Behaviour Research and Therapy 77 (2016) 68–77

Contents lists available at ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Symptom exacerbations in trauma-focused treatments: Associations with treatment outcome and non-completion

Sadie E. Larsen [a, b, *], Shannon Wiltsey Stirman [c], Brian N. Smith [d, e], Patricia A. Resick [f]

[a] Psychology Service, Clement J. Zablocki VA Medical Center, Milwaukee, WI, USA
[b] Department of Psychiatry and Behavioral Medicine, Medical College of Wisconsin, USA
[c] National Center for PTSD, VA Palo Alto Healthcare System and Stanford University, USA
[d] National Center for PTSD, VA Boston Healthcare System, USA
[e] Department of Psychiatry, Boston University School of Medicine, USA
[f] Duke University, USA

ARTICLE INFO

Article history:
Received 16 December 2014
Received in revised form
20 November 2015
Accepted 15 December 2015
Available online 18 December 2015

Keywords:
Posttraumatic stress disorder
Cognitive processing therapy
Prolonged exposure
Symptom exacerbations
Cognitive behavioral therapy

ABSTRACT

Trauma-focused treatments are underutilized, partially due to clinician concerns that they will cause symptom exacerbation or dropout. We examined a sample of women undergoing Prolonged Exposure (PE), Cognitive Processing Therapy (CPT), and a version of CPT (CPT-C) without a written trauma narrative to investigate the possibility of symptom exacerbation. Participants (n = 192) were drawn from two RCT's. Participants were administered self-report measures of PTSD symptoms (i.e., the PTSD Symptom Scale or Posttraumatic Diagnostic Scale [PSS/PDS]) and the Clinician-Administered PTSD Scale. Exacerbations were defined as increases greater than 6.15 points on the PSS/PDS. A minority of participants experienced PTSD exacerbations during treatment, and there were no significant differences across treatment type (28.6% in CPT, 20.0% in PE, and 14.7% in CPT-C). Neither diagnostic nor trauma-related factors at pre-treatment predicted symptom exacerbations. Those who experienced exacerbations had higher post-treatment PSS/PDS scores and were more likely to retain a PTSD diagnosis (both small but statistically significant effects). However, even those who experienced an exacerbation experienced clinically significant improvement by end of treatment. Further, symptom exacerbations were not related to treatment non-completion. These results indicate that trauma-focused treatments are safe and effective, even for the minority of individuals who experience temporary symptom increases.

Published by Elsevier Ltd.

Cognitive Processing Therapy (CPT) and Prolonged Exposure (PE) are extensively researched, empirically-supported, cognitive-behavioral treatments for PTSD following a variety of trauma types (Chard, Ricksecker, Healy, Karlin, & Resick, 2012; Foa, Keane, Friedman, & Cohen, 2009; Resick, Nishith, Weaver, Astin, & Feuer, 2002). They are among the most commonly used trauma-focused treatments for PTSD, and are being implemented across Veterans Affairs hospitals as frontline treatments (Karlin et al., 2010). Although both treatments have been shown to be effective, these and other trauma-focused treatments are underutilized relative to their efficacy and the prevalence of PTSD (Cook, Schnurr, & Foa, 2004; Hamblen et al., 2015; Rosen et al., 2004). For example, one study found that only 6.3% of new patients in six VA specialty PTSD clinics received CPT or PE (Shiner et al., 2013).

This discrepancy is due in part to clinician and researcher apprehensions about using such treatments. Concerns have long been raised in the literature about whether direct trauma processing may lead to exacerbations in symptoms or to increased dropout. Kilpatrick and Best (1984), for example, argued that exposure may lead to increased levels of anxiety and to higher levels of dropout among sexual assault victims. Likewise, Cloitre, Koenen, Cohen, and Han (2002) suggest that childhood sexual abuse victims may be particularly likely to have difficulties with exposure therapy, which may lead them to drop out. Pitman et al. (1991; 1996a; 1996b) presented case studies and outcome data showing that PE was effective, but then concluded that Eye Movement Desensitization and Reprocessing Therapy may be better tolerated by patients and therapists, without explicit rationale for this assertion (for a discussion, see Cahill & Frueh, 1997). Tarrier et al. (1999) presented a study comparing imaginal exposure (IE) to cognitive therapy (CT)

* Corresponding author. Behavioral Health Center, 1155 North Mayfair Road, Wauwatosa, WI 53226, USA.
E-mail address: selarsen@mcw.edu (S.E. Larsen).

http://dx.doi.org/10.1016/j.brat.2015.12.009
0005-7967/Published by Elsevier Ltd.

for PTSD, and found that there were no significant differences between the treatments. However, in their discussion they noted that more participants worsened after IE than CT (although this difference disappeared at follow-up). Devilly and Foa (2001) suggested that the authors may have conflated "worsening" with "not improving". Researchers have also been more closely examining clinician concerns as barriers to the provision of exposure-based trauma treatments in regular practice (e.g., Becker, Zayfert, & Anderson, 2004). For instance, Feeny, Hembree and Zoellner (2003) identified one of the primary "myths" about exposure treatments: "Exposure therapy leads to symptom worsening and high dropout rates" (p. 85). More recently, Cook et al. (2013) reported that some clinicians in VA residential PTSD treatment programs were reluctant to use PE or CPT due to the potential risk of symptom exacerbation or dropout.

Thus, trauma-focused treatments involving direct exposure to the trauma can be described as having a "PR problem" (Devilly & Huther, 2008; Olatunji, Deacon, & Abramowitz, 2009), and clinician attitudes toward trauma-focused therapies contribute to low utilization (Becker et al., 2004). If exacerbations are common or negatively impact outcomes or increase dropout, it will be important to understand who is likely to experience exacerbations and modify treatment guidelines accordingly. If symptom exacerbations and dropouts are not common, or if they do not ultimately have a negative impact on treatment, it is equally important to disseminate this information to professionals and the public. However, only six studies have directly examined this question to date.

