# Cognitions in prolonged exposure therapy for posttraumatic stress disorder[1]

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**ABSTRACT.** This quasi-experimental study investigates the impact of prolonged exposure treatment on cognitive changes in patients with posttraumatic stress disorder (PTSD). Seventy-seven PTSD patients with mixed traumas went through 8 to 12 sessions of imaginal exposure and exposure in vivo without any cognitive interventions. Reexperiencing symptoms and the most important cognition (*i.e.,* its frequency and credibility) were assessed weekly. Exposure treatment resulted in significant reductions of negative trauma-related cognitions, although these were not paid attention to during treatment. Reductions in cognitions were associated with reductions in PTSD symptoms. Pre to post-treatment reductions in cognitions did not predict PTSD at follow up after controlling for pre to post-treatment reductions in PTSD symptoms. Finally, reductions in the frequency of reexperiences and trauma-related cognitions were preceded by reductions in the distress caused by the reexperiences. The credibility of trauma-related cognitions was the last variable to decline. Distress caused by the reexperiences being the first symptom to decline may mean that reductions of negative trauma-related cognitions are the result of PTSD symptom reductions. This complicated relationship between cognitions and symptom change surely merits further exploration.

**KEYWORDS.** Posttraumatic stress disorder. Dysfunctional cognitions. Treatment. Quasi-experiment.

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**RESUMEN**. Este estudio cuasi-experimental investiga el impacto del tratamiento de exposición prolongada en los cambios cognitivos en pacientes con trastorno por estrés postraumático (TEPT). Setenta y siete pacientes con TEPT con traumas mixtos pasaron por 8-12 sesiones de exposición en imaginación y en vivo, sin ninguna intervención cognitiva. La reexperimentación de los síntomas y las cogniciones más importantes (*i.e.* su frecuencia y credibilidad) fueron evaluadas semanalmente. El tratamiento de exposición redujo de forma significativa las cogniciones negativas relacionadas con el trauma, aunque no se prestó atención a éstas durante el tratamiento. La reducción en cogniciones se relacionó con la reducción en los síntomas del TEPT. La reducción en las cogniciones pre y post-tratamiento no predijo el TEPT en el seguimiento habiendo controlado la reducción en los síntomas del TEPT pre y post-tratamiento. Finalmente, la disminución en la frecuencia de la reexperimentación y de las cogniciones relacionadas con el trauma fue precedida por la reducción del sufrimiento causado por la reexperimentación. La credibilidad de las cogniciones relacionadas con el trauma fue la última variable que disminuyó. El hecho de que el sufrimiento causado por la reexperimentación fuera el primer síntoma en disminuir puede significar que la reducción de las cogniciones relacionadas con el trauma es el resultado de la reducción de los síntomas del TEPT. Esta complicada relación entre las cogniciones y el cambio en los síntomas, sin duda, merece ser explorada en el futuro.

**PALABRAS CLAVE.** Trastorno por estrés postraumático. Cogniciones disfuncionales. Tratamiento. Cuasi-experimento.

In 1939, Mowrer noted that the conditioned response (CR) decreased when the conditioned stimulus (CS) was repeatedly presented in the absence of the unconditioned stimulus (UC): extinction. Throughout the years, this process has been regarded as the mechanism responsible for the reduction of anxiety in exposure therapy (Davis, Ressler, Rothbaum, and Richardson, 2006; Watson and Rayner, 1920). Extinction learning is considered to be a «low-level process» that occurs automatically and unconsciously. Exposure treatments were once assumed to involve extinction learning only. Neuroscience and animal studies indeed show that subcortical areas of the brain, especially the amygdala, are involved in fear conditioning and extinction (LeDoux, 1996). This does not mean though, that cognitions are of no importance in extinction learning, as the amygdala has reciprocal connections to the medial prefrontal cortex, an area involved in planning and emotion regulation.

