed at zero. Results revealed no significant differences between the saturated and constrained models, S-B $\Delta \chi^2(1, N = 477) = 2.64, p > .05$. This indicated that this path did not contribute to the model, and that T2 hyperarousal did not predict T3 PTG for PCI. The non-significant model path from the fully saturated model was ($\beta = 0.24, p = .07$).

**Discussion**

Utilizing data gathered from a large representative sample of Israeli Jews and PCI, this study sought to evaluate the following three questions: 1) whether the PTSD and PTG constructs were similarly represented when assessed between these groups, 2) whether there was a temporal relationship between PTSD symptoms and PTG, and 3) whether ethnic minority individuals were more likely to benefit from PTG than those in
the majority culture. The results indicated that, as has been found previously (King et al., 1998), PTSD was best represented by a four-factor correlated model of reexperiencing, avoidance, emotional numbing, and hyperarousal. Importantly, each of these factors, save for hyperarousal, were equivalently measured between Jews and PCI. PTSD and PTG were related over time. PTG was not found to lead to less PTSD symptom severity in any of the four PTSD clusters, and this was true for both Jews and PCI. In contrast, PTSD symptom severity was generally predictive of PTG such that PTSD assessed earlier in the study was related to later reported PTG for both groups. This finding calls into serious question the salutogenic utility of the PTG construct.

The Best Fitting Model of PTSD and PTG for Jews and PCI

The results demonstrated that PTSD evidenced excellent cross-cultural generalizability. This is of particular importance given that many studies are conducted in regions throughout the world that assume constructs developed in Western societies will be applicable across disparate cultural groups (Hui & Triandis, 1985; Vandenberg & Lance, 2000). Not only was the factor structure equivalent for the Jewish sample to studies conducted within the United States (King, et al., 1998), it was generally similar between the Jewish sample and the non-Western PCI sample. This suggests that PTSD is a real manifestation of psychological distress and not merely a Western culturally-specific construction.

The four-factor correlated model for PTSD was the best fitting among the models tested for each group. These findings are consistent with a growing literature suggesting that the current conceptualization of PTSD in the DSM-IV nomenclature does not
represent the actual structure of PTSD symptoms. Indeed in a summary of the CFA literature, Palmieri, Weathers, Difede and King (2007) identified this as the most frequently occurring PTSD factor structure. It is not surprising that this model would fit the data best. The major difference between this model and the one used to diagnose PTSD is the separation of active avoidance and emotional numbing. This separation follows theoretical arguments that avoidance represents an effortful process to reduce distress and that numbing is a consequence of a failure to do so (Foa, Zinbarg, & Rothbaum, 1992). Hence, the avoidance factor as it is currently included in the *DSM-IV*, is not measuring the actual structure of PTSD.

The four-factor model found in this study does not necessarily represent the actual PTSD syndrome as this and other studies are still constrained by the item pool used to assess PTSD. In this regard, the 17 items included in the CFA models have not changed significantly from the item pool that were used when PTSD was first included as a diagnostic entity in the *DSM-III*, 30 years ago (American Psychiatric Association, 1980; Wilson, 1994). This naturally limits our conceptualization of the syndrome. Other important diagnostic features for the disorder have emerged, or have gained greater empirical attention since that time. For example, current and prevailing psychotherapy models for PTSD address issues of safety, trust, power and control, self-esteem, and difficulties with intimacy (Resick, Nishith, Weaver, Astin, & Feuer, 2002). Guilt (Kubany, 1994), anger-out, anger control (Olatunji, Ciesielski, Tolin, 2010) anger-in (Ulrich & Wieland, 2006), dissociation and identity disturbances (Feeny, Zoellner, & Foa, 2000; Herman, 1992), sleep disturbances (Germain, Buysse, Shear, Fayyad, &
Austin, 2004), and behavioral avoidance are among several additional factors that are not represented, or represented inadequately in the current item pool. Clearly, additional work needs to be done with an expanded item pool to better understand the full dimensionality of this construct.

