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Behavior Therapy 51 (2020) 386-400

Behavior Therapy

www.elsevier.com/locate/bt

# Weekly Changes in Blame and PTSD Among Active-Duty Military Personnel Receiving Cognitive Processing Therapy

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Funding for this work was made possible by the U.S. Department of Defense through the U.S. Army Medical Research and Materiel Command, Congressionally Directed Medical Research Programs, Psychological Health and Traumatic Brain Injury Research Program awards W81XWH-08-02-0109 (Alan Peterson), W81XWH-08-02-0116 (Patricia Resick), and W81XWH-08-02-0114 (Brett Litz). This work was supported in part by Career Development Award No. IK2RX002965 (Kirsten Dillon) from the U.S. Department of Veterans Affairs Rehabilitation R&D (Rehab RD) Service.

The funding source played no role in the study design; the collection, analysis, and interpretation of data; the writing of this paper; or the decision to submit this paper for publication.

The views expressed herein are solely those of the authors and do not reflect an endorsement by or the official policy or position of the U.S. Army, the Department of Defense, the Department of Veterans Affairs, or the U.S. government.

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Both negative posttraumatic cognitions and posttraumatic stress disorder (PTSD) symptoms decrease over the course of cognitive-behavior therapy for PTSD; however, further research is needed to determine whether cognitive change precedes and predicts symptom change. The present study examined whether weekly changes in blame predicted subsequent changes in PTSD symptoms over the course of cognitive processing therapy (CPT). Participants consisted of 321 active duty U.S. Army soldiers with PTSD who received CPT in one of two clinical trials. Symptoms of PTSD and blame were assessed at baseline and weekly throughout treatment. Bivariate latent difference score modeling was used to examine temporal sequential dependencies between the constructs. Results indicated that changes in self-blame and PTSD symptoms were dynamically linked: When examining cross-construct predictors, changes in PTSD symptoms were predicted by prior changes in self-blame, but changes in self-blame were also predicted by both prior levels of and prior changes in PTSD. Changes in other-blame were predicted by prior levels of PTSD, but changes in other-blame did not predict changes in PTSD symptoms. Findings highlight the dynamic relationship between self-blame and PTSD symptoms during treatment in this active military sample.

COGNITIVE-BEHAVIORAL THEORIES of posttraumatic stress disorder (PTSD) emphasize the role of negative posttraumatic cognitions, such as excessive selfblame and negative thoughts about self, others, and the world, in the development and maintenance of symptoms (Ehlers & Clark, 2000; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999; LoSavio, Dillon, & Resick, 2017). Civilian studies have demonstrated that PTSD severity is associated with higher levels of negative posttraumatic cognitions (e.g., Beck, Jones, Reich, Woodward, & Cody, 2015; Foa et al., 1999) and that posttraumatic cognitions longitudinally predict PTSD symptoms (Carper et al., 2015; Dunmore, Clark, & Ehlers, 2001; O'Donnell, Elliott, Wolfgang, & Creamer, 2007), supporting the theory that cognitions contribute to the development and maintenance of PTSD.

Cognitive processing therapy (CPT) is an evidencebased, cognitive-behavioral therapy that was developed based on the assumption that reductions in negative posttraumatic cognitions will lead to subsequent reductions in PTSD symptoms (Resick, Monson, & Chard, 2017). CPT has been identified as one of the leading treatments for PTSD among veterans and active duty military personnel (Department of Veterans Affairs and Department of Defense, 2017). In CPT, inaccurate, negative beliefs about the causes (e.g., "The trauma was my fault") and implications (e.g., "I can never trust anyone again") of the traumatic event are referred to as "stuck points," and theorized to keep patients "stuck" with their PTSD symptoms and impede their recovery from the trauma. CPT uses Socratic questioning and a series of worksheets to teach patients new skills to help them modify these negative posttraumatic beliefs and develop more balanced and helpful beliefs about the trauma (Resick, Monson, et al., 2017). Two types of cognitions that are prioritized in CPT are erroneous self- and other-blame. Inaccurate beliefs about one's culpability for the traumatic event, or that another person who was not the perpetrator of the event was at fault, are conceptualized as primary reasons why patients are unable to recover. When present, such inaccurate beliefs are targeted early in the treatment.

In support of the theory underlying CPT, several researchers have found that negative posttraumatic cognitions decrease over the course of treatment (Dondanville et al., 2016; Iverson, King, Cunningham, & Resick, 2015; Resick et al., 2008). A recent meta-analysis reported large effect sizes for reductions in trauma-related negative cognitions from pre- to posttreatment (Holliday, Holder, & Surís, 2018b). However, the majority of these studies examined PTSD symptoms and posttraumatic cognitions as concurrent outcomes of CPT treatment without testing whether changes in cognitions temporally precede changes in PTSD symptoms.

Researchers have recently begun to examine the temporal relationship between changes in cognitons and PTSD symptoms during treatment. One study evaluated changes in cognitions as a mediator of changes in PTSD symptoms in a sample of veterans receiving CPT in a residential treatment program (Schumm, Dickstein, Walter, Owens, & Chard, 2015). Using the Post-Traumatic Cognitions Inventory (PTCI; Foa et al., 1999) to assess traumarelated cognitions, the researchers found that preto midtreatment reductions in self-blame and

*Keywords:* CPT; PTSD; negative posttraumatic cognitions; blame; treatment processes

negative beliefs about the self preceded mid- to posttreatment reductions in PTSD symptoms. More recently, Holliday, Holder, and Surís (2018a) found that, from baseline to 6-months posttreatment, changes in self-blame cognitions (using the PTCI as well) predicted and preceded changes in PTSD symptoms among veterans with military sexual trauma-related PTSD. Both of these studies represent important steps in examining mechanisms of change in CPT; however, both are limited by the fact that cognitions and PTSD symptoms were not assessed frequently during therapy. It is possible that across therapy sessions and at different parts of therapy, negative cognitions may differentially affect changes in PTSD symptoms. A requirement for demonstrating mechanisms of symptom change is to assess the presumed mediating variable and outcome across treatment sessions (Kazdin, 2007).

