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Mechanisms of Change in Trauma-Focused Treatment for PTSD: The Role of Rumination

Hannah Schumm [a,*], Antje Krüger-Gottschalk [b], Anne Dyer [c], Andre Pittig [d], Barbara Cludius [a], Keisuke Takano [a], Georg W. Alpers [e], Thomas Ehring [a]

[a] Department of Psychology, LMU Munich, Leopoldstr. 13, Munich, Germany
[b] Institute of Psychology, WWU Münster, Fliednerstr. 21, 48149, Münster, Germany
[c] ZISG Mannheim, Central Institute of Mental Health, J 5, 68159, Mannheim, Germany
[d] Translational Psychotherapy, Department of Psychology, Friedrich–Alexander University Erlangen–Nürnberg (FAU), Nägelsbachstr. 49a, 91051, Erlangen, Germany
[e] Department of Psychology, School of Social Sciences, University of Mannheim, L 13, 17, 68163, Mannheim, Germany

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ABSTRACT

Objective: Cognitive behavioral therapy (CBT) has been well established in the treatment of posttraumatic stress disorder (PTSD). In recent years, researchers have begun to investigate its underlying mechanisms of change. Dysfunctional cognitive *content*, i.e. excessively negative appraisals of the trauma or its consequences, has been shown to predict changes in PTSD symptoms over the course of treatment. However, the role of change in cognitive *processes*, such as trauma-related rumination, needs to be addressed. The present study investigates whether changes in rumination intensity precede and predict changes in symptom severity. We also explored the extent to which symptom severity predicts rumination.

Method: As part of a naturalistic effectiveness study evaluating CBT for PTSD in routine clinical care, eighty-eight patients with PTSD completed weekly measures of rumination and symptom severity. Lagged associations between rumination and symptoms in the following week were examined using linear mixed models.

Results: Over the course of therapy, both ruminative thinking and PTSD symptoms decreased. Rumination was a significant predictor of PTSD symptoms in the following week, although this effect was at least partly explained by the time factor (e.g., natural recovery or inseparable treatment effects). Symptom severity predicted ruminative thinking in the following week even with time as an additional predictor.

Conclusions: The present study provides preliminary evidence that rumination in PTSD is reduced by CBT for PTSD but does not give conclusive evidence that rumination is a mechanism of change in trauma-focused treatment for PTSD.

1. Are reductions in rumination a mechanism of change in trauma-focused cognitive behavioral therapy for PTSD?

There is ample evidence for the efficacy and effectiveness of cognitive behavioral treatments for PTSD (Bisson et al., 2007; Bradley, Greene, Russ, Dutra, & Westen, 2005; Stewart & Chambless, 2009; Watts et al., 2013). However, whereas 44–67% of patients no longer meet the diagnostic criteria for PTSD after treatment, a sizeable portion of patients remains burdened by symptoms after completing treatment (Bradley et al., 2005; Cusack et al., 2016). One pathway to increasing the efficacy of existing treatments and reducing non-response may be to focus on understanding mechanisms of change in existing treatments. A mechanism of change is defined as "steps or processes through which therapy (or some independent variable) actually unfolds and produces the change" (Kazdin, 2007, p. 3, p. 3). By identifying mechanisms that are responsible for symptom reduction in therapy, treatments can be further refined and distilled to their essential elements, thereby increasing treatment efficiency. Moreover, knowing which elements of therapy are essential may help therapists to individualize treatment in a way that will maximize benefit for patients.

* Corresponding author.
E-mail addresses: Hannah.Schumm@psy.lmu.de (H. Schumm), antje.krueger@uni-muenster.de (A. Krüger-Gottschalk), Anne.Dyer@zi-mannheim.de (A. Dyer), andre.pittig@fau.de (A. Pittig), Barbara.Cludius@psy.lmu.de (B. Cludius), Keisuke.Takano@psy.lmu.de (K. Takano), alpers@uni-mannheim.de (G.W. Alpers), thomas.ehring@lmu.de (T. Ehring).

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In his seminal article, Kazdin (2007) postulated several key requirements for a process to qualify as a mechanism of change. Against the background of a plausible theoretical rationale, a strong and specific association between process and intervention is required – one that is characterized by a dose-response relationship and can be consistently replicated across studies, including those using an experimental design to establish causality. Importantly, the proposed mechanism must also be shown to occur *before* a change in treatment outcome (e.g., symptom reduction), and to predict symptom change. This last criterion in particular, regarding temporal precedence, has often been overlooked in studies testing potential mechanisms of change. In other words, "demonstrating a timeline between cause and an effect, albeit obvious, is the Achilles' heel of treatment studies" (Kazdin, 2007, p. 5).

Current evidence-based treatments for PTSD are typically based on cognitive models of the disorder (Ehlers & Clark, 2000; Foa & Cahill, 2001; Resick & Schnicke, 1992). On the basis of these models, it can be hypothesized that cognitive factors are an important mechanism of change in PTSD treatment. In the following, empirical evidence for both cognitive *content* as well as cognitive *processes* as mechanisms of PTSD treatment is reviewed. Cognitive content refers to *what* one is thinking, for example excessively negative appraisals of the trauma and/or its consequences (e.g., "Nowhere is safe"; "It was my fault that the trauma happened"; "My symptoms mean I'm going crazy"). Cognitive process on the other hand describes *how* one is thinking about it, for example in an abstract or repetitive way.

