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Negative Emotionality and Disconstraint Influence PTSD Symptom Course via Exposure to New Major Adverse Life Events

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Abstract

Identifying the factors that influence stability and change in chronic posttraumatic stress disorder (PTSD) is important for improving clinical outcomes. Using a cross-lagged design, we analyzed the reciprocal effects of personality and PTSD symptoms over time and their effects on stress exposure in a sample of 222 trauma-exposed veterans (ages 23 – 68; 90.5% male). Personality functioning and PTSD were measured approximately 4 years apart, and self-reported exposure to major adverse life events during the interim was also assessed. Negative emotionality positively predicted future PTSD symptoms, and this effect was partially mediated by exposure to new events. Constraint (negatively) indirectly affected PTSD via its association with exposure to new events. There were no significant effects of positive emotionality nor did PTSD symptom severity exert influences on personality over time. Results indicate that high negative affect and disconstraint influence the course of PTSD symptoms by increasing exposure to stressful life events.

Keywords

stress exposure; personality; posttraumatic stress; longitudinal; veterans

Although most individuals who develop posttraumatic stress disorder (PTSD) after a traumatic life event recover, a substantial minority develop chronic symptoms that persist for years and fluctuate over time (Chapman et al., 2012; Perkonigg et al., 2005; Solomon & Mikulincer, 2006). Chronic PTSD is a dynamic and environmentally-sensitive condition characterized by periods of symptom exacerbation and relative remission. Identifying predictors of stability and change in PTSD is critical for understanding why symptoms persist and reemerge over time, detecting individuals at risk for chronic trajectories, and improving outcomes for intractable cases. To advance understanding of the longitudinal

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course of PTSD, this study examined the reciprocal and stress-related effects of personality traits and PTSD symptoms over time in a sample of trauma-exposed veterans.

Reciprocal Influences of Personality and PTSD

Psychopathology researchers have had a long-standing interest in the interplay of personality and psychopathology, including in relation to PTSD. PTSD shows large concurrent relations with neuroticism and negative emotionality (*Pearson's r* = 0.49; Kotov et al., 2010), which are closely related constructs that refer to the tendency to experience high levels of distress and a range of negative affect. In contrast, personality traits that measure the tendency to have a positive, outgoing disposition (e.g., extraversion/ positive emotionality) or behave in a controlled, cautious manner (e.g., conscientiousness/low disconstraint) show relatively weaker negative associations with PTSD (*Pearson's rs* = −0.25 and −0.27, respectively; Kotov et al., 2010). Understanding the relations of personality traits with PTSD is important, because personality may predict the course and severity of symptoms by affecting remission and relapse (Milan, Zona, Acker, & Turcios-Cotto, 2013; Stein, Jang, Taylor, Vernon, & Livesley, 2002). Although a number of studies have examined personality as a risk factor for the development of PTSD (e.g., Bramsen, Dirkzwager, & Van der Ploeg, 2000), the influence of personality on the course of symptoms remains poorly understood. Thus, the first major aim of this study was to examine whether personality traits relate to the course of PTSD symptoms.

Although personality traits are generally assumed to be relatively stable, there is evidence that premorbid levels of personality functioning can be fundamentally altered by psychopathology (i.e., the scar hypothesis; reviewed by Ormel et al., 2013). In a population-based study of twin pairs, the onset of major depression in individuals without a history of depression predicted an increase in neuroticism from premorbid to postmorbid levels (Kendler, Neale, Kessler, Heath, & Eaves, 1993). In a similar fashion, the experience of PTSD symptoms may alter personality functioning by fundamentally changing how an individual tends to think, feel, and act. For example, PTSD could exacerbate an individual's tendency to view the world as dangerous, and this shift in perception could persist after symptoms abate. A previously extraverted and optimistic individual might become more socially withdrawn and feel less hopeful about future, thereby resulting in an overall decrease in trait positive affect. Similarly, viewing the world as dangerous may have long-term effects on trait negative affect by causing an individual to be more suspicious of and hostile towards others, or influence trait constraint by reducing an individual's tendency to be spontaneous and adventuresome. To our knowledge, longitudinal research has yet to examine the influence of PTSD on personality traits over time, and, ideally, longitudinal studies that examine the reciprocal influences of PTSD symptoms and personality traits are needed. Therefore, the second major aim of this study was to examine the extent to which PTSD influences personality traits over time.

PTSD and Personality as Risk Factors for Stress Exposure

Individuals with PTSD are significantly more likely to report experiences of re-traumatization than are individuals who were initially traumatized but did not develop the

disorder (e.g., Breslau, Davis, & Andreski, 1995). PTSD may predict exposure to subsequent trauma and stress through a number of mechanisms. For one, individuals with PTSD may only appear to be at elevated risk of stressful life events, because they tend to be more sensitive and reactive to stressors than individuals without PTSD and, thus, report more events. Another possibility is that the psychological and environmental factors that contributed to the initial trauma (e.g., personality traits, disadvantaged neighborhoods) may continue to influence PTSD symptoms indirectly over time. For example, elevated PTSD symptoms may appear to cause stressful life events, because they indirectly measure a high chronic context of stress that persists over time. Additionally, symptoms of PTSD may actually contribute to adverse life events. For example, chronic hyperarousal and irritability may increase the tendency for individuals to engage in physical altercations with others, whereas intrusive memories and avoidance symptoms may interfere with job performance or create relationship conflict. Moreover, hyperarousal symptoms may desensitize an individual to internal threat cues (Messman-Moore & Long, 2003) and avoidance/numbing symptoms may cause an individual to ignore internal threat cues (Krause, Kaltman, Goodman, & Dutton, 2006), both of which may decrease the ability of an individual to detect threat in the environment and avoid (re-)victimization.

