The Relationship Between Posttraumatic and Depressive Symptoms During Prolonged Exposure With and Without Cognitive Restructuring for the Treatment of Posttraumatic Stress Disorder

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Objective: In the present study, we examined the relationship between posttraumatic and depressive symptoms during prolonged exposure (PE) treatment with and without cognitive restructuring (CR) for the treatment of posttraumatic stress disorder (PTSD).

Method: Female assault survivors (N = 153) with PTSD were randomized to either PE alone or PE with added CR (PE/CR). During treatment, bi-weekly self-report measures of posttraumatic and depressive symptoms were administered.

Results: Multilevel mediational analyses indicated that during PE, changes in posttraumatic symptoms accounted for 80.3% of changes in depressive symptoms, whereas changes in depressive symptoms accounted for 45.0% of changes in posttraumatic symptoms. During PE/CR, changes in posttraumatic symptoms accounted for 59.6% of changes in depressive symptoms, and changes in depressive symptoms accounted for 50.7% of changes in posttraumatic symptoms.

Conclusions: This pattern of results suggests that PE primarily affects posttraumatic symptoms, which in turn affect depressive symptoms. In contrast, PE/CR results in a more reciprocal relationship between posttraumatic and depressive symptoms.

Keywords: posttraumatic stress disorder, depression, prolonged exposure

Posttraumatic stress disorder (PTSD) is a common and chronic psychiatric condition with prevalence rates of 6.8% in the general population (Kessler et al., 2005) and high comorbidity rates (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Individuals with PTSD typically report elevated depressive symptoms (e.g., Erickson, Wolfe, King, King, & Sharkansky, 2001), and comorbid major depressive disorder (MDD) is common among individuals with PTSD (e.g., Brown, Campbell, Lehman, Grisham, & Mancill, 2001).

Evidence regarding the temporal relationship between posttraumatic and depressive symptoms has been mixed. Some studies suggest that posttraumatic symptoms lead to depression. For example, in the National Comorbidity Survey, 78.4% of individuals with both PTSD and MDD reported that the onset of PTSD preceded that of MDD (Kessler et al., 1995). In contrast, other studies have suggested that depressive symptoms lead to posttraumatic symptoms more consistently than vice versa (King, King, McAndie, Shalev, & Doron-LaMarca, 2009; Schindel-Allon, Aderka, Shahar, Stein, & Gilboa-Schechtman, 2010). Finally, some studies have found that PTSD and MDD develop simultaneously (e.g., Shalev et al., 1998) and that reciprocal relations exist between posttraumatic and depressive symptoms (Erickson et al., 2001).

Treatments for PTSD have been found to reduce both posttraumatic and depressive symptoms (see Harvey, Bryant, & Tarrier, 2003, for a review). Specifically, prolonged exposure (PE), a widely used treatment for PTSD, has been shown to significantly reduce posttraumatic and depressive symptoms across diverse populations and settings (Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). Research has shown that PE can alleviate posttraumatic and depressive symptoms among adults treated in academic and community settings (Feske, 2008; Foa et al., 2005), among female assault (Foa & Rauch, 2004) and rape (Resick, Nishith, Weaver, Astin, & Feuer, 2002) survivors, among veterans (Rauch et al., 2009; Tuerk et al., 2011), and among individuals previously treated with medication (Rothbaum et al., 2006).

Despite the large body of research documenting the effects of PE on both posttraumatic and depressive symptoms, little is known about the relationship between reductions in the two during PE. There are several possible ways in which posttraumatic and depressive symptoms may interact during treatment. Changes in posttraumatic symptoms may lead to subsequent changes in depressive symptoms but not vice versa. Conversely, changes in depressive symptoms may lead to subsequent changes in posttraumatic symptoms but not vice versa. Alternatively, changes in posttraumatic symptoms may lead to subsequent changes in depressive symptoms, and changes in depressive symptoms may lead to subsequent changes in posttraumatic symptoms. Finally, changes in posttraumatic and depressive symptoms may be unrelated.

Understanding the relationship between posttraumatic and depressive symptoms during PE is important as it can help us better understand how PE works and can aid in the development of more effective treatments.