2. Modification of posttraumatic cognitive *content* as a mechanism of change in PTSD

Cognitive models of PTSD posit that dysfunctional cognitive content plays a crucial role in the development and maintenance of PTSD (Ehlers & Clark, 2000; Foa & Cahill, 2001; LoSavio, Dillon, & Resick, 2017). This hypothesis has received strong empirical support (for recent reviews, see Brown et al., 2019; LoSavio et al., 2017). Specifically, dysfunctional trauma-related cognitions have been shown to be strongly associated with symptom severity as well as functional impairment (Lyons, Haller, Curry, & Norman, 2019; for a recent meta-analysis see Gómez de La Cuesta, Schweizer, Diehle, Young, & Meiser-Stedman, 2019). This relationship has been demonstrated across gender, culture, and age (L. A. Brown et al., 2019). Moreover, dysfunctional cognitions in the early aftermath of a traumatic event have been found to predict PTSD symptom severity months and years later (Beierl, Böllinghaus, Clark, Glucksman, & Ehlers, 2019; Ehring, Ehlers, & Glucksman, 2008; Murray, Ehlers, & Mayou, 2002; O'Donnell, Elliott, Wolfgang, & Creamer, 2007).

Given the important role of dysfunctional trauma-related cognitions in the maintenance of PTSD, researchers have suggested that the modification of these posttraumatic cognitions may be an important mechanism of change in PTSD treatment. This hypothesis is supported by a number of studies showing that dysfunctional posttraumatic cognitions decline during trauma-focused treatments, changes in posttraumatic cognitions are associated with reductions in symptomatology (Cooper, Zoellner, Roy-Byrne, Mavissakalian, & Feeny, 2017; Dondanville et al., 2016; Iverson, King, Cunningham, & Resick, 2015; Kleim, Grey, et al., 2013; Littleton, Buck, Rosman, & Grills-Taquechel, 2012) and more importantly, they precede as well as predict symptom amelioration (Kleim, Grey, et al., 2013; Kumpula et al., 2017; McLean, Yeh, Rosenfield, & Foa, 2015; Schumm, Dickstein, Walter, Owens, & Chard, 2015; Wiedemann et al., 2020). These studies serve as precedent for the present study, and are therefore elaborated in more detail in the following. In a seminal study by Kleim, Graham, Bryant, and Ehlers (2013), patients receiving weekly cognitive therapy for PTSD were given questionnaires at every session measuring dysfunctional cognitions and PTSD symptoms. Results showed that a change in negative appraisals predicted symptom change in the following week, but not vice versa. These findings were replicated in the context of trauma-focused psychotherapy implemented in a residential program (Schumm et al., 2015), and in four studies evaluating prolonged exposure for PTSD in an outpatient setting (Cooper et al., 2017; Kumpula et al., 2017; McLean et al., 2015; Zalta et al., 2014). Lastly, Wiedemann et al. (2020) were able to show that changes in dysfunctional cognitions also preceded so-called sudden gains, i.e., large and stable symptom improvements that occur from one therapy session to the next. Importantly, researchers have shown that the effect of change in posttraumatic cognitions predicting symptom change in the context of PTSD is distinctly stronger in cognitive behavioral interventions compared to a minimal attention group, client-centered therapy, or a psychopharmaceutical intervention (Cooper et al., 2017; McLean et al., 2015; Zalta et al., 2014). In sum, past research has provided strong evidence for changes in cognitive *content* as a mechanism of change in PTSD treatment, and serves as an example for the investigation on the role of cognitive processes as a mechanism of change.

3. Modification of cognitive *processes* as a potential mechanism of change in PTSD

The role of cognitive *processes*, on the other hand, has been investigated to a far lesser extent. For example, trauma-related rumination, defined as repetitive negative thinking about the trauma and/or its consequences (Michael, Halligan, Clark, & Ehlers, 2007), has been suggested as playing a key role in the development and maintenance of PTSD (Ehring, Frank, & Ehlers, 2008; Elwood, Hahn, Olatunji, & Williams, 2009; Valdez & Lilly, 2017; for a recent review, see; Moulds, Bisby, Wild, & Bryant, 2020).

In their cognitive model of PTSD, Ehlers and Clark (2000) conceptualize rumination as a dysfunctional cognitive strategy triggered by dysfunctional trauma-related appraisals, intrusive trauma-related memories, and a perceived sense of current threat. Rumination has been conceptualized as a cognitive avoidance strategy in which a person dwells on the traumatic event and its consequences, without fully processing the traumatic memory itself (Ehlers & Steil, 1995; Michael et al., 2007). Although trauma survivors engage in rumination to control the sense of current threat, rumination is thought to maintain PTSD by directly producing symptoms (e.g., triggering intrusions or increasing anxiety; for empirical evidence see Birrer & Michael, 2011; Michael et al., 2007), preventing change in the negative appraisals as well as preventing change in the nature of the trauma memory. Therefore, when ruminative thinking declines, naturally occurring recovery processes such as elaboration of the trauma memory and change in negative appraisals can occur. Consequently, symptom severity is expected to decline.

