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Change in Negative Cognitions Associated with PTSD Predicts Symptom Reduction in Prolonged Exposure

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Abstract

Objective—The goal of the current study was to examine mechanisms of change in Prolonged Exposure (PE) therapy for post-traumatic stress disorder (PTSD). Emotional Processing Theory of PTSD proposes that disconfirmation of erroneous cognitions associated with PTSD is a central mechanism in PTSD symptom reduction; but to date, the causal relationship between change in pathological cognitions and change in PTSD severity has not been established.

Method—Female sexual or nonsexual assault survivors (N = 64) with a primary diagnosis of PTSD received 10 weekly sessions of PE. Self-reported PTSD symptoms, depression symptoms, and PTSD-related cognitions were assessed at pre-treatment, each of the 10 PE treatment sessions, and post-treatment.

Results—Lagged mixed-effect regression models indicated that session-to-session reductions in PTSD-related cognitions drove successive reductions in PTSD symptoms. By contrast, the reverse effect of PTSD symptom change on change in cognitions was smaller and did not reach statistical significance. Similarly, reductions in PTSD-related cognitions drove successive reductions in depression symptoms whereas the reverse effect of depression symptoms on subsequent cognition change was smaller and not significant. Notably, the relationships between changes in cognitions and PTSD symptoms were stronger than the relationships between changes in cognitions and depression symptoms.

Conclusions—To our knowledge, this is the first study to establish change in PTSD-related cognitions as a central mechanism of PE treatment. These findings are consistent with Emotional Processing Theory and have important clinical implications for the effective implementation of PE.

Keywords

cognitions; prolonged exposure; treatment mechanism; posttraumatic stress disorder; emotional processing theory

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Prolonged Exposure therapy (PE) is highly effective in the treatment of posttraumatic stress disorder (PTSD) for patients with a wide variety of traumatic experiences (e.g., Foa et al., 2005; Paunovic & Ost, 2001; Teurk et al., 2011). Thus, the key question facing researchers now is not whether PE works, but *how* it works. Investigating potential mechanisms of change will enable us to both test the theoretical underpinnings of PE and hone treatment delivery in order to maximize it efficacy and efficiency.

Emotional Processing Theory (EPT; Foa & Kozak, 1986; Foa & Cahill, 2001), the theoretical foundation of PE, posits that chronic PTSD occurs due to cognitive and behavioral avoidance, which maintains erroneous perceptions about the world as utterly dangerous and oneself as totally incompetent. Thus, the goal of PTSD treatment is to modify these PTSD-related cognitions by presenting information that disconfirms them; this process is termed “emotional processing” (see Zalta & Foa, 2012 for a discussion of EPT and applications to PE). In PE, disconfirming information is achieved via exposure. Through in vivo and imaginal exposure, patients learn that avoided situations are safe and that they are capable of coping with distressing situations and memories. Accordingly, EPT hypothesizes that symptom reduction in PTSD via PE results from reduction in PTSD-related cognitions. This proposed mechanism of therapeutic action is not specific to PE. Evidence suggests that changes in erroneous cognitions lead to improvement in Cognitive Therapy for PTSD (Kleim et al., 2013) as well as other anxiety disorders (Teachman, Marker, & Clerkin, 2010). Given that PE has been described as a treatment of habituation, we sought to examine the role of cognitive changes in PE.

Few studies have examined the relationship between cognition and symptom change in PE. Foa and Rauch (2004) showed that PE resulted in significant reductions in PTSD-related cognitions from pre to post treatment and these reductions were correlated with PTSD symptom improvement from pre to post treatment. However, they did not examine the directional relationship between cognition and symptom change in PE. In a study of exposure therapy for PTSD, Hagenaars and colleagues (2010) concluded that “reductions of negative trauma-related cognitions are the result of PTSD symptom reductions” (p. 421). However, this study has several methodological limitations. Only study completers were used to conduct mediation analyses, which potentially biased their results. Their analysis of the causal relationship between cognitions and symptoms measured symptom change during the follow-up period after the majority of treatment change occurred. They also conducted some analyses using only individuals’ most important cognition and reexperiencing symptoms. These limitations preclude strong conclusion from this study.