To our knowledge, only one published study has examined the relationship between posttraumatic and depressive symptoms in...
the treatment of PTSD (Aderka, Foa, Applebaum, Shafran, & Gilboa-Schechtman, 2011). This study examined prolonged exposure treatment (PE) among children and adolescents, treating either previous depression and PTSD symptoms as a mediator of later PTSD or depression reduction, respectively. Aderka et al. (2011) found that changes in posttraumatic symptoms accounted for 64.1% in changes in depressive symptoms, whereas changes in depressive symptoms accounted for only 11.0% of the changes in posttraumatic symptoms. Importantly, Aderka et al. examined lagged mediation rather than contemporaneous changes, therefore establishing temporal precedence of the mediator. These findings suggest that the effects of PE follow a sequential pattern in which treatment first reduces PTSD symptoms, which leads to a reduction in depressive symptoms.

The aim of the present study was to evaluate whether the pattern of influence of PE on PTSD and depressive symptoms in adults is similar to that found in youths. Thus, we examined the relationship between posttraumatic and depressive symptoms during PE in an adult sample. Based on the findings in a pediatric sample (Aderka et al., 2011), and the fact that PE was specifically developed to target posttraumatic symptoms, we hypothesized that in PE changes in posttraumatic symptoms would account for subsequent changes in depressive symptoms to a greater extent than vice versa.

The relationship between posttraumatic and depressive symptoms also may vary across treatments. Specifically, it is possible that the addition of cognitive restructuring (CR) to PE may affect the relationship between posttraumatic and depressive symptoms. To examine this hypothesis, we explored the PTSD–depression relationship during PE with and without added CR. Due to the paucity of research on patterns of symptom change, our examination regarding potential differences between the treatments was exploratory, and we did not have explicit hypotheses about the PTSD–depression relationship in PE with added CR (PE/CR).

For both PE and PE/CR, we examined the four possible relationships between posttraumatic and depressive symptoms over time: (a) changes in posttraumatic symptoms account for changes in depressive symptoms but not vice versa, (b) changes in depressive symptoms account for changes in posttraumatic symptoms but not vice versa, (c) changes in both posttraumatic and depressive symptoms account for changes in each other, and (d) changes in posttraumatic symptoms are unrelated to changes in depressive symptoms.

Method

Participants

Participants were recruited through a variety of sources including flyers, newspaper advertisements, and referrals from police departments and victim advocacy workers. Eligible participants were adult women with a primary diagnosis of PTSD related to childhood sexual abuse or to a sexual or nonsexual assault that occurred at least 3 months prior to the evaluation. Exclusion criteria were as follows: being in an abusive relationship; current diagnosis of organic mental disorder, schizophrenia, or psychotic disorder; unmedicated, symptomatic bipolar disorder; current substance dependence; illiteracy in English; current suicidal intent or plan; and recent history of serious self-injurious behavior (e.g., cutting). For additional details, see Foa et al. (2005).

A total of 210 eligible women consented to participate; 20 of these women withdrew before being assigned a treatment condition, and 11 were removed from the study after randomization. Thus, our intention-to-treat (ITT) sample comprised 179 women. Twenty-six were assigned to waitlist (WL), 74 to PE/CR, and 79 to PE. Only individuals from the PE and PE/CR conditions (n = 153) were included in the present study. A total of 90 were treated at the Center for Treatment and Study of Anxiety in Philadelphia, and 63 were treated at the Women Organized Against Rape clinic. For flow of patients through the study and reasons for removal, see Foa et al. (2005).

Participants in the ITT sample had a mean age of 31 years and were predominately Caucasian (49.2%) or African American (43.6%), single (61.6%), with at least some college education (70.0%). Sexual assault during adulthood was the most prevalent index trauma; the average time since the index trauma was 9 years. Psychiatric comorbidity was common, with 67% of the sample having at least one comorbid Axis I disorder. The most common comorbid conditions were major depressive disorder (41.2%), social phobia (20.4%), and specific phobia (20.4%). For complete demographic information, see Foa et al. (2005).

