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Investigation of optimal dose of early intervention to prevent posttraumatic stress disorder: A multiarm randomized trial of one and three sessions of modified prolonged exposure

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Abstract

Background: Posttraumatic stress disorder (PTSD) is linked to a specific event, providing the opportunity to intervene in the immediate aftermath of trauma to prevent the development of this disorder. A previous trial demonstrated that trauma survivors who received three sessions of modified prolonged exposure therapy demonstrated decreased PTSD and depression prospectively compared to assessment only. The present study investigated the optimal dosing of this early intervention to test one versus three sessions of exposure therapy in the immediate aftermath of trauma.

Methods: Participants (n = 95) recruited from a Level 1 Trauma Center were randomly assigned in a 1.5:1.5:1 ratio in a parallel-group design to the three conditions: one-session exposure therapy,

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CONFLICT OF INTERESTS

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

three-session exposure therapy, and assessment only. Follow-up assessments were conducted by study assessors blind to study condition.

Results: Mixed-effects model results found no significant differences in PTSD or depression symptoms between the control condition and those who received one or three exposure therapy sessions across 1–12-month follow-up assessment. Results indicate that the intervention did not interfere with natural recovery. Receiver operating characteristic curve analyses on the screening measure used for study inclusion (Predicting PTSD Questionnaire; PPQ) in the larger sample from which the treatment sample was drawn (n = 481) found that the PPQ was a poor predictor of likely PTSD at all follow-up time points (Area under the curve's = 0.55–0.62).

Conclusions: This likely impacted study results as many participants demonstrated natural recovery. Recommendations for future early intervention research are reviewed, including strategies to identify more accurately those at risk for PTSD and oversampling more severe trauma types.

Keywords

early intervention; prolonged exposure; PTSD; secondary prevention

1 | INTRODUCTION

A majority of people will experience a traumatic event, with a 68.6% rate of lifetime trauma exposure in the United States (Goldstein et al., 2016). Fortunately, most people naturally recover after a traumatic event without intervention, but a significant minority will go on to develop posttraumatic stress disorder (PTSD; Goldstein et al., 2016). Unlike other psychiatric disorders, PTSD can be directly linked to a causal event and cannot be diagnosed until 1 month following the event, providing an opportunity for early identification and intervention for those at risk.

Brief cognitive-behavioral therapy (CBT) within 2–5 weeks of a traumatic event has been shown to be effective in reducing PTSD in recent motor/industrial accident and assault survivors (Bryant, Harvey, Dang, Sackville, & Basten, 1998; Foa, Hearst-Ikeda, & Perry, 1995; Foa, Zoellner, & Feeny, 2006). Recent meta-analytic findings suggest that multisession interventions delivered within 3 months of trauma are more effective than treatment as usual for reducing PTSD, but no significant differences were found compared with usual care at 7–12 months (Roberts et al., 2019). Authors note routine use of early intervention is not supported given many will demonstrate natural recovery and suggest that early intervention should target higher-risk individuals. Psychological debriefing (PD) involved a single group session to everyone exposed to trauma; results indicate it may interfere with natural recovery (Rose, Bisson, Churchill, & Wessely, 2002). Accurate screening to identify those at risk for PTSD would help target those most in need. Numerous studies have identified risk factors that are linked to subsequent PTSD such as previous trauma history (King, King, Foy, & Gudanpwski, 1996) subjective severity of the trauma (Andrews, Brewin, Rose, & Kirk, 2000), peritraumatic dissociation (Breh & Seidler, 2007), childhood history of trauma (Yehuda, Halligan, & Grossman, 2001), and family history of

psychiatric disorders (Inslicht et al., 2010). However, a widely accepted risk assessment tool is not yet available.

Exposure-based therapy may be an effective early intervention for PTSD. As noted previously, CBT therapy that involves exposure techniques initiated 2–5 weeks following trauma has been shown to be effective in reducing PTSD in recent trauma survivors (Bryant et al., 1998; Foa et al., 1995; Foa et al., 2006). As PTSD can be conceptualized as a failure of fear extinction after trauma exposure (Rothbaum & Davis, 2003), therapeutic assistance in achieving extinction may assist in recovery for those at risk. Translational research suggests that extinction training immediately after fear learning may be more effective than delayed extinction training. Specifically, rats who underwent a fear conditioning and subsequent extinction training showed greater reductions of fear if extinction occurred 10 min after fear learning rather than the more typical 72 hr later (Myers, Ressler, & Davis, 2006). This result has been replicated in humans, showing lower fear-potentiated startle when extinction training occurred 10 min rather than 72 hr after fear learning (Norrholm et al., 2008).