Three of the four symptom clusters for PTSD were measured equally between the Jewish and PCI samples. The symptoms of reexperiencing, active avoidance, and emotional numbing remained unchanged over time, and that the strength of the factor loadings was invariant for both groups. A different picture emerged for the hyperarousal symptom cluster. Although this construct demonstrated temporal stability for Jews and Arabs, the strength of the associations among the factor loadings was heterogeneous across these groups. For the purpose of trying to isolate the item or items that were to blame for this apparent discrepancy, additional post-hoc S-B Δχ^2 tests were conducted. The PTG factor loadings were constrained at equivalence between groups, which served as the base model, and each of the standardized hyperarousal factor loadings were constrained at equality across groups, each in turn.

The only item that was heterogeneous between groups was item D4 (hypervigilance), S-B Δχ^2(1, N = 1613) = 18.52, p < .001. The individual item loadings from the saturated configural invariance model (Measurement Model 1) for Jews across each measurement wave were .61, .64, and .70. For PCI they were .47, 28, and .40. Clearly, the item demonstrated lower factor loadings for PCI across all study waves. This suggested that the item may have measured a different construct for each of the cultural groups. It may also have been possible that the item itself assessed a similar trait, the
strength of which was differentially expressed in either group. It appears that the hyperarousal construct may more closely adhere to the commonly found factor structure in Western samples for Jews than for PCI.

This study provides additional evidence for the four-factor PTSD model and initial evidence for this factor structure following terrorism exposure. The PTSD factor structure that provided the best fit to the data in this study has been supported in other diverse samples such as cancer survivors (DuHamel et al. 2004) and English & Spanish speaking survivors of community violence (Marshall, 2004). All but one of these constructs were equivalently measured between Jews and PCI, which further points to the generalizability of the underlying four-factor structure of PTSD. Furthermore, although not fully invariant across groups, the cross-cultural application of this model was supported by well-fitting configural invariance models. That is, although the models were not entirely equivalent in terms of the strength of the parameter estimates across groups, the four-factor model was the best fitting among those that were tested.

The PTG construct was found to be equivalently measured between the Jewish and PCI samples. For each group, the four-items loaded onto one latent factor, and this model fit the data reasonably well for both groups. This finding is especially important given that growth is a potentially ephemeral construct. The measurement of PTG has not received much attention in the terrorism context. This investigation, although limited by the short-item scale, provides evidence that the PTG construct was measured adequately within a sample that has experienced ongoing exposure or threat of exposure to terrorism. It also provides evidence that the construct evidenced generalizability to a non-Western
sample. Although this study did not use Posttraumatic Growth Inventory (Tedeschi and Calhoun, 1996), it nevertheless contributes to an emerging literature demonstrating the cross-cultural application of PTG (Hooper, Marotta, Depuy, 2009; Taku Calhoun, Tedeschi, Gil-Rivas, Kilmer, & Cann, 2007; Jaarsma, Pool, Sanderman, Ranchor, 2006).

*Evaluating the Relationship between PTSD and PTG*

Contrary to the promise of PTG posited by Frankl (1963), growth in this study was not related to less clinical psychopathology. The benefits of PTG for the participants in this study, if any were accrued, was a belief of increased personal meaning, greater strength, and closeness with friends and family. However, these perceived benefits did not translate to reductions in psychological distress. The psychiatric utility of PTG, and its potential salutary benefit for the participants in this study is unknown. The results of the cross-lagged models for PTSD and PTG demonstrated a clear and relatively consistent finding: When a significant relationship was present, PTSD symptom severity was associated with greater PTG. In contrast, across this one year study period, at no point was self-reported PTG related to less PTSD symptom severity. These findings strongly suggest that PTG does not represent a salutogenic process.

A positive and significant cross-sectional relationship was generally demonstrated between the PTSD symptom clusters and PTG. These findings are consistent with previous studies measuring the association between PTSD and PTG (Butler et al., 2005; Hobfoll et al., 2006; Laufer & Solomon, 2006) and adds to the growing number of studies suggesting that PTG and psychopathology are positively related to one another within
terrorism contexts (Hall et al., 2009, 2010; Hobfoll et al., 2008; 2009). The effect sizes for the cross-sectional associations when significant were medium (Cohen, 1988), and stronger than the cross lagged effects.