In line with the Ehlers and Clark (2000) model, a strong association between rumination and PTSD has consistently been shown across studies and samples, with higher levels of rumination being linked to greater severity of posttraumatic stress symptoms (Ehring, Frank, & Ehlers, 2008; Michael et al., 2007; Steil & Ehlers, 2000; for a recent meta-analysis, see Szabo, Warnecke, Newton, & Valentine, 2017). This effect remains significant after controlling for negative affect (Arditte Hall et al., 2019; Seligowski, Rogers, & Orcutt, 2016), and even fully accounts for the relationship between negative affect and PTSD (W. J. Brown, Hetzel-Riggin, Mitchell, & Bruce, 2018). Furthermore, a number of studies have shown that the severity of rumination in the early aftermath of a traumatic event also predicts PTSD severity months and years later (Beierl et al., 2019; Ehring, Ehlers, & Glucksman, 2008; García, Vázquez, & Inostroza, 2019; Kleim, Ehlers, & Glucksman, 2012; Mayou, Ehlers, & Bryant, 2002, 2001; Murray et al., 2002). Moreover, experimental studies have shown that a rumination induction following a trauma film or distressing life event scripts increase analogue posttraumatic stress symptoms (Ehring, Fuchs, & Kläsener, 2009; Laposa & Rector, 2012, but see Kubota & Nixon, 2017, for differing results). Taken together, there is strong evidence supporting the view that rumination is an important process involved in the development and, in particular, the

maintenance of PTSD.

Unlike dysfunctional trauma-related appraisals as cognitive *content*, rumination as a cognitive *process* is rarely directly targeted in established treatments for PTSD. Nevertheless, given the important role of rumination in the maintenance of PTSD, we suggest that a reduction in trauma-related rumination may serve as an important mechanism of change in current PTSD treatments as naturally occurring recovery processes can then take place and lead to symptom reduction. In line with this idea, researchers have proposed that targeting cognitive processes such as rumination more directly in PTSD may be beneficial (W. J. Brown et al., 2018; Erwin et al., 2018; Mitchell, Contractor, Dranger, & Shea, 2016). Evidence from studies on depression have shown that rumination is responsive to treatment and decreases to a significant extent (for an overview, see Watkins, 2018). However, to our knowledge no study to date has investigated the relationship generally, or the temporal association between a reduction of rumination and a reduction of PTSD symptoms during trauma-focused treatment in particular.

4. The current study

The aim of the present study was to investigate whether changes in rumination precede and predict changes in PTSD symptomatology, i.e., testing one of the conditions for rumination as a mechanism of change in PTSD treatment. To this end, ruminative thinking and PTSD symptoms were measured at each session during the course of trauma-focused cognitive behavioral therapy. We then examined lagged associations between rumination and symptoms while controlling for autocorrelations, in parallel manner to the studies described above investigating cognitive content as change mechanism (Kleim, Grey, et al., 2013; Schumm et al., 2015). We also explored whether rumination decreased over the course of therapy, which has so far only been shown by Wisco, Sloan, and Marx (2013). We hypothesized that rumination would predict PTSD symptom severity in the following session (H1), controlling for symptom severity of the previous session. To further explore the relationship between rumination and PTSD, we also tested the reverse relationship, i.e., to what extent symptom severity predicts rumination. Since ruminative thinking in PTSD also includes thinking about one's symptoms and is often triggered by a worsening of symptoms, a reduction of symptoms should lead to a reduction of rumination. Therefore, a reciprocal cycle is assumed, and we hypothesized that PTSD symptom severity would predict the degree of rumination in the following session (H2), controlling for the degree of rumination in the previous session.

5. Method

5.1. Participants

Participants were recruited for an effectiveness study that aimed to evaluate an empirically established treatment protocol for PTSD in routine clinical care and to identify predictors of therapy outcome (details of the intervention and its effectiveness are described elsewhere; Krüger-Gottschalk et al., in preparation). Treatment was carried out at the outpatient center of the University of Münster and the outpatient center of the Otto Selz Institute at the University of Mannheim between February 2014 and April 2016 (Krüger-Gottschalk et al., in preparation). No pre-registration was made. The study was approved by the local ethics committees at the Universities of Münster and Mannheim. Inclusion criteria were a primary diagnosis of PTSD assessed with the Clinician-Administered PTSD Scale for DSM-5 (CAPS; Weathers, Blake, et al., 2013), and age of at least 18 years. Patients needing treatment for a current substance dependence, psychotic disorder, or immediate suicide risk, or who had a BMI lower than 17.5 (all assessed via Structured Clinical Interview for DSM-IV [SCID-IV, Wittchen, Zaudig, & Fydrich, 1997]), were excluded from the study. A total of 89 patients took part in the treatment study. This sample size was determined by the effect size the original study aimed to detect. Demographic and clinical characteristics of the sample are shown in Table 1.

All patients referred to the outpatient centers were screened, and eligible patients were informed about the study. When patients were eligible to take part in the study, written informed consent was obtained and they started treatment at the next possible date. Participation in the study was voluntary.

5.2. 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, and all patients followed the same treatment manual that was based on a modularized phase-based approach (see Table 2). Each patient underwent each component, but the number of sessions dedicated to each component and the selection of modules within each component differed from patient to patient. Therapy was provided either by licensed CBT therapists or by therapists in advanced postgraduate training. The average of received sessions of 50-min length was $M = 35.91$ ($SD = 20.72$, range 1–80, $n = 80$), unfortunately, these data were only available for 80 patients. 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 sessions than in previously reported trials on cognitive therapy for PTSD (e.g., Ehlers et al., 2013), but the range and average number of sessions is typical for outpatient treatment for PTSD in the German healthcare system.

