

Cognitive Change Predicts Symptom Reduction With Cognitive Therapy for Posttraumatic Stress Disorder

Birgit Kleim

King's College London and University of Zurich

Nick Grey

NIHR Biomedical Research Centre for Mental Health, South Maudsley NHS Foundation Trust and King's College London

Jennifer Wild

University of Oxford and King's College London

Fridtjof W. Nussbeck

University of Zurich

Richard Stott

King's College London

Ann Hackmann

University of Oxford

David M. Clark and Anke Ehlers

University of Oxford and King's College London

Objective: There is a growing body of evidence for the effectiveness of trauma-focused cognitive behavior therapy (TF-CBT) for posttraumatic stress disorder (PTSD), but few studies to date have investigated the mechanisms by which TF-CBT leads to therapeutic change. Models of PTSD suggest that a core treatment mechanism is the change in dysfunctional appraisals of the trauma and its aftermath. If this is the case, then changes in appraisals should predict a change in symptoms. The present study investigated whether cognitive change precedes symptom change in Cognitive Therapy for PTSD, a version of TF-CBT. **Method:** The study analyzed weekly cognitive and symptom measures from 268 PTSD patients who received a course of Cognitive Therapy for PTSD, using bivariate latent growth modeling. **Results:** Results showed that (a) dysfunctional trauma-related appraisals and PTSD symptoms both decreased significantly over the course of treatment, (b) changes in appraisals and symptoms were correlated, and (c) weekly change in appraisals significantly predicted subsequent reduction in symptom scores (both corrected for the general decrease over the course of therapy). Changes in PTSD symptom severity did not predict subsequent changes in appraisals. **Conclusions:** The study provided preliminary evidence for the temporal precedence of a reduction in negative trauma-related appraisals in symptom reduction during trauma-focused CBT for PTSD. This supports the role of change in appraisals as an active therapeutic mechanism.

Keywords: PTSD, cognition, appraisals, cognitive therapy, mediation

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Birgit Kleim, Department of Psychology, King's College London, London, England, and Department of Psychology, University of Zurich, Zurich, Switzerland; Nick Grey, NIHR Biomedical Research Centre for Mental Health, South London and Maudsley NHS Trust and King's College London; Jennifer Wild, David M. Clark, and Anke Ehlers, Department of Experimental Psychology, University of Oxford, Oxford, and Department of Psychology, Institute of Psychiatry, King's College London; Fridtjof W. Nussbeck, Department of Psychology, University of Zurich; Richard Stott, Department of Psychology, King's College London; Ann Hackmann, Department of Psychiatry, University of Oxford and Department of Psychology, Institute of Psychiatry, King's College London; Fridtjof W. Nussbeck is now at the Department of Psychology, University of Bielefeld, Bielefeld, Germany.

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Correspondence concerning this article should be addressed to Anke Ehlers, Department of Experimental Psychology, University of Oxford, South Parks Road, Oxford OX1 3UD, United Kingdom or to Birgit Kleim, Department of Psychology, University of Zurich, Clinical Psychology and Psychotherapy, Binzmuhlestrasse 14/Box 26, CH-8050 Zurich, Switzerland. E-mail: anke.ehlers@psy.ox.ac.uk or b.kleim@psychologie.uzh.ch

There is a growing body of evidence for the efficacy and effectiveness of trauma-focused cognitive behavioral therapy (CBT) in the treatment of posttraumatic stress disorder (PTSD; e.g., Bisson et al., 2007; Stewart & Chambless, 2009). Trauma-focused CBT programs have been shown to lead to clinically significant improvement in PTSD (Bisson et al., 2007; Bradley, Greene, Russ, Dutra, & Watson, 2005) and are currently recommended as first line treatments for this condition (American Psychiatric Association, 2004; Australian Centre for Posttraumatic Mental Health, 2007; Foa, Keane, Friedman, & Cohen, 2005; National Institute for Health and Clinical Excellence, 2005; Stein et al., 2009; Department of Veterans Affairs and Department of Defense, 2010). Examples include Prolonged Exposure (Foa, Hembree, et al., 2005; Foa & Rothbaum, 1998; Foa, Rothbaum, Riggs, & Murdock, 1991), Cognitive Processing Therapy (Resick, Nishith, Weaver, Astin, & Feuer, 2002; Resick & Schnicke, 1992, 1993), and Cognitive Therapy for PTSD (CT-PTSD; Ehlers & Clark, 2000; Ehlers, Clark, Hackmann, McManus, & Fennell, 2005; Ehlers et al., 2003; Ehlers, Hackmann, et al., 2012).

Effect sizes (ES) for the reduction in PTSD with CBT in randomized controlled trials are very large (mean ES = 1.43; Bradley et al., 2005). On average, 67% of patients who complete trauma-focused treatments (and 56% of those who enter the treatment initially, based on intent to treat analyses) no longer meet diagnostic criteria for PTSD (Bradley et al., 2005). There is also evidence that these treatment packages can be successfully disseminated and produce similar changes in routine clinical settings (Cohen & Mannarino, 2008; Duffy, Gillespie, & Clark, 2007; Ehlers, Grey, et al., 2012; Foa, Hembree, et al., 2005; Gillespie, Duffy, Hackmann, & Clark, 2002).

Thus, there is compelling evidence that trauma-focused CBT leads to clinically significant change in PTSD symptoms, in secondary social and behavioral problems, as well as in overall quality of life (e.g., Schnurr et al., 2007). However, few studies to date have investigated the mechanisms by which CBT leads to therapeutic change. Such investigations are needed for several reasons. First, as Kazdin (2007) pointed out, studying the mechanisms of how a treatment works is a good investment for improving clinical practice and patient care. Uncovering change processes that account for a treatment's effects (or for considerable parts of it) can facilitate the refinement of existing therapy procedures to make the therapy more efficacious or efficient. Second, delineating key mechanisms of action may aid the individualization of treatment by helping clarify aspects that can profitably be tailored to the patient in an individualized way. Finally, establishing an empirical base for active change mechanisms can help ensure that critical aspects of a therapy are disseminated appropriately.

Among potential candidates for mechanisms of effective psychological treatments of PTSD, dysfunctional cognitions play a prominent role. Cognitive models of psychopathology postulate that cognitive change is central to treating psychological disorder, and that "all therapies work by altering dysfunctional cognitions, either directly or indirectly" (D. A. Clark, 1995, p. 158). In PTSD, several information processing theories highlight excessively threatening meanings (dysfunctional appraisals) of traumas that threaten the individual's view of the self and the world as core factors in the development and maintenance of the disorder and in treatment (e.g., Dalgleish, 2004; Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1993). The appraisals may be about what happened during the trauma, or about

the aftermath of the trauma, and include threatening appraisals about the self (such as "I am inadequate") or about other people or the world in general (such as "Other people are not what they seem"; "The world is a dangerous place") (Ehlers & Clark, 2000; Dunmore, Clark, & Ehlers, 2001). Consistent with a role of such problematic appraisals in therapeutic change, several studies have observed that treatment changes both dysfunctional appraisals and PTSD symptoms (e.g., Ehlers et al., 2005; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999; Hage-naars, van Moinnen, & de Rooij, 2010; Owens, Pike, & Chard, 2001; Vogt, Shepherd, & Resick, 2012). However, it remains to be demonstrated that change in cognitions mediates symptom change.