Evidence indicates that PTSD may be prevented in the immediate aftermath of trauma with a modified form of prolonged exposure (PE) therapy (Foa, Hembree, & Rothbaum, 2007). In the early intervention protocol, 60-min PE sessions are delivered in the emergency department (ED) immediately following trauma exposure (Rothbaum et al., 2008). A randomized controlled trial demonstrated that trauma survivors who received three sessions demonstrated significantly decreased self-reported PTSD and depressive symptoms at 1 and 3 months posttrauma compared with those in the assessment-only condition and were half as likely to be diagnosed with PTSD at 3 months posttrauma (Rothbaum et al., 2012). This intervention seemed to mitigate genetic risk for PTSD (Rothbaum, 2014).

The optimal dosing of this early intervention is unknown. Although a three-session protocol has been shown to be effective, a one-session protocol would be more easily disseminated. The present study investigates the optimal dosing of early intervention for PTSD prevention, comparing one or three sessions of treatment to an assessment-only control condition for trauma survivors reporting multiple risk factors for PTSD. We hypothesize that patients who receive one or three sessions of modified PE after trauma exposure will demonstrate lower PTSD symptoms at 1, 3, 6, and 12 months posttrauma compared with the assessment only condition. In addition, given the importance of accurately identifying PTSD risk to target interventions, the present study investigates the accuracy of the screening measure (Predicting PTSD Questionnaire; PPQ).

2 | METHODS

2.1 | Study design and assessors

Participants were recruited from an urban Level I Trauma Center. Participants were randomly assigned in a 1.5:1.5:1 ratio in a parallel groups design to the three conditions: one-session exposure therapy, three-session exposure therapy, and assessment only. Participants were compensated $50 for each visit. Study procedures were approved by the Institutional Review Board and the research oversight committee. Assessments were

completed by blinded study assessors, all of whom had a minimum of bachelor's education. Assessors underwent a training process supervised by a licensed clinical psychologist, meeting 80% agreement on all training materials as well as on 10% of baseline and follow-up assessments conducted with a gold-standard rater (PhD level clinical psychologist). All assessments were conducted in person and data was stored in REDCap.

2.2 | Participants

Participants included 95 trauma center patients (50.5% female, ages 18–63 with an average of 35.1). Table 1 shows participant demographic information and Table S1 shows the study flow diagram detailing participant retention to follow-up.

2.3 | Procedure

2.3.1 | Screening and enrollment—With participants' assent, assessors conducted preliminary screening, verifying that participants met inclusion criteria (18–65, endorsement of DSM-IV [American Psychiatric Association, 2000] Criterion A trauma experienced within the past 24 hr; [PPQ] ≥ 3), with no history of mania, schizophrenia, or other psychoses; prominent suicidality; substance dependence in the past month; intoxication; or altered mental status. Recruitment began October 14, 2013 and ended December 6, 2016 due to study completion. The final follow-up assessment was completed on June 10, 2017.

2.3.2 | Pretreatment assessment—Assessors collected demographics, baseline symptoms, and trauma history. After this assessment, sealed envelopes containing computer-generated patient random assignments were opened to reveal the assigned condition. The study therapist immediately provided the session one intervention before discharge from the trauma center to those assigned to the exposure therapy conditions.