Accordingly, other researchers have used more fine-grained, longitudinal designs to examine changes in cognitions and PTSD symptoms for other PTSD treatments, such as cognitive therapy for PTSD (CT-PTSD; Ehlers, Clark, Hackmann, McManus, & Fennell, 2005) and prolonged exposure (PE) therapy (Foa, Hembree, & Rothbaum, 2007). Kleim and colleagues (2013) examined session-by-session changes in cognitions (using a revised version of the PTCI) and PTSD symptoms over the course of CT-PTSD. Using bivariate latent difference score modeling, they found that weekly decreases in negative trauma-related cognitions predicted subsequent decreases in PTSD symptoms, whereas reductions in PTSD symptoms did not predict subsequent reductions in cognitions. Similarly, Zalta and colleagues (2014) examined whether changes in negative cognitions assessed with the PTCI were associated with changes in PTSD symptoms during PE in a sample of female victims of assault. They used a time-lagged mixedeffect regression approach to analyze session-bysession data and found that reductions in negative cognitions led to reductions in PTSD and depression symptoms, whereas the reverse was not true.

Other researchers have also used a time-lagged mixed-effect regression approach to examine the relationships between symptom and cognitive changes during PE. Kumpula et al. (2017) examined whether changes in specific types of posttraumatic cognitions on the PTCI differentially affected PTSD symptoms in a sample of outpatients. They found that reductions in cognitions about the world and the self led to reductions in PTSD symptoms. Cooper, Zoellner, Roy-Byrne, Mavissakalian, and Feeny (2017) compared the relationship between symptom reduction and cognitions in patients treated by PE versus sertraline, and found that changes in cognitions predicted changes in PTSD symptoms.

Taken together, these studies provide evidence that changes in cognitions often precede and predict changes in PTSD symptoms. However, to our knowledge, no studies have examined weekly changes over the course of CPT. Additionally, no studies have examined the relationship between cognitions and PTSD during treatment using an active duty military sample. Finally, there have been no studies examining other-blame during treatment. Because targeting both erroneous self- and other-blame are priorities in CPT, understanding the trajectory of other-blame during CPT is an important and understudied area.

The goal of the current study was to replicate and extend findings from prior studies to address these gaps in the literature. We investigated how both prior levels of and changes in blame cognitions and PTSD symptoms mutually influenced each other during CPT, using data collected weekly over the course of treatment. Expanding on prior studies, we included assessment of both self- and other-blame. In addition to assessing how prior changes in blame and PTSD are implicated in subsequent changes within and across constructs, we were also able to examine whether any lagged causal relationships varied at different parts (i.e., first half vs. second half) of the treatment process. Based on CPT theory, as well as prior research, we hypothesized that, over the course of treatment, there would be reductions in self-blame, other-blame, and PTSD symptoms. Furthermore, we predicted that reductions in blame would precede and predict subsequent changes in PTSD, but the reverse relationships would not be found.

#### Methods

## PARTICIPANTS

Data were drawn from two randomized clinical trials (RCTs) that examined CPT in active duty U. S. Army soldiers. In the first RCT (Resick et al., 2015), group CPT was compared with group present-centered therapy (PCT; Schnurr et al., 2005). The second RCT compared CPT delivered in group versus individual format (Resick, Wachen, et al., 2017). The sample, methods, and procedures of the parent trials are described in detail elsewhere (Resick, Wachen, et al., 2017; Resick et al., 2015). Participants in the current study included participants who received CPT in either the first (n = 56) or second RCT (n = 265); participants received CPT in either an individual (n = 133) or group (n = 188) format. Participants were 321 active duty U.S. Army soldiers, ages 18 or older, who had experienced a Criterion A traumatic event (as defined by the Diagnostic and Statistical Manual of Mental Disorders; DSM-IV-

TR; American Psychiatric Association, 2000) during a deployment to or around Iraq or Afghanistan. However, treatment may have focused on traumatic experiences that occurred outside of a deployment. Participants taking psychotropic medications maintained a stable dose for 6 weeks prior to study entry and were asked to keep this regimen unchanged throughout treatment. Participants were excluded if they reported current suicidal or homicidal risk meriting crisis intervention, active psychosis, or a moderate to severe traumatic brain injury.

The mean age of participants was 33 years (SD = 7.39). Participants were primarily male

(91%), and either White non-Hispanic (44%), Black non-Hispanic (28%), or Hispanic (26%), with the majority serving in the Army as members of the enlisted ranks (97%). Patient demographics for the total sample and by study are are shown in Table 1. None of the subsamples, as defined by treatment study or treatment modality, differed on any of the demographic variables; accordingly, they were all combined in subsequent analyses.

