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Repeated Trauma Exposure Does Not Impair Distress Reduction During Imaginal Exposure for Posttraumatic Stress Disorder

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Abstract

Background—Based on experimental research on threat extinction, individuals exposed to repeated traumatic events may have impaired outcome in exposure therapy compared to those who have experienced a single trauma.^[1] This study examined whether repeated trauma exposure predicts smaller changes in self-reported distress during imaginal exposure and worse outcome for patients with PTSD.

Methods—Adults ($N = 116$) with chronic PTSD received up to 10 sessions of PE. Trauma exposure was assessed via interview and number of traumatic events were summed for each participant. To examine reductions in distress during treatment, mean and peak values of distress during imaginal exposure were calculated for the first imaginal session (initial distress activation) and subsequent sessions (between-session change in distress). Change in PTSD symptoms from pre- to post-treatment and follow-up provided an additional index of outcome.

Results—In-session distress during imaginal exposure decreased over the course of treatment. PTSD symptoms also decreased over treatment, with gains being maintained through follow-up. Repeated trauma exposure was not significantly correlated with initial distress activation.

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Additionally, linear mixed-model analyses showed no significant association between repeated trauma exposure and between-session change in distress or PTSD symptoms.

Conclusions—Contrary to recent speculation, repeated trauma exposure did not predict less change in self-reported distress during imaginal exposure or worse PTSD outcomes. The bench-to-bedside linkage of threat extinction to exposure therapy is discussed, noting strengths and weaknesses. Patients with repeated trauma exposure show reductions in distress with exposure treatment and benefit from PE as much as patients with single-exposure trauma histories.

Keywords

trauma; extinction; posttraumatic stress disorder; treatment; Prolonged Exposure

Grounded in principles of extinction learning, prolonged exposure (PE) therapy has consistently been found to be efficacious in the treatment of posttraumatic stress disorder (PTSD).^[e.g., 2, 3] Extinction refers to the process of repeated presentation of a conditioned stimuli (CS) without the presence of the unconditioned stimuli (US), whereby fear, defined across indices of behavior (e.g., freezing), physiology (e.g., skin conductance response), and/or subjective feelings, decreases over time. Impaired extinction processes have been implicated in the development of chronic PTSD^[e.g., 4–6] and facilitating extinction learning is hypothesized to be a critical component of exposure-based PTSD treatment.^[e.g., 7]

From the rodent literature, chronic stress impairs threat extinction^[8, 9] and threat extinction recall.^[10] In rodent threat extinction paradigms, chronic stress is manipulated by exposing a group of rats to repeated incidents of prolonged stress (e.g., forced swim or chronic restraint) over multiple days or weeks and then comparing the stressed group to a control group on freezing responses within a Pavlovian fear conditioning task, threat extinction, and threat extinction recall. Although simplified approximations of the effects repeated trauma exposure might have on exposure-based treatments, these results have led to the conjecture that multiply traumatized patients with PTSD may benefit less from exposure-based therapies, such as prolonged exposure (PE), than patients exposed to a single, discrete traumatic event.

Compared to those with single incident trauma exposure, individuals who have experienced multiple traumatic events show decreased startle responding, but similar levels of self-reported aversion, to idiographic trauma scripts.^[11] According to Lang and McTeague,^[1] this blunted fear responding to trauma memories among those with a history of repeated trauma exposure may indicate deficient recruitment of the amygdala in the presence of fear stimuli. Such hyporeactivity may interfere with emotional engagement during extinction and reduce the corrective learning that extinction training typically affords, resulting in decreased efficacy of exposure therapy for individuals with repeated trauma exposure.^[1] Yet, using an experimental, threat extinction paradigm in a non-clinical sample, Norrholm and colleagues^[12] found that fear-potentiated startle to conditioned stimuli, specifically colored shapes, during early threat extinction was not strongly associated with degree of trauma exposure. Further, multiple studies have found strong response rates to PE within samples reporting high levels of prior trauma exposure.^[13, 14] Thus, it remains unclear whether

greater prior trauma exposure in individuals with PTSD alters responding during exposure therapy or PTSD treatment outcomes.