2.3.3 | Treatment—Six study therapists were trained in PE and this modified protocol and had a master's or doctoral degree in psychology. Intervention content was consistent with the previous trial (Rothbaum et al., 2012). Session one involved intervention introduction, imaginal exposure, processing, identifying behavioral exposures, normal reactions to trauma and identification of self-care, breathing retraining, and assignment of homework. Sessions 2 and 3 for those assigned to three sessions were conducted in an outpatient setting with all sessions distributed 1 week apart and involved a review of homework and then conducting imaginal exposure, processing, identification of behavioral exposures, and identification of self-care tasks. Participants assigned to the three-session condition remained with the same therapist for all three sessions. Consistent with the previous trial (Rothbaum et al., 2012), 20% of therapy sessions were rated for treatment integrity to ensure treatment adherence and competence. Using a scale of 1 (very poor) to 7 (excellent) mean therapist skill and adherence rating was rated as 5.80 (SD = 0.70). No negative side effects of the intervention were reported.

2.3.4 | Follow-up assessments—Participants were asked to return for in-person follow-up at 1, 3, 6, and 12 months by blinded assessors.

2.4 | Measures

2.4.1 | Predicting PTSD Questionnaire—The PPQ is a five-item measure designed to screen for the following PTSD risk factors: trauma history (yes–no), current trauma subjective severity (≥3 on a 0 [*not at all*] to 5 [*near death*] scale), current trauma peritraumatic dissociation (≥2 on a 0 [*not at all*] to 4 [*completely*] scale), childhood trauma exposure (yes–no), and family history of psychopathology (yes–no).

2.4.2 | Posttraumatic Stress Diagnostic Scale—The Posttraumatic Stress Diagnostic Scale (PDS) assesses prior trauma and PTSD diagnosis and severity during the past 2 weeks. In this study, the PDS was used to assess lifetime PTSD before the presenting traumatic event in the pretreatment assessment (Foa, Cashman, Jaycox, & Perry, 1997).

2.4.3 | Standardized Trauma Interview—The Standardized Trauma Interview is a clinician-administered interview that gathers information regarding demographic variables and index trauma characteristics (Foa & Rothbaum, 1998).

2.4.4 | PTSD Symptom Scale—The PTSD Symptom Scale (PSS) is a semistructured interview assessing PTSD diagnosis and severity and was used in this study to assess PTSD in response to the index trauma as a primary outcome measure (Foa, Riggs, Dancu, & Rothbaum, 1993).

2.4.5 | Beck Depression Inventory-II—The Beck Depression Inventory-II is a 21-item self-report inventory measuring depression severity administered as a secondary outcome measure for the study (Beck, Steer, Ball, & Ranieri, 1996).

2.5 | Data analytic plan

All analyses were conducted using R software (version 3.5.1). Comparisons between conditions were conducted within a multilevel model framework. Fixed effects were added for the intercept, which corresponded to the first assessment, time, and treatment condition. The treatment condition was captured with two variables representing the comparison between the one-session intervention and control and the three-session intervention and control. Interaction terms between each treatment condition variable and time were included to determine the extent that change over time varied across treatment conditions. Random effects were included to allow for interindividual variation across the intercept and slope. Time was scaled in months. Separate models were used for PTSD and depression symptoms. Missing data were handled with mixed-effects modeling. An intraclass correlation coefficient (ICC) was used to evaluate the effect of the therapist on outcomes. The ICC was consistently <.02, which suggests that therapist effects were minimal in this outcome analysis. The therapist was removed from the final model for parsimony.

The predictive power of the PPQ was evaluated in a larger sample from which the treatment outcome sample was drawn including the participants in the assessment only condition of the current study ($N = 481$). Treatment groups were excluded due to the potential effect intervention may have had on diagnostic rates. Receiver operating characteristic (ROC) curves were used to determine the cutoffs that maximized the sensitivity and specificity of

scores on the PPQ to predict likely PTSD diagnosis at 1-, 3-, 6-, and 12-month follow-ups. Likely PTSD was determined by using a diagnostic algorithm with scores on the PSS-I in which a score of 2 or greater on an item indicated the likely presence of that symptom. A likely diagnosis was then made according to DSM-IV-TR criteria if there was a score of 2 or greater on at least one reexperiencing symptom, three avoidance symptoms, and two hyperarousal symptoms.

