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Stress-Generative Effects of PTSD: Transactional Associations between PTSD and Stressful Life Events in a Longitudinal Sample

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Abstract

Longitudinal studies have demonstrated transactional associations between psychopathology and stressful life events (SLEs), such that psychopathology predicts the occurrence of new SLEs and SLEs in turn predict increasing symptom severity. The relationship between posttraumatic stress disorder (PTSD), specifically, and stress-generation remains unclear. This study used temporally sequenced data from 116 veterans (88% male) to examine if PTSD symptoms predicted new onset SLEs, and if these SLEs were associated with subsequent PTSD severity. SLEs were objectively rated using a clinician administered interview and consensus rating approach to assess the severity, frequency, and personal dependence (i.e., if the event was due to factors that were independent of or dependent on the individual) of new-onset SLEs. A series of mediation models were tested and results provided evidence for moderated mediation whereby baseline PTSD severity robustly predicted personally-dependent SLEs (unstandardized $B = .03$, $p = .006$), and dependent SLEs predicted increases in follow-up PTSD symptom severity (unstandardized $B = -.04$, $p = .006$) among those with relatively lower baseline PTSD severity. Personality traits marked by low constraint (i.e., high impulsivity) were also associated with an increased number of dependent SLEs, controlling for baseline PTSD severity. Results provide evidence for a stress-generative role of PTSD and highlight the importance of developing interventions to reduce the occurrence of personally-dependent stressors.

Symptoms of chronic posttraumatic stress disorder (PTSD) fluctuate over time and can be influenced by a variety of environmental factors, including new-onset adverse life events and daily life stressors, which tend to further exacerbate trauma-related symptoms (Cerda et al., 2013; Gehrman et al., 2015; Sadeh et al., 2015; Vazan et al., 2015). Evidence also suggests

that the relationship between new onset stressors and symptom exacerbation may be transactional, such that symptoms and stressors may interact with, and reciprocally influence, each other. Stress-generation is the concept that individuals are not merely recipients of life stress, but can also influence their surroundings in ways that contribute to new adversity (Hammen, 1991). Individuals with certain personality traits and/or psychopathology have been shown to “select themselves into” difficult environments (Harkness & Stewart, 2009; Kendler et al., 1999b; Liu & Alloy, 2010), and twin studies found genetic predispositions towards exposure to stressful life events (SLEs; (Foley et al., 1996; Kendler et al., 1999b; Kendler et al., 1993).

Longitudinal studies provide preliminary support for transactional associations between psychopathology and stress-generation. For example, a recent study of World Trade Center first-responders found that depressive symptoms at initial assessment predicted later SLEs, such as financial or interpersonal problems, which were in turn associated with greater depression severity at a follow-up assessment (Zvolensky et al., 2015). Similarly, in a sample of veterans with chronic PTSD, Sadeh et al. recently reported that the personality traits negative emotionality (NEM, i.e., negative affect, and reactivity) and disinhibition (i.e., impulsivity) measured at an initial assessment predicted reports of exposure to new adverse life events during a four year interval and that these events, in turn, were associated with greater PTSD severity at follow-up (Sadeh et al., 2015). In a subsequent study of the same cohort focused on the association between PTSD and risky, self-destructive behavior, Lusk et al. (Lusk et al., 2017) found that a composite measure of risky behavior at Time 1 predicted exposure to new adverse events (e.g., physical assault and motor vehicle accidents) and that new adverse event exposure mediated the relationship between risky behavior and future PTSD severity. Both of these studies were limited, however, by their reliance on a retrospective self-report measure of new onset life events over the previous four year interval. Critics of the use of retrospective self-reports of life stressors have argued that negative recall biases inflate endorsement of negative events and, as such, may be unreliable (Dekel et al., 2016; Koenen et al., 2007; Raphael, 1991).

Critics have also suggested that since self-report measures do not include an objective assessment of the severity of the event, or of the respondent’s role in it, data may be biased (Brown, 1973). To address this issue, investigators have developed semi-structured interviews designed to be administered by a trained professional to gather information about life events and to then further evaluate the type, severity, and role of the individual in the event (i.e., the dependence vs. independence of the individual’s role in the event) using an objective panel of raters (Brown & Harris, 1989; Kendler et al., 1999a). This approach is considered psychometrically superior to checklists because the panel making the determinations is blind to the respondent’s mental health status and life events are rated using a standardized system with clear guidelines. Studies comparing checklist versus interview measures suggest that interview measures are superior for detecting the presence or absence of severe life events (McQuaid et al., 1992) and for predicting treatment outcome (McQuaid et al., 2000).