Contemporary theories of posttraumatic stress disorder (PTSD) emphasise the importance of negative trauma-related cognitions with respect to the development and maintenance of PTSD (Ehlers and Clark, 2000; Foa, Ehlers, Clark, Tolin, and Orsillo, 1999). Foa and Rothbaum (1998), for example, proposed that mainly two basic dysfunctional cognitions are involved in this process: the trauma causes the victim to perceive the world as an extremely dangerous place, and oneself as incompetent. Ehlers and Clark (2000) posit that PTSD becomes persistent if the victim experiences a sense of serious current threat, which is caused by negative appraisals of the trauma and its sequelae. Normally, these negative trauma-related cognitions are disconfirmed in daily life by encountering new, incompatible, and thus corrective, information (Foa and Kozak, 1986).

However, some individuals engage in problematic strategies, like avoiding trauma-related thoughts, behaviors, and situations, thereby preventing the negative trauma-related cognitions to be changed. As a result, PTSD symptoms become chronic.

Although exposure therapy was developed from this theory, the underlying mechanism is not considered to be merely extinction. That is, as trauma victims that previously engaged in avoidance strategies (typical for all PTSD patients) are now presented with new information, trauma-related cognitions are disconfirmed (Foa and Rothbaum, 1998). Many patients improve after exposure treatment (Bradley, Greene, Russ, Dutra, and Westen, 2005), and indeed, there are some indications that extinction is not the only underlying mechanism of exposure therapy. If that was the case, adding cognitive techniques should increase exposure treatment efficacy, because they target a possible means of change ignored by exposure. However, «simple» exposure treatments with and without cognitive interventions were found to be equally effective in PTSD (Foa *et al.,* 2005; Foa and Rauch, 2004), but also in other anxiety disorders (Hofmann, Moscovitch, Kim, and Taylor, 2004). Moreover, plain exposure techniques were found to lead to changes in cognitions (Hofmann, 2000). It has therefore been suggested that symptom changes in exposure therapy are mediated by changes in cognitions, «and specifically changes in CS-US (harm) expectancies» (Hofmann, 2007, pp. 7).

The exact underlying mechanisms of change in exposure therapy thus remain unclear. Reduction of PTSD symptoms is assumed to occur as a result of extinction and changes in negative trauma-related cognitions without explicitly addressing any cognition. However, other treatment strategies do indeed directly target negative trauma-related cognitions. The most important one, cognitive therapy, is also based on the assumption that cognitions play a crucial role in the development and maintenance of PTSD. But here, cognitive interventions specifically aim at altering dysfunctional cognitions. That is, addressing negative trauma-related cognitions is the main ingredient in cognitive therapy for PTSD. Like exposure, cognitive therapy indeed proved to be effective (Ehlers *et al.,* 2003). Thus, although most theories agree on the involvement of negative cognitions in the development and maintenance of PTSD, they differ in the direct aim to change these cognitions in therapy. As both forms of therapy seem to be effective, the question remains what the exact role of cognitions is in exposure therapy.

In conclusion, cognitions may play a role in extinction learning and somehow mediate the effect of exposure therapy, for example by changing the expectancy of harm and danger. However, the precise mechanisms remain unclear. Moreover, Hayes (2004) noted three pitfalls in the literature on cognitive therapy. The first was already mentioned here, that is, the added value from cognitive interventions does not show by component analyses. Secondly, early improvement in symptoms often occurs before any cognitive intervention has taken place, and thirdly, changes in cognitive mediators may not precede symptom change. The latter point has hardly been investigated and was therefore addressed in the present study.

The present study aims to examine the role of negative trauma-related cognitions in prolonged exposure therapy for PTSD, and tries to determine causality in changes in PTSD symptoms and cognitions. We used a quasi-experimental design to fit the natural setting (Montero and León, 2007; Ramos-Alvarez, Moreno-Fernández, Valdés-

Conroy, and Catena, 2008). First, we aim at replicating the finding that trauma-related cognitions change as a result of prolonged exposure treatment without any cognitive interventions or reappraisals. It is expected that the severity of trauma-related cognitions to decline as a result of exposure treatment. Second, we studied whether this change in trauma-related cognitions is related to the change in PTSD symptoms. We expect the reduction of dysfunctional cognitions to be related to the reduction of PTSD symptoms. Third, it was investigated whether severe dysfunctional cognitions predicted PTSD treatment outcome. It was hypothesized that a reduction of dysfunctional cognitions would indeed predict reductions in PTSD symptoms, but that this effect would be the result of the relationship between cognitions and PTSD and therefore disappear after controlling for reductions of PTSD symptoms. Fourth, the course of both cognitions and reexperiences (being a core PTSD symptom) during treatment was studied, because this might tell us something about the order in which cognitions and reexperiences change, implying some underlying causality.

