Mechanisms of Change in Posttraumatic Stress Disorder

The Roles of Posttraumatic Cognitions, Rumination, and Sleep

Inaugural-Dissertation

zur Erlangung des Doktorgrades der Philosophie

an der Ludwig-Maximilians-Universität München

Fakultät für Psychologie und Pädagogik

Lehrstuhl für Klinische Psychologie und Psychotherapie

vorgelegt von

Hannah Magdalena Schumm

aus München

München, 2024

Erstgutachter: Prof. Dr. Thomas Ehring

Zweitgutachterin: PD Dr. Larissa Wolkenstein

Tag der mündlichen Prüfung: 09.02.2024

Mechanisms of Change in Posttraumatic Stress Disorder

The Roles of Posttraumatic Cognitions, Rumination, and Sleep

Danksagung

Ich möchte mich bei all denjenigen bedanken, die mich in meiner Promotionsphase unterstützt und begleitet haben, und ohne die die vorliegende Dissertation nicht möglich gewesen wäre.

Ich danke ganz besonders Prof. Dr. Thomas Ehring für die hervorragende Betreuung meiner Doktorarbeit. Deine Vorlesung „Methoden der Psychotherapieforschung“ im Master hat mich für die Forschung begeistert, und Deine erstklassige Betreuung während meiner Promotionsphase hat mich darin immer wieder bestärkt. Ich danke Dir für Deine wertvollen Rückmeldungen und Ideen, Deine Förderung und Ermutigungen, und die vielen Freiheiten, die Du mir immer gewährt hast. Ich bin unglaublich froh um diese Zeit!

Ebenso danke ich PD Dr. Barbara Cludius ganz herzlich für ihre Betreuung meiner Manuskripte und meiner wissenschaftlichen Laufbahn. Danke Dir für die hilfreichen und konstruktiven Rückmeldungen, zum Inhalt oder zum Prozess, und dass du mich immer wieder ermutigt hast. Ich hätte mir keine bessere Mentorin wünschen können.

Weiterhin danke ich PD Dr. Larissa Wolkenstein und Prof. Dr. Frank Padberg sehr herzlich für ihre Zeit und Begutachtung meiner Arbeit.

Meinen Kolleg*innen am Lehrstuhl für Klinische Psychologie und Psychotherapie danke ich für die wundervolle Arbeitsatmosphäre, die guten MAB-Zeiten, den tollen Zusammenhalt und Austausch, und dass ihr als Personen alle so seid, wie ihr seid! Ganz besonders danke ich dabei all meinen Mit-Doktorand*innen für ihre tatkräftige Unterstützung in allen pRaktischen Belangen, vor allem aber den Austausch und die lustigen Zeiten mit euch: ohne euch und unsere PhD-Intervision hätte ich die Höhen und Tiefen der Promotionsphase nicht so gut gemeistert.

Ich danke allen Studienteilnehmer*innen, bekannter- oder unbekannterweise, für ihre Teilnahme und Bereitschaft, an der Forschung mitzuwirken.

Besonders möchte ich mich auch bei allen Hilfskräften und den Therapeut*innen der Hochschulambulanz bedanken. Ohne euch hätte ich die ReCAP-Studie niemals so erfolgreich durchführen können, dafür bin ich euch allen zutiefst dankbar.

Ein unendlicher Dank geht an meine Eltern und meinen Bruder: danke für die Wurzeln und Flügel, und mein immerwährendes Zuhause.

Ich danke auch all meinen Freundinnen und Freunden. Die letzten Jahre hätte ich ohne euch nicht so resilient meistern können, oder es wäre doch alles sehr viel ernster gewesen. Danke

für eure aufmunternden Worte und Ablenkung bei schwierigen Phasen, und vor allem aber danke, dass ihr immer wieder Meilensteine mit mir gefeiert habt!

Aus tiefstem Herzen danke ich Thorsten, meinem ganz frischgebackenen Ehemann. Du warst da, wo ich die Zusage für die Promotionsstelle bekommen habe und während ich Manuskripte geschrieben habe. Du hast die Highs and Lows meines Projektes miterlebt, hast mit mir Reviewer verflucht und meine Meilensteine gefeiert, als wären es deine. Ich freue mich auf die Zukunft!

Abstract

There are several effective psychological treatments for Posttraumatic Stress Disorder (PTSD). However, not all patients appear to benefit from the available treatment options to a satisfying extent. A possible pathway to increasing efficacy and efficiency of existing treatments could be to focus on mechanisms of change, i.e. processes that are responsible for the desired change in symptoms. By establishing mechanisms of change, treatments could be further refined and distilled to their essential elements. For trauma-focused treatments, cognitive models of the disorder have long stressed the importance of cognitive factors and processes, such as posttraumatic cognitions and rumination. Additionally, the role of sleep has received considerable attention. The present thesis first gives a detailed overview of the current empirical evidence for each of these processes as possible mechanism of change. It is then the major aim of the thesis to further investigate the roles of posttraumatic cognitions, rumination, and sleep as possible mechanisms of change in trauma-focused treatment.

Studies I and **II** were conducted with a clinical sample of patients with PTSD who received trauma-focused treatment in a naturalistic setting.

Study I (N = 61) assessed posttraumatic stress symptoms and dysfunctional posttraumatic cognitions every five sessions. We investigated whether changes in posttraumatic cognitions would differentially predict changes in the different PTSD symptom clusters. We found that posttraumatic cognitions predicted subsequent total PTSD symptom severity, and that posttraumatic cognitions predicted three out of four symptom clusters as expected. However, all these effects were no longer statistically significant when the general effect for time was controlled for. Potential limitations might have influenced the results.

Study II (N = 89) assessed posttraumatic stress symptoms and rumination at every session, and examined whether changes in rumination would precede and predict changes in symptoms. We found three main results: first, rumination decreased over the course of therapy.

Second, rumination was a significant predictor of PTSD symptoms in the following week, although this effect was at least partly explained by the time factor. Third, we also found the reverse effect with changes in symptoms predicting changes in rumination. This study provided preliminary evidence for a possible role of rumination as mechanism of change, although further replication is warranted.

Study III focused on the role of sleep in a sample of refugees with PTSD (N = 70). Participants filled out measures of PTSD severity, sleep problems, social impairment, and quality of life. We examined the prevalence of sleep disturbances in this population, their association with PTSD severity and their contribution to social functioning and quality of life over and above the effect of other PTSD symptoms. Results showed a very high prevalence of sleep problems, and significant associations to both self-reported and clinician-rated PTSD severity. Contrary to expectations, sleep problems did not add to the prediction of social impairment or quality of life beyond the effect of other symptoms of PTSD. Several reasons for these in partly unexpected findings are discussed.

Using three studies with clinical samples of patients with PTSD, the present thesis aimed to further elucidate the roles of posttraumatic cognitions, rumination, and sleep, as possible mechanisms of change in trauma-focused treatment. Following different stages of previous research regarding the hypothesized processes, the three studies each tackled different research questions. Findings from these studies add to our knowledge on possible mechanisms in trauma-focused treatments, and hope to stipulate further research. Possible directions for future research into the three mechanisms are outlined. Moreover, potentials and implications for further research on mechanisms of change in general are discussed.

Table of Contents

1. General Introduction

Posttraumatic Stress Disorder

Posttraumatic Stress Disorder (PTSD) is a disabling psychiatric disorder that can result from experiencing a serious traumatic event (American Psychiatric Association [APA], 2013; World Health Organization [WHO], 2019). In the most recent version of the International Classification of Diseases (ICD-11; WHO, 2019), a traumatic event is described as an *event or situation (either short- or long-lasting) of an extremely threatening or horrific nature*, whereas the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013) further specifies such an event as *exposure to actual or threatened death, serious injury, or sexual violence*. PTSD is characterized by symptoms of re-experiencing of the traumatic event, avoidance, hyperarousal, as well as changes in mood and cognitions (APA, 2013; see Table 1.1).

From the time of its first inclusion in DSM-III (American Psychiatric Association, 1980), the diagnostic criteria for PTSD have undergone considerable adaptations. The latent structure of PTSD has been debated by researchers, leading to changes in the number of symptoms and clusters from the previous DSM version (Rasmussen et al., 2018). For the latest DSM version, a new symptom cluster "Negative alternations in cognitions and mood" was added, following accumulating evidence for the important role of dysfunctional posttraumatic cognitions in the development and maintenance of the disorder (Brown et al., 2019). Other criteria such as sleep disturbances, on the other hand, have been included in every version of the DSM since the original formulation of the disorder.

Representative epidemiological studies have estimated the 12-month prevalence of PTSD with 3.7% for the United States (Kessler et al., 2012) – with considerable larger estimates of 8% for women – and 2.3% for Germany (Maercker et al., 2008). Importantly, PTSD is a debilitating disorder associated with severe impairment in functioning and quality of life (Schnurr et al., 2009), causing high societal economic costs (McGowan, 2019). Shortly after exposure to a

traumatic event, many people experience some symptoms of posttraumatic stress. However, only a minority of people go on to develop the full presentation of the disorder (Breslau, 2009; Koenen et al., 2017).

Table 1.1. Diagnostic criteria of PTSD according to DSM-5.

Criterion	Symptom
Stressor	
A	Exposure to actual or threatened death, serious injury, or sexual violence
Re-experiencing	
B1	Recurrent, intrusive distressing memories of the traumatic event
B2	Recurrent distressing dreams of the traumatic event
B3	Feeling or acting as if the traumatic event were recurring
B4	Intense psychological distress at exposure to internal or external reminders
B5	Marked physiological reactions on exposure
Avoidance	
C1	Efforts to avoid memories, thoughts, or feelings associated with the event
C2	Efforts to avoid people, places or situations associated with the event
Negative alterations in mood and cognitions	
D1	Inability to remember an important aspect of the traumatic event
D2	Persistent and exaggerated negative beliefs about oneself, others, or the world
D3	Persistent, distorted cognitions about the cause of the traumatic event
D4	Persistent negative emotional state
D5	Markedly diminished interest or participation in significant activities
D6	Feelings of detachment or estrangement from others
D7	Persistent inability to experience positive emotions
Hyperarousal	
E1	Irritable behavior and angry outbursts
E2	Reckless or self-destructive behavior
E3	Hypervigilance
E4	Exaggerated startle response
E5	Problems with concentration
E6	Sleep disturbance
Duration	
F	Duration of the disturbance of more than 1 month
Functioning	
G	Clinically significant distress or impairment of functioning

Note. The descriptions of the criteria and symptoms were shortened for this table.

Psychological Treatments for PTSD

For those who go on to develop the full clinical picture, several psychological treatment options exist. A large body of evidence has demonstrated both the efficacy and the effectiveness of psychotherapies for PTSD (Bisson et al., 2007; Cusack et al., 2016; Lewis et al., 2020; Watts et al., 2013). It has been shown that those treatments are also effective in the long term (Weber et al., 2021), and for PTSD following various types of traumatic events (e.g. childhood abuse, Ehring et al., 2014; military trauma, Goodson et al., 2011). Moreover, functional outcomes have been shown to improve as well following treatment (for a recent meta-analysis, see Bonfils et al., 2022). According to recent meta-analyses, the best evidence currently exists for trauma-focused treatments, i.e., therapies that directly target the traumatic event (Cusack et al., 2016; Lewis et al., 2020). These therapies are therefore recommended as first-line treatments in several guidelines for PTSD (American Psychiatric Association, 2017; National Institute for Health and Care Excellence, 2018; Schäfer et al., 2019). Example for such trauma-focused treatments include Prolonged Exposure Therapy (Foa et al., 2014), Cognitive Processing Therapy (Resick et al., 2016), Eye Movement Desensitization and Reprocessing (EMDR, Shapiro & Trunk, 2022), and Cognitive Therapy for PTSD (Ehlers et al., 2005).

However, although 44-67% of patients of longer meet the diagnostic criteria for PTSD after treatment, a sizeable portion of patients does not achieve loss of diagnosis and/or remains burdened by symptoms after completing treatment (Bradley et al., 2005; Cusack et al., 2016; Larsen et al., 2019; Steenkamp et al., 2015). Additionally, dropout, the premature termination of treatment on the part of the client, is a frequent problem in trauma-focused psychotherapies (Bisson et al., 2013). A meta-analysis by Imel and colleagues (2013) estimated the dropout rate in clinical trials for PTSD at 36%, while estimates for dropout in naturalistic settings are even higher at 50% (Doran & DeViva, 2018). Taken together, although the efficacy and

effectiveness of trauma-focused psychological treatments for PTSD has been well established, not all patients appear to benefit from the available treatment options. Therefore, there is an urgent task for the future to optimize existing psychotherapies.

Mechanisms of Change

A possible pathway to increase both efficacy and efficiency of existing treatments and to reduce non-response, could be to focus on understanding mechanisms of change in current psychotherapies (Kazdin, 2009). Mechanisms of change are defined as "steps or processes through which therapy (or some independent variable) actually unfolds and produces the change" (Kazdin, 2007, p. 3), in the present case processes that are responsible for changes in PTSD symptoms following trauma-focused psychotherapy (Sripada et al., 2016). It has been argued that by identifying mechanisms that bring about therapeutic change, therapists could focus on these processes to maximize symptom reduction, existing treatments could be further refined and distilled to their essential elements, and thereby therapy efficacy and efficiency could be increased (Alpert et al., 2023; Cuijpers et al., 2019).

A distinction must be made between *moderators*, *mediators*, and *mechanisms of change*. Following Kazdin (2007, 2009), a *moderator* describes a characteristic that influences the relationship between an independent and dependent variable. A *mediator* statistically explains why and in what way a treatment has an effect on an outcome (i.e., an intervention has an effect on symptoms via the mediator). A mediator *can* point towards possible mechanisms of change, but could also be a proxy for variables unrelated to the processes causal for the change. As defined by Kazdin (2007, p. 3), a "mediator is a construct that shows important statistical relations between an intervention and outcome, but may not explain the precise process through which change comes about". Demonstrating statistical mediation is a necessary, yet not sufficient, condition for a hypothesized process to qualify as mechanism of change.

Another critical distinction to be made is between *mechanisms of change* and *active elements* or *active ingredients* of psychotherapy (Cohen et al., 2023; Lorenzo-Luaces et al., 2015). Current evidence-based trauma-focused interventions have been investigated as treatment packages, subsuming a variety of different treatment procedures or components (e.g., Ehlers et al., 2005; Resick et al., 2016). These different components can further be categorized into *inactive* and *active* components (Cohen et al., 2023; Cuijpers et al., 2019). *Inactive* components are thought to have no significant effect on the outcome, whereas *active components* (or sometimes called *active ingredients*) are thought to drive changes in a mechanism, that then result in changes in the outcome. Active components, just like mechanisms of change (see below), can include factors thought to be *specific* for a particular therapeutic approach (e.g., cognitive restructuring) as well as *non-specific* or *common* factors such as therapeutic alliance (Cuijpers et al., 2019). An active ingredient then describes "*the procedures, skills, techniques, and activities in which the therapist and client actively engage to drive the clinical effects"* (Cohen et al., 2023, p. 3), or "*a procedure isolated from a complete treatment package that focuses on changing a process that is expected to lead to change in the outcome*" (Bruijniks et al., 2018, p. 7). A *mechanism of change*, on the other hand, then constitutes the process through which an active element achieves its effect on an outcome (e.g., symptom reduction). In trauma-focused interventions, for example the active element of cognitive restructuring of so-called stuck points is assumed to drive change in posttraumatic cognitions (= mechanism of change), which is then theorized to drive change in PTSD symptoms (= outcome). Interestingly – and further complicating the matter – a single active element could affect multiple mechanisms, and a single mechanism may be activated by diverse active elements (Lorenzo-Luaces et al., 2015).

Criteria for Mechanisms of Change

For a process to count as mechanism of change, several requirements must be met (Kazdin, 2007, 2009). First, a strong association between the intervention and the hypothesized process, as well as between the process and the outcome (e.g., symptoms) must be demonstrated. Second, these associations should be shown to be specific, rather than very general. This means that not multiple mediators should account for the change, but rather a more specific connection should be shown. Third, the cause for a proposed mechanism must be strengthened by evidence from experimental studies, showing that manipulation of the process leads to changes in the outcome. Fourth, studies need to demonstrate a timeline, showing that changes in the proposed mechanism precede changes in the outcome. Accordingly, a process measured only at pretreatment or posttreatment cannot be shown to be a mechanism of change. This – critical – requirement has often been overlooked in previous studies. Fifth, a dose-response-relationship (gradient) between the process and outcome further corroborates the case for a proposed mechanism. Sixth, the results gained from studies should be replicated across different studies, conditions, and samples, yielding consistent results. It is considered crucial to show that the relations between interventions, process, and outcome are not sample-specific, but can be found across a wide range of populations. Seventh and last, the possible role of a mechanism should be argued against a background of a plausible theoretical rationale. There should be a coherent explanation as to how a mechanism operates, from which testable hypotheses can be derived.

Mechanisms of Change in Psychotherapy

In general, common vs. specific mechanisms of change can be distinguished. Common mechanisms describe processes that are argued to be causal across a wider range of treatments, such as for example therapeutic alliance. Specific mechanisms are thought to be specific for certain treatments or interventions, such as for example change in the nature of the trauma

memory would be considered a mechanism of change for trauma-focused treatments for PTSD only (Sripada et al., 2016). For the majority of psychological treatments, a variety of mechanisms are assumed to be at work at any given time, operating on different levels (e.g., cognitive, social, neurobiological), and possibly also influencing each other (Sripada et al., 2016). Considering trauma-focused treatments for PTSD, it is highly likely that therapeutic change is brought on by several different processes – especially since PTSD is such a complex and heterogenous disorder involving multiple different systems (Galatzer-Levy & Bryant, 2013). Enhancing our understanding of possible mechanisms of change in PTSD treatment is therefore the aim of the present thesis. Before possible mechanisms of change in trauma-focused therapy are elaborated in more detail, reasons for studying mechanisms of change in general are discussed.

Reasons to Study Mechanisms of Change

As research on mechanisms of change in psychotherapy requires considerable effort, resources, and time, the benefits of exploring such possible mechanisms should be made clear. Kazdin (2007, 2009) provides clear arguments as to why such research is necessary. First, there is a high number of available treatment options in general, and mechanism research could bring order and parsimony to the multitude of existing treatments. Second, investigating mechanisms of change can bring clarity to the associations between various treatments and the outcomes they have on a broad range of problems. Third, mechanistic research allows for optimizing therapeutic change. Without understanding what is crucial for a treatment success, and how a treatment brings about change, it remains unclear how to further refine existing treatments. By investigating treatment mechanisms, the essential elements can be explored, and interventions can be further improved to achieve their maximum effect on these elements. Additionally, by then focusing on these essential elements (and possibly omitting non-essential ones), psychotherapies could be shortened and hopefully the risk of dropout could be reduced. Fourth,

when more is known about the essential elements of a psychological treatment, the dissemination of the treatment and training of therapists in these treatments is facilitated. Fifth, a clearer picture of how change in therapy comes about can help in identifying moderators of treatment, and lead to better selection of patients suitable for treatment. Finally, looking at the bigger picture, understanding mechanisms of change in psychotherapy could have implications for our knowledge of general human functioning, beyond what is happening in therapy.

Mechanisms of Change in Trauma-Focused Treatments

In the last decade, researchers have begun to investigate possible mechanisms of change in trauma-focused treatments for PTSD (Kangaslampi & Peltonen, 2022; Nixon & Sloan, 2017; Sripada et al., 2016; Zalta, 2015). Cognitive models of PTSD – on which current evidence-based treatments are typically based – stress the importance of cognitive factors and processes for the development and maintenance of the disorder (Ehlers & Clark, 2000; Foa & Cahill, 2001; Resick & Schnicke, 1992). Consequently, cognitive factors such as dysfunctional appraisals or posttraumatic rumination have early on received attention as possible mechanisms of change in these treatments (Sripada et al., 2016). Both of these factors are discussed in detail below. Alongside these factors, other factors such as emotion regulation, organization of the trauma narrative and centrality of the traumatic event, fear extinction, anxiety sensitivity, self-efficacy, or attentional biases, have also been argued to be mechanisms of change (for overviews, see Kangaslampi & Peltonen, 2022; Sripada et al., 2016). Fairly consistent results have been found regarding the role of between-session fear extinction, yet more high-quality research is needed to establish its role as mechanism of change in PTSD treatments (Alpert et al., 2023). Emotion regulation (Sripada et al., 2016) and hope (Gallagher, 2017) have also yielded promising starting points for future investigations, whereas mixed or weak support has been found for the other factors (Alpert et al., 2023; Kangaslampi & Peltonen, 2022).

In addition, the role of sleep in PTSD treatments has received considerable attention in the last decade (Spoormaker & Montgomery, 2008; Zalta et al., 2020). Hence, the present thesis aimed to advance our understanding of the roles of **posttraumatic cognitions**, **rumination**, and **sleep** as mechanisms of change in trauma-focused treatments for PTSD. In the following, the current empirical evidence for each of these factors as mechanisms is reviewed.

Posttraumatic Cognitions

As pointed out above, cognitive models of PTSD posit that dysfunctional cognitive content – so-called posttraumatic cognitions – plays a crucial role in the development and maintenance of the disorder (e.g., Ehlers & Clark, 2000; Resick & Schnicke, 1992). Dysfunctional posttraumatic cognitions include negative appraisals of the traumatic event itself, or its sequelae (Ehlers & Clark, 2000). Common examples are thoughts like "I am going mad", "Nowhere is safe", or "Others cannot be trusted". In general, the assumption that posttraumatic cognitions play a central role in the PTSD has received strong empirical support (for reviews, see Brown et al., 2019; LoSavio et al., 2017). According to the influential cognitive model of PTSD by Ehlers and Clark (2000), posttraumatic cognitions together with a poorly elaborated trauma memory cause an ongoing sense of threat, even if the traumatic event itself has long since passed. This sense of current threat is then proposed to motivate a variety of behavioral and cognitive strategies intended to reduce the current threat, such as thought suppression, avoidance or rumination. These strategies, however, are argued to be maladaptive because they either directly produce PTSD symptoms, prevent change in dysfunctional cognitions, or prevent change in the trauma memory. Similarly, emotional processing theory, which has informed Prolonged Exposure Therapy, posits that posttraumatic cognitions motivate individuals to engage in avoidance behavior, which upholds a pathological fear structure and thereby maintains the disorder (Foa & Cahill, 2001). On the basis of these theoretical models, it has been proposed that the successful reduction of posttraumatic

cognitions during treatment should onset a cascade of events ultimately leading to symptom reduction (Sripada et al., 2016; Zalta, 2015).

Posttraumatic cognitions have been shown to be strongly and specifically associated with symptom severity (Kleim et al., 2013; Oh et al., 2016; ter Heide et al., 2017; for a meta-analysis see Gómez de La Cuesta et al., 2019), and a greater likelihood of meeting criteria for PTSD (Karl et al., 2009; Startup et al., 2007). This association has been shown to be robust across gender, culture, and age (for a review, see Brown et al., 2019), and has also been replicated across a wide range of samples (refugees, ter Heide et al., 2017; veterans and civilians, Gobin et al., 2018; naturalistic samples, Kleim et al., 2013; mothers with childhood adversity, Oh et al., 2016). Not only are posttraumatic cognitions related to PTSD symptoms in cross-sectional studies, they also emerged as a powerful predictor of posttraumatic stress symptoms months and years after the traumatic event (Beierl et al., 2019; Ehring, Ehlers, et al., 2008; Murray et al., 2002; O'Donnell et al., 2007). Further lending support to their purported role as mechanism of change, studies have shown that such dysfunctional cognitions decline during trauma-focused treatments, with such decreases having been observed for various trauma-focused treatments (Cognitive Processing Therapy, Dondanville et al., 2016; Iverson et al., 2015; Prolonged Exposure, Foa & Rauch, 2004; Nacasch et al., 2015; Cognitive Therapy, Kleim et al., 2013), including innovative web-based interventions (Littleton et al., 2012; Wild et al., 2016). Moreover, a dose-response-relationship has been demonstrated, showing that greater reductions in posttraumatic cognitions during treatment are associated with greater reductions in PTSD symptoms (Dondanville et al., 2016; Nacasch et al., 2015; Rauch et al., 2015). By and large, trauma-focused treatments have been shown to reduce posttraumatic cognitions, thereby establishing a robust association between the treatment and proposed mechanism.

Regarding the crucial step of establishing a timeline and showing that changes in the proposed mechanism come before changes in the outcome, Kleim and colleagues (2013) were the first to demonstrate that changes in posttraumatic cognitions preceded and predicted changes in symptomatology during Cognitive Therapy for PTSD. These findings were since replicated across different therapeutic approaches and settings such as residential programs and outpatient settings (Cooper, Zoellner, et al., 2017; Kumpula et al., 2017; McLean et al., 2015; Schumm et al., 2015; Zalta et al., 2014). Further support comes from a study by Wiedemann and colleagues (2020), documenting that changes in posttraumatic cognitions also preceded sudden gains, defined as large symptom improvements occurring from one therapy session to the next. Importantly, the predictive effect of posttraumatic cognitions on changes in posttraumatic stress symptoms has been proven to be distinctly stronger for trauma-focused cognitive-behavioral interventions compared to a minimal attention group, psychopharmaceutical condition, or client-centered therapy (Cooper, Zoellner, et al., 2017; McLean et al., 2015; Zalta et al., 2014). Moreover, the effect appears to be specific to PTSD symptoms (Cooper, Zoellner, et al., 2017), lending further support for the purported role of posttraumatic cognitions as mechanism of change.

In sum, empirical data give robust support for posttraumatic cognitions as mechanism of change in trauma-focused treatments for PTSD. As has been concluded by two recent reviews on mechanisms in PTSD treatment, improvements in posttraumatic cognitions are likely to be a core mechanism of change involved in several interventions for PTSD (Alpert et al., 2023; Kangaslampi & Peltonen, 2022). However, existing studies establishing the timeline have without exception focused on the effect on *total* PTSD symptom severity, even though the multidimensionality of the disorder is well-known (Galatzer-Levy & Bryant, 2013). It is therefore currently unclear whether the different symptom clusters are differentially affected by changes in the proposed mechanism.

Rumination

Another factor that has been implicated to play a central role in the development and maintenance of PTSD, and may also serve as mechanism of change in current PTSD treatments, is posttraumatic rumination (Elwood et al., 2009; Valdez & Lilly, 2017; for a recent review, see Moulds et al., 2020). Trauma-related rumination has been defined as repetitive and recurrent negative thinking about the traumatic event or its consequences (Michael et al., 2007), with patients dwelling on the traumatic event itself, their actions during the event, or the consequences including current symptoms. Although the *content* of posttraumatic rumination can be similar to those of posttraumatic cognitions, the focus hereby lies on the *process* of abstract, perseverative thinking that is often perceived as difficult to control.