## PROCEDURES

Both parent studies were approved by the Institutional Review Boards at Brooke Army Medical Center, the University of Texas Health Science

Table 1

Demographic Characteristics and Descriptives for Main Study Variables

Characteristic	Total sample ( <i>N</i> = 321)	Study 1 participants ( <i>n</i> = 56) <sup>a</sup>	Study 2 participants $(n = 265)^{b}$	t or $\chi^2$	p
Age [ <i>M</i> ( <i>SD</i> )]	33.04 (7.39)	32.34 (7.32)	33.19 (7.43)	0.78	.43
Months in service [M (SD)]	128.45 (74.49)	118.73 (72.32)	130.48 (76.12)	1.06	.29
Male [ <i>n</i> (%)]	294 (91)	52 (93)	242 (91)	0.21	.65
Married/cohabiting [n (%)]	235 (73)	47 (84)	188 (71)	3.47	.06
Ethnicity [n (%)]				7.81	.05
Black	90 (28)	11 (20)	79 (30)		
Hispanic	85 (26)	10 (18)	75 (28)		
White	142 (44)	34 (61)	108 (41)		
Other	5 (1)	1 (2)	4 (1)		
Education [n (%)]				0.84	.66
High school or less	85 (26)	17 (30)	68 (26)		
Some college	213 (66)	36 (64)	177 (66)		
College/graduate degree	24 (8)	3 (6)	21 (8)		
Enlisted rank [n (%)]	313 (97)	54 (96)	259 (97)	1.50	.70
Typical duty [ <i>n</i> (%)]				0.37	.83
Combat arms	119 (37)	20 (36)	99 (37)		
Combat support	76 (24)	12 (21)	64 (24)		
Combat service support	127 (39)	24 (43)	103 (39)		
Number of deployments $[n (\%)]$				4.58	.21
1	93 (29)	17 (30)	30 (32)		
2	112 (35)	24 (43)	50 (38)		
3	66 (21)	11 (20)	28 (21)		
4+	51 (16)	4 (7)	25 (19)		

Sample size and estimated sample statistics for main study measures (*M*, *SE*)

Time point	Ν	PCL-S total	CERQ Self-Blame	CERQ Other-Blame
Baseline	321	56.16 (0.59)	3.68 (0.12)	4.68 (0.14)
Week 1 assessment	277	54.69 (0.60)	4.21 (0.11)	5.38 (0.12)
Week 2 assessment	253	53.97 (0.64)	4.15 (0.12)	5.54 (0.13)
Week 3 assessment	236	52.42 (0.73)	4.11 (0.12)	5.54 (0.13)
Week 4 assessment	216	50.89 (0.82)	3.94 (0.13)	5.48 (0.13)
Week 5 assessment	199	48.51 (0.95)	3.69 (0.12)	5.42 (0.14)
Week 6 assessment	175	45.73 (1.03)	3.44 (0.13)	5.35 (0.14)

*Note.* Tests for all categorical variables are with df = 1-3. Tests for variables presented with means and standard deviations are with df = 319. PCL-S = PTSD Checklist—Stressor-Specific Version; CERQ = Cognitive Emotion Regulation Questionnaire—Short Form.

<sup>a</sup> All of these participants received group CPT.

<sup>b</sup> Participants in their trial received CPT in either individual (n = 133) or group (n = 132) format.

Center at San Antonio, VA Boston Healthcare System, and Duke University. Participants provided informed consent in person with trained study staff and then completed a comprehensive baseline assessment. Clinical assessments were administered by trained masters- and doctoral-level independent evaluators who were masked to treatment condition. CPT consisted of 12 sessions, conducted by masters- and doctoral-level clinicians, twice weekly for 6 weeks (individual sessions were 60 minutes and group sessions were 90 minutes). Participants were assessed on self-reported outcome measures at baseline and once per week over the course of therapy. (See Resick, Wachen, et al., 2017; Resick et al., 2015, for flowcharts of participants.)

## MEASURES

The PTSD Symptom Scale—Interview (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993) was used for PTSD diagnosis at baseline but not otherwise used in the current study. The PSS-I is a 17-item clinical interview that evaluates DSM-IV PTSD symptoms on frequency/severity using a 4-point scale ranging from 0 (*not at all*) to 3 (*very much*). A diagnosis of PTSD is assigned when at least one reexperiencing, three avoidance, and two arousal symptoms were endorsed. The scale has demonstrated excellent internal consistency ( $\alpha = 0.91$ ), test–retest reliability (r = 0.80), and interrater reliability (kappa = 0.91).

The PTSD Checklist—Stressor-Specific Version (PCL-S; Weathers, Litz, Herman, Huska, & Keane, 1993) was used to assess PTSD symptoms at baseline and weekly during treatment. The PCL-S is a 17-item self-report measure used to assess how much an individual is bothered by PTSD symptoms on a scale from 1 (*not at all*) to 5 (*extremely*). Total scores range from 17 to 85, and higher scores reflect greater PTSD symptom severity. The PCL has been found to have excellent psychometric properties with both civilian and veteran populations (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). Coefficient alpha was acceptable across all assessment points for total scores (average alpha = 0.92, range = 0.85–0.96).

Participants also completed the Cognitive Emotion Regulation Questionnaire—Short Form (CERQ; Garnefski & Kraaij, 2006) at baseline and weekly. This 18-item questionnaire assesses cognitive coping strategies after having experienced threatening or stressful events. For this study, the instructions were modified to ask participants about their thoughts over the past week with regard to their worst combat, operational, or other lifetime experience. The 18 items form nine subscales that reflect both maladaptive (e.g., self-blame, other-blame) and adaptive (e.g., positive refocusing, positive reappraisal) cognitive strategies. Items are rated on a 5-point scale ranging from 1 (almost never) to 5 (almost always). Individual subscale scores are obtained by summing the two item scores belonging to the particular subscale, with higher scores indicating greater usage of the strategy. The CERQ was previously evaluated for its psychometric properties in a large adult population and demonstrated acceptable internal reliability, though the two-item scales of the short form demonstrated lower reliability than the original four-item subscales, as would be expected (alphas of 0.68 and 0.77 for self-blame and other-blame, respectively; Garnefski & Kraaij, 2006). Relationships with outcome measures were comparable to the original CERQ. For this study, the self-blame and otherblame subscales were used. Self-blame was assessed with the items "I feel that I am the one who is responsible for what has happened," and "I think that basically the cause must lie with myself." Other-blame was assessed with the items "I feel that basically the cause lies with others," and "I feel that others are responsible for what has happened." In the present study, the average alpha for the self-blame subscale was .78 (range = .70-.84) and the average alpha for other-blame was .85 (range = .79-.91).