## Method

### *Participants*

Ninety-nine patients, referred to an outpatient clinic for anxiety disorders, met the Diagnostic and Statistical Manual of Mental Disorders, fourth edition revised (DSM-IV-TR) criteria for PTSD (American Psychiatric Association, 2000) as a primary diagnosis, and thus were included in the present study. Patients were excluded if they were suicidal, or involved in ongoing traumatization, or if they met the DSM-IV criteria for substance dependence or a psychotic disorder. Mean age was 35.70 ($SD$ = 11.40; range 18 to 63). Patients had suffered mixed traumas: sexual assault ($n$ = 22), nonsexual assault ($n$ = 29), both sexual assault and nonsexual violence ($n$ = 16), accidents ($n$ = 9), and other ($n$ = 23). Of the 99 patients 50 (50%) were traumatized repetitively, and 49 (50%) patients had experienced a single trauma. Nineteen patients (19%) were male, 80 (81%) were female. At the beginning of the treatment, the mean time since the traumatic event had taken place was 10 years and 6 months ($SD$ = 11 years, range 6 months to 52 years). Co morbidity was high: other anxiety disorder ($n$ = 56), mood disorder ($n$ = 32), substance abuse ($n$ = 2), somatoform disorder ($n$ = 3), eating disorder ($n$ = 3), cluster C personality disorder ($n$ = 15), mixed cluster personality disorder ($n$ = 3). Twenty-five patients had no co morbid DSM-IV disorder. Educational level was low in 25 patients (25%), medium in 43 patients (43%), and high in 31 patients (31%).

Of the 99 intent-to-treat (ITT) patients, 22 (22%) dropped out of treatment before the post-treatment assessment. Seventy-seven patients (78%) completed treatment and the post-treatment assessment. The mean number of sessions for the dropouts was 3.90 ($SD$ = 3.29). Dropouts tended to be lower educated relative to the completers ($t_{(97)}$ = -1.68, $p$ = .10). Dropouts and completers did not differ in co morbidity ($\chi^2_{(2, N = 99)}$ = 1.24, $p$ = .54), age ($t_{(97)}$ = .09, $p$ = .93), multiple or single trauma $\chi^2_{(2, N = 99)}$ = .09, $p$ = .76), or pre-treatment PTSD symptoms ($t_{(99)}$ = 1.50, $p$ = .14). Dropouts tended to have higher scores on the Posttraumatic Cognitions Inventory (PTCI) subscale n*egative self* and on the PTCI total ($t_{(99)}$ = 1.75, $p$ = .09 and $t_{(99)}$ = 1.66, $p$ = .10 respectively), but not on the

PTCI subscales *Negative world* and *Self blame* ($t_{(99)}$ = .22, $p$ = .84 and $t_{(99)}$ = .89, $p$ = .38 respectively).

*Measures*

Diagnosis was made by means of Structured Clinical Interview for DSM-IV-TR (SCID-I and SCID-II). SCID-I and SCID-II are standardized, semi structured, diagnostic interviews for diagnosing all major psychiatric disorders. SCID-I is used to detect the presence of axis I disorders (First, Spitzer, Gibbon, and Williams, 1996) and SCID-II checks for the presence of axis II personality disorders (First, Gibbon, Spitzer, Williams, and Benjamin, 1997). Each SCID-I or SCID-II item corresponds to the DSM-IV-TR criteria of the specific psychiatric disorder. There are 4 ways to score each item: *«absent», subthreshold, threshold* or *inadequate information.* The reliability of the SCID-I in different patient samples showed to be good. Overall kappa's were .61 for current and .68 for lifetime diagnosis. The reliability of the Dutch version of the SCID-II showed to be good (Arnzt *et al.,* 1992). Kappa's ranged from .77 for obsessive-compulsive personality disorder to .82 for avoidant personality disorder in an outpatient population. Weighted kappa for all personality disorders was .80.