Two prospective studies to date have tested the hypothesis that PTSD symptoms predict subsequent stress exposure. A cross-lagged study of urban adolescents reported that PTSD symptoms predicted greater exposure to violent events during a two-year follow-up, and this event exposure mediated the relationship of initial PTSD symptoms with later severity (Milan et al., 2013). Similarly, a large 3-wave longitudinal study of urban adults found that PTSD symptoms predicted greater future non-assaultive trauma (e.g., serious illness), when taking into account baseline levels of PTSD and trauma exposure (Lowe, Walsh, Uddin, Galea, & Koenen, 2014). These findings suggest that exposure to stress was not random but instead was partially accounted for by PTSD. Another recent longitudinal study examined the effects of anxiety disorders, in general, on exposure to life stress in a sample of 627 adolescents (Uliaszek et al., 2012). Anxiety disorders predicted higher episodic (but not chronic) life stress one year post-assessment after controlling for baseline levels of life stress, and neuroticism partially accounted for this relationship. These preliminary findings suggest that anxiety symptoms, including symptoms of PTSD, can be risk factors for future stress exposure, including potentially traumatic events that are assaultive (e.g., sexual violence, physical attack) and non-assaultive (motor vehicle accidents, life threatening illnesses) in nature.

Personality functioning is another potential risk factor for stress exposure that could, in turn, affect the course of PTSD symptoms. Specifically, individuals high in neuroticism may experience interpersonal conflict as a result of their chronic disposition to negative affect, whereas individuals low in constraint may engage in dangerous behaviors that increase exposure to potentially traumatic accidents or assaults. Alternatively, the tendency to experience low positive affect could result in loneliness or poor work performance resulting from a lack of sociability and agency. While this hypothesis is supported by evidence that trait neuroticism predicts higher rates of negative life events prospectively (Gunthert, Cohen, & Armeli, 1999; Kercher, Rapee, & Schniering, 2009; Magnus, Diener, Fujita, & Pavot, 1993), the effects of constraint and positive affect on stress exposure have not been

investigated. Thus, the third major goal of the study was to examine whether PTSD symptoms and personality traits independently and/or jointly increase the likelihood of exposure to adverse life events.

Stress Exposure Effects on the Stability of PTSD and Personality

Experiencing new stressful life events may influence the stability of PTSD symptoms and personality traits over time. A limited number of studies have prospectively examined changes in PTSD as a function of new stress exposure. In a small sample of veterans with PTSD, biweekly assessment of PTSD symptoms in the 6 weeks before and after the September 11, 2001 attacks revealed an increase in symptom severity (Niles, Wolf & Kutter, 2003). Another more recent study of veterans with PTSD reported that PTSD symptoms increased shortly after the Boston Marathon bombing among individuals who reported high levels of emotional distress about the event (Miller, Wolf, Hein, Prince, & Reardon, 2013).[1] Consistent with these findings, a longitudinal epidemiological study of young adults reported that new trauma exposure was the strongest predictor that differentiated individuals who displayed chronic symptoms from those who remitted at 34–50 month follow-up (Perkonigg et al. 2005). One question not addressed by these studies is whether exposure to new major adverse events actually mediates stability in PTSD severity over time. Further, research has yet to examine whether stress exposure influences the temporal stability of personality traits or reciprocal relations of PTSD and personality over time. Thus, the fourth and final goals of this study were (a) to determine whether exposure to traumatic and stressful life events prospectively predicts increases in PTSD symptoms and personality traits, and (b) to determine if stress/trauma exposure mediates the temporal stability of PTSD symptoms, personality traits, and/or PTSD-personality relations.

Study Aims

We recruited a sample of trauma-exposed veterans with elevated rates of PTSD symptoms. We assessed personality functioning and symptom severity at two time points approximately 4 years apart and measured exposure to new major adverse life events during the intervening period. Because retrospective recall over such a long time period can be associated with problems remembering and/or memory biases (see Monroe, 2008), we focused specifically on a small set of very traumatic and/or major adverse events (e.g., motor vehicle accident, assault, disabling illness), and limited our analysis to event frequencies and not on subjective appraisals of the threatfulness of these events.

Per our first two aims, we hypothesized that PTSD and broad domains of personality (high negative affect, low positive affect, and low conscientiousness/disconstraint) would exert reciprocal influences on each other over time. Our final three aims focused on stress exposure during the follow-up period and we hypothesized that: (3) PTSD and personality traits at Time 1 would predict new events during the follow-up, (4) greater event exposure during the follow-up would predict greater Time 2 PTSD severity and more extreme manifestations of personality traits, and (5) new event exposure would mediate the relation

[1]Miller et al. (2013) was based on a subset of participants from this study (n = 71), which did not examine the effects of personality functioning on major adverse life event exposure or PTSD, and it used different measures.

of (a) Time 1 PTSD with Time 2 PTSD, (b) Time 1 personality with Time 2 personality, and (c) the reciprocal relations between PTSD severity and personality traits over time.

We tested these hypotheses in two ways. In our primary analyses, we used a broad measure of major stress exposure, specifically the total number of new major event types that occurred during the follow-up period. In a secondary exploratory analysis, we ran separate models for assaultive and non-assaultive events to examine whether PTSD and personality showed different relations with these particular event types. Research suggests that assaultive events are moderately heritable, whereas non-assaultive traumatic events are not (reviewed in Afifi, Asmundson, Taylor, & Jang, 2010). Thus, we hypothesized that PTSD symptoms, trait negative affect, and disconstraint would positively predict assaultive events, given that individuals with greater symptom severity and higher levels of these traits show tendencies toward aggression, hostility, and impulsivity that may increase exposure to assaultive environments.

Methods

Sample

Participants consisted of 242 veterans who were recruited from VA Boston Healthcare System and screened by telephone using the PTSD Checklist-Civilian Version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993). Veterans were invited to participate if their responses on the phone screen suggested that they met criteria for probable PTSD according to the DSM-IV scoring rule, defined as at least one B symptom, three C symptoms, and two D symptoms endorsed at a severity of 3 or higher on the PCL-C 5-point Likert-like scale (Keen, Kutter, Niles, & Krinsley, 2008). A more thorough structured clinical interview administered during the initial in-person assessment was used as the primary assessment of PTSD (as described below) and indicated that 55% of participants met criteria for current PTSD. Twenty participants were excluded from analyses because they had problems completing the protocol, were not exposed to a Criterion A event, or withdrew before completing the measures.