In their cognitive model of PTSD, Ehlers and Clark (2000) conceptualize rumination as one of the dysfunctional strategies intended to control the sense of current threat induced by posttraumatic cognitions and a poorly elaborated trauma memory. Rumination is thought to be a form of cognitive avoidance in which a person dwells on the event, without fully processing the traumatic memory itself (Ehlers & Steil, 1995; Michael et al., 2007). It has further been theorized to directly produce symptoms via triggering intrusions (Birrer & Michael, 2011), and to maintain the disorder via preventing change in both posttraumatic cognitions and the trauma memory. Consequently, decreases in rumination over the course of therapy could allow for elaboration of the trauma memory and change in negative appraisals to occur. As a result, posttraumatic stress symptoms are expected to decline.

Rumination is highly prevalent in PTSD, and trauma survivors with a PTSD diagnosis spend significantly more time ruminating than trauma survivors without PTSD (Michael et al., 2007). Studies have shown a strong relationship between rumination and posttraumatic stress symptoms, and these results have been replicated across different samples (Ehring, Frank, et al., 2008; Michael et al., 2007; Steil & Ehlers, 2000; for a meta-analysis, see Szabo et al., 2017).

This relationship is also characterized by a dose-response-relationship (Szabo et al., 2017). Additionally, severity of rumination following a traumatic event is a predictor of PTSD symptoms months and years later (Beierl et al., 2019; Ehring & Ehlers, 2014; García et al., 2019; Kleim et al., 2012). Lending further support for its role as mechanism of change, experimental studies give evidence that a rumination induction following laboratory trauma such as a trauma film or distressing life event scripts increase analogue posttraumatic stress symptoms (Ehring, Fuchs, et al., 2009; Laposa & Rector, 2012; but see Ehring, Szeimies, et al., 2009, for differing results). Regarding the association between intervention and proposed mechanism, so far only one study by Wisco and colleagues (2013) has shown that rumination can decline following a brief exposure intervention for PTSD.

Taken together, empirical studies provide indications for rumination as mechanism of change in trauma-focused treatments. However, essential criteria such as showing an association between the intervention and the proposed mechanism across different studies, and – especially critical – demonstrating a timeline of change between the mechanism and outcome, have not been met yet.

Sleep

Sleep disturbances encompass difficulties falling asleep, staying asleep, early morning awakenings, nightmares, and, as a consequence, limited length and quality of sleep. For a long time, sleep disturbances in PTSD have been regarded as secondary symptom only, and were expected to resolve once the "root problem" of PTSD had been taken care of (Spoormaker & Montgomery, 2008). Accordingly, no specific assessment, diagnosis, or treatment for sleep disturbances was considered necessary. Nowadays, however, sleep disturbances are viewed as core feature of the disorder, at times even as hallmark symptom (Germain, 2013; Spoormaker & Montgomery, 2008). Since the original introduction of the disorder into the DSM, sleep problems have been part of the diagnostic criteria (APA, 1980). Moreover, it has been argued

that sleep problems are an important factor in the development and maintenance of the disorder (Cox et al., 2017). An amelioration of sleep disturbances over the course of therapy could therefore also drive symptom change.

Sleep is essential for cognitive flexibility, integration of new information, general learning processes including emotional processing, and memory consolidation (Goldstein & Walker, 2014) – all processes that are thought to be crucial for effective cognitive-behavioural treatments for PTSD. During psychotherapy, new memory traces are formed that need to be consolidated and integrated, and sleep is thought to foster such processes (Walker, 2009). If sleep problems were to improve over the course of therapy, this could in turn allow for more effective learning processes, thereby promoting symptom change. Moreover, sleep has been argued to be especially important in reducing the degree of emotional arousal associated with certain memories, and to lead to successful affective depotentiation of memories (Pace-Schott et al., 2011; Van Der Helm et al., 2011; Walker & van der Helm, 2009). Since PTSD is characterized by affect-laden intrusive memories causing emotional distress, sleep appears to be especially crucial in this disorder. Further, sleep problems are thought to disrupt extinction learning as well as safety learning (Pace-Schott et al., 2023), two processes that are essential in most trauma-focused therapies, especially in exposure-based treatments. An improvement of existing sleep disturbances could lead to the toning down of emotional memories and enhance extinction learning, thereby driving an improvement in symptoms. This notion is supported by a study by Richards and colleagues (2022), demonstrating that more REM sleep predicted more rapid extinction learning following fear conditioning in trauma-exposed participants. Lastly, when considering how sleep problems may affect treatment response in patients with PTSD, mood and concentration difficulties resulting from poor sleep may pose a hindrance in engaging in intensive trauma-focused psychotherapy. Therefore, if existing sleep problems are alleviated during trauma-focused therapy – be it via targeted interventions or as

a side effect of other interventions not aimed directly at improving sleep – this could allow for learning and memory processes important for treatment success to take place, and thereby serving as a mechanism of change.

The conception of sleep problems as core feature of PTSD is supported by empirical data. Studies recording subjective sleep quality show that 70-90% of patients with PTSD report sleep problems (Koffel et al., 2016; Maher et al., 2006; Ohayon & Shapiro, 2000), sleep too few hours (Taylor et al., 2020), and up to two thirds suffer from nightmares (Pruiksma et al., 2016). These findings have been replicated across diverse trauma samples, such as veterans, survivors of childhood sexual abuse, or mixed trauma (Cox et al., 2017). Additionally, findings regarding subjective sleep quality have been corroborated by physiological data (Baglioni et al., 2016; Richards et al., 2020).

Further considering the criteria put forward by Kazdin (2007) for a process to qualify as mechanism of change, a strong association between sleep problems and PTSD severity has been shown (Cox et al., 2018; Germain et al., 2004), with the association being characterized by a dose-response-relationship (DeViva et al., 2004). Further evidence for sleep as mechanism of change comes from studies showing that sleep problems are not merely a consequence of increased burden by posttraumatic stress symptoms such as intrusions and such, but that sleep problems predict PTSD in the long term. It has repeatedly been demonstrated that disturbed sleep before or in the immediate aftermath of a traumatic event increases the likelihood of developing PTSD (Babson et al., 2011; Kobayashi & Mellman, 2012), whereas the opposite was not observed (i.e., posttraumatic stress symptoms did not predict subsequent sleep problems, Wright et al., 2011). In veterans, pre-deployment sleep problems were shown to predict PTSD symptoms two years after their mission over and above the effect of PTSD symptoms (Koffel et al., 2013). Similar results have been replicated for earthquake survivors (Fan et al., 2017). These results have further been supported by objective data, showing that

fragmented Rapid Eye Movement (REM) sleep immediately after a traumatic event predicted PTSD symptoms months later (Mellman et al., 2007). Using newer assessment methods such as ecological momentary assessment (EMA), which allows for investigating the interplay between sleep problems and posttraumatic stress symptoms at a day-to-day level, recent studies have further corroborated the role of sleep as possible mechanism in PTSD. For example, Short and colleagues (2017) demonstrated that poor sleep quality was associated with increased PTSD symptom severity the next day, which could not be accounted for by prior evening's symptom severity. Results have since been replicated for different populations, including first-responders from the World Trade Center attacks (Dietch et al., 2019), and veterans (DeViva et al., 2020). The latter study also demonstrated that increased daytime PTSD symptoms did not predict sleep problems the following night, thereby further showing the specific, unidirectional association between sleep disturbances and posttraumatic stress symptoms.

Fulfilling another criterion for establishing sleep's role as mechanism in PTSD, experimental studies have manipulated sleep problems and investigated its effect on (analogue) PTSD symptoms. Kleim and colleagues (2016) demonstrated that experimental sleep deprivation following an analogue traumatic stressor significantly increased the number of intrusive memories. In contrast, for participants that slept following the analogue trauma, sleep reduced the affective tone and emotional distress associated with these intrusive memories. These findings have recently been replicated by Zeng et al. (2021), lending strong support for sleep as mechanism of change.

The high prevalence of sleep disturbances in patients with PTSD has led researchers to take a closer look on the possible adverse effects on trauma-focused treatment, with several studies giving evidence that sleep disturbances negatively impact treatment response. Looking at anxiety disorders in general as well as PTSD, baseline sleep disturbances have been shown to be independently associated with worse treatment response in cognitive-behavioural

therapies (Marcks et al., 2010; Ramsawh et al., 2016) and that experimental manipulation of sleep negatively impacts treatment response (Pace-Schott et al., 2012). Taylor and colleagues (2020) demonstrated that baseline markers of poor sleep such as insomnia, nightmares, and excessive daytime sleepiness, predicted higher PTSD symptoms across treatment course. Similarly, it was shown that poorer sleep quality at baseline predicted slower rate of symptom improvement in patients with PTSD and comorbid depression (Lommen et al., 2016). Importantly, researchers have begun to establish a timeline of changes in the hypothesized mechanism of change, i.e., sleep, and PTSD symptoms. A seminal study by Zalta and colleagues (2020) is providing first evidence that improvement of sleep problems during PTSD treatment improves the likelihood of treatment success, as patients with more improvements in sleep problems over the course of therapy had more positive treatment outcomes. However, sleep disturbances were only measured at two timepoints, whereas it has been argued that three measurement points are required at the very least to demonstrate a timeline (Kraemer et al., 2002). To provide strong evidence for a timeline, future studies should ensure to measure both sleep and posttraumatic stress symptoms more frequently over the course of treatment.

In sum, there is considerable evidence for the role of sleep as mechanism of change. Currently, evidence establishing that changes in sleep problems precede and predict changes in symptomatology over the course of trauma-focused treatment, is still lacking, and needs to be investigated. Another very important criterion to establish a mechanism following Kazdin (2007), is to demonstrate consistency of findings across samples and populations. The majority of above-mentioned studies have focused on civilian and veteran samples (e.g., Short et al., 2017; Zalta et al., 2020). However, a group that is at high risk for PTSD and with high prevalences of sleep disturbances, are refugees. Currently, around 90 million people are thought to be forcibly displaced (United Nations High Commissioner for Refugees [UNHCR], 2021), with the prevalence of PTSD in this group being estimated at 30% (Steel et al., 2009).

Additionally, studies show an extremely high prevalence of sleep disorders in refugees in general, ranging from 39 to 99% (Richter et al., 2020). Yet, for refugees with PTSD, there is so far only rudimentary empirical evidence for the role of sleep as mechanism of change. Only two studies have investigated the prevalence of sleep disturbances in refugees with PTSD (Sandahl et al., 2017, 2021), showing a high prevalence of sleep disturbances in this group. However, one of these studies measured sleep disturbances with a single item with questionable psychometric properties (Sandahl et al., 2017). When looking at the criterion to establish an association between mechanism and outcome, only a single study has provided preliminary findings (Sandahl et al., 2021). To further corroborate the role of sleep as mechanism of change, an extension of above-mentioned findings coming from non-refugee samples with PTSD to the group of refugees is therefore necessary.

Aims of the Thesis

The present thesis aimed to improve our understanding of possible mechanisms of change in trauma-focused treatments for PTSD. Specifically, the roles of posttraumatic cognitions, rumination, and sleep disturbances as purported mechanisms of change were investigated in more detail. To this end, three studies including patients with PTSD were conducted.

Study I

As has been described in detail above, there is considerable and robust evidence for the role of posttraumatic cognitions as mechanism of change in trauma-focused treatments for PTSD. However, existing studies establishing the timeline have without exception investigated the effect of posttraumatic cognitions on *total* PTSD symptom severity, even though the multidimensionality of the disorder is well-known (Galatzer-Levy & Bryant, 2013). As is reflected in the diagnostic criteria for the disorder, PTSD consists of several distinct symptom

clusters that markedly differ from each other (APA, 2013). These symptom clusters show different trajectories of change (Schell et al., 2004), and relate differentially to factors such as depression (Wang et al., 2015), suicidal ideation (Hellmuth et al., 2012), impulsivity (Armour et al., 2016), alcohol and substance misuse (Bonn-Miller et al., 2013; Walton et al., 2018), as well as quality of life (Lunney & Schnurr, 2007) and social functioning (Monson et al., 2012). Moreover, studies show that different aspects of emotion regulation and rumination – both processes that have been implicated as possible mechanisms of change – show unique relationships to the symptom clusters (Mitchell et al., 2016; Short et al., 2016). **Study I** therefore aimed to extend previous findings on the role of posttraumatic cognitions as mechanism of change by investigating the relationship between cognitions and symptom clusters over the course of trauma-focused psychotherapy in more detail.

Study II

Rumination has been proposed to play a central role in the maintenance of PTSD, and empirical studies cautiously support this notion (Moulds et al., 2020). However, important criteria for its role as mechanism of change have not been investigated yet, and more research is necessary. **Study II** aimed to further investigate rumination as mechanism of change in trauma-focused treatment, by showing an association between the intervention and the proposed mechanism. Moreover, **Study II** set out to establish a timeline between rumination and symptom reduction, by testing whether changes in rumination preceded and predicted changes in symptomatology during trauma-focused treatment in a naturalistic sample.

Study III

Considerable empirical evidence hints at sleep as possible mechanism of change, as sleep disturbances are thought to hinder learning and memory processes crucial for trauma-focused treatment (Goldstein & Walker, 2014; Pace-Schott et al., 2023). Improvements in sleep

could therefore lead to changes in symptoms of posttraumatic stress, by fostering essential learning processes.

To establish the role of sleep as possible mechanism of change, establishing a timeline by repeatedly measuring both the hypothesized mechanism and the outcome over the course of therapy would be the required next step. However, as put forward above, research is still at an earlier stage for the subgroup of refugee samples. Considering that substantially less treatment options exist for this population, investigating treatment mechanisms and ultimately refining treatments appears to be especially relevant here, so that limited treatment options can be used at maximum efficiency. For this sample – a group at high risk for both PTSD and sleep disturbances (Richter et al., 2020) – other criteria such as the relevance of an purported mechanism, or a strong association between process and outcome, have not been met yet. Compared to Study I and Study II, **Study III** therefore addresses different – and somewhat more basic – criteria to establish a mechanism of change. **Study III** aimed to extend previous findings on the relevance of sleep problems in patients with PTSD, by exploring the prevalence of sleep disturbances in refugees with PTSD currently living in Germany. Moreover, **Study III** investigated the association between sleep disturbances and both clinician-rated and self-reported PTSD symptoms, extending earlier findings to a different population. Such an extension of findings can further corroborate the role of sleep as mechanism of change, and form the starting point for eventually examining whether changes in sleep disturbances precede and predict changes in symptomatology over the course of treatment.

As mentioned above, Kazdin (2007) in his highly influential article argued that investigating mechanisms of change can help in understanding the broad outcomes on different problems (e.g., social, emotional, behavioral) that psychotherapeutic interventions often have. Further, Kazdin (2007) argued that understanding the mechanisms could also assist in understanding human functioning on a broader scope. Taking these arguments into account,

Study III also investigated the predictive effect of sleep disturbances on broader outcomes, namely social functioning and quality of life.

The obtained results of all three studies are then discussed and integrated, taking limitations of the studies into account. Moreover, implications for future research on mechanisms of change in PTSD treatment are outlined.

2. Study I: Do changes in dysfunctional posttraumatic cognitions differentially predict PTSD symptom clusters?

This chapter is a post-peer-review, pre-copyedit version of a brief report published in *Journal of Consulting and Clinical Psychology*:

Schumm, H., Krüger-Gottschalk, A., Ehring, T., Dyer, A., Pittig, A., Takano, K., Alpers, G.W., Cludius, B. (2023) Do changes in dysfunctional posttraumatic cognitions differentially predict PTSD symptom clusters? *Journal of Consulting & Clinical Psychology*. 10.1037/ccp0000817.

© 2023, American Psychological Association. This paper is not the copy of record and may not exactly replicate the final, authoritative version of the article. Please do not copy or cite without authors' permission. The final article will be available, upon publication, via its DOI: 10.1037/ccp0000817

Abstract

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 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 CBT 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 time point 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.

Conclusions: The present study provides preliminary evidence that dysfunctional posttraumatic cognitions predict PTSD symptom clusters differentially. However, different findings when employing a traditional vs. a more rigorous statistical approach makes interpretation of findings difficult.

Introduction

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 has 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 (H1). 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 overlap. 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 (H2). 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 (H3). 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, H4) 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 JARS (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 pre-registered.

Participants

Participants were recruited for a multi-center 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., in preparation; 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 DSM-5 (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 BMI 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 current study could be obtained. Analyses were therefore based on 61 participants. Demographic and clinical characteristics of the sample are shown in Table 2.1.

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 2.2). Therapy was provided either by licensed CBT therapists or by therapists in advanced postgraduate training. Based on the data from 80 patients, the average number of sessions (50 min each) was M = 35.91 (SD = 20.72, range 1-80)[1].

[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

Measures

PTSD-Checklist for DSM-5 (PCL-5)

The 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 five-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, i.e., cluster B (items 1-5), cluster C (items 6-7), cluster D (items 8-14), and cluster E (items 15-20).

Posttraumatic Cognitions Inventory (PTCI) – Short Version

The 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 seven-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

cognitive therapy for PTSD (e.g., Ehlers et al., 2013, *M* = 10.6). However, these studies have typically employed sessions of 90-minute 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 healthcare 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)

paper-pencil questionnaires assessing symptom severity and posttraumatic cognitions. Therapy sessions were scheduled weekly, although not necessarily seven 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 was 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 Supplementary Material.

Results

Descriptive Statistics

Descriptive statistics of the investigated variables are shown in Table 2.1. On average, 5.79 observations per participant (*SD* = 3.62) were available. The correlation between the PCL-5 and PTCI across all time points 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 2.3). However, with time as additional predictor to control for the general effect of time, this effect was non-significant. Regarding the prediction of the different symptom clusters (see Table 2.4), as hypothesized cluster B (re-experiencing), cluster C (avoidance) and 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 2.5). 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 reductions 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* was 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 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 non-significant 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 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. Lastly, 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. 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). 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.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. https://doi.org/https://doi.org/10.1176/appi.books.9780890425596

Bates, D., Mächler, M., Bolker, B. M., & Walker, S. C. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, *67*(1), 1–48. https://www.jstatsoft.org/index.php/jss/article/view/v067i01/v67i01.pdf

Beierl, E. T., Böllinghaus, I., Clark, D. M., Glucksman, E., & Ehlers, A. (2019). Cognitive paths from trauma to posttraumatic stress disorder: A prospective study of Ehlers and Clark's model in survivors of assaults or road traffic collisions. *Psychological Medicine*, 1–10. https://doi.org/10.1017/S0033291719002253

Brown, L. A., Belli, G. M., Asnaani, A., & Foa, E. B. (2019). A review of the role of negative cognitions about oneself, others, and the world in the treatment of PTSD. *Cognitive Therapy and Research*, *43*(1), 143–173. https://doi.org/10.1007/s10608-018-9938-1

Cooper, A. A., Zoellner, L. A., Roy-Byrne, P., Mavissakalian, M. R., & Feeny, N. C. (2017). Do changes in trauma-related beliefs predict PTSD symptom improvement in prolonged exposure and sertraline? *Journal of Consulting and Clinical Psychology*, *85*(9), 873–882. https://doi.org/10.1037/ccp0000220

Cusack, K., Jonas, D. E., Forneris, C. A., Wines, C., Sonis, J., Middleton, J. C., Feltner, C., Brownley, K. A., Olmsted, K. R., Greenblatt, A., Weil, A., & Gaynes, B. N. (2016). Psychological treatments for adults with posttraumatic stress disorder: A systematic review and meta-analysis. *Clinical Psychology Review*, *43*, 128–141. https://doi.org/10.1016/j.cpr.2015.10.003

Ehlers, A., & Boos, A. (1999). Fragebogen zu Gedanken nach traumatischen Erlebnissen (PTCI). In A. Ehlers (Ed.), *Posttraumatische Belastungsstörung* (pp. 92–93). Hogrefe.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder.

Behaviour Research and Therapy, *38*(4), 319–345. https://doi.org/10.1016/S0005-7967(99)00123-0

Ehlers, A., Grey, N., Wild, J., Stott, R., Liness, S., Deale, A., Handley, R., Albert, I., Cullen, D., Hackmann, A., Manley, J., McManus, F., Brady, F., Salkovskis, P., & Clark, D. M. (2013). Implementation of cognitive therapy for PTSD in routine clinical care: Effectiveness and moderators of outcome in a consecutive sample. *Behaviour Research and Therapy*, *51*(11), 742–752. https://doi.org/10.1016/j.brat.2013.08.006

Falkenström, F., Finkel, S., Sandell, R., Rubel, J. A., & Holmqvist, R. (2017). Dynamic models of individual change in psychotherapy process research. *Journal of Consulting and Clinical Psychology*, *85*(6), 537–549. https://doi.org/10.1037/ccp0000203

Falkenström, F., Solomonov, N., & Rubel, J. A. (2022). How to Model and Interpret Cross-Lagged Effects in Psychotherapy Mechanisms of Change Research: A Comparison of Multilevel and Structural Equation Models. *Journal of Consulting and Clinical Psychology*, *90*(5), 446–458. https://doi.org/10.1037/ccp0000727

Galatzer-Levy, I. R., & Bryant, R. A. (2013). 636,120 ways to have posttraumatic stress disorder. *Perspectives on Psychological Science*, *8*(6), 651–662. https://doi.org/10.1177/1745691613504115

Gómez de La Cuesta, G., Schweizer, S., Diehle, J., Young, J., & Meiser-Stedman, R. (2019). The relationship between maladaptive appraisals and posttraumatic stress disorder: A meta-analysis. *European Journal of Psychotraumatology*, *10*(1). https://doi.org/10.1080/20008198.2019.1620084

Kazak, A. E. (2018). Editorial: Journal article reporting standards. *American Psychologist*, *73*(1), 1. https://doi.org/10.1037/AMP0000263

Kleim, B., Grey, N., Wild, J., Nussbeck, F. W., Stott, R., Hackmann, A., Clark, D. M., & Ehlers, A. (2013). Cognitive change predicts symptom reduction with cognitive therapy for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *81*(3),

383–393. https://doi.org/10.1037/a0031290

Krüger-Gottschalk, A., Knaevelsrud, C., Rau, H., Dyer, A., Schäfer, I., Schellong, J., & Ehring, T. (2017). The German version of the Posttraumatic Stress Disorder Checklist for DSM-5 (PCL-5): Psychometric properties and diagnostic utility. *BMC Psychiatry*, *17*(1). https://doi.org/10.1186/s12888-017-1541-6

Kumpula, M. J., Pentel, K. Z., Foa, E. B., LeBlanc, N. J., Bui, E., McSweeney, L. B., Knowles, K., Bosley, H., Simon, N. M., & Rauch, S. A. M. (2017). Temporal sequencing of change in posttraumatic cognitions and PTSD symptom reduction during prolonged exposure therapy. *Behavior Therapy*, *48*(2), 156–165. https://doi.org/10.1016/j.beth.2016.02.008

Lewis, C., Roberts, N. P., Andrew, M., Starling, E., & Bisson, J. I. (2020). Psychological therapies for post-traumatic stress disorder in adults: Systematic review and meta-analysis. *European Journal of Psychotraumatology*, *11*(1), 1729633. https://doi.org/10.1080/20008198.2020.1729633

Schell, T. L., Marshall, G. N., & Jaycox, L. H. (2004). All symptoms are not created equal: The prominent role of hyperarousal in the natural course of posttraumatic psychological distress. *Journal of Abnormal Psychology*, *113*(2), 189–197. https://doi.org/10.1037/0021-843X.113.2.189

Schumm, H., Krüger-Gottschalk, A., Dyer, A., Pittig, A., Cludius, B., Takano, K., Alpers, G. W., & Ehring, T. (2021). Mechanisms of change in trauma-focused treatment for PTSD: The role of rumination. *Behaviour Research and Therapy*, *148*, 104009. https://doi.org/10.1016/j.brat.2021.104009

Schumm, J. A., Dickstein, B. D., Walter, K. H., Owens, G. P., & Chard, K. M. (2015). Changes in posttraumatic cognitions predict changes in posttraumatic stress disorder symptoms during cognitive processing therapy. *Journal of Consulting and Clinical Psychology*, *83*(6), 1161–1166. https://doi.org/10.1037/ccp0000040

Shrout, P., & Lane, S. P. (2012). Psychometrics. In M. R. Mehl & T. S. Conner (Eds.), *Handbook of Research Methods for Studying Daily Life* (pp. 302–320). Guilford Press.

Zalta, A. K., Gillihan, S. J., Fisher, A. J., Mintz, J., Mclean, C. P., Yehuda, R., & Foa, E. B. (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology*, *82*(1), 171–175. https://doi.org/10.1037/a0034735

Table 2.1. 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[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%)
Pre-Treatment PTSD symptom severity (CAPS[c]) (*n* = 78)	38.32 (10.41)
Pre-Treatment self-reported PTSD symptom severity (PCL-5)	38.42 (19.36)
Self-reported symptom severity (PCL-5) across all time points[d]	29.23 (20.66)
Self-reported dysfunctional cognitions (PTCI)[d]	72.80 (25.65)
Repeated measures correlation PTCI & PCL-5 sum score	$r = 0.67$ ($p < .001$
Repeated measures correlation PTCI & PCL-5 cluster B	$r = 0.54$ ($p < .001$
Repeated measures correlation PTCI & PCL-5 cluster C	$r = 0.58$ ($p < .001$
Repeated measures correlation PTCI & PCL-5 cluster D	$r = 0.65$ ($p < .001$
Repeated measures correlation PTCI & PCL-5 cluster E	$r = 0.58$ ($p < .001$

Note. PCL-5 = PTSD Symptom Checklist for DSM-5; PTCI = Posttraumatic Cognitions Inventory

[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 time points (*N* = 317) and all participants (*N* = 61).

Table 2.2. 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

Table 2.3. Results of LMMs for Total PCL-5 score as Dependent Variable

	Model 1				Model 2				Model 3			
Variable	β[1]	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. PCL-5 = PTSD Symptom Checklist for DSM-5; PTCI = Posttraumatic Cognitions Inventory. *Lagged* variables represent the autocorrelations between a score at timepoint i and timepoint i+1.

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

Table 2.4. Results of LMMs for PCL-5 Cluster Scores as Dependent Variables (DV)

	Model 1				Model 2			
DV: PCL-5 Cluster B								
Variable	β[1]	SE	t	p	β	SE	t	p
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								
Variable	β	SE	t	p	β	SE	t	p
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								
Variable	β	SE	t	p	β	SE	t	p
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								
Variable	β	SE	t	p	β	SE	t	p
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. PCL-5 = PTSD Symptom Checklist for DSM-5; PTCI = Posttraumatic Cognitions Inventory. *Lagged* variables represent the autocorrelations between a score at timepoint i and timepoint i+1.
[1] β (=fixed effect) represents the magnitude of change in the outcome variable as the predictor increases by one standard deviation.