The goal of our study is to examine the causal relationship between PTSD-related cognitions and symptom reduction of PTSD during PE. Because erroneous cognitions about world and self are implicated in depression (Beck, Rush, Shaw, & Emery, 1797) and PE reduces depression as well as PTSD symptom severity (Foa et al., 2005), we also examined the causal relationship between depression and PTSD-related cognitions. We applied a rigorous assessment and statistical methodology that measured cognitions and symptoms at each PE session and examined lagged associations between cognitions and symptoms while accounting for autocorrelations (i.e., the extent to which each measure predicted itself over time). Based on EPT, we hypothesized that changes in PTSD-related cognitions would predict subsequent changes in PTSD symptoms but not vice versa. We hypothesized a similar pattern for depression.

Method

Participants

Participants were female sexual or nonsexual assault survivors with a primary diagnosis of DSM-IV posttraumatic stress disorder (PTSD) for a minimum of one year. Participants were enrolled in a treatment study that included assessment of hypothalamic-pituitary-adrenal (HPA) axis functioning to determine psychological and biological mechanisms of PE. The data in the current manuscript are the first to be published from this treatment study. Individuals were initially screened by phone; those who met the preliminary criteria underwent a baseline assessment including a diagnostic interview (SCID-I; First, Spitzer, Gibbon, & Williams, 1995). All participants provided written informed consent. Participants were excluded if they a) had a history of schizophrenia, bipolar disorder, or cognitive dysfunction; b) had a history of alcohol or drug abuse or dependence within the previous 3 months; c) met criteria for mental retardation or pervasive developmental disorder; d) had a medically unstable condition that would interfere with participation or biological measurements; e) had an ongoing intimate relationship with a perpetrator; f) presented with serious suicide risk; g) were pregnant; h) were taking psychotropic medications or medications that could interfere with HPA axis functioning except maintenance SSRIs; or i) were employed in positions that altered patterns of sleep-wakefulness.

Participants who met criteria for the study and consented were randomly assigned to receive immediate PE treatment or to weekly phone contact for 10 weeks (minimal attention control condition; MA). Randomization was biased such that four out of every six participants were assigned to the immediate treatment condition. Participants in the MA condition were subsequently offered PE. Only individuals who entered PE treatment (immediate or delayed) were included in the current study. The final sample (N = 64) included 53 women who received immediate PE and 11 women who received delayed PE after completing the MA condition. There were no significant differences between those who received immediate versus delayed treatment with respect to age, race, education, income, and pre- and post-treatment PTSD symptoms, depression symptoms, and negative cognitions. Therefore, the data during PE were combined for the two groups. The ethnic composition of the sample was 56.3% Black or African American, 39.1% White, 1.6% Asian, 1.6% Native Hawaiian or Pacific Islander, and 1.6% unknown. The mean age was 39.1 years (SD = 12.6, range 19–74). This study was approved by the Human Subjects Committee of the University of Pennsylvania's Institutional Review Board.

Measures

Post-Traumatic Cognitions Inventory (PTCI)—This 36-item measure assesses negative cognitions associated with PTSD including self-blame, negative beliefs about oneself, and negative beliefs about the world (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999). Individuals rate the extent to which they agree or disagree with each statement on a 1 (*totally disagree*) to 7 (*totally agree*) scale. A total score was calculated as the sum of all items. The PTCI has been shown to discriminate between traumatized individuals with and without PTSD (Foa et al., 1999). In our sample, test-retest reliability between the first and second treatment session was .91.