5.3. Procedure

At baseline, clinical interviews were administered and patients completed sociodemographic and clinical questionnaires. At the start of every treatment session, patients completed paper-pencil questionnaires

Table 1
Demographic and clinical characteristics of the sample at baseline and descriptive statistics for study variables.

	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[b]	12 (14.3%)
Secondary school[c]	47 (56%)
primary school	3 (3.6%)
no degree	4 (4.8%)
other	10 (11.9%)
Previous inpatient psychiatric stay ($n = 73$)	
Yes	37 (50.7%)
No	36 (49.3%)
Previous outpatient psychotherapy ($n = 68$)	
Yes	42 (61.8%)
No	26 (38.2%)
Comorbidity (assessed via Structured Clinical Interview for DSM-IV)[6]	
No additional diagnosis	42 (49.4%)
One additional diagnosis	22 (25.9%)
Two or more additional diagnoses	21 (24.7%)
Pre-Treatment CAPS-Score ($n = 78$)	38.32 (10.41)
PCL-5[a]	29.51 (20.18)
RIQ-R[a]	7.26 (5.12)

[a] Across all time points ($N = 2372$) and all participants ($N = 88$).
[b] High school: 12–13 years of schooling in the German school system.
[c] Secondary school: 9–10 years of schooling in the German school system.

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

assessing symptom severity and rumination. Therapy sessions were scheduled weekly, although not necessarily seven days apart. 78.92% of observations were obtained from consecutive weeks. Additionally, therapy sessions nearing the end of therapy were spaced out to longer intervals as part of relapse prevention, especially in longer therapies. Patients were asked to complete the questionnaires for the duration of their therapy. Only one patient (1.1%) did not complete any weekly measures.

5.4. Measures

5.4.1. Clinician-Administered PTSD scale for DSM-5 (CAPS-5)

The CAPS-5 (Weathers, Blake, et al., 2013; German translation by Schnyder, 2013) is a structured clinical interview used to assess post-traumatic stress symptoms in the past month. Symptoms are rated on a 5-point Likert scale ranging from 0 (absent) to 4 (extreme), with a symptom being considered present if it is rated with 2 or higher (Weathers et al., 2018). At least one item from each of the clusters "intrusive symptoms" and "avoidance" and at least two items from each of the clusters "changes in mood and cognition" and "hyperarousal" must be present for a diagnosis of PTSD to be made. The CAPS is considered to be the gold standard in PTSD assessment and has shown good psychometric properties across a wide range of research settings and clinical samples (Weathers et al., 2001, 2018).

5.4.2. PTSD-checklist for DSM-5 (PCL-5)

The PCL-5 (Weathers, Litz, et al., 2013; German version: Krüger-Gottschalk et al., 2017) is a 20-item self-report measure assessing PTSD symptom severity in which distress caused by each symptom is rated on a five-point-scale ranging from 0 (not at all) to 4 (extremely). For study aims, it was adapted to assess symptom severity in the past week (as opposed to past month). The German version has high internal consistency ($\alpha = 0.95$) as well as high test-retest reliability ($r = 0.91$, Krüger-Gottschalk et al., 2017). The reliability both within- and between-person in the present sample was good (both *RkRn*[1] and *Rcn*[2] >0.95). The severity score is calculated as a sum of all items (range 0–80). A cut-off of 33 is indicative of probable PTSD (Krüger-Gottschalk et al., 2017). As the DSM-5 criteria for PTSD do not include rumination, neither the CAPS-5 nor the PCL-5 contain any items assessing rumination.

5.4.3. Responses to Intrusions Questionnaire (RIQ)

The rumination subscale of the short Responses to Intrusions Questionnaire (RIQ-R; Clohessy & Ehlers, 1999) was used. The RIQ-R was developed in a series of studies and has been shown to possess good reliability and predictive validity, including internal consistencies of $\alpha = 0.80$-0.86 (Ehring, Ehlers, & Glucksman, 2006; Ehring, Frank, & Ehlers, 2008; Steil & Ehlers, 2000). The rumination subscale consists of seven items measuring the frequency of trauma-related rumination in the past week rated on a scale from 0 (never) to 3 (always), of which a sum score is obtained. Internal consistency in the present sample was good (both RkRn = 0.99, Rcn = 0.78). Participants are instructed to report on what they do when memories of their traumatic event "pop into their mind." Items then assess the extent to which participants engage in repetitive negative thoughts focused on why the trauma happened or how they could have prevented it (e.g., "I dwell on how the event could have been prevented") as well as on how they were before the trauma or who they might have been if the trauma had not happened (e.g., "I think about how life would have been different if the event had not occurred"). The subscale has shown good psychometric properties in previous studies (Ehring & Ehlers, 2014; Kleim et al., 2012). For the present study, a rumination scale consisting of only seven items based on a recommendation by Anke Ehlers (2014, personal communication) was used.

5.5. 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 weekly measurements nested within patients. All models assumed random effects for the intercept and slope, which were allowed to vary across participants. Models were estimated in R (Version 3.4.1; R Core Team, 2021) using packages lme4 and lmerTest (Bates, Mächler, Bolker, & Walker, 2015; Kuznetsova, Brockhoff, & Christensen, 2017) with the restricted maximum likelihood estimation. The level of significance was set as alpha = 0.05. Data of participants who completed fewer than five questionnaire packages were fully excluded, leaving data of 88 participants for analyses. Furthermore, data points that were less than three days apart or more than two months apart were excluded, as they were deemed unreliable. The PCL-5 and the RIQ-R were person-mean centered to predict either the PCL-5 or the RIQ-R at the next assessment occasion. All predictors were standardized with the grand mean and SD to avoid convergence errors.