Procedure

Evaluations. The Structured Clinical Interview for DSM–IV Axis I Disorders With Psychotic Screen (SCID-I; First, Spitzer, Gibbon, & Williams, 1995) was administered at pretreatment to establish diagnoses and assess exclusion criteria and comorbid conditions. Participants completed the PTSD Symptom Scale—Self-Report (PSS–SR; Foa, Riggs, Dancu, & Rothbaum, 1993) and the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) prior to each even-numbered therapy session (i.e., bi-weekly) to monitor treatment progress and to determine treatment termination; these repeated assessments across the course of treatment allow for tests of the current study hypotheses regarding the relation between posttraumatic and depressive symptoms over time.

Treatments. Both treatments were delivered in individual weekly sessions that lasted 90–120 min. Participants who reported at least a 70% reduction in PSS–SR scores (n = 27) ended treatment after Session 9; the remaining participants were offered three extension sessions (and 13 participants declined).1 No differences in pre-treatment demographic or clinical measures were found between those who ended treatment at Session 9 and those who received additional sessions (Foa et al., 2005). Each session began with homework review and ended with homework assignment. Brief descriptions of the treatments follow; for more detailed descriptions, see Foa, Hembree, and Rothbaum (2007) and Foa et al. (2005). The mean number of therapy sessions attended was 8.14 (SD = 3.77).

PE. Session 1 included presentation of the treatment rationale and program overview, information gathering, and breathing retraining. In Session 2, the therapist provided education about common reactions to trauma and introduced in vivo exposure to a

1 Due to this feature of the design, our sample size at Session 10 was 62 and at Session 12 was 20.
collaboratively developed hierarchy of previously avoided situations. Throughout the treatment, participants were assigned homework to approach items on the hierarchy in a systematic, gradual fashion. Imaginal exposure began in Session 3. In this procedure, participants were asked to revisit imaginatively their most distressing traumatic memory and recount it aloud in present tense, repeating the recounting as necessary to allow total revisiting of 45 min. Following imaginal exposure, therapists briefly discussed the patient’s experiences (processing). Participants were instructed to listen daily to an audio recording that was made of the recounting. Sessions 4–9 (or 12) were conducted in a similar fashion. In the final session, participants summarized what they had learned in treatment and discussed their progress. Therapists and participants also discussed future plans and what to do if the participant’s symptoms increased.

**PE/CR.** The procedures used in the PE/CR treatment were identical to PE alone with two exceptions. First, in Session 3 therapists presented the idea that posttraumatic symptoms are maintained in part by trauma-related thoughts and beliefs. Participants were taught to identify and challenge erroneous and unhelpful beliefs and were instructed to record and challenge these beliefs for homework using a daily diary. Imaginal exposure was introduced in Session 4. Second, instead of the discussion about the anxiety experienced during the imaginal and in vivo exposure, patients in the PE/CR group received about 25 min of formal CR using the Socratic method developed by Beck (Beck & Emory, 1985). Participants in PE/CR were given the same amount of exposure homework as those in PE, and they also practiced CR using their diaries.

**Treatment adherence.** Weekly supervision meetings were used to monitor adherence to the treatment protocol. In addition, using adherence manuals, 141 therapy sessions (11.5% of 1,227 sessions) were randomly selected, and videotapes were rated for fidelity to the treatment manual. Ten raters trained to conduct the adherence ratings reviewed session videotapes, rated each essential component as present or absent, and monitored for protocol violations. Of these sessions, 29 (21%) were rated independently by two raters. Interrater reliability was .88; therapists on average completed 97% of the components prescribed in the protocol.

**Measures**

The SCID-I (First et al., 1995) is a semistructured interview used to assess major Axis I disorders as well as to screen for the presence of psychotic symptoms. In the current study, it was used to assess exclusion criteria and comorbid conditions as well. Specifically, for PTSD diagnosis, the SCID-I has shown excellent inter-rater reliability (Lobbestael, Leurgans, & Arntz, 2011) and has been found to be superior to other diagnostic instruments in screening for trauma (Elhai, Franklin, & Gray, 2008).

The PSS–SR (Foa et al., 1993) is a self-report version of the PTSD Symptom Scale—Interview (PSS–I; Foa, Riggs, Dancu, & Rothbaum, 1993). It includes 17 items that measure the frequency and severity of PTSD symptoms in the past week, according to Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.; DSM-IV–TR; American Psychiatric Association, 2000) criteria. In the present study, the PSS–SR was internally consistent (Cronbach’s $\alpha = .91$) and stable over a period of 1 month ($r = .74$).

