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Changes in Negative Cognitions Mediate PTSD Symptom Reductions During Client-Centered Therapy and Prolonged Exposure for Adolescents

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Abstract

Objective—To assess whether changes in negative trauma-related cognitions play an important role in reducing symptoms of posttraumatic stress disorder (PTSD) and depression during prolonged exposure therapy for adolescents (PE-A).

Method—Secondary analysis of data from a randomized controlled trial comparing PE-A with client-centered therapy (CCT) for PTSD. Participants were 61 adolescent female sexual assault survivors ages 13–18 who received 8–14 weekly sessions of PE-A or CCT at a community rape crisis center. PTSD severity was assessed at baseline, mid-treatment, post-treatment, and 3-months post-treatment. Participants also completed self-report measures of negative posttraumatic cognitions and depressive symptoms at the same assessment points.

Results—Cross lag panel mediation analyses showed that change in negative trauma-related cognitions mediated change in PTSD symptoms and depressive symptoms whereas change in PTSD and depressive symptoms did not mediate change in negative cognitions.

Conclusion—Our findings support EPT and suggest that change in negative trauma-related cognitions is a mechanism of both PE-A and CCT.

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Keywords

Adolescents; negative cognitions; prolonged exposure; mechanisms; post-traumatic stress

Prolonged exposure (PE) therapy is an efficacious and effective evidence-based treatment for posttraumatic stress disorder (PTSD) and associated problems (e.g., Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). PE is derived from emotional processing theory (EPT; Foa et al., 2006; Foa & Kozak, 1986), which provides a theoretical account for the development of PTSD and the mechanisms involved in natural and therapeutic recovery from PTSD. EPT emphasizes the role of negative trauma-related cognitions about the self (e.g., I'm incompetent) and the world (e.g., the world is completely dangerous, no one can be trusted) in the development and maintenance of PTSD. According to EPT, natural or therapeutic recovery occurs when inaccurate, negative trauma-related cognitions are disconfirmed via thinking and talking about the trauma and/or approaching trauma reminders in daily life, and by realizing that thinking and confronting trauma reminders do not result in the anticipated harm (e.g., "being attacked again" or "falling apart"; Cahill and Foa, 2007). In PE, modification of these cognitions is achieved by helping patients to confront trauma-related stimuli and situations (in-vivo exposure) and to revisit and process the traumatic memory (imaginal exposure) in the absence of harm.

Several lines of research support the notion that negative trauma-related cognitions are a key mechanism of recovery from PTSD. Negative trauma related perceptions have been consistently associated with PTSD symptom severity (e.g., Foa, Tolin, Ehlers, Clark, & Orsillo, 1999; Moser, Hajcak, Simons, & Foa, 2007; Dunmore, Clark, & Ehlers, 2001) and decreases in negative cognitions were highly correlated with reductions in PTSD symptoms during PE (Foa & Rauch, 2004) and during other forms of cognitive behavioral therapy (CBT; Smith et al., 2007; Kleim et al., 2013). Moreover, reductions in negative trauma-related perceptions temporally preceded decreases in PTSD symptoms during PE among women with assault-related PTSD (Zalta et al., 2013) and individuals with comorbid PTSD and alcohol dependence (McLean, Su, & Foa, 2014), while the reverse was not true. These latter results suggest that changes in negative cognitions cause PTSD symptom reduction during treatment.

To date, no study has examined the cognitive mediation hypothesis among adolescents receiving treatment for PTSD. Given that adolescence is a unique developmental period, marked by rapid development and integration of cognitive and emotional regulation (Steinberg, 2005), it is possible that cognitive factors play a less relevant role in adolescents' recovery from PTSD than adults'. Thus, it is important to examine, rather than assume, that the mechanisms of treatment are the same across age groups. The first aim of the present study was to determine whether previous findings on the relationship between changes in negative trauma-related cognitions and changes in PTSD severity extend downward to adolescents receiving PTSD treatment. Given that PE has been found effective in reducing symptoms of depression in adults (e.g., Foa et al., 2005) and in adolescents (Foa, McLean, Capaldi, & Rosenfield, 2014), and that changes in negative automatic thoughts have been found to mediate recovery from depression in adolescents receiving CBT (Kaufman, Rohde,

Seeley, Clarke, & Stice, 2005), a second aim of this study was to examine the relationship between changes in negative trauma-related cognitions and changes in depressive symptoms during PE.