Although PTG demonstrated temporal metric invariance between the two samples, PTG was not shown to be stable over the three measurement occasions for PCI. The autoregressive pathway between PTG at T2 and T3 was not significant in all but the emotional numbing model. Given that the cross-lagged pathways were also not significant, it is unlikely that these effects attenuated the autoregressive path by accounting for more variance in the outcome variable. The report of PTG at T3 was not significantly related to the PTG reported earlier, which indicates a lack of stability (i.e., test retest reliability) for this construct. It may be possible that as this group gained temporal distance from the Intifada period, the PTG that they reported began to wane.

PCI did not experience the same degree of terrorism exposure as the Jews in this study so perhaps they were not exposed to as great a degree of stress necessary to continue experiencing PTG (Carboon et al., 2005; Erbes et al, 2005; Morris, Shakespeare-Finch, Rieck, & Newbery, 2005; Wild & Pavio, 2003; Tedeschi & Calhoun, 2006). A recent study on war-related trauma exposure and stressful life events demonstrated that both were associated with greater PTG. As the number of stressors increased, so did self-reported PTG, even in a sub-sample of people who all reported being exposed to trauma (Hall et al., 2010). Hence, greater exposure to trauma may have predicted greater and sustained PTG within the PCI sample.
It is important to note that throughout this study period, the threat and actual experience of terrorism was ongoing. Although the mean levels of terrorism were not particularly high following the first measurement wave, the threat of terrorist attack continued (Hobfoll et al., 2009). The participants in this study reported PTG in response to terrorism exposure occurring during the second Intifada, and because this epoch was marked with significant and widespread terror attacks, PTG in this context may be different than growth reported following single-incident trauma exposure.

Tedeschi and Calhoun suggested (2004) that true growth may need time to occur, and the assessments of PTG even one year following the end of the Intifada may represent continued active coping (e.g., making meaning of events), spurred on by psychological distress. It is possible that the PTG reported in this study may not be linked to stable life changes (Frazier et al., 2001; McMillen et al., 1997). For people exposed to the pressure of ongoing life threat, there may be little chance of entering a resolution phase where they are free from psychopathology, at which point their PTSD symptoms would be unrelated to PTG (Tedeschi & Calhoun, 2006).

The findings in this study suggest that the beneficial aspects of PTG found in other studies were not manifested within this terrorism milieu (Helgeson et al., 2006). These results support the Janus Face model of PTG, which suggests that PTG has two faces, one protective, and the other self-deceptive (Zoellner & Maercker, 2006). PTG may represent an attempt to make meaning from an event and suffering that makes little sense. As was theorized by Hobfoll and colleagues (2007), it may not be possible for psychological benefits of PTG to be demonstrated when the possibility of taking action is
not possible. Within this context, the participants were not able to take any action to ameliorate their suffering. When PTG is measured in people who are able to take action in the face of a threat, PTG may serve a protective function (Hall & Hobfoll, 2006; Hall et al., 2008; Hobfoll et al., 2007).

It is not certain whether the PTG reported in this and other studies actually represents true changes in the individual. A competing explanation is that PTG may serve a self-protective function by allowing people to justify their suffering by thinking they have grown (McFarland & Alvaro, 2000). Self-enhancement processes serve people by enabling them to maintain and create positive views of their current selves by derogating themselves in the past (Wilson & Ross, 2001). Alternatively, those who have reported greater stress and trauma have also reported greater PTG (Carboon et al., 2005; Hall et al., 2010; Erbes et al, 2005; Morris et al., 2005; Wild & Pavio, 2003). It may therefore be possible that the PTG reported in this study, secondary to PTSD, may be a reflection of actual growth. However, at no point in this study was PTG related to the amelioration of suffering according to how it was operationalized in this investigation.

**Ethnicity as a Moderator of the Relationship between PTG and PTSD**

Being a member of an ethnic minority group did not differentially predict the association between PTSD and PTG. The PCI sample did not evidence better functioning as a result of self-reported PTG. This finding is consistent with previous research in Israel (Hall et al., 2009; Hall et al., 2010; Hobfoll et al., 2008; Hobfoll et al., 2009), but is contradictory to several studies conducted within the United States (Helgeson et al.,
Given that the majority of studies reporting a salutogenic relationship between ethnicity and PTG have taken place in the United States, minority status has largely been defined as being either Latino or African American (Helgeson et al., 2006). Although ethnic minorities in the United States and PCI share a similar history of being underprivileged, marginalized, and have experienced generational economic inequality (Canetti-Nisim et al., 2008; Smooha, 2004), these apparent commonalities did not lead to similar results to the published literature.

