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Changes in trauma-related cognitions predict subsequent symptom improvement during prolonged exposure in patients with childhood abuse-related PTSD

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ABSTRACT

Change in negative posttraumatic cognitions is a proposed mechanism through which Prolonged Exposure (PE) leads to symptom reduction of posttraumatic stress disorder (PTSD). A strong case for posttraumatic cognitions as a change mechanism in PTSD treatment can be made by establishing temporal precedence of change in cognitions. The current study examines the temporal relationship between change in posttraumatic cognitions and PTSD symptoms during PE, using the Posttraumatic Cognitions Inventory. Patients with DSM-5 defined PTSD following childhood abuse (N = 83) received a maximum of 14–16 sessions of PE. Clinician-rated PTSD symptom severity and posttraumatic cognitions were assessed at baseline, week 4, 8, and 16 (post-treatment). Using time-lagged mixed effect regression models, we found that posttraumatic cognitions predicted subsequent PTSD symptom improvement. Notably, when using the items of an abbreviated version of the PTCI (PTCI-9), we found a mutual relationship between posttraumatic cognitions and PTSD symptom improvement. Crucially, the effect of change in cognitions on PTSD symptom change was greater than the reverse effect. The current findings corroborate change in posttraumatic cognitions as a change process during PE, but cognitions and symptoms cannot be completely separated. The PTCI-9 is a short instrument that appears suitable to track cognitive change over time.

1. Introduction

Negative posttraumatic cognitions have a central position in theoretical models of the development and maintenance of posttraumatic stress disorder (PTSD; Ehlers & Clark, 2000; Rauch & Foa, 2006). Negative cognitions about the self (e.g., "I am weak" or "I am inadequate") and the world ("The world is a dangerous place") are thought to induce a sense of current threat, accompanied by intrusions, heightened arousal, and other emotions. Strategies to control this sense of threat (such as suppression of thoughts and feelings or avoiding situations or places) may alleviate symptoms in the short term but maintain negative cognitions, and thereby PTSD symptoms, in the long term. Many empirical studies have shown that negative posttraumatic cognitions are positively related to PTSD symptom severity (see for a meta-analysis Gómez de La Cuesta et al., 2019). Furthermore, prospective studies have indicated that negative posttraumatic cognitions predict later PTSD symptom severity (Dekel et al., 2013; Dunmore et al., 2001; Ehring et al., 2008; Shahar et al., 2013). Interestingly, one of the changes in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) was the inclusion of 'persistent negative beliefs about oneself, others or the world' as a symptom criterion (Friedman, 2013). Recent network studies

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provide preliminary evidence that negative alterations in cognitions and mood form a central symptom cluster of PTSD which has strong connections to the other PTSD symptoms, such as re-experiencing or avoidance symptoms (Bartels et al., 2019; McBride et al., 2020). Given these findings, negative posttraumatic cognitions are important targets in the treatment of PTSD.

Prolonged exposure therapy (PE) is one of the treatments of choice for PTSD (Lewis et al., 2020; Mavranezouli et al., 2020; McLean et al., 2022). During PE, patients are repeatedly confronted with trauma-related stimuli they typically avoid, both trauma-related memories (imaginal exposure) and trauma-related objects and situations (in vivo exposure). Although the effectiveness of PE has been well established, less is known about the mechanisms that drive symptom reduction. Identifying these processes (i.e., mechanisms of change) may inform efforts to optimize treatment efficacy. Change in negative posttraumatic cognitions has been proposed to be a mechanism of change for a range of evidence-based trauma-focused treatments, including PE (Cooper, Clifton, & Feeny, 2017; Kangaslampi & Peltonen, 2022; Sripada et al., 2016; Zalta, 2015). Many studies have shown that PE reduces negative trauma-related cognitions and concurrently improves PTSD symptoms (see for a review, Brown et al., 2019).

If negative trauma-related cognitions are indeed a change mechanism during PE, cognitions and symptoms need to not only be related, but cognitive change needs to precede symptom change (Kazdin, 2007; Sripada et al., 2016). Establishing a timeline requires repeated assessments of the proposed mechanism and the treatment outcome measures; only then can temporal precedence be determined. The evidence for the temporal sequence of change in posttraumatic cognitions and symptoms during PE is not unequivocal. Some studies found that PTSD symptom alleviation was preceded by reductions in negative trauma-related cognitions and not vice versa (Cooper, Zoellner, et al., 2017; McLean, Yeh, et al., 2015; Zalta et al., 2014), but other studies found that cognitions and symptoms mutually influenced each other (Kumpula et al., 2017; McLean, Su, & Foa, 2015; Rauch et al., 2021). Various treatments were given alongside or in comparison with PE. Populations of these studies also differed (e.g., military veterans with PTSD, women with PTSD). However, none of these differences seem to be able to explain these discrepant findings and possible causes for these discrepancies should be further investigated.