Following the findings with adults, we hypothesized that: 1) change in negative cognitions would mediate change in PTSD symptoms during PE treatment; and 2) change in negative cognitions would also mediate change in depressive symptoms during PE treatment.

Method

Participants

The current study analyzed data from the original RCT described in Foa et al. (2013). Participants were 61 adolescent girls between the ages of 13 and 18 (M age = 15.3, SD = 1.5) who were seeking treatment at a rape crisis center in Philadelphia, Women Organized Against Rape (WOAR). For inclusion in the study, participants must have a primary DSM-IV diagnosis of current or subthreshold (i.e., 1 re-experiencing symptom, 2 avoidance symptoms, 2 arousal symptoms) PTSD resulting from sexual abuse that had occurred 3 or more months prior to intake. Participants were excluded if they met criteria for current suicidal ideation with intent, another disorder that is primary relative to PTSD (e.g., psychotic disorder, thought disorder, conduct disorder, alcohol or substance dependence disorder), or pervasive developmental disorder. Participants were also excluded if they initiated psychotropic medication within the previous 12 weeks or were receiving inpatient psychiatric treatment. See Figure 1.

Procedure

Adolescents who were referred to WOAR completed an intake assessment with a WOAR counselor. Those who met study criteria were invited to participate in the study. At the baseline assessment, participants and their guardians signed consent and assent forms and completed a 2–3 hour evaluation with an independent evaluator (IE), who was a doctoral-level clinician from the University of Pennsylvania trained to assess PTSD symptom severity. Participants also completed self-report measures of PTSD symptom severity and posttraumatic cognitions. After baseline assessment, participants and their guardians attended 1–3 preparatory sessions in which case management issues (e.g., legal issues, parental involvement, interest in receiving treatment) were discussed. If any safety concerns (e.g., suicidal plans) were identified, participants were excluded from the study and given appropriate referrals. Participants were subsequently randomized to receive either PE modified for adolescents (PE-A, $n=31$) or a supportive counseling condition, Client Centered Therapy (CCT, $n=30$). Participants also completed assessments at mid-treatment (following session 7), post-treatment (following session 14), and 3-, 6-, and 12-months post-treatment. This investigation focused on the effect of changes in negative perceptions during treatment on changes in outcomes at the next assessment (see Data Analysis below); therefore we did not include the 6- and 12-month post-treatment assessments in the present analyses.

Treatment

Both study treatments were delivered by master's level therapists at WOAR who attended a 4-day PE-A training and a 2-day CCT training. Therapists received biweekly group supervision. New participants were assigned to counselors on a rotating basis and counselors would implement whichever study treatment the participant had been randomly assigned to.

Prolonged Exposure for Adolescents (PE-A) consists of up to 14 weekly 60–90 minute treatment sessions comprised of 8 developmentally-appropriate modules that utilize PE techniques typically applied to adults with PTSD. In the first phase, therapists build rapport, explain the treatment rationale, introduce breathing retraining, and provide psychoeducation about healthy sexuality and personal safety. The second phase initiates in-vivo exposure, which consists of confronting safe, trauma-related situations and is completed as homework assignments. During sessions, therapists conduct imaginal exposure, which involves repeated revisiting and recounting of the trauma memory. Participants are asked to talk or write about the trauma, and then the therapist helps them process the memory and modify negative trauma-related perceptions that contribute to maintenance of PTSD symptoms. In the final phase, therapists address relapse prevention and generalization of skills learned in treatment. For more details, see the PE-A manual (Foa, Chrestman, & Gilboa-Schechtman, 2009).