Researchers have started to use the framework of mediation analysis for studying mechanisms of therapeutic change in anxiety disorders, building on the work of Baron and Kenny (1986), Kraemer and colleagues (Kraemer, Wilson, Fairburn, & Agras, 2002; Kraemer & Kupfer, 2002), and others. Initially, most of these studies assessed mediators and treatment outcome at the same time points, mostly at the end of treatment (e.g., DeRubeis et al., 1990; Hofmann, 2004; Vögele, Ehlers, Meyer, Frank, Hahlweg, & Margraf, 2010; but see Stice, Rohde, Seeley, & Gau, 2010). As a consequence, they could not directly test an important requirement for a variable to be a mediator of therapeutic effects, namely, that it must change in the direction of improvement before the change in the symptoms occurs.

Some studies on CBT for other anxiety disorders, such as social phobia or panic disorder, have begun to directly test the temporal precedence of cognitive change over change in outcome (Hoffart, Borge, Sexton, & Clark, 2009; Hofmann, 2004; Hofmann et al., 2007; Teachman, Marker, & Smith-Janik, 2008) and found, for instance, that treatment changes negative cognitive appraisals, such as estimated social costs, which in turn then produce the observable changes in symptoms. For PTSD, it remains to be investigated whether changes in dysfunctional trauma-related cognitions precede PTSD symptom reduction during trauma-focused CBT.

The purpose of the present study was to investigate changes in dysfunctional appraisals as a possible mechanism of change in CT-PTSD. CT-PTSD is based on Ehlers and Clark's (2000) model of PTSD. This model suggests that people with PTSD perceive a serious current threat that has two sources: (a) excessively negative appraisals of the trauma and/or its sequelae and (b) characteristics of trauma memories that lead to reexperiencing symptoms. The problem is maintained by cognitive strategies and behaviors (such as thought suppression, rumination, safety-seeking behaviors) that are intended to reduce the sense of current threat, but maintain the problem by preventing change in the appraisals or trauma memory, and/or by increasing symptoms. CT-PTSD targets the three factors specified in the model (see D. M. Clark & Ehlers, 2004; Ehlers et al., 2010; and Ehlers et al., 2005, for details). For each patient, an individualized version of the model is developed, by identifying the relevant appraisals, memory characteristics and triggers, and behavioral and cognitive strategies that maintain his/her PTSD. These maintaining factors are addressed with the procedures described in Ehlers et al. (2010). This treatment, like other forms of TF-CBT such as Cognitive Processing Therapy (Resick & Schnicke, 1992, 1993), thus directly aims to change maladaptive appraisals as a primary treatment goal and therefore appears to be suitable for studying the temporal relationship between cognitive and symptom change.

In this study, temporal patterns of change in dysfunctional appraisals and PTSD symptom reduction were analyzed using a novel approach, bivariate latent growth modeling (see King et al., 2006; McArdle & Nesselroade, 2002; Teachman et al., 2008). This approach allowed us to simultaneously model latent changes in trauma-related negative appraisals and PTSD symptoms over the course of treatment. In line with the theoretical assumption of information processing theories that CT-PTSD works by modifying negative trauma-related cognitions, we hypothesized that: (Hypothesis i) dysfunctional trauma-related appraisals would decrease during the course of CT, as would PTSD symptoms; (Hypothesis ii) change in appraisals and symptoms would be significantly associated with each other; (Hypothesis iii) change in appraisals would predict subsequent symptom reduction in the next session (both corrected for the general decrease over time). We thus reasoned that a greater change than expected due to the general change process in appraisals between session t (time) and session $t + 1$ would predict a greater change in symptoms in the following week ($t + 2$), and thus mediate the therapeutic effects. To further test the causal relationship between change in appraisals and change in symptoms, we also tested the reverse relationship, namely to what extent symptom change predicts change in appraisals (reverse mediation), which would *not* be expected if change in appraisal mediates change in symptoms.

Method

Participants

Patients were drawn from an effectiveness study of consecutive referrals to a United Kingdom National Health Service outpatient clinic between April 2001 and August 2008 (Ehlers, Grey et al., 2012). The trauma clinic offers treatment for survivors of trauma in adulthood who suffer from PTSD and receives referrals from general practitioners and community mental health Teams. The study was approved by the local ethics committee. All patients underwent an initial clinical assessment conducted by a trained clinician including the Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*; SCID; First, Spitzer, Gibbon, Williams, & Benjamin, 1994) for assessing Axis I and II diagnoses. Patients who needed treatment for current substance dependence, psychosis, or immediate suicide risk or who could not conduct the therapy in English were seen by other services within the same National Health Service Trust. Patients on psychotropic medication were asked to stay on a stable dose for at least 2 months before treatment commenced and were asked to remain on this dose for the duration of treatment. Liaison with the patient's family doctor or psychiatrist ensured that medication intake remained stable or was appropriately reduced after the course of treatment.

Of 577 patients who completed the assessment during the study period (see Ehlers, Grey et al., 2012, for details), 408 were suitable for trauma-focused treatment. The main reasons for not being suitable were that the patient did not have PTSD ($n = 42$) or first needed treatment for another primary problem such as alcohol dependence or immediate suicide risk ($n = 73$). The main reasons for not being offered CT-PTSD despite suitability for treatment ($n = 78$) were that the patient did not want treatment or participated in a trial where they received another psychological treatment. A total of 330 patients were offered a course of CT-PTSD.

To be included in the present study, patients needed to have attended at least five sessions and completed the weekly questionnaires used in this study, as a sufficient number of measurement points was needed to assess the pattern of cognitive and symptom change. The final sample size was 268, as 29 patients attended fewer than five sessions, and 33 had incomplete questionnaires. Demographic and clinical sample and trauma characteristics are shown in Table 1. The sample was ethnically mixed and had suffered predominantly interpersonal trauma (including physical assault, sexual assault, terrorist attack, and torture, 55.6%). As expected with the recruitment through a routine clinical service, the majority of patients had comorbid Axis I and Axis II disorders, mainly mood (48.9%; in particular, major depression), anxiety disorders (43.7%; in particular, panic disorder with and without agoraphobia), or substance abuse (21.3%).

Treatment Protocol

Patients received a course of Cognitive Therapy for PTSD (CT-PTSD, Ehlers et al., 2005, 2003), a trauma-focused CBT program, which has shown to be effective in five randomized controlled trials (Duffy, Gillespie, & Clark, 2007; Ehlers et al., 2005, 2003; Ehlers, Hackmann, et al., 2012; Smith et al., 2007) and several effectiveness studies (Brewin et al., 2010; Ehlers, Grey, et al., 2012; Gillespie, Duffy, Hackmann, & Clark, 2002). For patients who need treatment for one or two traumas (they may have had further traumas that do not require treatment), treatment is delivered in up to 12 weekly sessions plus up to three monthly booster sessions, with a mean of 10 weekly sessions in previous trials (Ehlers et al., 2005, 2003). Treatment duration could be extended if patients needed treatment for more than two traumas or for other disorders in their own right. The relative weight given to different treatment procedures differs from patient to patient, depending on the case formulation (see <http://oxcadat.psy.ox.ac.uk/downloads> for details). The first session usually comprised establishing treatment goals, education about PTSD symptoms, identification of main intrusive memories and maintaining factors (appraisals, cognitive strategies such as thought suppression, rumination, hypervigilance, safety behaviors), initial shared case formulation (which is revised throughout treatment), introduction of reclaiming your life assignments, a thought suppression experiment with homework, and the rationale for trauma memory work. Reclaiming your life assignments and work on dropping further maintaining behaviors are continued throughout treatment and are usually addressed in part of each session. The following three to five sessions are usually devoted to the updating your trauma memory procedure (depending on the duration of the traumas and the number of hot spots to be updated), followed by stimulus discrimination and a site visit, which is usually completed around Session 9. Work on changing problematic appraisals is closely integrated with the memory work and is included in every session.