First, to explore the change in PTSD symptoms over the course of treatment, we estimated an LMM with the PCL-5 total score as the dependent variable and time (formatted as days passed since the first weekly assessment) as a predictor at Level 1 (i.e., within-patient level), with random intercept and slope. The model is specified as follows:

$$Level\ 1: PCL_{ij} = \beta_{0j} + \beta_{1j} * time_{ij} + r_{ij}$$

$$Level\ 2: \beta_{0j} = \gamma_{00} + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + u_{1j}$$

where PCL_{ij} represents the PCL-5 score of the participant *j* at time *i*, $time_{ij}$ represents the number of days passed since the first observation of the participant, and r_{ij} refers to the residual. The intercept (β_{0j}) and slope (β_{1j}) had random effects (u_{0j}, u_{1j}) at the between-person level (Level 2), which allowed the intercept and slope to vary across individuals around the means (i.e., fixed effects) of γ_{00} and γ_{10}.

To explore whether there is a significant change in rumination during the course of psychotherapy, we estimated the same model with the RIQ-R as the dependent variable.

To model the relationships between rumination and PTSD symptom severity, time-lagged mixed models were employed. LMM analyses tested whether symptom severity for person j in week i+1 ($PCL_{(i+1)j}$) was predicted by rumination in week i ($RIQ\text{-}R_{ij}$), after controlling for symptom severity in week i (PCL_{ij}).

[1] RkRn indicates the generalizability of between person differences averaged over time, with time nested within people (Shrout & Lane, 2012).

[2] Rcn indicates the generalizability of within person variations averaged over items (Shrout & Lane, 2012).

$$Level\ 1: PCL_{(i+1)j} = \beta_{0j} + \beta_{1j} * RIQR_{ij} + \beta_{2j} * PCL_{ij} + r_{ij},$$

$$Level\ 2: \beta_{0j} = \gamma_{00} + u_{0j},$$

$$\beta_{1j} = \gamma_{10} + u_{1j}$$

$$\beta_{2j} = \gamma_{20} + u_{2j}$$

In a second step, time since start of treatment was added as an additional predictor (e.g., Curran & Bauer, 2011), because we expected that there are time-related confounds (e.g., natural recovery) that inflate the association between rumination and PTSD symptoms. Also, we assumed that the treatment would have a direct effect on each rumination and PTSD symptoms; therefore, control of the treatment (i.e., time) effect was important to evaluate the unique effect of rumination on PTSD (and that of PTSD symptoms on rumination). Like the other models, the intercept and slopes had random effects at Level 2.

$$Level\ 1: PCL_{(i+1)j} = \beta_{0j} + \beta_{1j} * RIQ - R_{ij} + \beta_{2j} * PCL_{ij} + \beta_{3j} * time_{ij} + r_{ij}$$

However, some authors have highlighted that this is a rather conservative statistical approach, which can also result in overcontrolling (Falkenström, Finkel, Sandell, Rubel, & Holmqvist, 2017). Therefore, results of analyses with time versus analyses without time as an additional predictor are reported and compared in the current study.

Next, we investigated the time-lagged influence of PTSD symptom severity on rumination. We estimated the same model with the RIQ-R as the dependent variable and time since start of treatment as an additional predictor.

6. Results

6.1. Descriptive statistics

Demographic and clinical characteristics of the sample are shown in Table 1. Based on the data of 69 participants, 78.3% endorsed an interpersonal trauma as index trauma. On average, 30.84 observations per participant ($SD = 21.14$) were available. For descriptive statistics of the investigated variables, please see Table 3. The correlation between PCL-5 and the rumination subscale of the RIQ across all time points and participants was 0.61 ($p < .001$).

Table 3
Results of linear mixed models.

	β[a]	*SE*	*t*	*p*
Dependent variable: Symptom severity (PCL-5)				
Intercept	27.33	2.07	13.21	<.001
Time	−11.37	1.07	−10.61	<.001
Changes in rumination over time (RIQ-R)				
Intercept	6.88	0.57	12.08	<.001
Time	−2.30	0.37	−6.2	<.001
Rumination as a predictor for PTSD Symptoms (PCL-5)				
Intercept	29.57	1.92	15.41	<.001
PCL-5 lagged	8.23	0.46	17.91	<.001
RIQ-R lagged	1.16	0.33	3.56	<.001
Rumination as a predictor for PTSD Symptoms (with time as predictor)				
Intercept	28.44	1.95	14.58	<.001
Time	−4.35	0.68	−6.38	<.001
PCL-5 lagged	6.11	0.48	12.75	<.001
RIQ-R lagged	0.57	0.33	1.69	.098
PTSD symptoms as a predictor for rumination (RIQ-R)				
Intercept	7.10	0.50	14.07	<.001
Time	−0.82	0.27	−3.64	<.001
PCL-5 lagged	0.54	0.12	4.4	<.001
RIQ-R lagged	0.89	0.11	8.01	<.001

Note. PCL-5 = PTSD Symptom Checklist for DSM-5; RIQ-R = Responses to Intrusions Questionnaire, Rumination Subscale.

