

Guest Reviewers

Trauma-Related Cognitions and Cognitive Emotion Regulation as Mediators of PTSD Change Among Treatment-Seeking Active-Duty Military Personnel With PTSD

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Trauma-related cognitions about the self and the world have been identified as a mediator of posttraumatic stress disorder (PTSD) change during prolonged exposure (PE) therapy. However, the extent to which negative cognitions mediate PTSD change in other PTSD treatments is unclear. In addition, previous studies have not tested alternate mediators of PTSD change during PE. In a sample of 216 treatment-seeking active-duty military personnel with PTSD, the present study examined the specificity of the negative cognition mediation effect in both PE and present-centered therapy (PCT). In addition, we examined another possible mediator, cognitive emotion regulation. Lagged mediational analyses indicated that negative cognitions about the self and world and the unhelpful cognitive emotion regulation strategy of *catastrophizing* each significantly mediated change in PTSD from baseline to 6-month follow-up. In a combined model, the mediating effect of catastrophizing was greater than negative cognitions about the world, and similar to negative cognitions about the self. Moderated mediation analyses revealed that the effect of catastrophizing was greater in PE than in PCT. Findings show that trauma-related cognitions and, to a greater degree, the emotion regulation strategy catastrophizing, both mediate PTSD change. Further research is needed to determine whether these mediating variables represent mechanisms of therapeutic change.

Keywords: PTSD; prolonged exposure therapy; present-centered therapy; trauma-related cognitions; mediation

PROLONGED EXPOSURE (PE) therapy is a well-established treatment for posttraumatic stress disorder (PTSD) that was derived from emotional processing theory (EPT; Foa & Cahill, 2001; Foa & Kozak, 1986). EPT provides a theoretical framework for the development of PTSD and the mechanisms involved in the maintenance and the natural and therapeutic recovery from PTSD. Similar to early schema theories of trauma sequelae (e.g., Epstein, 1991; Horowitz, 1976, 1986; Janoff-Bulman, 1992; McCann & Pearlman, 1990), EPT emphasizes the role of trauma-related cognitions about the self and the world in the development and maintenance of PTSD. According to EPT, natural or therapeutic recovery occurs when inaccurate, negative trauma-related cognitions are disconfirmed via thinking and talking about the trauma and/or approaching trauma reminders in daily life and realizing that the anticipated harm does not occur (e.g., “being attacked again” or “falling apart”; Cahill & Foa, 2007). In PE, modification of these cognitions is believed to result from helping patients confront trauma-related stimuli and situations (in vivo exposure) and revisit (imaginal

exposure) and process the traumatic memory in the absence of feared outcomes.

Several studies to date suggest that changes in negative trauma-related cognitions (e.g., “I’m incompetent”; “The world is completely dangerous”; “No one can be trusted”) mediate PTSD change during treatment, and are thus a precondition for change in PTSD symptom severity. For example, reductions in negative trauma-related beliefs temporally preceded decreases in PTSD symptoms in a trial of PE among women with assault-related PTSD (Zalta et al., 2014) and among civilians with comorbid mixed-trauma-related PTSD and alcohol dependence (McLean, Su, & Foa, 2015), whereas the reverse (i.e., PTSD change preceding negative cognition change) was not found. Similar results were found in a study of adolescents with sexual assault-related PTSD receiving either PE or supportive counseling: change in negative trauma-related cognitions mediated change in PTSD symptoms, whereas change in PTSD did not mediate change in cognitions (McLean, Yeh, Rosenfield, & Foa, 2015). These latter results suggest that changes in negative cognitions may mediate reductions in PTSD symptoms across PTSD treatments, rather than in PE specifically. However, more research is needed to examine the role of negative cognitions in other PTSD treatments, including non-trauma-focused interventions. In addition, the research to date has only included civilian samples.

Another important limitation of the existing literature is that studies to date have not included tests of alternative mediators. Although there is a strong theoretical justification for focusing on negative cognitions, there may be other, yet-to-be-identified factors that also significantly mediate PTSD treatment change. Testing alternate candidate mediators of change may reveal the specificity of trauma-related cognitions or call into question the uniqueness of their role as mediators of therapeutic recovery. Either way, we gain important information that may help refine PTSD theories and improve the efficacy/efficiency of treatment.

One possible alternate mediator of PTSD treatment change is cognitive emotion regulation, defined as the belief in one’s own ability to manage posttraumatic recovery demands (Cieslak, Benight, & Lehman, 2008). Negative cognitive emotion-regulation strategies, such as catastrophizing and rumination, and positive coping strategies, such as reappraisal, have been differentially associated with maladjustment in previous research. Cross-sectional studies have shown that lower cognitive emotion regulation is linked with higher PTSD symptom

severity (Cieslak et al., 2008; Held, Owens, Schumm, Chard, & Hansel, 2011; Pietrzak, Harpaz-Rotem, & Southwick, 2011). In addition, emotion regulation has been shown to mediate treatment change during cognitive-behavioral treatment for several disorders, including social anxiety disorder (Goldin et al., 2012) and trichotillomania (Woods, Wetterneck, & Flessner, 2006), but has not yet been examined as a mediator of PTSD treatment change.