All of the previously mentioned studies on the temporal relationship between posttraumatic cognitions and symptoms during PE used the Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999) to assess negative trauma-related cognitions. With 36 items, the PTCI may be considered a lengthy measure which is inconvenient for repeated assessments due to higher patient and therapist/researcher burden. Recently, a shorter version of the PTCI consisting of 9 items (PTCI-9; Wells et al., 2019) was developed and its psychometric properties were tested within different samples, such as trauma-exposed undergraduates, military veterans, and female civilians (Wells et al., 2019; Whiteman et al., 2020). Although the PTCI-9 appears to be a valid and reliable measure, it has not yet been used to track cognitive changes during treatment. As noted above, a shorter measure may be especially useful in the context of repeated assessment during treatment, as the burden for tracking these cognitions is lower, whilst still giving clinicians insights into the content and change of specific cognitions.

The aim of the present study was to examine the temporal relationship between changes in negative trauma-related cognitions and PTSD symptom change during PE in patients with PTSD related to childhood abuse. All prior studies in this field have investigated the relationship between PTCI changes and DSM-IV defined PTSD. In the current study, participants met DSM-5 defined PTSD criteria, and the Clinician Administered PTSD Scale for DSM-5 (CAPS-5; Boeschoten et al., 2018) was used for the assessment of symptom change. We expected that a decrease in trauma-related cognitions would lead to a subsequent decrease in clinician-rated PTSD symptoms but not vice versa. Additionally, we carried out the same analyses but instead used the nine items of the short version of the PTCI (PTCI-9; Wells et al., 2019), derived from the full PTCI. We expected the same pattern of findings. Furthermore, we carried out a sensitivity analysis in which we repeated the main analysis, but now after excluding the symptom cluster 'negative changes in cognitions and mood' from the CAPS-5 total score. Given that the conceptualization of PTSD has been broadened in the DSM-5 to include this criterion, this sensitivity analysis tests whether any relationship between trauma-related cognitions and PTSD symptom change holds when the conceptual overlap is minimized.

2. Methods

2.1. Design

We use data from the IMPACT study, a multicenter randomized controlled trial that compared PE, intensive PE (iPE), and phase-based therapy (PBT) in a sample of childhood abuse-related posttraumatic stress disorder (CA-PTSD). The study was approved by the Medical Ethical Committee of Leiden University Medical Center (NL57984.058.16) and the study trial is registered at the ClinicalTrials.gov registry, number NCT03194113. For more details about the design and the main outcomes, we refer to the published study protocol and the primary outcome paper (Oprel et al., 2018, 2021).

2.2. Participants

All participants met the following inclusion criteria: (1) age 18–65, (2) diagnosis of PTSD established with the Clinician Administered PTSD Scale for DSM-5 (CAPS-5; Boeschoten et al., 2018) with at least moderate severity of PTSD symptoms (CAPS-5 score $\geq$ 26), and at least one specific memory of the traumatic event, (3) an index traumatic event related to sexual abuse and/or physical abuse that occurred before the age of 18 years old and was committed by a primary caretaker or an authority figure and (4) proficiency in the Dutch language. Exclusion criteria were: (1) involvement in a compensation case or legal procedures concerning admission or stay in The Netherlands, (2) pregnancy, (3) severe non-suicidal self-injury (NSSI) which required hospitalization during the past three months, (4) severe suicidal behavior: a suicide attempt during the past three months or acute suicidal ideations with serious intent to die with a specific plan for suicide and preparatory acts, (5) severe disorder in the use of alcohol or drugs in last three months, (6) cognitive impairment (estimated IQ $<$ 70), (7) changes in psychotropic medication in the two months before inclusion, and (8) engagement in any current psychological treatment. For the current paper, we only used data from participants who were randomly allocated to the PE or iPE condition (n = 99, see also (Hoeboer et al., 2022). Participants in these conditions completed measures of trauma-related cognitions and PTSD symptoms at multiple timepoints during PE treatment, allowing us to study temporal change. Although the PBT condition also included PE, trauma-related cognitions and PTSD symptoms were only assessed once during PE. This precludes the study of temporality of cognitions and symptoms during PE within this condition. Therefore our primary analyses were conducted on the PE and iPE conditions. Moreover, participants who only had data on one measurement (i.e., only a baseline measurement [T0]; n = 17) were excluded from the analysis.