Empirically, this question can be examined in PTSD by comparing individuals with and without extensive trauma histories on clinical indices of responding during and following PE. Imaginal exposure is thought to mirror extinction processes; namely the trauma memory (CS) is repeatedly presented in the absence of the traumatic event (US) occurring. During imaginal exposure, the patient repeatedly recounts the memory, with very little input from the therapist. This memory is recounted multiple times in the session and is repeated over the course of multiple sessions. Unlike experimental threat extinction paradigms, imaginal exposure has strong verbal components (i.e., patient recounting the trauma out loud) and the subjective experience of the patient is discussed with the therapist at the end of each imaginal exposure session. Consistent with an extinction perspective of therapeutic learning, greater reduction of across session distress to one's trauma memory is associated with better outcomes in exposure-based PTSD treatment.^[15–18] That said, patients not reporting reliable decreases in distress to their trauma memory across sessions also make clinically significant changes.^[15] Relatedly, in a small sample of combat veterans with PTSD, treatment responders and non-responders both showed diminished responding during trauma-relevant virtual scenarios, as measured via fear-potentiated startle at pre-, mid, and post-treatment.^[19] These latter findings suggest that across-session distress reduction is unlikely to be the sole mechanism of change in exposure-based therapies. Indeed, exposure therapy involves cognitive changes in harm expectancies,^[20] and alterations in negative cognitions have been found to predict reductions in PTSD symptoms.^[21,22] The dialectic, according to LeDoux^[23], is that exposure therapy involves both extinction of implicit, trauma-related memories and changes in maladaptive beliefs that result from more explicit, or conscious, memory processes.

No study to date has examined Lang and McTeague's^[1] hypothesis that repeated trauma exposure predicts worse distress reduction and worse PTSD outcomes for exposure-based treatments such as PE. In a PTSD treatment-seeking sample of individuals characterized by a range of discrete (i.e., single incident) to extensive (i.e., multiple incident) trauma histories, we examined the extent to which repeated trauma exposure predicts both in-session distress ratings during imaginal exposure across sessions of PE and changes in PTSD symptoms across pre-, post-treatment, and follow-up. Although self-report ratings of distress do not capture fear responding in the same way as behavioral or psychophysiological measures of arousal, they do represent the individual's perception of distress. Studies that look at reductions in self-reported distress as a potential indicator of extinction have high clinical utility given that they are the most common indicator of responding used in clinical settings. If, as Lang and McTeague^[1] suggest, more prior traumatic events is associated with hyporeactivity to fear stimuli, repeated trauma exposure should also be associated with lower initial self-reported distress at the first imaginal exposure session and a shallower slope for decreases in self-reported distress across imaginal exposure sessions. If individuals who have extensive trauma histories are less likely to benefit from PE, then higher prior trauma exposure should be associated with worse PTSD treatment outcomes. Thus, such findings would have theoretical, translational science, and therapeutic implications for those with repeated trauma exposure, potentially arguing

against the use of exposure for these individuals. If, however, the amount of prior trauma exposure is not strongly associated with initial self-reported distress, between-session distress reduction, or changes in PTSD severity over the course of treatment and follow-up, then prior trauma exposure is unlikely to be a contraindication for exposure therapy.

Materials and Methods

Participants

Participants were recruited from two large, metropolitan communities using a wide range of recruitment sources, including clinical referrals and community advertising. Eligible participants were between ages 18 and 65, English-speaking, and had a primary DSM-IV diagnosis of chronic PTSD related to trauma exposure that occurred at least twelve weeks prior to the initial evaluation. Exclusion criteria included: a current diagnosis of schizophrenia or delusional disorder; medically unstable bipolar disorder, depression with psychotic features, or depression requiring immediate psychiatric treatment; a diagnosis of alcohol or substance abuse within the previous three months; an ongoing intimate relationship with the perpetrator (in cases of sexual or physical assault); a change in the dose of psychiatric medication within the previous three months; an unwillingness to discontinue current antidepressant medication or psychotherapy; current use of sertraline; a previous, failed trial of either PE or sertraline; or a medical contraindication for taking sertraline, such as pregnancy or lactation.