A small study ($N = 20$) reported that 30% of veterans experienced symptom exacerbations in a group CBT treatment for PTSD that included IE (Mott et al., 2013). In a review of several studies, Hembree et al. (2003) found that dropout rates did not differ between active treatments for PTSD. One study examined transient depression spikes in treatment for PTSD—either sertraline or prolonged exposure—and found that 22% of participants experienced transient depression spikes but did not have significantly worse post-treatment outcomes (Keller, Feeny, & Zoellner, 2014). Another study examined whether the onset of IE, typically in session 3, was related to significant symptom exacerbations in PE (Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002). The authors compared two treatments: PE and a modified treatment in which IE started at a later point in treatment, so that any difference between the two treatments in exacerbations at session 4 could be attributed to the onset of IE. They found that there were not significantly more exacerbations at the start of exposure than in the comparable treatment (15.4% vs. 2.9%, *ns*). Further, those who experienced such exacerbations did not have significantly worse outcomes or drop out of treatment at higher rates than those who had no exacerbations.

Finally, two studies examined exacerbations from pre- to post-treatment. Ehlers et al. (2014) reported that far fewer participants experienced symptom deterioration (i.e., statistically reliable change) in active cognitive trauma treatments than on a wait list (1.6% vs. 20% on the Clinician-Administered PTSD Scale, though none experienced deterioration on a self-report PTSD measure). Jayawickreme et al. (2013) examined whether PE in four controlled clinical trials (one of which is included in this study; Resick et al., 2002) led to pre-to-post-treatment worsening (defined as a pre- to post-treatment increase larger than the standard error of the difference between two measurements). They found that PE did not lead to reliable worsening. However, examining pre-post worsening assumes gradual change over the course of treatment, and therefore it is not sufficiently sensitive to capture the short-term changes that are also important to study (Hayes, Laurenceau, Feldman, Strauss, & Cardociotto, 2007), because they may affect treatment outcome (Hayes et al., 2007) and may still be a concern to clinicians.

Thus far, the impact of other trauma-focused treatments on short-term symptom change during treatment has not been explored. By expanding on the Foa et al. (2002) study, we sought to examine symptom exacerbations in a sample of women who engaged in one of three trauma-focused treatments for PTSD: 1) PE, which involves repeated and prolonged IE to the trauma memory itself as well as *in vivo* exposures; 2) CPT, a cognitive therapy that does not involve formal exposure, but requires patients to write and read back to themselves two narrative accounts of their worst trauma; and 3) another form of CPT (CPT-C), which does not involve a written account but requires discussion of the context in which the trauma occurred and discussion of beliefs about the trauma and its impact. Thus, the examination of PE, CPT, and CPT-C allowed us to assess whether different degrees of "exposure" to traumatic memories lead to symptom increases, and to understand the impact that those increases have on treatment engagement and outcome. We examined the frequency of weekly symptom exacerbations for each treatment, as well as whether they predicted worse post-treatment outcomes or higher rates of therapy non-completion. Given concerns about the tolerability of trauma-focused treatments for people who have experienced childhood sexual abuse (Cloitre et al., 2002) or other forms of sexual trauma, people with co-morbidities, and those who are highly avoidant, we also examined whether trauma-related experiences or diagnostic and symptom-related factors would lead to increased symptom exacerbations. This study is an important step toward informing clinicians and individuals with PTSD about the potential risks and benefits of trauma-focused treatments for PTSD.

1. Method

1.1. Participants

This study combined data from two randomized controlled trials of cognitive-behavioral therapy for PTSD with female victims of interpersonal violence (Resick et al., 2008; Resick et al., 2002). The first study compared CPT, PE, and a wait-list condition; those in the wait-list were randomly assigned to either CPT or PE after 6 weeks. The second study was a dismantling study comparing full CPT to its components: a written account only condition and a cognitive therapy only condition (CPT-C). These two studies were conducted consecutively with recruitment from the same location, and thus are similar in many ways. Participants were women who had either experienced an incident of lifetime completed rape (Resick et al., 2002) or sexual or physical assault (Resick et al., 2008) at least 3 months prior, were diagnosed with PTSD using the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1990, 1995), and were stable on any medications. Exclusion criteria included current psychosis, developmental disabilities, suicidal intent, current parasuicidal behavior, current drug/alcohol dependence, illiteracy, and being in a current abusive relationship. Women with a history of substance dependence were included if and when they had been abstinent from the substance(s) for 6 months. An earlier study found no significant differences in demographic variables between the two samples (Lester, Resick, Young-Xu, & Artz, 2010).

In this study, we retained those participants who engaged in CPT, PE, or CPT-C (see below), including those in the Resick et al. (2002) study who were initially assigned to the wait list and later to either CPT or PE. Additionally, for inclusion in the current investigation, participants must have completed at least two within-therapy data points, meaning that they must have completed at least four sessions of therapy. Although this selection strategy excludes participants who dropped out early, it yields the

data needed to be able to study within-treatment symptom changes. A total of 37 participants dropped out of treatment prior to session 4 (or were missing data for at least sessions 2 and 4): 5 from CPT-C (13% of that treatment), 19 from CPT (16%), and 14 from PE (19%), (χ^2 (2, N = 229) = .772, p = .68). Of these participants, 23 completed only one or two sessions, thus dropping out before the start of imaginal exposure or written trauma narrative. PTSD symptoms at pre-treatment did not differ between those who were included in the study (M = 29.31, SD = 8.66) and those who were excluded (M = 29.54, SD = 10.16), t (226) −.141, p = .89.