The final sample of our primary analyses consisted of 83 participants (18 male, 64 female, 1 other) between the ages of 20 and 60 years (M = 36.4, SD = 11.4). Thirty-two participants (61.4%) had at least one parent who was born in a non-Western country. The mean duration of PTSD was 16.0 years (SD = 12.1). Regarding the traumatic events, 63 participants (75.9%) experienced childhood sexual abuse, 50 participants (60.2%) experienced childhood physical abuse, 19 participants (22.9%) experienced adulthood sexual abuse and 25 participants (30.1%) experienced adulthood physical abuse. Forty-three (51.8%) were randomized to the PE condition and 40 participants (48.2%) to the iPE condition. For a complete description of the sample, see Oprel et al.

(2021).

2.3. Measures

2.3.1. Posttraumatic cognitions

Posttraumatic cognitions were measured with the Dutch translation of the Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999; Van Emmerik et al., 2006). The PTCI is a 36-item, self-report questionnaire, and each item is rated on a 7-point Likert scale that ranges from 1 (*totally agree*) to 7 (*totally disagree*). For the total score of the PTCI, sum scores of the answers to 33 items are used and scores range from 33 to 231. The PTCI was shown to have good internal reliability, test-retest reliability, and strong convergent and discriminant validity (Foa et al., 1999, van Emmerik et al., 2006). Evidence in favor of the three-factor structure of the PTCI (i.e., negative cognitions about the self, world, and self-blame) has been inconsistent, which questions the validity of these subscales (Beck et al., 2004; Hyland et al., 2015; Sexton et al., 2018; Whiteman et al., 2020). Therefore, we decided to use total scores only in the current study. Internal consistency of the PTCI in the current sample was good, both at baseline (Cronbach's $\alpha = 0.95$, McDonald's $\omega = 0.95$), and after four weeks (Cronbach's $\alpha = 0.96$, McDonald's $\omega = 0.97$), eight weeks (Cronbach's $\alpha = 0.97$, McDonald's $\omega = 0.97$), and 16 weeks (Cronbach's $\alpha = 0.97$, McDonald's $\omega = 0.97$).

We also calculated PTCI-9 total scores (Wells et al., 2019). The PTCI-9 consists of items 1, 7, 22, 23, 25, 27, 31, 33, and 36 of the PTCI. Item selection for the PTCI-9 was, among other things, based on the highest loading items within each subscale (Wells et al., 2019). PTCI-9 total scores are calculated by taking the mean of these nine items (vs. the sum in the original PTCI), with total scores ranging from 1 to 7. The PTCI-9 was not administered separately but calculated from the full version of the PTCI. The 33-item and the 9-item PTCI total scores at baseline (T0) had a strong, significant correlation ($r = 0.94$, $p < .001$). We also calculated the correlation between the PTCI-9 and the original PTCI without the nine items that were included in the PTCI, to control for the item overlap. This correlation was also strong and significant ($r = 0.89$, $p < .001$). Over the different timepoints, the strength of these correlations increased slightly. Internal consistency of the PTCI-9 in our sample was good, both at baseline (Cronbach's $\alpha = 0.83$, McDonald's $\omega = 0.84$), after four weeks (Cronbach's $\alpha = 0.88$, McDonald's $\omega = 0.87$), eight weeks (Cronbach's $\alpha = 0.88$, McDonald's's $\omega = 0.88$), and 16 weeks (Cronbach's $\alpha = 0.90$, McDonald's $\omega = 0.90$).

2.3.2. PTSD symptoms

The severity of PTSD symptoms was measured with the Dutch version of the Clinician Administered PTSD Scale for DSM-5 (CAPS-5; Boeschoten et al., 2018). The CAPS-5 is a 20-item clinical interview covering DSM-5 PTSD diagnostic criteria and PTSD symptom severity during the past week. Scores range from 0 to 80, where lower scores indicate lower PTSD symptom severity. The internal consistency of the CAPS-5 total score at baseline in the current study was moderately high, Cronbach's $\alpha = 0.75$. We also calculated a CAPS-5 score that excluded symptoms referring to negative posttraumatic cognitions (i.e., CAPS-5 total score – CAPS-5 item 9 and item 10), hereafter referred to as CAPS-$5^{excl9\text{-}10}$.