The sample ($N = 116$) was comprised of patients randomly assigned to PE or treatment choice, and chose PE, as part of a larger clinical trial that utilized a doubly randomized preference design to compare PE and sertraline.^[24] The sample was primarily Caucasian (54.3%) and female (75.9%). Mean age was 36.6 years ($SD = 11.3$). Participants sought PTSD treatment for the following types of index traumas, defined as the most currently distressing event: sexual assault (30.2%), non-sexual assault (20.7%), childhood sexual abuse (19.0%), an accident, such as a motor vehicle crash or a natural disaster, (13.8%), childhood non-sexual abuse (7.8%), having a loved one die or be exposed to violence (6.0%), or combat or war (2.6%). The mean time since index trauma was 12.02 years ($SD = 12.25$, range 0.24 – 45.70 years) and the mean number of distinct types of non-index traumatic events participants reported experiencing was 3.72 ($SD = 2.31$, range 0 – 11).

Measures

PTSD Symptom Scale-Interview Version (PSS-I).^[25]—The PSS-I is a 17-item, semi-structured interview that assesses DSM-IV PTSD symptoms using a 0 (*not at all*) to 3 (*5 or more times per week/very much*) scale, rated for the two previous weeks. Reliability was assessed in the current study by rerating 10% of the cases. Inter-rater reliability was high for PTSD severity ($ICC = .98$).

Structured Clinical Interview for DSM-IV Axis I Disorders with Psychotic Screen (SCID-IV).^[26]—The SCID-IV is a semi-structured clinical interview that was used to assess for exclusion criteria and to assess for comorbid disorders. Ten percent of the cases from the current study were rerated for diagnostic reliability. There was good diagnostic

agreement for current anxiety disorders ($\kappa = 1.00$, $p_{pos} = 1.00$, $p_{neg} = 1.00$), major depressive disorder ($\kappa = .68$, $p_{pos} = .88$, $p_{neg} = .80$), substance abuse disorders ($p_{pos} = .00$, $p_{neg} = 1.00$), and other diagnoses ($p_{pos} = .00$, $p_{neg} = 1.00$). In the present sample, rates of current and lifetime Axis I disorders were 63.8% and 94.0%, respectively.

Trauma History—The number of *DSM-IV* Criterion A lifetime traumatic events experienced was assessed using a standardized trauma history interview.^[27] Participants were asked whether they had witnessed and/or experienced a number of potential Criterion A traumatic events, the number of times each of these events occurred, and the degree of fear, helplessness, and horror felt at the time on a 3 point scale (0 = *not at all*, 1 = *somewhat*, 2 = *very much*). Queried events were as follows: natural disasters, serious accidents or injuries, sudden, life-threatening illness, unexpected death or murder of a loved one, military combat, sexual or physical assault, and childhood sexual or physical abuse. Total number of Criterion A events was calculated by summing the total reports for each event endorsed. Individuals who did not report any traumatic events in addition to the index trauma had a score of 0 on this measure. The distribution of counts of Criterion A events was negatively skewed, with 24.7% of patients indicating that they had experienced an event “too many times to count” or more than 100 times. In other words, roughly a quarter of the sample experienced some form of chronic abuse or violence, and thus, we transformed the raw counts of prior Criterion A events into ranks. The variable was coded such that “0” represented having only one prior criterion A event (i.e., the index trauma); individuals with the next highest number of criterion A events (i.e., the index trauma plus one additional trauma) were assigned the value “1”, and so forth, in ascending order of trauma exposure.

Subjective Units of Distress (SUDs).^[28]—SUDs is a state measure of how much anxiety or distress experienced in response to a specific situation rated on a 0 (*no discomfort at all, or complete relaxation*) to 100 (*extremely upset, the most you have been in your life*) scale. SUDs ratings were assessed every 5 min during imaginal exposure. Mean ratings, capturing overall distress throughout imaginal exposure, and peak ratings, capturing highest SUDs during imaginal exposure, for each session were utilized. SUDs ratings are commonly used as a state measure of fear activation and change in the exposure therapy literature^[29] and correspond reasonably with physiological indicators of fear.^[30–32] In a separate sub-study of patients in this sample ($n = 74$), the correlation between feeling “distress,” “scared,” or “afraid” and SUDs during recounting of the trauma narrative on the Positive and Negative Affective Scale^[33] was strong (respectively, $r = .68$, $r = .67$, $r = .62$, $ps < .001$); whereas, there were more moderate correlations with feeling “nervous” or “jittery” and SUDs ($r = .37$, $r = .31$, $ps < .05$), pointing to good discriminant validity.