The final sample of 192 (including those in PE (n = 60), CPT (n = 98), and CPT-C (n = 34)) had a mean age of 33.93 (SD = 11.08). Participants had an average of 14.51 (SD = 2.62) years of education, and 51.8% reported income under $20,000. Seventy-eight percent were Caucasian, 19% were African American, and 3% were Asian, American Indian, or endorsed "other". Forty-four percent were single, 25% were either married or cohabiting, and 30% were separated, divorced, or widowed. The average number of years since the index (worst) assault was 11.43 (SD = 11.85). The only major clinician-assessed comorbid diagnoses were Major Depressive Disorder (45%) and Panic Disorder (14%). Substance abuse/dependence was rare given our exclusion criteria: alcohol abuse (1%), alcohol dependence (1%), cannabis abuse (.5%), and cannabis dependence (.5%). Thirty-four percent had experienced childhood physical abuse (CPA), and 42% had experienced childhood sexual abuse (CSA).

We analyzed whether demographics, baseline symptoms, and comorbid diagnoses differed across the three treatments. The only significant difference was in time since assault (F (2, 187) = 3.39, p = .036), with more months since assault in CPT-C (M = 189.86, SD = 193.33) than in CPT (M = 134.86, SD = 142.07) or PE (M = 111.85, SD = 95.26; note that only PE and CPT-C were significantly different from each other; Tukey's HSD p = .028).

1.2. Measures

PTSD Symptom Scale (PSS)/Posttraumatic Diagnostic Scale (PDS). Both studies included a self-report measure of PTSD symptoms: the PSS (Foa, Riggs, Dancu, & Rothbaum, 1993) in Study 1, and its modified version, the PDS (Foa, 1995) in Study 2. These measures have been combined in previous studies (e.g., Lester, Artz, Resick, & Young-Xu, 2010; Stein, Dickstein, Schuster, Litz, & Resick, 2012). The scales are nearly identical, containing 17 items corresponding to the DSM-IV (APA, 1994) symptoms of PTSD, with very slightly different wording between measures. Participants rate frequency/severity of each symptom in the past week from 0 (*not at all or only one time*) to 3 (*5 or more times per week or almost always*). A total score is obtained by summing the scores of the 17 items. Both versions of the scale have demonstrated good reliability and validity with trauma groups (Foa, Cashman, Jaycox, & Perry, 1997; Foa et al., 1993). These measures were administered at baseline, weekly (every other session) during treatment, and at post-treatment.

Clinician-Administered PTSD Scale (CAPS; Blake et al., 1990, 1995). Both studies also used the CAPS to assess clinician-rated DSM–IV PTSD diagnosis. For each symptom, a clinician rates two separate dimensions, frequency and intensity, on a scale ranging from 0 (*never*) to 4 (*daily or almost daily*), and from 0 (*none*) to 4 (*extreme*), respectively. Items rated with a frequency of one or higher and an intensity of two or higher were considered diagnosable symptoms (Blake et al., 1995). CAPS diagnoses and symptom severity scores have demonstrated reliability and validity (Weathers, Keane, & Davidson, 2001).

Child abuse measures. Child sexual abuse was assessed with the Sexual Abuse Exposure Questionnaire (SAEQ; Rowan, Foy, Rodriguez, & Ryan, 1994). The SAEQ is a retrospective self-report measure of CSA. This study utilized the 10-item overall exposure portion of the questionnaire, in which respondents identify whether they experienced each of 10 sexual abuse events. Affirmative answers are summed to determine an overall exposure score. The overall exposure portion of the SAEQ has demonstrated reliability and validity in a treatment-seeking sample, including 2-week test–retest reliability ranging from .73 to .93 and statistically significant relationships with PTSD diagnoses and symptom severity (Rowan et al., 1994). The Physical Punishment Scale of the Assessing Environments-III (AE–III–PP; Berger, Knutson, Mehm, & Perkins, 1988) was used to assess childhood physical abuse victimization. The AE–III–PP examines the experience of punishment during childhood (before 16 years of age) with 12 true or false items. Punitive behaviors in the AE–III–PP range from mild (e.g., spanked) to physically damaging (e.g., severely beaten). A total score is computed by summing the positively endorsed items, with a higher score reflecting more physical abuse experiences. The AE–III–PP has demonstrated reliability and validity, including acceptable test–retest reliability over a 2-month period and score differences between groups with and without verified physical abuse (Berger et al., 1988; Feindler, Rathus, & Silver, 2003).

For all following analyses, CSA was examined as a continuous variable and CPA as a dichotomous variable (CPA defined as a score of 5 or higher on the AE–III–PP Scale), consistent with the scoring conventions established by the measure's creators (Berger et al., 1988; Rowan et al., 1994). For purposes of describing our sample above, however, we dichotomized both measures (CSA was dichotomized by using endorsement of any SAEQ items that indicated physical contact).

1.3. Procedure

See Fig. 1 for an overview of all three treatments, including when assessments were conducted and when exposure and narrative writing was introduced.

Assessments. Both studies received Institutional Review Board approval. In both studies, participants were briefly screened, then discussed and signed an informed consent form prior to assessment. Participants completed several questionnaires (including the PSS/PDS, SAEQ, and AE–III–PP) and were evaluated for diagnosis by a trained clinician prior to engaging in treatment. Participants then filled out the PSS/PDS once per week (every other session) prior to sessions. These self-report measures were rated for the previous week period and were used to determine whether participants experienced PTSD symptom exacerbations. Post-treatment measures were given 2 weeks following the end of treatment, at which point participants were again evaluated for diagnosis by a trained clinician who was blind to treatment status (for full information regarding assessments, please refer to the original studies: Resick et al., 2002, 2008).