2.4. Procedure

Participants were recruited in two outpatient units specialized in the treatment of trauma-related disorders. Potential participants had to complete a baseline assessment in which they received detailed information about the treatment and in which eligibility was checked. Eligible participants were then randomized (1:1:1) to receive PE, iPE, or PBT.

In the PE condition, participants received 16 weekly sessions of 90 min of PE. In the iPE condition, participants received three 90-min PE sessions per week over four weeks (12 sessions in total), followed by two monthly booster sessions. Sessions in the iPE condition were alternately provided by two therapists for practical reasons. The same treatment manual was used for PE and iPE. The manual was based on the protocol by Foa et al. (2007). The first session consisted of psychoeducation on PTSD and the construction of a case conceptualization. The second to the last session consisted of imaginal exposure (repeatedly recounting the traumatic event) and exposure in vivo (repeatedly approaching trauma-related stimuli). As homework assignment, participants were instructed to listen to audiotapes of the imaginal exposure and to complete in vivo assignments during the week. All therapists in the study had to complete PE training and pass an exam with pilot patients. Therapists also received weekly group supervision (supervisors: RAdK and AvM). Independent observers rated a random selection of the PE sessions (135 sessions; ~10% of all PE sessions) on treatment adherence based on the Dutch translation of the original adherence rater checklist scale. Protocol adherence was high (Mean session elements completed = 90%, *SD* = 18%). Measurements of the CAPS-5 and PTCI took place at baseline (T0), after four weeks (T1), after eight weeks (T2), and after 16 weeks (T3). At T3, data on the PTCI was available for 62 participants, and data on the CAPS-5 was available for 68 participants. The reasons for missed measures were diverse. Of the 21 participants who did not complete all measures, ten dropped out of therapy.

2.5. Statistical analyses

To increase comparability between the PTCI and PTCI-9 variables, we first standardized these scores. In the first analysis, we used a time-lagged mixed effect regression model with CAPS-5 scores (time point X) as the dependent variable and the autoregressive effect of CAPS-5 scores (time point X-1) and cross-lagged standardized PTCI scores (time point X-1) as the independent variables. In the second analysis, we used a time-lagged mixed effect regression model with standardized PTCI scores (time point X) as the dependent variable and the autoregressive effect of standardized PTCI scores (time point X-1) and cross-lagged CAPS-5 scores (time point X-1) as independent variables.

For the first sensitivity analysis, we carried out the same analyses, but now with the PTCI-9 instead of the PTCI. To control for conceptual overlap between the predictor and outcome variable, we also re-ran these analyses, but now excluding the symptoms referring to negative cognitions from the PTSD symptom total score (i.e., items 9 and 10 of the CAPS-5). Finally, we checked whether the effect of PTCI scores on subsequent PTSD symptom reduction was different between conditions by adding condition and the interaction between condition and cross-lagged PTCI as independent variables in the model. We checked this with a similar model for the reversed effect of PTSD symptoms on subsequent PTCI scores. All models were tested with maximum likelihood estimation using the lme4 package (v1.1-28; Bates et al., 2015) in R (Version 4.0.1). Maximum likelihood estimation can handle missing data and can take the effect of dropout into account. The alpha level was set at 0.05 (two-sided).

The data-analysis plan of this study was registered at OSF (Center for Open Science; Kooistra et al., 2023). Note that we had to change our statistical approach from a dynamic panel model to a mixed effect regression model due to convergence issues of the dynamic panel model. We have not made changes to the dependent and independent variables in the models.

Based on peer-review, we conducted additional post-hoc analyses including the PBT condition. All information related to these additional analyses is included in the supplemental file.