Posttraumatic Stress Diagnostic Scale (PDS)—This 17-item self-report measure assesses PTSD symptom severity (Foa, Cashman, Jaycox, & Perry, 1997). Participants are asked to rate how much they have been bothered by each of the DSM-IV PTSD symptoms on a scale ranging from 0 (*not at all or only one time*) to 3 (*5 or more times a week / almost always*). The PDS is highly correlated with other measures of trauma-related

psychopathology and has strong psychometric properties (Foa et al., 1997). In our sample, test-retest reliability between the first and second treatment session was .71.

Beck Depression Inventory (BDI)—This 21-item inventory measures cognitive and vegetative symptoms of depression (Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961). Individuals are asked to report the extent to which they have been experiencing symptoms in the past week. Scores on the BDI correlate highly with clinician ratings of depression among female physical and sexual assault survivors (Foa, Riggs, Dancu, & Rothbaum, 1993). In our sample, test-retest reliability between the pre-treatment and first treatment session was .81.

Procedure

Self-reported PTSD symptoms, depression symptoms, and PTSD-related cognitions were assessed at pre-treatment (week 0), each of the 10 PE treatment sessions (weeks 1 – 10), and post-treatment (week 11) for a total of 12 time points. PE consisted of 10 weekly 90-minute sessions (Foa, Hembree, & Rothbaum, 2007). PE includes in vivo exposure to safe situations that are trauma reminders; imaginal exposure to the trauma memory followed by a discussion aimed at processing the patient's thoughts and feelings associated with the trauma; breathing retraining; and education about common reactions to trauma.

Statistical approach

A useful way to examine causal relationships between contemporaneous time-varying phenomena is lagged regression modeling where a causal effect of variable X_1 on X_2 is established when X_1 at time 1 significantly contributes to the behavior of X_2 at time 2 while controlling for the effect of X_2 at time 1 (Granger, 1969). In the present data, 12 time points were available, including a pre-therapy baseline (time 1), 10 sessions of psychotherapy (times 2 – 11), and a post-therapy endpoint (time 12). For each index (PTCI, PDS, and BDI), two variables were generated with 11 total time points: a set of dependent variables from time 2 to time 12, and a set of lagged variables from time 1 to time 11. To assess potential causal effects of each variable, we ran four mixed-effect regression models with linear shapes of change and random effects for intercept and slope: 1) the time-lagged effect of PCTI on PDS; 2) the time-lagged effect of PDS on PTCI; 3) the time-lagged effect of PTCI on BDI; and 4) the time-lagged effect of BDI on PTCI. All models were estimated with restricted maximum likelihood estimation via the lme4 package in R (version 2.15.1), allowing for inclusion of all participants who began PE.[1]

An initial step assessed the linear effect of time to determine change in the dependent variables over the study period. All three variables – the PTCI ($\beta = -6.54$, $SE = 0.73$, $p < .001$, $d = 1.61$), the PDS ($\beta = -2.04$, $SE = 0.16$, $p < .001$, $d = 2.36$), and the BDI ($\beta = -1.45$, $SE = 0.15$, $p < .001$, $d = 1.77$) – exhibited significant reductions over the course of treatment. These linear effects of time were then retained in the lagged models. Thus, for each of the 4 models, the time-varying outcome variable was predicted by the linear effect of time, time-lagged values of the same variable (autocorrelation), and time-lagged values of the predictor variable.

[1]We conducted a pattern-mixture analysis (Gallop & Tosca, 2009) to assess the effect of missingness on the dependent variables. Non-significant effects of missingness on change-over-time slopes in the dependent variables supports the assumption that data are missing at random (MAR). Having demonstrated that data were MAR, we proceeded with full information maximum likelihood estimation for the raw data with missingness intact.

