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BRIEF REPORT

Do Changes in Dysfunctional Posttraumatic Cognitions Differentially Predict PTSD Symptom Clusters?

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Objective: In recent years, it has been suggested that the modification of dysfunctional posttraumatic cognitions plays a central role as a mechanism of change in cognitive behavioral therapy (CBT) for posttraumatic stress disorder (PTSD). Indeed, several studies have shown that changes in dysfunctional posttraumatic cognitions precede and predict symptom change. However, these studies have investigated the influence on *overall* symptom severity—despite the well-known multidimensionality of PTSD. The present study therefore aimed to explore differential associations between change in dysfunctional conditions and change in PTSD symptom clusters. ***Method:*** As part of a naturalistic effectiveness study evaluating trauma-focused cognitive behavioral therapy for PTSD in routine clinical care, 61 patients with PTSD filled out measures of dysfunctional posttraumatic cognitions and PTSD symptom severity every five sessions during the course of treatment. Lagged associations between dysfunctional cognitions and symptom severity at the following timepoint were examined using linear mixed models. ***Results:*** Over the course of therapy, both dysfunctional cognitions and PTSD symptoms decreased. Posttraumatic cognitions predicted subsequent total PTSD symptom severity, although this effect was at least partly explained by the time factor. Moreover, dysfunctional cognitions predicted three out of four symptom clusters as expected. However, these effects were no longer statistically significant when the general effect for time was controlled for. ***Conclusion:*** The present study provides preliminary evidence that dysfunctional posttraumatic cognitions predict PTSD symptom clusters differentially. However, different findings when employing a traditional versus a more rigorous statistical approach make interpretation of findings difficult.

What is the public health significance of this article?
This study highlights how posttraumatic cognitions, that is, dysfunctional appraisals about the traumatic event and its consequences, predict changes in posttraumatic stress disorder (PTSD) over the course of psychotherapy. It was closely investigated how these cognitions affect different PTSD symptom subgroups. This can inform clinicians and practitioners in their treatment planning.

Keywords: cognitions, posttraumatic stress disorder, mechanisms of change, trauma-focused treatment

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The data reported in this article were collected as part of a larger data collection (at several points in time). Findings from the data collection will be reported in three separate articles. Manuscript 1 (in preparation) focuses on pre-, post-, and follow-up measures of posttraumatic stress disorder (PTSD) symptom severity and other measures of psychosocial functioning. Its aim is to investigate the effectiveness of a trauma-focused treatment on different variables and to study possible predictors of outcome. Manuscript 2 (published) investigates posttraumatic rumination as possible mechanism of change in PTSD treatment. It focuses on variables *ruminative thinking* and *PTSD severity*, both measured weekly. Manuscript 3 (the present article) focuses on five-weekly measures of PTSD symptom severity and dysfunctional posttraumatic cognitions, which are not reported elsewhere. Furthermore, demographic and clinical variables also to-be-presented in MS 1 are reported here to describe the sample to the reader.

Parts of the data have already been published in H. Schumm et al. (2021).

Hannah Schumm played a lead role in conceptualization, data curation, formal analysis, writing–original draft, and writing–reviewing and editing. Antje Krüger-Gottschalk played a lead role in funding acquisition, investigation, methodology

continued

Numerous studies have shown that trauma-focused psychological treatments for posttraumatic stress disorder (PTSD) are effective (Cusack et al., 2016). In the last decade, research has started to focus on investigating the processes of change during treatment, especially on the role of dysfunctional posttraumatic cognitions (Kleim et al., 2013). Dysfunctional cognitions about the trauma and its consequences have been shown to be strongly associated with PTSD symptom severity (Gómez de La Cuesta et al., 2019) and to predict PTSD symptoms prospectively (Beierl et al., 2019). Given the robust evidence for a central role of dysfunctional cognitions in the etiology and maintenance of PTSD, researchers have begun to look into changes in cognitions as possible mechanism of change in trauma-focused psychotherapies. In a seminal study by Kleim et al. (2013), patients receiving cognitive therapy for PTSD were given questionnaires measuring dysfunctional cognitions and PTSD symptoms at every session. Results showed that a change in dysfunctional posttraumatic cognitions predicted symptom change in the following week unidirectionally. Since then, several studies have replicated these findings (J. A. Schumm et al., 2015; Zalta et al., 2014). However, existing studies are limited in that they have primarily conceptualized PTSD as monolithic disorder. Thus, they invariably investigated the influence of posttraumatic cognitions on *overall* PTSD symptom severity—although the multidimensionality and heterogeneity of the disorder have been well-established (Galatzer-Levy & Bryant, 2013). It is therefore currently unknown whether the symptom clusters of PTSD—namely *re-experiencing*, *avoidance*, *alterations in arousal and reactivity*, and *changes in mood and cognition* (American Psychiatric Association, 2013)—show differential associations with dysfunctional posttraumatic cognitions. Accordingly, more fine-grained analyses have been called for (Brown et al., 2019). Hence, the aim of the present study was to investigate the relationship between dysfunctional posttraumatic cognitions and the symptom clusters of PTSD in more detail.