The BDI (Beck et al., 1961) is a 21-item self-report inventory measuring cognitive, affective, and physiological symptoms of depression in the past week. In the present study, the BDI was internally consistent (Cronbach’s $\alpha = .93$).

**Analytic Strategy**

Our data formed a multilevel structure. The lower level, or Level 1 data, consisted of the repeated measures that were collected during treatment sessions (i.e., posttraumatic and depressive symptoms). The Level 1 data were nested within Level 2 units (i.e., participants). This data structure is appropriate for hierarchical linear modeling (HLM) techniques (Raudenbush, 2001). HLM allows the number of observations to vary between participants and handles missing data effectively.

To examine mediation of change, we used the mixed models module of SPSS Version 18. We followed the procedures reported by Kenny, Korchmaros, and Bolger (2003), which have been previously applied to mediational analysis of anxiety and depression during treatment (Aderka et al., 2011; Moscovitch, Hofmann, Suvak, & In-Albon, 2005). As described by Kenny et al., four statistical criteria must be present in order to establish mediation: (1) The predictor variable must be significantly related to the outcome variable; (2) the predictor variable must be significantly related to the mediator; (3) when the outcome is regressed simultaneously on the predictor and mediator, the mediator must be significantly related to the outcome; and (4) the relationship between the predictor and the outcome must be significantly attenuated when the mediator is included. In order to perform a more rigorous test for mediation, we “lagged” the mediator variable in all analyses. Thus, we examined whether changes in the mediator variable at time $t$ accounted for changes in the outcome variable at time $t + 1$. Due to multiple comparisons, we set significance levels at .01 in all analyses.

**Results**

**Treatment Effects on Posttraumatic and Depressive Symptoms**

To examine whether the two treatment conditions had differential effects on posttraumatic symptoms, we conducted a repeated-measures analysis of variance (ANOVA). The independent variables were Treatment (a within-subjects, two-level variable: pre vs. post) and Condition (a between-subjects, two-level variable: PE vs. PE/CR). The dependent variable was posttraumatic symptoms (PSS–I scores). Results indicated a significant main effect for treatment, $F(1, 106) = 504.04, p < .001$, partial $\eta^2 = .83$, such that participants’ posttraumatic symptoms were significantly lower at post-treatment ($M = 10.15, SD = 9.04$) compared to pre-treatment ($M = 31.72, SD = 7.50$). The main effects for Condition and the Treatment $\times$ Condition interaction were both non-significant, $F(1, 106) = 0.26, p = .61$, partial $\eta^2 = .00, ns$; $F(1, 106) = 2.68, p = .11$, partial $\eta^2 = .03$, respectively. These results indicate that PE and PE/CR reduced posttraumatic symptoms to a similar extent.

We used a similar ANOVA model to examine reductions in depressive symptoms. Results indicated a significant main effect for treatment, $F(1, 100) = 257.25, p < .001$, partial $\eta^2 = .72$, such
that participants’ depressive symptoms were significantly lower at post-treatment ($M = 7.99$, $SD = 9.39$) compared to pre-treatment ($M = 23.72$, $SD = 8.99$). The main effects for Condition and the Treatment $\times$ Condition interaction were both non-significant, $F(1, 100) = 0.14$, $p = .71$, partial $\eta^2 = .00$; $F(1, 100) = 2.23$, $p = .14$, partial $\eta^2 = .02$, respectively, indicating that PE and PE/CR reduced depressive symptoms to a similar extent. A comprehensive examination of the differences between conditions including an intent-to-treat analysis can be found in Foa et al. (2005).

**Multilevel-Mediation Analyses**

**Rates of change.** Time was modeled using measurement number (i.e., 1, 2, 3, 4, 5, 6). Prior to running analyses, we compared a model with a linear time variable, a non-linear, natural log time variable, and a quadratic time variable. As these models were non-nested, we compared them using the Akaike information criterion (AIC; Akaike, 1987) and the deviance statistic. Results indicated that a linear model was the best-fitting model. We also examined a model with both a linear time variable and a quadratic variable, and a model with both a linear time variable and a non-linear, natural log variable. No significant non-linear effects were found (all $p$s > .05), suggesting that changes in both post-traumatic and depressive symptoms occur at a similar rate throughout treatment. Thus, we used a linear time variable in all analyses.