Treatment. For both studies, participants engaged in twice-weekly therapy sessions that were 12 h-long sessions total (for CPT and CPT-C) or two 60-min sessions and seven 90- minute sessions total (for PE). CPT followed the manual written by Resick and Schnicke (1993), updated by Resick (2001) to include more generic wording on all forms.

CPT is a manualized protocol in which clients are taught to recognize and challenge dysfunctional cognitions about the trauma and trauma-related beliefs. The treatment starts with psychoeducation, treatment rationale, and an assignment to write about the meaning of the event. Clients are then introduced to the relationship between events, thoughts, and emotions. At the end of session 3, clients are asked to write a detailed account of their most traumatic events, and are encouraged to experience their emotions as they write the account and read it back to themselves daily. They

Base-line	Session 1	Session 2	Session 3	Session 4	Session 5	Session 6	Session 7	Session 8	Session 9	Session 10	Session 11	Session 12	Follow-up
PSS/PDS Demo-graphics Child abuse		PSS/PDS		PSS/PDS		PSS/PDS		PSS/PDS	PSS/PDS (for PE only)	PSS/PDS		PSS/PDS	PSS/PDS CAPS

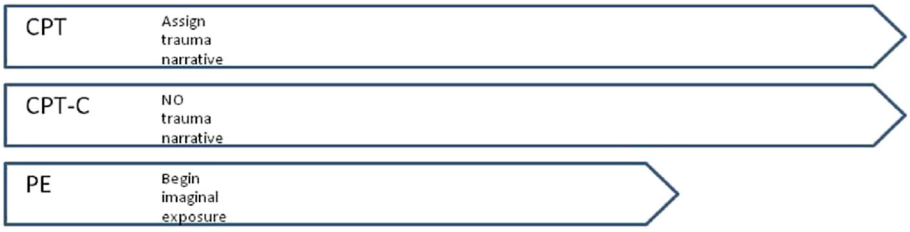

Fig. 1. Treatments with timing of assessments and beginning of exposure and narrative writing.

then read it aloud in session and are asked to repeat the process for the next session. Clients are taught to challenge unrealistic thoughts and beliefs using a series of questions and to look for unhealthy common patterns of thinking followed by generating alternative, more fact-based statements. Sessions 7 to 12 focus on thoughts and beliefs related to five themes: safety, trust, power/control, esteem, and intimacy.

The CPT-C protocol is identical, except that it excludes the written trauma account, and expands the time spent on identifying thoughts and feelings and challenging unrealistic trauma-related beliefs (thus having an equal number of sessions).

PE (Foa, Hearst, Dancu, Hembree, & Jaycox, 1994) includes four major components, starting with psychoeducation and rationale for the treatment as well as breathing retraining in the first session. Starting in the second session, clients are introduced to the subjective units of distress scale; they generate an *in vivo* exposure hierarchy and are given their first exposure homework. During sessions 3 to 9, imaginal exposure is also implemented for at least 45 min within the session, along with emotional processing of the exposure experience. Clients listen to the imaginal exposure tape and practice *in vivo* exposure daily.

1.4. Analysis plan

We used Foa et al.'s (2002) definition of symptom exacerbation, which is a change greater than 6.15 points on the PSS/PDS. This figure reflects a change greater than the standard error of the difference for this measure (i.e., the reliable change index; Jacobson & Truax, 1991). We first examined the frequency of exacerbations at any point in treatment.[1] We also replicated Foa et al. (2002) by examining frequencies of exacerbations at session 4 (i.e., a PSS/PDS score at least 6.15 points higher at session 4 compared to session 2). The assessment, which takes place just prior to session 4, is the first assessment following session 3, when imaginal exposure begins in PE and the written trauma account is assigned in CPT (but no written accounts occur in CPT-C). Therefore, if the imaginal exposure or written narrative leads to an increased likelihood of symptom exacerbation, significantly more CPT or PE than CPT-C participants would show a symptom exacerbation at the beginning of session 4. We also compared frequency of session 4 symptom exacerbations across treatments using chi-square analyses.

Next, binary logistic regression was used to examine potential predictors of session 4 symptom exacerbation. First we examined the following demographic variables as possible predictors: race/ethnicity, (dichotomized) income, marital status, and years of education. We examined several aspects of the event itself: CSA as a continuous variable, CPA as a dichotomous variable (though results did not differ when examined as dichotomous or continuous), the nature of the index trauma (physical or sexual assault), and time since index trauma (divided into fewer than 12 months, between 1 and 5 years, and more than 5 years for ease of interpretation, though results did not differ when examined as a continuous variable). Treatment type was examined as a predictor variable, with CPT and PE entered separately, each compared to CPT-C (though results did not differ when comparing to both other treatments). Diagnostic variables were dichotomous variables indicating current presence of comorbid depression, panic disorder, or alcohol use disorder. Scores on the avoidance symptom cluster of the PSS/PDS were treated as a continuous variable.

To examine whether and how symptom exacerbations would affect individual symptom trajectories (PSS/PDS) over the course of treatment, we used mixed-effects modeling. As indicated by the deviance statistic and the amount of within-subject variance accounted for, the PSS/PDS data best fit a linear rather than a curvilinear pattern. We also included CSA and CPA as level 2 predictors. Logistic regressions were used to examine whether these factors were associated with the retention of a PTSD diagnosis at post-treatment. Logistic regressions were also used to predict treatment dropout.