Aims and Hypotheses

The goals of this study were to evaluate the association between PTSD symptoms and new onset SLEs (i.e., stress generation) and determine if SLEs were associated with subsequent PTSD. This study improves upon prior methodology by using objective, clinician-determined ratings of the severity, frequency, and personal dependence of SLEs. We also sought to extend prior research by examining if PTSD and temperament traits were differentially associated with personally dependent or independent SLEs.

To evaluate this, we first conducted a temporally-sequenced mediation model in which PTSD symptoms predicted intervening clinician-rated dependent and independent SLEs, which in turn predicted subsequent PTSD symptoms. We hypothesized that PTSD symptoms would be most strongly associated with new dependent SLEs. Second, we tested if this mediated effect might be dependent on the initial level of PTSD symptoms (i.e., moderated mediation). We had two competing hypotheses about this. Specifically, we reasoned that the hypothesized mediation model might be strongest among those with the greatest baseline PTSD symptom severity because greater PTSD severity might exacerbate the negative effects of dependent SLEs on later PTSD. Alternatively, we reasoned it was also possible that the mediated effect might be strongest among those with less severe baseline PTSD symptoms because the contribution of new life stress to later PTSD might be more evident in this less symptomatic group, whereas those with high PTSD might be expected to simply show a chronic course of PTSD regardless of new stressors. Third and finally, to evaluate the relative contribution of PTSD versus personality to SLEs, we also examined how associations between PTSD and SLEs changed when personality variables were added to the model. We expected that NEM would be positively associated with dependent SLEs while positive emotionality (PEM, i.e., positive affect and social affiliativeness) and constraint (CON, i.e., the opposite of impulsivity) would be inversely associated with dependent SLEs.

Methods

Participants

Participants for this study were veterans involved in waves two and three of data collection from a longitudinal study of chronic PTSD. We focused on this cohort because they were the only ones to complete the Stressful Life Events interview (administered at wave three, described below). Results spanning the first two waves of data collection were reported previously (Lusk et al., 2017; Sadeh et al., 2015). The time 2 (T2) sample consisted of 167 veterans who screened positive for probable PTSD per *DSM-IV*, as determined by phone administration of the PTSD Checklist (PCL; Weathers et al., 1993). 116 (69%) of those participants completed the time 3 (T3) assessment an average of 20.03 months ($SD = 3.69$, range: 13.14 – 32.62) later, yielding a final sample of 116 for this report.

Of the final sample, participants were mostly male ($n = 102$, 87.9%), with an average age at T2 of 56.23 years ($SD = 9.54$; range: 27 – 70). Self-reported race yielded: 89 (76.7%) white, 28 (24.1%) black or African-American, 1 (0.9%) Asian, 9 (7.8%) American Indian or Alaska Native, and 2 (1.7%) reported being of unknown decent.¹ Additionally, 2.6% of participants self-reported their ethnicity as Hispanic or Latino. Participants reported serving

in the Vietnam (59.5%), Korean (0.9%), Operation Desert Storm (9.5%), OEF/OIF (12.1%), and other (18.1%) eras of military service.

Measures

Clinician Administered PTSD Scale (CAPS; Blake et al., 1990)—The CAPS, the 30-item gold-standard structured diagnostic interview for the assessment of *DSM-IV* PTSD, was administered at all time points to index current PTSD severity and diagnosis. The interview includes 17 PTSD criteria rated on two 5-point scales to assess symptom frequency and intensity. Severity scores for PTSD symptomology experienced in the past month were calculated by summing the frequency and intensity ratings for each of the 17 symptoms. PTSD diagnoses followed the frequency 1 and intensity 2 rule in the context of the *DSM-IV* symptom cluster algorithm (Weathers et al., 1999). Interviews were videotaped, and 30% of the T2 recorded interviews were independently coded by a second rater, resulting in an intraclass correlation coefficient for severity scores of $r = .99$ and $\kappa = .93$ at T2. The primary rater's scores were used for all analyses.