3. Results

3.1. Descriptives

We tested baseline differences between the participants who completed all measures ($n = 62$) and those who did not ($n = 21$). The

two groups did not significantly differ on age, $t(81) = 1.42$, $p = .160$, or gender, $\chi 2\ (2) = 3.13$, $p = .209$. The group who completed all measures scored significantly lower on the CAPS-5 at baseline ($M_{T0} = 39.5$, $SD_{T0} = 8.4$) compared to the group who did not ($M_{T0} = 44.4$, $SD_{T0} = 9.5$), $t(81) = -2.23$, $p = .028$. Furthermore, the group who completed all measures also scored significantly lower on the PTCI at baseline ($M_{T0} = 129.1$, $SD_{T0} = 36.6$) compared to the group who did not ($M_{T0} = 152.6$, $SD_{T0} = 40.1$), $t(81) = -2.47$, $p = .016$.

CAPS-5 scores decreased over the course of treatment ($M_{T0} = 40.7$, $SD_{T0} = 8.9$; $M_{T3} = 17.3$, $SD_{T3} = 15.5$). Scores of the PTCI ($M_{T0} = 135.1$, $SD_{T0} = 38.7$; $M_{T3} = 102.2$, $SD_{T3} = 44.6$) and items of the PTCI-9 ($M_{T0} = 3.8$, $SD_{T0} = 1.2$; $M_{T3} = 2.9$, $SD_{T3} = 1.4$) also decreased over the course of treatment. See Table 1 for more details.

3.2. Mixed regression models of posttraumatic cognitions and PTSD symptom change

The results of the mixed effect regression models can be found in Table 2. We first assessed the autoregressive effect of CAPS-5 and the cross-lagged effect of the standardized PTCI on CAPS-5. We found that both the auto-regressive effect, $b = 0.49$, $SE = 0.08$, $t = 6.28$, $p < .001$, Cohen's $d = 0.96$, and the cross-lagged effect, $b = 4.27$, $SE = 1.14$, $t = 3.75$, $p = < .001$, Cohen's $d = 0.70$, were significant. In other words, more negative posttraumatic cognitions at timepoint X-1 were related to a smaller reduction in PTSD symptoms at timepoint X. This effect did not differ for PE compared to iPE, $b = 0.27$, $SE = 0.83$, $t = 0.31$, $p = .750$. The reversed effect of CAPS-5 on the next measurement's PTCI was not significant, $b = 0.01$, $SE = 0.00$, $t = 1.61$, $p = .109$. This effect also did not differ for PE compared to iPE, $b = 0.04$, $SE = 0.09$, $t = 0.46$, $p = .647$.

3.3. Sensitivity analyses

Using the PTCI-9 as the independent variable, we found that the PTCI-9 significantly predicted subsequent CAPS-5 scores, $b = 4.30$, $SE = 1.02$, $t = 4.22$, $p < .001$, Cohen's $d = 0.79$. The reversed effect of CAPS-5 on the next measurement's PTCI-9 was also significant, albeit with a smaller effect size, $b = 0.01$, $SE = 0.00$, $t = 2.54$, $p = .012$, Cohen's $d = 0.39$.

Using the PTCI as the independent variable and CAPS-5$^{excl9\text{-}10}$ as the dependent variable, we found that the PTCI significantly predicted subsequent CAPS-5$^{excl9\text{-}10}$ scores, $b = 3.50$, $SE = 0.99$, $t = 3.52$, $p < .001$, Cohen's $d = 0.64$. The reverse effect, CAPS-5$^{excl9\text{-}10}$ predicting PTCI, was not significant, $b = 0.01$, $SE = 0.00$, $t = 1.72$, $p = .087$. The results of these sensitivity analyses can be found in Table 3.

4. Discussion

The present study aimed to assess the temporal relation between posttraumatic cognitions and PTSD symptom improvement during PE in patients with PTSD following childhood abuse. We primarily tested whether a change in trauma-related cognitions (full version PTCI) predicted subsequent DSM-5 defined PTSD symptom change (CAPS-5). Secondly, we tested whether a change in trauma-related cognitions, as measured with the items of an abbreviated version of the trauma-related cognitions questionnaire (PTCI-9), predicted PTSD symptom change. Our results indicate that the reduction of negative trauma-related cognitions indeed precedes PTSD symptom change. When using the items of the PTCI-9, we found a bidirectional relationship, but notably, the effect of cognitions on symptoms was greater than the reverse (i.e., the effect of symptoms on cognitions). The interpretation of our findings did not change when we controlled for the conceptual overlap between trauma-related cognitions and DSM-5 defined PTSD symptoms.