Finally, for further exploration and descriptive purposes, we 1) used paired-samples t-tests to specifically examine those who experienced symptom exacerbations, to see whether they showed improvement from pre- to post-treatment and 2) examined the outcomes of participants who experienced particularly large symptom exacerbations (i.e., an exacerbation two times greater than the established cutoff for clinical significance; see Foa et al., 2002).

2. Results

2.1. Symptom exacerbation frequencies: comparison across conditions

In this sample, a nontrivial minority of patients experienced a symptom exacerbation at some point in the treatment (CPT = 28.6%

[1] One person was missing data at more than half the assessment points despite being a therapy completer; this was counted as missing data. Four others were missing less than half the assessments; therefore, we counted presence or absence of exacerbations based on the data available. Sensitivity analyses revealed that excluding these four participants had no effect on the results of the basic analyses. For therapy dropouts, exacerbations were also calculated based on the data available.

Table 1
Participants who experienced an exacerbation by session.

	PE		CPT		CPT-C	
	n	%	*n*	%	*n*	%
Session 4	9	15	13	13.4	1	2.9
Session 6	2	3.6	7	7.9	1	3.1
Session 8	2	3.8	0	0	3	9.4
Session 9/10[a]	0	0	10	12	1	3.2
Session 12	N/A[b]	N/A[b]	3	3.7	0	0

Note. percentages are calculated excluding missing data. Several participants had more than one exacerbation (CPT $n = 5$, CPT-C $n = 1$, PE $n = 1$). PE = Prolonged Exposure; CPT = Cognitive Processing Therapy; CPT-C = version of CPT without written narrative.

[a] Session 9 is the final assessment session for PE; otherwise assessments were conducted at each even session.

[b] PE ends after session 9 so participants cannot have a session 10–12 exacerbation.

at any point, 13.4% at session 4; CPT-C = 14.7% at any point, 2.9% at session 4; and PE = 20.0% at any point, 15% at session 4). See Table 1 for the numbers and percentages of participants experiencing an exacerbation at each session.[2] Tests to compare the percent of session 4 exacerbations across treatments indicated non-significant differences between treatments (omnibus χ^2 (2, $N = 191$) = 3.32, $p = .19$). Given a priori interest in differences between treatments, we examined each pairwise comparison, finding non-significant trends between CPT and CPT-C: χ^2 (1, $N = 131$) = 2.89, $p = .089$, $\varphi = .15$; between PE and CPT-C: χ^2 (1, $N = 94$) = 3.32, $p = .068$, $\varphi = -.19$; and no difference between CPT and PE: χ^2 (1, $N = 157$) = .08, $p = .78$, $\varphi = -.02$. Although symptom exacerbations were more common in CPT and PE than CPT-C, this difference did not reach statistical significance (possibly due to the smaller sample size for CPT-C) and corresponded to a small effect size. The same pattern of results was found when we examined symptom exacerbations at any point during treatment (omnibus χ^2 (2, $N = 192$) = 3.28, $p = .19$). Given the lack of statistically significant differences across treatments, and in order to increase power, all further analyses utilized the full sample, including treatment type as a predictor or covariate where appropriate.

We also examined the magnitude and length of symptom exacerbations. The average increase in symptoms for those who experienced an exacerbation was 11.4 points on the PSS/PDS (range: 7–35, $SD = 5.3$). We examined how long exacerbations lasted by examining how many assessment points it took after a 6.15 point symptom exacerbation before participants experienced at least a 6.15 point symptom decrease. Most (64%) who experienced an exacerbation had a corresponding symptom decrease by the next assessment. On average, it took 1.56 assessments/weeks ($SD = 1.14$), although 9% ($n = 4$; 1 in CPT, 1 PE, and 2 CPT-C) never showed a corresponding symptom decrease. Of those who experienced a second exacerbation, all had a corresponding symptom decrease by the next assessment session.

[2] Though this manuscript focuses on within-treatment exacerbations, we separately examined exacerbations that occurred between pre-treatment assessment and session 2, thus preceding any exposure/narrative writing. 14.7% of the treatment sample experienced such an exacerbation (these are not included in the counts of exacerbations "at any point"). Of the 37 therapy participants who did not have enough data to be included in this study, 21 did not have enough data available to calculate whether they had an exacerbation; of the 16 who did, only one experienced an exacerbation. Thus, at least given the data available, early exacerbations do not appear to contribute to dropout or sample bias.

Table 2
Results of logistic regressions to examine predictors of session 4 symptom exacerbations.

Variable	Wald χ^2	*df*	*p*	*O.R.*	*95% CI*
Model 1: Demographic					
Caucasian	0.000	1	.999	1.001	0.342–2.930
Low Income	0.413	1	.520	0.677	0.206–2.223
Married/Cohabiting	0.704	1	.402	1.462	0.602–3.548
Years of Education	1.584	1	.208	0.900	0.763–1.061
Model 2: Trauma-Related					
CSA	2.983	1	.084	1.201	0.976–1.480
CPA	0.836	1	.361	0.702	0.329–1.499
Sexual Assault	1.748	1	.186	2.085	0.702–6.197
Time since index trauma	0.014	1	.907	0.958	0.470–1.955
Model 3a: Treatment-Related					
PE	0.408	1	.523	1.450	0.463–4.536
Model 3b: Treatment-Related					
CPT	2.490	1	.115	2.320	0.816–6.599
Model 4: Diagnostic and Symptom-Related					
MDD	1.104	1	.293	.749	.437–1.284
Panic	1.010	1	.315	.585	.206–1.663
Alcohol Use Disorder	3.323	1	.068	3.540	.909–13.782
Avoidance	.798	1	.372	1.056	.937–1.192

Note. Low Income = Below $30,000; CSA = Child Sexual Abuse; CPA = Child Physical Abuse, dichotomous variable; PE = Prolonged Exposure (as compared to CPT-C); CPT = Cognitive Processing Therapy (as compared to CPT-C); Sexual Assault = sexual assault at index trauma; MDD = Major Depressive Disorder.