Stressful Life Events Interview (SLE; Kendler et al., 1998)—The SLE is a semi-structured clinical interview used to determine the occurrence of 11 SLEs that directly affected the participant during the two years between T2 and T3. SLEs included assault, divorce/separation, major financial problems, serious housing problems, serious illness or injury, job loss, legal problems, loss of confidant, serious marital problems, robberies, and serious difficulties at work. Additionally, events directly affecting someone in the participant's network (e.g., spouse, child, parent, siblings) that also had an impact on the participant were recorded. Interviewers considered the context, frequency, and nature of each event to determine its long-term contextual threat (LTCT) and the dependence of each event on the individual. LTCT was rated on a 4-point scale representing minor, low moderate, high moderate, and severe levels of threat, adapted from criteria introduced in Brown (1989). Dependence was operationally defined as how probable it was that the participant's own behavior contributed to or caused the SLE. Those determinations were rated on a 4-point scale: clearly independent, probably independent, probably dependent, and clearly dependent. Following Kendler (1999), interpersonal events were assumed to be at least probably dependent unless there was significant evidence suggesting otherwise. Although the SLE also inventories personal illness or injury, the high rate of chronic illness in our sample made it difficult to discern the number of new-onset personal health events during the two year interval the interview covered. There was also significant ambiguity when attempting to determine the personal dependence of these health events because we did not have information to determine, for example, if an individual's health diagnosis was due primarily to poor health behaviors (e.g., smoking or nutrition), genetics, or environmental factors. For these reasons, we excluded personal health events from analysis.

Interviews were videotaped, and ratings were made by consensus of at least three study team members. This approach allowed the consensus team to refer to the Stressful Life Events manual for exemplars and discuss decisions about dependence/independence and long-term

¹Self-reported race was not mutually exclusive, therefore percentages may add up to greater than 100.

contextual threat in a fashion that is not possible for an interviewer during an assessment. Study members were instructed to rate events based on what most people would be expected to feel about an event, rather than taking into account the respondent's beliefs about or reaction to the event, as per methods described by Brown and Harris (1989). Ratings of the dependence of SLEs were dichotomized into clearly/probably dependent versus clearly/probably independent to form two SLE severity scales. For these analyses, a total severity score for dependent and independent events was calculated by summing the frequency and severity (i.e., LTCT) of each respective event type.

Structured Clinical Interview for DSM-IV (SCID; First et al., 1994)—A selection of modules from the SCID-IV, including Axis I mood disorders and alcohol-use disorders (abuse or dependence), were administered by doctoral- or masters-level psychologists at T2 and T3. Typical administration allows for skipping the remaining items in a module if a participant does not endorse the initial items for that module. For our purposes, all items in the aforementioned modules were administered to allow for the calculation of dimensional ratings for these diagnoses. Severity scores were calculated by summing the number of symptoms endorsed for each diagnosis. Diagnostic determination followed criteria defined by *DSM-IV*. Interviews were videotaped, and approximately 30% of recorded interviews were independently rated by a second rater, resulting in an intraclass correlation coefficient for major depressive disorder symptom severity of $r = .96$ at T2 and an intraclass correlation coefficient for alcohol use disorder severity of $r = .99$ at T2. The primary rater's scores were used for all analyses.

Multidimensional Personality Questionnaire – Brief Form (MPQ-BF; Patrick et al., 2002)—Participants completed the 155-item self-report MPQ-BF at T2 to assess temperament traits. This survey assesses 11 primary trait scales (e.g., Well-being, Alienation, Aggression) that contribute to three higher-order temperament scales labeled Negative Emotionality (NEM), Positive Emotionality (PEM), and Constraint (CON). The higher order temperament scales reflect weighted contributions of the primary trait scales based on the results of factor analyses conducted in the original validation sample (Patrick et al., 2002). NEM is derived from the three primary trait scales labeled Stress Reaction, Alienation, and Aggression. The primary trait scales of Well-Being, Social Potency, Achievement, and Social Closeness contribute to PEM, while CON is derived from Control, Harm Avoidance, and Traditionalism. Six participants yielded invalid profiles on the validity and consistency scales (per Patrick et al., 2002) included in the assessment and were therefore excluded from any analyses utilizing temperament scales.

Procedure

Participants were initially recruited through a designated database of veterans who had consented to be contacted for research, flyers, and clinician referrals. Research assistants conducted a telephone screen with interested veterans to determine eligibility, which included screening positive for probable PTSD on the PCL (Weathers et al., 1993) using the *DSM-IV* algorithm. Specifically, participants needed to endorse one symptom in the B cluster, three symptoms in the C cluster, and two symptoms in the D cluster to screen into the study, with at least moderate symptom severity considered item endorsement.

Participants then completed a T1 assessment that is not the focus of this study (Lusk et al., 2017; Sadeh et al., 2015). At T2, data collection (self-report inventories and videotaped diagnostic interviews) occurred over two sessions, each lasting approximately 3–4 hours, scheduled no more than one week apart. T3 data collection occurred in one 3–4 hour session and included a videotaped assessment of current psychiatric symptoms and of intervening SLEs between T2 and T3. All participants provided written informed consent at each time point and were compensated for study participation. The study was approved by the VA Boston Healthcare System institutional review boards.