Generally, the association between PTSD and PTG was not as strong for the PCI sample compared to the Jewish sample. When significant associations were noted, they only existed between T1 and T2, and not from T2 to T3. The only association that was significant in the PCI sample, and not in the Jewish sample, was between emotional numbing and PTG. Emotional numbing at T1 was related to greater PTG at T2. It may be possible that this association further supports the assertion that PTG fulfills a self-enhancing purpose. It is difficult to imagine that a person who reports being alienated from friends and family could later report experiencing greater connectedness. Previous longitudinal research has demonstrated that psychological distress leads to alienation of social support networks (Kaniasty & Norris, 2008).

**Strengths and Limitations of the Current Study**

This study has several notable strengths. The longitudinal design provided a means to test the potential temporal relationships between PTSD and PTG. This overcomes a serious limitation to the majority of PTG studies that have been cross-sectional. Following an extensive literature review, no other study was identified that has
examined the relationship between PTSD symptomatology and PTG in a longitudinal framework within people who faced community-wide exposure to terrorism and war.

A second strength of this study was to evaluate the factor structure of PTSD and PTG and to demonstrate the equivalence of these constructs between different cultural groups. Psychological constructs cannot be assumed to universally apply to all cultures and in all regions of the world (Vandenberg & Lance, 2000). The examination of the factor structure of PTSD and PTG enabled more meaningful modeling of the study variables, and provided the ability to draw conclusions about cross-cultural similarities and differences. The multi-group cross-lagged panel models were designed to identify possible cross-cultural similarities and differences. Additionally, the large sample size allowed for ample statistical power to examine the multigroup models reliably. Further, this study extends the literature by examining these processes in a sample that has experienced ongoing and repeated psychological trauma. Since the literature is mixed regarding the benefit of PTG, this is an important contribution as PTSD and PTG may be differently related in various samples with different types of trauma exposure.

Finally, although not generalizable to all situations, many regions of the world face ongoing war and terrorism, and so these results may be important for other areas of the world that are facing ongoing war and threat of war. Taken together, this study contributes significantly to what is known about the factor structure of PTSD, how PTSD and PTG are related, and the role of ethnic minority status in this relationship.

The study also had several limitations. The study design, although longitudinal, cannot discern strict casual relatedness among the study variables since the effects of
unmeasured influences cannot be ruled out (Kazdin, 2003). This study captures one discrete period of time and measures reactions naturalistically during the course of one year. The temporal relatedness between variables may indicate potential causal mechanisms, but caution should be used when interpreting the pathways in the models.

PTG may take time to begin to translate to reductions in psychopathology (Hall et al., 2009; Hall et al., 2010; Hobfoll et al., 2007; Tedeschi & Calhoun, 2004; Tedeschi & Calhoun, 2006) so people in this study may not have had enough time free from all exposure for the possible salutogenic aspects of PTG to occur (Frazier et al., 2001). It must be acknowledged that the PTSD and PTG reported in this study were in response to ongoing exposure and threat of future exposure to terrorism. In the short-term, the participants in this study may be using PTG as a coping tool (Hall et al., 2010; McFarland & Alvaro, 2000). Although PTG did not relate to a reduction of PTSD, it is also important to acknowledge that the growth reported may be linked to other indicators of positive psychological functioning that were left unmeasured in this study such as well being or depression.

The PTG construct was not measured fully so inferences regarding the relationship between PTSD and PTG can be made for only three of the five domains of PTG commonly studied. The use of this short instrument was necessary in this telephone survey to decrease participant burden and include all relevant constructs important to the overall project. In a CFA study of the 21 item instrument, Taku, Cann, Calhoun, and Tedeschi (2008) found that rather than one unitary construct, the PTG measurement model that best fit the data for the PTGI was a five-factor correlated structure. Although
the items used in this study to measure PTG were found to correlate highly with the PTGI (Tedeschi & Calhoun, 1996; Hall & Hobfoll, 2008) the full dimensionality of the construct was not captured by these four items. However, these items did hold up reasonably well to CFA as a unitary construct, and the face validity of the items would suggest that the dimensions of personal meaning, strength, and closeness with friends and family were represented in this study. This study is unable to speak to the potential relationship between PTSD and growth in the areas of spirituality and new life possibilities.