The remaining sample consisted of 222 veterans ages 23 to 68 (M = 50.8, SD = 10.7). Participants were primarily male (n = 201, 90.5%) and most self-identified as White (n = 175, 78.8%), followed by Black or African-American (n = 43, 19.4%), and/or American Indian or Alaskan Native (n = 15, 6.8%). Seven participants reported unknown racial origin (n = 7, 3.2%) and eight (3.6%) endorsed Hispanic or Latino ethnicity. The majority of participants were either unemployed or receiving disability payments (n = 142, 63.9%); the remainder were employed full- or part-time (n =53, 23.9%), retired (n = 20, 9.0%), students (n = 3, 1.4%), or did not provide employment information (n = 4, 1.8%).

Participants completed an initial assessment (T1) and a follow-up assessment approximately 4 years later (T2). The study was not originally designed as a longitudinal investigation, and 148 of the initial 222 participants returned for T2.[2] The average interval between T1 and T2 was 3.9 years (SD = 0.8) and ranged from 2.4 to 6.1 years.[3] At T1, all participants had

[2]Of the people who did not participate in T2, 62% refused, 19% were deceased, and 19% were unable to be reached or scheduled.

PTSD data and 7 (3%) participants were missing personality data due to invalid response profiles (see below for details).[4] At T2, 73 (33%) participants were missing PTSD data, 77 (35%) were missing personality data (4 due to invalid response profiles), and 71 (32%) were missing major adverse event data. Following similar work (Allison, 2003; Brown & Rosellini, 2011), we used analytic techniques (direct maximum likelihood estimation) that modeled missing data for the cross-lagged and path analyses, which allowed us to include all available data (e.g., any participant with any data at T1), yielding a final sample size of 222 participants for these analyses. Individuals with follow-up data vs. those without did not differ on T1 PTSD symptom severity ($t_{(220)} = 1.0$, $M_{Difference} = 3.7$, +/−95% $CI = -3.4/10.8$, $p = .31$), current PTSD diagnosis ($x^2_{(1)} = 0.6$, $p = .67$), or scores on the personality indicators (positive affect: $t_{(212)} = 0.2$, $M_{Difference} = 0.5$, +/−95% $CI = -4.3/5.3$, $p = .83$, negative affect: $t_{(212)} = 1.4$, $M_{Difference} = 3.8$, +/−95% $CI = -1.4/8.9$, $p = .15$, constraint: $t_{(212)} = -1.7$, $M_{Difference} = -3.3$, +/−95% $CI = -7.1/0.5$, $p = .09$). They also did not differ on gender ($x^2_{(1)} = 2.1$, $p = .15$), income ($t_{(208)} = -1.3$, $M_{Difference} = -0.3$, +/−95% $CI = -0.6/0.1$, $p = .19$), or on any of the race/ethnicity categories (smallest $p = .19$).[5]

Measures

PTSD Symptoms—At the time of the phone screening, participants completed the self-report PTSD Checklist-Civilian Version (Weathers et al., 1993), which was used to assess for probable PTSD. The PCL-C consists of 17 items derived from the DSM-IV PTSD criteria and participants were asked to rate the severity of their symptoms on a 5-point scale.

Current PTSD symptom severity was assessed at each time point using the Clinician Administered PTSD Scale (CAPS; Blake et al., 1990), a 30-item diagnostic interview used to assess the frequency and intensity of the 17 DSM-IV PTSD criteria each on a 5-point scale. Past-month dimensional severity scores were used in analyses and calculated at each time point by summing the frequency and intensity ratings for each of the 17 symptoms (Weathers et al., 1999). A clinician determined the index Criterion A event to be combat trauma for 101 (45.5%) participants, actual or threatened physical assault for 34 (15.3%) participants, sexual trauma prior to age 18 for 19 (8.6%) participants, sudden death of a friend of loved one for 17 (7.7%) participants, motor vehicle or other accident for 15 (6.8%) participants, and sexual trauma as an adult for 10 (5.0%) participants. Each of the other index traumas (e.g., life threatening illness, witnessing assault, being stalked) was endorsed by less than 4% of the sample. Five (2.3%) participants endorsed a different index trauma at the follow-up assessment. At the initial assessment, 55% percent of the sample met criteria for current PTSD, and 57% of the sample with follow-up data met criteria for current PTSD at the second assessment. CAPS scores spanned a range of severity (T1: $M = 53.7$, $SD = 25.4$, $Min/Max = 3/123$; T2: $M = 52.4$, $SD = 25.9$, $Min/Max = 0/110$), were not excessively

[3]Given that the duration of the follow-up period varied across participants, we examined whether follow-up length was associated with the study variables. Follow-up length was not related to any of the T1 or T2 variables, including exposure to new adverse events ($r = .12$). It was also unrelated to mean level changes in personality traits and PTSD symptoms over time. Consequently, we did not include it as a covariate in analyses.

[4]PTSD scores were retained for individuals with invalid personality scores, because the PTSD scores were obtained from clinical interview and did not rely solely on self-report.

[5]Supplementary analyses showed that removing participants with incomplete follow-up data did not change the results or produce new findings.

skewed or kurtotic (values ranged from −.61 to .11), and showed excellent internal consistency (Cronbach's alpha coefficient = .87 for both the initial and follow-up assessments). Secondary ratings of the video-taped interviews were completed by an independent rater for approximately one-third of participants (T1 *n* = 68, T2 *n* = 50) and used to calculate inter-rater reliability, which was high (intraclass correlation coefficient = .93).