Results

Lagged regression models

Table 1 presents the lagged-effect models for the PTCI and PDS. Accounting for the autocorrelations and cross-lagged effects, linear effects for time were non-significant for both the PTCI and PDS. The autocorrelations for both the PTCI and PDS were significant and produced large effects (*ds* = 3.14 and 1.52, respectively). Importantly, the cross-lagged effect of PTCI on successive PDS values was medium to large and statistically significant ($p = .001$, $d = 0.66$), indicating that preceding levels of PTSD-related cognitions drive successive levels of PTSD symptoms. By contrast, the inverse effect of PDS on successive PTCI values was significantly smaller ($z = 2.94$, $p = .003$) and not statistically significant ($p = .06$, $d = 0.35$).

Table 2 presents the lagged-effect models for the PTCI and BDI. The linear effect of time for PTCI was non-significant when accounting for the autocorrelations and cross-lagged effects; however, the linear effect for time in BDI remained significant. The autocorrelations for both the PTCI and BDI were significant and produced large effects (*ds* = 2.64 and 1.72, respectively). The cross-lagged effect of PTCI on successive BDI values was significant ($p = .04$, $d = 0.38$), whereas the inverse was not ($p = .58$, $d = 0.10$). Thus, preceding levels of PTSD-related cognitions appear to drive successive levels of depression symptoms, but not vice versa.

Assessment of active versus minimal attention conditions on change in cognitions

To examine whether the change in negative cognitions are attributable to PE and not to the passage of time, we conducted a two-sample *t*-test to test the difference between the mean change in PTCI from pre treatment (week 0) to post treatment (week 11) for the group that received only PE versus the group that received minimal attention first. The PE group exhibited a mean change in PTCI scores of −50.57 ($t = 3.39$, $p = .005$), whereas the minimal attention group exhibited a mean change of −3.76 ($t = .23$, $p = .82$). This difference was significant ($t = 3.80$, $p < .001$, $d = 0.68$), indicating that changes in PTCI during PE were due to treatment.

Discussion

To our knowledge, this is the first study to examine mechanisms of change in PE using a session-by-session approach that accounts for both autocorrelations and cross-lagged effects. The current findings indicate that reductions in negative PTSD-related cognitions lead to subsequent improvement in PTSD and, to a lesser extent, depression symptoms during PE. The reduction of PTSD symptoms led to changes in subsequent cognitions that were on par with the effect of cognition change on depression symptoms. However, this effect was significantly smaller than the effect of cognitions on subsequent PTSD symptoms and did not reach statistical significance. Controlled analyses using the minimal attention group showed that the reduction of PTSD-related cognitions over time was driven by PE.

Our findings are consistent with EPT's premise that pathological, unrealistic perceptions are a central mechanism in maintaining PTSD and that improvement in PTSD symptom severity via PE is caused by modifying these cognitions. Future research examining the extent to which the reduction of avoidance leads to change in PTSD-related cognitions in PE would lend additional support to EPT.

Several methodological differences may explain why our findings differ from those of Hagenaars and colleagues (2010). Most notably, Hagenaars and colleagues examined the extent to which cognition change during treatment predicted symptoms scores at follow-up

while controlling for pre to post treatment symptom change (i.e., the time period in which the majority of treatment related change occurred). Hagenaars and colleagues also showed that during treatment, reexperiencing symptoms change faster than individuals' worst trauma cognition; however, this analysis failed to examine the extent to which other PTSD-related cognitions changed during this period (i.e., the cognitions we would expect to change earliest in treatment). Though further research is needed, we feel confident that our methodological approach is better suited to address the question as to whether cognitive change is a mechanism of PE.

Understanding the central mechanisms of treatment can help promote effective implementation. Our findings suggest that if patients experience fewer treatment gains during PE, treatment procedures may not be effectively reducing PTSD-related cognitions. One possibility is that exposures may not be accurately targeting patients' idiographic cognitions. Assessment with the PTCI can be a helpful way to identify the patient's most central erroneous beliefs on a case by case basis. It is also possible that therapeutic procedures are targeting the proper cognitions, but other factors interfere with cognitive change such as the use of anxiolytic substances during exposure (van Minnen, Arntz, & Keijsers, 2002).