## DATA ANALYTIC STRATEGY

To examine the degree to which PTSD and blame are dynamically related, we used the changes to changes extension of the latent difference score model (Grimm, An, McArdle, Zonderman, & Resnick, 2012), a simplified version of which is presented in Figure 1. This specialized structural equation framework combines elements of latent growth curve models and autoregressive crosslagged models. At each assessment, the observed scores of both outcomes (indicated by boxes in Figure 1) are modeled as a function of a latent true score and a residual term that reflects random error of measurement (indicated by circles with "e" in the figure). Once specified, the latent true score at any given time point is further specified in the model using a series of constraints as the sum of the previous time point's true score plus a latent change score representing the change in latent true score since the preceding assessment-that is, change at each time point is explicitly defined as the part of the true score that was not present at the previous time point. By explicitly modeling the change between every assessment, the latent change scores themselves can become the focus of prediction, and they can also be used as predictors



**FIGURE 1** Simplified version of the full bivariate latent difference score model linking changes in PTSD severity (IPCL-S) to changes in either self- or other-blame (ICERQ), with the third time point emphasized for clarity. All identically labeled paths are constrained to equality. After the third time point, the pattern of estimated paths continues identically for the four remaining assessments. Within-week assessment error corvariances omitted for clarity. Note. PCL-S = PTSD Checklist—Stressor-Specific Version; W = week; BL = baseline; A = effect of prior level of subscripted variable on its subsequent changes; B = effect of prior changes in subscripted variable on its subsequent changes; C = constant change over time in subscripted variable;  $D_{C^*P}$  = effect of prior changes in self- or other-blame on later changes in PTSD;  $D_{P*C}$  = effect of prior changes in self- or other-blame on later changes in PTSD;  $D_{P*C}$  = effect of prior levels of self- or other-blame on later changes in PTSD on later changes in self- or other-blame; CERQ = Cognitive Emotion Regulation Questionnaire—Short Form

of later changes for the same variable or a linked variable within a dynamic system. Additonally, all possible correlations between the intercepts and slopes of the two linked variables are directly estimated within the model, as is the covariance between the error terms across constructs within each assessment, the latter of which are constrained to be equal over time. Error terms within each construct are constrained to be unrelated over time.

As illustrated in the highlighted box, within a bivariate model, changes in either construct can be modeled by up to five parameters. Focusing on changes in PTSD prior to Week 2 ( $\Delta PCL - S_{W2}$ -W1), possible predictors in the model include ("A") prior levels of PTSD; ("B") prior changes in PTSD; ("C") the constant change in PTSD over time; ("D") prior changes in self- or other-blame; and ("E") prior levels of self- or other-blame. Changes in self- and other-blame prior to the Week 2 assessment are analogously modeled. Parameters labeled A, B, and C emanate "within construct," meaning they are autoregressive parameters derived from components of the focal variable itself to predict its own changes. Parameters D and E emanate "across constructs," meaning they are cross-lagged parameters that link the two constructs together in a dynamic system. Together, parameters A, B, D, and E allow researchers to examine whether levels of or changes in the same variable or a linked variable are important determinants of change in a focal variable. The parameters labeled  $D_{C^*P}$  and  $D_{P^*C}$  are the most germane to our hypotheses because they allow us to draw conclusions about whether one variable is a leading indicator of another; a significant  $D_{C^*P}$  in the absence of a significant  $D_{P^*C}$  parameter would allow us to conclude that changes in blame precede and drive changes in PTSD, and the opposite pattern would lead to the opposite conclusion.

After establishing scalar invariance for all three measures, an iterative series of univariate models were estimated separately to determine the best representation of each construct prior to estimating bivariate models. Using the best-fitting univariate models, we then tested a series of bivariate models that sequentially allowed for increased coupling across constructs. -2LL tests were used to compare nested models, and Information Criteria were used to compare non-nested models. Model fit was deemed acceptable given comparative fit index (CFI) values greater than .95 (Hu & Bentler, 1999). Root mean square error of approximation (RMSEA) tends to be biased in models with small degrees of freedom (a problem common in longitudinal models), so it was used only as a secondary indicator of model fit. Possible clustering effects associated with treatment in a group format were not included in the models because the primary outcome papers for both parent studies (Resick, Wachen, et al., 2017; Resick et al., 2015) examined them in preliminary analyses. Both reported those effects were negligible and dropped them from the final analyses. We also tested moderation effects of both study and treatment modality by running separate multiple groups models on the retained parameters in the final selected bivariate models. We found no evidence of moderation by study, and were unable to fully examine moderation due to treatment modality due to an unresolvable nonpositive definite matrix for one of the groups. Analyses were conducted using Mplus version 7.3 (Muthén & Muthén, 1998–2012) using the full information maximum likelihood estimator, which accounts for missing data by using all available data and borrowing information about the correlation between variables in complete cases to produce the most likely estimates of the parameters of interest.

#### Results

## ATTRITION ANALYSIS

An attrition analysis was conducted to determine whether treatment completers and noncompleters differed on the demographic variables presented in Table 1 as well as main study outcomes, with dropout status coded at the final treatment session (Week 6, n = 175). Compared to participants who dropped out of treatment, those who remained were older,  $M_{age} = 31.23$  vs. 34.57, t(319) =4.137, p < .001, but they did not differ on any other demographic variables. Consequently, age was controlled for in the univariate and bivariate models we examined, but doing so did not affect any substantive conclusions, so it was subsequently dropped from the models discussed hereafter. Completers versus noncompleters did not differ in terms of PCL-S total scores or on CERQ self-blame or other-blame scores at any of the seven assessments.