Client-Centered Therapy (CCT; Cohen & Mannarino, 1996) consists of 14 weekly 60–90 minute treatment sessions that utilize Rogerian principles such as active listening, reflection, establishment of an empathic and trusting therapeutic alliance, and unconditional support. In the first few sessions, therapists gather information about difficulties in daily functioning and explain treatment rationale to patients. The trauma is discussed briefly, and therapists provide participants with information on common reactions to sexual assault. Participants are asked to keep a diary documenting daily difficulties, feelings, and thoughts. Beginning in session 5, participants are asked to direct the content of sessions and to discuss any trauma- or nontrauma-related difficulties. Therapists encourage positive coping techniques and provide psychoeducation about healthy sexuality and personal safety. In the final sessions, therapists review lessons learned from treatment.

Therapists' adherence to both treatment protocols was monitored by trained adherence raters who were otherwise uninvolved in the study. Raters reviewed a randomly selected 20% of video-recorded treatment sessions and assessed adherence to essential components of each treatment and monitored protocol violations. Adherence to prescribed components of the treatments was 90.8% for PE-A and 90.5% for CCT.

Measures

Child PTSD Symptom Scale-Interview (CPSS-I; Foa et al., 2001) is a 24-item measure of PTSD symptom severity that maps on DSM-IV criteria. It is administered to children and adolescents between the ages of 8 and 18. The measure yields a PTSD total score that ranges from 0 to 51 (0–10=below threshold, 11–15=subclinical, 16–20=mild, 21–25=moderate, 26–30=moderately severe, 31–40=severe, 41–51=extremely severe), as well as three subscores for re-experiencing, avoidance, and hyperarousal symptoms. The measure has high internal

consistency (Cronbach's $\alpha=.83-.89$; $\alpha=.84$ in the present sample at baseline). Test-retest reliability (.84-.86), and convergent and divergent validity are also high (Foa et al., 2001).

Child Post-Trauma Attitudes Scale (C-PTAS; Johnson et al., 1996) is a 30-item self-report measure of negative post-traumatic cognitions developed by Foa and colleagues. It is administered to children and adolescents ages 8 and above who have experienced traumatic events. The items measure three subgroups of negative post-traumatic cognitions, including dangerousness (e.g. "It is important to be on guard all the time"), incompetence (e.g. "I can't deal with everyday problems anymore"), and isolation (e.g. "I have people in my life who really care about me"). Items are rated on a Likert scale that ranges from 0 (Don't agree) to 4 (Totally agree). The measure yields a total score that ranges from 0 to 120. The CPTAS has adequate one-month test-retest reliability ($r=.66$), convergent validity with measures of PTSD and depression ($r=.46$; $r=.55$), discriminant validity with measures of overall functioning ($r=.11$), and the Cronbach's α in the present sample was .75 (Rosenfield & Foa, 2015).

The Children's Depression Inventory (CDI; Kovacs, 1985) is a 27-item self-report measure of depression severity. It is administered to children and adolescents ages 7 to 17. The measure yields a total score that ranges from 0 to 54 (0-13, no depressive disorder; 14-19, possible depressive disorder; 20-54, depressive disorder). It has good internal consistency (Cronbach's α at baseline=.83) and test-retest reliability (Smucker, Craighead, Craighead, & Green, 1986).

Data Analysis

We used longitudinal cross lag panel mediation analyses (see Figure 2) to assess possible bi-directional mediation between changes in CPTAS and changes in outcomes (CPSS and CDI) over time (see Olatunji et al., 2013). In these analyses, the mediator and the outcome at each time point (baseline, mid-treatment, and post-treatment) predict both the mediator and the outcome at the next time point (mid-treatment, post-treatment, and 26-week follow-up). Unlike typical cross-sectional mediation analyses (e.g., Baron & Kenny, 1986) and concurrent (as opposed to lagged) longitudinal mediation analyses (e.g., MacKinnon, 2008), this approach separates mediator-to-outcome effects from outcome-to-mediator effects (i.e., mediation vs. "reverse" mediation), allowing each effect to be examined individually. Thus, cross lag panel mediation analysis provides more accurate estimates of the mediated pathway.