Patients received an average of $M(SD) = 11.56(3.87)$ sessions, range 5–48 sessions of CT during a mean period of 108 days (range 40 days to 16 months). Only 14 patients (5.2%) received more than 18 sessions. Session duration was on average $M(SD) = 88(15)$ min. For the purpose of the present analysis, we consider data from the patients' first 10 sessions, as this ensured that the majority of patients contributed to each data point (only 26.9% of the sample had fewer than 10 sessions).

Table 1
Demographic and Clinical Sample Characteristics of PTSD Patients (N = 268)

Variable	N	%	M	SD
Sex				
Male	111	41.4		
Female	157	58.6		
Ethnicity				
Caucasian	157	58.6		
Black	65	24.3		
Other or mixed race	46	17.2		
Marital status				
Married/cohabitating	99	36.9		
Divorced/separated/widowed	48	17.9		
Never married/single	112	41.8		
No information	9	3.4		
Main trauma				
Interpersonal violence	149	55.6		
Accident/disaster	63	23.5		
Witnessed harm to others	27	10.1		
Other	29	10.8		
Level of education				
None	43	16.0		
GCSE (age 16) ^a	106	39.6		
A level (age 18) ^b	40	14.9		
University	62	23.1		
No information	17	6.3		
Current employment				
Employed/self-employed	114	42.5		
Sick leave	33	12.3		
Disability/retired	24	9.0		
Unemployed	82	30.6		
No information	15	5.6		
Comorbid Axis 1 disorder				
Present	198	73.9		
Absent	70	26.1		
Comorbid Axis 2 disorder				
Present	81	30.2		
Absent	187	69.8		
Age				
	In years		38.67	11.26
Time since main trauma				
	In months		36.95	57.72
PTSD symptom severity				
	PDS total score pretreatment		33.69	8.86
Depression				
	BDI total score pretreatment		26.15	11.60

Note. PTSD = posttraumatic stress disorder; PDS = Posttraumatic Diagnostic Scale (range: 0–51); BDI = Beck Depression Inventory (range: 0–63); GCSE = general certificate of secondary education; A level = advanced level.

^a Equivalent to middle school degree. ^b Equivalent to high school degree.

Therapists

Therapists were clinical psychologists, psychiatrists, or nurse therapists with varying levels of prior experience with CBT who had received training in CT-PTSD. The majority of patients were seen by a staff therapist who had completed professional training in their discipline including a general course in CBT. Trainee therapists received close individual supervision by an experienced therapist. All cases were discussed in weekly group supervision meetings to ensure adherence to the treatment protocol. Therapists kept detailed notes about each treatment session, and an independent rater rated the extent to which the session focused on the PTSD treatment model, on a scale from 1 to 3 (1 = *mainly followed trauma-focused protocol*, 2 = *equal focus on trauma-focused protocol and other issues*, 3 = *main focus on other problem*). The mean rating for all sessions was $M = 1.37$, $SD = 0.40$, and 90.1% of the patients mainly focused on the PTSD

treatment protocol in their course of treatment (mean rating of below 2). The most common other problems addressed in the sessions were comorbid disorders and other stressors such as social problems (e.g., financial, housing, legal issues) or physical health problems.

Questionnaires

Patients completed the following questionnaires at initial assessment and every treatment session.

Posttraumatic Diagnostic Scale (PDS). PTSD symptom severity with the Posttraumatic Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997), a standardized self-report measure of PTSD symptom severity with high internal consistency and test-retest reliability and good agreement with interview measures of PTSD that has been widely used with clinical and nonclinical samples of traumatized individuals. The PDS asks participants to

rate 17 items regarding how much they were bothered by each of the PTSD symptoms specified in *DSM-IV* ranging from 0 (*never*) to 3 (*5 times per week or more/very severely*). Internal consistency in this sample was $\alpha = .85$ (initial assessment).

Posttraumatic Cognition Inventory (PTCI)—Short Version. The Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999) is a self-report instrument with good internal consistency and concurrent and discriminant validity (J. G. Beck et al., 2004; Foa et al., 1999) that measures thoughts that are characteristic of people with PTSD. It assesses trauma-related negative appraisals about the self, including negative interpretations of PTSD symptoms, about others, and self-blame (“It happened to me because of the way I acted.”) Participants are asked to rate each item on a scale from 1 (*totally disagree*) to 7 (*totally agree*). The PTCI has been shown to be reliable and to distinguish between trauma survivors with and without PTSD (Foa et al., 1999). A shortened version of 22 items was used for this study. It contains 15 of the original PTCI items that showed the highest loading on the Thoughts About Self (12 items, e.g., “I am inadequate”; “I will never be able to feel normal emotions again”; “My reactions since the event mean I am going crazy”) and Thoughts About Others factors (three items, e.g., “I have to be especially careful because you can never know what will happen next,” “I have to be on guard all the time”) and seven further items regarding excessive self-blame (e.g., “If people know what happened they would look down on me”), preoccupation with unfairness (e.g., “I will never be able to forgive the people who did this to me”), and negative appraisals of changes in the body (“I cannot accept the way my body has changed since the trauma”) from an item pool generated for a revision of the PTCI. A mean score was calculated for each session ($\alpha = .94$; initial assessment).

Beck Depression Inventory (BDI). Severity of depression at initial assessment was assessed with the Beck Depression Inventory (BDI; A. T. Beck & Steer, 1987), a widely used and validated standardized questionnaire measure of depression. Respondents are asked to decide between four different response choices reflecting different degrees of symptom severity. Items are then scored from 0 to 3, with the sum of the item scores representing the total BDI score. Internal consistency in this sample was $\alpha = .93$ (initial assessment).