Table 2.5. Results of LMMs for Dysfunctional Cognitions DV (Testing Reverse Relationships)

	Model 1				Model 2			
DV: PTCI								
Variable	β[1]	SE	t	p	β	SE	t	p
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								
Variable	β	SE	t	p	β	SE	t	p
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								
Variable	β	SE	t	p	β	SE	t	p
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								
Variable	β	SE	t	p	β	SE	t	p
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

Supplementary Material

Statistical Analyses

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 assessment) as a predictor at Level 1 (i.e., within-person 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}.

Second, to explore whether there is a significant change in dysfunctional posttraumatic cognitions during the course of psychotherapy, we estimated the same model with the PTCI as the dependent variable.

The model is specified as follows:

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

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

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

Next, to model the relationship between cognitions and PTSD *total* symptom severity, time-lagged mixed models were employed to test whether total symptom severity for person j

at timepoint i+1 ($PCL_{(i+1)j}$) was predicted by posttraumatic cognitions at timepoint i ($PTCI_{ij}$), after controlling for symptom severity at timepoint i (PCL_{ij}).

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

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

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

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

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 posttraumatic cognitions and PTSD symptoms. Therefore. controlling for the time effect – so-called "detrending" was applied to protect against the detection of only spurious relationships. Also, we assumed that the treatment would have a direct effect on each cognitions and PTSD symptoms; therefore, control of the treatment (i.e., time) effect was important to evaluate the unique effect of cognitions on PTSD. Like the other models, the intercept and slopes had random effects at Level 2.

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

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

Then, the differential associations over time between posttraumatic cognitions and each of the PTSD symptom *clusters* were investigated. Again, time-lagged mixed models were employed to test whether symptom severity of cluster B, C, D & E, respectively, for person j at timepoint i+1 (Cluster $X_{(i+1)j}$) was predicted by posttraumatic cognitions at timepoint i ($PTCI_{ij}$), after controlling for severity of the respective cluster at timepoint i (PCL_{ij}).

For cluster B (and analogue for clusters C, D & E), the model was specified as follows:

$$Level\ 1: Cluster\ B_{(i+1)j} = \beta_{0j} + \beta_{1j} * PTCI_{ij} + \beta_{2j} * Cluster\ B_{ij} + r_{ij}$$

Like the other models, the intercept and slopes had random effects at Level 2. Again, in a second step, time since start of treatment was added as additional predictor to control for possible time-related confounds. The model for cluster B (and analogue for clusters C, D & E), is then specified as follows (with random effects of intercepts and slopes at Level 2):

$$Level\ 1: Cluster\ B_{(i+1)j} = \beta_{0j} + \beta_{1j} * PTCI_{ij} + \beta_{2j} * Cluster\ B_{ij} + \beta_{3j} * time_{ij} + r_{ij}$$

Again, results of analyses with and without time as additional predictor are reported and compared in the current study.

For exploratory aims, we also tested all reverse relationships between posttraumatic cognitions and the PTBS symptoms clusters. All models were tested both with and without controlling for time.

3. Study II: Mechanisms of Change in Trauma-Focused Treatment for PTSD: The Role of Rumination

This chapter is a post-peer-review, pre-copyedit version of an article published in *Behaviour Research and Therapy*:

Schumm, H., Krüger-Gottschalk, A., Dyer, A., Pittig, A., Cludius, B., Takano, K., Alpers, G. W. & Ehring, T. (2022). Mechanisms of change in trauma-focused treatment for PTSD: The role of rumination. *Behaviour Research and Therapy*, 148, 104009.

The final authenticated version is available online at:

https://doi.org/10.1016/j.brat.2021.104009

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-nine 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.

Introduction

There is ample evidence for the efficacy and effectiveness of cognitive behavioral treatments for PTSD (Bisson et al., 2007; Bradley et al., 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). 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.

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.

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 et al., 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 et al., 2019; for a recent meta-analysis see Gómez de La Cuesta et al., 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 et al., 2019; Ehring, Ehlers, et al., 2008; Murray et al., 2002; O'Donnell et al., 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 et al., 2017; Dondanville et al., 2016; Iverson et al., 2015; Kleim, Grey, et al., 2013; Littleton et al., 2012) and more importantly, they precede as well as predict symptom amelioration (Kleim, Grey, et al., 2013; Kumpula et al., 2017; McLean et al., 2015; Schumm et al., 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 et al. (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.

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 et al., 2007), has been suggested as playing a key

role in the development and maintenance of PTSD (Ehring, Frank, et al., 2008; Elwood et al., 2009; Valdez & Lilly, 2017; for a recent review, see Moulds et al., 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, et al., 2008; Michael et al., 2007; Steil & Ehlers, 2000; for a recent meta-analysis, see Szabo et al., 2017). This effect remains significant after controlling for negative affect (Arditte Hall et al., 2019; Seligowski et al., 2016), and even fully accounts for the relationship between negative affect and PTSD (W. J. Brown et al., 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, et al., 2008; García et al., 2019; Kleim et al., 2012; Mayou et al., 2001, 2002; 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 et al., 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 et al., 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.

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 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 et al. (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.

Method

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 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 3.1.

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.

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 3.2). 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-minute 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.

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 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.

Measures

Clinician-Administered PTSD Scale for DSM-5 (CAPS-5)

The CAPS-5 (Weathers et al., 2013; German translation by Schnyder, 2013) is a structured clinical interview used to assess posttraumatic 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).

PTSD-Checklist for DSM-5 (PCL-5)

The PCL-5 (Weathers 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 ($\alpha = .95$) as well as high test-retest reliability ($r = .91$, Krüger-Gottschalk et al., 2017). The reliability both within- and between-

person in the present sample was good (both R_{kR}[4]$_n$ and R_{cn}[5] >.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.

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 α = .80-.86 (Ehring et al., 2006; Ehring, Frank, et al., 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 R_{kRn} = .99, R_{cn} =.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.

Statistical Analyses

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

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

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, 2020) using packages lme4 and lmerTest (Bates et al., 2015; Kuznetsova et al., 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}).

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

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

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

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

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 et al., 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.

Results

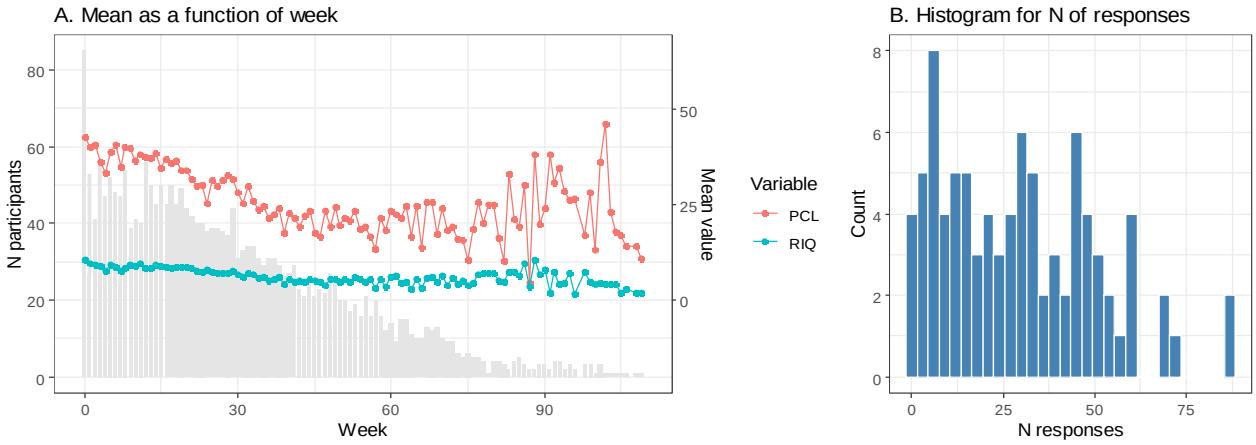
Descriptive Statistics

Demographic and clinical characteristics of the sample are shown in Table 3.1. 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 3.3. The correlation between PCL-5 and the rumination subscale of the RIQ across all time points and participants was 0.61 ($p < .001$).

Change in Rumination and PTSD Symptoms Over Time

Results of all mixed models are presented in Table 3.3. 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 Figure 3.1 for illustration.

Figure 3.1. 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.

Rumination as a Predictor for PTSD Symptoms

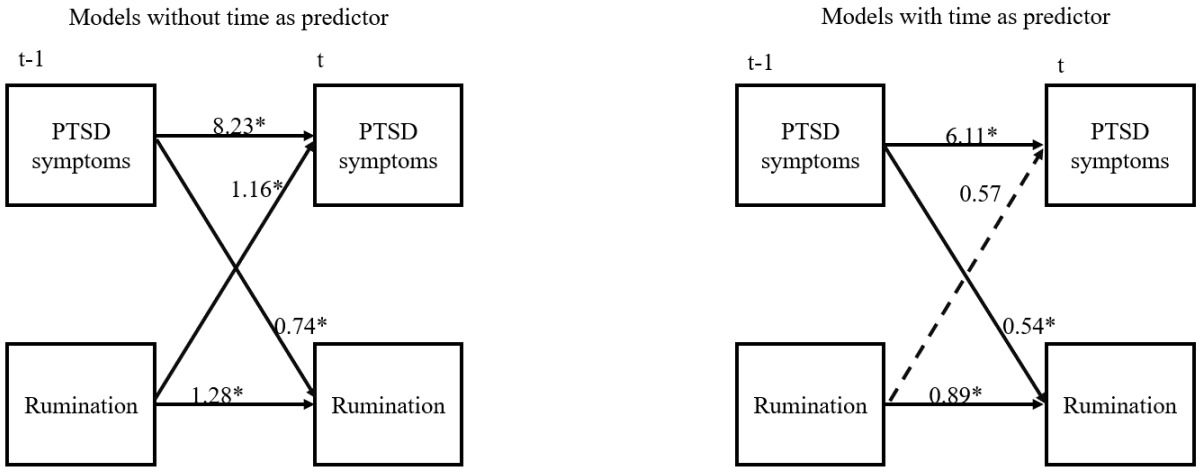
To test whether changes in rumination predict subsequent changes in symptoms of PTSD, an LMM with symptom severity (PCL-5) as the dependent variable and lagged rumination (RIQ-R) scores, as well as lagged PCL scores as predictors, was estimated. Results show that rumination in the preceding week was 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. Figure 3.2 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.

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 Figure 3.2.

Figure 3.2. Estimated Auto- and Cross-Lagged Effects for Rumination as Predictor for PTSD Symptoms and Vice Versa



* $p < .05$

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 and colleagues (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 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, et al., 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 et al., 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 and colleagues (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 et al., 2017; Tanner et al., 2013), and it has been suggested that only specific aspects of rumination are related to PTSD (Claycomb et al., 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 and colleagues (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 et al., 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).

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 et al., 2020). Last, we used a single measure of rumination and could not capture different facets of rumination or trait rumination.

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 et al., 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).

References

Arditte Hall, K. A., Davison, E. H., Galovski, T. E., Vasterling, J. J., & Pineles, S. L. (2019). Associations between trauma-related rumination and symptoms of posttraumatic stress and depression in treatment-seeking female veterans. *Journal of Traumatic Stress*, *32*(2), 260–268. https://doi.org/10.1002/jts.22385

Bates, D., Mächler, M., Bolker, B. M., & Walker, S. C. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software, 67*(1), 1–48. https://doi.org/10.18637/jss.v067.i01

Arditte Hall, K. A., Davison, E. H., Galovski, T. E., Vasterling, J. J., & Pineles, S. L. (2019). Associations between trauma-related rumination and symptoms of posttraumatic stress and depression in treatment-seeking female veterans. *Journal of Traumatic Stress*, *32*(2), 260–268. https://doi.org/10.1002/jts.22385

Bates, D., Mächler, M., Bolker, B. M., & Walker, S. C. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software, 67*(1), 1–48. https://doi.org/10.18637/jss.v067.i01

Beierl, E. T., Böllinghaus, I., Clark, D. M., Glucksman, E., & Ehlers, A. (2019). Cognitive paths from trauma to posttraumatic stress disorder: A prospective study of Ehlers and Clark's model in survivors of assaults or road traffic collisions. *Psychological Medicine,* 1–10. https://doi.org/10.1017/S0033291719002253

Birrer, E., & Michael, T. (2011). Rumination in PTSD as well as in traumatized and non-traumatized depressed patients: A cross-sectional clinical study. *Behavioural and Cognitive Psychotherapy, 39*(4), 381–397. https://doi.org/10.1017/S1352465811000087

Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder: Systematic review and meta-analysis. *British Journal of Psychiatry, 190,* 97–104.

https://doi.org/10.1192/bjp.bp.106.021402

Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry*, *162*(2), 214–227. https://doi.org/10.1176/appi.ajp.162.2.214

Brown, L. A., Belli, G. M., Asnaani, A., & Foa, E. B. (2019). A review of the role of negative cognitions about oneself, others, and the world in the treatment of PTSD. *Cognitive Therapy and Research*, *43*(1), 143–173. https://doi.org/10.1007/s10608-018-9938-1

Brown, W. J., Hetzel-Riggin, M. D., Mitchell, M. A., & Bruce, S. E. (2018). Rumination mediates the relationship between negative affect and posttraumatic stress disorder symptoms in female interpersonal trauma survivors. *Journal of Interpersonal Violence*. https://doi.org/10.1177/0886260518818434

Claycomb, M. A., Wang, L., Sharp, C., Ractliffe, K. C., & Elhai, J. D. (2015). Assessing relations between PTSD's dysphoria and reexperiencing factors and dimensions of rumination. *PLoS ONE*, *10*(3). https://doi.org/10.1371/journal.pone.0118435

Clohessy, S., & Ehlers, A. (1999). PTSD symptoms, response to intrusive memories and coping in ambulance service workers. *British Journal of Clinical Psychology*, *38*(3), 251–265. https://doi.org/10.1348/014466599162836

Cooper, A. A., Zoellner, L. A., Roy-Byrne, P., Mavissakalian, M. R., & Feeny, N. C. (2017). Do changes in trauma-related beliefs predict PTSD symptom improvement in prolonged exposure and sertraline? *Journal of Consulting and Clinical Psychology*, *85*(9), 873–882. https://doi.org/10.1037/ccp0000220

Cusack, K., Jonas, D. E., Forneris, C. A., Wines, C., Sonis, J., Middleton, J. C., Feltner, C., Brownley, K. A., Olmsted, K. R., Greenblatt, A., Weil, A., & Gaynes, B. N. (2016). Psychological treatments for adults with posttraumatic stress disorder: A systematic review and meta-analysis. *Clinical Psychology Review, 43*, 128–141.

https://doi.org/10.1016/j.cpr.2015.10.003

Dondanville, K. A., Blankenship, A. E., Molino, A., Resick, P. A., Wachen, J. S., Mintz, J., Yarvis, J. S., Litz, B. T., Borah, E. V., Roache, J. D., Young-McCaughan, S., Hembree, E. A., & Peterson, A. L. (2016). Qualitative examination of cognitive change during PTSD treatment for active duty service members. *Behaviour Research and Therapy*, *79*, 1–6. https://doi.org/10.1016/j.brat.2016.01.003

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*(4), 319–345. https://doi.org/10.1016/S0005-7967(99)00123-0

Ehlers, A., Grey, N., Wild, J., Stott, R., Liness, S., Deale, A., Handley, R., Albert, I., Cullen, D., Hackmann, A., Manley, J., McManus, F., Brady, F., Salkovskis, P., & Clark, D. M. (2013). Implementation of cognitive therapy for PTSD in routine clinical care: Effectiveness and moderators of outcome in a consecutive sample. *Behaviour Research and Therapy*, *51*(11), 742–752. https://doi.org/10.1016/j.brat.2013.08.006

Ehlers, A., & Steil, R. (1995). Maintenance of Intrusive Memories in Posttraumatic Stress Disorder: A Cognitive Approach. *Behavioural and Cognitive Psychotherapy*, *23*(3), 217–249. https://doi.org/10.1017/S135246580001585X

Ehring, T., & Ehlers, A. (2014). Does rumination mediate the relationship between emotion regulation ability and posttraumatic stress disorder? *European Journal of Psychotraumatology*, *5*(1), 23547. https://doi.org/10.3402/ejpt.v5.23547

Ehring, T., Ehlers, A., & Glucksman, E. (2006). Contribution of cognitive factors to the prediction of post-traumatic stress disorder, phobia and depression after motor vehicle accidents. *Behaviour Research and Therapy*, *44*(12), 1699–1716. https://doi.org/10.1016/j.brat.2005.11.013

Ehring, T., Ehlers, A., & Glucksman, E. (2008). Do cognitive models help in predicting the

severity of posttraumatic Stress disorder, phobia, and depression after motor vehicle accidents? A prospective longitudinal study. *Journal of Consulting and Clinical Psychology*, *76*(2), 219–230. https://doi.org/10.1037/0022-006X.76.2.219

Ehring, T., Frank, S., & Ehlers, A. (2008). The role of rumination and reduced concreteness in the maintenance of posttraumatic stress disorder and depression following trauma. *Cognitive Therapy and Research*, *32*(4), 488–506. https://doi.org/10.1007/s10608-006-9089-7

Ehring, T., Fuchs, N., & Kläsener, I. (2009). The effects of experimentally induced rumination versus distraction on analogue posttraumatic stress symptoms. *Behavior Therapy*, *40*(4), 403–413. https://doi.org/10.1016/j.beth.2008.10.001

Ehring, T., & Watkins, E. R. (2008). Repetitive negative thinking as a transdiagnostic process. *International Journal of Cognitive Therapy*, *1*(3), 192–205. https://doi.org/10.1521/ijct.2008.1.3.192

Ehring, T., Welboren, R., Morina, N., Wicherts, J. M., Freitag, J., & Emmelkamp, P. M. G. (2014). Meta-analysis of psychological treatments for posttraumatic stress disorder in adult survivors of childhood abuse. *Clinical Psychology Review*, *34*(8), 645–657. https://doi.org/10.1016/j.cpr.2014.10.004

Ehring, T., Zetsche, U., Weidacker, K., Wahl, K., Schönfeld, S., & Ehlers, A. (2011). The Perseverative Thinking Questionnaire (PTQ): Validation of a content-independent measure of repetitive negative thinking. *Journal of Behavior Therapy and Experimental Psychiatry*, *42*(2), 225–232. https://doi.org/10.1016/j.jbtep.2010.12.003

Elhai, J. D., Grubaugh, A. L., Kashdan, T. B., & Frueh, B. C. (2008). Empirical examination of a proposed refinement to DSM-IV posttraumatic stress disorder symptom criteria using the national comorbidity survey replication data. *Journal of Clinical Psychiatry*, *69*(4), 597–602. https://doi.org/10.4088/JCP.v69n0411

Elwood, L. S., Hahn, K. S., Olatunji, B. O., & Williams, N. L. (2009). Cognitive vulnerabilities to the development of PTSD: A review of four vulnerabilities and the proposal of an integrative vulnerability model. *Clinical Psychology Review*, *29*(1), 87–100. https://doi.org/10.1016/j.cpr.2008.10.002

Erwin, M. C., Mitchell, M. A., Contractor, A. A., Dranger, P., Charak, R., & Elhai, J. D. (2018). The relationship between distress tolerance regulation, counterfactual rumination, and PTSD symptom clusters. *Comprehensive Psychiatry*, *82*, 133–140. https://doi.org/10.1016/j.comppsych.2018.01.012

Falkenström, F., Finkel, S., Sandell, R., Rubel, J. A., & Holmqvist, R. (2017). Dynamic models of individual change in psychotherapy process research. *Journal of Consulting and Clinical Psychology*, *85*(6), 537–549. https://doi.org/10.1037/ccp0000203

Foa, E. B., & Cahill, S. P. (2001). Psychological theories: Emotional processing. In N. J. Smelser & P. B. Baltes (Eds.), *International Encyclopedia of Social & Behavioral Sciences* (pp. 12363–12369). Elsevier.

García, F. E., Duque, A., & Cova, F. (2017). The four faces of rumination to stressful events: A psychometric analysis. *Psychological Trauma: Theory, Research, Practice, and Policy*, *9*(6), 758–765. https://doi.org/10.1037/tra0000289

García, F. E., Vázquez, C., & Inostroza, C. (2019). Predictors of post-traumatic stress symptoms following occupational accidents: A longitudinal study. *Anxiety, Stress and Coping*, *32*(2), 168–178. https://doi.org/10.1080/10615806.2019.1566533

Gómez de La Cuesta, G., Schweizer, S., Diehle, J., Young, J., & Meiser-Stedman, R. (2019). The relationship between maladaptive appraisals and posttraumatic stress disorder: A meta-analysis. *European Journal of Psychotraumatology*, *10*(1). https://doi.org/10.1080/20008198.2019.1620084

Iverson, K. M., King, M. W., Cunningham, K. C., & Resick, P. A. (2015). Rape survivors'

trauma-related beliefs before and after cognitive processing therapy: Associations with PTSD and depression symptoms. *Behaviour Research and Therapy, 66,* 49–55. https://doi.org/10.1016/j.brat.2015.01.002

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology, 3*(1), 1–27. https://doi.org/10.1146/annurev.clinpsy.3.022806.091432

Kleim, B., Ehlers, A., & Glucksman, E. (2012). Investigating cognitive pathways to psychopathology: Predicting depression and posttraumatic stress disorder from early responses after assault. *Psychological Trauma: Theory, Research, Practice, and Policy, 4*(5), 527–537. https://doi.org/10.1037/a0027006

Kleim, B., Graham, B., Bryant, R. A., & Ehlers, A. (2013). Capturing intrusive re-experiencing in trauma survivors' daily lives using ecological momentary assessment. *Journal of Abnormal Psychology*, *122*(4), 998–1009. https://doi.org/10.1037/a0034957

Kleim, B., Grey, N., Wild, J., Nussbeck, F. W., Stott, R., Hackmann, A., Clark, D. M., & Ehlers, A. (2013). Cognitive change predicts symptom reduction with cognitive therapy for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *81*(3), 383–393. https://doi.org/10.1037/a0031290

Krüger-Gottschalk, A., Alpers, G., Pittig, A., Kuck, S., Dyer, A., & Ehring, T. (in preparation): Effectiveness of phase-based trauma-focused cognitive behavioural therapy in a routine clinical setting [Unpublished manuscript]. Institute of Psychology, University of Muenster.

Krüger-Gottschalk, A., Knaevelsrud, C., Rau, H., Dyer, A., Schäfer, I., Schellong, J., & Ehring, T. (2017). The German version of the Posttraumatic Stress Disorder Checklist for DSM-5 (PCL-5): Psychometric properties and diagnostic utility. *BMC Psychiatry, 17*(1). https://doi.org/10.1186/s12888-017-1541-6

Kubota, R., & Nixon, R. D. V. (2017). An analogue investigation into the effect of trauma-related rumination on trauma intrusions and the moderating role of trait rumination and depression. *Journal of Experimental Psychopathology*, *8*(4), 413–442. https://doi.org/10.5127/jep.058516

Kumpula, M. J., Pentel, K. Z., Foa, E. B., LeBlanc, N. J., Bui, E., McSweeney, L. B., Knowles, K., Bosley, H., Simon, N. M., & Rauch, S. A. M. (2017). Temporal sequencing of change in posttraumatic cognitions and PTSD symptom reduction during prolonged exposure therapy. *Behavior Therapy*, *48*(2), 156–165. https://doi.org/10.1016/j.beth.2016.02.008

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). lmerTest package: Tests in linear mixed effects models. *Journal of Statistical Software*, *82*(13), 1–26. https://doi.org/10.18637/jss.v082.i13

Laposa, J. M., & Rector, N. A. (2012). The prediction of intrusions following an analogue traumatic event: Peritraumatic cognitive processes and anxiety-focused rumination versus rumination in response to intrusions. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(3), 877–883. https://doi.org/10.1016/j.jbtep.2011.12.007

Lewis, C., Roberts, N. P., Andrew, M., Starling, E., & Bisson, J. I. (2020). Psychological therapies for post-traumatic stress disorder in adults: Systematic review and meta-analysis. *European Journal of Psychotraumatology*, *11*(1), 1729633. https://doi.org/10.1080/20008198.2020.1729633

Littleton, H., Buck, K., Rosman, L., & Grills-Taquechel, A. (2012). From survivor to thriver: A pilot study of an online program for rape victims. *Cognitive and Behavioral Practice*, *19*(2), 315–327. https://doi.org/10.1016/j.cbpra.2011.04.002

LoSavio, S. T., Dillon, K. H., & Resick, P. A. (2017). Cognitive factors in the development, maintenance, and treatment of post-traumatic stress disorder. *Current Opinion in*

Psychology, *14*, 18–22. https://doi.org/10.1016/j.copsyc.2016.09.006

Lyons, R., Haller, M., Curry, I., & Norman, S. B. (2019). The relationship between negative trauma-related cognitions and psychosocial functioning in veterans with posttraumatic stress disorder and alcohol use disorder. *Substance Abuse*, *41*(1), 132–138. https://doi.org/10.1080/08897077.2019.1635957

Mayou, R. A., Bryant, B., & Ehlers, A. (2001). Prediction of psychological outcomes one year after a motor vehicle accident. *American Journal of Psychiatry*, *158*(8), 1231–1238. https://doi.org/10.1176/appi.ajp.158.8.1231

Mayou, R. A., Ehlers, A., & Bryant, B. (2002). Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study. *Behaviour Research and Therapy*, *40*(6), 665–675. https://doi.org/10.1016/S0005-7967(01)00069-9

McLean, C. P., Yeh, R., Rosenfield, D., & Foa, E. B. (2015). Changes in negative cognitions mediate PTSD symptom reductions during client-centered therapy and prolonged exposure for adolescents. *Behaviour Research and Therapy*, *68*, 64–69. https://doi.org/10.1016/j.brat.2015.03.008

Michael, T., Halligan, S. L., Clark, D. M., & Ehlers, A. (2007). Rumination in posttraumatic stress disorder. *Depression and Anxiety*, *24*(5), 307–317. https://doi.org/10.1002/da.20228

Mitchell, M. A., Contractor, A. A., Dranger, P., & Shea, M. T. (2016). Unique relations between counterfactual thinking and DSM-5 PTSD symptom clusters. *Psychological Trauma: Theory, Research, Practice, and Policy*, *8*(3), 293–300. https://doi.org/10.1037/tra0000089

Moulds, M. L., Bisby, M. A., Wild, J., & Bryant, R. A. (2020). Rumination in posttraumatic stress disorder: A systematic review. *Clinical Psychology Review*, *82*(September), 101910. https://doi.org/10.1016/j.cpr.2020.101910

Murray, J., Ehlers, A., & Mayou, R. A. (2002). Dissociation and post-traumatic stress disorder: Two prospective studies of road traffic accident survivors. *British Journal of Psychiatry*, *180*(APR.), 363–368. https://doi.org/10.1192/bjp.180.4.363

O'Donnell, M. L., Elliott, P., Wolfgang, B. J., & Creamer, M. (2007). Posttraumatic appraisals in the development and persistence of posttraumatic stress symptoms. *Journal of Traumatic Stress*, *20*(2), 173–182. https://doi.org/10.1002/jts.20198

R Core Team (2021). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. https://www.R-project.org/

Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology*, *60*(5), 748–756. https://doi.org/10.1037/0022-006X.60.5.748

Schiepek, G., Gelo, O., Viol, K., Kratzer, L., Orsucci, F., Felice, G., Stöger-Schmidinger, B., Sammet, I., Aichhorn, W., & Schöller, H. (2020). Complex individual pathways or standard tracks? A data-based discussion on the trajectories of change in psychotherapy. *Counselling and Psychotherapy Research*, *20*(4), 689–702. https://doi.org/10.1002/capr.12300

Schnyder, U. (2013). German version of the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) [Unpublished manuscript]. University Hospital Zurich, Switzerland.