Statistical Analyses

We first evaluated descriptive statistics pertaining to T2 and T3 PTSD diagnosis and severity, depression severity, and independent and dependent SLEs. We then conducted our mediation models using the SPSS Process macro (version 2.16.1; Hayes, 2013). This analytic approach produces bias-corrected bootstrapped parameter estimates for the indirect effect and, as such, does not assume a normal distribution for the confidence interval surrounding the point estimate of the indirect effect (Hayes, 2013). The indirect path is interpreted in the same manner as other regression coefficients in the model and represents the magnitude of the indirect association between the independent and dependent variables via the mediator. We set the number of bootstrapped samples to 5000 for all analyses. In the mediation analysis addressing our first aim, we included T2 PTSD severity as a predictor of T3 PTSD severity via independent and dependent SLEs (i.e., two potential mediators; model “4” in Hayes, 2013). As our second aim queried if the potential indirect effects might be conditional on the level of T2 PTSD severity, we next conducted a test of moderated mediation (Hayes, 2013, 2015) to examine if the indirect effect of T2 PTSD severity on T3 PTSD severity via independent or dependent SLEs varied as a function of level of T2 PTSD severity. In this sense, T2 PTSD severity served as both an independent variable and a moderator of the indirect effect (i.e., model “74” in Hayes, 2013). In these analyses, independent and dependent SLEs were evaluated in separate models as there is no mechanism for including multiple mediators in this test of moderated mediation.

Given that the majority of prior research on the stress generative hypothesis was conducted in depression samples, we repeated our analytic approach using T2 and T3 depression symptom counts as the independent and dependent variables, respectively, in place of PTSD, in order to compare the pattern of associations for PTSD versus depression. Finally, to evaluate temperament based predictors of independent and dependent SLEs (i.e., our third aim), we ran a multiple regression in which we evaluated the effects of PEM, NEM, and CON on SLEs.

For the moderated mediation models, we used an index of the significance of the model that is calculated by the Process macro following Hayes (2015). This provides a bootstrapped 95% confidence interval (CI) for the distribution of the index of moderated mediation; if the 95% CI does not include 0 (the null), there is evidence that the mediated effect is conditional on levels of the moderator (i.e., significant moderated mediation). As Process largely returns unstandardized parameter estimates (and 95% CIs), we report unstandardized beta (B) coefficients in the results unless otherwise noted. For the sake of comparison, we obtained

completely standardized parameter estimates by reanalyzing the data in Mplus 7.11 (Muthén & Muthén, 2013) for models that were significant in Process. Analyses focused on temperament were conducted in SPSS version 21 ("IBM SPSS Statistics Version 21," 2012).

Finally, for models with significant effects, we examined potential confounds of the main associations of interest by including demographic and mental health related variables as covariates in the model. Specifically, age, sex, self-reported (at T3) mental health treatment (psychotherapy or psychiatric medication management), T2 alcohol-use disorder symptom severity (a summary of alcohol abuse and dependence symptoms on the SCID), and T2 major depression symptom severity were included as covariates (of the mediator and the dependent variable) in follow-up regression models. We focused on major depression and alcohol-use disorders because they are common comorbidities of PTSD and because both might be expected to influence SLEs.

Results

Descriptive Statistics and Bivariate Associations

Descriptive statistics pertaining to demographic information, PTSD severity, and independent and dependent SLEs are listed in Table 1. The prevalence of current PTSD diagnosis was 60.3% ($n = 70$) at T2 and 34.5% ($n = 39$) at T3; 72.2% of the participants who lost the PTSD diagnosis from T2 to T3 missed the threshold for diagnosis by 1 or 2 symptoms at T3. We also compared participants who completed both T2 and T3 assessments with those who completed T2 but not T3. T2 PTSD severity, NEM, and CON did not significantly differ between groups, but PEM was significantly higher in the group that completed T3 ($t(141) = -2.264, p = .025$).