As hypothesized and in line with cognitive theories (Ehlers & Clark, 2000; Rauch & Foa, 2006), we found that posttraumatic cognitions predicted subsequent PTSD symptom reduction. This finding is in line with earlier work (Cooper, Zoellner, et al., 2017; Kumpula et al., 2017; McLean et al., 2015a, 2015b, 2019; Rauch et al., 2021; Zalta et al., 2014). When we used the full version of the PTCI to measure negative posttraumatic cognitions, we did not find the reversed effect, i.e., PTSD symptoms did not predict subsequent changes in cognitions. Some earlier work also found a unidirectional temporal relationship between PTSD cognitions and symptoms (Cooper, Zoellner, et al., 2017; McLean,

Table 1
Descriptive information on PTCI, PTCI-9, and CAPS-5 measures by each measurement timepoint.

	n_{PTCI}	PTCI	PTCI-9	n_{CAPS}	CAPS-5
		M (*SD*)	*M* (*SD*)		*M* (*SD*)
T0	83	135.1 (38.7)	3.8 (1.3)	83	40.7 (8.9)
T1	81	124.1 (45.2)	3.6 (1.4)	81	29.6 (15.1)
T2	73	114.5 (45.8)	3.3 (1.4)	75	23.5 (16.2)
T3	62	102.2 (44.6)	2.9 (1.4)	68	17.3 (15.5)

Note. n_{PTCI} = the number of participants who have completed the Posttraumatic Cognition Inventory; PTCI = the original Posttraumatic Cognitions Inventory; PTCI-9 = items of the short version of the Posttraumatic Cognitions Inventory; n_{CAPS} = the number of participants who have completed the Clinician Administered PTSD Scale for DSM-5; CAPS-5 = total score of the Clinician Administered PTSD Scale for DSM-5; Following the scale instructions, total scores of the PTCI-33 are calculated by *summing* 33 items whereas total scores of the PTCI-9 are calculated by *averaging* 9 items.

Table 2
Time-lagged mixed effect models with cognitions and change in symptoms.

Model and variable	B	*SE*	*t*	*p*	*d*
Predicting CAPS-5 from time-lagged PTCI					
Intercept	9.40	4.03	2.33	.021	
Time	−0.47	1.10	−0.43	.668	−0.11
CAPS-5 autoregression	0.49	0.08	6.28	<.001	0.96
Lagged PTCI	4.27	1.14	3.75	<.001	0.70
Predicting PTCI from time-lagged CAPS-5					
Intercept	−0.43	0.21	−2.03	.043	
Time	0.01	0.06	0.25	.801	0.05
PTCI autoregression	0.79	0.06	14.23	<.001	2.27
Lagged CAPS-5	0.01	0.00	1.61	.109	0.25

Note. CAPS-5 = total score of the Clinician Administered PTSD Scale for DSM-5; PTCI = the original Posttraumatic Cognitions Inventory.

Table 3
Sensitivity analyses.

Model and variable	B	*SE*	*t*	*p*	*d*
Predicting CAPS-5 from time-lagged PTCI-9					
Intercept	8.97	3.84	2.34	.020	
Time	−0.51	1.09	−0.47	.668	−0.11
CAPS-5 autoregression	0.50	0.07	7.21	<.001	1.11
Lagged PTCI-9	4.30	1.02	4.22	<.001	0.79
Predicting PTCI-9 from time-lagged CAPS-5					
Intercept	0.46	0.21	−2.19	.030	
Time	−0.01	0.06	−0.09	.926	−0.02
PTCI-9 autoregression	0.76	0.05	14.68	<.001	2.27
Lagged CAPS-5	0.01	0.00	2.54	.012	0.39
Predicting CAPS-5$^{excl9\text{-}10}$ from time-lagged PTCI					
Intercept	6.97	3.59	1.94	.521	
Time	−0.16	1.02	−0.16	.848	−0.04
CAPS-5$^{excl9\text{-}10}$ autoregression	0.51	0.08	6.82	<.001	1.05
Lagged PTCI	3.50	0.99	3.52	<.001	0.64
Predicting PTCI from time-lagged CAPS-5$^{excl9\text{-}10}$					
Intercept	−0.44	0.21	−2.12	.035	
Time	0.02	0.06	0.29	.776	0.06
PTCI autoregression	0.80	0.01	14.98	<.001	2.40
Lagged CAPS-5$^{excl9\text{-}10}$	0.01	0.00	1.72	.087	0.27

Note. CAPS-5 = total score of the Clinician Administered PTSD Scale for DSM-5; CAPS-5$^{excl9\text{-}10}$ = total score of the Clinician Administered PTSD Scale for DSM-5 minus item 9 and item 10; PTCI-9 = the short version of the Posttraumatic Cognitions Inventory; PTCI = the original Posttraumatic Cognitions Inventory.