2.2. Predictors of symptom exacerbations

Next, we examined whether we could predict who was most likely to experience session 4 symptom exacerbations. Variables that were explored included demographics, trauma-related variables, treatment, diagnostic variables, and the avoidance symptom cluster score on the pre-treatment PSS/PDS. As indicated in Table 2, none of the demographic, diagnostic or trauma-related variables examined significantly predicted exacerbations, though some were marginally significant (CSA and alcohol abuse). Receiving CPT or PE, which require a verbal or written account of the trauma, as opposed to CPT-C, which does not, did not predict exacerbations. We also examined these same variables predicting exacerbations at any point in treatment, with the same results (no significant predictors; CSA was the only variable to reach marginal significance).

2.3. Symptom exacerbations and post-treatment outcomes

Next, we examined whether symptom exacerbations had an impact on change in PSS/PDS. Linear mixed-effects modeling, with maximum likelihood estimation, was conducted to examine whether exacerbations would affect individual PSS/PDS symptom trajectories after the time of the exacerbation including all treatment types together. We included several level 2 predictors, with session 4 exacerbations being the only significant predictor of trajectory: CPA (.03, $SE = 1.49$, $p = .98$), CSA (.21, $SE = .30$, $p = .48$),[3] and session 4 exacerbations (10.17, $SE = 4.56$, $p = .03$), such that those who experienced a session 4 exacerbation continued to have a slightly higher PSS/PDS score across sessions ($M = .80$). There was also a significant interaction of exacerbations by time (−1.73, $SE = .75$, $p = .02$), but this did not appear clinically meaningful (those with a session 4 exacerbation had lower scores than those without an exacerbation at session 10 only). We also examined whether symptom exacerbations at any point in the first half of treatment predicted the slope of change over the remainder of

[3] Model results were the same regardless of whether CSA and CPA were dichotomous or continuous.

Table 3
Results of logistic regressions to examine predictors of post-treatment PTSD diagnosis (using CAPS).

Variable	Wald χ^2	*df*	*p*	*O.R.*	*95% CI*
Model 1: Session 4 Exacerbations					
Session 4 Exacerbations	3.539	1	.060	14.342	0.894–229.992
Pre-treatment PSS/PDS	3.406	1	.065	1.045	0.997–1.095
Treatment Type	0.063	1	.802	0.931	0.532–1.628
Interaction (Exacerbation × Treatment Type)	0.915	1	.339	0.425	0.074–2.452
Model 2: Any Exacerbations					
Exacerbations at Any Point	4.030	1	.045	8.017	1.051–61.175
Pre-treatment PSS/PDS	3.342	1	.068	1.046	0.997–1.097
Treatment Type	0.001	1	.976	1.010	0.525–1.942
Interaction (Exacerbation × Treatment Type)	.116	1	.734	0.810	0.241–2.723

treatment. Again, early exacerbations were the only significant predictor: CPA (.17, $SE = 1.46$, $p = .91$), CSA (.16, $SE = .29$, $p = .59$), and early exacerbations (13.56, $SE = 3.77$, $p = .00$), such that those who experienced an early exacerbation continued to have a slightly higher PSS/PDS score across sessions ($M = 2.6$). Again, the interaction of exacerbations by time (−.79, $SE = .63$, $p = .01$) was significant but not clinically meaningful (the scores of both groups were more similar at session 10 than the other sessions).

For the overall sample, the difference in post-treatment PSS/PDS between those who experienced a session 4 exacerbation ($M = 13.95$, $SD = 9.63$) and those who did not was very small ($M = 11.08$, $SD = 9.21$; $t(169) = -1.33$, $p = .19$). A difference in post-treatment PSS/PDS was found between those who experienced any exacerbation ($M = 15.53$, $SD = 10.85$) and those who did not ($M = 10.18$, $SD = 8.40$; $t(169) = -3.28$, $p = .001$). Though the latter difference is statistically significant, all post-treatment means fell within norms for a non-PTSD population ($M = 12.54$, $SD = 10.54$) rather than norms for a population with PTSD ($M = 33.59$, $SD = 9.96$; Foa et al., 1997). Moreover, differences between means of the two populations are less than the reliable change index amount (6.15 points on the PSS/PDS).

We also examined CAPS-determined PTSD diagnosis; 79% of the sample no longer had a PTSD diagnosis according to the CAPS at post-treatment (see Table 3). We used logistic regression to separately examine whether session 4 exacerbations or exacerbations at any point predicted post-treatment PTSD diagnosis. Exacerbations at session 4 were marginally significant predictors and exacerbations at any point were significant predictors of retained PTSD diagnosis at post-treatment, such that individuals who experienced exacerbations at any point were 8 times more likely to have a PTSD diagnosis at the end of treatment. The wide confidence intervals suggest some imprecision in this estimate, however, most likely driven by the relatively low number of exacerbations in the total sample, as well as the low percent of retained PTSD diagnoses. Given the inconsistency between the magnitude of the odds ratio for the exacerbation and PTSD association and our findings regarding changes in mean PTSD score, we examined the concordance between exacerbations and PTSD diagnosis to provide additional context for our results. Most (63%) of the sample experienced neither an exacerbation nor a PTSD diagnosis at post-treatment; the large observed association between exacerbation and PTSD in the logistic regression analyses appears to be explained by 11% of the sample who experienced an exacerbation and had a post-treatment PTSD diagnosis. Looked at in a different way, among those who experienced an exacerbation, 53% no longer had a PTSD diagnosis at the end of treatment. Among those who did not experience an exacerbation, 87% no longer had PTSD diagnosis at the end of treatment. Treatment type was not a significant predictor (nor was the interaction of treatment type and exacerbations).