PTSD Mediation Models

Simple Mediation (Aim 1)—We examined the indirect effects of T2 PTSD severity on T3 PTSD severity via total scores on independent and dependent SLEs using the SPSS Process macro (Hayes, 2013). This analysis revealed that T2 PTSD predicted greater dependent SLEs ($B = .03, p = .006$) and explained 6.5% of the variance in dependent SLEs ($F[1, 112] = 7.75, p = .006$). T2 PTSD also predicted T3 CAPS ($B = .69, p < .0001$), with the model explaining 53% of the variance in T3 PTSD ($F[3, 110] = 41.34, p < .001$). Dependent SLEs did not predict T3 PTSD symptoms, indicating no indirect effect of T2 PTSD on T3 PTSD via dependent SLEs. T2 PTSD did not predict independent SLEs ($R^2 = .001, F[1, 112] = .14, p = .71$) so there were also no indirect effects of T2 PTSD on T3 PTSD (see Table 2).

Moderated Mediation (Aim 2)—We next examined the moderated mediation model in which T2 PTSD severity served as both a predictor of T3 PTSD and a moderator of the indirect effect via dependent SLEs. This model yielded a significant direct effect of T2 PTSD on T3 PTSD (as in the prior model), and a significant interaction between T2 PTSD and dependent SLEs on T3 PTSD via dependent SLEs (indirect $B = -.04, p = .003$; see Table 3; significant beta coefficients are reported to two decimal points or the first non-zero integer as necessary for interpretation). The relationship between dependent SLEs and T3 PTSD was only significant among those with the lowest T2 PTSD symptoms. The index of

moderated mediation did not include 0 in the 95% CI (point estimate = $-.002$, 95% CI: $-.0040$ to $-.0002$), suggesting that the indirect effect was conditional on PTSD severity. This model explained 56.7% of the variance in T3 PTSD ($F[3, 110] = 47.76$, $p < .001$) and suggested that the indirect effect of T2 PTSD on T3 PTSD via dependent SLEs was evident only for those with relatively lower T2 PTSD symptoms: at 1 SD below the T2 CAPS mean (CAPS score of 27.15), the indirect effect was significant ($B = .05$; 95% CI: $.005$ to $.15$), but at higher levels of CAPS scores, the 95% CI for the indirect effect included the null value (see Figure 1). The unstandardized parameter estimates for the moderated mediation model are shown in Figure 2; as standardized estimates are not produced by the Process macro, we estimated these separately in Mplus and these values appear below each line in Figure 2.^{2,3} In a separate model we evaluated moderated mediation using the independent SLEs as the mediator and found no evidence for significant indirect effects at any level of the moderator (see Table 3).

Depression Mediation Models

We next followed the same analytic approach to evaluate if depression symptom counts at T2 predicted depression symptom counts at T3 via independent or dependent SLEs. There were no direct effects of T2 depression on either dependent or independent SLEs. Dependent SLEs were associated with greater T3 depression ($B = .26$, $p = .01$) and T2 depression was associated with T3 depression ($B = .50$, $p < .0001$), but no indirect effects emerged (see Table 2). The model explained 36% of the variance in T3 depression ($F[3, 108] = 19.93$, $p < .001$). There was no evidence of moderated mediation (T2 depression $\times$ SLE) for either dependent or independent SLEs (see Table 3).

Contribution of T2 Temperament to Subsequent SLEs (Aim 3)

Finally, we wondered about the relative contribution of temperament as compared to PTSD on independent and dependent SLEs. Controlling for T2 PTSD (which was significantly associated with dependent SLEs, as above), the only T2 temperament variable that predicted subsequent dependent SLEs was CON (standardized $\beta = -.22$, $p = .02$; see Table 4). In total, this model explained 12% of the variance in dependent SLEs ($F[4, 103] = 3.50$, $p = .01$).⁴ No temperament variables were significantly associated with independent SLEs ($R^2 = .02$, $F[4, 103] = 0.62$, $p = .65$; see Table 4).

²In a follow-up model, we also examined if age, sex, mental health treatment at T3 (psychotherapy or psychiatric medication management), alcohol-use disorder symptom, or T2 major depression symptom might better account for the moderated indirect effect on T3 PTSD symptoms via dependent SLEs. These five variables were included as covariates of both dependent SLEs and T3 PTSD symptoms and the moderated mediation model was re-run. Results revealed that none of these covariates were significantly associated with dependent SLEs whereas T2 alcohol-use symptoms were associated with T3 PTSD symptoms ($p = .02$) and mental health treatment was also associated with T3 PTSD symptoms ($p = .007$). The moderated effect on T3 PTSD symptoms was still significant ($p = .003$) with these covariates included in the model and the moderated indirect effect also remained unchanged relative to the results reported in the main text: there was an indirect effect of T2 PTSD on T3 PTSD via dependent SLEs at lower levels of T2 PTSD symptoms.