Outcome was measured by the following instruments:

– Clinician-Administered PTSD Scale-1 (CAPS-1). The CAPS-1 is a structured interview developed to test for the presence of the 17 DSM-IV-TR criteria for PTSD (Blake *et al.,* 1995). Each symptom is scored on two dimensions: frequency and intensity. Both scales are to be rated on a 5 point scale, ranging from 0 (*never* and *not at all*) to 4 (*every day* and *extremely*). The inter rater reliability for all three subscales of the CAPS-1 is good ($r$ = .92 to .99 for frequency, $r$ > .98 for intensity (Blake *et al.*, 1990). The internal consistency for all items of the CAPS-1 is high ($\alpha$ = .94; Blake *et al.,* 1995) and the concurrent validity is adequate (correlation with Mississippi Scale for Combat-related PTSD: $r$ = .70 to .91, correlation with MMPI PTSD subscale $r$ = .77 to .84; Blake *et al.,* 1990; Blake *et al.,* 1995; Keane, Caddell, and Taylor, 1988; Keane, Malloy, and Fairbank, 1984). We used a Dutch version of the CAPS-1, which was adapted to the DSM-IV-TR criteria of PTSD (Hovens, Luinge, and Van Minnen, 2005).
– Posttraumatic Stress Symptom Scale, Self Report (PSS-SR). The PSS-SR is a 17 item self report questionnaire that measures the frequency of PTSD symptoms (Foa, Riggs, Dancu, and Rothbaum, 1993). Each item corresponds to one of the DSM-IV-TR criteria for PTSD, and has three subscales: *Reexperiencing symptoms, Avoidance symptoms* and *Arousal symptoms.* Each item has to be answered on a 4-point Likert scale, ranging from 0 (*never*) to 3 (*very often* or *more than 5 times a week*). Analyses showed a high internal consistency (Cronbach's alpha for the total score was .91), and a good test-retest reliability of the overall severity (.74) (Foa *et al.,* 1993). The Dutch version also shows good internal consistency ($\alpha$ = .92).
– Posttraumatic Cognitions Inventory (PTCI). The PTCI (Foa *et al.,* 1999) measures possible cognitions after a traumatic event. It consists of 33 items that can be

rated on a 7-point scale ranging from 1 *(totally disagree)* to 7 *(totally agree)*. The items can be classified into three categories: *Negative cognitions about the self, Negative cognitions about the world,* and *Self-blame.* All three subscales show an excellent internal consistency (alpha = .97, .88 and .86 respectively; alpha = .97 for the total score) and good test-retest reliability ($\rho$ = .75, .89 and .89 respectively; $\rho$ = .74 for the total score) and all three subscales discriminate between traumatized individuals with and without PTSD (Foa *et al.,* 1999).

– Most important cognition. At the first assessment, all patients had to state their most important trauma-related cognition. If it proved difficult to pick the most important cognition, the PTCI was used as guidance and the cognition that was rated highest was chosen. This cognition was then rated on its frequency in the preceding week, and on its credibility. Patients rated frequency, ranging from 0 *(did not occur at all)* to 10 *(occurred all the time)* and credibility, ranging from 0 *(not credible at all)* to 10 *(convinced the cognition is true)*. The same cognition was rated like this at every treatment session and at every assessment, providing an insight in the weekly change of this cognition during treatment.

– Most important reexperience. At the first assessment, all patients had to state their most important trauma-related reexperience. This reexperience was then rated on its frequency in the preceding week, and on the amount of distress it provoked in that week. Patients rated both frequency, ranging from 0 *(did not occur at all)* to 10 *(occurred all the time)* and distress, ranging from 0 *(did not provoke any distress)* to 10 *(provoked very much distress)*.The same intrusion was rated like this at every treatment session and at every assessment, providing an insight in the weekly change of this intrusion during treatment.

*Procedure*

The participants cooperated on a voluntary basis and completed informed consent forms after the procedure was explained. All assessments were conducted by trained, independent assessors. Pre-treatment screening (establishing diagnosis, comorbidity, and inclusion and exclusion criteria) comprised the SCID-I and SCID-II. Within a week, included patients subsequently completed all pretreatment questionnaires and took part in the CAPS-1 interview.