The aim of the current study was to test two candidate mediators of PTSD change (negative cognitions and emotion regulation) during PE and present-centered therapy (PCT)—a non-trauma-focused, evidence-based treatment for PTSD—among active-duty military personnel seeking treatment for PTSD. This study used data from a large randomized controlled trial (RCT) comparing Massed-PE (10 sessions delivered over 2 weeks), Spaced-PE (10 sessions delivered over 8 weeks), and PCT (10 sessions delivered over 8 weeks) (Foa et al., 2018). The current study only included participants randomized to Spaced-PE and PCT, because these treatments were both delivered in the same 8-week time frame. Based on prior research, we hypothesized that: (1) change in negative cognitions will mediate change in PTSD for PE and PCT; (2) change in emotion regulation will mediate change in PTSD for PE and PCT; and (3) change in negative cognitions will mediate change in PTSD to a greater degree than change in emotion regulation for PE and PCT. Our exploratory aim was to examine whether treatment condition moderated any mediation effects.

Method

PARTICIPANTS

Participants were treatment-seeking active-duty military personnel ($N = 216$) who had returned from deployments to Afghanistan, Iraq, or nearby locations and were diagnosed with PTSD. The parent RCT ($N = 366$) included four study conditions; the current study included the 216 participants who were randomized to either 10 sessions of Spaced-PE (PE) or 10 sessions of PCT. Inclusion criteria were: (a) adult (ages 18–65) men and women active-duty military personnel, activated Reservist, or activated National Guard seeking outpatient treatment for PTSD; (b) diagnosis of PTSD according to criteria in the fourth edition, text revision of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000)*; (c) exposure to a *DSM-IV-TR* Criterion A traumatic event that was a combat-related event or high magnitude operational experience that occurred during a military deployment. Exclusion criteria were: (a) current bipolar disorder or psychotic disorder; (b) current alcohol

dependence; (c) moderate or severe traumatic brain injury; (d) current suicidal ideation severe enough to warrant immediate attention; and (e) other disorders severe enough to warrant immediate treatment.

MEASURES

PTSD Symptom Scale–Interview Version (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993)

The PSS-I is a 20-minute, 17-item, clinician-administered interview that evaluates *DSM-IV* PTSD symptoms in the past 2 weeks on frequency and severity. Items are rated on a 4-point scale (0 = *not at all* to 3 = *very much*). The PSS-I has excellent test-retest reliability (.80) and interrater reliability ($K = .91$; Foa & Tolin, 2000). The psychometric properties of the PSS-I are comparable to the Clinician Administered PTSD Scale (Weathers et al., 2013) employed in studies of veterans. The Cronbach's α for the PSS-I in the current sample was .68 at baseline.

Posttraumatic Cognitions Inventory (PTCI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999)

The PTCI is a 36-item, self-report measure of negative trauma-related cognitions associated with PTSD. It includes three subscales: Self-Blame, Negative Cognitions About the Self, and Negative Cognitions About the World (Foa et al., 1999). Individuals rate the extent to which they agree or disagree with each statement on a 1 (*totally disagree*) to 7 (*totally agree*) scale. The PTCI has been shown to discriminate between traumatized individuals with and without PTSD (Foa et al., 1999). The Cronbach's α for the PTCI in the current sample was .95.

Cognitive Emotion Regulation Questionnaire–Short (CERQ-Short; Garnefski & Kraaij, 2006)

The CERQ is an 18-item self-report measure of cognitive emotion regulation that focuses on a broad range of cognitive components of emotion regulation, referring to an individual's thoughts in response to the experience of threatening or stressful events, rather than behavioral aspects of coping (Garnefski, Kraaij, & Spinhoven, 2002). The CERQ-Short includes nine subscales measuring nine conceptually distinct cognitive emotion regulatory strategies (self-blame, other-blame, rumination, catastrophizing, positive refocusing, planning, positive reappraisal, putting into perspective, and acceptance). Because the CERQ has nine 2-item subscales, running models for each subscale would increase the risk of Type 1 error. Therefore, we only included the subscales with adequate internal reliability (Cronbach's $\alpha = 0.7$) in the model: acceptance ($\alpha = .80$), positive refocusing ($\alpha = .82$), catastrophizing ($\alpha = .76$) and blames other ($\alpha = .74$).

INTERVENTIONS

Prolonged Exposure Therapy (PE)

PE (Foa, Hembree, & Rothbaum, 2007) is a manualized cognitive-behavioral therapy consisting of imaginal exposure (repeated recounting of the most disturbing traumatic memory) followed by processing thoughts and feelings related to the imaginal experience; in vivo exposure (approaching trauma-related situations), psychoeducation about PTSD; and controlled breathing training. Between sessions, participants listen to audio recordings of the imaginal recounting daily and complete in vivo exercises. Ten 90-minute sessions of PE were delivered over 8 weeks: twice-weekly during the first and last weeks and once weekly during the 6 intervening weeks.

Present-Centered Therapy (PCT)

PCT is a non-trauma-focused, manualized treatment that controls for nonspecific therapeutic factors (Frost, Laska, & Wampold, 2014; Schnurr et al., 2005). Therapists help participants identify stressors and discuss them in a supportive, nondirective manner. Treatment focuses on current life problems that may or may not be PTSD-related. Ten 90-minute sessions were scheduled over 8 weeks, similarly to PE.