Prolonged Exposure

PE was delivered in 10 weekly, 90–120-min sessions according to a manual that specified the structure and content of each session.^[34] Sessions included education, breathing retraining, *in vivo* exposure to avoided trauma-related reminders, imaginal exposure involving repeated revisiting of trauma memories during session, processing of the imaginal exposure examining trauma-related feelings and beliefs, and assignment and review of homework. PE was provided by Masters- or Doctoral-level clinicians who had some CBT

experience and were trained in delivering PE prior to treating patients on the trial. Weekly supervision included case discussion and videotape review. Trained raters reviewed 10% of videotapes. PE providers completed 90% of the essential components and no protocol violations were observed. PE sessions were also rated for therapist competence (e.g., engaged in interactive exchange with client) on a 3-point scale (1 = *Inadequate*, 3 = *Adequate or Better*). Overall PE therapist competence was very good ($M = 2.73$, $SD = .32$).

Imaginal exposure (IE)—IE involved repeated revisiting of the target trauma memory for 30–45 min during PE Sessions 3–10. Standardized instructions included asking patients to close their eyes, visualize the trauma as if it were happening again, and retell the narrative out loud in the present tense. Over time, focus shifted to the most distressing aspects of the trauma memory and potentially to other distinct trauma memories. Therapists elicited SUDs ratings prior to the beginning of IE, every 5 min during IE, and at the end of IE.

Procedures

Independent evaluators obtained informed consent and then completed diagnostic interviews. Next, potentially eligible patients completed a physical exam and a drug and pregnancy screen. Eligible patients were then randomized as part of the larger trial. Those in PE received up to 10 sessions of PE and were then reassessed by independent evaluators on the PSS-I at post-treatment and at 3- and 6-month follow-up.

Analytic Plan

We used bivariate correlations to examine the relationship between initial distress activation, as measured via mean and peak SUDs during Session 3, and repeated trauma exposure. We used linear mixed models to evaluate the association between prior trauma exposure and the intercept and slope of SUDs reported during in-session IE, which took place during Sessions 3–10. Mean and peak SUDs were examined in separate analyses. We also used linear mixed models to examine the association between prior trauma exposure and change in PTSD symptoms from pre- to post-treatment and 3- and 6-month follow-up. Following recommendations for longitudinal mixed-model analyses,^[35] a model with session as a fixed effect in an unconditional growth model having random intercepts and fixed slopes was the best fit for examining reductions in distress across IE sessions. For the data examining change in PTSD symptoms over time, a model with time point (pre, post, 3- and 6-month follow-up) as a fixed effect in an unconditional growth model having random intercepts and fixed slopes was the best fit. All models were initially examined under the assumptions of unstructured, compound symmetry, and first-order-autoregressive variance-covariance matrices. Variance-covariance matrices provided the best fit in every model and are reported here. Statistical analyses were conducted using SPSS Statistics version 19.0 (SPSS Inc., Chicago). Analyses were intent-to-treat, using restricted maximum likelihood for handling missing data. Alpha was set at $\alpha = .05$.

We estimated power for the linear mixed models^[36], based on a minimum of four data points for predicting change in PTSD symptoms over time, with Type-I error level for a two-sided test set at .05 and power at .80. Given the observed average correlation between PTSD

at all time points ($r = .47$) and the observed dropout rate of 19% from pre- to post-treatment, we were powered to detect at least a small to moderate effect ($d = 0.3$).

Results

Table 1 presents the means, standard deviations, and range of predictor and outcome variables for the total sample.

Repeated Trauma History and Initial Distress Activation

Table 2 shows a weak relationship between prior trauma exposure and initial distress activation, as measured by mean and peak SUDS at Session 3 during the first imaginal exposure.