One week after the abovementioned assessments, patients entered a standardized prolonged exposure treatment program (Freeman and Dattilio, 1993) comprising 8 to 12 weekly sessions that lasted 45 minutes. Note, however, that in 10 cases treatment was ended before the 8th session because the patients concerned had already achieved (full) recovery: they no longer met the DSM-IV-TR PTSD criteria according to the CAPS-1, their PSS-SR total score had dropped below 10, and their scores on the Subjective Unit of Distress Scale (SUDs, ranging from 0 to 10) recorded during the 3 last exposure sessions and the subsequent homework assignments were low (< 5). The mean number of session of these early completers was 4.38 (SD = 1.71).

The first therapy session included a presentation of the treatment rationale, education about the disorder and common reactions to trauma and information gathering. The subsequent sessions consisted of 30 minutes imaginal exposure: patients were asked to

close their eyes and talk about the traumatic event in the first person and in the present tense, recollecting as many sensory details as vividly as possible, *i.e.,* as if the trauma was happening «here and now». Each imaginal exposure session was audiotaped and patients were instructed to listen to the tape at home five times a week. From the 4th session onwards in vivo exposure assignments were an integrated part of the treatment. These included exposure to fearful stimuli associated with the trauma, like visiting trauma-related places or listening to trauma-related sounds. Each session started with a review of the patients' homework and ended with a new homework assignment. At the start of each treatment session patients completed the PSS-SR, and indicated the frequency and credibility of their most important cognition as well as the frequency of and distress caused by their most important reexperience. Treatment fidelity was rated after each session and all therapists involved were supervised weekly by the second author. Following treatment conclusion, all patients again completed the abovementioned questionnaires and participated in the post-treatment CAPS-1 interview.

Follow-up assessments comprising all questionnaires and the CAPS-1 were conducted six months after the post-treatment assessment.

*Statistical analyses*

To address the first research question (the effect of treatment on cognitions), repeated measures ANOVAs were conducted. The ITT sample was used for this analysis with last observation carried forward (LOCF) scores in order to have a conservative estimation of the treatment effect and to compare this effect across studies.

All subsequent analyses were done using the completers sample because the primary objective was to investigate mediation of treatment change, and not to demonstrate treatment effect. These mediation analyses are restricted to completers, as it is necessary to study changes in the dependent variable as well as in the mediator over the same time interval for all participants (Kraemer, Wilson, Fairburn, and Agras, 2002).

If cognitions serve as mediators of exposure treatment, changes in cognitions should be related to changes in PTSD symptoms, and should have a main or interactive effect on treatment outcome (Hofmann, 2004). To establish the association between changes in dysfunctional cognitions and changes in PTSD symptoms (second research question), correlations between pre to post-treatment PTCI residual gain scores and pre to post-treatment PSS residual gain scores were calculated. Residual gain scores control for pre-treatment differences and measurement error, occurring as a result of repeated measures on the same instrument (Beutler and Hamblin, 1986; Steketee and Chambless, 1992).

To address the third research question (whether reductions in dysfunctional cognitions predicted reductions in PTSD), stepwise linear regression analyses were conducted to calculate the impact of pre- to post-treatment changes in PTCI predicted PTSD at follow up (FU), when controlling for pre- to post-treatment changes in PTSD.

Finally, if cognitions play a causal role in the working mechanism of exposure therapy, reductions of dysfunctional cognitions should precede reductions in PTSD symptoms (Hayes, 2004). To this end, multilevel analyses were used to examine the course of cognitions and reexperiences during treatment. MLWIN was used for the latter multilevel analysis, SPSS for the other analyses.

## Results

*Treatment effect: PTSD symptoms and cognitions*

Mean posttraumatic stress symptoms and posttraumatic cognitions at the different time points are listed in Table 1. Repeated measures analyses showed that treatment was successful in reducing PTSD symptoms from pre-treatment to FU ($F_{(2, 97)} = 54.63$, $p <$ .001, $\eta^2 = .53$). *Post-hoc* analyses with Bonferroni corrections showed that these reductions were significant from pre- to post-treatment ($p < .001$), and not from post to FU *(p = .43)*.