Multidimensional Personality Questionnaire – Brief Form—(MPQ-BF; Patrick, Curtin, & Tellegen, 2002). At both time points, participants completed the widely-used and well-validated MPQ-BF (Patrick et al., 2002), a 155-item self-report personality inventory derived from the full-length 276-item MPQ (Tellegen, 1982). The MPQ-BF is composed of 11 primary trait scales that converge into three higher-order temperament scales that assess negative emotionality (NEM), positive emotionality (PEM), and constraint (CON). Negative emotionality assesses tendencies toward distress, anxiety, aggression, hostility, and estrangement from/ suspicion of others. Positive emotionality assesses a positive disposition, sociability, agency, and social dominance. Constraint measures impulsivity (reversed), thrill-seeking or fearlessness (reversed), and conformity to social norms. Negative emotionality, positive emotionality, and constraint were created by summing the raw scores of the primary trait scales that make up each higher-order temperament scale and used as explanatory variables in analyses. Invalid response profiles were determined by the MPQ-BF validity scales (Variable-Response Inconsistency, VRIN; True-Response Inconsistency, TRIN; and Unlikely Virtues). As defined by Patrick et al. (2002), response profiles were considered invalid if they met one of these criteria: VRIN > 3 SDs above the sample mean, TRIN > +/− 3.21 SDs from the sample mean, or VRIN > 2 SDs above the sample mean and TRIN > +/− 2.28 SDs from the sample mean.

Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000)—At the follow-up assessment, participants were given a form with a list of major adverse life events from the TLEQ and asked to mark whether or not each of the events listed had occurred since the date of their initial assessment. We did not measure the dependency of the event (i.e., how much of a role the individual played in creating the event), and thus only included events in our analyses that an individual's symptoms or personality could have possibly influenced. More specifically, we included any event where an individual's personality or psychopathology symptoms could directly or indirectly influence the likelihood that an event would occur. For instance, personality and psychopathology may increase the likelihood an individual will associate with people who are at risk for stressful events, select into environments that are dangerous or stressful, and/or react strongly to stressful events, all of which could directly or indirectly cause a myriad of adverse outcomes (e.g., health problems, accidents or premature deaths of close others, assault, etc.). Consequently, we excluded the natural disaster item of the TLEQ, because exposure to this type of event is highly random (i.e., exposure is almost entirely outside an individual's control) and, therefore, it is theoretically distinct from the other events on the TLEQ. Also, it rarely occurred in this sample (< 6%) and when this item was included in the total count variable, the results did not change and no new results emerged. We did not assess whether the events met full criteria for the DSM-IV definition of trauma (e.g., A1 and A2), and consequently

we refer to them as major adverse life events rather than traumatic events. A count variable was created by summing the number of different types of events participants endorsed during the follow-up period from the following list: motor vehicle accident, combat or warfare, sudden death of friend/loved one, life-threatening/disabling event to loved one, life-threatening illness, robbery/ or assaulted with a weapon, assaulted by an acquaintance or stranger, witnessed severe assault to an acquaintance or stranger, threatened with death or serious harm, unwanted sexual contact, sexual harassment, stalked, miscarriage, abortion. The TLEQ demonstrates excellent convergent validity with interview-based measures of trauma exposure, and the total number of events has been shown to correlate positively with PTSD (Kubany et al., 2000).

In addition to a total event type, we also created assaultive and non-assaultive event variables. These variables were created to differentiate stress exposure that involved directly experiencing or witnessing physical assault from experiences that are not violent in nature. Assaultive events were operationalized as exposure to robbery/ or assaulted with a weapon, assaulted by an acquaintance or stranger, witnessed severe assault to an acquaintance or stranger, and threatened with death or serious harm. Non-assaultive events were operationalized as event types that did not involve physical assault to self or others, including a motor vehicle accident, life-threatening/disabling event to loved one, and life-threatening illness.

Procedure

At each assessment, participants completed a battery of self-report questionnaires and diagnostic interviews. The diagnostic interviews were administered by doctoral- and masters-level clinical psychologists and clinical psychology trainees who underwent extensive training on the interview and rating procedures. Relevant institutional review boards approved the study procedures prior to data collection, and participants gave written informed consent after they were provided with a detailed description of the study. Participants received monetary compensation for their involvement.

Data Analysis

Analyses were conducted with the software programs SPSS version 22 (SPSS, Chicago, IL) and Mplus 7.11 (Muthen & Muthen, 2013). Cross-lagged and path analyses analyzed with Mplus used all available data (under direct maximum likelihood estimation), and simple descriptive statistics analyzed with SPSS had some missingness. To provide context for the cross-lagged and path model analyses, we performed descriptive repeated measures ANOVAs with time as the within-subjects variable were used to test for changes in mean levels of the personality traits and PTSD symptoms over time using SPSS, and partial eta squared is reported as a measure of effect size. In Mplus, we specified cross-lagged panel models to assess temporal relations between the study variables assessed at two time points using the robust maximum likelihood estimator (MLR). This analytic approach allowed us to account for non-normality of variables and include all 222 participants (with missing data modeled directly using direct maximum likelihood estimation). The role that intervening major adverse life events play in mediating change in personality functioning and PTSD

symptom severity over time was examined using Model INDIRECT in Mplus. Given that the models have zero degrees of freedom, model fit was always perfect.

Per our first two aims, cross-lagged models were analyzed to assess the reciprocal effects of personality functioning and PTSD symptoms. Each model contained PTSD symptoms and one of the MPQ personality factors measured at T1 and T2. This allowed us to model the influence of 1) each personality factor on future PTSD severity while taking into account baseline PTSD symptoms and 2) PTSD symptoms on future personality functioning while taking into account baseline levels of that trait. Autoregressive paths were also included in the models. Standardized parameter estimates are depicted in Figure 1.

Next, we added exposure to new events between the T1 and T2 assessments as a mediator in the cross-lagged models to address aims 3–5. We tested the indirect paths from T1 personality and PTSD symptoms to these variables at T2 via exposure to new events in a separate analysis for each personality factor. This allowed us to examine the extent to which PTSD and personality functioning predict exposure to new events as well as how new events influence future PTSD symptoms and personality functioning. We also provide the 95% bootstrapped confidence intervals for significant indirect effects.