Repeated Trauma History and Across Session Distress Reduction

We next looked at prior trauma exposure as a predictor of reductions in distress across sessions of imaginal exposure.¹ As can be seen in Table 3, there was an effect of Session in both models, such that both mean and peak SUDs declined across sessions. Notably, neither Trauma Amount nor the Trauma Amount $\times$ Session effect was statistically significant for either mean or peak SUDs models, suggesting that higher rank-ordered amount of prior trauma exposure was not reliably associated with between-subject variability in the slope of mean or peak SUDs across sessions.

Repeated Trauma History and PTSD Symptom Reduction

Table 4 presents the results of linear mixed-model analyses predicting change in PTSD symptoms from pre- to post-treatment and 3- and 6-month follow-up. Table 5 presents the pattern of change between each time-point (i.e., pre-treatment to post-treatment; post-treatment to follow-up).

There was an effect of Time such that PTSD significantly declined from pre- to post-treatment, with gains being maintained through six-month follow-up. Notably, neither Trauma Amount nor the Trauma Amount $\times$ Time interaction was significant, suggesting that higher prior trauma exposure was not reliably associated with change in PTSD symptoms over time.

Discussion

The present study is the first to examine whether cumulative trauma exposure in individuals with PTSD negatively affects initial and change in self-reported distress during imaginal exposure and later PTSD outcomes over the course of PE and follow-up. This is a highly relevant clinical question posed by Lang and McTeague,^[1] who theorized that individuals with multiple trauma exposures would show impaired extinction during exposure therapy. More specifically, we examined the relationship between the amount of prior trauma

¹We examined two additional exploratory models for both dependent variables: one in which quadratic growth terms were added, and one in which trauma amount was log-transformed to correct for positive skew. In both cases, the final model results were statistically indistinguishable from those presented here. We present the simpler, a priori models here. The exploratory results are available from the corresponding author upon request.

exposure and initial, self-reported distress, between-session changes in distress during imaginal exposure, and changes in PTSD symptom severity from pre- to post-treatment and 3- and 6-month follow-up. Amount of prior trauma was not related to initial distress, as measured via mean and peak distress during the first imaginal exposure session. Further, although overall mean and peak distress ratings decreased across sessions, there was no relationship between prior trauma exposure and between-session changes in either mean or peak distress during imaginal exposure. Moreover, repeated trauma exposure did not significantly predict PTSD outcomes at post-treatment, 3-, or 6-month follow-up. Thus, higher prior trauma exposure did not adversely alter imaginal exposure trajectories or outcomes following exposure therapy.

These results are consistent with the large body of data finding consistently strong response rates to PE within samples that report high levels of prior trauma exposure.^[13,14] Additionally, these findings extend the work of Norrholm and colleagues,^[12] who found a lack of a strong association between prior trauma exposure and experimental threat extinction of fear-potentiated startle, to processes that occur during PE (i.e., changes in self-reported distress and PTSD symptoms). Moreover, these results are consistent with findings indicating that pre-treatment factors (e.g., age, gender, trauma type, time since trauma, comorbidity) are not reliable predictors of PTSD treatment outcome.^[37–39]

However, in prior research, chronic stress impaired threat extinction in rodents;^[8–10] and, in humans, more extensive trauma histories predicted lower levels of fear potentiated startle during idiographic trauma scripts^[11]. The present results, though, call into question Lang and McTeague's^[1] related assertion that individuals with PTSD and extensive trauma histories may be unlikely to benefit from exposure therapy. Notably, in experimental threat extinction paradigms, the US is often an aversive stimulus such as a shock, loud noise, or air puff and indices of fear focus on freezing behavior, startle response, or skin conductance response. It is unclear how much these threat extinction paradigms elicit the emotion of fear rather than mild distress or annoyance. However, in clinical exposure procedures, self-reported experience of distress, even if correlated with subjective fear and physiological arousal,^[34,35] only provides one index of potential fear. Thus, additional indices such as skin conductance response would add confidence to the present findings and provide a more direct link to experimental threat extinction paradigms.