Analyses were performed using multilevel modeling (MLM) which includes all subjects regardless of missing data, hence increasing power and generalizability. We examined whether the within-subject change in outcomes over time (Time) was mediated by within-subjects changes in CPTAS over time. Since change over time was curvilinear (improvement slowing over time), we used the square root of time (in weeks) as our Time variable, which yielded models that had lower AICs and BICs than linear time models. We performed two bi-directional mediation analyses: one between CPTAS and CPSS, and one between CPTAS and CDI (Figures 2 and 3, respectively). Since we expected that participants in PE-A would have greater changes in CPTAS than participants in CCT, we

expected moderated mediation. Hence, we tested for moderation of the path from Time to CPTAS, and if present (indicating moderated mediation), we calculated mediation separately for those given PE-A and those given CCT, following Tein, Sandler, MacKinnon, and Wolchik (2004). Mediation tests were conducted using the program PRODCLIN, which calculates the 95% confidence interval for the mediated pathway. If the 95% CI for the mediated pathway does not include 0, mediation is significant. For each significant mediated pathway, we also calculated the proportion of the total effect of treatment on outcome that was accounted for by the mediator (the proportion mediated [PM] = $a*b/c$, where “c” is the total effect of treatment on outcome; Shrout & Bolger, 2002).

Results

Overall Analyses

We obtained data from participants at 225 (92%) out of a possible 244 total assessments (pre-, mid-, post-treatment and 3-month follow-up assessments for 61 participants). As expected, outcomes 3-months post-treatment were superior in PE-A compared to CCT across all three measures, $b = 11.70$, $t(123) = 5.19$, $p < .001$ for CPSS, $b = 6.69$, $t(86) = 3.39$, $p = .001$ for CDI, and $b = 10.67$, $t(112) = 3.59$, $p < .001$ for CPTAS. In addition, the slopes of improvement were faster in PE-A than in CCT for all three measures, $b = 5.71$, $t(120) = 4.73$, $p < .001$ for CPSS, $b = 2.54$, $t(68) = 2.45$, $p = .017$ for CDI, and $b = 5.64$, $t(90) = 3.70$, $p < .001$ for CPTAS. Since these slopes are the “a” paths in the mediation analyses, the “a” paths were moderated by treatment condition, indicating moderated mediation (Tein et al., 2004). Hence, we tested the mediated pathways separately for the two treatment conditions.

Mediation Analyses

The Relationship between Changes in CPTAS and Changes in CPSS over Time—Cross lag panel mediation analyses showed that, as expected, $CPTAS_t$ predicted $CPSS_{t+1}$ at the next assessment, controlling $CPSS_t$ at the previous assessment (see Figure 2), $b = .14$, $t(157) = 2.27$, $p = .025$ (this “b” path did not differ by treatment condition). Since the effect of Time on $CPTAS_t$ was significant for those given PE-A (Figure 2) and the effect of $CPTAS_t$ on $CPSS_{t+1}$ was significant, CPTAS was a significant mediator of the effect of Time on CPSS for those in PE-A, $a*b = -1.08$, 95% CI: $[-.138, -2.139]$, $P_M = .26$ (i.e., CPTAS partially accounted for the within-subjects change in CPSS over time for those receiving PE-A). The effect of Time on $CPTAS_t$ was also significant for those receiving CCT, though significantly smaller than for PE-A, $b_{diff} = 5.64$, $t(90) = 3.70$, $p < .001$ (see Figure 2). Thus, CPTAS was a trend-level (marginally significant) mediator ($p < .07$) of the effect of Time on CPSS for those in CCT, $a*b = -.29$, 93.5% CI: $[-.001, .715]$, $P_M = .11$. The proportion of the total treatment effect on CPSS that was mediated by CPTAS was 11% in CCT and 26% in PE-A.

In contrast, $CPSS_t$ was not significantly related to $CPTAS_{t+1}$ at the next assessment when controlling for $CPTAS_t$ as the previous assessment (Figure 2). Hence, none of the “reverse” mediation pathways through $CPSS_t$ to $CPTAS_{t+1}$ were significant.