PROCEDURES

This study utilized data collected as part of a larger RCT evaluating the efficacy of PE for PTSD treatment in active-duty military personnel (Foa et al., 2018). Following informed consent, eligibility was determined during a baseline evaluation consisting of a clinical interview and self-report measures. Participants randomized to the Spaced PE and PCT conditions were assessed twice during treatment (Week 2 and Week 4), posttreatment (Week 8) and at 2 weeks and 3 months, and 6 months after treatment. The institutional review boards of Brooke Army Medical Center, The University of Texas Health Science Center at San Antonio, and the University of Pennsylvania approved the protocol.

DATA ANALYTIC APPROACH

Lagged mediational analyses were conducted to evaluate the relationship between putative mediators (posttraumatic cognitions and cognitive emotion regulation) and PTSD symptoms from baseline to 6-month follow-up. In these analyses, the mediator at time point t (baseline, midtreatment, posttreatment, 2-week follow-up, 10-week follow-up, and 3-month follow-up) predicted the PTSD symptoms at the next time point $t+1$ (midtreatment, posttreatment, 2-week follow-up, 10-week

follow-up, 3-month follow-up and 6-month follow-up). This approach controls for the temporal precedence of mediator versus outcome variables in longitudinal design.

An individual mediator model was used to examine the indirect effect of the mediators separately (Model A in Figure 1). To test negative cognitions as a mediator, we examined whether PSS-I at time $t+1$ was mediated by PTCI at time t . In this model, time is the predictor variable, PTCI is the mediator, and PSS-I is the outcome variable. To test emotion regulation as a mediator, we examined whether PSS-I at time $t+1$ was mediated by CERQ at time t .

A multiple mediator model (Preacher & Hayes, 2008) examined the relative strength of the mediation effect between PTCI and CERQ (Model B in Figure 1). In this model, time was the predictor variable, PTCI and CERQ subscales were mediators, and PSS-I was the outcome variable. Because PTSD decreased more quickly during treatment than during the follow-up phase (Foa et al., 2018), a log-transformation was applied to time. Pairwise contrasts of the indirect effects of the two mediators were examined to assess the significance and magnitude of the influence of each mediator. To examine the exploratory aim, a moderated multiple mediator model was applied to test whether treatment condition moderated the mediating effect (Model C in Figure 1). Bootstrapping (with 5,000 bootstrap samples) was used to estimate bias-corrected 95% confidence intervals (CI) and confirm the indirect (mediating) effect. Bootstrapping computes more accurate confidence intervals of indirect effects than the more commonly used methods, such as the causal steps strategy (Baron & Kenny, 1986), as it does not assume that the sampling distribution is normal (Preacher & Hayes, 2008). This is especially relevant for indirect effects, as their distributions are skewed from zero (Shrout & Bolger, 2002). Analyses were conducted using SPSS, version 24 with an SPSS macro named PROCESS (Preacher & Hayes, 2004; Preacher, Rucker, & Hayes, 2007). Age and gender were controlled in all analyses.

Results

Participants' demographic characteristics and descriptive results on study variables are presented in Table 1.

INDIVIDUAL MEDIATOR ANALYSES

The total effect of Time on PTSD symptoms was significant ($B = -1.743$, $SE = 0.307$, $p < .001$), and the model significantly explained 3.5% (R^2 , $p < .001$) of variance in PTSD. An individual mediator analysis was applied to three subscales of PTCI and four