Results

Descriptive Statistics

Estimated means, standard deviations, and bivariate correlations for the study variables are listed in Table 1. Overall, 82% of the sample reported exposure to at least one major adverse life event during the follow-up period, and the mean number of new event types was two. Approximately, 36.5% of participants reported the sudden death of a friend or loved one, 24.3% reported threatened or actual physical assault, 22.3% reported a motor vehicle accident, 20.3% reported a life-threatening or disabling event happened to a loved one, 19.6% reported the onset of a life-threatening illness, and 8.1% reported witnessing a severe assault. Other adverse life events (e.g., unwanted sexual contact, sexual harassment, combat exposure) were endorsed by less than 4% of the sample.

The mean severity of PTSD symptoms in the sample did not change from T1 to T2; however there was a decrease in negative emotionality ($F_{(1, 127)} = 31.12$, $p < .001$, $n^2_p = .20$), and increases in positive emotionality ($F_{(1, 127)} = 6.12$, $p = .014$, $n^2_p = .05$) and constraint ($F_{(1, 127)} = 5.29$, $p = .023$, $n^2_p = .04$) over time. At both time points, negative emotionality and constraint were negatively correlated, whereas positive emotionality was not significantly related to either of these personality factors. Bivariate relations among the psychopathology-personality variables showed that T1 PTSD correlated positively with negative emotionality and negatively with positive emotionality at both time points, but was uncorrelated with constraint at either time point. In contrast, T1 negative emotionality was positively associated with T2 PTSD, and T1 constraint was negatively associated with T2 PTSD. In contrast, T1 positive emotionality was not significantly related to T2 PTSD.

Cross-Lagged Models

Per our first two aims, we tested the hypothesis that personality and PTSD symptoms exert reciprocal effects over time such that each predicts subsequent levels of the other. As hypothesized and illustrated in Figure 1, T1 negative emotionality positively predicted T2 PTSD (β = .17, , *SE* = .06, *p* = .008), indicating that higher T1 negative emotionality led to increases in T2 PTSD symptoms over and above the influence of T1 PTSD on T2 PTSD. The cross-lagged path from T1 PTSD to T2 negative emotionality was not significant, however. The full model explained 52% of the variance in negative emotionality and 37% in PTSD at T2.

Contrary to hypotheses, T1 positive emotionality did not predict T2 PTSD, and T1 PTSD did not predict T2 positive emotionality. Overall, the full model explained 55% of the variance in T2 positive emotionality and 35% of the variance in T2 PTSD. Neither of the direct paths from T1 constraint to T2 PTSD or from T1 PTSD to T2 constraint was significant. The full cross-lagged model explained 55% of the variance in constraint and 35% in PTSD at T2.

Stress Exposure Analysis

To address aims 3–5, we entered exposure to new major adverse events between T1 and T2 as a mediator in the cross-lagged path analyses. The primary analysis used total number of new events, and secondary analysis examined assaultive and non-assaultive events. A separate model was tested for each personality factor, including those that did not show zero-order relations with PTSD symptoms, in order to test for inconsistent mediation (i.e., when mediated effects have different signs; MacKinnon, Fairchild, & Fritz, 2007).

The first model examined relations among negative emotionality, PTSD, and new events, and Figure 2 depicts the standardized parameter estimates for this analysis. A significant direct path emerged from T1 negative emotionality to new events (β = .24, *SE* = .09, *p* = .012), indicating that higher negative emotionality at T1 predicted greater exposure to future adverse events. In contrast, the direct path from T1 PTSD to new events was not significant.[6] Significant paths also emerged from new events to T2 negative emotionality (β = .24, *SE* = .05, *p* < .001) and from new events to T2 PTSD (β = .25, *SE* = .06, *p* < .001), such that exposure to new adverse events between T1 and T2 increased levels of both T2 negative emotionality and T2 PTSD. With the addition of new events to the model, the direct effect of T1 negative emotionality on T2 PTSD was no longer significant. The indirect paths from T1 negative emotionality to T2 negative emotionality (β = .06, *SE* = .03, *p* = .029, 95% CIs = .01, .11), and from T1 negative emotionality to T2 PTSD (β = .06, *SE* = .03, *p* = .04, 95% CIs = .01, .12) via new events were both significant, indicating that exposure to intervening stressors mediated the effects of T1 negative emotionality on both T2 negative emotionality and T2 PTSD severity. The full model explained 6% of the variance in exposure to new

[6]We examined whether the differential associations of the personality factors and PTSD symptoms with new adverse events was due to the use of self-report vs. a structured clinical interview to assess these constructs, respectively. We used the total score from the self-report PCL-C administered during the phone screen and examined the effects of T1 negative emotionality, constraint, and PTSD symptoms on new adverse events. Significant direct paths to new adverse events emerged for T1 negative emotionality (*p* = .04) and T1 constraint (*p* = .013), but not PCL-C total, suggesting that the stronger effects of the personality factors than PTSD symptoms on new event exposure was not an artifact of shared method variance.

events, 57% of the variance in T2 negative emotionality, and 42% of the variance in T2 PTSD.

Analysis of the mediation model for positive emotionality did not change the results of the cross-lagged analysis (the autoregressive paths remained significant and the cross-lagged paths remained non-significant). The only new result was a significant direct path from exposure to new events to T2 PTSD (β = .27, *SE* = .05, *p* < .001), with greater exposure predicting greater T2 PTSD severity. The model explained 2% of the variance in new events, 55% of the variance in T2 positive emotionality, and 42% of the variance in T2 PTSD.

Next, we examined relations between constraint, PTSD symptoms, and new events, and the standardized parameter estimates are depicted in Figure 3. This analysis produced a significant direct path from T1 constraint to new events (β = −.20, *SE* = .07, *p* = .003), with lower levels of T1 constraint predicting greater exposure to future adverse events. Consistent with the other models, the direct path from T1 PTSD to new events was not significant. Exposure to new events did not predict T2 constraint. As with the other models involving this same pathway, new events predicted T2 PTSD (β = .27, *SE* = .06, *p* < .001). The indirect path from T1 constraint to T2 PTSD via new event exposure was also significant (β = −.05, *SE* = .02, *p* = .017, 95% CIs = −.10, −.01), which indicates that exposure to adverse events between T1 and T2 mediated the effect of T1 constraint on T2 PTSD severity. The full model explained 4% of the variance in new events, 55% of the variance in T2 constraint, and 41% of the variance in T2 PTSD.