## INITIAL ANALYSES

Table 1 includes the observed sample means and standard deviations for each outcome variable at each time point. Investigation of the individual-level data demonstrated wide variability in the individual participant trajectory plots over time. At the aggregated group level, PCL-S total scores appear to have decreased steadily across time. CERQ selfblame scores appeared to decrease only slightly from baseline to the last treatment assessment, but that appears to be because they increased significantly between the baseline and Week 1 assessments. Self-blame scores decreased steadily thereafter and dropped to subbaseline levels over the course of treatment (Week 1-Week 6). CERQ other-blame scores similarly increased between the baseline and Week 1 assessments, but otherwise changed very little over the course of treatment. In fact, on average, Week 1 and Week 6 other-blame scores were essentially the same.

Table 2

Model Parameters From Final Univariate and Bivariate Latent Difference Score Models Including Self-Blame

		Univariate models	Raw metric bivariate model	Common metric bivariate model
Fixed effects	Parameter	Unstandardized co		
Predicting changes in PCL-S scores				
Prior levels → subsequent changes (within construct)	A <sub>PCL-S</sub>	-	_	_
Prior changes → subsequent changes (within construct)	B <sub>PCL-S</sub>	-	-	-
Constant change (within construct)	C <sub>PCL-S</sub>	-1.58***	-1.47***	-0.09***
Prior changes $\rightarrow$ subsequent changes (across construct)				
Early treatment (baseline–Week 3)	D <sub>C*P</sub>		1.43***	0.17***
Late treatment (Week 4–Week 6)	D <sub>C*P</sub>		5.28**	0.62**
Prior levels → subsequent changes (across construct)			-	-
Early treatment (baseline–Week 2)	E <sub>C*P</sub>			
Middle treatment (Week 3–Week 4)	E <sub>C*P</sub>		-	-
Late treatment (Week 5–Week 6)	E <sub>C*P</sub>		-	-
Predicting changes in CERQ Self-Blame scores				
Prior levels → subsequent changes (within construct)	A <sub>CERQ</sub>	-0.34***	42***	42***
Prior changes → subsequent changes (within construct)	B <sub>CERQ</sub>	0.59***	-0.06	-0.06
Constant change (within construct)	C <sub>CERQ</sub>	1.35***	4.15***	2.07***
Prior changes → subsequent changes (across construct)				
Early treatment (baseline–Week 3)	D <sub>P*C</sub>		0.26***	2.19***
Late treatment (Week 4–Week 6)	D <sub>P*C</sub>		0.26***	2.19***
Prior levels → subsequent changes (across construct)				
Early treatment (baseline–Week 2)	E <sub>P*C</sub>		037**	31**
Middle treatment (Week 3–Week 4)	E <sub>P*C</sub>		-0.042**	-0.35**
Late treatment (Week 5–Week 6)	E <sub>P*C</sub>		-0.046***	-0.39***

*Note.* Parameters marked with "--" were not estimated in final selected model. PCL-S = PTSD Checklist—Stressor-Specific Version; A = effect of prior level of subscripted variable on its subsequent changes; B = effect of prior changes in subscripted variable on its subsequent changes; C = constant change over time in subscripted variable;  $D_{C^*P}$  = effect of prior changes in self-blame on later changes in PTSD; CERQ = Cognitive Emotion Regulation Questionnaire—Short Form. \* p < .05, \*\* p < .01, \*\*\* p < .001.

## CHANGE SCORE MODELING

Results from the best-fitting univariate and bivariate models discussed hereafter are displayed in Table 2 for models including self-blame and Table 3 for models including other-blame. To demonstrate the concordance between the models and the observed data, plots of the actual mean scores over time versus the mean scores estimated by univariate and bivariate models, including selfblame and PTSD severity, are presented in Figure 2. The same outcomes are plotted in Figure 3 for the models, including other-blame. These graphs show that the vast majority of the univariate and bivariate models discussed hereafter reproduce the data well.

#### Univariate Models

We first examined separate univariate models for the two CERQ subscales and the PCL-S, using only the within-construct parameters described earlier, to determine the best representation of changes in each construct prior to estimating bivariate models. For each variable, we specified a proportional change model (including only the parameters labeled A in Figure 1), a constant change model (including only the C parameters), a dual-change model (including both the A and C parameters), and a changes to changes model (including the A, B, and C parameters). Additonal models examining quadratic trends for both self-blame and otherblame were also examined given the shape of their observed data trajectories. Parameters associated with these models are presented under the heading "Univariate Models" in Tables 2 and 3.

At the univariate level, changes in PCL-S total scores were best represented by the constant change model, CFI = .94, RMSEA (90% CI) = .10 [.08, .12]. This model predicted a constant 1.58-point decrease in PCL-S scores at each time point, and the resulting predicted univariate trajectory reproduced the actual means fairly well (see Figure 2), considering the latter followed a relatively linear pattern of decline. Changes in self-blame were best represented by a changes to changes model, CFI = .89, RMSEA (90% CI) = .15 [.13, .17], and were predicted by the constant change component (C<sub>CERQ</sub> = 1.35, p < .001), by prior levels of self-blame (A<sub>CERO</sub> = -0.34, p < .001), and by prior

Tab	le	3
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Model Parameters From Final Univariate and Bivariate Latent Difference Score Models Including Other-Blame