Total PTCI scores also declined from pre-treatment to follow-up ($F_{(2, 90)} = 54.55$, $p < .001$, $\eta^2 = .58$). *Posthoc* analyses with Bonferroni corrections showed that PTCI reductions were significant from pre- to post-treatment ($p < .001$), and not from post-treatment to FU ($p = .27$). All PTCI subscales also declined from pre-treatment to FU (*Self*: $F_{(2, 97)} = 32.59$, $p < .001$, $\eta^2 = .46$; *World*: $F_{(2, 97)} = 21.01$, $p < .001$, $\eta^2 = .35$; *Self-blame*: $F_{(2, 97)} = 6.15$, $p < .01$, $\eta^2 = .14$). *Post-hoc* analyses with Bonferroni corrections showed that reductions of the *Self* and *World* subscales were significant from pre- to post-treatment ($p < .001$), and not from post-treatment to FU ($p = .91$ and .10 for *Self* and *World* respectively). The reduction of the *Self-blame* subscale was significant from pre-treatment to FU ($p < .01$)[3] and tended to be significant from pre- to post-treatment ($p = .09$).

**TABLE 1.** Posttraumatic stress symptoms and posttraumatic cognitions at pre-treatment, post-treatment and follow up ($N = 99$).

|              | Pre-treatment | Post-treatment | Follow up     |
|--------------|---------------|----------------|---------------|
|              | M (SD)        | M (SD)         | M (SD)        |
| PSS-SR*      | 26.27 (9.10)  | 14.97 (12.06)  | 14.07 (12.14) |
| CAPS*        | 73.54 (20.11) | 32.97 (30.21)  | 27.08 (27.43) |
| PTCI total*  | 124.99 (34.58)| 91.33 (36.41)  | 84.66 (38.80) |
| PTCI self*   | 3.76 (1.21)   | 2.60 (1.21)    | 2.41 (1.29)   |
| PTCI world*  | 4.77 (1.38)   | 3.70 (1.57)    | 3.58 (1.70)   |
| PTCI blame** | 2.71 (1.43)   | 2.39 (1.39)    | 2.06 (1.26)   |

*Notes.* PSS-SR = Posttraumatic Stress Symptoms-Self Rating scale, CAPS = Clinician Administered PTSD Scale, PTCI = Posttraumatic Cognitions Inventory.
*$p < .001$. **$p < .01$

*Association cognitions and PTSD symptoms*

The reduction of negative trauma-related cognitions (PTCI) was highly related to the reduction in PTSD symptoms from pre- to post-treatment ($r = .60$, $p < .001$), and from pre-treatment to FU ($r = .70$, $p < .001$). Pre- to post-treatment reductions of the PTCI subscales *Self* and *World* were also related to a pre- to post-treatment reduction in PTSD

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[3] Completer analyses revealed similar results, including posthoc significant and nonsignificant analyses.

symptoms ($r = .62$, $p < .001$ and $r = .46$, $p = .001$ for self and world respectively). The pre- to post-treatment reduction of the PTCI subscale *Self-blam*e tended to be related to a pre- to post-treatment reduction in PTSD symptoms ($r = .26$, $p = .08$). Pre-treatment to FU reductions of all PTCI subscales were related to a pre-treatment to FU reduction in PTSD symptoms ($r = .71$, $p < .001$, $r = .52$, $p < .001$, and $r = .32$, $p < .05$ for self, world, and self-blame respectively).

*Predictive value cognitions on FU PTSD symptoms*

Associations between pre- to post-treatment PTCI and PSS reductions do not allow any conclusions about causality or temporal precedence. Therefore, additional analyses were done using the FU data, as suggested by Hofmann (2004). Pre- to post-treatment *PTCI* changes (first step) and pre- to post-treatment *PSS changes* (second step) were entered as independent variables in a hierarchical regression analysis with PSS at FU as dependent variable. Changes in PTCI predicted FU PSS ($\beta = .52$, $p < .001$, $\Delta R^2 = .27$), but the effect disappeared after changes in PSS were entered (PTCI: $\beta = .16$, $p = .24$; PSS: $\beta = .60$, $p < .001$, $\Delta R^2 = .61$). This suggests that the predictive value of changes in PTCI on treatment outcome is explained by the correlation between changes in PTCI and PSS. The same analyses were done with pre- to post-treatment changes in the PTCI subscales (first step) and pre- to post-treatment PSS changes (second step). Results were similar in that changes in the *Self* subscale (but not the *World* and *Self-blame* subscales) significantly predicted PSS at FU ($\beta = .54$, $p = .001$, $\Delta R^2 = .31$), but this effect disappeared after changes in PSS were entered (PSS: $\beta = .56$, $p < .001$, $\Delta R^2 = .61$; all PTCI subscales: $\beta < .23$, all $p > .15$).