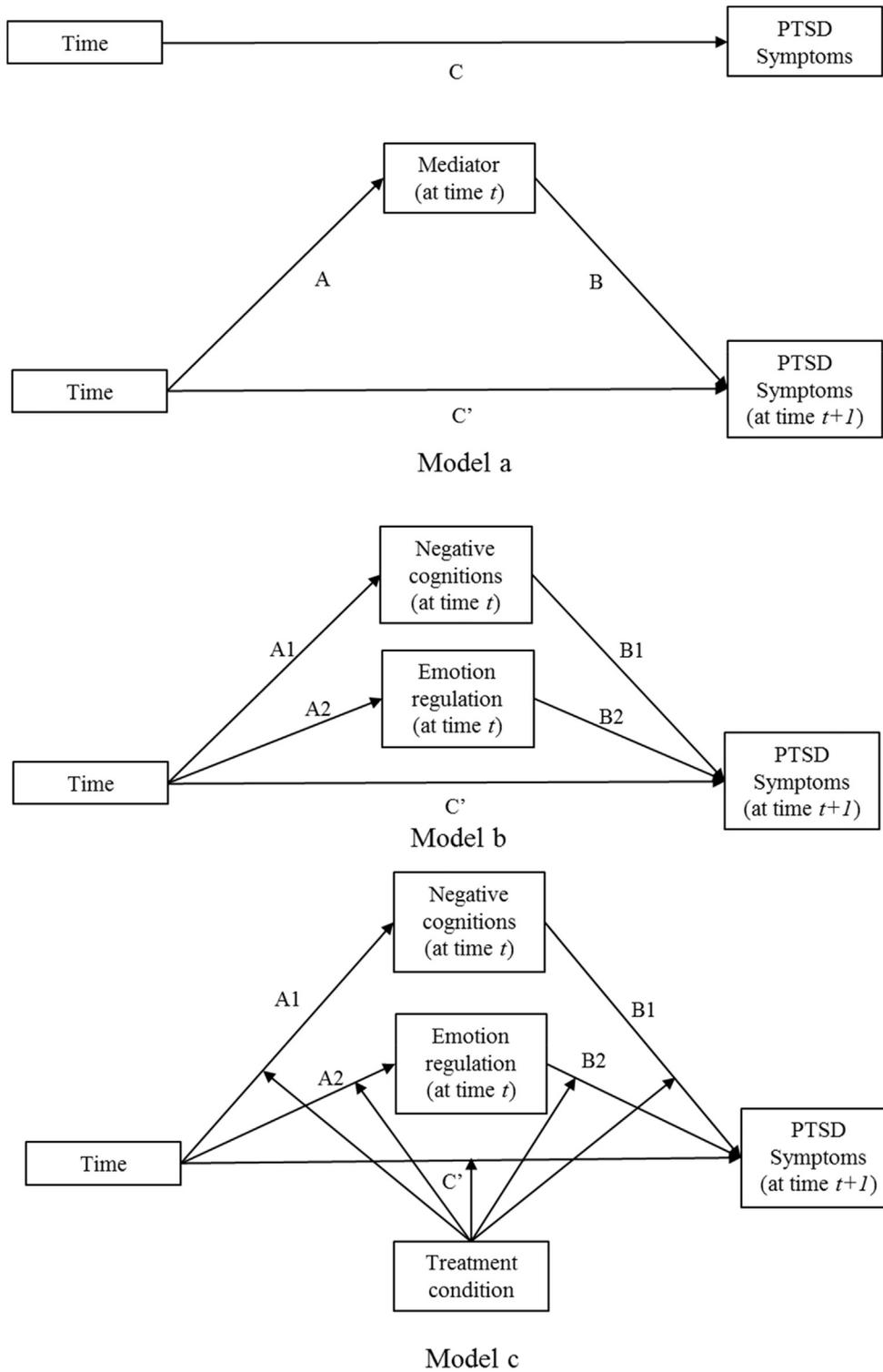


FIGURE 1 Hypothesized model. PTSD = posttraumatic stress disorder.

emotion regulation subscales of CERQ. With regard to the PTCL, there was significant indirect effect through negative cognitions about the self (indirect effect = -0.439, SE = 0.150, 95% CI = -0.732 to

-0.138), as well as negative cognitions about the world (indirect effect = -0.283, SE = 0.121, 95% CI = -0.538 to -0.050). However, self-blame was not a mediator of the relationship between Time and PTSD

Table 1
Demographic Characteristics by Condition

	Total Sample (<i>N</i> = 216)	Spaced PE (<i>n</i> = 109)	PCT (<i>n</i> = 107)
Age, mean (<i>SD</i>)	32.78 (7.20)	32.89 (7.05)	32.54 (7.45)
Sex			
Men	190	99 (90.8)	91 (85.0)
Women	26	10 (9.2)	16 (15.0)
Marital Status			
Not married	59	28 (25.7)	31 (29.0)
Married or cohabiting	157	81 (74.3)	76 (71.0)
Education			
High school	70	38 (34.9)	32 (29.9)
College experience	141	69 (63.3)	72 (67.3)
Postgraduate	5	2 (1.8)	3 (2.8)
Ethnicity			
Hispanic	44	20 (18.3)	24 (22.4)
Non-Hispanic	172	89 (81.7)	83 (77.6)
Race/ethnicity			
Asian	2	2 (1.8)	0
Black	40	28 (25.7)	22 (20.6)
White	130	62 (56.9)	68 (63.6)
Other	34	17 (15.6)	17 (15.9)
Enlisted			
E-1 to E-3	2	2 (1.8)	0
E-4 to E-6	171	86 (78.9)	85 (81.7)
E-7 to E-9	35	18 (16.5)	17 (16.3)
Warrant officer	2	1 (0.9)	1 (1.0)
Officer	3	2 (1.8)	1 (1.0)
No. of times deployed			
1 or 2	130	64 (58.7)	66 (61.7)
> 3	86	45 (41.3)	41 (38.3)
Time in military, mean (<i>SD</i>), <i>y</i>	10.84 (6.23)	11.01 (6.30)	10.68 (6.19)

Note. PE = prolonged exposure therapy; PCT = present-centered therapy; E-1 to E-3 = junior enlisted; E-4 to E-6 = junior noncommissioned officers; E-7 to E-9 = senior noncommissioned officers.

as Time was not significantly associated with self-blame ($p = .162$) that self-blame did not reduce during the treatment and follow-up. In terms of emotion regulation, the results showed that decreased catastrophizing significantly mediated (indirect effect = $-.959$, $SE = 0.146$, 95% CI = -1.260 to -0.682) the relationship between Time and decreased PTSD. Positive refocusing, acceptance, and other blames did not significantly mediate the relationship between Time and PTSD (all $ps > .05$).

MULTIPLE MEDIATOR ANALYSES

The parameter estimates for the total, direct, and specific indirect effects with bias-corrected 95% CI for the multiple mediator model are reported in Table 2. *Self-Blame*, *Negative Cognitions about the Self*, *Negative Cognitions about the World*, *Catastrophizing*, *Positive Refocusing*, *Acceptance*, and *Blames Others* were added simultaneously into the model; see Figure 2. The seven variables, taken as a set, mediated the relationship between Time and PTSD, and the total indirect effect of Time on PTSD

was significant (total indirect effect = -0.830 , $SE = 0.269$, 95% CI = -1.358 to -0.302). Negative cognitions about the self (indirect effect = -0.328 , $SE = 0.118$, 95% CI = -0.564 to -0.104), negative cognitions about the world (indirect effect = -0.065 , $SE = 0.042$, 95% CI = -0.166 to -0.003) and catastrophizing (indirect effect = -0.556 , $SE = 0.108$, 95% CI = -0.784 to -0.362) were the significant mediators of the Time-PTSD relationship, with the presence of all variables in the model. The model significantly explained 31.2% (R^2 , $p < .001$) of variance in PTSD. In terms of the magnitude of the indirect effect, pairwise contrasts between the three mediators indicated that negative cognitions about the self and catastrophizing had a nonsignificantly different mediating effect on PTSD (comparison = 0.228 , $SE = 0.141$, 95% CI = -0.050 to 0.498 ; see Table 2). Both catastrophizing and negative cognitions about the self had a significantly greater mediating effect than negative cognitions about the world (negative cognitions about the self vs. world, comparison = -0.263 , $SE = 0.110$, 95%