		Univariate models	Raw metric bivariate model	Common metric bivariate model	
Fixed effects	Parameter	Unstandardized coefficients			
Predicting changes in PCL-S scores					
Prior levels → subsequent changes (within construct)	A <sub>PCL-S</sub>	_	_	_	
Prior changes → subsequent changes (within construct)	B <sub>PCL-S</sub>	_	_	_	
Constant change (within construct)	C <sub>PCL-S</sub>	-1.58***	-1.57***	-0.09***	
Prior changes → subsequent changes (across construct)	D <sub>C*P</sub>		_	_	
Prior levels $\rightarrow$ subsequent changes (across construct)	E <sub>C*P</sub>		-	-	
Predicting changes in CERQ Self-Blame scores					
Prior levels → subsequent changes (within construct)	A <sub>CERQ</sub>	-0.34***	70***	70***	
Prior changes → subsequent changes (within construct)	B <sub>CERQ</sub>	_	_	_	
Constant change (within construct)	C <sub>CERQ</sub>	1.35***	2.17**	1.09**	
Prior changes → subsequent changes (across construct)	D <sub>P*C</sub>		_	_	
Prior levels → subsequent changes (across construct)	E <sub>P*C</sub>		0.03**	0.27**	

*Note.* Parameters marked with "--" were not estimated in final selected model. PCL-S = PTSD Checklist—Stressor-Specific Version; A = effect of prior level of subscripted variable on its subsequent changes; B = effect of prior changes in subscripted variable on its subsequent changes; C = constant change over time in subscripted variable;  $D_{C^*P}$  = effect of prior changes in other-blame on later changes in PTSD;  $E_{C^*P}$  = effect of prior level in other-blame on later changes in PTSD; CERQ = Cognitive Emotion Regulation Questionnaire—Short Form. \* p < .05, \*\* p < .01, \*\*\* p < .001.

changes in self-blame ( $B_{CERQ} = 0.59$ , p < .001). Though this univariate model did not fit the data particularly well (see Figure 2), we elected to include it in bivariate models given the possibility that prediction of changes in self-blame could be enhanced by the addition of the coupling parameters. If self-blame and PTSD severity are dynamically related (i.e., if prior levels of and/or prior changes in PTSD severity are important predictors of changes in self-blame), then this univariate model would not predict changes in self-blame very well. The addition of the coupling parameters, if they are important determinants of subsequent changes in self-blame, can dramatically improve model fit (Grimm et al., 2012). Changes in other-blame were best represented by the dual-change model, CFI = .93, RMSEA (90% CI) = .08 [.06, .10], and were predicted by the constant change component  $(C_{CERO} = 4.35, p < .001)$  and by prior levels of other-blame (A<sub>CERO</sub> = -0.79, p < .001); together these parameters predicted a trajectory that increased after the first session and stayed relatively flat thereafter, which is largely consistent with the observed means (see Figure 3).

#### Self-Blame/PTSD Bivariate Model

Beginning with the univariate models described above as baseline specifications, we then tested a series of bivariate models linking the PCL-S scores to each of the CERQ subscales. In these models, we iteratively allowed for increased coupling across constructs, starting with the prior level predictors ( $E_{C^*P}$ , then  $E_{P^*C}$ ), followed by the prior changes predictors ( $D_{C^*P}$ , then  $D_{P^*C}$ ), the latter being the parameters of most interest to our hypotheses. After establishing which cross-coupling parameters were significant, we iteratively relaxed constraints on the prior level and prior change parameters to test whether they were (a) consistent across treatment, (b) different between the first and second half of treatment, or (c) different between the first, second, and final thirds of treatment.

The final selected self-blame/PTSD bivariate model fit the data well, CFI = .95, RMSEA (90%) CI) = .08 [.07, .09]; parameters from this model are presented in Table 2 under the heading "Raw Metric Bivariate Model," next to the parameters from the best-fitting univariate models for comparison. In the bivariate model (as in the univariate model), changes in PCL-S scores were predicted by a negative constant change parameter ( $C_{PCL-S}$  = -1.47, p < .001); however, they were also predicted by prior changes in CERQ self-blame scores, and these prior changes predicted larger PCL-S changes later in treatment (Week 4–Week 6:  $D_{C^*P} = 5.28$ , p = .003) than they did earlier in treatment (baseline–Week 3:  $D_{C^*P} = 1.43$ , p = .001)—that is, PCL-S scores decreased (improved) at each assessment, and larger prior changes (decreases) in self-blame were associated with larger subsequent decreases (improvements) in PTSD severity. This provides strong evidence that earlier changes in selfblame precede and drive later changes in PCL-S scores. As can be seen in the upper graph in Figure 2, the estimated means based on the bivariate model mirror the observed means even better than the means in the univariate model, indicating the usefulness of including prior changes in self-blame in the model of changes in PCL-S changes. Overall, PCL-S scores dropped 10.3 points on average, a standardized effect size using the baseline standard deviation (Cohen's d) of 0.99, which is considered a large effect.

With respect to the other half of the final selected bivariate model, changes in CERQ self-blame scores were predicted by the constant change parameter ( $C_{CERQ} = 4.15$ , p < .001), prior levels of self-blame ( $A_{CERQ} = -0.42$ , p < .001), prior changes in PCL-S total scores ( $D_{P^*C} = 0.26$ , p < .001), and prior levels of PCL-S scores, an effect that varied over the course of treatment ( $E_{P^*C} = -0.37$ , -0.42, -0.46 during the first, second, and final thirds of treatment, respectively). Higher prior levels of both self-blame and PTSD were associated with larger subsequent improvements in self-blame, as were larger prior changes in PTSD. The largest week-to-week change in selfblame was an increase that occurred after the first treatment session, after which self-blame steadily decreased in a fairly linear manner. Relative to baseline, the overall decrease was small (-0.25,Cohen's d = -0.12). The presence of both significant cross-coupling parameters  $(D_{P^*C} \text{ and } D_{C^*P})$ indicates that self-blame and PCL-S total scores are dynamically linked; changes in PCL-S total scores are driven by prior changes in self-blame, but changes in self-blame themselves are at least partially driven by both prior levels of and prior changes in the PCL-S total scores. As shown in the bottom graph in Figure 2, the bivariate model's estimated means are nearly identical to the observed data, a substantial improvement over the univariate model's estimated means, indicating the value of modeling these two constructs as a dynamic system.