The Relationship between Changes in CPTAS and Changes in CDI over Time

—Mediation analyses indicated that, as expected, $CPTAS_t$ was a significant predictor of CDI_{t+1} at the next assessment, controlling for CDI_t at the previous assessment (Figure 3), $b = .12$, $t(111) = 2.22$, $p = .028$ (this “b” path also did not differ by treatment condition). Further, because the effect of Time on $CPTAS_t$ was significant for those given PE-A, and because $CPTAS_t$ was related to CDI_{t+1} , the mediated pathway from Time to CDI_{t+1} through $CPTAS_t$ was significant for those given PE-A, $a*b = -.92$, 95% CI: $[-.120, -1.831]$, $P_M = .64$. For those given CCT, the effect of Time on $CPTAS_t$ was also significant, though smaller than in PE-A, $b_{diff} = 5.64$, $t(90) = 3.70$, $p < .001$ as noted above. Thus, the mediated pathway from Time to CDI_{t+1} through $CPTAS_t$ trended towards significance ($p < .07$) for those given CCT, $a*b = -.25$, 93.5% CI: $[-.001, -.613]$, $P_M = .18$. The proportion of the total treatment effect on CDI that was mediated by CPTAS was 18% in CCT and was 64% in PE-A.

Figure 3 also shows that CDI_t was not a significant predictor of $CPTAS_{t+1}$ at the next assessment, so none of the mediated pathways through CDI_t to $CPTAS_{t+1}$ was significant.

Discussion

This study investigated a key assumption of emotional processing theory for PTSD (Foa et al., 2006; Foa & Kozak, 1986), namely, that reductions in negative trauma-related cognitions are a key driver of recovery from PTSD. This is the first study to show that change in negative trauma-related cognitions is a mechanism of PE in an adolescent-only sample. Our results are consistent with findings from prior adult studies that examined the relationships between negative cognitions and symptoms of PTSD (e.g., Foa et al., 1999).

The pattern of findings was similar for CCT (mediation was significant at a trend-level), indicating that reductions in negative cognitions may be an important mechanism of therapeutic recovery for a variety of PTSD interventions. This is consistent with EPT, which posits that change in negative cognitions underlies PTSD recovery in general, whether this be natural recovery or therapeutic recovery, or whether it is due to PE or to another treatment (Foa, Huppert & Cahill, 2006).

In addition to driving PTSD symptom reduction, the results indicated that changes in negative trauma-related cognitions are also responsible for improvement in depressive symptoms. Interestingly, change in negative cognitions accounted for an even greater proportion of the change in depressive symptoms as compared to PTSD symptoms. On the surface, this result appears to contrast with Zalta et al. (2013), which found that changes in negative cognitions had a stronger effect on subsequent PTSD symptoms than on subsequent depressive symptoms. The inconsistency between these findings could be explained by methodological differences. For example, Zalta et al. used the post-traumatic cognitions inventory (Foa et al., 1999), which has not been directly compared with the CPTAS and may tap slightly different types of trauma-related cognitions. In addition, Zalta et al. only included participants who received PE in their analysis, and their lag between assessments was much shorter (1 week vs. 6–12 weeks in the current study). Finally, Zalta et al. only examined the cross-lag relation between the mediator and outcome (equivalent to the “b” paths in our model), and did not calculate mediation nor the proportion mediated. Examining

only the “b” paths in our models shows that, like Zalta et al., the relation between negative cognitions and later symptoms appears stronger for PTSD symptoms ($b = .14$) than for depressive symptoms ($b = .12$), although this difference was small (about 17%) in the present study. Indeed, although the proportions mediated were greater for depressive symptoms than for PTSD symptoms, this does not imply that the mediated effect is greater for depressive symptoms than for PTSD symptoms. The mediated effect ($a*b$) was actually larger for PTSD symptoms (1.08 in PE-A) than for depressive symptoms (.92 in PE-A). The difference is that the total effect of treatment on PTSD symptoms (4.17) was larger than on depressive symptoms (1.44); hence the proportion of the total effect mediated by CPTAS was smaller for PTSD than depression symptoms. This suggests that there are other important processes by which treatment impacts PTSD symptoms besides CPTAS.