Analytic Strategy

Model specification. We adopted a three-step approach recommended in the literature to estimate bivariate latent growth models (e.g., McArdle & Hamagami, 2001; McArdle & Nesselroade, 2002). In the first step, we modeled latent change processes from session to session in cognitions and symptoms separately, entailing a test of the best representation of latent trajectories, including linear, log linear, and quadratic trajectories. Timing of the first set of questionnaires varied somewhat between patients, hence resulting in a more variable time period between the initial score (t1) and second session (t2) measurement point than between subsequent assessments. Therefore, we included an additional variable (“step”) in the statistical model. The intercept captures patients’ initial scores. The step variable captures the difference between t1 and t2. The latent change as a trend is modeled starting with t2 (Session 2). First, we estimated growth models with unrestricted trajectories. Based on this model, we identified the simplest latent growth model describing this course over time and fit the model to our data (Step 1). Means of latent variables

indicating change over time (i.e., slopes) were expected to be negative, indicating a decrease in negative cognitions and symptoms over the course of therapy. In Step 2, we calculated a dual linear growth curve model. In this model, we simultaneously estimated latent growth curves for cognitions and symptoms, as well as their association. We hypothesized that individuals presenting to therapy with more negative cognitions also show higher symptom scores (correlation of intercepts). We also hypothesized that decline in negative cognitions would be associated with decline in symptoms (correlation of slopes). In a final step (Step 3), we focused on temporal precedence and correlated residuals of negative cognition scores at each time point with the subsequent residual symptom scores and vice-versa. This allowed us to determine whether a larger than average change on one variable is associated with a larger than average change on the other variable at a later time point. Residuals capture parts of scores that cannot be predicted by the general trajectory, i.e., an individual showing greater decline in appraisals at a given time-point than expected by the overall sample trajectory. If greater decrease in negative appraisals is associated with greater symptom reduction in the next session, it can be assumed that changes on the first variable cause changes on the latter. We expected to find significant associations between negative cognition residuals and subsequent symptom residuals, but not vice versa.

Model identification, restrictions, and estimation. Model restrictions were imposed in order to identify model parameters and to simplify model interpretation: residual variances within constructs, as well as covariances of subsequent residuals within constructs across time (“autocovariances”), were set equal to each other. Similarly, covariances of residuals and subsequent residuals of the other construct’s indicators (“cross-lag covariances”) were set equal to each other across time. All analyses were conducted using Mplus 6.0 (Muthén & Muthén, 2010) applying the robust maximum likelihood estimator (MLR), as MLR allows for cases with missing data. The minimal data coverage was .56 for variances (PTCI at session 10) and .48 for covariances (PTCI at Sessions 7 and 10); the mean data coverage (variances and covariances) was .67.

Results

Change in Cognitions and PTSD Symptoms Over Time

Mean scores for negative appraisals (PTCI) and PTSD symptom (PDS) scores for measurement occasions t1 (initial scores) to t10 (Session 10) were used in the models (and will be referred to in the following as PTCI1 to PTCI10 and PDS1 to PDS10). The mean score for negative trauma-related cognitions (PTCI) dropped from $M (SD) = 4.29 (1.57)$ at t1 (initial score) to $M (SD) = 3.38 (2.36)$ at t10 (session 10). The mean PDS item score dropped from $M (SD) = 2.08 (0.23)$ at t1 to $M (SD) = 1.07 (0.72)$ ¹ at t10. This is equivalent to a drop from 35.36 to 18.19 on the PDS sum score.

Statistical models were estimated for the two separate trajectories over time. For both, cognitions and symptoms, a decrease from

¹ Software for structural equation models sometimes provides aberrant results if sum scores with large variances are entered instead of mean scores. Replacing the sum score by the mean score does not alter correlation or standardized regression coefficients.

t1 to t2 (step variable) was identified that was different in height from, and did not correlate with, the following linear decline over time (t2 to t10). We thus fitted a model that included the identified step from t1 to t2, followed by a linear decline from t2 (session 2) onwards. The models fitted adequately to the data (PTCI: $\chi^2 = 107.65$, $df = 55$; $p < .001$, comparative fit index (CFI) = .97; root-mean-square error of approximation [RMSEA] = .06; PDS: $\chi^2 = 135.03$, $df = 55$; $p < .001$, CFI = .96; RMSEA = .07). A comparison of the fit statistics for the latent trajectories suggested that the linear trajectory entailed the best model fit (PTCI model: AIC = 3,698.28, BIC = 3,791.55, sample-size adjusted BIC = 3,709.11, PDS model: Akaike information criterion [AIC] = 2,291.67, Bayesian information criterion [BIC] = 2,385.22, sample-size adjusted BIC = 2,302.79), better than the models entailing quadratic, log linear, or exponential trajectories (for example, quadratic PTCI model: 3,784.47, BIC = 3,877.74, sample-size adjusted BIC = 3,795.31, quadratic PDS model: AIC = 2,401.41, BIC = 2,494.97, sample-size adjusted BIC = 2,412.54).

Table 2 presents estimates of the two growth models, including initial scores (intercepts) for trauma-related negative appraisals (intercept PTCI = 4.29), and PTSD symptoms (intercept PDS = 2.08). The step variables for both negative appraisals and symptoms show that there was a considerable decline from t1 to t2. In line with Hypothesis i, the slope variables showed a significant linear decline over time from t2 to t10, symptom slope (slope PDS = $-.07$), and negative appraisals slope (slope PTCI = $-.08$, all mean values are significant at $p < .001$). Note that the absolute decrease is larger for the PDS as its scale is smaller in range (0 to 3 compared to 1 to 7 for the PTCI).

Intercept (i.e., initial score) and slope (decrease from session to session) did not correlate with each other on either variable (PDS: $r = -.07$; $p = .502$, PTCI: $r = -.05$; $p = .523$), indicating that PTSD symptoms/negative appraisals did not decrease more rapidly or slowly in patients with more severe symptoms/negative negative appraisals.

Association Between Negative Appraisals and Symptom Change Over Time

In the second step, we combined the two latent growth models allowing for correlations between all latent variables. The model fit indicated acceptable fit, $\chi^2 = 349.47$, $df = 200$, $p < .001$ ($\chi^2/df = 1.75$), CFI = .97, RMSEA = .05, square-root-mean residual [SRMR] = .04. As expected, the intercepts of negative appraisals and symptoms were correlated ($p < .001$), indicating that patients with more severe PTSD symp-

toms also endorsed more negative appraisals. In line with Hypothesis ii, negative appraisal and symptom slopes were correlated ($p < .001$), a reduction in negative appraisals was associated with a reduction in PTSD symptoms over the course of therapy. The results are depicted in Table 3.

Predictive Relationships Between Negative Appraisal and Symptom Change Processes

The final model (see Figure 1) extends the model reported in Step 2 by adding autoregressive effects (i.e., prediction of scores from scores at previous session) and regressions between adjacent residuals (i.e., from the same session) of negative appraisal and symptom variables. This model fit the data well ($\chi^2 = 255.88$, $df = 196$, $p = .003$; $\chi^2/df = 1.30$; CFI = .99, RMSEA = .03, SRMR = .03). Table 2 presents correlations between the latent variables. Individuals with more severe initial PTSD symptoms showed greater negative appraisal scores ($r_{\text{intercept PTCI, intercept PDS}} = .57$, $p < .001$). Patients with greater initial PTCI scores showed a steeper decline in negative appraisals from Session 1 to Session 2 ($r_{\text{intercept PTCI, step PDS}} = -.65$, $p < .001$), and those who showed steeper declines in PTCI also showed steeper declines in PTSD symptoms ($r_{\text{slope PTCI, slope PDS}} = .78$, $p < .001$).