Table 2
Unstandardized Total, Direct and Indirect Effect of Time on PTSD Through Posttraumatic Cognitions and Emotional Regulation

Effects on PTSD	Variables	Point estimate	SE	p	Bias-corrected 95% CI	
					Lower	Upper
Total effect (c)	Time	-1.743	0.307	< .001	-2.346	-1.140
Direct effect (c')	Time	-0.830	0.269	< .01	-1.358	-0.302
Indirect effect	Negative cognitions about the self	-0.328	0.118	-	-0.564	-0.104
	Negative cognitions about the world	-0.065	0.042	-	-0.166	-0.003
	Self-blame	0.034	0.031	-	-0.013	0.107
	Catastrophizing	-0.556	0.108	-	-0.784	-0.362
	Blames others	0.005	0.014	-	-0.019	0.041
	Positive refocusing	0.0003	0.010	-	-0.021	0.021
	Acceptance	-0.003	0.013	-	-0.033	0.024
	Total indirect effect	-0.328	0.118	-	-0.564	-0.104
Contrasts						
Negative cognitions about the self vs. the world		-0.263	0.11	-	-0.49	-0.06
Negative cognitions about the self vs. Catastrophizing		0.228	0.141	-	-0.05	0.498
Negative cognitions about the world vs. Catastrophizing		0.491	0.111	-	0.285	0.721

Note: PTSD = posttraumatic stress disorder; SE = standard error; CI = confidence intervals.

CI = -0.490 to -0.060; negative cognitions about the world vs. catastrophizing, comparison = 0.491, SE = 0.111, 95% CI = 0.285 to 0.721; see Table 2).

A post-hoc analysis was conducted to determine whether the mediating effect of catastrophizing was an artifact of its overlap with PTSD reexperiencing symptoms. The results of the mediation analyses, omitting the PTSD reexperiencing symptoms, showed that negative cognitions about the self (indirect effect =

-0.282, SE = 0.097, 95% CI = -0.478 to -0.101), negative cognitions about the world indirect effect = -0.062, SE = 0.036, 95% CI = -0.146 to -0.007), and catastrophizing (indirect effect = -0.271, SE = 0.069, 95% CI = -0.415 to -0.146) were still significant mediators between the relationship of Time and PTSD avoidance and arousal symptoms. The model significantly explained 32.2% (R^2 , $p < .001$) of variance in avoidance and arousal symptoms.

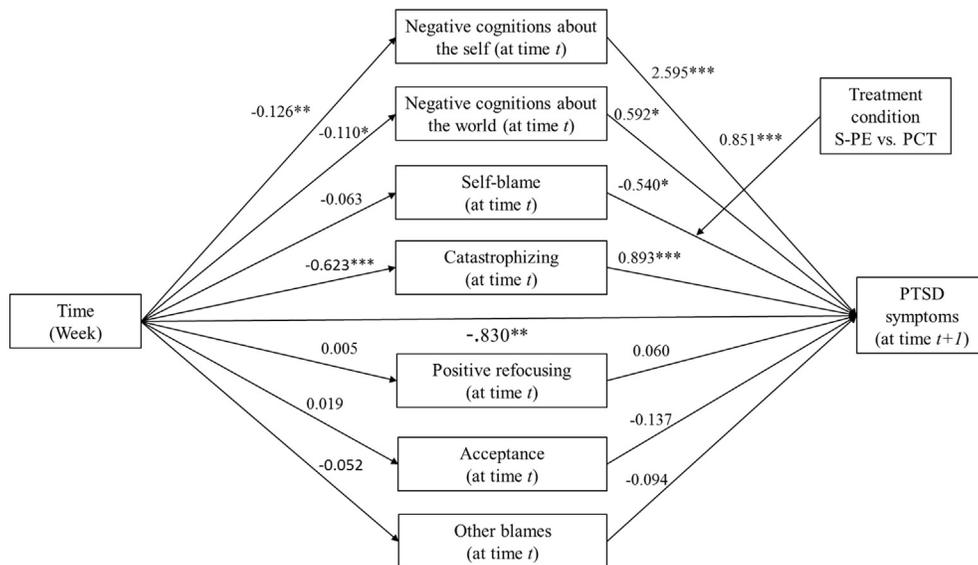


FIGURE 2 Multiple mediator model. The estimate point of the treatment condition reported was the coefficient of catastrophizing × condition interaction with both variables mean centered. S-PE = spaced prolonged exposure therapy; PCT = present-centered therapy; PTSD = posttraumatic stress disorder; ** $p < .01$, *** $p < .001$.