3 | RESULTS

3.1 | Treatment outcomes

Comparisons of demographic and clinical variables at baseline were conducted. There were no significant differences in age ($F(1,93) = 0.11$; $p = .747$), time of baseline assessment since trauma ($F(1,74) = 1.40$; $p = .241$), baseline PTSD symptoms from a prior trauma ($F(1,88) = 0.24$; $p = .627$), baseline depression ($F(1,93) = 0.71$; $p = .402$), childhood trauma exposure ($F(1,73) = 0.62$; $p = .434$), or gender ($\chi^2(2) = 0.36$; $p = .836$) across the groups. Results indicate that random assignment created three comparable treatment conditions. A comparison of dropout rates suggested that there were no significant differences in dropout at 1 month ($\chi^2(2) = 2.02$; $p = .365$) or at any later assessment point (p's = .235–.977).

Mixed-effect models examined differences in the treatment F conditions relative to the control condition for PTSD and depression symptoms (Table 2). Descriptives for PTSD and depression measures at all time points are provided in Table 3. Comparisons across groups for PTSD symptoms as the primary outcome were prespecified. For PTSD symptoms, there was a significant effect for time, suggesting that symptoms declined from 1 through 12 months. PTSD symptoms at 1 month were not significantly different between those who received one treatment session or three treatment sessions relative to the control condition. There was no significant time × condition interaction, suggesting PTSD symptom improvement over time did not differ between the control condition and those who received one or three sessions. Effect sizes for comparisons at each assessment point were negligible to small range between the control and one-session groups (d's = .03–.35) and the control and three-session groups (d's = .07–.25; Table 4). Importantly, these results indicate that the intervention did not interfere with natural recovery.

Comparable results were obtained for depression symptoms. There were no significant time × condition interactions for the one- or three-session conditions, suggesting no group differences in the rate of depression symptom change across time. Effect size comparisons at each assessment point were in the negligible to small range between the control and one-session groups (d's = .12–.45) and the control and three-session groups (d's = .05–.35).

3.2 | PTSD prediction

The proportion of the sample that met criteria for likely PTSD declined across assessments such that at 1-month follow-up 28.4% of the sample had likely PTSD and at 12-month follow-up, 12.0% of the sample had likely PTSD. ROC curves suggested that the PPQ was a poor predictor of likely PTSD status at all timepoints (Table 5; area under the curve's = 0.51–0.55). A score of 4 was determined to have the best balance of sensitivity and

specificity at all timepoints. However, this score had values that were below existing guidelines for determining the adequate performance of a measure (Youngstrom, 2013). These findings suggest that the PPQ was not useful in predicting PTSD.

4 | DISCUSSION

The current study examined the effects of one or three sessions of modified PE in the ED immediately following trauma exposure compared with an assessment-only control on the development of PTSD symptoms over 12 months. Results identified a significant effect of time for PTSD symptoms, decreasing from 1-month through 12-month follow-up in all groups. The rate of PTSD symptom reduction over time did not differ between control and intervention conditions. Results were comparable for depression symptoms across groups over time. The results in the present study did not replicate the previously identified effects of the three-session modified PE protocol for reducing PTSD and depression symptoms following trauma exposure. These results indicate that the intervention did not interfere with the natural recovery from trauma as in PD (Mayou, Ehlers, & Hobbs, 2000).

In considering possible reasons for the null effect, timing and treatment fidelity were considered. The same treatment protocol was used in both studies; treatment fidelity procedures were identical across the studies with an average fidelity rating of 6.19 (SD = 0.83) and 5.80 (SD = 0.70), respectively, across the previous and current trial, indicating comparable treatment fidelity. Translational research suggests that the timing of extinction training is important (Myers et al., 2006; Norrholm et al., 2008). Comparison of timing indicates that the present trial administered early intervention closer to the time of trauma, with an average of 368.95 min since trauma compared to an average of 751.95 min for the intervention group in the previous trial, and assessment conducted at 665.55 min for the assessment group in the previous trial (Rothbaum et al., 2012). Future research should investigate early intervention timing; if administered during the window of cellular and synaptic consolidation processes for the initial trauma memory (Alberini & Kandel, 2015), glucocorticoid release and decay could interfere with the formation of the extinction memory if early intervention is administered when levels are still high. However, we believe data in the current trial suggest that low overall risk is the most likely explanation for the null effect. A comparison of the assessment only groups across the trials suggests that the previous trial had higher PTSD symptom severity across follow-up time points compared with the current trial, with a PSS average score of 24.54 at 1 month compared with 18.35 in the present trial, and 20.33 and 18.85 at 3-month, respectively. As such, we believe the present sample had lower PTSD risk overall and likely higher rates of natural recovery, contributing to difficulty identifying a significant effect of early intervention due to sample characteristics.