Our findings do not imply that the reduction of negative PTSD-related cognitions is the only active ingredient in PE. Indeed, EPT proposes two mechanisms underlying PE, fear activation and disconfirmation of pathological cognitions (Foa & Cahill, 2001). Studies in humans and animals consistently show that fear (emotional) activation has a causal role in fear extinction and reduction of pathological anxiety (e.g., Bouton, Kenney, & Rosengard, 1990; Chambless, Foa, Groves, & Goldstein, 1979). Examining these two mechanisms in a single study would help to elaborate the ways in which PE produces therapeutic benefit to optimize efficacy.

Several limitations should be considered when interpreting these results. Our sample size was relatively small for the use of lagged mixed regression models, limiting statistical power. Replication of this finding in a larger sample would be important to lend confidence to these results. Having an untreated control group that followed the same repeated measures assessment as the treated group would have ruled out the possibility that factors unrelated to treatment explain the observed relationship between changes in cognitions and symptoms. Notably, we were able to establish that reduction in PTSD-related cognition from pre- to post-treatment was attributable to PE using a randomized minimal attention control group. We also do not know the extent to which the current findings apply to other trauma populations. Evidence suggests that PE is effective for many different types of trauma and that trauma type does not moderate the relationship between PTSD-related cognitions and PTSD (Foa et al., 1999). However, replication in other samples is needed to establish the generalizability of our results.

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References

Beck, AT.; Rush, AJ.; Shaw, B.; Emery, G. Cognitive therapy of depression. New York: Guilford; 1979.

Beck AT, Ward CH, Mendelsohn M, Mock J, Erbaugh J. An inventory for measuring depression. Archives of General Psychiatry. 1961; 4:561–571. [PubMed: 13688369]

Bouton ME, Kenney FA, Rosengard C. State-dependent fear extinction with two benzodiazepine tranquilizers. Behavioral Neuroscience. 1990; 104:44–55. [PubMed: 2317285]

Chambless DL, Foa EB, Groves GA, Goldstein AJ. Flooding with Brevital in the treatment of agoraphobia: Countereffective? Behaviour Research and Therapy. 1979; 17:243–251. [PubMed: 526241]

First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I disorders - Patient edition (Version 2.0). New York: Biometrics Research Department; 1995.

Foa, EB.; Cahill, SP. Psychological therapies: Emotional processing. In: Smelser, NJ.; Bates, PB., editors. International encyclopedia of the social and behavioral sciences. Oxford, UK: Elsevier; 2001. p. 12363-12369.

Foa EB, Cashman L, Jaycox L, Perry K. The validation of a self-report measure of posttraumatic stress disorder: The posttraumatic diagnostic scale. Psychological Assessment. 1997; 9:445–451.

Foa EB, Ehlers A, Clark DM, Tolin DG, Orsillo SM. The posttraumatic cognitions inventory (PTCI): Development and validation. Psychological Assessment. 1999; 11:303–314.

Foa EB, Hembree EA, Cahill SP, Rauch SA, Riggs DS, Feeny NC, Yadin E. 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. 2005; 73:953–964. [PubMed: 16287395]

Foa, EB.; Hembree, EA.; Rothbaum, BO. Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences, therapist guide. New York: Oxford University Press; 2007.

Foa EB, Kozak MJ. Emotional processing of fear: Exposure to corrective information. Psychological Bulletin. 1986; 99:20–35. [PubMed: 2871574]

Foa EB, Rauch SA. Cognitive changes during prolonged exposure versus prolonged exposure plus cognitive restructuring in female assault survivors with posttraumatic stress disorder. Journal of Consulting and Clinical Psychology. 2004; 72:879–884. [PubMed: 15482045]

Foa EB, Riggs DS, Dancu CV, Rothbaum BO. Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. Journal of Traumatic Stress. 1993; 6:459–473.