Table 2 presents autocorrelations of residuals, that is, scores of one variable predicting the score of the same variable at the next time point. Negative appraisal and symptom residuals at the same measurement occasion were correlated (e.g., $r_{\text{PDS1,PTCI1}} = .44$, $p < .001$). That is, patients who showed lower scores than predicted by the latent trajectory at a particular occasion of measurement on the PTCI also showed lower scores on PDS at the same occasion of measurement.

Cross-lagged regressions of residual scores were calculated to test the hypothesis regarding temporal precedence. Consistent with Hypothesis iii, residual PTCI scores were significant predictors of later time-lagged residual PDS scores (e.g., $\beta_{\text{PDS3,PTCI2}} = .12$; $p = .014$). Individuals with less negative appraisals had less severe PTSD symptoms at the following session, after controlling for both the general decrease in negative appraisals and symptoms over time and for the autopredictive effect of symptoms at the preceding time point. This association remained significant when we excluded symptoms of emotional numbing and avoidance from the PDS score, that is, those with more change in negative appraisals had less severe PTSD symptoms at the following session ($\beta_s = .09-.14$, $p < .006$).

Table 2

Variances and Correlations of Latent Variables in the Model Combining PDS and PTCI

	Intercept PTCI	Step PTCI	Slope PTCI	Intercept PDS	Step PDS	Slope PDS
Intercept PTCI	1.42					
Step PTCI	-0.65	.32				
Slope PTCI	-0.05	.01	.01			
Intercept PDS	0.57	-.08	.001	.15		
Step PDS	0.13	.11	.16	.03	.07	
Slope PDS	0.07	-.02	.78	-.07	.02	.002

Note. Variances are displayed in the diagonal. All variances are significant. Significant correlation coefficients are printed in bold type. PDS = Posttraumatic Diagnostic Scale; PTCI = Posttraumatic Cognitions Inventory.

Table 3
 Mean Values of Latent Variables, Autoregressive, and Crossover Regressions of Residual Scores in the Final Model (Step 3):
 Unstandardized and Standardized Model Characteristics

	Unstandardized parameter	SE	p	Standardized parameter
Mean values of latent variables				
Intercept PDS	2.08	0.03	<.001	5.33
Step PDS	-0.97	0.14	<.001	-3.78
Slope PDS	-0.07	0.01	<.001	-1.35
Intercept PTCI	4.29	0.08	<.001	3.61
Step PTCI	-1.29	0.25	<.001	-2.28
Slope PTCI	-0.08	0.01	<.001	-0.87
Range of values for autoregressive correlations				
PDS2 on PDS1 to PDS10 on PDS9	.19	.05	<.001	.14 to .19
PTCI2 on PTCI1 to PTCI10 on PTCI9	.28	.05	<.001	.27
Range of values cross-over regressions				
PDS2 on PTCI1 to PDS10 on PTCI9	.07	.03	.014	.12 to .13
PTCI2 on PDS1 to PTCI10 on PDS9	.01	.06	.832	.01

Note. This is based on mean scores for each item for PDS (Posttraumatic Diagnostic Scale, range: 0–3) and PTCI (Posttraumatic Cognition Inventory, range: 0–7) scores. Autoregressions and crossover regressions were set equal to each other for all time points. Autoregressions = regression of residual scores of one variable (e.g., residual PDS score at Session 2, PDS2) on its precursor (e.g., PDS residual score at Session 1). Crossover regressions = regression of residual scores of one variable (e.g., PDS3) on the other construct’s residual score from the previous session (e.g. PTCI2).

Predictions in the opposite direction were not significant (e.g., $\beta_{PTCI3,PDS2} = .01$; $p = .835$). PTSD symptom severity at one time-point did not predict negative appraisals at the next time-point, controlling for general decrease over time. This was also the case for a PTSD symptom score that included avoidance items only ($\beta = .01$, $p > .589$).

Discussion

Trauma-focused CBT is among the most effective treatments for PTSD (Bisson et al., 2007; Bradley et al., 2005). Empirical data on the mechanisms of therapeutic change in these treatments are needed. The present study tested whether the reduc-

tion in negative appraisals, that is, trauma-related negative cognitive appraisals, represents a mechanism in PTSD symptom reduction during CT-PTSD, a version of trauma-focused CBT. Our results showed that (a) negative trauma-related appraisals and PTSD symptoms both decreased significantly during treatment, (b) both change processes were associated, and (c) change in negative appraisals predicted symptom change in the following week, independent of a general decrease of each variable over time. This prediction was not significant in the other direction, that is, PTSD symptom change did not predict negative appraisal change in the following week, again when calculated independently of both variables’ general decrease

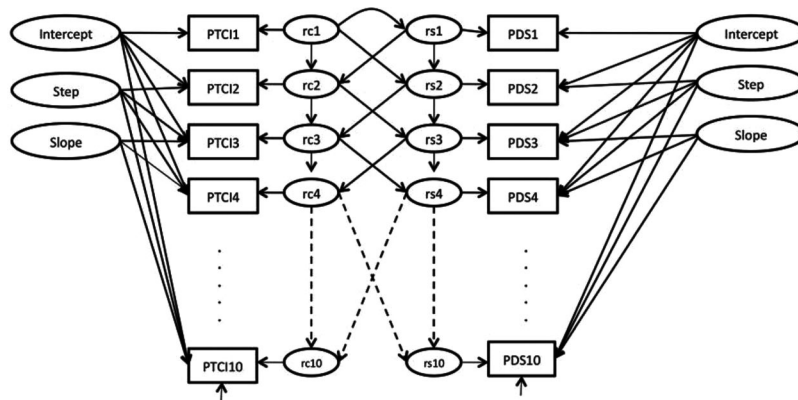


Figure 1. Bivariate latent difference score model of the Posttraumatic Diagnostic Scale (PDS, Foa et al., 1998) and the Posttraumatic Cognitions Inventory (PTCI, Foa et al., 1999, brief version) across treatment Sessions 1 to 10 ($N = 268$). For clarity of presentation, no loading parameters are depicted (all loading parameters on intercept and step = 1; step loading parameters follow a linear trend: 1, 2, 3, etc.). Dotted lines depict crossover regressions, that is, regression of residual scores of one variable on the other construct’s residual score from the previous session. Autoregressions and crossover regressions were set equal to each other for all time points. PTSD = posttraumatic stress disorder; rc = residual score cognition (PTCI); rs = residual score PTSD symptoms (PDS).

over time. The present sample was ethnically mixed, recruitment took place through a routine clinical service, and most patients had comorbid disorders. This makes it likely that the current findings generalize to other populations of trauma survivors seen in routine clinical practice.