*Course of trauma-related cognitions and reexperiences*

A multivariate multilevel model was used to determine the course of reexperiences and cognitions during treatment. Each of the four variables (frequency cognition, credibility cognition, frequency reexperience, and distress elicited by the reexperience) was treated as outcome. Figure 1 shows the course of the most important cognition (frequency and credibility) and reexperience (frequency and distress) during therapy. As can be seen in Figure 1 the reduction of all four variables is fast at the start and slows down later in time, a pattern often found in longitudinal data analysis, which suggests to use a logarithmic transformation of time as explanatory variable to make the relationship approximately linear. Since the interest is in the slopes of this time variable a random intercept and slopes model was used for each of the four response variables. In order to test which variable declines fastest we compared five models. In the first model all slopes are free and slopes were constraint to be equal to see whether that leads to simpler but as effective models.

**FIGURE 1.** Course of PTSD reexperiencing symptoms and cognitions during therapy.
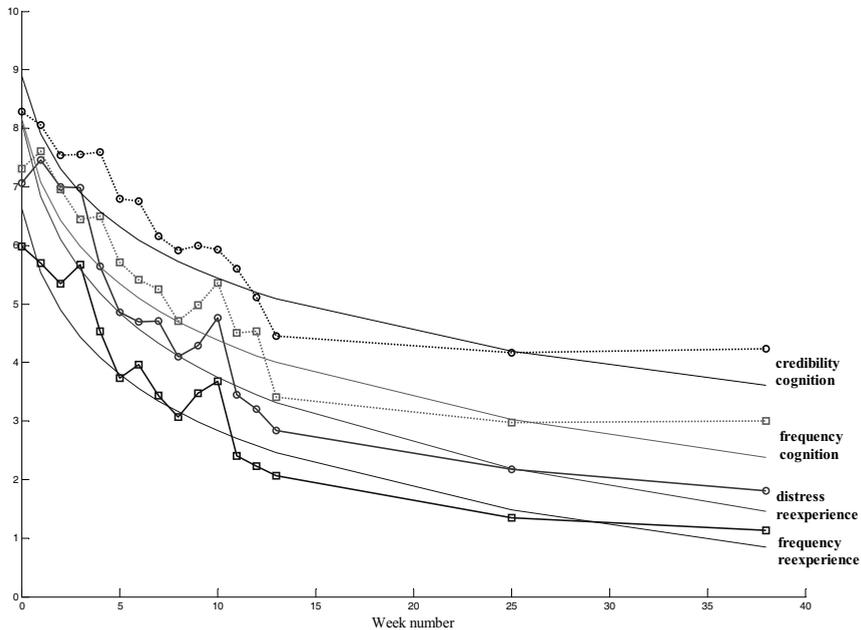


*Notes.* Non-smooth lines with marker show the observed values on the four variables; smooth lines represent the lines fitted using the multivariate multilevel regression model. The Y-axis reflects respons ratings (frequency, credibility and distress), all rated from 0 to 10.

Results are shown in Table 2, showing that all models lead to a significant poorer fit compared to model 1 except for model 4, where the slopes of frequency of cognition and frequency of reexperience are equal ($\beta$ = -1.58). The slope of distress of reexperience is -1.81 which indicates a faster decrease and the slope of credibility of cognitions is -1.44 which indicates the slowest decrease.

**TABLE 2.** Five models for course of cognitions and reexperiences.

| Model | Deviance | LR | df | p |
|---|---|---|---|---|
| (1) FC, CC, FR, IR | 13764.66 | | | |
| (2) FC=CC=FR=IR | 13872.96 | 108.30 | 24 | < .0001 |
| (3) FC = FR, CC=IR | 13830.24 | 65.58 | 23 | < .0001 |
| (4) FC=FR, CC, IR | 13774.99 | 10.33 | 9 | .3244 |
| (5) FC, FR, CC=IR | 13821.01 | 56.35 | 9 | < .0001 |

*Notes.* FC = slope for frequency of cognition; CC = slope for credibility of cognition; FR = slope for frequency of reexperience; IR = slope for distress elicited by reexperience. A '='-sign represents a model where the slopes are constraint to be equal. The Likelihood ratio test (LR) test against the first model to see whether the constraint leads to a diminished fit.