**Effect of time.** In the PE condition, our time variable explained 52.9% of the changes in posttraumatic symptoms and 35.7% of the changes in depressive symptoms during treatment ($r_{PTSD}^2 = .529; r_{DEP}^2 = .357$). In the PE/CR condition, this variable explained 51.7% of the changes in posttraumatic symptoms and 38.1% of the changes in depressive symptoms during treatment ($r_{PTSD}^2 = .517; r_{DEP}^2 = .381$). We found that in the PE condition, the effect size of time on posttraumatic symptoms was 3.29, and the effect size of time on depressive symptoms was 1.64. In the PE/CR condition, the effect size of time on posttraumatic symptoms was 2.00, and the effect size of time on depressive symptoms was 1.45. These effect sizes are highly similar to those reported by Foa et al. (2005).

**PE condition.** We employed a model in which time was the predictor, posttraumatic symptoms were the mediator, and depressive symptoms were the outcome. This model tested the extent to which posttraumatic symptoms mediated the effect of time on depressive symptoms. In these analyses, posttraumatic symptoms were lagged so that posttraumatic symptoms at time $t$ predicted depressive symptoms at time $t + 1$. Consistent with the guidelines of Kenny et al. (2003), we first regressed depressive symptoms on time (Path C in Figure 1 and Table 1). Results indicated that depressive symptoms significantly decreased during treatment ($\beta = -3.32$, $SE = .04$, $t = -8.06$, $p < .001$). We then regressed posttraumatic symptoms on time (Path A in Figure 1 and Table 1). Results indicated that posttraumatic symptoms also significantly decreased during treatment ($\beta = -4.48$, $SE = .05$, $t = -9.17$, $p < .001$). Next, we regressed depressive symptoms on both time and posttraumatic symptoms simultaneously. The effect of posttraumatic symptoms (Path B in Figure 1 and Table 1) was significant indicating that reductions in posttraumatic symptoms significantly predicted subsequent reductions in depressive symptoms ($\beta = .05$, $SE = .01$, $t = 7.22$, $p < .001$). The effect of time (Path $C'$ in Figure 1 and Table 1) was non-significant ($\beta = -.06$, $SE = .04$, $t = -1.54$, $p = .13$), indicating that time did not have an additional affect on depressive symptoms above the effect of posttraumatic symptoms. Thus, posttraumatic symptoms fully mediated the relationship between time and depressive symptoms. As can be seen in Figure 1 and Table 1, the original pathway between time and depressive symptoms (Path C) was reduced from –0.32 to –0.06 when the mediator (posttraumatic symptoms) was entered (Path $C'$).

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2 We examined explained variance for the time variable following Kreft and de Leeuw’s (1998) recommendations for HLM analyses. Specifically, the formula used was as follows: \[(null\ model\ error - model\ with\ time\ error)/null\ model\ error\].

3 We computed effect sizes based on Feingold’s (2009) recommendations for multilevel models. Specifically, we used the formula \[d = B(time) \times \text{number of time periods/} \text{raw pre SD}J\], which has the advantage of being in the same metric commonly used in clinical trials.
We also examined mediation using the program Proclin (MacKinon, Fritz, Williams, & Lockwood, 2007). This program provides a powerful test for mediation that can adjust for covariance between Paths A and B. We found clear evidence for mediation as the confidence interval (CI) for the indirect effect was [–1.89, –0.68] and did not include zero. Of the total effect of time on depressive symptoms, 80.3% was mediated by posttraumatic symptoms.4

We examined the reverse model in which depressive symptoms mediated the effects of time on posttraumatic symptoms. In these analyses, depressive symptoms were lagged so that depressive symptoms at time \( t \) predicted posttraumatic symptoms at time \( t + 1 \). Table 2 and Figure 2 present the results of these analyses. In the reverse model, the effect of time on posttraumatic symptoms (Path C') remained significant after regressing posttraumatic symptoms on time and depressive symptoms simultaneously, which indicates that depressive symptoms only partially mediate the effect of time on posttraumatic symptoms. The mediational effect of depressive symptoms was significant (CI [–2.00, –0.72]) and accounted for 45.0% of the total effect of time on posttraumatic symptoms. In sum, changes in posttraumatic symptoms accounted for subsequent changes in depressive symptoms to a greater extent than vice versa. This pattern of results suggests that PE may work primarily by reducing posttraumatic symptoms, which in turn reduce depressive symptoms.