We then examined whether the results for the total number of event types used in the main analyses replicated for assaultive and non-assaultive events. We found that results replicated for assaultive but not non-assaultive events. T1 negative emotionality and T1 constraint exerted direct effects on exposure to assaultive (negative emotionality: β = .30, *SE* = .09, *p* < .001; constraint: β = −.22, *SE* = .06, *p* < .001) but not non-assaultive events. Assaultive and non-assaultive events both predicted T2 PTSD (Assaultive: β = .17, *SE* = .06, *p* = .004; Non-Assaultive: β = .19, *SE* = .06, *p* = .003) and T2 negative emotionality (Assaultive: β = .20, *SE* = .06, *p* < .001; Non-Assaultive: β = .11, *SE* = .06, *p* = .043). Exposure to new assaultive events between T1 and T2 mediated the effect of T1 personality on T2 PTSD severity levels for negative emotionality (β = .05, *SE* = .03, *p* = .049, 95% CIs = .00, .11) and constraint (β = −.04, *SE* = .02, *p* = .019, 95% CIs = −.08, .00). New assaultive events also mediated the effect of T1 negative emotionality on T2 negative emotionality (β = .06, *SE* = .03, *p* = .014, 95% CIs = .01, .11). Non-assaultive events did not mediate personality-PTSD relations.

Discussion

With nearly 40% of individuals with PTSD reporting persistent symptoms (Santiago et al., 2013), understanding the factors that maintain chronic symptomatology is critically important for improving clinical outcomes. This study examined the reciprocal effects of personality traits and PTSD symptoms over time and tested hypotheses about the stress-related effects of personality traits and PTSD on one another via exposure to new major

adverse events. Analyses revealed several findings that advance our understanding of the progression of PTSD symptoms in trauma-exposed individuals, most notably that high negative affect and disconstraint play key roles in determining the course of PTSD symptoms by conferring risk for exposure to new major adverse events.

Reciprocal Effects of Personality and PTSD

Examination of the reciprocal effects of personality and PTSD symptoms converged with previous research showing that high negative affect is a risk factor for PTSD (Miller, 2003; Miller et al., 2012; Parslow et al., 2006) and extended this work by demonstrating that it also influences the course of PTSD symptoms. Although negative emotionality and positive emotionality were both correlated with concurrent PTSD symptoms, only the cross-lagged effect of negative emotionality on future PTSD symptom severity was significant. These findings suggest that, although low positive affect characterizes individuals with greater PTSD symptoms, it does not necessarily function to maintain these symptoms over time. Relative to trait negative affect, bivariate relations between constraint/conscientiousness and PTSD symptoms are generally weaker (Kotov et al., 2010), and we did not find a direct relation between constraint and future PTSD in this sample.

Findings do not support the hypothesis that PTSD influences the expression of personality traits over time, given that symptom severity did not show any direct effects on future personality functioning. However, alterations in personality functioning related to psychopathology may be most evident when comparing premorbid and postmorbid levels of personality. Recruitment of a trauma-exposed sample with PTSD symptoms precluded this type of analysis and may have limited our ability to detect changes in personality functioning related to PTSD. Thus, prospective studies that measure premorbid and postmorbid personality functioning are needed to evaluate whether the onset of PTSD shifts dispositional tendencies towards more extreme personality variants. Further, although PTSD did not influence the broad domains of normal personality traits assessed in this study, it may predict changes in the course or severity of personality disorders.

Stress Exposure, Personality, and PTSD Symptom Course

To our knowledge, this is the first study to explicitly test the simultaneous influences of personality, PTSD, and stress exposure on the course of PTSD. We hypothesized that initial PTSD severity, low positive affect, trait negative affect, and disconstraint would all positively predict new major adverse events during follow-up. Contrary to expectations, PTSD severity did not confer risk for exposure to new events. Rather, we found that trait negative affect and disconstraint played a greater role in stress exposure than PTSD symptoms. Secondary analyses indicated that these personality traits conferred risk for new assaultive events, but not non-assaultive events. This finding is consistent with research suggesting that assaultive events are more genetically-mediated, and therefore more influenced by individual difference characteristics, than non-assaultive events (e.g., Afifi et al., 2010). The relation of negative emotionality and constraint, but not positive emotionality, with assaultive events may reflect the tendency for individuals high on negative affect and disconstraint to associate with others who are aggressive and impulsive, initiate physical altercations, and select into dangerous environments that put them at risk

for assault. Research has identified personality traits that increase risk for traumatization and the development of PTSD, including high neuroticism (e.g., Breslau, Davis, & Andreski, 1995). Our findings extend this work by demonstrating that personality-based risk factors for stress exposure also influence the course of PTSD.

Consistent with previous research, exposure to major adverse events predicted greater future PTSD symptoms after adjusting for baseline symptom severity, and this finding held for both assaultive and non-assaultive event exposure. These findings converge with studies that find exposure to new major adverse events predicts PTSD symptom course (Perkonigg et al. 2005) and PTSD symptoms are exacerbated by new onset stressors (Miller et al., 2013). New events also positively predicted negative emotionality in our study, suggesting that stress exposure influenced personality functioning as well as psychopathology severity.

As predicted, major adverse events mediated the effects of personality on the course of PTSD, which is a novel finding that helps clarify the role of individual differences in personality and stress exposure on symptom presentation in trauma-exposed individuals. High negative affect and disconstraint represent potentially distinct pathways that influence PTSD severity via stress exposure. Interestingly, our results suggest that, although exposure to assaultive and non-assaultive events both increase subsequent PTSD, only assaultive events mediate the relations of negative emotionality and constraint with future PTSD severity. Thus, exposure to assaultive events appears to be particularly important for understanding the influence of negative affect and disconstraint on the course of PTSD. Our findings suggest that attending to personality-based heterogeneity associated with trait negative affect and disconstraint is necessary for developing a comprehensive understanding of chronic symptom presentations.