Yeh, et al., 2015; Zalta et al., 2014). Given that temporal precedence is one of the requirements for establishing a mechanism of change (Kazdin, 2007), our findings provide support for cognitions as a change mechanism. However, we found a bidirectional temporal relationship between PTSD cognitions and symptoms when we measured cognitions using the items from the PTCI-9. A bidirectional temporal relationship was also found in some earlier studies (Kumpula et al., 2017; McLean, Su, & Foa, 2015; Rauch et al., 2021), suggesting that cognitions and symptoms mutually influence each other. Of note, when looking at the effect sizes of our analyses, we see a similar pattern across our analyses: the effect of posttraumatic cognitions on subsequent PTSD symptoms is almost twice as large as the reversed effect. We thus find evidence that change in cognitions predict subsequent symptom reduction, but it is difficult to disentangle posttraumatic cognitions as a change mechanism from the overarching PTSD symptomatology.

We extend earlier work by also testing the predictive value of the items from an abbreviated version of the PTCI, i.e., the PTCI-9. This shortened instrument could promote the tracking of cognitive changes during trauma-focused treatment in routine clinical care. Given the very high correlation between the PTCI and the PTCI-9, the PTCI-9 appears to be a suitable alternative to the PTCI. That said, whether the temporal relationship between cognitions and symptoms was uni- or bidirectional depended on the version of the PTCI. This difference cannot be easily explained. The broader range of items in the PTCI appear to be less affected by PTSD symptom change. Importantly, our findings imply that it matters how cognitions are measured and future research should be aware of this. More work with the PTCI-9 is warranted to see how findings with this instrument relate to findings when the full version is used and to validate its use in PTSD research, specifically in treatment studies.

It is important to note that change in negative posttraumatic cognitions is not proposed to be a mechanism of change that is specific to PE. Any treatment that accomplishes change in trauma-related cognitions proposedly leads to reductions in PTSD symptoms (Rauch & Foa, 2006). Indeed, empirical studies show that changes in trauma-related cognitions are relevant in other treatments too, such as cognitive (processing) therapy, written exposure therapy, present-centered therapy, and pharmacotherapy (Gobin et al., 2018; Kleim et al., 2013; Lee et al., 2021; McLean et al., 2019; Scher et al., 2017; Schumm et al., 2015). This is further supported by our exploratory analyses which included the PBT condition (STAIR followed by PE). Change in cognitions predicted subsequent changes in PTSD symptoms across all conditions and we found no significant interaction between treatment conditions and the effect of cognitions on PTSD symptoms.

Our current findings underline the importance of cognitive change for symptom alleviation, but future work should address whether promoting cognitive changes could improve treatment efficacy. Interestingly, in Inhibitory Learning Theory (ILT) it is proposed that maximizing expectancy violation could improve exposure efficacy (Craske et al., 2008, 2014, 2022). Expectancy violation (or prediction error) is thought to be crucial for the learning of inhibitory non-threat associations and refers to a mismatch between the expectancy of an aversive outcome and its non-occurrence. Translated to exposure therapy for PTSD, testing specific expectancies (e.g., "If I think back at the trauma, I will lose control and hurt someone") and directing attention to the non-occurrence of this outcome might promote new learning. Although related, expectancy violation and change in posttraumatic cognitions might be distinct processes. Repeated violation of specific expectancies is thought to lead to a change in the more generalized beliefs (Knowles & Tolin, 2022). For example, repeated violation of the expectancy to lose control upon exposure to the trauma memory may lead to change in the more general belief that one is weak or inadequate. Whether the application of ILT principles indeed improves the efficacy of exposure therapy for PTSD remains to be established (De Kleine et al., 2017). Future works should address whether promoting cognitive changes *within* sessions (i.e., expectancy violation) can improve treatment efficacy in PTSD. Moreover, aside from tracking changes in both expectancies and beliefs, it would be interesting to investigate established change mechanisms concurrently, such as belief change and between-session habituation (Cooper, Clifton, & Feeny, 2017). Studies with larger sample sizes are necessary for this.