To investigate these findings more fully, we specifically examined those participants who experienced exacerbations. Although exacerbations at any point predicted retained post-treatment PTSD diagnosis (above), those with exacerbations still showed significant improvements from pre-to post-treatment on the PSS/PDS (see Table 4). Using Cohen's *d* for paired samples t-tests, both effect sizes were large. These changes indicate clinically significant changes as well, in that both improved by over twice the reliable change index amount (6.15 points on the PSS/PDS), and both post-treatment outcomes fell within norms for a non-PTSD population ($M = 12.54$, $SD = 10.54$) rather than norms for a PTSD population ($M = 33.59$, $SD = 9.96$; Foa et al., 1997).

2.4. Symptom exacerbations and non-completion

We then examined whether symptom exacerbations led to higher rates of therapy non-completion.[4] Overall, 14.6% of this sample did not complete treatment. Logistic regressions were conducted, including the same variables used to examine PTSD diagnosis described above (see Table 5). Exacerbations (either at session 4 or at any point in treatment) did not predict non-completion. We also examined early treatment PSS/PDS scores to determine whether those who did not complete therapy had different levels of PTSD symptoms than those who did; symptom levels did not differ at pre-treatment $t(189) = 1.0$, $p = .32$, $d = .21$ or at session 2 $t(190) = .81$, $p = .81$, $d = .05$, session 4 treatment $t(189) = -.88$, $p = .38$, $d = .17$, or session 6 $t(176) = .19$, $p = .85$, $d = .05$ (all effect sizes negligible to small). Thus, symptom levels did not appear to be related to therapy non-completion.[5]

2.5. Individuals with high exacerbation of symptoms

Finally, we examined those participants who experienced an exacerbation of two times greater than the established cutoff for clinical significance (i.e., an exacerbation of 12.3 points or greater; see Foa et al., 2002). Fourteen participants in our sample experienced such an exacerbation (7% of our sample compared to 8% of the Foa et al. sample). We consider these analyses exploratory given the small sample size. Nine were in CPT, one in CPT-C, and four in PE. Of those 14, four did not complete treatment (29% vs. 16.7% in Foa et al.'s sample). This is a higher proportion than the proportion of our full sample that did not complete treatment (14.6%). A paired sample t-test comparing pre- to post-treatment PSS/PDS among

[4] Therapy non-completion here is defined as not completing the full treatment protocol. Given that participants who dropped out before session four were excluded from the study, this analysis focuses on non-completion/dropout after session four. Non-completion did not differ by treatment type (χ^2 (2, $N = 192$) = 1.61, $p = .45$).

[5] Another study using the same datasets found that timing of treatment dropout (at any point after randomization to treatment) did not differ by treatment (Gutner, Gallagher, Baker, Sloan, & Resick, in press).

Table 4
Paired Sample T-tests comparing Pre- and Post-treatment PSS/PDS in Participants with Symptom Exacerbations.

	n	*df*	*t*	*p*	*M* (pre-treatment)	*SD*	*M* (post-treatment)	*SD*	*d*
Session 4 exacerbations	21	20	6.13	.000	28.38	7.03	13.95	9.63	1.34
Exacerbations any session	39	38	8.23	.000	30.00	8.06	15.05	10.57	1.76

Note. PSS/PDS = PTSD Symptom Scale/Posttraumatic Diagnostic Scale.

Table 5
Results of logistic regressions to examine predictors of dropout.

Variable	Wald χ^2	*df*	*p*	*O.R.*	*95% CI*
Model 1: Session 4 Exacerbations					
Session 4 Exacerbations	0.168	1	.682	1.855	0.097–35.629
Pre-treatment PSS/PDS	1.173	1	.279	0.974	0.929–1.022
Treatment Type	0.625	1	.429	1.275	0.698–2.328
Interaction (Exacerbation × Treatment Type)	0.739	1	.390	0.462	0.079–2.689
Model 2: Any Exacerbations					
Exacerbations at Any Point	0.881	1	.348	2.816	0.324–24.481
Pre-treatment PSS/PDS	1.035	1	.309	0.976	0.930–1.023
Treatment Type	1.310	1	.252	1.490	0.753–2.950
Interaction (Exacerbation × Treatment Type)	2.497	1	.114	0.365	0.105–1.274

those with large exacerbations found a significant change from $M = 29.25$ at pre-treatment to $M = 14.92$ at post-treatment ($t(12) = 4.271$, $p = .001$). Thus, like all participants with an exacerbation, those with very large exacerbations were able to see significant improvements in symptoms, ending in the symptom range for a non-PTSD population (though they were also less likely to complete treatment).

3. Discussion

This study examined whether trauma-focused treatments were associated with symptom exacerbations. Results are similar to those found by Foa et al. (2002), with remarkably similar percentages of participants experiencing session 4 exacerbations in both studies, despite differences in methodology (i.e., weekly sessions in Foa et al. vs. twice weekly sessions in these studies). Further extending the literature on symptom exacerbations, we also examined symptom exacerbations at any point in treatment, along with consequences and predictors of such exacerbations in three separate treatments. In this sample, a minority of patients in CPT, PE, and CPT-C experienced an exacerbation of PTSD symptoms during treatment for PTSD. Results were generally similar across treatments with three different levels of "exposure" to a trauma memory.[6] People who experienced exacerbations were not less likely to complete treatment than those who did not experience an exacerbation, however, they showed somewhat slower rates of recovery, and were more likely to still have a PTSD diagnosis at post-treatment. Nonetheless, on average, they still experienced large, clinically- and statistically-significant changes from pre- to post-treatment and ended within normative symptom levels for a non-PTSD population. These findings thus contribute to a literature that demonstrates that trauma-focused treatments can be tolerated without lasting symptom exacerbation (Foa et al., 2002; Hembree et al., 2003; Jayawickreme et al., 2013; Keller et al., 2014). It is worth noting that many of these patients had also experienced multiple traumas, including child sexual or physical abuse, and co-occurring diagnoses (i.e. there was no exclusion for personality disorder, etc). However, neither trauma-related nor diagnostic factors were associated with a greater likelihood of symptom exacerbation. Thus, although concerns have been raised about the appropriateness of trauma-focused treatments for clients with childhood trauma (Cloitre et al., 2002) or co-occurring diagnoses, this study adds to a small literature that suggests that these individuals can tolerate and benefit from trauma-focused treatments (Resick, Suvak, & Wells, 2014).