Sample characteristics such as trauma type may be related to the absence of a treatment effect identified. In the previous study (Rothbaum et al., 2012), the intervention was most effective in sexual assault survivors (Rothbaum et al., 2012). In that trial, 40.58% and 27.90% of participants enrolled in the intervention and assessment groups, respectively, presented following a sexual assault index trauma, compared with 9.5% in the present study (8.3% in one-session group, 14.3% in three-session group, and 4.2% in control group).

Comparatively, in the present study, 42.1% of participants were presenting following a motor vehicle collision. The higher effect size of treatment previously identified is notable given that rape is more commonly associated with PTSD compared with other traumatic events (e.g., Goldstein et al., 2016); as such the previous trial had more sexual assault survivors who may be more at risk for developing PTSD and, therefore, more responsive to this early intervention approach. Trauma type may be an especially important factor to consider when developing more accurate PTSD screening measures- a necessary aim for studies seeking to improve the effects of early interventions (Roberts et al., 2019).

A meta-analysis of 27 RCTs of multisession early psychological interventions within 3 months of the index trauma found that these interventions may be more effective than treatment as usual in reducing PTSD symptoms 3–6 months postintervention, but did not identify group differences at posttreatment or 7–12 months postintervention (Roberts et al., 2019). Authors interpreted results as suggesting there is limited evidence to support routine use of psychological intervention following traumatic events and note that many will demonstrate natural recovery, thus interventions likely would demonstrate greater impact when targeted at symptomatic individuals. The present results appear to echo this conclusion and demonstrate the importance of accurately screening for risk and targeting early intervention efforts to high-risk participants.

4.1 | Lessons learned for early intervention research

Given a majority of trauma survivors will follow a natural recovery trajectory, it is crucial to accurately assess PTSD risk to target prevention resources. ROC curve analyses suggest that unfortunately the PPQ used in the present study was a poor predictor of likely PTSD. Effective screening measures and/or oversampling of traumas with a greater likelihood of PTSD are important for future research. Fortunately, the empirical literature on prediction of PTSD has grown noticeably in recent years. A recent study using data accessible at the time of ED presentation, including electronic medical records and subjective reports, was effective at predicting future PTSD (Schultebraucks et al., under review). Results indicated a three-class model in which 56.23% of participants were classified as “resilient,” 32.89% as “recovery,” and 10.88% as “nonremitting,” further highlighting that natural recovery is the rule rather than the exception following a traumatic event.

A separate 9-item measure, the injured trauma survivor screen, demonstrates good sensitivity and strong specificity in predicting later PTSD diagnosis in trauma patients (Hunt et al., 2017). Furthermore, skin conductance response in the ED (Hinrichs et al., 2019) and peritraumatic nausea have also been shown to be prospective predictors of PTSD (Michopoulos et al., 2019). Another study found that automated electronic screening of risk factors in the medical record demonstrated good sensitivity and specificity for predicting PTSD symptoms (Russo, Katon, & Zatzick, 2013), demonstrating the potentially strong reach and low cost.

Limitations should be noted. An effect size of 0.38 was observed in prior work that found a significant difference between treatment and control conditions (Rothbaum et al., 2012); 110 individuals were needed per group to detect such an effect. As such the present study may have been underpowered to detect the true effect of this early intervention. In addition, the

effects observed in the current study were small and notably smaller than what was observed previously (Rothbaum et al., 2012). The lack of significance in the current study may have been attributed to limited power. Alternatively, the effects observed from the prior study (Rothbaum et al., 2012) and that observed in the current study may suggest that the effect of this early intervention on preventing PTSD may be small for trauma survivors overall. This study sample included a lower percentage of sexual assault survivors, whose previous research suggests benefit most from this early intervention (Rothbaum et al., 2012). In addition, the PPQ used for study inclusion and enrollment did not effectively identify those at elevated risk for PTSD. Using DSM-5 rather than DSM-IV criteria for PTSD could have influenced the results, although the lack of depression-related findings suggests otherwise, given the primary difference between DSM-IV and DSM-5 PTSD is the addition of negative alterations in cognitions and mood cluster. Importantly, the interventions did not appear to interfere with the natural recovery from trauma. Future research would benefit from testing this intervention in high-risk samples, including at a sexual assault crisis center or in combat zones.