Over the course of trauma-focused cognitive behavioral therapy, dysfunctional cognitions, and PTSD symptoms were measured every fifth session and lagged associations between cognitions and symptoms were examined. Our hypotheses regarding associations between dysfunctional cognitions and the different symptom clusters are derived from the cognitive model of PTSD by Ehlers and Clark (2000). Therefore, we expected dysfunctional cognitions to significantly predict changes in Cluster D (negative alterations in cognitions and mood) over the course of therapy (Hypothesis 1). In the cognitive model of PTSD, dysfunctional cognitions are assumed to be directly linked to emotions. Additionally, dysfunctional cognitions are conceptually close to this cluster, with even some symptom overlaps. Further derived from the model by Ehlers and Clark (2000), which proposes a close relationship between dysfunctional appraisals and characteristics of the trauma memory, we expected dysfunctional cognitions to predict changes in Cluster B (re-experiencing) over the course of therapy (Hypothesis 2). Next, as dysfunctional posttraumatic cognitions are assumed to motivate avoidance behavior, we expected them to predict changes in Cluster C (avoidance) over the course of therapy (Hypothesis 3). Whereas the cognitive model does assume that general feelings of anxious arousal—subsumed under a *sense of current threat*—are partly produced by dysfunctional cognitions, it does not make any assumptions regarding the relationship between dysfunctional cognitions and the *symptom cluster* hyperarousal and its components such as irritable behavior, sleeping disturbances, concentration problems, etc. Therefore, no association with Cluster E (alterations in arousal and reactivity, Hypothesis 4) was expected.

Method

Transparency and Openness

We report how we determined our sample size, all data exclusions (if any), all manipulations, and all measures in the study, and we follow journal article reporting standards (Kazak, 2018). Data were analyzed using R (Version 4.0.1; R Core Team, 2020) and the lme4 package (Bates et al., 2015). This study's design and its analysis were not preregistered.

Participants

Participants were recruited for a multicenter effectiveness study that aimed to evaluate an empirically established treatment protocol for PTSD in routine clinical care (for details of the intervention and its effectiveness, see Krüger-Gottschalk et al., 2022; H. Schumm et al., 2021). The study was approved by the local ethics committee at Münster University. Inclusion criteria were a primary diagnosis of PTSD assessed with the Clinician-Administered PTSD Scale for *Diagnostic and Statistical Manual of Mental Disorders, fifth edition* (CAPS; Weathers et al., 2013) and age of at least 18 years. Patients with a diagnosis of current substance dependence, psychotic disorder, or immediate suicide risk, or who had a body mass index lower than 17.5 (all assessed via Structured Clinical Interview for *DSM-IV* [SCID-IV]), were excluded from the study. A total of 89 patients took part in the treatment study. From 18 participants (20.2%), no process measures relevant for the present study could be obtained. Analyses were therefore based on 61 participants. Demographic and clinical characteristics of the sample are shown in Table 1.

Treatment

All patients were treated with trauma-focused cognitive behavioral therapy. Due to the nature of a naturalistic trial, there was no randomization to different treatments nor a control condition. All patients followed the same treatment manual that was based on a modularized phase-based approach (see Table 2). Therapy was provided either by licensed cognitive behavioral therapy therapists or by therapists in advanced postgraduate training. Based on the data from 80 patients, the

and project administration, supporting role in writing–reviewing and editing, and an equal role in conceptualization. Thomas Ehring played a lead role in conceptualization, funding acquisition, investigation, methodology, project administration and supervision, and a supporting role in writing–reviewing and editing. Anne Dyer played a supporting role in conceptualization, investigation, and writing–reviewing and editing. Andre Pittig played a supporting role in conceptualization, investigation, methodology, and writing–reviewing and editing. Keisuke Takano played a supporting role in formal analysis and writing–reviewing and editing, and an equal role in data curation. Georg W. Alpers played a supporting role in investigation, methodology and writing–reviewing and editing, and an equal role in conceptualization and funding acquisition. Barbara Cludius played a equal role in supervision and writing–reviewing and editing.