Schnyder, U., & Moergeli, H. (2002). German version of clinician-administered PTSD scale. *Journal of Traumatic Stress*, *15*(6), 487–492. https://doi.org/10.1023/A:1020922023090

Schumm, J. A., Dickstein, B. D., Walter, K. H., Owens, G. P., & Chard, K. M. (2015). Changes in posttraumatic cognitions predict changes in posttraumatic stress disorder symptoms during cognitive processing therapy. *Journal of Consulting and Clinical Psychology*, *83*(6), 1161–1166. https://doi.org/10.1037/ccp0000040

Seligowski, A. V., Rogers, A. P., & Orcutt, H. K. (2016). Relations among emotion

regulation and DSM-5 symptom clusters of PTSD. *Personality and Individual Differences*, *92*, 104–108. https://doi.org/10.1016/j.paid.2015.12.032

Shrout, P., & Lane, S. P. (2012). Psychometrics. In M. R. Mehl & T. S. Conner (Eds.), *Handbook of Research Methods for Studying Daily Life* (pp. 302–320). Guilford Press.

Steil, R., & Ehlers, A. (2000). Dysfunctional meaning of posttraumatic intrusions in chronic PTSD. *Behaviour Research and Therapy*, *38*(6), 537–558. https://doi.org/10.1016/S0005-7967(99)00069-8

Stewart, R. E., & Chambless, D. L. (2009). Cognitive–behavioral therapy for adult anxiety disorders in clinical practice: A meta-analysis of effectiveness studies. *Journal of Consulting and Clinical Psychology*. https://doi.org/10.1037/a0016032.supp

Szabo, Y. Z., Warnecke, A. J., Newton, T. L., & Valentine, J. C. (2017). Rumination and posttraumatic stress symptoms in trauma-exposed adults: A systematic review and meta-analysis. *Anxiety, Stress and Coping*, *30*(4), 396–414. https://doi.org/10.1080/10615806.2017.1313835

Tanner, A., Voon, D., Hasking, P., & Martin, G. (2013). Underlying structure of ruminative thinking: Factor analysis of the ruminative thought style questionnaire. *Cognitive Therapy and Research*, *37*(3), 633–646. https://doi.org/10.1007/s10608-012-9492-1

Topper, M., Emmelkamp, P. M. G., Watkins, E. R., & Ehring, T. (2017). Prevention of anxiety disorders and depression by targeting excessive worry and rumination in adolescents and young adults: A randomized controlled trial. *Behaviour Research and Therapy*, *90*, 123–136. https://doi.org/10.1016/J.BRAT.2016.12.015

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*, *27*(3), 247–259. https://doi.org/10.1023/A:1023910315561

Valdez, C. E., & Lilly, M. M. (2017). Posttraumatic rumination: Content, correlates, and

processes. *Journal of Clinical Psychology*, *73*(6), 707–721. https://doi.org/10.1002/jclp.22371

Watkins, E. R. (2018). *Rumination-focused cognitive-behavioral therapy for depression.* Guilford Publications.

Watkins, E. R., Taylor, R. S., Byng, R., Baeyens, C., Read, R., Pearson, K., & Watson, L. (2012). Guided self-help concreteness training as an intervention for major depression in primary care: a Phase II randomized controlled trial. *Psychological Medicine*, *42*(7), 1359–1371. https://doi.org/10.1017/S0033291711002480

Watts, B. V., Schnurr, P. P., Mayo, L., Young-Xu, Y., Weeks, W. B., & Friedman, M. J. (2013). Meta-analysis of the efficacy of treatments for posttraumatic stress disorder. *Journal of Clinical Psychiatry*, *74*(6). https://doi.org/10.4088/JCP.12r08225

Weathers, F., Blake, D., Schnurr, P., Kaloupek, D., Marx, B., & Keane, T. (2013). The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5). Interview available from the National Center for PTSD at www.ptsd.va.gov

Weathers, F. W., Bovin, M. J., Lee, D. J., Sloan, D. M., Schnurr, P. P., Kaloupek, D. G., Keane, T. M., & Marx, B. P. (2018). The clinician-administered PTSD scale for DSM-5 (CAPS-5): Development and initial psychometric evaluation in military veterans. *Psychological Assessment*, *30*(3), 383–395. https://doi.org/10.1037/pas0000486

Weathers, F. W., Keane, T. M., & Davidson, J. R. T. (2001). Clinician-administered PTSD scale: A review of the first ten years of research. *Depression and Anxiety*, *13*(3), 132–156. https://doi.org/10.1002/da.1029

Weathers, F.W., Litz, B.T., Keane, T.M., Palmieri, P.A., Marx, B.P., & Schnurr, P.P. (2013). The PTSD Checklist for DSM-5 (PCL-5) - LEC-5 and extended criterion A. Retrieved from https://www.ptsd.va.gov/professional/assessment/documents/PCL-5_LEC_criterionA.pdf

Wiedemann, M., Stott, R., Nickless, A., Beierl, E. T., Wild, J., Warnock-Parkes, E., Grey, N., Clark, D. M., & Ehlers, A. (2020). Cognitive processes associated with sudden gains in cognitive therapy for posttraumatic stress disorder in routine care. *Journal of Consulting and Clinical Psychology*, *88*(5), 455–469. https://doi.org/10.1037/ccp0000488

Wild, J., El-Salahi, S., Tyson, G., Lorenz, H., Pariante, C. M., Danese, A., Tsiachristas, A., Watkins, E. R., Middleton, B., Blaber, A., & Ehlers, A. (2018). Preventing PTSD, depression and associated health problems in student paramedics: Protocol for PREVENT-PTSD, a randomised controlled trial of supported online cognitive training for resilience versus alternative online training and standard practice. *BMJ Open*, *8*(12), 22292. https://doi.org/10.1136/bmjopen-2018-022292

Wisco, B. E., Sloan, D. M., & Marx, B. P. (2013). Cognitive emotion regulation and written exposure therapy for posttraumatic stress disorder. *Clinical Psychological Science*, *1*(4), 435–442. https://doi.org/10.1177/2167702613486630

Wittchen, H.-U., Zaudig, M., & Fydrich, T. (1997). SKID. Strukturiertes Klinisches Interview für DSM-IV. Achse I und II. Handanweisung. Göttingen: Hogrefe.

Zalta, A. K., Gillihan, S. J., Fisher, A. J., Mintz, J., Mclean, C. P., Yehuda, R., & Foa, E. B. (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology*, *82*(1), 171–175. https://doi.org/10.1037/a0034735

Table 3.1. 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[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 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[1]	29.51 (20.18)
RIQ-R[1]	7.26 (5.12)

[1]Across all time points (*N* = 2372) and all participants (*N* = 88).

[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.

Table 3.2. 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

*Table 3.3***.** Results of Linear Mixed Models

Dependent variable: Symptom severity (PCL-5)				
	β[1]	*SE*	*t*	*p*
Intercept	27.33	2.07	13.21	< .001
Time	-11.37	1.07	-10.61	< .001
Changes in rumination over time (RIQ-R)				
	β	*SE*	*t*	*p*
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)				
	β	*SE*	*t*	*p*
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)				
	β	*SE*	*t*	*p*
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)				
	β	*SE*	*t*	*p*
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.

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

4. Study III: Sleep Problems in Refugees with Posttraumatic Stress Disorder and their Association with Social Functioning and Quality of Life.

This chapter is a pre-peer-review, pre-copyedit version of a manuscript submitted to *Journal of Traumatic Stress.* This article may be used for non-commercial purposes in accordance with Wiley Terms and Conditions for Use of Self-Archived Versions.

Schumm, H., Steil, R., Lechner-Meichsner, F., Morina, N., Weise, C., Mewes, R., Kuck, S., Reuter, J., Giesebrecht, J., Cludius, B., & Ehring, T. (2023). Sleep Problems in Refugees with Posttraumatic Stress Disorder and their Association with Social Functioning and Quality of Life. Manuscript submitted to *Journal of Traumatic Stress*.

Abstract

Objective: Many patients with posttraumatic stress disorder (PTSD) suffer from sleep problems, leading to impairments in social functioning and subjective quality of life. Refugees are a population at high risk for sleep problems, both due to stressful life circumstances and to high PTSD prevalence in this population. However, only limited data on the prevalence of sleep problems in refugees with diagnosed PTSD are available. This study examined the prevalence of subjective sleep problems in a heterogenous group of refugees with PTSD, and their association with other symptoms of PTSD. Additionally, the present study investigated the contribution of sleep problems to social functioning and quality of life over and above the effect of other PTSD symptoms in this population.

Method: Seventy refugees from different countries of origin and currently settled in Germany were included in the study. All participants met criteria for PTSD according to the Clinician-Administered PTSD Scale for DSM-5 and filled out measures of PTSD severity, sleep problems, social impairment, and quality of life.

Results: We found a very high prevalence of sleep problems, with 100% of participants scoring above the clinical cut-off on the Pittsburgh Sleep Quality Index. Subjective sleep problems were significantly associated with both clinician-rated and self-rated PTSD severity after controlling for overlapping items. Contrary to expectations, sleep problems did not predict social impairment or quality of life beyond the effect of other symptoms of PTSD.

Conclusions: These findings highlight the widespread prevalence of sleep problems among refugees. Future studies should assess the causal nature of the relationship between sleep problems and measures of psychosocial functioning in more detail, as well as its dynamic change over time.

Introduction

Many patients with posttraumatic stress disorder (PTSD) suffer from severe sleep problems (Koffel et al., 2016). Sleep problems encompass difficulties initiating and maintaining sleep, early awakenings, nightmares, and – as a consequence – reduced length and quality of sleep. Both nightmares and sleep disturbances in general are listed as part of the diagnostic criteria for PTSD in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), whereas the International Classification of Diseases (ICD-11) guideline only lists nightmares as diagnostic feature (World Health Organization, 2019). Both guidelines conceptualize nightmares as re-experiencing symptom, however, as a result, sleep is often disturbed due to awakenings and difficulties to fall asleep again. Empirical data on subjective sleep problems show that 70-90% of patients with PTSD report sleep problems (Koffel et al., 2016; Maher et al., 2006), sleep on average two to three hours less than needed to feel rested (Taylor et al., 2020), 69% of patients suffer from nightmares (Pruiksma et al., 2016), and only a minority of patients report satisfying sleep quality (Belleville et al., 2009). Objective data corroborate these findings, showing that sleep in patients with PTSD is physiologically disturbed (e.g., shortened slow-wave sleep and changes in rapid eye movement sleep; Baglioni et al., 2016). Moreover, sleep problems are highly correlated with PTSD severity (Cox et al., 2018) and associated with increased suicidality as well as higher levels of co-morbid depressive symptoms (Krakow et al., 2000). Furthermore, sleep problems often persist following PTSD treatment, even when other PTSD symptoms remit (Pruiksma et al., 2016; Zalta et al., 2020). Finally, sleep problems have been argued to hinder successful engagement in PTSD treatment (López et al., 2017).

A population with a high prevalence of sleep problems in general and at high risk for PTSD are refugees. Worldwide, the United Nations High Commissioner for Refugees (UNHCR) estimated that a total of 89.3 million people were forcibly displaced by the end of

2021 (UNHCR, 2021). Of those, around 30% are estimated to suffer from PTSD (Steel et al., 2009). It has been argued that the risk of a complex symptom presentation is significantly higher for refugees compared to non-refugees with PTSD (Jowett et al., 2021). Refugees differ from many other trauma-exposed populations in that they have often experienced prolonged and repeated traumatic events in their country of origin and during their flight, and have additionally witnessed such events in family members. Moreover, compared to non-refugee patients with PTSD, refugees are at an even higher risk of suffering from sleep problems due to the profoundly stressful nature of their life circumstances continuing post-migration (Richter et al., 2020). After arriving in the host country, refugees face challenges such as lack of resources, family separation, acculturation, and asylum policies. Such continuing stressors have been subsumed under the concept of post-migration living difficulties (PMLD), and have been shown to negatively impact psychosocial functioning of refugees (Li et al., 2016). Moreover, refugees frequently live in highly unstable conditions, often in housing facilities with several hundreds of people, sharing a room with strangers. Not surprisingly, studies report an extremely high prevalence of sleep problems among refugees. For example, Sandahl et al. (2017) conducted a study in a sample of over 700 refugees with PTSD and found that almost all participants (99.1%) had trouble sleeping. In a study by Lies et al. (2019), 75.5% of refugees had moderate to severe sleep difficulties. Similar results are reported by Al-Smadi et al. (2019) and Lies et al. (2021), and a recent review established prevalences of sleep problems in refugees between 39-99% (Richter et al., 2020). Yet, only two studies (Sandahl et al., 2017, 2021) have investigated the prevalence of sleep problems in refugees with diagnosed PTSD. However, in the study by Sandahl et al. (2017) sleep problems were measured with a single item with unknown psychometric properties. Also, a single item can hardly capture the multi-faceted nature of sleep problems. Thus, the first aim of this study was to investigate the prevalence of sleep problems among refugees with PTSD in Germany, using a multidimensional, validated questionnaire.

Second, we aimed to assess the relationship between sleep problems and PTSD severity in this sample, while controlling for overlapping items in order to avoid artificial inflation of the associations. Previous studies have often neglected to correct for this (e.g., Lies et al., 2019).

Another aim of this study was to investigate the contribution of sleep problems to social functioning and quality of life, over and above the effect of other symptoms of PTSD. This is based on previous results in non-refugee patients with PTSD showing that sleep problems have detrimental effects on social functioning and quality of life (Krakow et al., 2002). Poor sleep quality is associated with disturbed social and occupational functioning, and exponentially increases distress and daily dysfunction for these patients (Belleville et al., 2009; Giosan et al., 2015). Moreover, sleep difficulties have been shown to mediate effects of PTSD severity on quality of life in samples of veterans (McCarthy et al., 2019). Similarly, studies have demonstrated that sleep problems are associated with worse psychosocial functioning in refugees in general (Lies et al., 2019) as well as with PTSD (Sandahl et al., 2021). This is a highly relevant finding in this population, considering the need for refugees to adapt to a new environment and culture. Refugees are often confronted with numerous post-migration living difficulties, and adequate social functioning is immensely important to cope with these challenges. Impaired sleep – in addition to other symptoms of posttraumatic stress – might further deplete the emotional and cognitive resources needed to learn a new language, adapt to a foreign culture, and cope in a new workplace. Additionally, good physical and mental wellbeing, reflected in perceived quality of life, is essential for successful integration in the host country. Sleep problems might affect quality of life negatively. Even though the two previous studies give important insights into the association between psychosocial functioning and sleep problems in refugees, there are some methodological shortcomings. In the study by Lies et al. (2019), PTSD diagnosis was not employed as an inclusion criterion, and a single item instead of a multidimensional instrument was used to assess sleep problems. To our knowledge, the

study by Sandahl et al. (2021) is therefore the only study assessing sleep problems in refugees with PTSD with a multi-dimensional and validated instrument. This study investigated the role of sleep problems as predictors for treatment response of psychosocial functioning following an intensive treatment program. It therefore remains unclear whether sleep problems are also predictive of psychosocial impairment at baseline. Additionally, Sandahl et al. (2021) did not control for refugee-specific factors such as residence status or time since arrival in the host country, although previous research has identified these variables as risk factors for refugee mental health (Steel et al., 2006). Lastly, to our knowledge, the effect of sleep problems on quality of life in refugees with PTSD has not been studied at all to date. Thus, the possible detrimental effect of sleep problems in addition to other PTSD symptoms on both psychosocial functioning and quality of life among this population needs to be further explored.

The present study included a treatment-seeking sample of refugees with PTSD, and assessed PTSD symptom severity, sleep problems, social impairment, and quality of life. PTSD diagnosis was established via a gold-standard structured interview and sleep problems were assessed using a validated multidimensional instrument. First, we explored the prevalence of self-reported sleep problems. Second, we expected a positive relationship between subjective sleep problems and other self-reported (H1) as well as clinician-rated PTSD symptoms (H2). Third, we expected self-reported sleep problems to positively predict social impairment over and above the effect of other self-reported PTSD symptoms (H3). Fourth, we expected self-reported sleep problems to predict lower quality of life over and above the effect of other self-reported PTSD symptoms (H4).

Method

Participants and Procedure

Data were drawn from a large multi-center randomized controlled trial comparing imagery rescripting with treatment as usual in refugees with PTSD. A full description of the

trial and the intervention can be found in the study protocol (Steil et al., 2021). The ethics committee of the German Psychological Association has approved the study (SteilRegina2019-10- 18-VA, SteilRegina2020-02-26AM), and all participants provided written informed consent. Patients were included if they had a primary diagnosis of PTSD according to DSM-5, came to Germany as a refugee, were between 18 and 65 years old, were able to communicate with a therapist with or without the help of an interpreter, and were motivated to undergo trauma-focused treatment. Exclusion criteria were a life-time diagnosis of psychosis, bipolar disorder or substance dependence, acute suicide risk or risk of harm to others, or the start of new psychopharmacological medication within the previous month. Baseline data from 70 participants (65.71% male, 31.43 % female, 2.86% other) included from February 2020 until July 2022 were used for the present study. Participants ranged in age from 18 to 62 years (M = 31.12 years, SD = 11.25). At baseline, diagnostic interviews were conducted to assess inclusion and exclusion criteria as well as PTSD symptom severity. Additionally, patients filled out a set of questionnaires. In the following, interviews and questionnaires relevant for the present research questions are described in detail.

Measures

Translation of measures

The clinical interview was available in English and German. For participants not fluent in either language, a trained interpreter translated the questions and answers during the interview. The questionnaires were available in English, German, Arabic, and Farsi in previously validated versions. In cases where the questionnaire was not available in the language of the participant (e.g., Pashto) or the participant's literacy was not sufficient to complete the instrument, an interpreter read the questions out loud and noted the answers.

PTSD Symptom Severity

The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5; Weathers et al., 2013; German version by Müller-Engelmann et al., 2020) is a structured clinical interview used to assess diagnosis and severity of posttraumatic stress symptoms in the past month. Symptoms are rated on a 5-point Likert scale ranging from 0 (absent) to 4 (extreme). A total severity score is calculated by summing the severity scores for all 20 symptoms. 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 samples (Müller-Engelmann et al., 2020).

The International Trauma Questionnaire (ITQ; Cloitre et al., 2018) is an 18-item self-report measure assessing PTSD symptom severity and disturbances in self-organization according to the ICD-11 (World Health Organization, 2019), and has good psychometric properties (Cloitre et al., 2018; German: Christen et al., 2021; Arabic: Vallières et al., 2018; Farsi: Andisha et al., 2023). Distress caused by each symptom in the past month is rated on a five-point-scale ranging from 0 (not at all) to 4 (extremely). We first calculated a total score according to the scoring guidelines that includes the six items assessing PTSD core symptoms. The ITQ exhibited adequate internal consistency for the six PTSD items in the present sample, with Cronbach's $\alpha = .73$.

For both measures of PTSD severity, we then calculated modified total scores that excluded items assessing nightmares or sleep problems. This was done in order to avoid overlap with the severity of sleep problems and artificially inflate associations. Regarding the ITQ, the item P1 ("upsetting dreams") was excluded, for the CAPS, the items B2 ("nightmares") and E6 ("sleep disturbances") were excluded. For all analyses, the modified total scores were used.

Sleep Problems

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989; German: Hinz et al., 2017; Arabic: Suleiman et al., 2010; Farsi: Farrahi Moghaddam et al., 2012) is a 19-item self-

report measure of sleep problems in the past month with good psychometric properties. It contains seven components (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction). Each component is based on a 0-3 severity scale indicating the frequency of each problem yielding a total score ranging from 0-21. A total score of 5 or higher indicates clinically significant sleep problems. The PSQI showed good internal consistency in the current sample (Cronbach's α = .76).

Social Impairment

The General Health Questionnaire (GHQ-28; Goldberg et al., 1997; German: Klaiberg et al., 2004; Arabic: El-Rufaie & Daradkeh, 1996; Farsi: Malakouti et al., 2007) is a screening instrument for general psychological health, containing four subscales (somatic symptoms, anxiety, depression, social impairment). For the present analyses, only the subscale social impairment consisting of seven items was used. Items are rated on a 4-point scale ranging from 0 (not at all) to 3 (much more than usual). In the present sample, the subscale social impairment showed good internal consistency in the current sample (Cronbach's α = .79)

Health-Related Quality of Life

The European Quality of Life 5 Dimensions questionnaire (EQ-5D-5L; EuroQol Group, 2019) is a self-report measure assessing health related quality of life on five dimensions (mobility, self-care, usual activities, pain/discomfort, and anxiety/depression). The EQ-5D has been used across a wide range of settings and shown excellent psychometric properties (for an overview, see Feng et al., 2021). As country-specific utiliy scores, which are usually recommended for analyzing the EQ-5D, are not justified here due to the heterogenity of the sample in terms of countries of origin, a total sum score of the severity levels on each dimension (level sum score) was calculated. The EQ-5D showed rather low internal consistency in the current sample (Cronbach's α = .65).

Data analysis

Analyses were conducted in R (Version 4.2.0; R Core Team, 2022). Descriptive statistics were used to examine sleep problem prevalences. For the first and second hypothesis, Pearson correlation analyses between sleep problems and self-reported as well as clinician-rated PTSD severity, respectively, were conducted. As described above, modified total scores without items relating to nightmares or sleep problems were used. For hypotheses three and four, two separate hierarchical multiple regression analyses were conducted with social impairment (GHQ-28 subscale) and quality of life (EQ-5D) as outcome variables. In each hierarchical multiple regression analysis, the same six predictors were entered in three steps. In the first step, the demographic variables age, residence status (secure vs. insecure), time since arrival in Germany, as well as housing situation (apartment alone or with friends/family vs. large housing facility) were entered. The first three factors had been identified as common risk factors for refugee mental health in previous research (Steel et al., 2006). Additionally, it appeared important to control for the current housing situation due to the likely effect on sleep problems. In the second step, self-reported PTSD severity (ITQ modified total score) was entered. Self-reported (instead of clinician-rated) PTSD severity was used as a predictor here to ensure that differences between predictors would not arise due to different measurement methods. Finally, sleep problems (PSQI total score) were entered in the third step. This statistical approach allows to test the effects of certain predictors while controlling for the influence of other predictors.

Results

Descriptive Statistics

Demographic and clinical characteristics of the sample are shown in Table 4.1, as well as descriptive statistics of the investigated variables. Roughly two thirds of the sample were male. Participants came from a wide array of countries, with the largest group coming from

Afghanistan and the second-largest from Syria. More than half were living in large community housing facilities.

Prevalence of Sleep Problems

The global PSQI score in the current sample ranged from six to 21, with 100% of participants scoring above the clinical cut-off of five points (see Table 4.1). The large majority of participants rated their sleep quality as fairly bad or very bad and reported that they sleep (much) less than the recommended 8 hours per night. Most participants stated that it was a (very big) problem to have enough energy throughout the day to fulfill everyday tasks.

Relationship Between Sleep Problems and PTSD Severity

In line with our hypotheses, we found a positive, moderate correlation between self-reported sleep quality (PSQI) and other self-reported PTSD symptoms (ITQ, $r = 0.30$, $p = .016$), and a moderate correlation between self-reported sleep quality and other clinician-rated PTSD symptoms (CAPS, $r = 0.47$, $p < .001$).

Role of Sleep Problems in Social Impairment

Results of the hierarchical multiple regressions predicting social impairment are presented in Table 4.2. In the first step, only residence status was a significant predictor. When PTSD severity was entered into the model in the second step, it significantly predicted social impairment and improved the model ($F = 18.13$, $p < .001$), accounting for an additional 21% of the variance. Contrary to our hypotheses, the addition of sleep problems into the model in the third step did not significantly improve the model ($F = 1.39$, $p = .241$), and sleep problems did not predict social impairment.

Role of Sleep Problems in Quality of Life

Results of the hierarchical multiple regressions predicting quality of life are presented in Table 4.2. Demographic variables were not significantly predictive in the first step. When PTSD severity was entered into the model in the second step, it significantly improved the model (F

= 17.80, $p < .001$) and predicted quality of life, accounting for additional 22% of the variance. Again contrary to our hypotheses, the addition of sleep problems as predictor in the third step did not significantly improve the model ($F = 1.02$, $p = .323$), and sleep problems did not predict quality of life.

Discussion

The purpose of the present study was to gain a better understanding of the prevalence of sleep problems among refugees with PTSD in Germany, using a multidimensional, validated questionnaire. Furthermore, we investigated the relationship between sleep problems and other PTSD symptoms, and the contribution of sleep problems to social functioning and quality of life beyond the effect of other PTSD symptoms.

Regarding our first study aim, we found a very high prevalence of severe sleep problems and the whole sample exceeded the cut-off for clinically relevant sleep problems. Patients generally rated their sleep quality to be very poor, and reported a lack of energy for daily tasks. Our findings regarding subjective overall sleep quality are similar to those by Sandahl et al. (2017, 2021), who found nearly identical results in refugees with PTSD in Denmark. The present results extend previous findings to refugees in Germany, specifically to refugees with PTSD, using a validated sleep inventory. Surprisingly, we found sleep problems in the same range as Sandahl et al. (2021) although – different to their study – we did not employ sleep problems as an inclusion criterion. The fact that sleep problems were not part of our study's inclusion criteria also strengthens the generalizability of the significance of sleep problems to treatment-seeking refugees with PTSD. Other studies that have described sleep problems in refugees have consistently reported lower prevalence rates with only about half of participants scoring above clinical cut-off points for insomnia (e.g., Al-Smadi et al., 2019; Lies et al., 2021). However, these studies did not specifically assess refugees with PTSD. As outlined above, sleep problems and frequent nightmares are widespread in PTSD, and higher compared to non-PTSD

samples. The fact that we included only refugees with PTSD in this study might explain why we found a higher prevalence of sleep problems.