The results showed that negative cognitions mediate change in PTSD and depressive symptoms and not the other way around (symptoms mediating change in cognitions). This is important because it indicates that the relationship between negative cognitions and symptoms of PTSD and depression appears to be causal and one-directional.

In terms of the treatment specificity of these findings, as hypothesized, mediation appeared to be slightly greater in the PE than the CCT conditions (the proportion of change in CPSS and CDI that was mediated by negative cognitions was 26% and 64%, respectively, in PE, and 11% and 18% in CCT). This difference was due to the expected greater effect of PE, compared to CCT, on the mediator, CPTAS (the “a” paths) (Tein et al., 2004). The relation between CPTAS and outcome did not differ by treatment condition, indicating that, for those in CCT for whom CPTAS did change, their outcomes changed equivalently to the outcomes for those in PE.

A few limitations should be considered when interpreting these findings. First, our variables were only assessed at baseline, mid-treatment (after session 7), post-treatment, and at 3-months post-treatment. The longer lag time between assessment points probably decreased the magnitude of the lagged relations between cognitions and outcomes, and makes it difficult to monitor the precise trajectory of changes in negative trauma-related cognitions and symptoms of PTSD and depression. Second, because our sample consisted entirely of female adolescents whose PTSD was related to sexual assault, the generalizability of our findings to other samples (e.g. adolescent males) may be limited. Third, depressive symptoms and levels of negative cognitions were measured via self-report, which are vulnerable to numerous biases. Finally, because CPTAS was observed and not randomly manipulated, the causal relation between CPTAS and outcome is only suggestive, and may actually result from other third variable causes.

Despite these limitations, this study is the first to show that the previously documented relationship between changes in negative trauma-related cognitions and changes in PTSD severity extends downward to adolescents receiving PTSD treatment. The findings highlight the importance of negative trauma-related cognitions, which were found to mediate change in PTSD and depressive symptoms during PTSD treatment. Finally, given that negative trauma-related cognitions mediated change in PTSD symptoms for both PE-A and CCT, the results suggest that negative trauma-related cognitions may be a mechanism of change

across broad range of treatment modalities, a possibility that should be investigated in future research.

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Highlights

- Change in negative cognitions mediated change in PTSD and depressive symptoms
- Change in PTSD and depressive symptoms did not mediate change in negative cognitions
- The mediating effect of negative cognitions was stronger in the exposure group
- Negative cognitions are a mechanism of prolonged exposure for adolescents with PTSD

Figure 1.
Consort diagram of participant flow through the protocol.

Figure 2.

Schematic of multilevel mediation model of the relationship between PTSD symptoms and negative trauma-related cognitions during treatment.

Note. Coefficients represent unstandardized regression coefficients. Mediated Pathways in bold are significant. The treatment boxes indicate the moderating effect of treatment condition (PE vs. CCT) on the mediation paths to which its arrows points. Path coefficients above the arrows emanating from Time are for PE-A. Those below the arrows are for CCT. T CPTAS = Child Post-Trauma Attitudes Scale, CPSS = Child PTSD Symptom Scale-Self-Report.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Figure 3.

Schematic of multilevel mediation model of the relationship between PTSD symptoms and depressive symptoms during treatment.

Note. Coefficients represent unstandardized regression coefficients. Mediated Pathways in bold are significant. The treatment boxes indicate the moderating effect of treatment condition (PE vs. CCT) on the mediation paths to which its arrows points. Path coefficients above the arrows emanating from Time are for prolonged exposure therapy for adolescents (PE-A). Those below the arrows are for client-centered therapy (CCT). CPTAS = Child Post-Trauma Attitudes Scale, CDI = Children's Depression Inventory.

* $p < .05$, ** $p < .01$, *** $p < .001$.