To draw inferences about the relative size of effects of the bivariate coupling parameters in this



FIGURE 2 Actual/observed means, estimated univariate model means, and estimated means from final bivariate model linking PCL-S total scores to CERQ Self-Blame scores. *Note.* PCL-S = PTSD Checklist—Stressor-Specific Version; CERQ = Cognitive Emotion Regulation Questionnaire—Short Form



FIGURE 3 Actual/observed means, estimated univariate model means, and estimated means from final bivariate model linking PCL-S total scores to CERQ Other-Blame scores. Note. PCL-S = PTSD Checklist—Stressor-Specific Version; CERQ = Cognitive Emotion Regulation Questionnaire—Short Form

reciprocal relationship, we divided the PCL-S and CERO self-blame scores at each assessment by the number of items in each respective measure so that they would be on the same scale and reran the analysis. The resulting model, presented in Table 2 under the heading "Common Metric Bivariate Model," fits the data identically, but has the advantage of making the unstandardized parameters directly comparable with one another. Comparing the prior change coupling parameters (D<sub>P\*C</sub> and  $D_{C^*P}$  shows that the effect of prior changes in PTSD on later changes in self-blame are 12.88 and 3.53 times larger during the first and second half of treatment, respectively, than the effect of prior changes in self-blame on later changes in PTSD. An additional model constraining the sizes of D<sub>P\*C</sub> and  $D_{C^*P}$  to be equal across all time points resulted in poorer model fit,  $\chi^2$  *difference* (2) = 9.74, p = .007, indicating that the observed differences in the size of those parameters was significant.

#### Other-Blame/PTSD Bivariate Model

The final selected other-blame/PTSD severity bivariate model fit the data very well, CFI = .94, RMSEA (90%) CI) = .07 [.06, .08], and reproduced the observed means at least as well as each construct's univariate model (see Figure 3). Parameters from the raw metric and the common metric bivariate models are listed in Table 3. Changes in PTSD severity were predicted only by the constant change parameter ( $C_{PCL-S}$  = -1.57, p < .001), which had nearly the same estimate as the univariate model of changes in PCL-S scores. Changes in CERQ other-blame scores were predicted by the constant change parameter ( $C_{CERO} = 2.17$ , p = .006), prior levels of other-blame (A<sub>CERQ</sub> = -0.70, p < .001), and prior levels of PCL-S scores, an effect that did not vary over the course of treatment  $(E_{P^*C} = 0.32, p = .002)$ . Though prior levels of PCL-S scores significantly predicted changes in other-blame, it does not appear that its inclusion in the model dramatically improved prediction of the observed data relative to the univariate model (see Figure 3) that is, its effect was very small. In fact, according to the common metric model, the effect of prior levels of other-blame on subsequent changes in other-blame was 2.6 times larger than the effect of prior levels of PTSD severity.

#### Discussion

Studies examining the temporal course of changes in cognitions and PTSD symptoms are essential to understanding the dynamic processes that occur during treatment. While previous studies have examined session-by-session changes in traumarelated cognitions during PE (Kumpula et al., 2017; Zalta et al., 2014) and CT-PTSD (Kleim et al., 2013), as well as pre-, mid-, and posttreatment changes in CPT (Schumm et al., 2015), this was the first study to examine weekly change during CPT. Futhermore, the present study built on earlier research by including assessment of other-blame and using a sample of active military personnel. We hypothesized that, over the course of treatment, there would be reductions in blame and PTSD symptoms. Furthermore, we predicted that reductions in blame would precede and predict subsequent changes in PTSD, whereas the reverse relationships would not be significant.

As expected, we found that PTSD symptoms reduced over the course of CPT, although scores decreased at different rates over the course of treatment. The difference in the rate of PTSD improvement over therapy is explained by the predicted model, which indicated that change in PCL-S over treatment was a combination of two effects. The linear decrease in PCL-S scores represented a *direct* effect of treatment on PTSD symptoms. The model also predicted an *indirect* effect of prior changes in self-blame. Reduction in PTSD symptom severity ratings was the sum of these two components. After the first session, selfblame *worsened*, reducing the amount of overall PTSD symptom severity improvement. During the second half of treatment, self-blame steadily improved, adding to reductions in PTSD symptom severity.

Both self- and other-blame increased initially. This may be explained by the fact that, in the early stages of treatment, there is a focus on identification of blame cognitions. For many, this is contrary to their avoidance strategies and may be the first time that they have confronted these cognitions. After an initial increase, self- and other-blame had different trajectories over the course of treatment. Self-blame scores peaked after the first treatment session, after the assignment to write about the causes and consequences of the worst traumatic event, and then decreased steadily to subbaseline levels by the end of treatment, a change that was moderate in size. Other-blame increased early in treatment, but then changed very little later in treatment.