The present study failed to identify a significant effect of this early intervention approach using either one or three sessions of modified PE. Potential reasons for not replicating findings in the previous trial (Rothbaum et al., 2012) include lower enrollment of sexual assault survivors and lack of predictive power of the screening measure used for study inclusion; future early intervention efforts should focus on oversampling at-risk trauma types and use of contemporary screening measures to identify those at-risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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TABLE 1

Demographic information and clinical characteristics

	1-Session (*n* = 36)	**3-Session (*n* = 35)**	**Assessment (*n* = 24)**
Male, *n* (%)	19 (52.8)	16 (45.7)	12 (50)
Female, *n* (%)	17 (47.2)	19 (54.3)	12 (50)
Age, mean (SD)	33.83 (12.99)	34.97 (13.91)	37.00 (14.20)
Ethnicity			
White, *n* (%)	7 (19.4)	3 (8.6)	2 (8.3)
Black, *n* (%)	25 (69.4)	30 (85.7)	19 (79.2)
Other, *n* (%)	4 (11.1)	1 (2.9%)	2 (8.3)
Marital status			
Single, *n* (%)	22 (61.2)	16 (45.7)	14 (58.3)
Married or cohabitating *n* (%)	7 (19.4)	17 (48.6)	7 (29.2)
Divorced or separated, *n* (%)	7 (19.4)	2 (5.7)	3 (12.5)
Minutes since presenting trauma	345.35 (223.63)	431.03 (358.42)	307.87 (231.33)
Trauma type			
Sexual assault, *n* (%)	3 (8.3)	5 (14.3)	1 (4.2)
Nonsexual assault, *n* (%)	4 (11.1)	2 (5.7)	4 (16.7)
Motor vehicle crash, *n* (%)	16 (44.4)	13 (37.1)	11 (45.8)
Motorcycle Crash, *n* (%)	1 (2.8)	3 (8.6)	1 (4.2)
Pedestrian vs. auto, *n* (%)	3 (8.3)	3 (8.6)	3 (12.5)
Gunshot wound, *n* (%)	2 (5.6)	3 (8.6)	0
Stabbing, *n* (%)	3 (8.3)	1 (2.9)	2 (8.3)
Fire/burn, *n* (%)	0	0	0
Industrial/home accident, *n* (%)	1 (2.8)	2 (5.7)	0
Fall, *n* (%)	1 (2.8)	2 (5.7)	2 (8.3)
Animal bite/attack, *n* (%)	0	1 (2.9)	0
Sports injury, *n* (%)	0	0	0
Bike accident/bike vs. auto, *n* (%)	2 (5.6)	0	0
Other, *n* (%)	0	0	0
Unknown, *n* (%)	0	0	0
Prior trauma exposure	24 (66.7)	27 (77.1)	15 (62.5)

	1-Session (*n* = 36)	3-Session (*n* = 35)	Assessment (*n* = 24)
Childhood trauma exposure	20 (55.6)	16 (45.7)	12 (50.0)

Abbreviations: BDI, Beck Depression Inventory; PDS, Posttraumatic Diagnostic Scale; *SD*, standard deviation.

TABLE 2

A multilevel model comparing groups from 1-month assessment to 12-month assessment

Outcome	Coefficient	*SE*	*p* Value
PTSD symptoms			
Intercept	18.58	3.31	<.001
Time	−2.19	0.98	.030
1-Session treatment	0.86	4.22	.839
3-Session treatment	−0.94	4.39	.831
1-Session treatment × time	0.15	1.27	.909
3-Session treatment × time	1.02	1.29	.433
Depression symptoms			
Intercept	18.43	2.26	<.001
Time	−0.48	0.26	0.066
1-Session treatment	0.421	2.91	0.885
3-Session treatment	−1.96	2.94	0.507
1-Session treatment × time	−0.24	0.33	0.468
3-Session treatment × time	0.09	0.33	.785

Abbreviations: PTSD, posttraumatic stress disorder; *SE*, standard error.