In sum, the equations and the Figure 1 show that distress by the reexperience was the variable to decline fastest during treatment. Frequency of that reexperience and frequency of the negative cognitions then followed. Credibility of the negative cognition was the latest variable to decline.

## Discussion

The present study investigated the role of negative trauma-related cognitions in prolonged exposure therapy for PTSD. It was found that negative-trauma related cognitions declined through therapy, and this decline was related to a reduction in PTSD symptoms. Negative cognitions did not predict treatment outcome. That is, their initial predictive value disappeared after controlling for pre-treatment PTSD symptoms. The course of the most important trauma-related cognition and reexperience revealed a difference in reduction of these variables. That is, distress caused by the most important reexperience was the fastest declining variable, followed by frequency of the most important reexperience and frequency of the most important cognition. The reduction in the credibility of the most important cognitions was slowest.

The fact that negative trauma-related cognitions decline as a result of exposure treatment and that this decline is related to the reduction in PTSD symptoms confirms earlier findings (Foa and Rauch *et al.*, 2004; Foa 2005). Apparently, exposure therapy has an effect on cognitions without explicit cognitive challenge procedures. This change seems to be persistent, as there was no relapse. This finding is in line with other studies that failed to show an augmentation effect of cognitive interventions on top of exposure (Foa *et al.*, 2005; Marks, Lovell, Noshirvani, Livanou, and Thrasher, 1998; Resick, Nishith, Weaver, Astin, and Feuer, 2002). It is also in line with PTSD theories stating that trauma-related cognitions can be disconfirmed by encountering new, incompatible, and thus corrective, information (Ehlers and Clark, 2000; Foa and Kozak, 1986). In this view, exposure enables a change in problematic appraisals.

The relation between cognitions and PTSD suggests that trauma-related cognitions are at least seriously intertwined with PTSD symptoms. They indirectly seem to confirm the important role of cognitions in the maintenance of the disorder.

The most interesting finding concerns the course of reexperiences and cognitions during therapy. These analyses actually permit carefull interpretations about temporal precedence and causality. That is, at least in exposure therapy, cognitive changes follow changes in the distress caused by the reexperiences, and the credibility of the cognitions was even the slowest symptom to change. This finding has implications for the mediating role of cognitions on exposure therapy. That is, one of the criteria of a mediational relationship is that changes in the mediator variable precede changes in the dependent variable (Kraemer *et al.*, 2002). Our data suggests a relationship the other way around: cognitions change as a result of a reduction in reexperiencing symptoms. Patients may actually adapt their way of thinking to their experiences. We need to emphasize that it is not stated that general cognitive changes are irrrelevant. Note that we assessed frequency of and belief in one specific, most important cognition. Instead, general cognitive changes still could have caused reductions in the distress elicited by the

reexperiences. It is argued before that exposure therapy is in fact a cognitive intervention changing levels of harm expectancy (Hofmann, 2008). Interestingly, conditioning theories also emphasize the role of harm expectancy. That is, exposure is specifically violating expectancies for the unconditioned stimulus: Extinction is considered to result from a mismatch between the expectancy of an aversive event (harm expectancy) and the absence of its occurance (Rescorla and Wagner, 1972) or from a change in the extend to which the aversive event is associated with the conditioned stimulus (Gallistel and Gibbon, 2000).

Some limitations of our study must be noted. First, all patients were treated with exposure therapy. It would be interesting to study the course of PTSD symptoms and cognitions in both exposure and cognitive therapy to investigate the underlying mechanisms of and similarities and differences in these treatments. It is possible that in cognitive therapy a change in cognitions precedes PTSD symptom change, although it may still be the other way around as well. Secondly, although the weekly assessment of symptoms allowed us to study their causal rol, assessment of symptoms were retrospective over one week and used self-report. Results should be replicated using other assessment methods, for example by recording cognitions and reexperiences at home, because the first symptom changes may take place between sessions. It would also be interesting to include implicit measures in order to establish at what level symptoms start to reduce.

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