Gallop R, Tasca GA. Multilevel modeling of longitudinal data for psychotherapy researchers: II: The complexities. Psychotherapy Research. 2009; 19:438–452. [PubMed: 20183399]

Granger CWJ. Investigating causal relations by econometric models and cross-spectral methods. Econometrica: Journal of the Econometric Society. 1969; 37:424–438.

Hagenaars MA, van Minnen A, de Rooij M. Cognitions in prolonged exposure therapy for posttraumatic stress disorder. International Journal of Clinical and Health Psychology. 2010; 10:421–434.

Kleim B, Grey N, Wild J, Nussbeck FW, Stott R, Hackmann A, Ehlers A. Cognitive change predicts symptom reduction with cognitive therapy for posttraumatic stress disorder. Journal of Consulting and Clinical Psychology. *in press*. Epub ahead of print.

Paunovic N, Ost LG. Cognitive-behavior therapy vs exposure therapy in the treatment of PTSD in refugees. Behaviour Research & Therapy. 2001; 39:1183–1197. [PubMed: 11579988]

Teachman BA, Marker CD, Clerkin EM. Catastrophic misinterpretations as a predictor of symptom change during treatment for panic disorder. Journal of Consulting and Clinical Psychology. 2010; 78:964–973. [PubMed: 20954759]

Teurk PE, Yoder M, Grubaugh A, Myrick H, Hamner M, Acierno R. Prolonged exposure therapy for combat-related posttraumatic stress disorder: An examination of treatment effectiveness for veterans of the wars in Afghanistan and Iraq. Journal of Anxiety Disorders. 2011; 25:397–403. [PubMed: 21131170]

van Minnen A, Arntz A, Keijsers GPJ. Prolonged exposure in patients with chronic PTSD: predictors of treatment outcome and dropout. BehaviourResearch and Therapy. 2002; 40:439–457.

Zalta, AK.; Foa, EB. Exposure therapy: Promoting emotional processing of pathological anxiety. In: O'Donohue, W.; Fisher, JE., editors. Cognitive behavior therapy: Core principles for practice. New York: John Wiley & Sons; 2012.

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Table 1

Time-lagged mixed effect regressions of PTCI and PDS

PTCI on time-lagged PDS					
	B	***SE***	***t***	***p***	***d***
Intercept	10.85	7.34	1.48	.14	0.27
Time	−1.84	2.26	−0.82	.42	0.15
PTCI Autocorrelation	0.84	0.05	17.46	< .001	3.14
Lagged PDS	0.37	0.19	1.92	.06	0.35
PDS on time-lagged PTCI					
	B	*SE*	*t*	*p*	*d*
Intercept	0.26	2.69	0.10	.92	0.02
Time	−0.90	0.82	−1.10	.28	0.20
PDS Autocorrelation	0.60	0.07	8.44	< .001	1.52
Lagged PTCI	0.07	0.02	3.67	.001	0.66

Note. PTCI = Post-traumatic Cognitions Inventory; PDS = Posttraumatic Stress Diagnostic Scale; *d* = Cohen's *d*, where $d = t * \text{sqrt}(2/n)$.

Table 2

Time-lagged mixed effect regressions of PTCI and BDI

PTCI on time-lagged BDI					
	β	***SE***	***t***	***p***	***d***
Intercept	17.84	7.06	2.53	.01	0.46
Time	−3.32	2.24	−1.48	.14	0.27
PTCI Autocorrelation	0.86	0.06	14.68	< .001	2.64
Lagged BDI	0.15	0.26	0.56	.58	0.10
BDI on time-lagged PTCI					
	β	*SE*	*t*	*p*	*d*
Intercept	1.49	1.94	0.77	.44	0.14
Time	−1.62	0.61	−2.64	.01	0.48
BDI Autocorrelation	0.69	0.07	9.55	< .001	1.72
Lagged PTCI	0.03	0.02	2.13	.04	0.38

Note. PTCI = Post-traumatic Cognitions Inventory; BDI = Beck Depression Inventory; d = Cohen's *d*, where $d = t * \text{sqrt}(2/n)$.