[a] β (=fixed effect) represents the magnitude of change in the outcome variable as the predictor increases by one standard deviation.

6.2. Change in rumination and PTSD symptoms over time

Results of all mixed models are presented in Table 3. We first tested whether there was a significant decrease in rumination and symptom severity over time. To explore whether there was a significant change in PCL-5 scores during the course of therapy, a linear mixed model was estimated with time (formatted as days passed since start of treatment) as a predictor for the outcome. Results showed that time was a significant predictor, indicating that PTSD symptoms decreased over the course of therapy. To explore whether rumination decreased during the course of trauma-focused therapy, a linear mixed model with rumination (RIQ-R) as the dependent variable and time as a predictor was estimated. Results show that time was a significant predictor, showing that trauma-related rumination declined during the course of treatment. See Fig. 1 for illustration.

6.3. Rumination as a predictor for PTSD symptoms

To test whether changes in rumination predict subsequent changes in symptoms of PTSD, an LMM with symptom severity (PCL-5) as the dependent variable and lagged rumination (RIQ-R) scores, as well as lagged PCL scores as predictors, was estimated. Results show that rumination in the preceding week was a significant predictor of subsequent PTSD symptoms. However, when time was added as a predictor to control for the general effect of time on symptoms, rumination was no longer a significant predictor. Only time and symptom severity at the previous assessment point significantly predicted symptom severity in the following session. Fig. 2 represents the estimated auto- and cross-regressive effects for each model. We also tested whether the number of completed assessments moderated the effect of rumination on symptoms, but found that the moderation effect was non-significant.

6.4. PTSD symptoms as a predictor for rumination

To test whether symptom severity would predict the degree of rumination in the following week, an LMM with rumination score (RIQ-R) as the dependent variable and lagged symptom severity scores (PCL-5) and lagged rumination scores (RIQ-R) as predictors was estimated. The inverse effect of symptom severity on successive rumination scores was significant, both with and without time as an additional predictor. Additional analyses showed that the effect was not moderated by number of completed assessments. Please see also Fig. 2.

7. Discussion

We investigated the change of posttraumatic stress symptoms and ruminative thinking and their association over the course of trauma-focused psychological treatment. Specifically, we examined whether the degree of rumination predicted PTSD symptom severity in the following week. We also tested the inverse relationship, namely whether symptom severity would predict the degree of rumination in the following week. Based on the cognitive model of PTSD (Ehlers & Clark, 2000), we assumed that a decrease in rumination would predict a decrease in symptom severity and vice versa.

Results showed that PTSD symptoms decreased significantly over the course of the trauma-focused treatment in a naturalistic setting (for a detailed description of the study findings regarding treatment outcome, see Krüger-Gottschalk et al., in preparation). Further, we found that rumination decreased significantly over the course of the trauma-focused treatment. So far, only one previous study has measured changes in rumination over the course of PTSD treatment. Wisco et al. (2013) found that rumination, measured by two items, significantly decreased from post-treatment to follow-up three months later following five sessions of expressive writing, but did not decrease significantly in

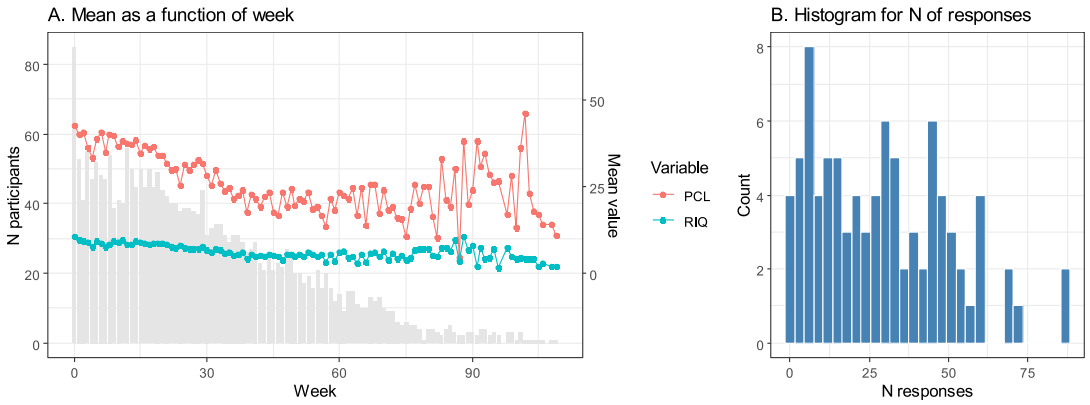

Fig. 1. A. PTSD Symptoms and Rumination Over the Course of Therapy, B. Quantity of Patients by Number of Completed Questionnaires
Note. PCL= PTSD Symptom Checklist for DSM-5; RIQ = Responses to Intrusions Questionnaire, Rumination Subscale. Please note that mean levels of the PCL and RIQ show higher variance at later stages of the therapy due to only a small number of questionnaires completed.