The current study is not without its limitations. Firstly, we did not measure our variables at the session level, but rather at a 4-week interval during treatment. Using more frequent assessments (such as at the session level) increases the accuracy of the timeline between the proposed mechanism and the proposed outcome (see also Hagenaars et al., 2010). More frequent assessments would have been especially useful in the iPE condition. Secondly, due to a lack of measurement timepoints, our statistical analyses were not able to separate within-person from between-person effects, while this is important when investigating mechanisms of change (Falkenström et al., 2020). In the context of negative trauma-related cognitions and PTSD symptoms, this means that change in posttraumatic cognitions affects change in PTSD symptoms, *within* one person. Between-person effects demonstrate that, *on average*, those with more change in negative trauma-related cognitions have more change in PTSD symptoms, but do not clarify whether a change in trauma-related cognitions is related to fluctuations in PTSD symptoms over time within a patient. Dynamic panel models can distinguish within-from between-person effects and we have used this approach previously to investigate within- and between-session habituation as PE's mechanisms of change (Hoeboer et al., 2022). We found that, when looking at within-person effects, within-session habituation was predictive of PTSD symptom reduction, contrary to what previous studies using combined within- and between-effect statistical approaches found (for a review, see Asnaani et al., 2016). The use of the short PTCI-9 may increase the feasibility of more frequent assessments of cognitions and facilitate taking the crucial next step of separating within-from between-person effects. A third limitation is that participants in our study did not actually fill out the PTCI-9. To calculate the PTCI-9 score, we used the necessary items from the administered PTCI. We cannot exclude the possibility that participants would have filled out the items differently if only those nine items had been administered. Finally, a limitation of the current study is the missing data at later timepoints. Although the reasons for missing data varied, it was not completely random (we found significant differences in PTSD symptomatology and posttraumatic cognitions at baseline between participants who completed all measures and those who did not), which limits generalizability.

The strengths of this study include the fact that we investigated the temporality of cognitive change on PTSD symptoms in the context of a randomized clinical trial with few exclusion criteria (Oprel et al., 2021). This resulted in a culturally diverse, multimorbid sample with PTSD following childhood abuse, which serves the ecological validity of the current findings. No previous study has assessed the temporality of posttraumatic cognitions and symptom reduction in a sample of patients with PTSD related to childhood abuse specifically. These are patients who have often experienced multiple traumas early in life which affects how they view themselves and the world. Given their chronicity, these negative cognitions may be more difficult to treat. Yet, here we show that even in this early-traumatized sample cognitions change during PE and predict symptom reduction. Finally, this study is the first on this topic that assessed PTSD using DSM-5 criteria.

To conclude, negative trauma-related cognitions predict subsequent PTSD symptom reduction during PE, also when taking the conceptual changes made in the DSM-5 into account. Negative trauma-related cognitions can also be tracked with an abbreviated version of the PTCI. Symptoms and cognitions might mutually affect one another and more research is necessary to further elucidate the temporal relation between cognitive changes and symptom alleviation. Nevertheless, the findings confirm the importance of posttraumatic cognitions for achieving PTSD symptom improvement as changes in cognitions may indicate subsequent symptom reduction. As such, clinicians are

encouraged to track cognitions throughout treatment, as this can inform them on treatment effectiveness and assist in tailoring interventions (e.g., repeated exposure in vivo assignments to challenge a specific cognition). We highlight the need to assess negative posttraumatic cognitions on a session level which can be accomplished more easily with the use of short measures such as the PTCI-9.

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CRediT authorship contribution statement

Marike J. Kooistra: Conceptualization, Methodology, Formal analysis, Writing – original draft, Writing – review & editing. **Chris M. Hoeboer:** Conceptualization, Methodology, Software, Formal analysis, Investigation, Writing – review & editing. **Danielle A.C. Oprel:** Investigation, Writing – review & editing. **Maartje Schoorl:** Conceptualization, Writing – review & editing, Supervision. **Willem van der Does:** Conceptualization, Writing – review & editing, Supervision. **Jackie June ter Heide:** Writing – review & editing. **Agnes van Minnen:** Conceptualization, Writing – review & editing, Supervision. **Rianne A. de Kleine:** Conceptualization, Methodology, Writing – original draft, Writing – review & editing, Supervision.

Declaration of competing interest

None.

Data availability

Data will be made available on request.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.brat.2023.104284.

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