There are also notable differences between threat extinction paradigms and imaginal exposure therapy itself. Rodent studies of the effects of chronic stress on extinction typically utilize single rather than complex or compound conditioned stimuli that are not ecologically relevant or evolutionarily primed. Similarly, the hyporesponsivity found by McTeague and colleagues^[11] in those with cumulative trauma exposure may be a byproduct of their paradigm, which may have lacked the feature-rich stimulus, response, and meaning elements that are incorporated into imaginal exposure. Moreover, imaginal exposure involves multiple and prolonged, 30–45 min exposures to trauma memories, and thus likely evokes more distress than the four, 20-word trauma scripts that were presented once each in the study by McTeague and colleagues.^[11] That said, as discussed above, imaginal exposure includes verbal and cognitive elements not included in threat extinction paradigms, widening the gap between bench studies and clinical exposure therapy studies. Thus, future work on both

sides of the “bench-to-bedside” translation are needed not only to identify precise mechanisms at play but also to extend findings to the elaborated context of real world trauma exposure.

The current study is not without limitations. First, SUDs ratings are subjective and are susceptible to outside influences such as demand characteristics. Although SUDs ratings provide a convenient way of potentially assessing fear and are more commonly used in clinical practice than psychophysiological indices, they are a measure of subjective distress and are not specific to fear, though highly correlated with it. No behavioral or physiological indicators of threat extinction were included in this study. These additional indicators would add further confidence to the translation back to experimental threat extinction studies. Though reductions in freezing or physiological responses alone, without the subjective experience, also potentially fail to fully capture the emotion of fear.^[23] As LeDoux^[23] notes, reductions in physiological responses may not necessarily mean that the conscious emotion of fear has decreased, highlighting the value of patient self-report as a potential clinical indicator of extinction. We also did not utilize an experimental threat extinction paradigm to test extinction learning. A next important step is for experimental threat extinction paradigms to be conducted repeatedly throughout the course of exposure therapy, examining how changes in responding during imaginal exposure and changes in symptom reduction correspond with changes in experimental threat extinction. We chose to examine prior trauma history not as a dichotomous variable (cumulative vs. discrete), as McTeague and colleagues did,^[11] but instead as a continuous variable capturing increasing amount of trauma exposure. Our decision was based on arguments that more complex symptom profiles are thought to develop following sustained periods of repeated trauma exposure^[42] and that a continuous variable would provide more information than a dichotomous one. Thus, we sought to answer the question of whether those with increasingly more extensive trauma histories are at risk for not benefiting from exposure therapy. Lastly, we did not examine the relationship between PTSD symptoms and the number of trauma exposures in childhood and adulthood separately; and cumulative trauma in childhood, but not adulthood, has been associated with more severe PTSD and problems with emotion and interpersonal regulation.^[43] However, the majority (approximately 60%) of our sample reported a history of either childhood physical or sexual abuse, suggesting that our sample was not limited to those experiencing repeated trauma exposure only in adulthood.

Conclusion

In conclusion, the current findings suggest that the patterns of reduction in distress and PTSD symptoms of those with varying degrees of trauma exposure do not differ from one another within PE. These results have theoretical and translational science implications related to threat extinction and build on an emerging body of literature suggesting that PE can be successfully used with individuals who have experienced a large number of prior traumatic events. Clinicians and patients alike can use these findings to inform treatment planning and case conceptualization, knowing that exposure-based PTSD treatments are likely to be effective, even for patients who have extensive trauma histories.

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Table 1

Means and standard deviations for predictor and outcome variables

	<i>M</i>	<i>SD</i>	<i>Range</i>
Criterion A traumatic events ^{<i>I</i>}	13.00		5.00 – 42.00
Mean Initial Exposure Distress (Session 3 SUDs)	64.73	<i>18.81</i>	13.91–95.38
Mean Last Exposure Distress (Session 10 SUDs)	41.78	<i>21.07</i>	5.00–80.56
Peak Initial Exposure Distress (Session 3 SUDs)	81.85	<i>16.84</i>	30.00–100.00
Peak Last Exposure Distress (Session 10 SUDs)	52.91	<i>24.64</i>	5.00–100.00
Pre-treatment PTSD Severity (PSS-I)	29.41	<i>6.90</i>	15.00–46.00
Post-treatment PTSD Severity (PSS-I)	10.49	<i>9.41</i>	0–37.00
3-month Follow-up PTSD Severity (PSS-I)	8.17	<i>8.82</i>	0–40.00
6-month Follow-up PTSD Severity (PSS-I)	8.36	<i>9.20</i>	0–36.00

Note. Overall means and standard deviations for the full sample are presented.