TABLE 3

The mean and standard deviation of PTSD and depression symptoms across timepoints

	Baseline Mean (*SD*)	1 Month Mean (*SD*)	3 Months Mean (*SD*)	6 Months Mean (*SD*)	12 Months Mean (*SD*)
Assessment					
PTSD	14.12 (10.91)	18.35 (13.93)	11.64 (10.34)	14.64 (13.73)	12.43 (9.52)
% Likely PTSD	-	52.9%	23.5%	21.4%	21.4%
Depression	19.00 (11.37)	18.88 (13.89)	13.84 (12.54)	17.21 (17.97)	15.00 (15.26)
1-Session					
PTSD	14.29 (11.36)	17.48 (11.12)	14.14 (9.54)	12.95 (10.72)	11.05 (11.24)
% Likely PTSD	-	34.5%	28.6%	23.8%	20.0%
Depression	21.11 (13.96)	14.10 (8.23)	15.14 (8.82)	11.29 (9.94)	9.80 (10.19)
3-Session					
PTSD	13.71 (11.55)	16.43 (13.25)	14.95 (12.62)	15.53 (16.34)	12.05 (12.69)
% Likely PTSD	-	34.8%	22.2%	23.5%	20.0%
Depression	17.40 (10.51)	14.57 (10.82)	14.50 (11.50)	13.94 (14.16)	12.05 (11.62)

Note: Depression was assessed via the Beck Depression Inventory at all timepoints. Posttraumatic Stress Diagnostic Scale was used to assess PTSD symptom severity before the presenting traumatic event in pretreatment assessment. PTSD Symptom Scale was used to assess PTSD in response to the index trauma at all follow-up assessment timepoints.

Abbreviations: PTSD, posttraumatic stress disorder; *SD*, standard deviation.

TABLE 4

Measures of effect size across conditions and within timepoints

PSS				
Between conditions				
	1 Month	**3 Months**	**6 Months**	**12 Months**
Assessment—1-Session	0.07	0.25	0.14	0.03
Assessment—3-Session	0.14	0.35	0.06	0.13
Across timepoints				
	1–3 Months	**3–6 Months**	**6–12 Months**	**1–12 Months**
Assessment	0.48	0.13	0.35	0.83
1-Session	0.47	0.12	0.09	0.76
3-Session	0.04	0.11	0.16	0.40
BDI				
Between conditions				
	1 Month	**3 Months**	**6 Months**	**12 Months**
Assessment—1-Session	0.45	0.12	0.43	0.41
Assessment—3-Session	0.35	0.05	0.20	0.22
Across timepoints				
	1–3 Months	**3–6 Months**	**6–12 Months**	**1–12 Months**
Assessment	0.28	0.13	0.33	0.30
1-Session	0.15	0.27	0.07	0.73
3-Session	0.05	0.32	0.10	0.46

Note: All effect sizes are Cohen's.

Abbreviations: BDI, Beck Depression Inventory; PSS, PTSD Symptom Scale.

TABLE 5

AUC, sensitivity, and specificity on the PPQ

Cut score	Sensitivity	Specificity	PPV	NPV
1-Month AUC = 0.51				
3	0.54	0.52	0.31	0.74
4	0.37	0.68	0.31	0.73
5	0.13	0.88	0.31	0.72
3-Month AUC = 0.54				
3	0.58	0.53	0.18	0.87
4	0.45	0.71	0.22	0.87
5	0.08	0.89	0.12	0.84
6-Month AUC = 0.51				
3	0.52	0.50	0.14	0.86
4	0.39	0.66	0.16	0.87
5	0.09	0.89	0.12	0.86
12-Month AUC = 0.55				
3	0.58	0.53	0.14	0.90
4	0.42	0.69	0.16	0.90
5	0.15	0.90	0.17	0.89

Note: The sample consists of those who did not receive treatment.

Abbreviations: AUC, area under the curve; NPV, negative predictive value; PPQ, Predicting PTSD Questionnaire; PPV, positive predictive value.