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Table 1
Characteristics of the Sample at Baseline and Descriptive Statistics for Study Variables

Variable	*n* (%)/*M* (*SD*)
Gender (available from $n = 84$)	
Female	68 (81%)
Male	16 (19%)
Age in years ($n = 86$)	36.06 (12.94)
Employment ($n = 82$)	
Employed	40 (48.8%)
Unemployed	20 (24.4%)
Retired	5 (6.1%)
Other	11 (13.4%)
Highest educational level ($n = 73$)	
University degree	9 (10.7%)
High school[a]	12 (14.3%)
Secondary school[b]	47 (56%)
Primary school	3 (3.6%)
No degree	4 (4.8%)
Other	10 (11.9%)
Previous inpatient psychiatric stay ($n = 68$)	
Yes	37 (50.7%)
No	36 (49.3%)
Previous outpatient psychotherapy ($n = 85$)	
Yes	42 (61.8%)
No	26 (38.2%)
Comorbidity (assessed via SCID-IV)	
No additional diagnosis	42 (49.4%)
One additional diagnosis	22 (25.9%)
Two or more additional diagnoses	21 (24.7%)
Pretreatment PTSD symptom severity (CAPS[c]) ($n = 78$)	38.32 (10.41)
Pretreatment self-reported PTSD symptom severity (PCL-5)	38.42 (19.36)
Self-reported symptom severity (PCL-5) across all timepoints[d]	29.23 (20.66)
Self-reported dysfunctional cognitions (PTCI)[d]	72.80 (25.65)
Repeated measures correlation PTCI and PCL-5 sum score	$r = 0.67$ ($p < .001$)
Repeated measures correlation PTCI and PCL-5 Cluster B	$r = 0.54$ ($p < .001$)
Repeated measures correlation PTCI and PCL-5 Cluster C	$r = 0.58$ ($p < .001$)
Repeated measures correlation PTCI and PCL-5 Cluster D	$r = 0.65$ ($p < .001$)
Repeated measures correlation PTCI and PCL-5 Cluster E	$r = 0.58$ ($p < .001$)

Note. PCL-5 = PTSD Symptom Checklist for *Diagnostic and Statistical Manual of Mental Disorders, fifth edition*; PTCI = Posttraumatic Cognitions Inventory; SCID-IV = Structured Clinical Interview-IV; CAPS = Clinician-Administered PTSD Scale; PTSD = posttraumatic stress disorder.
[a] High school: 12–13 years of schooling in the German school system. [b] Secondary school: 9–10 years of schooling in the German school system. [c] CAPS = Clinician-Administered PTSD Scale for *DSM-V* (German Version by Schnyder, 2013). [d] Averaged across all timepoints ($N = 317$) and all participants ($N = 61$).

average number of sessions (50 min each) was $M = 35.91$ ($SD = 20.72$, range 1–80).[1]

Measures

PTSD Checklist for DSM-5

The PTSD Checklist (PCL-5; German version: Krüger-Gottschalk et al., 2017) is a 20-item self-report measure assessing PTSD symptom severity. Distress caused by each symptom is rated on a 5-point scale ranging from 0 (*not at all*) to 4 (*extremely*). To enable its use in each treatment session, it was adapted to assess symptom severity in the past week (as opposed to past month). The reliability both within- and between-person in the present sample was good (both R_{kRn}^2 and $R_{cn}^3 > .95$). The total severity score is calculated as a sum of all items (range 0–80). Additionally, symptom cluster severity scores can be obtained by summing the scores for the items for each cluster, that is, Cluster B (Items 1–5), Cluster C (Items 6–7), Cluster D (Items 8–14), and Cluster E (Items 15–20).

Posttraumatic Cognitions Inventory–Short Version

The Posttraumatic Cognitions Inventory (PTCI; German version by Ehlers & Boos, 1999) is a self-report measure designed to assess trauma-related cognitions about the self. Each item is rated on a 7-point Likert scale ranging from 1 (*totally disagree*) to 7 (*totally agree*). For the present study, a shortened version with 22 items based on Kleim et al. (2013) was used. A total score was used for all analyses. The reliability both within- and between-person in the present sample was acceptable ($R_{kRn} = 94$ and $R_{cn} = .75$).