^{*I*} Median and interquartile range are presented for this variable, since it is a rank ordered variable.

Table 2

Bivariate Correlations for the Sample.

Variable	1.	2.	3.	4.	5.	6.	7.	8.
1. Criterion A Traumatic Events ¹	-							
2. Mean Initial Exposure Distress (Session 3 SUDs)	.08	-						
3. Mean Last Exposure Distress (Session 10 SUDs)	.01	.22	-					
4. Peak Initial Exposure Distress (Session 3 SUDs)	.02	.88*	.18	-				
5. Peak Last Exposure Distress (Session 10 SUDs)	.02	.12	.95*	.09	-			
6. Pre-treatment PTSD Severity (PSS-1)	.05	.20	-.13	.10	-.13	-		
7. Post-treatment PTSD Severity (PSS-1)	-.09	-.00	.31*	-.02	.27*	.25*	-	
8. 3-month Follow-up PTSD Severity (PSS-1)	-.10	.03	.29*	-.03	.27*	.15	.69*	-
9. 6-month Follow-up PTSD Severity (PSS-1)	-.12	.04	.19	-.00	.19	.21	.68*	.84*

Note.

* $p < .05$;

PSS-1: PTSD Symptom Scale Interview

Table 3

Fixed Effects Estimates and Variance-Covariance Estimates for Models of the Predictors of Mean and Peak SUDs from Session 3–10.

	Mean SUDs		Peak SUDs	
Fixed Effects				
Parameter	Estimate	SE	Estimate	SE
Intercept	63.89 [*]	2.48	81.28 [*]	2.45
Session	−2.89 [*]	0.31	−3.92 [*]	0.36
Trauma Amount	−0.01	0.07	−0.04	0.07
Trauma Amount × Session	−0.004	0.01	−0.002	0.01
Random Parameters				
Residual	169.07 [*]	10.41	223.21 [*]	9.24
Intercept (variance)	236.83 [*]	39.43	206.04 [*]	35.92

Note.

* $p < .05$;

SUDs = Subjective Units of Distress; SE = Standard Error.

Table 4

Fixed Effects Estimates and Variance-Covariance Estimates of the Predictors of PTSD Symptom Severity Across Pre-treatment, Post-treatment, 3-month, and 6-month Follow-up.

PSS-I		
Fixed Effects		
Parameter	Estimate	SE
Intercept	32.01 *	1.56
Time	-7.46 *	0.58
Trauma Amount	-0.01	0.04
Trauma Amount $\times$ Time	-0.02	0.02
Random Parameters		
Residual	76.00 *	6.93
Intercept (variance)	20.74 *	6.61

Note.

* $p < .01$;

PSS-I = PTSD Symptom Scale Interview; SE = Standard Error.

Table 5

Fixed Effects Estimates and Variance-Covariance Estimates of the Predictors of PTSD from Pre-treatment to Post-treatment, Post-treatment to 3-month Follow-up, and 3-month to 6-month Follow-up.

PSS-I						
Fixed Effects						
Parameter	Pre- to Post-Tx	Post-Tx to 3-Mo	3-Mo to 6-Mo	Estimate	SE	SE
Intercept	Estimate 47.70 *	Estimate 13.02 *	Estimate 8.62 *	Estimate 8.62 *	SE 3.30	SE 3.30
Time	Estimate -18.73 *	Estimate -1.37	Estimate 1.07	Estimate -1.37	SE .86	SE .86
Trauma Amount	Estimate .01	Estimate -.04	Estimate .08	Estimate -.05	SE .09	SE .09
Trauma Amount × Time	Estimate .00	Estimate .02	Estimate .03	Estimate .03	SE .02	SE .02
Random Parameters						
Residual	Estimate 49.59 *	Estimate 22.43 *	Estimate 4.04	Estimate 13.25	SE 2.33	SE 2.33
Intercept (variance)	Estimate 16.19 *	Estimate 6.87	Estimate 70.93 *	Estimate 13.41	SE 63.27	SE 11.57

Note.

* $p < .01$;

PSS-I = Posttraumatic Symptom Scale Interview; Tx = Treatment; Mo = Month SE = Standard Error