³Given that not all T2 participants returned for the T3 assessment, we re-ran this moderated mediation model for dependent SLEs in Mplus and employed full information maximum likelihood estimation (i.e., direct ML) to address concerns regarding missing data at T3. Doing so yielded path coefficients that were virtually unchanged (within .01) from that reported in Figure 2 for the sample with complete T2 and T3 data.

⁴As with the moderated mediation model, we also re-ran this model with a number of potential covariates included as a predictor of T2 dependent SLEs. Specifically, we include age, sex, T2 alcohol-use disorder severity and T2 major depression symptom severity. None of these variables were associated with dependent SLEs in this model while CON ($p = .045$) and T2 PTSD symptoms ($p = .026$) remained significantly associated.

Discussion

Results of this study provide support for bidirectional and transactional relationships between PTSD and SLEs such that PTSD robustly predicted greater dependent, but not independent, SLEs; in contrast to our first hypothesis, but consistent with our second, the effects of dependent SLEs on subsequent residualized increases in PTSD symptoms were evident only among those with relatively less severe initial PTSD. These results suggest that individuals with lower initial PTSD severity may be more sensitive to the effects of subsequent life stressors, whereas those with higher initial PTSD severity may exhibit chronically elevated symptoms regardless of intervening stressors. Alternatively, the lack of an association between SLEs and subsequent PTSD symptoms for the high baseline PTSD severity group could simply reflect a measurement/ceiling effect for the group. However, this interpretation is unlikely, as even the highest CAPS score of 110 in the high baseline PTSD severity group at T2 was well below the maximum possible CAPS score of 136. Nevertheless, PTSD appears to be a risk factor for stress generation which is, in turn, a mechanism for symptom maintenance and exacerbation for a subset of the PTSD population. Results for PTSD are generally consistent with research suggesting transactional relationships between depression (Zvolensky et al., 2015) and stress-generation and extend such work, for the first time, to PTSD.

Longitudinal models of PTSD symptom course typically include subgroups or class trajectories defined by delayed-onset (Andersen et al., 2014; Maercker et al., 2013; Pietrzak et al., 2014) and/or increasing severity (Andersen et al., 2014; Lowe et al., 2014; Osofsky et al., 2015; Pietrzak et al., 2014). Prior research suggests that the number of post-trauma life stressors predicts membership in these trajectory groups (Lowe et al., 2014). Our findings lend support to the idea that these worsening trajectories may be partially driven by new SLEs, and further suggest that these events are driven in part by posttraumatic psychopathology. Additionally, analyses showed that this association was significant for behaviorally dependent, but not independent, SLEs. This suggests that we are observing fundamental stress-generative effects of PTSD, as results are unlikely to be explained by the effects of environmental factors outside of an individual's influence (such as neighborhood crime level or familial health issues).

Independent of T2 PTSD severity, individuals with low levels of constraint (i.e. high impulsivity) showed an increased number of dependent SLEs. Though Lusk et al. and Sadeh et al. have previously shown associations between poor constraint and risky behavior on new onset adverse life events (Lusk et al., 2017; Sadeh et al., 2015), these results are unique in that we were able to disentangle dependent versus independent SLEs, and show that disconstraint predicts only dependent SLEs. Additionally, we reduced potential biases in participants' self-reports of life stressors (which may be influenced by current psychopathology) by using objective ratings of the nature of the events.

We have previously suggested that a subset of individuals with PTSD display an externalizing phenotype defined by high levels of anger, aggression, alienation (e.g., NEM) and low levels of constraint (Miller et al., 2003; Miller et al., 2004; Miller & Resick, 2007; Wolf et al., 2012). This subtype is marked by recklessness, disregard for social mores, rules,

and laws, and comorbid psychopathology in the realm of substance use and cluster B personality disorders (Miller & Resick, 2007; Wolf et al., 2012). Given this, and our findings that both PTSD symptoms and low CON contribute independently to stress generation in the domain of dependent SLEs, we suspect that individuals with the externalizing subtype of PTSD and/or externalizing posttraumatic psychopathology may be more prone to the stress-generative effects of PTSD. Notably, only low constraint, and not negative emotionality, predicted an increasing number of dependent SLEs, suggesting that the stress-generative quality of PTSD may be a result of increased impulsivity and less related to the negative affect temperament component of externalizing.