**Discussion**

The present study examined the relationship between posttraumatic and depressive symptoms during PE and PE/CR for PTSD. We found that during PE, changes in posttraumatic symptoms fully mediated the effect of treatment on depressive symptoms and accounted for 80.3% of changes in depressive symptoms, whereas changes in depressive symptoms partially mediated the effect of treatment on posttraumatic symptoms, and accounted for 45.0% of changes in posttraumatic symptoms. This pattern of results suggests that the primary effect of PE is on posttraumatic symptoms which in turn affect depressive symptoms. In contrast, for PE/CR we found that posttraumatic symptoms partially mediated the effect of treatment on depressive symptoms, and vice versa; changes in posttraumatic symptoms accounted for 59.6% of changes in depressive symptoms, and changes in depressive symptoms accounted for 50.7% of changes in posttraumatic symptoms. Thus, during PE/CR posttraumatic and depressive symptoms affected each other to a similar extent.

Our findings for PE are consistent with a previous study that examined PE among children and adolescents (Aderka et al., 2011). In that study, posttraumatic symptoms fully mediated the effects of PE on depressive symptoms, whereas depressive symptoms only partially mediated the effects of PE on posttraumatic symptoms. Thus, in both pediatric and adult populations, PE appears to reduce posttraumatic symptoms which in turn reduce depressive symptoms. However, it seems that changes in posttraumatic symptoms and depressive symptoms are more closely linked in adult populations than in pediatric ones. In the present study, 80.3% of changes in depressive symptoms were accounted for by posttraumatic symptoms, compared to 64.1% among youths, and 45.0% of changes in posttraumatic symptoms were accounted for by changes in depressive symptoms, compared to 11.0% among youths (Aderka et al., 2011). This discrepancy may be the result of developmental differences, or differences in types of trauma experienced and time since the trauma. More research is necessary to

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4 We calculated percent mediation according to the guidelines of Kenny et al. (2003), which have been previously applied to mediational analyses of anxiety and depression (Aderka et al., 2011; Moscovitch et al., 2005). According to Kenny et al., the total effect in lower level mediation models is the sum of the direct effect (C'), the indirect effect (AB), and the covariance between A and B (the covariance between the ordinary-least-squares estimates for Paths A and B). Thus, the formula used to calculate percent mediation was as follows: 100 \( \times ([C' + AB + Cov(AB)] - C')/[C' + AB + Cov(AB)] \), or 100 \( \times (\text{total effect} - \text{direct effect})/\text{total effect} \).
replicate these differential findings for youths and adults in PE and to understand their causes.

The pattern of symptom change observed during PE is consistent with several theoretical accounts of the relationship between anxiety and depression. For instance, the helplessness–hopelessness theory (Alloy, Kelly, Mineka, & Clemens, 1990) posits that feelings of helplessness are primarily related to anxiety, whereas feelings of hopelessness are primarily related to depression. According to this theory, experiencing helplessness repeatedly or for long periods of time can lead to hopelessness (Alloy et al., 1990). Thus, as individuals begin to better cope with their symptoms during treatment, their helplessness is reduced, which can result in subsequent reductions in hopelessness. In support of this potential mechanism, hopelessness has been found to mediate symptom change observed during PE (Resick, 2012). Our findings are also consistent with the anhedonia model of depression, which stresses the causal role of anhedonia in the development of depression (Loas, 1996). Thus, reductions in trauma-related fear and avoidance may lead to the increased experience of pleasurable life events, thus reducing anhedonia and subsequent depression.