In general, sleep problems among refugees may well be brought on by their highly stressful lives in unstable circumstances, as daily stress has been shown to worsen sleep quality (Petersen et al., 2013). Additionally, with often sleeping in shared rooms in crowded housing facilities, the external sleeping circumstances are also far from ideal. In exploratory analyses, we tested whether participants differed in sleep problems depending on their housing situation. However, differences between the groups were non-significant. This may be due to the fact that we only assessed the housing situation in general, but not the current room/sleeping situation. Within one type of housing, there may be considerable differences that affect the sleeping environment. Future studies should record the current sleeping situation in more detail.

Regarding our second study aim, we found significant moderate relationships between subjective sleep problems and other self-reported as well as clinician-rated PTSD symptoms. In line with our hypotheses, poorer sleep was associated with greater PTSD severity across both assessment types. These results regarding self-rated PTSD severity are consistent with findings by Cox et al. (2018) in non-refugee patients with PTSD. When looking at trauma-affected refugees without PTSD diagnosis, our findings compare to those of Lies et al. (2019). Considering refugees with diagnosed PTSD, our results are in line with those of Sandahl et al. (2021). Moreover, we extend previous findings by showing a similar pattern for clinician-rated PTSD severity, whereby PTSD severity was assessed with the gold-standard interview. It should be noted that the associations we found apply to the relationship between sleep problems and other symptoms of PTSD, i.e. both measures of PTSD were adjusted to no longer contain items measuring sleep problems in order to avoid artificial inflation of the associations. The present results therefore allow for a closer estimation of the association between sleep problems and other symptoms of PTSD in refugees. Most likely, the associations found are characterized

by a reciprocal relationship: a high burden of PTSD symptoms during the day, such as intrusive memories, feelings of anxiety and generally hyperarousal, might lead to impaired sleep at night. In turn, un-restful sleep at night has been shown to exacerbate daytime PTSD symptoms (Short et al., 2017).

Regarding our third and forth study aim, results of the present study did not provide support for the hypotheses that sleep problems predict social impairment or quality of life beyond the effect of other posttraumatic stress symptoms in refugees with PTSD. To our knowledge, no study to date has investigated the role of sleep problems in the prediction of quality of life in refugees in general or with PTSD. When predicting social impairment, only one study has explored the role of sleep problems beyond the effect of PTSD in refugees with PTSD. Sandahl et al. (2021) found both baseline sleep problems and improvements in sleep quality to be predictive of psychosocial functioning following an extensive 12-month treatment for refugees with PTSD. In the present study, we aimed to extend these findings by investigating the role of sleep problems as predictor for psychosocial functioning and well-being at one time point, rather than as predictor for treatment response, and to include refugee-specific factors as additional control variables. Several explanations could account for the lack of significant results: First, methodological aspects need to be considered. There was indication of restricted variance and ceiling effects in subjective sleep problems, with generally very high sleep problems and 100% of the sample scoring above the clinical cut-off. Absent, low, or moderate sleeping problems were not present in our sample. Therefore, due to its too little variance, the predictor variable might not have added significant value in the prediction of social impairment or quality of life, leading to non-significant findings. Second, as was tested for the second study aim, self-reported PTSD severity and sleep problems – both predictors in the regression models for the psychosocial outcomes – were moderately correlated with each other. Such (albeit only small) multicollinearity can reduce the power of a statistical model to identify independent

variables that are statistically significant, leading to non-significant findings (Pettit & Belsley, 1992). Third, the fact that PTSD severity accounted for the largest proportion of variance in both social impairment and quality of life, beyond which sleep problems did not reach significance, confirms the enormous impact of PTSD severity on psychosocial measures. The impact of PTSD on quality of life and social functioning is consistently reported in the literature, both for refugee and non-refugee samples (Teodorescu et al., 2012; Zatzick et al., 1997). Next to this large effect, the additional effect of sleep problems might be negligible. Additionally, our sample did not seek treatment for sleep problems per se, but for PTSD in general. Patients often reported to be highly burdened by "bad memories". Therefore, for this sample, sleep problems might have been less central to subjective social functioning and quality of life. Fourth, when comparing our results to those of Sandahl et al. (2021) who investigated the predictive effect of sleep problems on social functioning, it should be noted that they employed a different measure of social functioning. Their measure also covers some more general aspects of functioning (e.g., taking care of oneself). Also, Sandahl et al. (2021) included a sample which constituted of refugees specifically seeking treatment for PTSD and sleep problems. In their sample, sleep problems may well be much more central in the perceived mental burden than in our sample, and therefore add significantly to subjective social functioning. Both aspects may explain differences between the findings of the two studies, and future studies should ensure comparability across samples and measures.

Interestingly, we found residence status to be the only significant demographic predictor for social impairment, and none of the demographic factors were significant predictors for quality of life. We included these demographic factors since previous research had identified them as relevant in the prediction of refugee mental health, albeit with mixed findings (Koch et al., 2019; Sandahl et al., 2021; Steel et al., 2006). Visa status and time since arrival in the host country are often included in refugee research as they are thought to serve as proxies for current

stability of life circumstances or degree of cultural integration. However, these variables might insufficiently capture the degree of subjective living security – which is probably more central for constructs of psychosocial functioning than objective measures. Additionally, time since arrival in the host country might be ill-suited to capture the degree of cultural integration, and current employment or knowledge of the host country language could serve as better proxies. We tested these in additional exploratory analyses, but both factors were also non-significant. Possibly, some demographic factors are more or less important for subgroups of refugees differing in their country of origin. These subgroups often diverge in their length and difficulty of the flight, access to psychosocial care, and obtainment of visa. As our sample is heterogenous in terms of origin, general statements about the role of certain demographic predictors are difficult here. Future studies should explore the importance of demographic for different subgroups, and to include measures of perceived stability and integration in the host country.

Several limitations of the present study should be noted. First, we almost solely relied on self-report measures, and there was a lack of objective measures of sleep quality. This warrants caution with the study findings, as objective and subjective measures of sleep sometimes show discrepant results in PTSD samples (Werner et al., 2016). However, other authors have shown that patients with PTSD do not globally overestimate their sleep problems (Ghadami et al., 2014). Still, there is a need for replication with objective sleep measures. Second, whereas all translated questionnaires employed here have previously been sucessfully used, no translated questionnaires were available for Pashto-speakers (n = 11). Also, when literacy was not sufficient, some participants required the support of an interpreter to complete the measures. The validity of such interpreter-assissted questionnaires and clinical interviews needs to be further established.

To conclude, the present study has extended previous findings regarding sleep problems and their relationship to PTSD severity to refugees in Germany. Additionally, we have

replicated earlier findings investigating sleep problems in refugees with PTSD, and extended these findings for refugees in Germany seeking treatment for PTSD. With the present findings we hope to stipulate further research into the relationship between sleep problems and quality of life as well as social impairment in this very specific sample. Findings from non-refugee samples have shown that sleep problems lead to and impairments in occupational functioninig, and thereby to high societal economic costs (Giosan et al., 2015). It is therefore of interest to further investigate the role of sleep problems in the ongoing cultural and occupational integration of refugees, even though the present study did not find a significant contribution of sleep problems to a specific measure of social functioning. Keeping in mind that 80% of our sample reported insufficient energy for daily tasks due to sleep problems, the already challenging post-migration living difficulties that refugees are encountering may seem insurmountable to individuals. Lastly, in our findings PTSD severity emerged as strongest predictor for quality of life and social functioning. PTSD treatment is therefore advised to alleviate these impairments in daily functioning. However, sleep problems have been argued to be a risk factor for a poor response to PTSD treatment in non-refugee samples (López et al., 2017; Taylor et al., 2020). Poor sleep quality and resulting mood and concentration difficulties possibly pose a hindrance in engaging in trauma-focused psychotherapy. As a result, the already scarce treatment options for refugees might not be used efficiently, thereby adding to the risk of chronification of the disorder and ensuing high societal costs due to disability. Future studies should assess whether sleep problems sufficiently ameliorate alongside PTSD symptoms in trauma-focused treatment for refugees, or whether additional interventions are needed.

References

Al-Smadi, A. M., Tawalbeh, L. I., Gammoh, O. S., Ashour, A., Tayfur, M., & Attarian, H. (2019). The prevalence and the predictors of insomnia among refugees. *Journal of Health Psychology, 24*(8), 1125–1133. https://doi.org/10.1177/1359105316687631

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. https://doi.org/https://doi.org/10.1176/appi.books.9780890425596

Andisha, P., Shahab, M. J., & Lueger-Schuster, B. (2023). Translation and validation of the Dari International Trauma Questionnaire (ITQ) in Afghan asylum seekers and refugees. *European Journal of Psychotraumatology*, *14*(1), 2158428. https://doi.org/10.1080/20008066.2022.2158428

Baglioni, C., Nanovska, S., Regen, W., Spiegelhalder, K., Feige, B., Nissen, C., Reynolds, C. F., & Riemann, D. (2016). Sleep and mental disorders: A meta-analysis of polysomnographic research. *Psychological Bulletin*, *142*(9), 969–990. https://doi.org/10.1037/bul0000053

Belleville, G., Guay, S., & Marchand, A. (2009). Impact of sleep disturbances on PTSD symptoms and perceived health. *Journal of Nervous and Mental Disease*, *197*(2), 126–132. https://doi.org/10.1097/NMD.0b013e3181961d8e

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4

Carpenter, J. S., & Andrykowski, M. A. (1998). Psychometric evaluation of the Pittsburgh Sleep Quality Index. *Journal of Psychosomatic Research, 45*(1), 5–13. https://doi.org/10.1016/S0022-3999(97)00298-5

Christen, D., Killikelly, C., Maercker, A., & Augsburger, M. (2021). Item Response Model Validation of the German ICD-11 International Trauma Questionnaire for PTSD and

CPTSD. *Clinical Psychology in Europe*, *3*(4), 1–19. https://doi.org/10.32872/CPE.5501

Cloitre, M., Shevlin, M., Brewin, C. R., Bisson, J. I., Roberts, N. P., Maercker, A., Karatzias, T., & Hyland, P. (2018). The International Trauma Questionnaire: development of a self-report measure of ICD-11 PTSD and complex PTSD. *Acta Psychiatrica Scandinavica*, *138*(6), 536–546. https://doi.org/10.1111/acps.12956

Cox, R. C., Alex McIntyre, W., & Olatunji, B. O. (2018). Interactive effects of insomnia symptoms and trauma exposure on PTSD: Examination of symptom specificity. *Psychological Trauma: Theory, Research, Practice, and Policy*, *10*(5), 508–514. https://doi.org/10.1037/TRA0000336

El-Rufaie, O. E. F., & Daradkeh, T. K. (1996). Validation of the arabic versions of the thirty- and twelve-item general health questionnaires in primary care patients. *British Journal of Psychiatry*, *169*(5), 662–664. https://doi.org/10.1192/bjp.169.5.662

EuroQol Group. (2019). EQ-5D. EQ-5D. Retrieved from https://euroqol.org/

Farrahi Moghaddam, J., Nakhaee, N., Sheibani, V., Garrusi, B., & Amirkafi, A. (2012). Reliability and validity of the Persian version of the Pittsburgh Sleep Quality Index (PSQI-P). *Sleep and Breathing*, *16*(1), 79–82. https://doi.org/10.1007/s11325-010-0478-5

Feng, Y. S., Kohlmann, T., Janssen, M. F., & Buchholz, I. (2021). Psychometric properties of the EQ-5D-5L: a systematic review of the literature. *Quality of Life Research*, *30*(3), 647–673. https://doi.org/10.1007/s11136-020-02688-y

Ghadami, M. R., Khaledi-Paveh, B., Nasouri, M., & Khazaie, H. (2014). PTSD-related paradoxical insomnia: an actigraphic study among veterans with chronic PTSD. *Journal of Injury and Violence Research*, *7*(2), 54. https://doi.org/10.5249/jivr.v7i2.607

Giosan, C., Malta, L. S., Wyka, K., Jayasinghe, N., Evans, S., Difede, J., & Avram, E. (2015). Sleep Disturbance, Disability, and Posttraumatic Stress Disorder in Utility Workers.

Journal of Clinical Psychology, *71*(1), 72–84. https://doi.org/10.1002/jclp.22116

Goldberg, D. P., Gater, R., Sartorius, N., Ustun, T. B., Piccinelli, M., Gureje, O., & Rutter, C. (1997). The validity of two versions of the GHQ in the WHO study of mental illness in general health care. *Psychological Medicine*, *27*(1), 191–197. https://doi.org/10.1017/S0033291796004242

Hinz, A., Glaesmer, H., Brähler, E., Löffler, M., Engel, C., Enzenbach, C., Hegerl, U., & Sander, C. (2017). Sleep quality in the general population: psychometric properties of the Pittsburgh Sleep Quality Index, derived from a German community sample of 9284 people. *Sleep Medicine*, *30*, 57–63. https://doi.org/10.1016/J.SLEEP.2016.03.008

Jowett, S., Argyriou, A., Scherrer, O., Karatzias, T., & Katona, C. (2021). Complex post-traumatic stress disorder in asylum seekers and victims of trafficking: treatment considerations. *BJPsych Open*, *7*(6). https://doi.org/10.1192/bjo.2021.1007

Klaiberg, A., Schumacher, J., & Brähler, E. (2004). General Health Questionnaire 28 (GHQ-28): Teststatistische Überprüfung einer Deutschen Version in einer bevölkerungsrepräsentativen Stichprobe. *Zeitschrift Fur Klinische Psychologie, Psychiatrie Und Psychotherapie*, *52*(1), 31–42.

Koch, T., Liedl, A., & Ehring, T. (2019). Emotion Regulation as a Transdiagnostic Factor in Afghan Refugees. *Psychological Trauma: Theory, Research, Practice, and Policy*. https://doi.org/10.1037/tra0000489

Koffel, E., Khawaja, I. S., & Germain, A. (2016). Sleep disturbances in posttraumatic stress disorder: Updated review and implications for treatment. *Psychiatric Annals*, *46*(3), 173–176. https://doi.org/10.3928/00485713-20160125-01

Krakow, B., Artar, A., Warner, T. D., Melendrez, D., Johnston, L., Hollifield, M., Germain, A., & Koss, M. (2000). Sleep disorder, depression, and suicidality in female sexual assault survivors. *Crisis*, *21*(4), 163–170. https://doi.org/10.1027//0227-5910.21.4.163

Krakow, B., Melendrez, D., Johnston, L., Warner, T. D., Clark, J. O., Pacheco, M., Pedersen, B., Koss, M., Hollifield, M., & Schrader, R. (2002). Sleep-disordered breathing, psychiatric distress, and quality of life impairment in sexual assault survivors. *Journal of Nervous and Mental Disease*, *190*(7), 442–452. https://doi.org/10.1097/00005053-200207000-00004

Li, S. S. Y., Liddell, B. J., & Nickerson, A. (2016). The Relationship Between Post-Migration Stress and Psychological Disorders in Refugees and Asylum Seekers. *Current Psychiatry Reports*, *18*(9), 1–9. https://doi.org/10.1007/S11920-016-0723-0/METRICS

Lies, J., Jobson, L., Mascaro, L., Whyman, T., & Drummond, S. P. A. (2021). Postmigration stress and sleep disturbances mediate the relationship between trauma exposure and posttraumatic stress symptoms among Syrian and Iraqi refugees. *Journal of Clinical Sleep Medicine : JCSM : Official Publication of the American Academy of Sleep Medicine*, *17*(3), 479. https://doi.org/10.5664/JCSM.8972

Lies, J., Mellor, A., Jobson, L., & Drummond, S. P. A. (2019). Prevalence of sleep disturbance and its relationships with mental health and psychosocial issues in refugees and asylum seekers attending psychological services in Australia. *Sleep Health*, *5*(4), 335–343. https://doi.org/10.1016/j.sleh.2019.06.002

López, C. M., Lancaster, C. L., Gros, D. F., & Acierno, R. (2017). Residual Sleep Problems Predict Reduced Response to Prolonged Exposure among Veterans with PTSD. *Journal of Psychopathology and Behavioral Assessment*, *39*(4), 755–763. https://doi.org/10.1007/s10862-017-9618-6

Maher, M. J., Rego, S. A., & Asnis, G. M. (2006). Sleep disturbances in patients with post-traumatic stress disorder: Epidemiology, impact and approaches to management. In *CNS Drugs* (Vol. 20, Issue 7, pp. 567–590). CNS Drugs. https://doi.org/10.2165/00023210-200620070-00003

Malakouti, S. K., Fatollahi, P., Mirabzadeh, A., & Zandi, T. (2007). Reliability, validity and factor structure of the GHQ-28 used among elderly Iranians. *International Psychogeriatrics, 19*(4), 623–634. https://doi.org/10.1017/S1041610206004522

McCarthy, E., DeViva, J. C., Norman, S. B., Southwick, S. M., & Pietrzak, R. H. (2019). Self-Assessed Sleep Quality Partially Mediates the Relationship Between PTSD Symptoms and Functioning and Quality of Life in U.S. Veterans: Results From the National Health and Resilience in Veterans Study. *Psychological Trauma: Theory, Research, Practice, and Policy*. https://doi.org/10.1037/TRA0000436

Müller-Engelmann, M., Schnyder, U., Dittmann, C., Priebe, K., Bohus, M., Thome, J., Fydrich, T., Pfaltz, M. C., & Steil, R. (2020). Psychometric properties and factor structure of the German version of the clinician-administered PTSD scale for DSM-5. *Assessment, 27*(6), 1128–1138. https://doi.org/10.1177/1073191118774840

Petersen, H., Kecklund, G., D'Onofrio, P., Nilsson, J., & Åkerstedt, T. (2013). Stress vulnerability and the effects of moderate daily stress on sleep polysomnography and subjective sleepiness. *Journal of Sleep Research, 22*(1), 50–57. https://doi.org/10.1111/j.1365-2869.2012.01034.x

Pettit, L., & Belsley, D. A. (1992). Conditioning Diagnostics: Collinearity and Weak Data in Regression. *Applied Statistics, 41*(3), 601. https://doi.org/10.2307/2348093

Pruiksma, K. E., Taylor, D. J., Wachen, J. S., Mintz, J., Young-McCaughan, S., Peterson, A. L., Yarvis, J. S., Borah, E. V., Dondanville, K. A., Litz, B. T., Hembree, E. A., & Resick, P. A. (2016). Residual sleep disturbances following PTSD treatment in active duty military personnel. *Psychological Trauma: Theory, Research, Practice, and Policy, 8*(6), 697–701. https://doi.org/10.1037/tra0000150

R Core Team. (2022). *R: A Language and Environment for Statistical Computing*. https://www.r-project.org/

Richter, K., Baumgärtner, L., Niklewski, G., Peter, L., Köck, M., Kellner, S., Hillemacher, T., & Büttner-Teleaga, A. (2020). Sleep disorders in migrants and refugees: a systematic review with implications for personalized medical approach. *EPMA Journal 2020 11:2*, *11*(2), 251–260. https://doi.org/10.1007/S13167-020-00205-2

Sandahl, H., Carlsson, J., Sonne, C., Mortensen, E. L., Jennum, P., & Baandrup, L. (2021). Investigating the link between subjective sleep quality, symptoms of PTSD, and level of functioning in a sample of trauma-affected refugees. *Sleep*, *44*(9). https://doi.org/10.1093/sleep/zsab063

Sandahl, H., Vindbjerg, E., & Carlsson, J. (2017). Treatment of sleep disturbances in refugees suffering from post-traumatic stress disorder. *Transcultural Psychiatry*, *54*(5–6), 806–823. https://doi.org/10.1177/1363461517746314

Short, N. A., Allan, N. P., & Schmidt, N. B. (2017). Sleep disturbance as a predictor of affective functioning and symptom severity among individuals with PTSD: An ecological momentary assessment study. *Behaviour Research and Therapy*, *97*, 146–153. https://doi.org/10.1016/j.brat.2017.07.014

Steel, Z., Chey, T., Silove, D., Marnane, C., Bryant, R. A., & Van Ommeren, M. (2009). Association of torture and other potentially traumatic events with mental health outcomes among populations exposed to mass conflict and displacement: A systematic review and meta-analysis. *JAMA - Journal of the American Medical Association*, *302*(5), 537–549. https://doi.org/10.1001/jama.2009.1132

Steel, Z., Silove, D., Brooks, R., Momartin, S., Alzuhairi, B., & Susljik, I. (2006). Impact of immigration detention and temporary protection on the mental health of refugees. *The British Journal of Psychiatry : The Journal of Mental Science*, *188*(JAN.), 58–64. https://doi.org/10.1192/BJP.BP.104.007864

Suleiman, K. H., Yates, B. C., Berger, A. M., Pozehl, B., & Meza, J. (2010). Translating the pittsburgh sleep quality index into arabic. *Western Journal of Nursing Research*, *32*(2), 250–268. https://doi.org/10.1177/0193945909348230

Taylor, D. J., Pruiksma, K. E., Hale, W., McLean, C. P., Zandberg, L. J., Brown, L., Mintz, J., Stacey Young-McCaughan, Peterson, A. L., Yarvis, J. S., Dondanville, K. A., Litz, B. T., Roache, J., & Foa, E. B. (2020). Sleep problems in active duty military personnel seeking treatment for posttraumatic stress disorder: Presence, change, and impact on outcomes. *Sleep*, *43*(10). https://doi.org/10.1093/sleep/zsaa065

Teodorescu, D. S., Siqveland, J., Heir, T., Hauff, E., Wentzel-Larsen, T., & Lien, L. (2012). Posttraumatic growth, depressive symptoms, posttraumatic stress symptoms, post-migration stressors and quality of life in multi-traumatized psychiatric outpatients with a refugee background in Norway. *Health and Quality of Life Outcomes*, *10*(1), 1–16. https://doi.org/10.1186/1477-7525-10-84

UNHCR - United Nations High Commissioner for Refugees. (2021). *Global Trends Report 2021*. https://www.unhcr.org/62a9d1494/global-trends-report-2021

Vallières, F., Ceannt, R., Daccache, F., Abou Daher, R., Sleiman, J., Gilmore, B., Byrne, S., Shevlin, M., Murphy, J., & Hyland, P. (2018). ICD-11 PTSD and complex PTSD amongst Syrian refugees in Lebanon: the factor structure and the clinical utility of the International Trauma Questionnaire. *Acta Psychiatrica Scandinavica*, *138*(6), 547–557. https://doi.org/10.1111/ACPS.12973

Weathers, F. W., Blake, D. D., Schnurr, P. P., Kaloupek, D. G., Marx, B., & Keane, T. M. (2013). *The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5). Interview available from the National Center for PTSD at www.ptsd.va.gov*.

Werner, K. B., Griffin, M. G., & Galovski, T. E. (2016). Objective and subjective measurement of sleep disturbance in female trauma survivors with posttraumatic stress

disorder. *Psychiatry Research*, *240*, 234–240. https://doi.org/10.1016/j.psychres.2016.04.039

World Health Organization. (2019). *International Statistical Classification of Diseases and Related Health Problems* (11th ed.). https://icd.who.int/

Zalta, A. K., Pinkerton, L. M., Valdespino-Hayden, Z., Smith, D. L., Burgess, H. J., Held, P., Boley, R. A., Karnik, N. S., & Pollack, M. H. (2020). Examining Insomnia During Intensive Treatment for Veterans with Posttraumatic Stress Disorder: Does it Improve and Does it Predict Treatment Outcomes? *Journal of Traumatic Stress*, *33*(4), 521–527. https://doi.org/10.1002/jts.22505

Zatzick, D. F., Marmar, C. R., Weiss, D. S., Browner, W. S., Metzler, T. J., Golding, J. M., Stewart, A., Schlenger, W. E., & Wells, K. B. (1997). Posttraumatic stress disorder and functioning and quality of life outcomes in a nationally representative sample of male Vietnam veterans. *American Journal of Psychiatry*, *154*(12), 1690–1695. https://doi.org/10.1176/ajp.154.12.1690

*Table 4.1***.** Demographic and Clinical Characteristics of the Sample at Baseline

Variable	*n*	*%*	*M*	*SD*
Gender				
Male	46	65.7%		
Female	22	31.4%		
Other	2	2.9%		
Age (years)			31.12	11.25
Country of origin[a]				
Afghanistan	26	38.8%		
Syria	10	14.9%		
Iraq, Nigeria, Sierra Leone	4 each	6% each		
Iran	3	4.5%		
Bosnia, Jordan, Turkey	2 each	3% each		
Cameroon, Eritrea, Gaza, Guinea, Kuwait, Nigeria, Paraguay, Saudi-Arabia, Tanzania	1 each	1.5% each		
Residence status[a]				
secure		65.7%		
insecure		34.3%		
Time since arrival in Germany (years)[b]			4.98	4.90
Formal education (years)[b]			8.88	4.24
<5		15.2%		
5-8		24.2%		
9-12		50%		
<12		10.6%		
Housing situation[a]				
Own apartment		28.4%		
Living with friends/family		11.4%		
Community housing facility		59.7%		
Currently employed[a]		34.3%		
Clinician-rated PTSD severity (CAPS)			40.77	11.32
Self-reported PTSD severity (ITQ)			16.34	5.79
Social impairment (GHQ-28 subscale)			12.43	4.61
Subjective quality of life (EQ-5D)			11.96	4.65

Table 4.1. (continued)

Variable	*n*	%	*M*	*SD*
Sleep problems (PSQI) – global score[c]			14.32	4.02
Subjective sleep quality (PSQI)[c]				
Very good		0%		
Fairly good		14.7%		
Fairly bad		38.2%		
Very bad		47.1%		
Average sleep per night (PSQI)[c]				
≥ 8h		8.7%		
6-8h		17.4%		
4-6h		47.8%		
< 4h		34.8%		
Enough energy for everyday tasks (PSQI)[c]				
No problem at all		11.9%		
Small problem		16.4%		
Somewhat of a problem		28.4%		
Very big problem		43.3%		

Note. CAPS = Clinician-Administered PTSD Scale for DSM-V; ITQ = International Trauma Questionnaire; GHQ-28 = General Health Questionnaire (subscale social impairment); EQ-5D = European Quality of Life Questionnaire; PSQI = Pittsburgh Sleep Quality Index.