A second post-hoc time-lagged analysis examined the temporary sequencing of the changes in negative cognitions and catastrophizing. Negative cognitions about the self mediated the relationship between Time and catastrophizing (indirect effect = -0.130 , $SE = 0.041$, 95% CI = -0.214 to -0.052), and the direct effect was still significant (direct effect = -0.333 , $SE = 0.088$, 95% CI = -0.507 to -0.160). The reverse model showed that catastrophizing mediated the relationship between Time and negative cognitions about the self (indirect effect = -0.144 , $SE = 0.022$, 95% CI = -0.189 to -0.101), and there was no significant direct effect (direct effect = -0.012 , $SE = 0.048$, 95% CI = -0.107 to -0.083), suggesting that changes in catastrophizing precede changes negative cognitions during PTSD treatment.

MODERATED MEDIATION (EXPLORATORY) ANALYSIS

Dummy-coded treatment condition (PE = 1, PCT = 0) was added to the model to examine whether individual paths of the mediation model were moderated by condition. We ran one model with condition tested as a moderator for each path simultaneously. The results indicated that treatment condition only moderated the path from catastrophizing to PTSD (condition \times catastrophizing: $B = 0.851$, $p < .001$, 95% CI = 0.370 – 1.332 ; see Figure 2). This interaction is illustrated by selecting the values of mean ± 1 SD in Figure 3. There was a steeper slope for the PE group, indicating a stronger

relationship between catastrophizing and PTSD, such that catastrophizing had a greater mediating effect for participants in PE than those in PCT.

Discussion

This study aimed to test the specificity of the finding that negative cognitions mediate PTSD recovery during PE (e.g., Zalta et al., 2014) in two ways: (a) by examining negative cognitions as a mediator of change in PE and in a non-trauma-focused treatment, PCT, and (b) by testing a credible alternate mediator of PTSD change, emotion regulation. Replicating prior research and extending those findings to an active-duty military sample, we found that negative trauma-related cognitions significantly mediated reductions in PTSD severity in the full sample, which supported our first hypothesis. Specifically, two of the three PTCI subscales, negative cognitions about the world and negative cognitions about the self, were significant mediators of PTSD change in those receiving PE or PCT. This is consistent with one prior study examining mediators of PE and supportive counseling in adolescents (McLean, Yeh, et al., 2015) and suggests that changes in negative cognitions may underlie therapeutic recovery in general, rather than in PE or in trauma-focused therapy specifically. Replication and extension to other treatments, including pharmacological approaches, would further improve our understanding of how trauma-related cognitions may drive PTSD recovery.

Consistent with our second hypothesis, the cognitive emotion-regulation strategy of catastrophizing also significantly mediated PTSD reductions in both PE and PCT. Moreover, and in contrast to our third hypothesis, in a combined mediator model the mediating effect of catastrophizing on PTSD change was greater than negative cognitions about the world but not significantly different than the effect of negative cognitions about the self. Catastrophizing involves excessive negative interpretations about the event or its consequences and believing a situation is worse than it truly is. The CERQ catastrophizing items used in this study were “I keep thinking about how terrible it is what I have experienced” and “I continually think how horrible the situation has been” (participants were prompted to answer questions in reference to their index trauma [worst event]). Some of the negative trauma-related cognitions about the self that are assessed using the PTCI are similar to catastrophizing (e.g., “My life has been destroyed by the trauma”). The distinction is that the PTCI assesses beliefs about the self, world, and others, whereas the CERQ is designed to measure strategies used to cope with the trauma. It should also be noted that the CERQ catastrophizing items appear to assess a

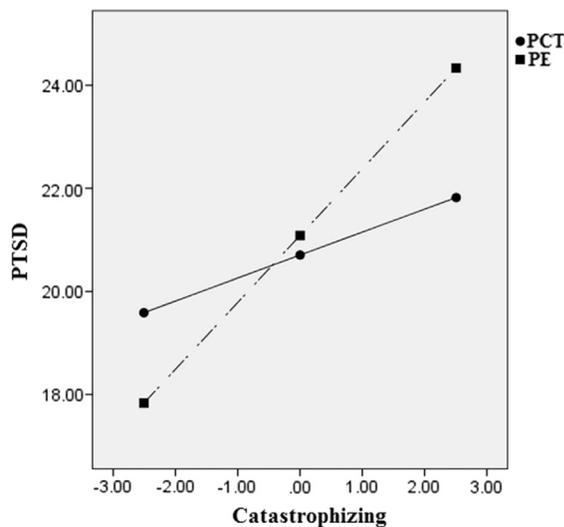


FIGURE 3 Moderation effect of treatment condition on the relationship between catastrophizing and total PTSD symptoms. Catastrophizing and PTSD were both mean centered. PCT = present-centered therapy; PE = prolonged exposure therapy; PTSD = posttraumatic stress disorder.

combination of catastrophizing and rumination, and that prior work suggests that rumination plays a role in maintaining PTSD symptoms (e.g., Michael, Halligan, Clark, & Ehlers, 2007). Although there is a separate CERQ subscale that assesses rumination, it did not meet our internal consistency criterion for inclusion in the present study, emphasizing the need for replication with more comprehensive measures of emotion regulation.

The CERQ catastrophizing items are also similar to items on the PSS-I measuring PTSD reexperiencing symptoms (i.e., Have you had recurrent or intrusive distressing thoughts or recollections about the trauma?), raising the possibility that the mediating effect of catastrophizing was an artifact of its overlap with reexperiencing symptoms. However, results of a post-hoc analysis showed that negative cognitions about the self, negative cognitions about the world, and catastrophizing were significant mediators of change in PTSD, even when the PSSI re-experiencing symptoms were omitted from the analysis.