Procedure

At baseline, clinical interviews were administered and patients filled in sociodemographic and clinical questionnaires. Every fifth treatment session, patients filled in paper–pencil questionnaires assessing symptom severity and posttraumatic cognitions. Therapy sessions were scheduled weekly, although not necessarily 7 days apart.

Statistical Analyses

Due to the nested data structure, linear mixed models (LMM) were used to test the hypotheses. Each model had a two-level structure with measurements every five sessions nested within-persons. All models assumed random effects for the intercept and slope, which were allowed to vary across participants. Models were estimated with the restricted maximum likelihood estimation; the level of significance was set as $\alpha = 0.05$. Furthermore, data points that were more than 10 weeks apart were excluded, as these data were deemed unreliable. The PTCI was person-mean centered to predict the PCL-5 sum score or cluster scores at the next assessment point. All predictors were standardized with the grand mean and *SD* to avoid convergence errors. For details of the LMM analyses (including equations), please see the Electronic Supplemental Material.

[1] Due to the naturalistic setting in the German health care system, which grants up to 80 therapy sessions, patients have received a higher number of therapy sessions than in previously reported trials on cognitive therapy for PTSD (e.g., Ehlers et al., 2013, $M = 10.6$). However, these studies have typically employed sessions of 90-min length, therefore the treatment dose remains comparable. The range and average number of sessions reported here is typical for routine outpatient treatment for PTSD in the German health care system.

[2] R_{kRn} indicates the generalizability of between person differences averaged over time, with time nested within people (Shrout & Lane, 2012).

[3] R_{cn} indicates the generalizability of within-person variations averaged over items (Shrout & Lane, 2012).

Table 2
Trauma-Focused Cognitive Behavioral Therapy Components and Content

Therapy phase	Content
Assessment	1. Assessment of inclusion and exclusion criteria, patient history, treatment planning
Phase 1	2. Establishing therapy goals
	3. Psychoeducation about onset and maintenance of PTSD
	4. Explanation of rationale for trauma memory work
	5. Targeting self- or therapy-harming behaviors
Phase 2	6. Imaginal exposure to traumatic events
	7. Cognitive processing of the imaginal exposure
	8. Trigger analyses and discrimination training
	9. Cognitive work on changing dysfunctional appraisals
Phase 3	10. Improving quality of life by reclaiming-your-life assignments
	11. Relapse prevention

Note. PTSD = posttraumatic stress disorder.

Results

Descriptive Statistics

Descriptive statistics of the investigated variables are shown in Table 1. On average, 5.79 observations per participant ($SD = 3.62$) were available. The correlation between the PCL-5 and PTCI across all timepoints and participants was $r = 0.72$ ($p < .001$).

Posttraumatic Cognitions as a Predictor for PTSD Symptom Clusters

Results showed that in line with hypotheses, dysfunctional cognitions in the preceding week were a significant predictor of subsequent *total* PTSD symptom severity (see Table 3). However, with time as additional predictor to control for the general effect of time, this effect was nonsignificant. Regarding the prediction of the different symptom clusters (see Table 4), as hypothesized Cluster B (re-experiencing), Cluster C (avoidance), and Cluster D (alterations in cognitions and mood) were significantly predicted by dysfunctional cognitions in the previous session. However, these effects were no longer statistically significant when the general effect for time was controlled for. As expected, Cluster E (alterations in arousal and reactivity) was not predicted by dysfunctional cognitions, neither with or without the control for time. In additional exploratory analyses, we investigated the reverse relationships between symptom levels and dysfunctional cognitions for all clusters (see Table 5). When time was not controlled for, all of the reverse relationships were significant. When time was controlled for, changes in cognitions were only predicted by changes in avoidance and in hyperarousal.

Discussion

We investigated whether changes in dysfunctional cognitions predicted reduction of PTSD symptom severity over the course of therapy, and whether PTSD symptom clusters were predicted differentially. Results showed that both PTSD severity and cognitions decreased over the course of the trauma-focused treatment. Next, the previously reported finding that dysfunctional cognitions predict changes in symptom severity over the course of therapy was partly replicated here. Regarding the differential effects on the various clusters, the clusters *re-experiencing*, *avoidance* and *changes in mood, and cognition* were significantly predicted by changes in dysfunctional cognitions, whereas *alterations in arousal and reactivity* were not. However, when time was controlled for, none of the clusters were predicted significantly by dysfunctional cognitions. As we found diverging results when time was controlled for versus when it was not, our results need to be discussed in light of the different statistical approaches. Both approaches have precedents in the literature that the present study builds upon. When time was *not* additionally controlled for—parallel to J. A. Schumm et al. (2015) or Kleim et al. (2013)—our results are in line with the majority of studies showing that changes in dysfunctional cognitions predict changes in overall PTSD symptom severity (Brown et al., 2019). But, when the time factor *was* controlled for—parallel to, for example, Zalta et al. (2014)—previous results could not be replicated. Adding time as an additional predictor in the analyses ("detrending") is applied to protect against the detection of only spurious relationships. However, some authors have highlighted that this is a conservative statistical approach, which can result in overcontrolling (Falkenström et al., 2017)—thereby leading to nonsignificant findings.