The modification of trauma-related negative appraisals characteristic of PTSD has been proposed as a key mechanism of change in CT-PTSD (e.g., Ehlers et al., 2005) and other forms of trauma-focused CBT (e.g., Foa, Rothbaum, Riggs, & Murdock, 1991; Resick & Schnicke, 1992, 1993). In line with this hypothesis, prior research has shown that changes in cognitive appraisals were positively related to treatment outcome and that the better outcome for trauma-focused CBT compared to supportive therapy was due to a greater change in appraisals (Ehlers et al., 2005; Ehlers, Hackmann, et al., 2012; Foa et al., 1999; Vogt et al., 2012). Our results extend these studies in an important way as we investigated the temporal patterns of change in negative appraisals and symptoms over the course of treatment, with focus on the hypothesis of a temporal precedence of change in negative appraisal before change in PTSD symptoms. The results were in line with this hypothesis. They demonstrated that change in negative appraisals above and beyond the general decrease over the course of therapy predicted PTSD symptom change at the next session. These results suggest that changes in PTSD symptoms are driven by changes in negative appraisals and support the notion that reduction of negative appraisals presents an active mechanism of change. Our results are thus in direct support of the theoretical assumption that cognitive change is central to treating PTSD (Ehlers & Clark, 2000; Foa & Riggs, 1993; Resick & Schnicke, 1993) and psychological disorders more generally (D. A. Clark, 1995). The requirement that change in the mediator occurs before the dependent variable changes is a crucial, but often neglected, requirement for mediators of treatment effects (Kazdin, 2007; Kraemer, Wilson, Fairburn, & Agras, 2002). Our finding that reductions in negative appraisals predict later reductions in PTSD symptoms is thus in direct support of cognitive mediation in CT-PTSD.

We also tested the reverse temporal relationship, namely that change in symptoms leads to a subsequent change in negative appraisals, as it is conceivable that a presumed mediator may be actually be caused by the outcome variable. If changes in negative appraisals are a mechanism of symptom change, then they should not just be a consequence of improvement in symptoms. Our finding that reductions in PTSD symptoms beyond the general decline in symptoms did not predict reductions in negative appraisals at the next treatment session provides further support for the direction of the relationship. Change in PTSD symptoms was directly preceded by change in negative appraisals, but not vice versa, a pattern that further supports cognitive mediation of treatment effects.

We included an initial "step" in symptom and negative appraisal reduction in our model, which increased the measurement model's fit to the data. This pattern of symptom and negative appraisal reduction may be due to two factors. First, there were individual differences in the timing of completion of the first questionnaire set. Second, change in both negative appraisals and symptoms may also show a steep initial decline when treatment commences, followed by a smaller, but more steady linear decline from Session 2 onwards. Such an initial symptom decrease is consistent with a number of studies that have reported rapid and large early re-

sponses in cognitive behavioral therapy (Hardi & Craighead, 1999; Strunk, Brotman, & DeRubeis, 2010), including a recent study on Cognitive Processing Therapy for PTSD (MacDonald, Monson, Doron-Lamarca, Resick, & Palfai, 2011). The present data are in line with these studies and document that some of the improvement in symptom severity, as well as reduction in the endorsement of negative appraisals may already occur very early in treatment. The initial treatment session normalized PTSD symptoms and provided patients with a conceptualization of their problems and treatment rationale. All these interventions are likely to induce hope and counteract negative interpretations such as "I have permanently changed for the worse" or "My reactions since the event mean I am going crazy."

Initial PTSD symptom severity was not significantly related to the rate of cognitive change throughout therapy sessions. Thus, patients with a wide range of PTSD symptom severities showed similar changes in their appraisals. The results were not due to a ceiling effect as there was a substantial range of initial symptoms, which was indicated by the moderate to large standard deviation in initial PDS scores. A similar picture emerged with respect to initial negative appraisal. Individual differences in pretreatment negative appraisals were not related to the rate of symptom change during treatment. This finding is in contrast to recent results by Moser, Cahill, and Foa (2010), who found in a sample of female assault survivors treated with prolonged exposure and cognitive restructuring that those with more severe initial negative appraisals (indexed with the PTCI) demonstrated a poorer treatment outcome. In the present sample, the rate with which patients' symptoms changed during treatment was unrelated to the initial severity of trauma-related negative appraisals. It is possible that the different results are a function of differences between the treatment protocols, for example, the procedures used in CT-PTSD to link the change in appraisals directly with the relevant moments in memory in the updating trauma memories procedure. Patients with very severe cognitive distortions of particular moments in memory and/or severe dissociation may be less able than other patients to link insights from cognitive restructuring with the relevant parts of the trauma memory and may thus require additional procedures such as the updating procedure used in CT-PTSD.

The present study is not without limitations. First, we chose to investigate the role of negative trauma-related appraisals in the process of change during CT-PTSD. Information processing models of PTSD propose a number of other treatment mechanisms (e.g., Ehlers & Clark, 2000; Foa & Rothbaum, 1998) such as changes in trauma memories and changes in cognitive-behavioral strategies thought to prevent change, such as avoidance, thought suppression, or safety behaviors. These proposed mediators, and their relationship with changes in appraisals, should be tested in future studies. Different mechanisms could operate in different ways, for example, together at the same time or during different components of therapy, depending on what specific techniques are applied (see Murphy, Cooper, Hollon, & Fairburn, 2008). It is also possible that change in one mediator promotes change in another, for example, changes in the perceivednowness of trauma memories may lead to decreases in appraisals of impending danger (Ehlers, Hackmann, & Michael, 2004). Dropping problematic behaviors such as excessive precautions is thought to lead to a reevaluation of problematic appraisals when the patient realizes that the predicted catastrophic outcomes do not occur (Ehlers &

Clark, 2000). Second, our model relied on the assumption that the group of patients under study changes as a group in a characteristic and rather uniform way. That is, negative trauma-related appraisals and PTSD symptoms are thought to change in a similar way across the sample. However, it is possible that there are subpopulations that change in idiographic ways, for instance, subpopulations with different rates of decline in negative appraisals and PTSD symptoms. Third, we did not link changes in negative appraisals and PTSD symptoms to the content of specific sessions. It would be intriguing to study the sessions preceding peak changes in appraisals or PTSD symptoms in greater detail, for instance, by applying sudden gain criteria (Tang & DeRubeis, 1999). Fourth, we did not have the resources to obtain fidelity or therapist competency ratings from recordings of the therapy sessions. This may have introduced error variance. However, the close supervision of all cases ensured that therapists followed the protocol. The analysis of session notes confirmed that for nearly all patients, treatment sessions mainly focused on the PTSD treatment protocol. Fifth, this study focused on an investigation of negative appraisal and symptom change within CBT and one important future research direction would be to investigate these changes in other types of psychotherapy. A final limitation is that this study focused on Sessions 1 to 10, following the argument that the main change should happen during this time, while some patients had more sessions, depending on comorbidities and number of traumas to be addressed. Our analyses therefore do not capture changes in negative appraisals and symptoms that may happen at later stages for patients requiring a greater number of sessions.

Despite these limitations, the present study presents a significant step forward in the investigation of active treatment mechanisms in the treatment of PTSD. In line with information processing models of PTSD (Ehlers & Clark, 2000; Foa & Riggs, 1993; Foa & Rothbaum, 1998), cognitive change during CT-PTSD predicted subsequent change in PTSD symptoms, but not vice versa. This finding, in conjunction with data from randomized trials (Ehlers et al., 2005; Ehlers, Hackmann, et al., 2012), supports change in trauma-related appraisals as a mediator of treatment effects. Furthermore, we showed that PTSD symptom changes during CT-PTSD were unrelated to the severity of negative appraisals and PTSD symptom severity at the outset of therapy, indicating similar mechanisms of change across the whole spectrum of initial symptom severity. Future studies will nevertheless need to shed light on whether further person and therapy variables act as moderators of this change.