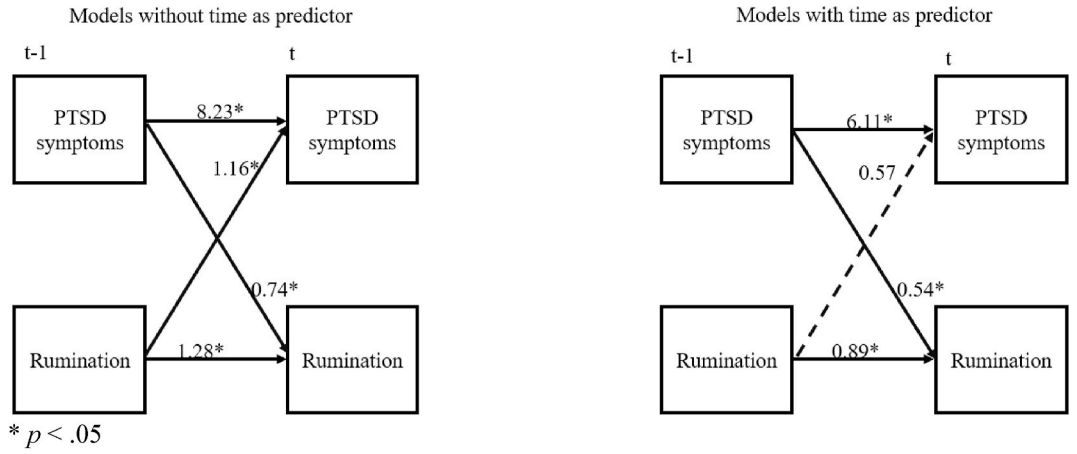

Fig. 2. *Estimated Auto- and Cross-Lagged Effects for Rumination as Predictor for PTSD Symptoms and Vice Versa*
* $p < .05$.

the waitlist-control condition. Although rumination was not directly targeted in the Wisco et al. (2013) study, nor was it directly targeted by the treatment approach used in the current study, several techniques can be expected to be responsible for the reduction of rumination. First, it has been suggested that rumination is associated with an abstract style of processing, serving the function of avoiding trauma-related memories and associated emotions (see e.g., Ehring, Frank, & Ehlers, 2008; Ehring & Ehlers, 2014; Ehring & Watkins, 2008; Michael et al., 2007). Therefore, interventions involving exposure to trauma-related material can be expected to reduce rumination by promoting a more concrete and experiential style of processing trauma-related information. Note that interventions fostering concrete thinking with an experiential focus have been shown to reduce rumination (e.g., Topper, Emmelkamp, Watkins, & Ehring, 2017; Watkins et al., 2012). Second, cognitive interventions can similarly be expected to reduce ruminative thinking by fostering a concrete thinking style that is centered on identifying and changing trauma-focused appraisals on a very specific and concrete level. Finally, psychoeducation about factors maintaining PTSD, as well as behavioral activation in reclaim-your-life-assignments, should have an indirect effect on reducing ruminative thinking. However, because of the flexible application of the different treatment elements in the current study, it cannot be assessed whether the changes in rumination are attributable to any specific elements. Furthermore, due to the lack of a control condition, it cannot be determined whether the observed effect is even due to active treatment ingredients at all or rather to non-specific factors.

Additionally, we examined how changes in PTSD symptoms and changes in rumination are interrelated. In the first step, rumination was found to be predictive of PTSD symptoms in the following week. Contrary to our expectations, rumination no longer significantly predicted symptom severity when time was added as predictor. We also tested the reverse relationship and found – in line with our hypothesis – that symptom severity significantly predicted rumination in the following week, and this association did remain significant after controlling for time.

These findings on the association between changes in rumination and PTSD symptoms are partly consistent with Ehlers and Clark's (2000) cognitive model. As predicted by the model, and suggested by previous research (Michael et al., 2007), symptoms such as intrusions may serve as cues to trigger ruminating about the traumatic event or its meaning ("Why did this happen to me?") as well as rumination about symptoms ("Why can't I get over it?"). It is therefore to be expected that a decrease in symptoms will lead to a decrease in rumination. Importantly, however, the cognitive model of PTSD also posits that rumination is a maintaining factor for symptoms of PTSD; thus, a decrease in ruminative thinking should also predict a decrease in symptom severity. However, this was not supported in our study as rumination did not predict a decrease in symptom severity when time was entered as an additional predictor.

There are several conceivable explanations for the unexpected results on the null effect of rumination on PTSD symptoms after controlling for time. From a methodological point of view, adding time as an

additional predictor to control for effects of time (detrending) may have removed relevant criterion variance, possibly leading to non-significant findings. If the time-trend is a mix of both the general effect of passing time and an interplay between rumination and symptomatology leading to symptom reduction, removing time-trends via detrending will remove both the effect of passing time and the interplay between rumination and symptomatology – including the very effect we are investigating. The effects of such statistical overcontrol have been demonstrated by Falkenström et al. (2017), showing a decrease in effect sizes by up to 70% when detrending is implemented. Thus, while detrending protects against the detection of only spurious relationships, it is a rather conservative analytical approach and we cannot rule out that it may have led to overcontrolling. Nevertheless, we have to conclude that the effect of rumination on symptom severity received less empirical support in our study – i.e., when applying this rigorous analytical approach – than the reverse effect of symptom severity on rumination.