The differential effects on the four PTSD clusters are in line with our hypotheses. As expected, changes in dysfunctional cognitions predicted changes in the clusters *avoidance* and *alterations in mood and cognitions*. Dysfunctional posttraumatic cognitions often contain irrational thoughts about a dangerous world and are therefore thought to motivate avoidance behavior. Additionally, dysfunctional cognitions are thought to be directly linked to emotions (Ehlers & Clark, 2000), thereby explaining the predictive effect. Also in line with our

Table 3
Results of LMMs for Total PCL-5 Score as Dependent Variable

	Model 1				Model 2				Model 3			
Variable	β[a]	*SE*	*t*	*p*	β	*SE*	*t*	*p*	β	*SE*	*t*	*p*
Intercept	27.33	2.07	13.21	<.001	26.79	2.53	10.61	<.001	27.26	2.53	10.78	<.001
Time	−11.37	1.07	−10.61	<.001					−5.89	1.07	−5.50	<.001
PCL-5 lagged					5.73	1.13	5.09	<.001	3.52	1.12	3.15	<.001
PTCI lagged					2.04	0.88	2.34	.039	0.66	0.83	0.8	.428

Note. *Lagged* variables represent the autocorrelations between a score at timepoint i and timepoint i + 1. PCL-5 = PTSD Symptom Checklist for *Diagnostic and Statistical Manual of Mental Disorders*, *fifth edition*; PTCI = Posttraumatic Cognitions Inventory; *SE* = standard error; PTSD = posttraumatic stress disorder; LMMs = linear mixed models.
[a] β(=fixed effect) represents the magnitude of change in the outcome variable as the predictor increases by one standard deviation.

Table 4
Results of LMMs for PCL-5 Cluster Scores as Dependent Variables (DV)

	Model 1				Model 2			
Variable	β[a]	*SE*	*t*	*p*	β	*SE*	*t*	*p*
DV: PCL-5 Cluster B								
Intercept	6.04	0.64	9.38	<.001	6.23	0.64	9.73	<.001
PCL-5 Cluster B lagged	1.13	0.35	3.26	.003	0.46	0.33	1.39	.170
PTCI lagged	0.97	0.29	3.36	.008	0.31	0.26	1.19	.234
Time					−2.16	0.34	−6.40	<.001
DV: PCL-5 Cluster C								
Intercept	2.77	0.30	9.10	<.001	2.87	.029	9.75	<.001
PCL-5 Cluster C lagged	0.64	0.16	4.11	<.001	0.24	0.16	1.62	.134
PTCI lagged	0.28	0.12	2.42	.016	0.03	0.11	0.26	.792
Time					−1.03	0.15	−6.79	<.001
DV: PCL-5 Cluster D								
Intercept	9.38	0.92	10.21	<.001	9.52	0.92	10.33	<.001
PCL-5 Cluster D lagged	1.63	0.44	3.7	<.001	0.99	0.44	2.23	.031
PTCI lagged	0.89	0.34	2.66	.021	0.39	0.32	1.12	.219
Time					−1.86	0.4	−4.68	<.001
DV: PCL-5 Cluster E								
Intercept	8.68	0.86	9.92	<.001	8.63	0.87	9.92	<.001
PCL-5 Cluster E lagged	1.60	0.45	3.59	<.001	0.64	0.47	1.37	.177
PTCI lagged	0.5	0.31	1.63	.126	−0.18	0.32	−0.55	.587
Time					−1.66	0.77	−2.15	.039

Note. *Lagged* variables represent the autocorrelations between a score at timepoint i and timepoint i + 1. PCL-5 = PTSD Symptom Checklist for *Diagnostic and Statistical Manual of Mental Disorders*, *fifth edition*; PTCI = Posttraumatic Cognitions Inventory; *SE* = standard error; PTSD = posttraumatic stress disorder; LMMs = linear mixed models.
[a] β(=fixed effect) represents the magnitude of change in the outcome variable as the predictor increases by one standard deviation.