[a] $n = 67$

[b] $n = 66$

[c] $n = 68$

Table 4.2. Hierarchical Regression Analyses Predicting Social Impairment and Quality of Life

Variables	R^2(adjusted)	Δ R^2	*B*	*SE* B	β	*p*
	Criterion variable: Social Impairment (GHQ-28)					
Step 1	.09 (.03)	.09				.11
Age			-.06	.13	-.06	.610
Residence status			.26	.13	.26	.048*
Time in Germany			-.19	.13	-.19	.172
Housing situation			-.15	.13	-.15	.251
Step 2	.30 (.25)	.21				<.001**
PTSD severity (ITQ)			.49	.12	.48	<.001**
Step 3	.26 (.18)	-.04				.241
Sleep problems (PSQI)			.15	.12	.16	.241
	Criterion variable: Quality of Life (EQ-5D)					
Step 1	.06 (-.00)	.06				.452
Age			.10	.13	.10	.461
Residence status			.18	.14	.18	.190
Time in Germany			-.18	.14	-.18	.201
Housing situation			-.12	.13	-.12	.383
Step 2	.27 (.21)	.22				<.001**
PTSD severity (ITQ)			.51	.12	.49	<.001**
Step 3	.29 (.21)	.02				.323
Sleep problems (PSQI)			.12	.12	.13	.323

Note. GHQ-28 = General Health Questionnaire (subscale social impairment); ITQ = International Trauma Questionnaire; PSQI = Pittsburgh Sleep Quality Index; EQ-5D = European Quality of Life Questionnaire.

5. General Discussion

The major goal of the present thesis was to further investigate the roles of posttraumatic cognitions, rumination, and sleep, as possible mechanisms of change in trauma-focused treatment. To this end, **Study I** investigated whether posttraumatic cognitions differentially predicted PTSD symptom clusters over the course of trauma-focused psychotherapy. **Study II** tested whether changes in rumination would decline during trauma-focused treatment as well as precede and predict changes in PTSD severity. The focus of **Study III** was on the role of sleep, examining its association with PTSD symptoms at baseline and its predictive value for broader outcomes in a specific sample.

In this chapter, the main findings will be summarized and interpreted in the context of earlier research. Additionally, implications for further research will be discussed.

Summary of Results

Data for **Study I** and **Study II** were drawn from an effectiveness study evaluating a phase-based therapy approach for patients with PTSD in a naturalistic setting. For **Study I**, over the course of therapy, posttraumatic cognitions and posttraumatic stress symptoms were measured every five sessions via questionnaires. To further clarify the role of posttraumatic cognitions as mechanism of change, it was then examined whether changes in posttraumatic cognitions would precede and predict both total PTSD severity and changes in the PTSD clusters. Results show that previous findings on the role of posttraumatic cognitions as mechanism of change for total PTSD score could only partly be replicated, depending on whether time was additionally controlled for or not in the statistical models. Moreover, previous findings were extended by showing that the four symptom clusters were predicted differentially. However, as changes in three out of four symptom clusters were predicted by changes in posttraumatic cognitions, this speaks for a rather broad effect on the different symptom clusters. In **Study II**, the role of rumination as possible mechanism of change was investigated. Patients filled out weekly measures of symptom severity and rumination, and it was examined whether

changes in rumination would precede and predict changes in symptomatology. It was shown that rumination decreased during the treatment. However, results only partially supported rumination's role as working mechanism, again depending on the choice of the statistical model. When time was not additionally controlled for, changes in rumination predicted changes in symptomatology, lending support to its role as mechanism of change.

Taken together, both **Study I** and **II** yielded support for the role of the proposed processes as mechanisms of change in trauma-focused treatment. However, results depended on the statistical analyses employed – both of which have precedents in the literature – and therefore need to be interpreted with caution and replicated.

Study III was based on a sample of refugees with diagnosed PTSD, currently living in Germany. This study aimed to replicate previous results on the association between sleep as proposed mechanism of change, and to extend these results for the especially affected subgroup of refugees with PTSD. Measures of PTSD symptom severity, both clinician-rated and self-reported, along with measures of subjective sleep problems, psychosocial functioning, and quality of life, were given at baseline. Results showed an extremely high prevalence of sleep problems, and significant associations to both measures of posttraumatic stress symptoms. However, sleep problems did not add additional value in the prediction of psychosocial functioning nor quality of life.

Advancing our understanding of posttraumatic cognitions, rumination, and sleep as treatment mechanisms

Posttraumatic cognitions

Coming back to the criteria for a mechanism of change proposed by Kazdin (2007), empirical evidence has provided substantial support for the role of posttraumatic cognitions as mechanism of change in trauma-focused treatment. Importantly, several studies have demonstrated that changes in posttraumatic cognitions precede and predict changes in

symptoms over the course of therapy, thereby fulfilling the critical criterion of establishing a timeline (e.g., Kleim et al., 2013; for an overview, see Alpert et al., 2023). **Study I** has extended results by investigating this effect on a more fine-grained level. One aspect that deserves further attention in research is that currently, processes investigated under the umbrella term of posttraumatic cognitions include a heterogenous variety of dysfunctional thought content such as appraisals, beliefs, or interpretations, of the traumatic event itself but also concerning its consequences. Not all of these types of thought may play an equally important role as mechanism of change. For example, McLean and colleagues (2019) demonstrated that only certain subtypes of posttraumatic cognitions, namely negative cognitions about the world and the self, significantly mediated change in posttraumatic stress symptoms during trauma-focused treatment, whereas the subtype of self-blame cognitions did not. Other authors have proposed that cognitions relating to hope could play an important role as mechanism (Gilman et al., 2012). Future studies should therefore focus on more detailed analyses on the specific types of posttraumatic cognitions. Additionally, the evidence base for this mechanism could be further strengthened by including assessment methods other than self-report questionnaires (e.g., behavioral tasks or observer ratings). Moreover, albeit weekly measurements of the purported mechanism are a very good starting point, such assessments can hardly capture the range, intensity and variability of cognitions that patients experience throughout a week. Further, the association with other symptoms of PTSD on a day-to-day level remains unclear. Higher-frequency measurements such as EMA might be a approach worthwhile of further investigation.

Rumination

As has been depicted in detail above, previous studies have provided considerable evidence for the role of rumination as mechanism of change, by showing, among other things, a strong association with posttraumatic stress symptoms, establishing its predictive value, and corroborating results with findings from experimental studies. However, evidence for an

association between intervention and proposed mechanism, as well as establishing a timeline of change, had so far been lacking. **Study II** yielded preliminary evidence that rumination does decline following a phase-based intervention, and that changes in rumination precede and predict changes in PTSD symptoms over the course of treatment. Yet, since the hypotheses were only partly supported depending on the choice of the statistical approach, future studies should aim to further corroborate rumination's role as mechanism of change. For such studies, a frequent assessment of both rumination and outcome would is again necessary. Although the use of EMA-methods could also prove a to be a fruitful avenue here, it continues to be difficult to measure internal processes that could be mechanisms of change with the required frequency. Further, future studies should also consider to employ measurements that are less confounded with the outcome than was the case in Study II, and are better suited to capture the characteristics of the ruminative thinking *process* such as recursiveness and perceived uncontrollability. The Perseverative Thinking Questionnaire (Ehring et al., 2011) could be such a potential alternative questionnaire. Lastly, for all such future studies, controlled designs are necessary to differentiate treatment effects from processes of natural recovery and to strengthen the evidence for rumination as mechanism of change.

Based on the findings of **Study I** and **II**, it is also recommended for the field to establish a consensus regarding the statistical approach. In both of these studies, results differed depending on the statistical model, with both approaches having predecessors in the literature. Until consensus regarding the best-suited model is reached, applying both statistical approaches – with and without controlling for the effect of time –and reporting both results could allow for comparison of results between studies.

Sleep

Regarding sleep, several criteria proposed by Kazdin (2007) to establish a mechanism of change have so far been met. Studies demonstrated a high relevance of the process in general,

a strong association with the outcome, and that disturbed sleep prior to a traumatic event predicts the development of PTSD. Findings from experimental studies lent further support for its role as treatment mechanism. Moreover, all this has been done against the background of a plausible theoretical rationale. **Study III** has extended previous results by demonstrating a high prevalence of sleep problems and a significant association with PTSD severity for a different, highly relevant, population. Thereby, evidence for the criterion of replication across different samples was accumulated. It now appears critical for the field to move on to establish a timeline of changes between the proposed mechanism, namely sleep, and changes in the outcome over the course of therapy. To this end, a frequent measurement of both process and outcome during trauma-focused interventions is necessary. Again, EMA could prove to be a promising approach, as it allows a frequent measurement and a closer investigation of the interplay between sleep quality and symptoms of posttraumatic stress. Of course, such results would then need to be replicated across different populations, including refugees, as this subgroup is at high risk for both PTSD and sleep disturbances.

Prospectively, it could then also be worthwhile for future studies to examine the effects of sleep problems on the different PTSD symptom clusters. Using an EMA design, Dietch and colleagues (2019) in a first study demonstrated that sleep problems especially affected intrusive re-experiencing and hyperarousal, but not the other symptom clusters.

Overlap in hypothesized mechanism and symptoms

A critical issue to consider when investigating both mechanisms of change for psychotherapy in general and in trauma-focused interventions, is that the hypothesized mechanism under investigation should be distinct from the assessed treatment outcome. When looking at the roles of posttraumatic cognitions and sleep problems as mechanisms, it should be noted that both are also part of the diagnostic criteria for PTSD according to DSM-5 (APA, 2013). While sleep problems have been part of the original formulation of the disorder since its

first introduction into the DSM (APA, 1980), posttraumatic cognitions have initially been regarded as important explanatory variables in cognitive models of the disorder (Brown et al., 2019), but not as symptoms per se. With the latest revision, dysfunctional posttraumatic cognitions and associated changes in mood were then introduced as separate symptom cluster (APA, 2013). By definition, a change in one of these hypothesized mechanisms is therefore a change in posttraumatic stress symptoms. Critically, some of the observed patterns from earlier and present studies could then be due to construct overlap. Hence, it is currently not entirely clear whether we are investigating "true" mechanisms of change, or mere symptom dynamics. As a recent review of mechanisms in PTSD treatment concluded, we "may also wonder whether change in cognitions is best understood as a mechanism leading to reduction in 'actual PTSS' [posttraumatic stress symptoms] or just one aspect of a cascade of changes in symptoms" (Kangaslampi & Peltonen, 2022, p. 268). Future studies therefore need to be careful in their choice of measurement instruments to avoid construct overlap. Possibly, currently employed instruments may need to be adapted to ensure a distinction of mechanism and outcome as best as possible, with such adaptations being informed by a clear theoretically-derived rationale. Alternatively, as was done with sleep items on PTSD measures in Study III, outcome measures could be adjusted to no longer contain items assessing the purported mechanism. A similar approach was employed by McLean et al. (2019): to distinguish whether the mediating effect of catastrophizing cognitions on PTSD symptoms was in part due to overlap with PTSD re-experiencing symptoms, the authors conducted secondary analyses using a PTSD score free of re-experiencing symptoms. While such an approach may seem like a pragmatic solution to the problem of overlap between proposed mechanism and symptoms, it should be noted that in both cases the outcome is then a different construct than PTSD in its current conceptualization.

Simultaneous investigation of several mechanisms

An important next step in mechanism research would be to test several processes of change simultaneously in one study. Given the complexity of current interventions, with treatments including several modules and techniques (for an example, see Table 2.2 for the approach employed in Study I and II), as well as the heterogeneity of the disorder itself (Galatzer-Levy & Bryant, 2013), it is highly unlikely that therapeutic change can be accounted for by one single mechanism. Testing multiple mechanisms in a single study or statistical model would then allow for the comparison of the specific effects and estimates of their relative contribution. In an initial study, McLean and colleagues (2019) tested the roles of posttraumatic cognitions and the cognitive emotion regulation strategy *catastrophizing* as mediators for PTSD change in a single model, thereby allowing for comparison of effects. In the combined model, the effect of catastrophizing was significantly greater than the effect of posttraumatic cognitions about the world, but comparable to negative cognitions about the self.

Moreover, it is also likely that different mechanisms influence each other in a sequence of processes, with additive or more complex effects. In their model on active elements and mechanisms in cognitive-behavioral therapies, Cohen et al. (2023) further differentiated mechanisms of change into *activating* and *downstream* mechanisms. The *activating* mechanism is the entry point into the system, directly triggered by an active therapy element. The *downstream* mechanism then refers "to any and all mechanisms that are part of a dynamic cascade of effects that result in change in the outcome(s)" (Cohen et al., 2023, p. 5). Further complicating the issue, what constitutes an *activating* vs. *downstream* mechanism may also differ between individuals.

Regarding the mechanisms investigated in the present thesis, several interplays seem possible. For example, for patients with excessive ruminative thinking, first alleviating rumination, and thereby decreasing cognitive avoidance, could then allow for processing and

critically questioning certain dysfunctional cognitive content. Only when cognitive resources are less occupied with repetitive negative thinking *processes*, could dysfunctional thought *content* substantially be re-evaluated. Indeed, results from the abovementioned study gave first evidence that changes in a different kind of repetitive negative thinking, namely catastrophizing, preceded changes in posttraumatic cognitions during PTSD treatment (McLean et al., 2019). Vice versa, dysfunctional meta-cognitive thought content regarding the usefulness of rumination could maintain the process itself. Considering the interplay between rumination and sleep, ruminative thinking could increase pre-bedtime arousal and thereby hindering restful sleep, as has been shown in empirical studies (Takano et al., 2012; Zoccola et al., 2009). Conversely, impaired concentration following sleep problems or sleep problems themselves could then be subject of dysfunctional interpretations of posttraumatic stress symptoms.

To gain a better understanding of how different mechanisms interactively lead to symptom change, future studies should therefore aim to the simultaneous assessment of several proposed mechanisms. Additionally, to model the interplay between active elements and different mechanisms, more complex statistical approaches like network analytic framework are likely needed (Hofmann et al., 2020; Roefs et al., 2022). Processes would then need to be assessed with high frequency so that a complex interplay between different processes and symptoms could then be disentangled. For most psychotherapy studies, such high-frequent measurement of several processes and outcomes may not be feasible. Moreover, these challenges currently "present a disconnect between complex mechanistic theories and the analytic tools currently available to test these theories" (Alpert et al., 2023, p. 13).

Implications for Future Research into Mechanisms of Change in PTSD Treatment

In addition to the aforementioned implications for future studies investigating posttraumatic cognitions, rumination, and sleep, the present thesis also points toward a number of open questions worthwhile to address in future research on mechanisms in PTSD treatment.

Such research continues to be important and deserves our research effort, as studying mechanisms of how treatments work could hopefully lead to further refining existing interventions, clinical practice, and patient care. Gathering empirical evidence for change mechanism can further ensure that the essential elements of interventions are disseminated appropriately (Kleim et al., 2013).

Active ingredients in trauma-focused treatments

A critical next research focus would be to investigate which treatment components lead to changes in the proposed mechanisms, thereby establishing so-called active ingredients of therapeutic approaches (for an example, see Farmer et al., 2017). Which interventions exactly precipitate and facilitate changes in the mechanisms? How could these interventions then ultimately be adapted to achieve their maximum effect on the mechanisms? Neither in Study I nor in Study II did we link changes in dysfunctional thought content or rumination, respectively, to content of specific sessions or specific interventions, and future studies should aim to address this gap. Further, it would be of interest whether changes in key mechanisms are obtained by specific versus non-specific treatment techniques. Are changes in the proposed mechanisms caused by the techniques designed to address them, or by components common to various treatments? For example, are changes in posttraumatic cognitions and following symptom reduction caused by explicit, formal cognitive restructuring, or does the experiences of a validating therapeutic alliance more implicitly alleviate dysfunctional cognitions about the self?

Such questions are more difficult to answer in naturalistic studies such as Study I and II, characterized by an individual case formulation and a greater heterogeneity of applied techniques. Internet-delivered programs could pose an interesting opportunity here, as content and techniques are delivered more uniformly across patients. Ultimately, if more is known about which techniques achieve the desired effect on established mechanisms of change, we should aim to further refine or possibly amend such techniques to achieve their maximum effect

on the mechanism. As has been argued above, treatments could then be further refined and distilled to their essential elements, and hopefully their efficacy increased.

Specific vs. non-specific mechanisms

The prevailing majority of recommended evidence-based treatments for PTSD are anchored in cognitive behavioral therapy, and share a common framework. Still, it would be worthwhile to investigate whether treatment effects of the various approaches are due to specific vs. non-specific mechanisms. Whereas the dispute of specific vs. non-specific has largely been led regarding treatment content (see also above), it is equally relevant for treatment mechanisms. A specific mechanism would refer to a process that is based on the underlying treatment theory, whereas non-specific mechanisms refer to treatment processes that are common to a wide range of interventions (for an overview of the proposed mechanisms in trauma-focused treatments, see Zalta, 2015). For example, Cognitive Processing Therapy argues that changes in dysfunctional posttraumatic cognitions cause symptom alleviation (Resick et al., 2016), whereas symptom change in Prolonged Exposure Therapy is thought to be attained via changes in habituation processes (Foa & Cahill, 2001). This claim has received empirical support (e.g., Gallagher & Resick, 2012; see Cooper, Clifton, et al., 2017 for a comprehensive review of other potential mechanisms in Prolonged Exposure Therapy). However, changes in symptomatology could also mainly be driven by mechanisms common to wide range of trauma-focused interventions such as a more general increase of self-efficacy and associated functional cognitions and emotions. Additionally, transdiagnostic mechanisms of change – i.e., mechanisms that also drive therapeutic change in treatments across a range of disorders – could come into play.

As has been argued above, due to the complexity of existing interventions, it is highly unlikely that therapeutic change is brought about by one single mechanism. Moreover, both specific and non-specific mechanisms likely interact to produce change in the treatment

outcome. For example, changes in posttraumatic cognitions may be a critical driver of symptom change only when the therapeutic alliance is high compared to when it is low. As a multitude of mechanisms on different levels, both derived from the treatment theory or common to many approaches, is likely to cause symptomatic change, identifying mechanisms of change for trauma-focused psychotherapies will continue to prove difficult. Collecting evidence on potential mechanisms of change should therefore by default be part of any trials on psychological interventions, ideally at several times during the treatment process. Additionally, future studies should ensure to assess both specific and non-specific possible mechanisms, to elaborate how these factors lead to therapeutic benefit.

Individual trajectories of change

So far, models investigating mechanisms of change in previous studies mostly relied on the assumption that the sample under study changes in a rather uniform way, and that the same mechanisms are at work for the whole sample. Also, in the present studies, it was implied that posttraumatic cognitions or rumination change in a similar way for all patients, or that the relationship between mechanism and outcome is unvarying across the sample. Yet, it is possible that change may not come about uniformly for all patients, and subgroups with different trajectories of change in the proposed mechanisms may exist. For example, Dillon and colleagues (2020) highlighted that changes posttraumatic cognitions are not uniform during treatment – which is likely also the case for other mechanisms. Hence, it could be worthwhile for future studies to not focus exclusively on the aggregated group level. Instead, it could be valuable to explore a more personalized analytic approach, and examine whether certain proposed mechanisms are especially relevant for subgroups of patients. It has been well established that certain patient characteristics influence how individuals benefit from treatment (e.g., Cloitre et al., 2016), and such factors may also influence the degree to which certain change mechanisms lead to symptom reduction. Considering for example the traumatic event,

future research should disentangle whether PTSD resulting from different traumatic events and different levels of trauma exposure change via the same mechanisms. Similar, it should be investigated whether comorbid disorders such as depression affect the role of certain mechanisms. Lastly, such moderating factors could also affect which treatment components achieve the desired effect on the proposed mechanisms. For example, for some patients, changes in posttraumatic cognitions could be best facilitated by mindfulness-interventions rather than cognitive restructuring, depending on so far unknown moderating variables. When investigating mechanisms of change in trauma-focused treatment, future studies should therefore also integrate research on possible moderators.

Conclusion

To refine established trauma-focused treatments and to increase their efficacy, it is necessary to understand via which mechanisms these interventions achieve their effects. The present thesis aimed to enhance our understanding of possible mechanisms of change in treatments for PTSD, and presented three empirical studies. In particular, this thesis focused on the roles of posttraumatic cognitions, rumination, and sleep. For these proposed mechanisms, the empirical evidence for their roles as treatment mechanisms was reviewed in detail. Three clinical studies with patients with PTSD then attempted to close current gaps in the research, and to stipulate further research. In summary, the present findings point toward the hypothesized processes to play an important role as mechanisms of change. Nevertheless, final conclusions cannot be drawn, as findings were at times mixed, and the studies are not without their limitations. Implications for further research on the proposed processes and for mechanisms in general have been proposed. By adding to our understanding of the roles of posttraumatic cognitions, rumination, and sleep, the present thesis contributes to our knowledge of mechanisms of change in trauma-focused treatment. Ultimately, increased knowledge on

treatment mechanisms may help in further refining existing treatments and improving clinical care.

Zusammenfassung

Wirkmechanismen in der Behandlung der Posttraumatischen Belastungsstörung:

Die Rolle posttraumatischer Kognitionen, Rumination und Schlaf

Zusammenfassung

Die Posttraumatische Belastungsstörung (PTBS) ist eine häufige psychische Störung, welche als Folge traumatischer Ereignisse auftreten kann (APA, 2013; WHO, 2019). Nach dem aktuellen *Diagnostischen und statistischen Manual psychischer Störungen* (DSM-5; APA, 2013) ist PTBS durch die vier Symptomcluster *intrusives Wiedererleben*, *Vermeidung von trauma-assoziierten Stimuli*, *negative Veränderungen in Kognitionen und Stimmung* sowie *Veränderungen in Erregung und Reaktivität* charakterisiert. Die Anzahl der Symptome und Cluster erfuhr dabei in den verschiedenen Versionen des DSM immer wieder eine Aktualisierung (Rasmussen et al., 2018). So wurde beispielsweise in der letzten Neuerung das Cluster *Negative Veränderungen in Stimmung und Kognitionen* neu mit aufgenommen, während andere Symptome wie zum Beispiel Schlafstörungen schon seit der ursprünglichen Formulierung Teil der Diagnosekriterien waren. PTBS ist dabei eine psychische Störung, die starke Einschränkungen in Funktionalität und Lebensqualität mit sich bringt (Schnurr et al., 2009) und hohe gesellschaftliche Kosten verursacht (McGowan, 2019). Die geschätzte 1-Jahres-Prävalenz beträgt laut repräsentativen Studien in den USA 8% (Kessler et al., 2012), für Deutschland wird diese mit 2,3% angegeben (Maercker et al., 2008).

Für die Wirksamkeit psychotherapeutischer Behandlungen der PTBS existieren zahlreiche Belege (Bisson et al., 2013; Cusack et al., 2016; Lewis et al., 2020; Watts et al., 2013). Dabei zeigt sich für *traumafokussierte* Behandlungen, d.h. Therapien, welche die PTBS durch die direkte Adressierung traumabezogener Erinnerungen, Kognitionen oder Emotionen zu verändern versuchen, eine Überlegenheit gegenüber *nicht-traumafokussierten* Ansätzen (Cusack et al., 2016). Während einerseits wirksame psychologische Therapien existieren, so erfüllen dennoch nach Abschluss traumafokussierter Therapien 33-56% der Patient:innen weiterhin die Diagnosekriterien für eine PTBS oder zeigen eine anhaltend hohe Symptombelastung (Bradley et al., 2005; Cusack et al., 2016; Larsen et al., 2019). Weiterhin stellt Dropout, d.h. der vorzeitige Abbruch einer Therapie, in traumafokussierten Behandlungen

ein häufiges Problem dar (Bisson et al., 2013). Es scheinen demnach nicht alle Patient:innen ausreichend von den verfügbaren Behandlungsmöglichkeiten zu profitieren, und es besteht die dringende Aufgabe, bestehende traumafokussierte Therapien weiter zu optimieren.

Ein möglicher Weg zur Verbesserung bestehender Interventionen besteht darin, den Fokus verstärkt auf *Wirkmechanismen* von Therapien zu richten. Ein Wirkmechanismus wird definiert als *Schritte oder Prozesse, durch welche eine Therapie ihre tatsächliche Wirkung entfaltet und die Veränderung bewirkt* (Kazdin, 2007). Durch die Erforschung von Mechanismen, die zur Symptomreduktion in Therapien führen, können Behandlungen verfeinert und auf ihre essentiellen Elemente destilliert werden. Im vorliegenden Fall bedeutet das, diejenigen (psychologischen) Mechanismen zu identifizieren, welche die Symptomreduktion in traumafokussierten Therapien bewirken. Damit ein Faktor jedoch als Wirkmechanismus gelten kann, müssen verschiedene Kriterien erfüllt und durch empirische Befunde gestützt werden (Kazdin, 2007). Im vergangenen Jahrzehnt wurden verstärkt Wirkmechanismen traumafokussierter Behandlungen untersucht (Kangaslampi & Peltonen, 2022; Nixon & Sloan, 2017). Das Ziel der vorliegenden Dissertation war es, zu dieser Forschung beizutragen und die Faktoren *posttraumatische Kognitionen, Rumination* sowie *Schlaf* hinsichtlich ihrer Rolle als Wirkmechanismen bei PTBS in drei Studien genauer zu untersuchen.

Das Ziel von **Studie I** bestand darin, die Rolle posttraumatischer Kognitionen als Wirkmechanismus in der PTBS-Behandlung genauer zu untersuchen. Bisher konnte eine Vielzahl empirischer Studien gute Belege für die Veränderung kognitiver *Inhalte* als relevanter Wirkmechanismus erbringen (für Übersichten, siehe Brown et al., 2019; LoSavio et al., 2017). Mehrere Studien zeigten, dass Veränderungen in Kognitionen den Symptomveränderungen im Therapieverlauf vorausgehen und diese vorhersagen (Kleim et al., 2013; Kumpula et al., 2017; Schumm et al., 2015; Zalta et al., 2014). Bestehende Studien waren jedoch dahingehend

limitiert, als dass sie ausnahmslos den Effekt von posttraumatischen Kognitionen auf die Gesamt-Symptomschwere untersuchten. PTBS zeichnet sich jedoch durch eine große Multidimensionalität und Heterogenität aus (Galatzer-Levy & Bryant, 2013), was sich wie oben dargestellt auch in den verschiedenen Symptom-Clustern widerspiegelt.