Together, these results highlight the importance of focusing on the prevention of new-onset SLEs among individuals with PTSD. Some interventions, such as assertiveness and risk-recognition trainings, have shown efficacy in preventing revictimization from traumas like sexual assault (Hill et al., 2011; Marx et al., 2001) and domestic abuse (Mears, 2003). Other interventions focused on managing daily stressors, such as anger management, present centered therapy, and Dialectical Behavior Therapy (Linehan, 1993), may also prove useful in reducing exposure to common SLEs. Given our effects for impulsivity, clinicians should also consider using behavioral indicators of low constraint to identify individuals at risk for stress-generative behaviors. When devising treatment plans for these individuals, clinicians may want to include training in behavioral control (e.g., problem solving skills, Dialectical Behavior Therapy) to try to reduce impulsive behavior that might lead to new SLEs and increased PTSD symptom severity.

In contrast to existing literature, we did not find a stress-generative role of T2 depressive symptoms in our sample: T2 depressive symptoms did not predict new independent or dependent SLEs. This may be due to our sample being enriched for PTSD, as screening positive for PTSD was an inclusion criterion for the study; we may have better ability to detect correlates of PTSD because there is greater representation and variability of PTSD symptom severity by virtue of our study inclusion criteria, which required a positive screen for PTSD. We also did not observe associations between high NEM or low PEM and SLEs, two temperament traits commonly linked to depression (Clark et al., 1994; Kendall et al., 2015; Naragon-Gainey & Watson, 2014). Additional research is necessary to further evaluate potential differential patterns of association between PTSD, depression, and SLEs.

These results should be considered in light of study limitations. Our sample size was modest and only a subset of the original participants was retained in the study through the final follow-up. This raises questions about the representativeness of the final sample to the broader veteran PTSD population and may have resulted in reduced statistical power. As well, results may not generalize to other populations that were under-represented in this study, including women, civilians, or veterans from more recent war eras. We also did not account for whether participants were receiving treatment, such as psychotherapy or medication use, which likely affects symptom course. Results require replication in larger and more heterogeneous samples. Additionally, T3 PTSD and SLEs were assessed at the same time, so it is possible that retrospective reports of SLEs were biased by T3 PTSD severity. However, our use of the Stressful Life Events interview and clinician-determined ratings reduced potential biases. Notably, the percentage of subjects meeting full criteria for

PTSD diagnosis dropped from 60.3% ($n = 70$) at T2 to 34.5% ($n = 39$) at T3. This decrease was likely an artifact of arbitrary thresholds that define presence/absence of the disorder given that the majority of veterans who no longer met PTSD diagnostic criteria at T3 just missed the threshold for the diagnosis by 1 or 2 symptoms. This is one of the reasons we analyzed PTSD symptom severity, rather than PTSD diagnosis.

In this first ever prospective study of potential bidirectional associations between PTSD and objectively-rated independent and dependent SLEs, we found that PTSD symptoms were implicated in the generation of dependent SLEs, and, that for some individuals, stress generation is a contributor to increasing posttraumatic psychopathology. Additionally, we found that a temperament style marked by impulsivity and disinhibition independently contributed to stress generation. These findings advance our understanding of factors that influence PTSD symptom course and illustrate the need for interventions designed to improve behavioral control and reduce the occurrence of personally-dependent stressors.

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Figure 1.

Figure 1 shows the magnitude of the indirect effect (unstandardized) and the 95% confidence interval for the effect of PTSD on subsequent PTSD via dependent life events as a function of initial level of PTSD severity (e.g., the magnitude of the indirect moderated mediation effect). The figure shows that the indirect effect was greater at lower levels of initial PTSD severity. PTSD = posttraumatic stress disorder; SD = standard deviation.

Figure 2.

Figure 2 shows the moderated mediation path model for PTSD in which PTSD evidenced a direct association with subsequent PTSD and an indirect one via intervening dependent stressful life events, with this indirect effect dependent on the severity of T2 PTSD symptoms. The unstandardized beta (and standard error) are presented on top of each path and the standardized beta (calculated separately in Mplus) estimate is listed below each path. When the unstandardized path coefficient is significant, so is the standardized path coefficient. The interaction between T2 PTSD and dependent life events is depicted by the filled circle at the center of the figure and the effect of this interaction variable on the relationship between dependent life events and T3 PTSD is depicted on the path stemming from the filled circle. T2 = time 2; T3 = time 3; PTSD = posttraumatic stress disorder; DEP = dependent; SLE = stressful life events. ** $p < .01$. *** $p < .001$.

Table 1

Descriptive Statistics and Bivariate Associations.