hypotheses, the cluster *re-experiencing* was predicted by changes in dysfunctional cognitions. Ehlers and Clark (2000) propose a close relationship between dysfunctional appraisals and characteristics of the trauma memory, which are thought to underlie intrusive re-experiencing. Additionally, thinking negatively about the trauma and its consequences may serve as a trigger and could thereby lead to intrusive memories, explaining why changes in dysfunctional cognitions predict changes in re-experiencing. Last,

Table 5
Results of LMMs for Dysfunctional Cognitions DV (Testing Reverse Relationships)

	Model 1				Model 2			
Variable	β[a]	*SE*	*t*	*p*	β	*SE*	*t*	*p*
DV: PTCI								
Intercept	70.96	3.23	21.97	<.001	71.09	3.27	21.77	<.001
PTCI lagged	1.88	1.50	1.25	.217	−1.94	1.35	−1.45	.156
PCL-5 Cluster B lagged	4.64	1.27	3.65	.001	2.10	1.30	1.61	.121
Time					−10.06	1.97	−5.1	<.001
DV: PTCI								
Intercept	70.98	3.24	21.92	<.001	70.77	3.26	21.74	<.001
PTCI lagged	1.47	1.49	0.99	.327	−1.92	1.30	−1.48	.146
PCL-5 Cluster C lagged	5.89	1.01	5.82	<.001	3.35	1.06	3.17	.002
Time					−9.40	2.15	−4.38	<.001
DV: PTCI								
Intercept	70.97	3.24	21.88	<.001	71.00	3.27	21.74	<.001
PTCI lagged	1.57	1.50	1.05	.301	−1.25	1.36	−0.92	.365
PCL-5 Cluster D lagged	5.48	1.47	3.73	.001	2.84	1.49	1.91	.073
Time					−8.50	1.78	−4.77	<.001
DV: PTCI								
Intercept	71.15	3.24	22	<.001	71.07	3.26	21.78	<.001
PTCI lagged	1.82	1.56	1.17	.249	−1.50	1.38	−1.09	281
PCL-5 Cluster E lagged	5.40	1.31	4.13	<.001	3.08	1.04	2.96	.042
Time					−9.57	1.84	−5.21	<.001

Note. DV = dependent variables; PCL-5 = PTSD Checklist-5; PTCI = Posttraumatic Cognitions Inventory; *SE* = standard error; PTSD = posttraumatic stress disorder; LMMs = linear mixed models.
[a] β (=fixed effect) represents the magnitude of change in the outcome variable as the predictor increases by one standard deviation.

we found that *alterations in arousal and reactivity* were not predicted by dysfunctional cognitions. With this cluster, the relationship may well be reversed: Previous studies have shown that hyperarousal drives changes in other symptom clusters of PTSD such as dysfunctional cognitions (e.g., Schell et al., 2004), and not the other way round as was hypothesized here. Additional exploratory analyses confirmed this assumption, showing that changes in hyperarousal significantly predicted changes in dysfunctional cognitions over the course of therapy (both without and with controlling for time; see Table 5, for all reverse models). Moreover, we found a close bidirectional relationship between all PTSD symptom clusters and dysfunctional cognitions. As posttraumatic cognitions are defined as dysfunctional interpretations of the traumatic event or its sequelae, including one's symptoms (Ehlers & Clark, 2000), cognitions are expected to decrease when symptoms ameliorate. Future studies should be designed to untangle the reasons for the reverse effects.

Several strengths of the study should be considered. A longitudinal design with repeated measures was employed, and PTSD diagnoses were established with the diagnostic gold standard. Additionally, our study sample as well as treatment dose was naturalistic. Limitations include the unknown variability in treatment content due to the modular approach, and a mainly female sample. Reassuringly, this gender ratio is rather typical for treatment-seeking civilian PTSD patients (Lewis et al., 2020). Additionally, we would like to draw the readers' attention to the current debate whether MLM are the best-suited analytic approach for investigating cross-lagged effects in psychotherapy research or whether random-intercept cross-lagged panel models, estimated within a Structural Equation Modeling framework might offer some benefits (Falkenström et al., 2022).

The present study found that changes in dysfunctional posttraumatic cognitions predicted changes in three of four PTSD symptom clusters, hinting at a more general effect on PTSD symptoms. Future studies should assess whether this effect is stable. Additionally, previous studies have been inconsistent in their statistical approach regarding the control for time, whereas our results show that this distinction is quite crucial. Future studies should therefore routinely employ both approaches and compare results.

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