Studie I untersuchte daher, welchen Einfluss die Veränderung dysfunktionaler Kognitionen auf die Veränderungen der verschiedenen PTBS-Symptomcluster im Therapieverlauf hat. Hierzu wurden während einer traumafokussierten Therapie bei $N = 61$ Patient:innen mit PTBS alle fünf Wochen PTBS-Symptomatik und dysfunktionale Kognitionen gemessen. Basierend auf theoretischen Modellen der PTBS (Ehlers & Clark, 2000) wurde eine differentielle Beeinflussung der verschiedenen Cluster durch posttraumatische Kognitionen postuliert. Die Ergebnisse der Analysen zeigten zwar einerseits, dass sie verschiedenen Cluster durchaus differentiell vorhergesagt wurden, jedoch war dieser Effekt abhängig von dem jeweils gewählten statistischen Modell. Zudem zeigte sich, dass die Beziehung zwischen den Clustern und posttraumatischen Kognitionen durch einen bi-direktionalen Zusammenhang charakterisiert war, welcher in zukünftigen Studien genauer untersucht werden sollte.

Das Ziel von **Studie II** bestand in der Untersuchung von traumabezogener Rumination, also eines kognitiven *Prozesses*, als möglichem Wirkmechanismus in der PTBS-Behandlung. Traumabezogener Rumination, definiert als grüblerisches, repetitives negatives Denken über das Trauma oder dessen Folgen (Michael et al., 2007), wird eine wichtige Rolle in der Entstehung und Aufrechterhaltung der PTBS zugeschrieben (Ehring, Frank, et al., 2008; Moulds et al., 2020; Szabo et al., 2017). Rumination wird dabei als dysfunktionale kognitive Vermeidungsstrategie verstanden, welche die PTBS langfristig aufrechterhält, indem sie Symptome verstärkt, Veränderungen in kognitiven Inhalten verhindert sowie eine Elaboration des Traumagedächtnisses blockiert (Ehlers & Clark, 2000). Bisher wurde jedoch die mögliche Rolle als Wirkmechanismus in der Therapie noch nicht untersucht.

Das Ziel der **Studie II** war es daher, zu untersuchen, ob Veränderungen in Rumination Veränderungen in der PTBS-Symptomatik im Therapieverlauf vorhergehen und diese vorhersagen. Hierzu wurden während einer traumafokussierten Therapie bei $N = 80$ Patient:innen mit PTBS wöchentlich PTBS-Symptomatik und Rumination gemessen, und sogenannte *cross-lagged associations*, d.h. der Effekt von Rumination zu Zeitpunkt t_x auf PTBS-Symptomatik zum Zeitpunkt t_{x+1}, untersucht. Die Ergebnisse zeigten zunächst, dass Rumination im Therapieverlauf abnahm. Weiterhin sagte Veränderung in Rumination auch Veränderung in der PTBS-Symptomatik vorher, was für die Rolle von Rumination als Wirkmechanismus spricht. Allerdings war auch dieser Effekt abhängig vom gewählten statistischen Modell. Eine wichtige Limitation der Studie bestand in dem Fehlen einer Kontrollgruppe, so dass die Ergebnisse zwar erste Hinweise für Rumination als Wirkmechanismus liefern, jedoch in weiteren Studien repliziert werden sollten.

Das Ziel von **Studie III** war es, die Rolle von Schlaf bei Geflüchteten mit PTBS genauer zu untersuchen. Viele Patient:innen mit PTBS leiden an Schlafstörungen (Koffel et al., 2016; Maher et al., 2006), was auch durch objektive Befunde untermauert wird (Baglioni et al., 2016). Zudem konnte ein starker Zusammenhang von Schlafstörungen zur PTBS-Schwere nachgewiesen werden (Cox et al., 2018). Studien deuten weiterhin darauf hin, dass Schlafstörungen vor traumatischen Ereignissen die Entwicklung einer PTBS vorhersagen können (Fan et al., 2017; Koffel et al., 2013), und dass die Verbesserung von Schlafstörungen während traumafokussierter Therapie auch mit besserem Ansprechen auf die Therapie einhergeht (Zalta et al., 2020). Eine Risikogruppe für PTBS und mit hoher Prävalenz von Schlafstörungen stellen Geflüchtete dar. Mehrere Studien belegen, dass 75-99% der Geflüchteten an Schlafstörungen leiden (Al-Smadi et al., 2019; Lies et al., 2019, 2021; Sandahl et al., 2017), jedoch liegen keine derartigen Daten für Deutschland vor. Zudem beziehen sich die Studienergebnisse meist auf Geflüchtete im Allgemeinen, nicht nur Geflüchtete mit PTBS.

Um zukünftig die Rolle von Schlaf als Wirkmechanismus in der PTBS-Behandlung für diese besonders vulnerable Gruppe genauer zu untersuchen, wurde in **Studie III** in einem ersten Schritt daher zunächst die Prävalenz von Schlafstörungen und deren Zusammenhang mit der PTBS-Schwere untersucht. Weiterhin wurde die Rolle von Schlafstörungen in der Vorhersage von psychosozialen Einschränkungen und subjektiver Lebensqualität als zusätzlicher Prädiktor neben PTBS untersucht.

Die Ergebnisse (N = 70) zeigten eine Prävalenz von 100% für klinisch relevante Schlafstörungen, sowie signifikante Zusammenhänge zur PTBS-Symptomatik (erfasst durch Selbstbericht sowie Fremdeinschätzung). Entgegen der Hypothesen waren Schlafstörungen jedoch kein signifikanter Prädiktor für psychosoziale Einschränkungen oder Lebensqualität. Möglicherweise leisten Schlafstörungen neben dem großen Einfluss der anderen posttraumatischen Symptome keinen zusätzlichen signifikanten Beitrag zur Prädiktion.

Zusammengefasst war es das Ziel der vorliegenden Dissertation, die Rolle verschiedener Prozesse – nämlich posttraumatischer Kognitionen, Rumination und Schlafstörungen – hinsichtlich ihrer Rolle als Wirkmechanismen eingehender zu untersuchen. Da der bisherige Forschungsstand zu den drei Prozessen verschieden weit fortgeschritten war, setzten die drei Studien entsprechend an verschiedenen Kriterien an. Im Allgemeinen zeigten die Ergebnisse die hohe Relevanz der untersuchten Prozesse, konnten jedoch keine eindeutigen Belege für deren Rolle als Wirkmechanismen erbringen. Eine Replikation in zukünftigen Studien ist daher angezeigt. Weiterhin wurden in der Arbeit die Limitationen der einzelnen Studien beleuchtet, sowie die teils unerwarteten Ergebnisse eingehend diskutiert. Schließlich wurden Implikationen für die weitere Forschung bezüglich der drei beleuchteten Prozesse, aber auch bezüglich Wirkmechanismus-Forschung bei PTBS im Allgemeinen, abgeleitet. Die Wirkmechanismus-Forschung sieht sich komplexen Herausforderungen gegenüber, ist jedoch ein weiterhin

vielversprechender Weg, bestehende PTBS-Behandlungen weiter zu verfeinern, um den Behandlungserfolg für Patient:innen mit PTBS zu maximieren.

References

Al-Smadi, A. M., Tawalbeh, L. I., Gammoh, O. S., Ashour, A., Tayfur, M., & Attarian, H. (2019). The prevalence and the predictors of insomnia among refugees. *Journal of Health Psychology*, *24*(8), 1125–1133. https://doi.org/10.1177/1359105316687631

Alpert, E., Shotwell Tabke, C., Cole, T. A., Lee, D. J., & Sloan, D. M. (2023). A systematic review of literature examining mediators and mechanisms of change in empirically supported treatments for posttraumatic stress disorder. *Clinical Psychology Review*, *103*, 102300. https://doi.org/10.1016/j.cpr.2023.102300

American Psychiatric Association. (1980). *Diagnostic and Statistical Manual of Mental Disorders (3rd ed.)*.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. https://doi.org/https://doi.org/10.1176/appi.books.9780890425596

American Psychiatric Association. (2017). *Clinical Practice Guideline for the Treatment of Posttraumatic Stress Disorder (PTSD) in Adults*. https://www.apa.org/ptsd-guideline

Armour, C., Contractor, A., Shea, T., Elhai, J. D., & Pietrzak, K. R. H. (2016). Factor structure of the PTSD Checklist for DSM-5: Relationships among symptom clusters, anger, and impulsivity. *Journal of Nervous and Mental Disease*, *204*(2), 108–115. https://doi.org/10.1097/NMD.0000000000000430

Babson, K., Feldner, M., Badour, C., Trainor, C., Blumenthal, H., Sachs-Ericsson, N., & Schmidt, N. (2011). Posttraumatic stress and sleep: Differential relations across types of symptoms and sleep problems. *Journal of Anxiety Disorders*, *25*(5), 706–713. https://doi.org/10.1016/j.janxdis.2011.03.007

Baglioni, C., Nanovska, S., Regen, W., Spiegelhalder, K., Feige, B., Nissen, C., Reynolds, C. F., & Riemann, D. (2016). Sleep and mental disorders: A meta-analysis of polysomnographic research. *Psychological Bulletin*, *142*(9), 969–990.

https://doi.org/10.1037/bul0000053

Beierl, E. T., Böllinghaus, I., Clark, D. M., Glucksman, E., & Ehlers, A. (2019). Cognitive paths from trauma to posttraumatic stress disorder: A prospective study of Ehlers and Clark's model in survivors of assaults or road traffic collisions. *Psychological Medicine*, 1–10. https://doi.org/10.1017/S0033291719002253

Birrer, E., & Michael, T. (2011). Rumination in PTSD as well as in traumatized and non-traumatized depressed patients: A cross-sectional clinical study. *Behavioural and Cognitive Psychotherapy*, *39*(4), 381–397. https://doi.org/10.1017/S1352465811000087

Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder: Systematic review and meta-analysis. *British Journal of Psychiatry*, *190*, 97–104. https://doi.org/10.1192/bjp.bp.106.021402

Bisson, J. I., Roberts, N. P., Andrew, M., Cooper, R., & Lewis, C. (2013). Psychological therapies for chronic post-traumatic stress disorder (PTSD) in adults. *The Cochrane Database of Systematic Reviews*, *2013*(12). https://doi.org/10.1002/14651858.CD003388.PUB4

Bonfils, K. A., Tennity, C. L., Congedo, B. A., Dolowich, B. A., Hammer, L. A., & Haas, G. L. (2022). Functional outcomes from psychotherapy for people with posttraumatic stress disorder: A meta-analysis. *Journal of Anxiety Disorders*, *89*, 102576. https://doi.org/10.1016/j.janxdis.2022.102576

Bonn-Miller, M. O., Boden, M. T., Vujanovic, A. A., & Drescher, K. D. (2013). Prospective investigation of the impact of cannabis use disorders on posttraumatic stress disorder symptoms among veterans in residential treatment. *Psychological Trauma: Theory, Research, Practice, and Policy*, *5*(2), 193–200. https://doi.org/10.1037/A0026621

Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-

analysis of psychotherapy for PTSD. *American Journal of Psychiatry*, *162*(2), 214–227. https://doi.org/10.1176/appi.ajp.162.2.214

Breslau, N. (2009). The epidemiology of trauma, PTSD, and other posttrauma disorders. *Trauma, Violence, and Abuse*, *10*(3), 198–210. https://doi.org/10.1177/1524838009334448

Brown, L. A., Belli, G. M., Asnaani, A., & Foa, E. B. (2019). A review of the role of negative cognitions about oneself, others, and the world in the treatment of PTSD. *Cognitive Therapy and Research*, *43*(1), 143–173. https://doi.org/10.1007/s10608-018-9938-1

Bruijniks, S. J. E., Sijbrandij, M., Schlinkert, C., & Huibers, M. J. H. (2018). Isolating therapeutic procedures to investigate mechanisms of change in cognitive behavioral therapy for depression. *Journal of Experimental Psychopathology*, *9*(4). https://doi.org/10.1177/2043808718800893/ASSET/IMAGES/LARGE/10.1177_2043808718800893-FIG1.JPEG

Cloitre, M., Petkova, E., Su, Z., & Weiss, B. J. (2016). Patient characteristics as a moderator of posttraumatic stress disorder treatment outcome: combining symptom burden and strengths. *BJPsych Open*, *2*(2), 101–106. https://doi.org/10.1192/BJPO.BP.115.000745

Cohen, Z. D., Barnes-Horowitz, N. M., Forbes, C. N., & Craske, M. G. (2023). Measuring the active elements of cognitive-behavioral therapies. *Behaviour Research and Therapy*, *167*, 104364. https://doi.org/10.1016/J.BRAT.2023.104364

Cooper, A. A., Clifton, E. G., & Feeny, N. C. (2017). An empirical review of potential mediators and mechanisms of prolonged exposure therapy. *Clinical Psychology Review*, *56*, 106–121. https://doi.org/10.1016/J.CPR.2017.07.003

Cooper, A. A., Zoellner, L. A., Roy-Byrne, P., Mavissakalian, M. R., & Feeny, N. C. (2017). Do changes in trauma-related beliefs predict PTSD symptom improvement in prolonged exposure and sertraline? *Journal of Consulting and Clinical Psychology*, *85*(9), 873–882.

https://doi.org/10.1037/ccp0000220

Cox, R. C., Alex McIntyre, W., & Olatunji, B. O. (2018). Interactive effects of insomnia symptoms and trauma exposure on PTSD: Examination of symptom specificity. *Psychological Trauma: Theory, Research, Practice, and Policy*, *10*(5), 508–514. https://doi.org/10.1037/TRA0000336

Cox, R. C., Tuck, B. M., & Olatunji, B. O. (2017). Sleep Disturbance in Posttraumatic Stress Disorder: Epiphenomenon or Causal Factor? *Current Psychiatry Reports*, *19*(4). https://doi.org/10.1007/s11920-017-0773-y

Cuijpers, P., Reijnders, M., & Huibers, M. J. H. (2019). The Role of Common Factors in Psychotherapy Outcomes. *Annual Review of Clinical Psychology*, *15*, 207–231. https://doi.org/10.1146/annurev-clinpsy-050718-095424

Cusack, K., Jonas, D. E., Forneris, C. A., Wines, C., Sonis, J., Middleton, J. C., Feltner, C., Brownley, K. A., Olmsted, K. R., Greenblatt, A., Weil, A., & Gaynes, B. N. (2016). Psychological treatments for adults with posttraumatic stress disorder: A systematic review and meta-analysis. *Clinical Psychology Review*, *43*, 128–141. https://doi.org/10.1016/j.cpr.2015.10.003

DeViva, J. C., Rosen, M. I., Cooney, N. L., & Black, A. C. (2020). Ecological momentary assessment of sleep and PTSD symptoms in a veteran sample. *Psychological Trauma: Theory, Research, Practice, and Policy*, *12*(2), 186–192. https://doi.org/10.1037/tra0000494

DeViva, J. C., Zayfert, C., & Mellman, T. A. (2004). Factors associated with insomnia among civilians seeking treatment for PTSD: an exploratory study. *Behavioral Sleep Medicine*, *2*(3), 162–176. https://doi.org/10.1207/s15402010bsm0203_5

Dietch, J. R., Ruggero, C. J., Schuler, K., Taylor, D. J., Luft, B. J., & Kotov, R. (2019). Posttraumatic stress disorder symptoms and sleep in the daily lives of world trade center

responders. *Journal of Occupational Health Psychology*, *24*(6), 689–702. https://doi.org/10.1037/ocp0000158

Dillon, K. H., Hale, W. J., LoSavio, S. T., Wachen, J. S., Pruiksma, K. E., Yarvis, J. S., Mintz, J., Litz, B. T., Peterson, A. L., & Resick, P. A. (2020). Weekly Changes in Blame and PTSD Among Active-Duty Military Personnel Receiving Cognitive Processing Therapy. *Behavior Therapy*, *51*(3), 386–400. https://doi.org/10.1016/j.beth.2019.06.008

Dondanville, K. A., Blankenship, A. E., Molino, A., Resick, P. A., Wachen, J. S., Mintz, J., Yarvis, J. S., Litz, B. T., Borah, E. V., Roache, J. D., Young-McCaughan, S., Hembree, E. A., & Peterson, A. L. (2016). Qualitative examination of cognitive change during PTSD treatment for active duty service members. *Behaviour Research and Therapy*, *79*, 1–6. https://doi.org/10.1016/j.brat.2016.01.003

Doran, J. M., & DeViva, J. (2018). A naturalistic evaluation of evidence-based treatment for veterans with PTSD. *Traumatology*, *24*(3), 157–167. https://doi.org/10.1037/TRM0000140

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*(4), 319–345. https://doi.org/10.1016/S0005-7967(99)00123-0

Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., & Fennell, M. (2005). Cognitive therapy for post-traumatic stress disorder: Development and evaluation. *Behaviour Research and Therapy*, *43*(4), 413–431. https://doi.org/10.1016/j.brat.2004.03.006

Ehlers, A., & Steil, R. (1995). Maintenance of Intrusive Memories in Posttraumatic Stress Disorder: A Cognitive Approach. *Behavioural and Cognitive Psychotherapy*, *23*(3), 217–249. https://doi.org/10.1017/S135246580001585X

Ehring, T., & Ehlers, A. (2014). Does rumination mediate the relationship between emotion regulation ability and posttraumatic stress disorder? *European Journal of*

Psychotraumatology, *5*(1), 23547. https://doi.org/10.3402/ejpt.v5.23547

Ehring, T., Ehlers, A., & Glucksman, E. (2008). Do cognitive models help in predicting the severity of posttraumatic Stress disorder, phobia, and depression after motor vehicle accidents? A prospective longitudinal study. *Journal of Consulting and Clinical Psychology*, *76*(2), 219–230. https://doi.org/10.1037/0022-006X.76.2.219

Ehring, T., Frank, S., & Ehlers, A. (2008). The role of rumination and reduced concreteness in the maintenance of posttraumatic stress disorder and depression following trauma. *Cognitive Therapy and Research*, *32*(4), 488–506. https://doi.org/10.1007/s10608-006-9089-7

Ehring, T., Fuchs, N., & Kläsener, I. (2009). The effects of experimentally induced rumination versus distraction on analogue posttraumatic stress symptoms. *Behavior Therapy*, *40*(4), 403–413. https://doi.org/10.1016/j.beth.2008.10.001

Ehring, T., Szeimies, A. K., & Schaffrick, C. (2009). An experimental analogue study into the role of abstract thinking in trauma-related rumination. *Behaviour Research and Therapy*, *47*(4), 285–293. https://doi.org/10.1016/j.brat.2008.12.011

Ehring, T., Welboren, R., Morina, N., Wicherts, J. M., Freitag, J., & Emmelkamp, P. M. G. (2014). Meta-analysis of psychological treatments for posttraumatic stress disorder in adult survivors of childhood abuse. *Clinical Psychology Review*, *34*(8), 645–657. https://doi.org/10.1016/j.cpr.2014.10.004

Ehring, T., Zetsche, U., Weidacker, K., Wahl, K., Schönfeld, S., & Ehlers, A. (2011). The Perseverative Thinking Questionnaire (PTQ): Validation of a content-independent measure of repetitive negative thinking. *Journal of Behavior Therapy and Experimental Psychiatry*, *42*(2), 225–232. https://doi.org/10.1016/j.jbtep.2010.12.003

Elwood, L. S., Hahn, K. S., Olatunji, B. O., & Williams, N. L. (2009). Cognitive vulnerabilities to the development of PTSD: A review of four vulnerabilities and the

proposal of an integrative vulnerability model. *Clinical Psychology Review*, *29*(1), 87–100. https://doi.org/10.1016/j.cpr.2008.10.002

Fan, F., Zhou, Y., & Liu, X. (2017). Sleep Disturbance Predicts Posttraumatic Stress Disorder and Depressive Symptoms: A Cohort Study of Chinese Adolescents. *The Journal of Clinical Psychiatry*, *78*(7), 19701. https://doi.org/10.4088/JCP.15M10206

Farmer, C. C., Mitchell, K. S., Parker-Guilbert, K., & Galovski, T. E. (2017). Fidelity to the Cognitive Processing Therapy Protocol: Evaluation of Critical Elements. *Behavior Therapy*, *48*(2), 195–206. https://doi.org/10.1016/j.beth.2016.02.009

Foa, E. B., & Cahill, S. P. (2001). Psychological theories: Emotional processing. In N. J. Smelser & P. B. Baltes (Eds.), *International Encyclopedia of Social & Behavioral Sciences* (pp. 12363–12369). Elsevier.

Foa, E. B., Hembree, E., & Rothbaum, B. (2014). *Handbuch der prolongierten Exposition. Basiskonzepte und Anwendung*. 192. https://www.gp-probst.de/

Foa, E. B., & Rauch, S. A. M. (2004). Cognitive changes during prolonged exposure versus prolonged exposure plus cognitive restructuring in female assault survivors with posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *72*(5), 879–884. https://doi.org/10.1037/0022-006X.72.5.879

Galatzer-Levy, I. R., & Bryant, R. A. (2013). 636,120 ways to have posttraumatic stress disorder. *Perspectives on Psychological Science*, *8*(6), 651–662. https://doi.org/10.1177/1745691613504115.

Gallagher, M. W. (2017). Transdiagnostic mechanisms of change and cognitive-behavioral treatments for PTSD. In *Current Opinion in Psychology* (Vol. 14, pp. 90–95). https://doi.org/10.1016/j.copsyc.2016.12.002

Gallagher, M. W., & Resick, P. A. (2012). Mechanisms of Change in Cognitive Processing Therapy and Prolonged Exposure Therapy for PTSD: Preliminary Evidence for the

Differential Effects of Hopelessness and Habituation. *Cognitive Therapy and Research*, *36*(6), 750–755. https://doi.org/10.1007/S10608-011-9423-6

García, F. E., Vázquez, C., & Inostroza, C. (2019). Predictors of post-traumatic stress symptoms following occupational accidents: A longitudinal study. *Anxiety, Stress and Coping*, *32*(2), 168–178. https://doi.org/10.1080/10615806.2019.1566533

Germain, A. (2013). Sleep disturbances as the hallmark of PTSD: Where are we now? *American Journal of Psychiatry*, *170*(4), 372–382. https://doi.org/10.1176/appi.ajp.2012.12040432

Germain, A., Buysse, D. J., Shear, M. K., Fayyad, R., & Austin, C. (2004). Clinical correlates of poor sleep quality in posttraumatic stress disorder. *Journal of Traumatic Stress*, *17*(6), 477–484. https://doi.org/10.1007/S10960-004-5796-6

Gilman, R., Schumm, J. A., & Chard, K. M. (2012). Hope as a change mechanism in the treatment of posttraumatic stress disorder. *Psychological Trauma: Theory, Research, Practice, and Policy*, *4*(3), 270–277. https://doi.org/10.1037/a0024252

Gobin, R. L., MacKintosh, M. A., Willis, E., Allard, C. B., Kloezeman, K., & Morland, L. A. (2018). Predictors of differential PTSD treatment outcomes between veteran and civilian women after cognitive processing therapy. *Psychological Trauma : Theory, Research, Practice and Policy*, *10*(2), 173–182. https://doi.org/10.1037/TRA0000266

Goldstein, A. N., & Walker, M. P. (2014). The role of sleep in emotional brain function. In *Annual Review of Clinical Psychology* (Vol. 10, pp. 679–708). NIH Public Access. https://doi.org/10.1146/annurev-clinpsy-032813-153716

Gómez de La Cuesta, G., Schweizer, S., Diehle, J., Young, J., & Meiser-Stedman, R. (2019). The relationship between maladaptive appraisals and posttraumatic stress disorder: A meta-analysis. *European Journal of Psychotraumatology*, *10*(1). https://doi.org/https://doi.org/10.1080/20008198.2019.1620084

Goodson, J., Helstrom, A., Halpern, J. M., Ferenschak, M. P., Gillihan, S. J., & Powers, M. B. (2011). Treatment of Posttraumatic Stress Disorder in U.S. Combat Veterans: A Meta-Analytic Review. *Http://Dx.Doi.Org/10.2466/02.09.15.16.PR0.109.5.573-599*, *109*(2), 573–599. https://doi.org/10.2466/02.09.15.16.PR0.109.5.573-599

Hellmuth, J. C., Stappenbeck, C. A., Hoerster, K. D., & Jakupcak, M. (2012). Modeling PTSD Symptom Clusters, Alcohol Misuse, Anger, and Depression as They Relate to Aggression and Suicidality in Returning U.S. Veterans. *Journal of Traumatic Stress*, *25*(5), 527–534. https://doi.org/10.1002/jts.21732

Hofmann, S. G., Curtiss, J. E., & Hayes, S. C. (2020). Beyond linear mediation: Toward a dynamic network approach to study treatment processes. *Clinical Psychology Review*, *76*, 101824. https://doi.org/10.1016/J.CPR.2020.101824

Imel, Z. E., Laska, K., Jakupcak, M., & Simpson, T. L. (2013). Meta-analysis of dropout in treatments for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *81*(3), 394–404. https://doi.org/10.1037/a0031474

Iverson, K. M., King, M. W., Cunningham, K. C., & Resick, P. A. (2015). Rape survivors' trauma-related beliefs before and after cognitive processing therapy: Associations with PTSD and depression symptoms. *Behaviour Research and Therapy*, *66*, 49–55. https://doi.org/10.1016/j.brat.2015.01.002

Kangaslampi, S., & Peltonen, K. (2022). Mechanisms of change in psychological interventions for posttraumatic stress symptoms: A systematic review with recommendations. *Current Psychology*, *41*(1), 258–275. https://doi.org/10.1007/s12144-019-00478-5

Karl, A., Rabe, S., Zöllner, T., Maercker, A., & Stopa, L. (2009). Negative self-appraisals in treaatment-seeking survivors of motor vehicle accidents. *Journal of Anxiety Disorders*, *23*(6), 775–781. https://doi.org/10.1016/j.janxdis.2009.03.001

References

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, *3*(1), 1–27. https://doi.org/10.1146/annurev.clinpsy.3.022806.091432

Kazdin, A. E. (2009). Understanding how and why psychotherapy leads to change. *Https://Doi.Org/10.1080/10503300802448899*, *19*(4–5), 418–428. https://doi.org/10.1080/10503300802448899

Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H. U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research*, *21*(3), 169–184. https://doi.org/10.1002/MPR.1359

Kleim, B., Ehlers, A., & Glucksman, E. (2012). Investigating cognitive pathways to psychopathology: Predicting depression and posttraumatic stress disorder from early responses after assault. *Psychological Trauma: Theory, Research, Practice, and Policy*, *4*(5), 527–537. https://doi.org/10.1037/a0027006

Kleim, B., Grey, N., Wild, J., Nussbeck, F. W., Stott, R., Hackmann, A., Clark, D. M., & Ehlers, A. (2013). Cognitive change predicts symptom reduction with cognitive therapy for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *81*(3), 383–393. https://doi.org/10.1037/a0031290

Kleim, B., Wysokowsky, J., Schmid, N., Seifritz, E., & Rasch, B. (2016). Effects of sleep after experimental trauma on intrusive emotional memories. *Sleep*, *39*(12), 2125–2132. https://doi.org/10.5665/sleep.6310