Second, the way in which rumination was assessed in the current study, i.e., using the Responses to Intrusions Questionnaire (RIQ), needs to be considered when interpreting the findings. Rumination is proposed to be a multidimensional construct (García, Duque, & Cova, 2017; Tanner, Voon, Hasking, & Martin, 2013), and it has been suggested that only specific aspects of rumination are related to PTSD (Claycomb, Wang, Sharp, Ractliffe, & Elhai, 2015). So far, there is no scientific consensus which factors constitute rumination in PTSD, with different studies proposing different facets (e.g., Claycomb et al., 2015; García et al., 2017). Moreover, García et al. (2017) have shown that different dimensions of rumination had differential validity in predicting posttraumatic stress symptoms. The measure used here may not reflect all of these facets well. Future research should aim to better define the different dimensions of posttraumatic rumination, and to combine measures to investigate which dimensions could predict symptoms of PTSD. Additionally, due to the nature of the questionnaire used, which specifically asks participants to indicate rumination *in response to* intrusions, it cannot completely be ruled out that the assessment of rumination may have been confounded with current symptom severity (see also Treynor, Gonzalez, & Nolen-Hoeksema, 2003, for a similar discussion regarding symptom confoundation in the assessment of depressive rumination).

Moreover, the treatment investigated in the current study did not directly target rumination. If replicated in future research, our results could imply that – in contrast to cognitive content variables – cognitive processes, such as rumination, may not be a mechanism of change in *current* trauma-focused treatments for PTSD. It remains to be tested whether novel interventions directly targeting rumination may nevertheless show a clinical benefit.

Lastly, an intriguing possibility may be that the effect of rumination on symptom severity may not be uniformly present in all participants, but only for a subgroup of patients. It could be hypothesized that effects of rumination on subsequent symptom decrease might be greater for individuals who score high on trait rumination. Investigating how treatment change comes about for the average individual might therefore blur effects (Schiepek et al., 2020).

7.1. Strengths and limitations

Our study shows a number of important strengths, including the use of a naturalistic sample of patients treated in routine clinical care and assessments of the relevant variables at every session. On the other hand, some limitations of the study need to be considered. As stated above, the uncontrolled design does not allow drawing definite conclusions about the efficacy of the intervention in reducing ruminative thinking. Second, caution is warranted regarding the validity of results for treatment lengths above 60 sessions, as above this point, only a small number of observations was available. However, additional analyses showed that the observed effects were not moderated by the number of completed assessments. Third, the unknown variability in treatment content due to the modular approach of the treatment needs to considered as limitation. Fourth, from our data we cannot differentiate between missed sessions and missed questionnaires. Additionally, our sample consisted mainly of female patients. Reassuringly, however, a recent meta-analysis did not find gender to moderate the relationship between rumination and PTSD symptoms (Szabo et al., 2017). In addition, the gender ratio in our sample is rather typical for treatment-seeking samples of civilian trauma survivors with PTSD (e.g., Ehring et al., 2014; Lewis, Roberts, Andrew, Starling, & Bisson, 2020). Last, we used a single measure of rumination and could not capture different facets of rumination or trait rumination.

7.2. Implications for further research

In sum, the present study provides preliminary evidence that ruminative thinking declines over the course of trauma-focused therapy in patients suffering from PTSD. In addition, although rumination was found to predict symptom severity in the following week, this effect was not found to be robust and fell below significance when we controlled for time. Moreover, the study showed that symptom severity robustly predicted rumination in the following week. More research is needed to test whether the unidirectional (instead of assumed bidirectional) association between symptoms and rumination during the course of treatment is due to methodological issues or can consistently be replicated. The latter may indicate that mechanisms of change in currently existing treatments may include a change of cognitive content rather than process. The lack of consistent support for rumination as a mechanism of change in current treatments does not rule out that novel treatments directly targeting rumination may be efficacious and clinically useful. As rumination has been found to be a transdiagnostic maintaining factor, this may be especially helpful for targeting comorbidity (e.g., depression) in addition to PTSD, with up to 55% of patients with PTSD also suffering from depression (Elhai, Grubaugh, Kashdan, & Frueh, 2008). Future studies would benefit from extending the weekly assessment with additional measures of rumination. A measure less biased by content or the activating event leading to rumination, such as the Perseverative Thinking Questionnaire (Ehring et al., 2011), could be informative, as could more frequent assessments of both rumination and PTSD symptoms using ecological momentary assessments (see Kleim, Graham, et al., 2013). Most importantly, it appears promising to test whether adding rumination-specific interventions is superior to the present treatment protocol for reducing rumination. One would expect the effect of such interventions to be larger than currently observed, which could then help to further clarify the role of rumination as mediator in PTSD treatment. Similarly, the role of rumination-focused interventions in the *prevention* of PTSD is timely. Emphasizing the central role of rumination in the context of PTSD, a large trial currently underway is investigating whether resilience training aimed to reduce rumination *before* exposure to possibly traumatic events is effective in preventing PTSD (Wild et al., 2018).

CRediT authorship contribution statement

Hannah Schumm: Conceptualization, Methodology, Formal analysis, Writing – original draft, Visualization. **Antje Krüger-Gottschalk:** Conceptualization, Methodology, Validation, Investigation, Writing – review & editing. **Anne Dyer:** Conceptualization, Investigation. **Andre Pittig:** Conceptualization, Investigation, Writing – review & editing. **Barbara Cludius:** Methodology, Writing – review & editing. **Keisuke Takano:** Methodology, Formal analysis, Data curation. **Georg W. Alpers:** Conceptualization, Investigation, Writing – review & editing. **Thomas Ehring:** Conceptualization, Methodology, Validation, Investigation, Writing – review & editing, Supervision.

Declaration of competing interest

None.

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