Taken together, these findings highlight that changes in blame and PTSD symptoms are not uniform during treatment, demonstrating the importance of examining weekly changes over the course of treatment. Such week-by-week information is informative to researchers and clinicians and illustrates that change during CPT does not necessarily occur as a linear reduction in symptoms, consistent with observations of nonlinear change across other treatments (e.g., Hayes, Laurenceau, Feldman, Strauss, & Cardaciotto, 2007). Recognition of variable change patterns during CPT might help clinicians better conceptualize their patients' progress. For example, if clinicans expect gradual reductions in self-blame over the course of CPT, they might think that initiating treatment was a mistake if they observe increases in self-blame initially. However, initial increases in self-blame may reflect increased self-awareness and not be an indication that treatment is not working or should be abandoned. We note that the initial increases in self-blame were followed by steady decreases, and those changes predicted subsequent improvements in PTSD symptom severity.

To our knowledge, this is the first study that has examined other-blame over the course of CPT, so it is difficult to discern the reason for the observed lack of change. One possible explanation relates to measurement. It was not possible to track at whom the other-blame was directed over time, and the measure did not differentiate erroneous from accurate blame. Therefore, the measure may not have adequately captured changes in other-blame (e.g., shifts from blaming one person to another). Another possibility is that the CPT treatment provided in this study did not adequately address other-blame. Patients in this study received a set number of sessions, and many received treatment in a group format. It may be that the active duty military sample needed more sessions or modified approaches to adequately address their otherblame.

Finally, our primary question was whether changes in blame would predict subsequent changes in PTSD symptoms. As hypothesized, we found that changes in PTSD symptoms were predicted by prior changes in self-blame; however, contrary to our predictions, we also found that changes in selfblame were predicted by both prior levels of and prior changes in PTSD symptoms. These results suggest that PTSD symptoms and self-blame cognitions are dynamically linked and reciprocal over the course of CPT treatment. Furthermore, when self-blame and PTSD symptoms were put on the same scale, the effect of prior changes in PTSD symptoms on self-blame was larger than the effect of prior changes in self-blame on PTSD. This finding diverges from earlier findings indicating that changes in cognitions predict PTSD symptom change and not vice versa (e.g., Kleim et al., 2013, Zalta et al., 2014). However, previous studies used the PTCI, which includes cognitions about the self and world as well as self-blame, while our measure focused solely on blame. Perhaps other dysfunctional cognitions reflected in the PTCI are responsible for the change in PTSD and are not reciprocal. While CPT theory emphasizes the role of reducing erroneous blame cognitions to reduce PTSD symptoms, these findings highlight that the relationship between self-blame cognitions and PTSD symptom change may be more complex. Changes in selfblame cognitions appear centrally important in the reduction of PTSD symptoms, but the relationship is more dynamic than hypothesized.

We also hypothesized that changes in PTSD symptoms would be predicted by changes in otherblame, and not vice versa. The former hypothesis was not supported, while the latter was. The inclusion of prior changes in other-blame to the model did not improve the prediction of changes in PTSD symptoms over time, nor did inclusion of prior changes in PTSD symptoms improve prediction of changes in other-blame. However, when predicting changes in other-blame, prior levels of PTSD symptoms had a significant, yet small, effect. Overall, the addition of either variable (PTSD symptoms or other-blame) did not dramatically improve the prediction of the other variable, suggesting that they are either not or minimally dynamically related over time as assessed in this study.

Future research is needed to replicate these results, and this study is not without limitations. One limitation is the use of the CERQ rather than the more commonly used PTCI (Foa et al., 1999). This difference makes it difficult to determine whether the observed findings differ from previous findings due to the use of a different measure or from differences between active duty military personnel versus civilians and veterans. The CERQ had the advantage of being a brief measure appropriate for weekly use, and assessing both selfand other-blame cognitions, which are not captured in the other measures-however, the CERO was not created specifically as a trauma-related measure. Another limitation is that the CERO measure used in this study included two-item subscales, which are not ideal from a measurement perspective. Furthermore, there was shared method variance due to the reliance on patient self-report measures for both PTSD symptoms and blame ratings. The use of weekly assessments of PTSD symptoms and blame cognitions meant that these measures were administered at every other treatment session because treatment was provided twice per week. It is possible that the results may have differed if we had assessed these variables at every session. Future research should reexamine the question of the dynamic relationship between blame and PTSD symptoms session by session during CPT using other measures and methods of measurement.

This study is also limited by the fact that there was a 54.5% attrition rate, which means that there were many participants who had missing data. Unlike research with civilian and veteran samples, military samples may face deployments, trainings, moves to new bases, or discharge during treatment, so dropout is sometimes not voluntary on the part of the participant. Importantly, attrition analyses did not find evidence of any significant differences between completers and noncompleters other than age, which did not alter results when entered as a covariate. Finally, we were unable to examine whether these results were moderated by treatment modality due to model estimation/convergence issues. It is possible that individual treatment could be tailored more flexibly to address specific blame cognitions, which could lead to different patterns of predictive relationships in the cross-lagged parameters. On the other hand, it is plausible that some members in the group format vicariously gained insight into cognitions that they were unwilling to share due to feelings of stigma or shame when other members of the group brought up similar cognitions. Future research should investigate these issues.

## CONCLUSIONS

The current findings suggest that, over the course of CPT, self-blame and PTSD symptoms are dynamically linked: Prior changes in self-blame cognitions predicted subsequent changes in PTSD symptoms and vice versa. Furthermore, prior changes in PTSD symptoms had a larger effect on changes in self-blame than the reverse. Changes in PTSD symptoms were not predicted by changes in other-blame, but changes in other-blame were predicted by prior levels of PTSD. These findings support and extend the theoretical underpinnings of CPT and emphasize both the clinical importance of identifying and targeting self-blame cognitions in treatment, and the importance of symptom change in facilitating cognitive change.

Conflict of Interest Statement

The authors declare no conflicts of interest.

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RECEIVED: January 2, 2019 Accepted: June 20, 2019 Available online: 28 June 2019