Although our findings were similar for both catastrophizing and negative cognitions, the greater mediating effect of catastrophizing on PTSD change suggests that lessening one's focus on how terrible the trauma was may drive recovery more than shifts in negative cognitions, per se. It is also possible that shifts in negative cognitions are facilitated by reductions in catastrophizing. When cognitive resources are less occupied due to catastrophizing, it may be easier to reevaluate unhelpful beliefs about the trauma and its impact. Indeed, the results of a post-hoc time-lagged analysis suggest that changes in catastrophizing precede changes in negative cognitions during PTSD treatment. Future studies further examining the temporal relationship between emotion regulation strategies and trauma-related cognitions during PTSD treatment could address this hypothesis,

Our exploratory analyses showed that the mediating effect of catastrophizing was stronger in PE than in PCT, suggesting that reductions in catastrophizing may be more important for recovery in PE than PCT, although the magnitude of this effect was not large. Learning to cope successfully while approaching trauma cues via imaginal and in vivo exposure is incompatible with catastrophizing. That could explain why catastrophizing played a more influential role in PE than in PCT, for which other yet-identified variables may be more influential in mediating recovery.

Several limitations to the current study should be noted. The primary limitation relates to the measure of cognitive emotion regulation. The CERQ-Short is a brief measure comprised of two-

item subscales. Although we only included those subscales with adequate internal consistency, conclusions should be tempered until replicated with a more robust measure of catastrophizing. Also, while this study of active-duty military personnel fills a gap in the literature, the current sample was predominantly male (88%), and thus the results may not generalize to female service members or veterans. We also note that our findings demonstrate statistical mediation only, which is necessary but insufficient to establish a mechanism. Future work focused on gathering experimental evidence of cause and delineating a mechanistic explanation is needed to establish whether these constructs are also mechanisms of PTSD change.

These limitations notwithstanding, the results of this study provide further evidence of the mediating role of negative trauma-related cognitions in PTSD treatment response. They also suggest that this role may not be specific to PE but could extend to non-trauma-focused evidence-based PTSD treatment. In addition, the results also show that the emotion regulation strategy of catastrophizing had a greater mediating effect than negative cognitions and that this was particularly true for PE. These novel findings of the mediating effect of catastrophizing demonstrate the value of testing alternate mediators, although replication with better measures is needed.

Conflict of Interest Statement

Dr. Foa receives royalties from the sale of Prolonged Exposure Therapy for PTSD: Emotional Processing of Traumatic Experiences Therapist Guide, and Reclaiming your Life from a Traumatic Experience Workbook by Oxford University Press. She also receives payment for training she conducts on prolonged exposure therapy. The other authors declare no conflicts of interest.

References

- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality & Social Psychology*, 51, 1173–1182. <https://doi.org/10.1037//0022-3514.51.6.1173>
- Cahill, S. P., & Foa, E. B. (2007). Psychological theories of PTSD. In M. J. Friedman, T. M. Keane, & P. A. Resick (Eds.), *Handbook of PTSD: Science and practice* (pp. 55–77). New York, NY: Guilford Press.
- Cieslak, R., Benight, C. C., & Lehman, V. C. (2008). Coping self-efficacy mediates the effects of negative cognitions on posttraumatic distress. *Behaviour Research and Therapy*, 46, 788–798. <https://doi.org/10.1016/j.brat.2008.03.007>
- Epstein, S. (1991). Cognitive-experiential self-theory: Implications for developmental psychology. In M. Gunnar & L. A. Sroufe (Eds.), *Self-processes and development: The Minnesota*