References

- American Psychiatric Association Work Group on ASD and PTSD. (2004). *Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder*. Washington, DC: Author.
- Australian Centre for Posttraumatic Mental Health. (2007). *Australian guidelines for the treatment of adults with acute stress disorder and posttraumatic stress disorder: Information for people with ASD and PTSD, their families and carers*.
- Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 6, 1173–1182. doi:10.1037/0022-3514.51.6.1173
- Beck, A. T., & Steer, R. A. (1987). *Beck Depression Inventory—Manual*. San Antonio, TX: The Psychological Corporation.
- Beck, J. G., Coffey, S. C., Palyo, S. A., Gudmundsdottir, B., Miller, L. M., & Colder, C. R. (2004). Psychometric properties of the Posttraumatic Cognitions Inventory (PTCI): A replication with motor vehicle accident survivors. *Psychological Assessment*, *16*, 3, 289–298. doi:10.1037/1040-3590.16.3.289
- Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder. *The British Journal of Psychiatry*, *190*, 97–104. doi: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*, 214–227. doi:10.1176/appi.ajp.162.2.214
- Brewin, C. R., Fuchkan, N., Huntley, Z., Robertson, M., Scragg, P., Thompson, M., . . . Ehlers, A. (2010). Effectiveness of a screen and treat program in improving mental health following the 2005 London bombings. *Psychological Medicine*, *40*, 2049–2057. doi:10.1017/S0033291710000206
- Clark, D. A. (1995). Perceived limitations of standard cognitive therapy: A reconsideration of efforts to revise Beck's theory and therapy. *Journal of Cognitive Psychotherapy*, *9*, 153–172.
- Clark, D. M., & Ehlers, A. (2004). Posttraumatic stress disorder: From cognitive theory to therapy. In R. L. Leahy (Ed.), *Contemporary cognitive therapy* (pp. 141–160). New York, NY: Guilford Press.
- Cohen, J., & Mannarino, A. P. (2008). Disseminating and implementing trauma-focused CBT in community settings. *Trauma, Violence, & Abuse*, *9*, 214–226. doi:10.1177/1524838008324336
- Dalgleish, T. (2004). Cognitive approaches to posttraumatic stress disorder: The evolution of multirepresentational theorizing. *Psychological Bulletin*, *130*, 228–260. doi:10.1037/0033-2909.130.2.228
- Department of Veterans Affairs and Department of Defense. (2010). *Management of post-traumatic stress*. Washington, DC: Department of Veterans Affairs. Retrieved from http://www.healthquality.va.gov/Post_Traumatic_Stress_Disorder_PTSD.asp
- DeRubeis, R. J., Evans, M. D., Hollon, S. D., Garvey, M. J., Grove, W. M., & Tuason, V. B. (1990). How does cognitive therapy work? Cognitive change and symptom change in cognitive therapy and pharmacotherapy for depression. *Journal of Consulting and Clinical Psychology*, *58*, 862–869. doi:10.1037/0022-006X.58.6.862
- Duffy, M., Gillespie, K., & Clark, D. M. (2007). Post-traumatic stress disorder in the context of terrorism and other civil conflict in Northern Ireland: Randomised controlled trial. *British Medical Journal*, *334*, 1147–1150. doi:10.1136/bmj.39021.846852.BE
- Dunmore, E., Clark, D. M., & Ehlers, A. (2001). A prospective investigation of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy*, *39*, 1063–1084. doi:10.1016/S0005-7967(00)00088-7
- Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*, 319–345. doi:10.1016/S0005-7967(99)00123-0
- Ehlers, A., Clark, D. M., Hackmann, A., Grey, N., Wild, J., Liness, S., . . . McManus, F. (2010). Intensive cognitive therapy for PTSD: A feasibility study. *Behavioural and Cognitive Psychotherapy*, *38*, 383–398. Retrieved from <http://oxcadat.psy.ox.ac.uk/downloads/CT-PTSD%20Treatment%20Procedures.pdf/view>
- Ehlers, A., Clark, D. M., Hackman, A., McManus, F., & Fennell, M. (2005). Cognitive therapy for posttraumatic stress disorder: Development and evaluation. *Behaviour Research and Therapy*, *43*, 413–431. doi:10.1016/j.brat.2004.03.006
- Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., Fennell, M., Herbert, C., & Mayou, R. (2003). A randomized controlled trial of cognitive therapy, self-help booklet, and repeated early assessment as early interventions for PTSD. *Archives of General Psychiatry*, *60*, 1024–1032. doi:10.1001/archpsyc.60.10.1024