Personality traits influence the types of environments an individual actively seeks out and the responses an individual evokes from the environment (Plomin & DeFries, 1977), both of which are potential explanations for the association between personality and stress exposure found in this study. Given that we did not assess whether participants played an active role in generating the events they reported, our findings may reflect the tendency for individuals with certain personality traits to chronically select into stressful environments or generate environmental stressors themselves. Further, the type and severity of adverse events we studied may have influenced the findings. A large body of research finds that individuals with depressive syndromes play an active role in generating stressful life events that are dependent in nature and less severe than those measured in this study (Hammen, 1991; Liu & Alloy, 2010). Thus, a more fine-grained assessment of particular event types than was possible in this study is necessary to test whether PTSD symptoms generate dependent stressful life events (e.g., divorce, job loss) and how such events relate to the progression of symptoms.

Limitations and Conclusions

As with any investigation, there are potential limitations to note. The primarily male (and 100% veteran) composition of the sample may have influenced the relative importance of certain personality traits over others. For example, men tend to score lower on measures of behavioral constraint than women (e.g., Roberts, Caspi, & Moffitt, 2001), which may have

strengthened our ability to detect relations between disconstraint and stress exposure. There is also evidence that the severity and types of stressful events that are associated with risk for psychopathology are different in men and women (e.g., Harkness et al., 2010). Thus, the generalizability of the findings to trauma-exposed women and examination of potential gender differences in the findings is an important area for future research. Given that the number of intervening adverse events was fairly low on average, the sample size was modest, and the strength of the indirect and cross-lagged effects were small, results require replication. Although our subsidiary analyses did not suggest that attrition or variability in the follow-up period affected the findings, replication is needed to definitively rule out potential confounds. Information about adverse events were collected at the second assessment and may have been influenced by method effects or retrospective biases associated with PTSD symptoms at follow-up, although looking at major adverse events likely improved the accuracy of event recall. Further, the findings cannot speak to the temporal ordering of changes in PTSD symptoms and exposure to stressful events as symptoms may have increased before event exposure. In addition, our design did not account for a history of stressful life events at Time 1, and thus, the possibility that the factors leading to stressful events at Time 1 are maintained over the interval and influence the occurrence of stressful life events at Time 2. The results of the study suggest that personality traits may be one of those factors, although there are likely others that were not tested in our models. Finally, this study only assessed posttrauma indicators of personality functioning and the findings cannot speak to whether pretrauma personality traits influence the development of PTSD.

This study also has several strengths, including the prospective cross-lagged design that strengthened our ability to make inferences about temporal relations between the study variables, a theory-driven analysis of the interplay between personality, psychopathology, and stress exposure, and recruitment of a trauma-exposed sample with a high representation of chronic PTSD symptoms. It advances understanding of the course of PTSD by identifying distinct personality-based mechanisms of stress exposure that appear to perpetuate symptoms over time.

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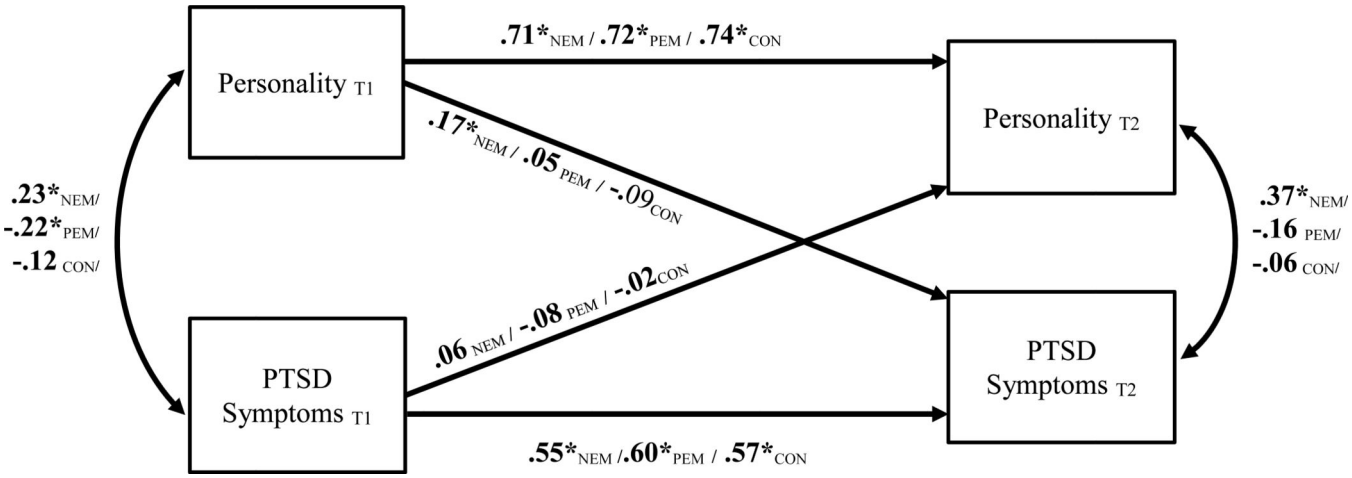

Figure 1.

Cross-lagged analysis of personality trait factors and posttraumatic stress severity. Standardized parameter estimates for the personality domains of Negative Emotionality/ Positive Emotionality/ Constraint. PTSD = Posttraumatic Stress Disorder. NEM = Negative Emotionality. PEM = Positive Emotionality. CON = Constraint. *$p < .01$.