As a more general limitation, this study relies solely on a unitary assessment method (i.e., self-report via telephone interview) and one assessment instrument per construct involved in the study. Given the nature of the biases potentially involved in reporting PTG, multiple assessments of the construct including objective behavior, subjective self-report and perhaps peer- or partner-report would improve the confidence in the findings of this study. Also, PTSD symptom severity and PTG was assessed using telephone interviews. Although these assessments were not used to make diagnoses per se, and recent study has shown that moderate to significant diagnostic overlap existed between telephone and in-person structured interviews (Hobfoll et al., under review), clinician versus lay assessment would be preferable.

It is not possible to examine whether there were different response rate for the Jews and PCI samples. As a result, it is possible that differential non-response bias in the two groups may have contributed to some of the results observed in this study. As an example of a potential mechanism for non-response, individuals who have experienced
greater exposure to terrorism and related economic deprivation may not have been able to respond to telephone interviews.

Another limitation was that participants’ subjective report of the severity of their traumatic event exposure was not measured. It may be entirely possible that variation exists in the degree to which terrorism exposure is felt by our participants as traumatic. Therefore the extent to which PTG was expressed could have been accounted for by this non-measured process (Carboon et al., 2005; Erbes et al, 2005; Morris et al., 2005; Wild & Pavio, 2003; Tedeschi & Calhoun, 2004).

**Directions for Future Research**

Based on the study’s limitations, future PTG research could add to the literature by including multiple assessments of PTG through self-report, observable behaviour, informant report, and in-person interviews. Clinician assessment of PTG could also aid in making direct linkages between pre- and post-trauma selves (e.g., assess changes) and their relationship to a potentially traumatic event. More complete assessment of PTG should be evaluated utilizing longitudinal methods so the findings in this investigation can be replicated. Moreover, truly prospective designs where a person’s pre-morbid functioning could be assessed before trauma exposure would also add significantly to understanding the role that PTG plays in post-event psychological functioning. Future research can also examine how PTG relates to psychological functioning following a variety of traumatic events, and in a variety of contexts. Because it is not entirely clear
whether people who report PTG have actually grown following trauma, research should also examine other factors that may account for describing oneself as having experienced PTG (e.g., self-enhancement).

**Conclusions**

The model for the best fitting factor structure for PTSD was different than what is currently found in the *DSM-IV*. It appears that the correlated four-factor model originally posited by King and colleagues (King et al., 1998) has garnered considerable empirical support. It has demonstrated excellent model fit in this, and the only other CFA study conducted with terrorism exposed populations (Palmieri et al., 2007). The cross-cultural application of the model further supports the generalizability of this factor structure. Clearly revisions to PTSD included in the *DSM-V* should incorporate the mounting empirical evidence of a lack of support for its current conceptualization. Although the assumption of cross-cultural applicability should continue to be tested (Hui & Triandis, 1985; Vandenberg & Lance, 2000), this study provides evidence of the cross-cultural applicability of the PTSD and PTG constructs in the Middle East.

PTG is an enticing construct that holds within it the hope for an alternative outcome to PTSD and psychological suffering following trauma. However, PTG has not consistently been shown to correlate with less suffering. In this study, as was seen in previous studies of terrorism (Hobfoll et al., 2006; Hobfoll et al., 2009), PTG failed to live up to its promise. Research has documented that the majority of people who experience a major traumatic event, such as terrorism, actually remain relatively unperturbed by the event (Bonanno, 2005). Resilience following traumatic life events is
therefore, at least following single incident traumas, a relatively common occurrence. However, PTG reaches beyond resilience and suggests that humans have the capacity for growth and may experience an evolution into a person who sees new possibilities and meaning in life, is stronger, and a more interpersonally and spiritually connected. What is PTG then if not a construct related to the amelioration of human suffering? It might serve the individual to believe that positive changes have occurred, to cope with an event through a meaning making process (Davis et al., 2000; McFarland & Alvaro, 2000). This may signal an over-accommodation of the traumatic event, such that the event changes the cognitive schema of the individual to reflect a belief in positive changes (Resick et al., 2006). In this case, the person believes themselves to be superior to how they were before the event took place. If this is true, PTG may be best characterized as a coping tool that in practice, does not lead to less distress.
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