We observed a sequential relationship between posttraumatic and depressive symptoms in PE (posttraumatic symptoms → depressive symptoms), and a more reciprocal relationship in PE/CR (posttraumatic symptoms ↔ depressive symptoms). A possible explanation for this difference between treatments is that PE and PE/CR may affect different posttraumatic and depressive factors. The imaginal and in vivo exposure in PE may affect unique PTSD factors (e.g., fear, guilt), and this reduction may affect shared PTSD/depression factors (e.g., negative beliefs of the self and the future) and unique depression factors (e.g., depressed mood). For example, in vivo exposures are very effective at reducing the distress that a person experiences in feared situations. This reduction in distress may change the way an individual perceives himself (e.g., “I am capable of dealing with these situations”), which may in turn lead to improved mood as the person becomes more hopeful. In contrast, the more language-focused, cognitive techniques in PE/CR may more directly target shared PTSD/depression factors (e.g., negative cognitions), which subsequently lead to reductions in unique PTSD and unique depression factors. For example, challenging one’s assumptions that he or she is “weak” for having PTSD may increase one’s willingness to complete in vivo exposure exercises, and can lead to a reduction in trauma-related fear. Concurrently, challenging these assumptions can also increase self-efficacy and reduce self-blame, which can result in improved mood. While testing for these possible pathways is beyond the scope of the present article, they represent potentially fertile ground for future study.

Importantly, both PE and PE/CR produced similar improvements in symptoms (Foa et al., 2005) and similar reductions in negative cognitions (Foa & Rauch, 2004). Thus, the two treatments evidence differing patterns of symptom change but result in similar degrees of change. This finding has the potential to inform clinical research, as it suggests that adding an additional active ingredient of treatment may affect the pattern of change but not the outcome. This is consistent with many studies that fail to find differences in outcome between cognitive therapy and prolonged exposure (e.g., Foa et al., 2005; Resick et al., 2002), as well as a recent study suggesting that the two types of treatment may work through different mechanisms of change (Gallagher & Resick, 2012). Future studies should examine differences in patterns of change in addition to outcome, as this may increase our understanding of how different treatments work.

Limitations and Future Directions

The present study has several limitations. First, our sample included only female assault victims. Thus, it cannot be known based on the present data whether the same patterns of interrelations exist among males or among individuals experiencing other traumas. Future studies can examine the relationship between posttraumatic and depressive symptoms in these populations. Second, the present study did not directly assess potential mediators between posttraumatic and depressive symptoms, such as hopelessness or anhedonia, that may play a role in the relationship between them; future studies can assess these mediators during treatment. Third, we examined mediation throughout the course of treatment. However, it is possible that different mediational relationships exist in different phases of treatment (e.g., early vs. late). Future studies can examine whether the relationships found in the present study change in different phases of treatment.

Table 2
Summary of Multilevel Regression Analyses for Reverse Mediation Models

<table>
<thead>
<tr>
<th>Step</th>
<th>Path</th>
<th>Predictor variable</th>
<th>Outcome variable</th>
<th>B</th>
<th>SE</th>
<th>β</th>
<th>SE β</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>C</td>
<td>Time</td>
<td>Posttraumatic symptoms</td>
<td>−4.74</td>
<td>0.43</td>
<td>−.41</td>
<td>.04</td>
<td>−11.02</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>Time</td>
<td>Depression a</td>
<td>−2.95</td>
<td>0.47</td>
<td>−.34</td>
<td>.05</td>
<td>−6.24</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>3</td>
<td>B</td>
<td>Depression a</td>
<td>Posttraumatic symptoms</td>
<td>0.56</td>
<td>0.08</td>
<td>.05</td>
<td>.01</td>
<td>6.88</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>C’</td>
<td>Time</td>
<td>Posttraumatic symptoms</td>
<td>−2.02</td>
<td>0.42</td>
<td>−.18</td>
<td>.04</td>
<td>−4.88</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Note. PE = prolonged exposure; PE/CR = prolonged exposure with added cognitive restructuring.

a Depressive symptoms were lagged so that depressive symptoms at time t predicted posttraumatic symptoms at time t + 1.
Despite these limitations, the current study represents the first longitudinal examination of the inter-relationship between posttraumatic and depressive symptoms during treatments for PTSD among adults. These findings highlight the importance of examining patterns of symptom change in psychotherapy research and suggest that despite similar outcomes, the techniques used in PE and PE/CR may result in different patterns of change in posttraumatic and depressive symptoms.

References