- Ehlers, A., Grey, N., Wild, J., Stott, R., Liness, S., Deale, A., . . . Clark, D. M. (2012). *Dissemination of cognitive therapy for PTSD into a National Health Service clinic: Effectiveness and moderators of treatment outcome*. Manuscript submitted for publication.
- Ehlers, A., Hackmann, A., Grey, N., Wild, J., Liness, S., Albert, I., . . . Clark, D. M. (2012). *A randomized controlled trial of intensive and weekly cognitive therapy for PTSD and emotion-focused supportive psychotherapy*. Manuscript submitted for publication.
- Ehlers, A., Hackmann, A., & Michael, T. (2004). Intrusive reexperiencing in posttraumatic stress disorder: Phenomenology, theory, and therapy. *Memory, 12*, 403–415. doi:10.1080/09658210444000025
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1994). *Structured clinical interview for DSM–IV Axis II Personality Disorders (SCID-II, Version 2.0)*. New York, NY: New York State Psychiatric Institute, Biometrics Research Department.
- Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment, 9*, 445–451. doi:10.1037/1040-3590.9.4.445
- Foa, E. B., Ehlers, A., Clark, D. M., Tolin, D. F., & Orsillo, S. M. (1999). The Posttraumatic Cognitions Inventory (PTCI): Development and validation. *Psychological Assessment, 11*, 303–314. doi:10.1037/1040-3590.11.3.303
- Foa, E. B., Hembree, E. A., Cahill, S. P., Rauch, S. A. M., Riggs, D. S., Feeny, N. C. & Yadin, E. (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology, 73*, 953–964. doi:10.1037/0022-006X.73.5.953
- Foa, E. B., Keane, T. M., Friedman, M. J., & Cohen, J. A. (2005). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (2nd ed.). New York, NY: Guilford Press.
- Foa, E. B., & Riggs, D. S. (1993). Posttraumatic stress disorder and rape. M. Oldham, M. B. Riba, & A. Tasman (Eds.), *American Psychiatric Press Review of Psychiatry* (pp. 273–303). Washington, DC: American Psychiatric Press.
- Foa, E. B., & Rothbaum, B. O. (1998). *Treating the trauma of rape: Cognitive-behavior therapy for PTSD*. New York, NY: Guilford Press.
- Foa, E. B., Rothbaum, B. O., Riggs, D. S., & Murdock, T. B. (1991). Treatment of posttraumatic stress disorder in rape victims: A comparison between cognitive-behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology, 59*, 715–723. doi:10.1037/0022-006X.59.5.715
- Gillespie, K., Duffy, M., Hackmann, A., & Clark, D. M. (2002). Community based cognitive therapy in the treatment of post-traumatic stress disorder following the Omagh bomb. *Behaviour Research and Therapy, 40*, 345–357. doi:10.1016/S0005-7967(02)00004-9
- Hagenaars, M. A., van Minnen, A., & de Rooij, M. (2010). Cognitions in prolonged exposure therapy for posttraumatic stress disorder. *International Journal of Clinical and Health Psychology, 10*, 421–434.
- Hoffart, A., Borge, F. M., Sexton, H., & Clark, D. M. (2009). Change processes in residential cognitive and interpersonal therapy for social phobia; a process outcome study. *Behavior Therapy, 40*, 10–22. doi:10.1016/j.beth.2007.12.003
- Hofmann, S. G. (2004). Cognitive mediation of treatment change in social phobia. *Journal of Consulting and Clinical Psychology, 72*, 393–399. doi:10.1037/0022-006X.72.3.392
- Hofmann, S. G., Meuret, A. E., Rosenfield, D., Suvak, M. K., Barlow, D. H., Gorman, J. M., . . . Woods, S. W. (2007). Preliminary evidence for cognitive mediation during cognitive-behavioral therapy of panic disorder. *Journal of Consulting and Clinical Psychology, 75*, 374–379. doi:10.1037/0022-006X.75.3.374
- Ilardi, S. S., & Craighead, W. E. (1999). Rapid early response, cognitive modification, and nonspecific factors in cognitive behavior therapy. *Clinical Psychology: Science and Practice, 6*, 295–299. doi:10.1093/clipsy/6.3.295
- Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology, 3*, 1–27. doi:10.1146/annurev.clinpsy.3.022806.091432
- King, L. A., King, D. W., McArdle, J. J., Saxe, G. N., Soron-LaMarca, S., & Orazem, R. J. (2006). Latent difference score approach to longitudinal trauma research. *Journal of Traumatic Stress, 19*, 771–785. doi:10.1002/jts.20188
- Kraemer, H. C., & Kupfer, D. J. (2006). Size of treatment effects and their importance to clinical research and practice. *Biological Psychiatry, 59*, 990–996. doi:10.1016/j.biopsych.2005.09.014
- Kraemer, H. C., Wilson, G. T., Fairburn, C. C., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry, 59*, 877–883. doi:10.1001/archpsyc.59.10.877
- McArdle, J. J., & Hamagami, F. (2001). Latent difference score structural models. In L. Collins & A. Sayer (Eds.), *New methods for the analysis of change* (pp. 137–175). Washington, DC: American Psychological Association. doi:10.1037/10409-005
- McArdle, J. J., & Nesselroade, J. R. (2002). Growth curve analysis in contemporary psychological research. In J. Schinka & W. Velicer (Eds.), *Comprehensive handbook of psychology: Vol. 2. Research methods in psychology*. New York, NY: Wiley.
- MacDonald, A., Monson, C., Doron-Lamarca, S., Resick, P. A., & Palfai, T. P. (2011). Identifying patterns of symptom change during a randomized controlled trial of cognitive processing therapy for military-related posttraumatic stress disorder. *Journal of Traumatic Stress, 24*, 268–276. doi:10.1002/jts.20642
- Moser, J. S., Cahill, S. P., & Foa, E. B. (2010). Evidence for poorer outcome in patients with negative trauma-related cognitions receiving prolonged exposure plus cognitive restructuring: Implications for treatment matching in posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 198*, 72–75. doi:10.1097/NMD.0b013e3181e81fac
- Murphy, R., Cooper, Y., Hollon, S. D., & Fairburn, C. G. (2009). How do psychological treatments work? Investigating mediators of change. *Behaviour Research and Therapy, 47*, 1–5. doi:10.1016/j.brat.2008.10.001
- Muthén, L. K., & Muthén, B. O. (2010). *Mplus user's guide* (6th ed.). Los Angeles, CA: Author.
- National Institute for Health and Clinical Excellence. (2005). *Posttraumatic stress disorder (PTSD): The management of PTSD in adults and children in primary and secondary care* (NICE Clinical Guideline 26). Retrieved from <http://guidance.nice.org.uk/CG26>
- Owens, G. P., Pike, J. L., & Chard, K. M. (2001). Treatment effects of cognitive processing therapy on cognitive distortions of female child sexual abuse survivors. *Behavior Therapy, 32*, 413–424. doi:10.1016/S0005-7894(01)80028-9
- Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 70*, 867–879. doi:10.1037/0022-006X.70.4.867
- Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology, 60*, 748–756. doi:10.1037/0022-006X.60.5.748
- Resick, P. A., & Schnicke, M. K. (1993). *Cognitive processing therapy for rape victims*. Newbury Park, CA: Sage.
- Schnurr, P. P., Friedman, M. J., Engel, C. C., Foa, E. B., Shea, M. T., Chow, B. K., . . . Bernardy, N. (2007). Cognitive behavioral therapy for posttraumatic stress disorder in women: A randomized controlled trial. *JAMA: The Journal of the American Medical Association, 297*, 820–830. doi:10.1001/jama.297.8.820
- Smith, P. Yule, W., Perrin, S., Tranah, T., Dalgleish, T., & Clark, D. M. (2007). Cognitive-behavioral therapy for PTSD in children and adoles-

- cents: A preliminary randomized controlled trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46, 1051–1061. doi:10.1097/CHI.0b013e318067e288
- Stein, D. J., Cloitre, M., Nemeroff, C. B., Nutt, D. J., Seedat, S., Shalev, A. Y., . . . Zohar, J. (2009). Cape Town consensus on posttraumatic stress disorder. *CNS Spectrums*, 14(Suppl. 1), 52–58.
- 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*, 77, 595–606. doi:10.1037/a0016032
- Stice, E., Rohde, P., Seeley, J. R., & Gau, J. M. (2010). Testing mediators of intervention effects in randomized controlled trials: An evaluation of three depression prevention programs. *Journal of Consulting and Clinical Psychology*, 78, 273–280. doi:10.1037/a0018396
- Strunk, D. R., Brotman, M. A., & DeRubeis, R. J. (2010). The process of change in cognitive therapy for depression: Predictors of early inter-session symptom gains. *Behaviour Research and Therapy*, 48, 599–606. doi:10.1016/j.brat.2010.03.011
- Tang, T. Z., & DeRubeis, R. J. (1999). Sudden gains and critical sessions in cognitive-behavioral therapy for depression. *Journal of Consulting and Clinical Psychology*, 67, 894–904. doi:10.1037/0022-006X.67.6.894
- Teachman, B. A., Marker, C. D., & Smith-Janik, S. B. (2008). Automatic associations and panic disorder: Trajectories of change over the course of treatment. *Journal of Consulting and Clinical Psychology*, 76, 988–1002. doi:10.1037/a0013113
- Vögele, C., Ehlers, A., Meyer, A. H., Frank, M., Hahlweg, K., & Margraf, J. (2010). Cognitive mediation of clinical improvement after intensive exposure therapy of agoraphobia and social phobia. *Depression and Anxiety*, 27, 294–301. doi:10.1002/da.20651
- Vogt, D. S., Shepherd, J. C., & Resick, P. A. (2012). Posttraumatic maladaptive beliefs scale: Evolution of the personal beliefs and reactions scale. *Assessment*, 19, 308–317. doi:10.1177/10731911110376161

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