	Descriptive Statistics		Bivariate Correlations			
	Mean	SD	PTSD T2	PTSD T3	Dep SLEs	Indep SLEs
Age	56.23	9.54	-.189*	-.141	-.186*	.025
sex (% male)	87.9%	-	-.118	-.086	.096	-.022
race (% white)	71.4%	-	.048	.017	.108	-.143
PTSD Sev T2	53.27	25.84	-	.728*	.254*	.038
PTSD Sev T3	42.48	24.64	-	-	.192*	.033
Dep SLEs	2.83	3.47	-	-	-	.144
Indep SLEs	5.08	4.70	-	-	-	-

Note: PTSD Sev = posttraumatic stress disorder severity; SD = standard deviation; Dep SLEs = dependent stressful life events; Indep SLEs = independent stressful life events.

* $p < .05$.

Table 2

Parameter Estimates from the Mediation Models of PTSD and Depression on Subsequent PTSD and Depression via Dependent and Independent Life Events.

	PTSD Model				Depression Model			
	B	SE	95% CI	p	B	SE	95% CI	p
IV→DV	.69	.06	.56,.81	.000	.50	.07	.36,.64	.000
IV→M _{dep}	.03	.01	.01,.06	.006	.08	.07	-.05,.22	.220
IV→M _{indep}	.01	.02	-.03,.04	.708	-.02	.09	-.19,.16	.849
M _{dep} →DV	.05	.48	-.91,1.00	.919	.26	.10	.05,.46	.013
M _{indep} →DV	.03	.36	-.68,.74	.928	.04	.08	-.12,.20	.617
IV→M _{dep} →DV	.00	.03	-.06,.04	.924	.02	.02	-.01,.09	.297
IV→M _{indep} →DV	.00	.01	-.01,.02	.975	-.00	.01	-.02,.01	.933

Note. All betas are unstandardized, as is provided by this statistical model. PTSD = posttraumatic stress disorder; SE = standard error; CI = confidence interval; IV = independent variable, which are PTSD severity and Depression severity at Time 2, respectively; DV = dependent variable, which are PTSD severity and Depression severity at Time 3, respectively; M = Mediator; Dep = dependent stressful life events; Indep = independent stressful life events.

Table 3

Parameter Estimates from the Moderated Mediation Models of PTSD and Depression on Subsequent PTSD and Depression via Dependent and Independent Life Events.

	PTSD Models				Depression Models			
	B	SE	95% CI	p	B	SE	95% CI	p
IV→DV	.84	.08	.68,.99	.000	.48	.09	.31,.66	.000
IV→M _{dep}	.03	.01	.01,.06	.006	.08	.07	-.05,.22	.220
IV→M _{indep}	.01	.02	-.03,.04	.708	-.02	.09	-.19,.16	.849
M _{dep} →DV	2.63	.97	.70,4.55	.008	.23	.17	-.10,.56	.171
M _{indep} →DV	.64	.88	-1.11,2.38	.471	-.02	.13	-.27,.23	.871
IVxM _{dep} →DV	-.04	.01	-.07,-.01	.003	.00	.02	-.03,.04	.822
IVxM _{indep} →DV	-.01	.01	-.04,.02	.458	.01	.02	-.02,.05	.403
IVxM _{dep} →M _{dep} →DV	-.002	.0009	-.004,.0002	-	.0003	.002	-.003,.006	-
IVxM _{indep} →M _{indep} →DV	-.0001	.0003	-.001,.0003	-	-.0002	.002	-.007,.002	-

Note. All betas are unstandardized, as is provided by this statistical model. Significant beta coefficients are reported to two decimal points or the first non-zero integer as necessary for interpretation. Moderated paths are denoted by an X between the two variables that form the interaction term. PTSD = posttraumatic stress disorder; SE = standard error; CI = confidence interval; IV = independent variable, which are PTSD severity and Depression severity at Time 2, respectively; DV = dependent variable, which are PTSD severity and Depression severity at Time 3, respectively; M = Mediator; Dep = dependent stressful life events; Indep = independent stressful life event

Table 4

Regression of PTSD and Temperament on Independent and Dependent SLEs

	Independent SLEs			Dependent SLEs		
	B	SE	β	B	SE	p
PTSD T2	-.003	.019	-.019	.859	.039	.007
Positive Emotionality T2	.013	.028	.046	.655	.026	.233
Negative Emotionality T2	.019	.026	.079	.459	-.005	.818
Constraint T2	-.044	.034	-.123	.220	-.061	.020

Note. B = unstandardized beta; SE = standard error; β = standardized beta; PTSD = posttraumatic stress disorder