- Symposia on Child Development* (Vol. 23, pp. 79-123). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Foa, E. B., & Cahill, S. P. (2001). Psychological therapies: Emotional processing. In N. J. Smelser, & P. B. Bates (Eds.), *International encyclopedia of the social and behavioral sciences* (pp. 12363-12369). Oxford, England: Elsevier.
- Foa, E. B., Ehlers, A., Clark, D. M., Tolin, D. F., & Orsillo, S. M. (1999). The Posttraumatic Cognitions Inventory (PTCI): Development and validation. *Psychological Assessment*, 11, 303-314. <https://doi.org/10.1037/1040-3590.11.3.303>
- Foa, E. B., Hembree, E., & Rothbaum, B. O. (2007). *Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences: Therapist guide*. New York, NY: Oxford University Press. <https://doi.org/10.1093/med:psych/9780195308501.001.0001>
- Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, 99, 20-35. <https://doi.org/10.1037/0033-2909.99.1.20>
- Foa, E. B., McLean, C. P., Zang, Y., Rosenfield, D., Yadin, E., Yarvis, J. S., ... Peterson, A. L.; for the STRONG STAR Consortium (2018). Effect of prolonged exposure therapy delivered over 2 weeks vs 8 weeks vs present-centered therapy on PTSD symptom severity in military personnel: A randomized clinical trial. *Journal of the American Medical Association*, 319, 354-364. <https://doi.org/10.1001/jama.2017.21242>
- Foa, E., Riggs, D., Dancu, C., & Rothbaum, B. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress*, 6, 459-474. <https://doi.org/10.1007/BF00974317>
- Foa, E. B., & Tolin, D. F. (2000). Comparison of the PTSD Symptom Scale-Interview Version and the Clinician-Administered PTSD Scale. *Journal of Traumatic Stress*, 13, 181-191. <https://doi.org/10.1023/A:1007781909213>
- Frost, N. D., Laska, K. M., & Wampold, B. E. (2014). The evidence for present-centered therapy as a treatment for posttraumatic stress disorder. *Journal of Traumatic Stress*, 27, 1-8. <https://doi.org/10.1002/jts.21881>
- Garnefski, N., & Kraaij, V. (2006). Cognitive Emotion Regulation Questionnaire-Development of a short 18-item version (CERQ-short). *Personality and Individual Differences*, 41, 1045-1053. <https://doi.org/10.1016/j.paid.2006.04.010>
- Garnefski, N., Kraaij, V., & Spinhoven, P. (2002). *Manual for the use of the Cognitive Emotion Regulation Questionnaire*. Leiderdorp, The Netherlands: DATEC.
- Goldin, P., Ziv, M., Jazaieri, H., Werner, K., Kraemer, H., Heimberg, R. G., & Gross, J. J. (2012). Cognitive reappraisal self-efficacy mediates the effects of individual cognitive-behavioral therapy for social anxiety disorder. *Journal of Consulting and Clinical Psychology*, 80, 1034-1040. <https://doi.org/10.1037/a0028555>
- Held, P., Owens, G. P., Schumm, J. A., Chard, K. M., & Hansel, J. E. (2011). Disengagement coping as a mediator between trauma-related guilt and PTSD severity. *Journal of Traumatic Stress*, 24, 708-715. <https://doi.org/10.1002/jts.20689>
- Horowitz, M. J. (1976). *Stress response syndromes*. Oxford, England: Jason Aronson.
- Horowitz, M. J. (1986). Stress-response syndromes: A review of posttraumatic and adjustment disorders. *Psychiatric Services*, 37, 241-249. <https://doi.org/10.1176/ps.37.3.241>
- Janoff-Bulman, R. (1992). *Shattered assumptions: Towards a new psychology of trauma*. New York, NY: Free Press.
- Michael, T., Halligan, S. L., Clark, D. M., & Ehlers, A. (2007). Rumination in posttraumatic stress disorder. *Depression and Anxiety*, 24(5), 307-317. <https://doi.org/10.1002/da.20228>
- McCann, I. L., & Pearlman, L. A. (1990). Vicarious traumatization: A framework for understanding the psychological effects of working with victims. *Journal of Traumatic Stress*, 3, 131-149. <https://doi.org/10.1007/BF00975140>
- McLean, C. P., Su, Y. J., & Foa, E. B. (2015). Mechanisms of symptom reduction in a combined treatment for comorbid posttraumatic stress disorder and alcohol dependence. *Journal of Clinical and Consulting Psychology*, 83, 655-661. <https://doi.org/10.1037/ccp0000024>
- McLean, C. P., Yeh, R., Rosenfield, D., & Foa, E. B. (2015). Changes in negative cognitions mediate PTSD symptom reductions during client-centered therapy and prolonged exposure for adolescents. *Behaviour Research and Therapy*, 68, 64-69. <https://doi.org/10.1016/j.brat.2015.03.008>
- Pietrzak, R. H., Harpaz-Rotem, I., & Southwick, S. M. (2011). Cognitive-behavioral coping strategies associated with combat-related PTSD in treatment-seeking OEF-OIF veterans. *Psychiatry Research*, 189, 251-258. <https://doi.org/10.1016/j.psychres.2011.07.019>
- Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers*, 36, 717-731. <https://doi.org/10.3758/bf03206553>
- Preacher, K. J., & Hayes, A. F. (2008). Contemporary approaches to assessing mediation in communication research. In A. F. Hayes, M. D. Slater, & L. B. Snyder (Eds.), *The Sage sourcebook of advanced data analysis methods for communication research* (pp. 13-54). Thousand Oaks, CA: Sage.
- Preacher, K. J., Rucker, D. D., & Hayes, A. F. (2007). Assessing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research*, 42, 185-227. <https://doi.org/10.1080/00273170701341316>
- Schnurr, P. P., Friedman, M. J., Engel, C. C., Foa, E. B., Shea, M. T., Resick, P. M., ... Chow, B. K. (2005). Issues in the design of multisite clinical trials of psychotherapy: VA Cooperative Study No. 494 as an example. *Contemporary Clinical Trials*, 26, 626-636. <https://doi.org/10.1016/j.cct.2005.09.001>
- Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, 7, 422-445. <https://doi.org/10.1037/1082-989x.7.4.422>
- Weathers, F. W., Blake, D. D., Schnurr, P. P., Kaloupek, D. G., Marx, B. P., & Keane, T. M. (2013). The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5). Interview available from the National Center for PTSD at <https://www.ptsd.va.gov/professional/assessment/adult-int/caps.asp>
- Woods, D. W., Wetterneck, C. T., & Flessner, C. A. (2006). A controlled evaluation of acceptance and commitment therapy plus habit reversal for trichotillomania. *Behaviour Research and Therapy*, 44, 639-656. <https://doi.org/10.1016/j.brat.2005.05.006>
- Zalta, A. K., Gillihan, S. J., Fisher, A. J., Mintz, J., McLean, C. P., Yehuda, R., & Foa, E. B. (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology*, 82, 171-175. <https://doi.org/10.1037/a0034735>

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