Kobayashi, I., & Mellman, T. A. (2012). Gender Differences in Sleep During the Aftermath of Trauma and the Development of Posttraumatic Stress Disorder. *Behavioral Sleep Medicine*, *10*(3), 180–190. https://doi.org/10.1080/15402002.2011.654296

Koenen, K. C., Ratanatharathorn, A., Ng, L., McLaughlin, K. A., Bromet, E. J., Stein, D. J.,

Karam, E. G., Meron Ruscio, A., Benjet, C., Scott, K., Atwoli, L., Petukhova, M., Lim, C. C. W., Aguilar-Gaxiola, S., Al-Hamzawi, A., Alonso, J., Bunting, B., Ciutan, M., De Girolamo, G., … Kessler, R. C. (2017). Posttraumatic stress disorder in the World Mental Health Surveys. *Psychological Medicine*, *47*(13), 2260–2274. https://doi.org/10.1017/S0033291717000708

Koffel, E., Khawaja, I. S., & Germain, A. (2016). Sleep disturbances in posttraumatic stress disorder: Updated review and implications for treatment. *Psychiatric Annals*, *46*(3), 173–176. https://doi.org/10.3928/00485713-20160125-01

Koffel, E., Polusny, M. A., Arbisi, P. A., & Erbes, C. R. (2013). Pre-deployment daytime and nighttime sleep complaints as predictors of post-deployment PTSD and depression in National Guard troops. *Journal of Anxiety Disorders*, *27*(5), 512–519. https://doi.org/10.1016/j.janxdis.2013.07.003

Kraemer, H. C., Wilson, G. T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry*, *59*(10), 877–883. https://doi.org/10.1001/archpsyc.59.10.877

Kumpula, M. J., Pentel, K. Z., Foa, E. B., LeBlanc, N. J., Bui, E., McSweeney, L. B., Knowles, K., Bosley, H., Simon, N. M., & Rauch, S. A. M. (2017). Temporal sequencing of change in posttraumatic cognitions and PTSD symptom reduction during prolonged exposure therapy. *Behavior Therapy*, *48*(2), 156–165. https://doi.org/10.1016/j.beth.2016.02.008

Laposa, J. M., & Rector, N. A. (2012). The prediction of intrusions following an analogue traumatic event: Peritraumatic cognitive processes and anxiety-focused rumination versus rumination in response to intrusions. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(3), 877–883. https://doi.org/10.1016/j.jbtep.2011.12.007

Larsen, S. E., Fleming, C. J. E., & Resick, P. A. (2019). Residual symptoms following

empirically supported treatment for PTSD. *Psychological Trauma: Theory, Research, Practice, and Policy*, *11*(2), 207–215. https://doi.org/10.1037/tra0000384

Lewis, C., Roberts, N. P., Andrew, M., Starling, E., & Bisson, J. I. (2020). Psychological therapies for post-traumatic stress disorder in adults: Systematic review and meta-analysis. *European Journal of Psychotraumatology*, *11*(1), 1729633. https://www.tandfonline.com/doi/full/10.1080/20008198.2020.1729633

Lies, J., Jobson, L., Mascaro, L., Whyman, T., & Drummond, S. P. A. (2021). Postmigration stress and sleep disturbances mediate the relationship between trauma exposure and posttraumatic stress symptoms among Syrian and Iraqi refugees. *Journal of Clinical Sleep Medicine : JCSM : Official Publication of the American Academy of Sleep Medicine*, *17*(3), 479. https://doi.org/10.5664/JCSM.8972

Lies, J., Jones, L., & Ho, R. (2019). The management of post-traumatic stress disorder and associated pain and sleep disturbance in refugees. *BJPsych Advances*, *25*(3), 196–206. https://doi.org/10.1192/bja.2019.7

Littleton, H., Buck, K., Rosman, L., & Grills-Taquechel, A. (2012). From survivor to thriver: A pilot study of an online program for rape victims. *Cognitive and Behavioral Practice*, *19*(2), 315–327. https://doi.org/10.1016/j.cbpra.2011.04.002

Lommen, M. J. J., Grey, N., Clark, D. M., Wild, J., Stott, R., & Ehlers, A. (2016). Sleep and treatment outcome in posttraumatic stress disorder: Results from an effectiveness study. *Depression and Anxiety*, *33*(7), 575–583. https://doi.org/10.1002/da.22420

Lorenzo-Luaces, L., German, R. E., & DeRubeis, R. J. (2015). It's complicated: The relation between cognitive change procedures, cognitive change, and symptom change in cognitive therapy for depression. *Clinical Psychology Review*, *41*, 3–15. https://doi.org/10.1016/J.CPR.2014.12.003

LoSavio, S. T., Dillon, K. H., & Resick, P. A. (2017). Cognitive factors in the development,

maintenance, and treatment of post-traumatic stress disorder. *Current Opinion in Psychology*, *14*, 18–22. https://doi.org/10.1016/j.copsyc.2016.09.006

Lunney, C. A., & Schnurr, P. P. (2007). Domains of quality of life and symptoms in male veterans treated for posttraumatic stress disorder. *Journal of Traumatic Stress*, *20*(6), 955–964. https://doi.org/10.1002/jts.20269

Maercker, A., Forstmeier, S., Wagner, B., Glaesmer, H., & Brähler, E. (2008). Posttraumatische Belastungsstörungen in Deutschland. Ergebnisse einer gesamtdeutschen epidemiologischen Untersuchung. *Nervenarzt*, *79*(5), 577–586. https://doi.org/10.1007/s00115-008-2467-5

Maher, M. J., Rego, S. A., & Asnis, G. M. (2006). Sleep disturbances in patients with post-traumatic stress disorder: Epidemiology, impact and approaches to management. In *CNS Drugs* (Vol. 20, Issue 7, pp. 567–590). CNS Drugs. https://doi.org/10.2165/00023210-200620070-00003

Marcks, B. A., Weisberg, R. B., Edelen, M. O., & Keller, M. B. (2010). The relationship between sleep disturbance and the course of anxiety disorders in primary care patients. *Psychiatry Research*, *178*(3), 487–492. https://doi.org/10.1016/J.PSYCHRES.2009.07.004

McGowan, I. W. (2019). The Economic Burden of PTSD. A brief review of salient literature. *International Journal of Psychiatry and Mental Health*, 20–26. https://doi.org/10.36811/ijpmh.2019.110003

McLean, C. P., Yeh, R., Rosenfield, D., & Foa, E. B. (2015). Changes in negative cognitions mediate PTSD symptom reductions during client-centered therapy and prolonged exposure for adolescents. *Behaviour Research and Therapy*, *68*, 64–69. https://doi.org/10.1016/j.brat.2015.03.008

McLean, C. P., Zang, Y., Gallagher, T., Suzuki, N., Yarvis, J. S., Litz, B. T., Mintz, J.,

Young-McCaughan, S., Peterson, A. L., & Foa, E. B. (2019). Trauma-related cognitions and cognitive emotion regulation as mediators of PTSD change among treatment-seeking active-duty military personnel With PTSD. *Behavior Therapy*, *50*(6), 1053–1062. https://doi.org/10.1016/j.beth.2019.03.006

Mellman, T. A., Pigeon, W. R., Nowell, P. D., & Nolan, B. (2007). Relationships between REM sleep findings and PTSD symptoms during the early aftermath of trauma. *Journal of Traumatic Stress*, *20*(5), 893–901. https://doi.org/10.1002/jts.20246

Michael, T., Halligan, S. L., Clark, D. M., & Ehlers, A. (2007). Rumination in posttraumatic stress disorder. *Depression and Anxiety*, *24*(5), 307–317. https://doi.org/10.1002/da.20228

Mitchell, M. A., Contractor, A. A., Dranger, P., & Shea, M. T. (2016). Unique relations between counterfactual thinking and DSM-5 PTSD symptom clusters. *Psychological Trauma: Theory, Research, Practice, and Policy*, *8*(3), 293–300. https://doi.org/10.1037/tra0000089

Monson, C. M., Macdonald, A., Vorstenbosch, V., Shnaider, P., Goldstein, E. S. R., Ferrier-Auerbach, A. G., & Mocciola, K. E. (2012). Changes in Social Adjustment With Cognitive Processing Therapy: Effects of Treatment and Association With PTSD Symptom Change. *Journal of Traumatic Stress*, *25*(5), 519–526. https://doi.org/10.1002/jts.21735

Moulds, M. L., Bisby, M. A., Wild, J., & Bryant, R. A. (2020). Rumination in posttraumatic stress disorder: A systematic review. *Clinical Psychology Review*, *82*(September), 101910. https://doi.org/10.1016/j.cpr.2020.101910

Murray, J., Ehlers, A., & Mayou, R. A. (2002). Dissociation and post-traumatic stress disorder: Two prospective studies of road traffic accident survivors. *British Journal of Psychiatry*, *180*(APR.), 363–368. https://doi.org/10.1192/bjp.180.4.363

Nacasch, N., Huppert, J. D., Su, Y. J., Kivity, Y., Dinshtein, Y., Yeh, R., & Foa, E. B. (2015). Are 60-minute prolonged exposure sessions with 20-minute imaginal exposure to traumatic memories sufficient to successfully treat PTSD? A randomized noninferiority clinical trial. *Behavior Therapy*, *46*(3), 328–341. https://doi.org/10.1016/j.beth.2014.12.002

National Institute for Health and Care Excellence. (2018). *Post-traumatic stress disorder (NICE guideline NG116)*. NICE. https://www.nice.org.uk/guidance/ng116

Nixon, R. D. V., & Sloan, D. M. (2017). Treating PTSD: Innovations and Understanding Processes of Change. *Behavior Therapy*, *48*(2), 143–146. https://doi.org/10.1016/j.beth.2016.06.003

O'Donnell, M. L., Elliott, P., Wolfgang, B. J., & Creamer, M. (2007). Posttraumatic appraisals in the development and persistence of posttraumatic stress symptoms. *Journal of Traumatic Stress*, *20*(2), 173–182. https://doi.org/10.1002/jts.20198

Oh, W., Muzik, M., McGinnis, E. W., Hamilton, L., Menke, R. A., & Rosenblum, K. L. (2016). Comorbid trajectories of postpartum depression and PTSD among mothers with childhood trauma history: Course, predictors, processes and child adjustment. *Journal of Affective Disorders*, *200*, 133–141. https://doi.org/10.1016/J.JAD.2016.04.037

Ohayon, M. M., & Shapiro, C. M. (2000). Sleep disturbances and psychiatric disorders associated with posttraumatic stress disorder in the general population. *Comprehensive Psychiatry*, *41*(6), 469–478. https://doi.org/10.1053/comp.2000.16568

Pace-Schott, E. F., Seo, J., & Bottary, R. (2023). The influence of sleep on fear extinction in trauma-related disorders. *Neurobiology of Stress*, *22*, 100500. https://doi.org/10.1016/j.ynstr.2022.100500

Pace-Schott, E. F., Shepherd, E., Spencer, R. M. C., Marcello, M., Tucker, M., Propper, R. E., & Stickgold, R. (2011). Napping promotes inter-session habituation to emotional stimuli.

Neurobiology of Learning and Memory, *95*(1), 24–36. https://doi.org/10.1016/j.nlm.2010.10.006

Pace-Schott, E. F., Verga, P. W., Bennett, T. S., & Spencer, R. M. C. (2012). Sleep promotes consolidation and generalization of extinction learning in simulated exposure therapy for spider fear. *Journal of Psychiatric Research*, *46*(8), 1036–1044. https://doi.org/10.1016/j.jpsychires.2012.04.015

Pruiksma, K. E., Taylor, D. J., Wachen, J. S., Mintz, J., Young-McCaughan, S., Peterson, A. L., Yarvis, J. S., Borah, E. V., Dondanville, K. A., Litz, B. T., Hembree, E. A., & Resick, P. A. (2016). Residual sleep disturbances following PTSD treatment in active duty military personnel. *Psychological Trauma: Theory, Research, Practice, and Policy*, *8*(6), 697–701. https://doi.org/10.1037/tra0000150

Ramsawh, H. J., Bomyea, J., Stein, M. B., Cissell, S. H., & Lang, A. J. (2016). Sleep Quality Improvement During Cognitive Behavioral Therapy for Anxiety Disorders. *Behavioral Sleep Medicine*, *14*(3), 267–278. https://doi.org/10.1080/15402002.2014.981819

Rasmussen, A., Verkuilen, J., Jayawickreme, N., Wu, Z., & McCluskey, S. T. (2018). When Did Posttraumatic Stress Disorder Get So Many Factors? Confirmatory Factor Models Since DSM–5. *Https://Doi.Org/10.1177/2167702618809370*, *7*(2), 234–248. https://doi.org/10.1177/2167702618809370

Rauch, S. A. M., King, A. P., Abelson, J., Tuerk, P. W., Smith, E., Rothbaum, B. O., Clifton, E., Defever, A., & Liberzon, I. (2015). Biological and symptom changes in posttraumatic stress disorder treatment: A randomized clinical trial. *Depression and Anxiety*, *32*(3), 204–212. https://doi.org/10.1002/da.22331

Resick, P. A., Monson, C. M., & Chard, K. M. (2016). *Cognitive Processing Therapy for PTSD: A Comprehensive Manual*. Guilford Publications.

Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault

victims. *Journal of Consulting and Clinical Psychology*, *60*(5), 748–756. https://doi.org/10.1037/0022-006X.60.5.748

Richards, A., Inslicht, S. S., Yack, L. M., Metzler, T. J., Russell Huie, J., Straus, L. D., Dukes, C., Hubachek, S. Q., Felmingham, K. L., Mathalon, D. H., Woodward, S. H., & Neylan, T. C. (2022). The relationship of fear-potentiated startle and polysomnography-measured sleep in trauma-exposed men and women with and without PTSD: Testing REM sleep effects and exploring the roles of an integrative measure of sleep, PTSD symptoms, and biological sex. *Sleep*, *45*(1). https://doi.org/10.1093/sleep/zsab271

Richards, A., Kanady, J. C., & Neylan, T. C. (2020). Sleep disturbance in PTSD and other anxiety-related disorders: an updated review of clinical features, physiological characteristics, and psychological and neurobiological mechanisms. *Neuropsychopharmacology : Official Publication of the American College of Neuropsychopharmacology*, *45*(1), 55–73. https://doi.org/10.1038/S41386-019-0486-5

Richter, K., Baumgärtner, L., Niklewski, G., Peter, L., Köck, M., Kellner, S., Hillemacher, T., & Büttner-Teleaga, A. (2020). Sleep disorders in migrants and refugees: a systematic review with implications for personalized medical approach. *EPMA Journal 2020 11:2*, *11*(2), 251–260. https://doi.org/10.1007/S13167-020-00205-2

Roefs, A., Fried, E. I., Kindt, M., Martijn, C., Elzinga, B., Evers, A. W. M., Wiers, R. W., Borsboom, D., & Jansen, A. (2022). A new science of mental disorders: Using personalised, transdiagnostic, dynamical systems to understand, model, diagnose and treat psychopathology. *Behaviour Research and Therapy*, *153*, 104096. https://doi.org/10.1016/J.BRAT.2022.104096

Sandahl, H., Carlsson, J., Sonne, C., Mortensen, E. L., Jennum, P., & Baandrup, L. (2021). Investigating the link between subjective sleep quality, symptoms of PTSD, and level of functioning in a sample of trauma-affected refugees. *Sleep*, *44*(9).

https://doi.org/10.1093/sleep/zsab063

Sandahl, H., Vindbjerg, E., & Carlsson, J. (2017). Treatment of sleep disturbances in refugees suffering from post-traumatic stress disorder. *Transcultural Psychiatry*, *54*(5–6), 806–823. https://doi.org/10.1177/1363461517746314

Schäfer, I., Gast, U., Hofmann, A., Christine, K., Lampe, A., Liebermann, P., Lotzin, A., Maercker, A., Rosner, R., & Wöller, W. (2019). S3-Leitlinie Posttraumatische Belastungsstörung. In *S3-Leitlinie Posttraumatische Belastungsstörung*. Springer Berlin Heidelberg. https://doi.org/10.1007/978-3-662-59783-5

Schell, T. L., Marshall, G. N., & Jaycox, L. H. (2004). All symptoms are not created equal: The prominent role of hyperarousal in the natural course of posttraumatic psychological distress. *Journal of Abnormal Psychology*, *113*(2), 189–197. https://doi.org/10.1037/0021-843X.113.2.189

Schnurr, P. P., Lunney, C. A., Bovin, M. J., & Marx, B. P. (2009). Posttraumatic stress disorder and quality of life: extension of findings to veterans of the wars in Iraq and Afghanistan. *Clinical Psychology Review*, *29*(8), 727–735. https://doi.org/10.1016/J.CPR.2009.08.006

Schumm, J. A., Dickstein, B. D., Walter, K. H., Owens, G. P., & Chard, K. M. (2015). Changes in posttraumatic cognitions predict changes in posttraumatic stress disorder symptoms during cognitive processing therapy. *Journal of Consulting and Clinical Psychology*, *83*(6), 1161–1166. https://doi.org/10.1037/ccp0000040

Shapiro, F., & Trunk, C. (2022). *EMDR - Grundlagen und Praxis: Handbuch zur Behandlung traumatisierter Menschen* (3. Auflage). https://books.google.com/books/about/EMDR_Grundlagen_und_Praxis.html?hl=de&id=2gtYEAAAQBAJ

Short, N. A., Allan, N. P., & Schmidt, N. B. (2017). Sleep disturbance as a predictor of

affective functioning and symptom severity among individuals with PTSD: An ecological momentary assessment study. *Behaviour Research and Therapy*, *97*, 146–153. https://doi.org/10.1016/j.brat.2017.07.014

Short, N. A., Norr, A. M., Mathes, B. M., Oglesby, M. E., & Schmidt, N. B. (2016). An examination of the specific associations between facets of difficultieas in emotion regulation and posttraumatic stress symptom clusters. *Cognitive Therapy and Research*, *40*(6), 783–791. https://doi.org/10.1007/s10608-016-9787-8

Spoormaker, V. I., & Montgomery, P. (2008). Disturbed sleep in post-traumatic stress disorder: Secondary symptom or core feature? *Sleep Medicine Reviews*, *12*(3), 169–184. https://doi.org/10.1016/j.smrv.2007.08.008

Sripada, R. K., Rauch, S. A. M., & Liberzon, I. (2016). Psychological mechanisms of PTSD and its treatment. *Current Psychiatry Reports*, *18*(11). https://doi.org/10.1007/s11920-016-0735-9

Startup, M., Makgekgenene, L., & Webster, R. (2007). The role of self-blame for trauma as assessed by the Posttraumatic Cognitions Inventory (PTCI): A self-protective cognition? *Behaviour Research and Therapy*, *45*(2), 395–403. https://doi.org/10.1016/j.brat.2006.02.003

Steel, Z., Chey, T., Silove, D., Marnane, C., Bryant, R. A., & Van Ommeren, M. (2009). Association of torture and other potentially traumatic events with mental health outcomes among populations exposed to mass conflict and displacement: A systematic review and meta-analysis. *JAMA - Journal of the American Medical Association*, *302*(5), 537–549. https://doi.org/10.1001/jama.2009.1132

Steenkamp, M. M., Litz, B. T., Hoge, C. W., & Marmar, C. R. (2015). Psychotherapy for Military-Related PTSD: A Review of Randomized Clinical Trials. *JAMA*, *314*(5), 489–500. https://doi.org/10.1001/JAMA.2015.8370

References

Steil, R., & Ehlers, A. (2000). Dysfunctional meaning of posttraumatic intrusions in chronic PTSD. *Behaviour Research and Therapy*, *38*(6), 537–558. https://doi.org/10.1016/S0005-7967(99)00069-8

Szabo, Y. Z., Warnecke, A. J., Newton, T. L., & Valentine, J. C. (2017). Rumination and posttraumatic stress symptoms in trauma-exposed adults: A systematic review and meta-analysis. *Anxiety, Stress and Coping*, *30*(4), 396–414. https://doi.org/10.1080/10615806.2017.1313835

Takano, K., Iijima, Y., & Tanno, Y. (2012). Repetitive thought and self-reported sleep disturbance. *Behavior Therapy*, *43*(4), 779–789. https://doi.org/10.1016/j.beth.2012.04.002

Taylor, D. J., Pruiksma, K. E., Hale, W., McLean, C. P., Zandberg, L. J., Brown, L., Mintz, J., Stacey Young-McCaughan, Peterson, A. L., Yarvis, J. S., Dondanville, K. A., Litz, B. T., Roache, J., & Foa, E. B. (2020). Sleep problems in active duty military personnel seeking treatment for posttraumatic stress disorder: Presence, change, and impact on outcomes. *Sleep*, *43*(10). https://doi.org/10.1093/sleep/zsaa065

ter Heide, F. J. J., Sleijpen, M., & van der Aa, N. (2017). Posttraumatic world assumptions among treatment-seeking refugees. *Transcultural Psychiatry*, *54*(5–6), 824–839. https://doi.org/10.1177/1363461517741811

UNHCR - United Nations High Commissioner for Refugees. (2021). *Global Trends Report 2021*. https://www.unhcr.org/62a9d1494/global-trends-report-2021

Valdez, C. E., & Lilly, M. M. (2017). Posttraumatic rumination: Content, correlates, and processes. *Journal of Clinical Psychology*, *73*(6), 707–721. https://doi.org/10.1002/jclp.22371

Van Der Helm, E., Yao, J., Dutt, S., Rao, V., Saletin, J. M., & Walker, M. P. (2011). REM sleep depotentiates amygdala activity to previous emotional experiences. *Current*

Biology, *21*(23), 2029–2032. https://doi.org/10.1016/j.cub.2011.10.052

Walker, M. P. (2009). The role of sleep in cognition and emotion. *Annals of the New York Academy of Sciences*, *1156*, 168–197. https://doi.org/10.1111/j.1749-6632.2009.04416.x

Walker, M. P., & van der Helm, E. (2009). Overnight Therapy? The Role of Sleep in Emotional Brain Processing. *Psychological Bulletin*, *135*(5), 731–748. https://doi.org/10.1037/a0016570

Walton, J. L., Raines, A. M., Cuccurullo, L. A. J., Vidaurri, D. N., Villarosa-Hurlocker, M. C., & Franklin, C. L. (2018). The relationship between DSM-5 PTSD symptom clusters and alcohol misuse among military veterans. *American Journal on Addictions*, *27*(1), 23–28. https://doi.org/10.1111/ajad.12658

Wang, L., Zhang, L., Armour, C., Cao, C., Qing, Y., Zhang, J., Liu, P., Zhang, B., Wu, Q., Zhao, Z., & Fan, G. (2015). Assessing the underlying dimensionality of DSM-5 PTSD symptoms in Chinese adolescents surviving the 2008 Wenchuan earthquake. *Journal of Anxiety Disorders*, *31*, 90–97. https://doi.org/10.1016/j.janxdis.2015.02.006

Watts, B. V., Schnurr, P. P., Mayo, L., Young-Xu, Y., Weeks, W. B., & Friedman, M. J. (2013). Meta-analysis of the efficacy of treatments for posttraumatic stress disorder. *Journal of Clinical Psychiatry*, *74*(6). https://doi.org/10.4088/JCP.12r08225

Weber, M., Schumacher, S., Hannig, W., Barth, J., Lotzin, A., Schäfer, I., Ehring, T., & Kleim, B. (2021). Long-term outcomes of psychological treatment for posttraumatic stress disorder: A systematic review and meta-analysis. *Psychological Medicine*, *51*(9), 1420–1430. https://doi.org/10.1017/S003329172100163X

Wiedemann, M., Stott, R., Nickless, A., Beierl, E. T., Wild, J., Warnock-Parkes, E., Grey, N., Clark, D. M., & Ehlers, A. (2020). Cognitive processes associated with sudden gains in cognitive therapy for posttraumatic stress disorder in routine care. *Journal of Consulting and Clinical Psychology*, *88*(5), 455–469. https://doi.org/10.1037/ccp0000488

References

Wild, J., Warnock-Parkes, E., Grey, N., Stott, R., Wiedemann, M., Canvin, L., Rankin, H., Shepherd, E., Forkert, A., Clark, D. M., & Ehlers, A. (2016). Internet-delivered cognitive therapy for PTSD: A development pilot series. *European Journal of Psychotraumatology*, *7*(1), 31019. https://doi.org/10.3402/ejpt.v7.31019

Wisco, B. E., Sloan, D. M., & Marx, B. P. (2013). Cognitive emotion regulation and written exposure therapy for posttraumatic stress disorder. *Clinical Psychological Science*, *1*(4), 435–442. https://doi.org/10.1177/2167702613486630

World Health Organization. (2019). *International Statistical Classification of Diseases and Related Health Problems (11th ed.)*. https://doi.org/https://icd.who.int/

Wright, K. M., Britt, T. W., Bliese, P. D., Adler, A. B., Picchioni, D., & Moore, D. (2011). Insomnia as predictor versus outcome of PTSD and depression among Iraq combat veterans. *Journal of Clinical Psychology*, *67*(12), 1240–1258. https://doi.org/10.1002/JCLP.20845

Zalta, A. K. (2015). Psychological mechanisms of effective cognitive–behavioral treatments for PTSD. *Current Psychiatry Reports*, *17*(4). https://doi.org/10.1007/s11920-015-0560-6

Zalta, A. K., Gillihan, S. J., Fisher, A. J., Mintz, J., Mclean, C. P., Yehuda, R., & Foa, E. B. (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology*, *82*(1), 171–175. https://doi.org/10.1037/a0034735

Zalta, A. K., Pinkerton, L. M., Valdespino-Hayden, Z., Smith, D. L., Burgess, H. J., Held, P., Boley, R. A., Karnik, N. S., & Pollack, M. H. (2020). Examining Insomnia During Intensive Treatment for Veterans with Posttraumatic Stress Disorder: Does it Improve and Does it Predict Treatment Outcomes? *Journal of Traumatic Stress*, *33*(4), 521–527. https://doi.org/10.1002/jts.22505

Zeng, S., Lau, E. Y. Y., Li, S. X., & Hu, X. (2021). Sleep differentially impacts involuntary intrusions and voluntary recognitions of lab-analogue traumatic memories. *Journal of Sleep Research*, *30*(3), e13208. https://doi.org/10.1111/JSR.13208

Zoccola, P. M., Dickerson, S. S., & Lam, S. (2009). Rumination predicts longer sleep onset latency after an acute psychosocial stressor. *Psychosomatic Medicine*, *71*(7), 771–775. https://doi.org/10.1097/PSY.0b013e3181ae58e8

List of Figures

Study II

List of Tables

General Introduction

Study I

Study II

Study III