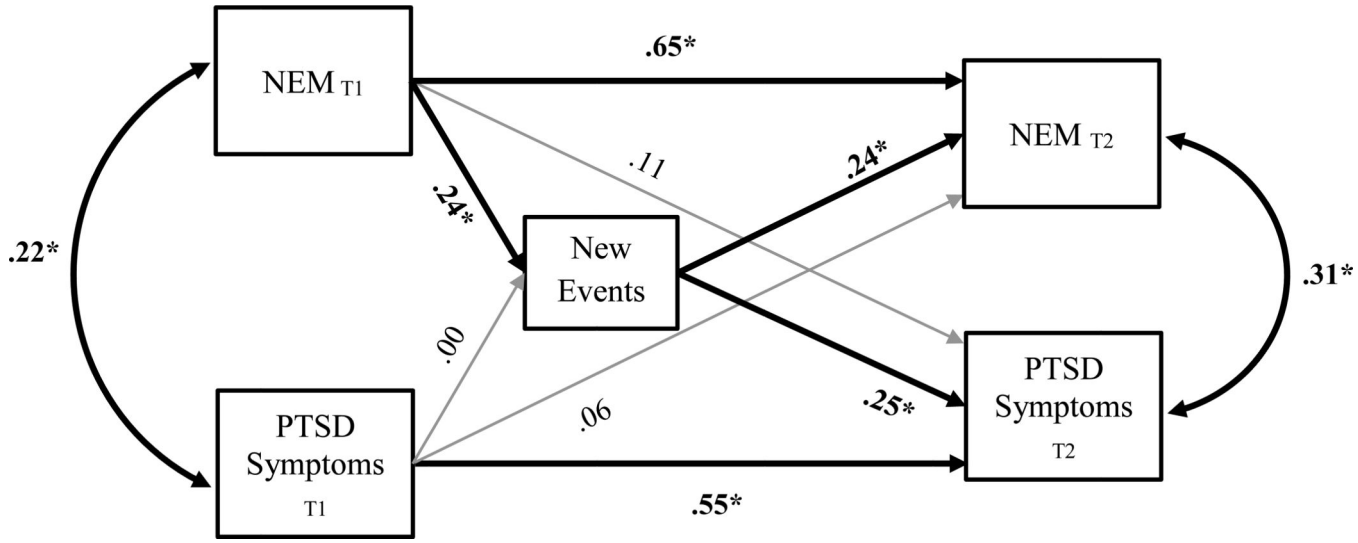

Figure 2.

Cross-lagged path analysis of trait negative emotionality and posttraumatic stress severity measured at baseline and four-year follow-up with intervening adverse life events examined as a mediator. Standardized parameter estimates are provided for each path. PTSD = Posttraumatic Stress Disorder. NEM = Negative Emotionality. *$p < .05$.

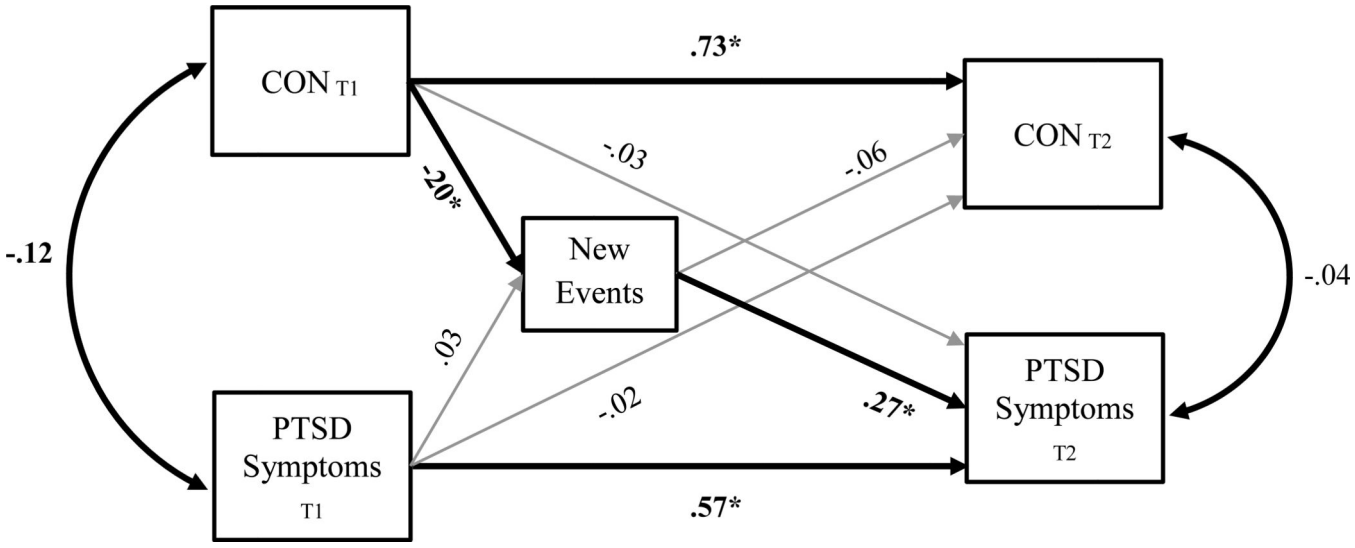

Figure 3.

Cross-lagged path analysis of trait constraint and posttraumatic stress severity measured at baseline and four-year follow-up with intervening adverse life events examined as a mediator. Standardized parameter estimates are provided for each path. PTSD = Posttraumatic Stress Disorder. CON = Constraint. *$p < .05$.

Table 1

Descriptive Statistics for the Study Variables

	Mean (SD)	1	2	3	4	5	6	7	8
Time 1									
[1] PTSD Symptoms	53.7 (25.4)								
[2] Negative Emotionality	54.7 (18.1)	.22*							
[3] Positive Emotionality	50.9 (16.9)	−.23*	−.08						
[4] Constraint	77.3 (13.3)	−.12	−.21*	−.02					
Time 2									
[5] PTSD Symptoms	53.5 (25.9)	.59*	.29*	−.09	−.16*				
[6] Negative Emotionality	47.4 (18.3)	.22*	.72*	−.08	−.23*	.45*			
[7] Positive Emotionality	53.4 (15.7)	−.25*	−.13	.74*	−.03	−.20*	−.12*		
[8] Constraint	79.9 (13.4)	−.10	−.15*	.02	.74*	−.16*	−.20*	.09	
[9] New Major Adverse Events	1.9 (2.0)	.07	.26*	.08	−.21*	.31*	.41*	.00	−.21*

Note. PTSD = Posttraumatic Stress Disorder.

* $p < .05$.