Although exacerbations did contribute to outcomes that were less positive than those experienced by individuals who did not experience exacerbations in some cases (i.e., retained PTSD diagnosis, slightly higher average PSS/PDS scores over time), there were very small numbers of participants who experienced exacerbations overall (and small numbers with a retained diagnosis at the end of treatment). Thus, given the imprecision of the estimates, the magnitude of the increased risk is uncertain and it is important to note that even for those who experienced exacerbations, PTSD symptoms decreased from baseline by the end of treatment. Moreover, even for those who experienced symptom exacerbations, it is unclear that these are due to the trauma-focused or exposure-based nature of the treatments. For instance, symptom exacerbations occurred in a few cases in CPT-C, which does not require a written account or exposure. Additionally, several participants experienced exacerbations that were not associated with the onset of imaginal exposure or the writing of a trauma narrative. It is possible that these exacerbations were simply due to delayed emotional engagement with the trauma memory; it is also not possible to rule out other negative life events that occurred during the course of treatment. However, it is worth noting that other studies have examined symptom exacerbations and found that they can occur in more "gentle" parts of treatment. For instance, Hayes et al. (2007) examined "spikes" (a pattern of rapid symptom increases followed quickly by rapid symptom decreases) that occur during an exposure-based treatment for depression and found that spikes that happened during the exposure-activation phase were associated with cognitive and emotional processing of depression-related material. However, many participants also experienced spikes during the stress management phase of treatment that could not be attributed to cognitive or emotional processing, and may not even have been related to treatment.

Symptom exacerbations also occur in trauma treatments that are explicitly non-exposure based, so symptom increases may at

[6] Small CPT-C sample sizes may have contributed to lack of significant differences across treatment. Similar findings across treatments may also be partially explained by the fact that the three treatments involve *some* discussion of the trauma and its consequences, thus eliciting negatively valenced emotional responses and providing more adaptive means of coping with them.

times have little to do with the treatment itself. For instance, in a study of Seeking Safety, a non-exposure-based treatment for co-occurring PTSD and substance abuse, 38% of participants experienced an "adverse event" during the study (note that "adverse events" are not necessarily symptom increases; Killeen et al., 2008). Only 17% of these adverse events were study-related, including worsening PTSD, depression, anxiety, or substance use, and the rate of adverse events was the same in the "Women's Health Education" control condition that did not address trauma or PTSD. Further, Ehlers et al. (2014) found that symptom deterioration happened more frequently among patients who were on the wait list than among those in active treatment. Finally, Lilienfeld (2007) cites some studies showing that a nontrivial minority of clients (3–10%) may deteriorate in treatment in general. Thus, it appears that symptom exacerbations and other adverse events happen across studies and may sometimes be unrelated to what occurs in treatment, whether trauma-focused or not; indeed, we cannot assume that exacerbations detected at session 4 are necessarily associated with trauma exposure.

This study adds to a small body of research that directly examines the possibility that trauma-focused, especially exposure-based therapies, may be too difficult for clients or make their symptoms worse. These results, combined with Foa et al. (2002), Hembree et al. (2003) and Resick et al. (2014), should serve to reassure clinicians that symptom exacerbations are not common and, when present, are not harmful to participants. These data, like the Foa et al. (2002) study, also show that symptom exacerbations, although slightly more common during the beginning of exposure, are not significantly related to the onset of exposure exercises. Finally, it should be noted that even those who experienced exacerbations still experienced statistically and clinically significant symptom improvement. Exacerbations were slightly less common in CPT-C (though not significantly so, perhaps because of a small sample size for CPT-C and unequal sample sizes across treatments), and did not predict worse outcomes.

In considering treatment options, it is important to differentiate between therapies that produce symptom increases in the short term and those that are truly harmful (Lilienfeld, 2007). Findings from this study suggest that three trauma-focused therapies, CPT, CPT-C, and PE, appear to not be harmful, and are in fact helpful. Studies have shown that rapid improvements in PTSD symptoms are far more common than exacerbations in both CPT and PE, ranging from 40 to 50% (Aderka, Appelbaum-Namdar, Shafran, & Gilboa-Schechtman, 2011; Doane, Feeny, & Zoellner, 2010; Kelly, Rizvi, Monson, & Resick, 2009). Further, Keller et al. (2014) showed that participants in PE experienced roughly equal rates of sudden gains and spikes in depression symptoms, yet the sudden gains significantly predicted better post-treatment outcomes, whereas the spikes did not significantly predict worse post-treatment outcomes. Indeed, when clinicians are weighing whether to engage in trauma-focused treatments, they would do well to consider the potential drawbacks of not engaging in exposure-based treatments. If temporary symptom exacerbations are sometimes signs of doing important therapeutic work (Hayes et al., 2007), then they should not be seen as problematic. Clients with PTSD who have been avoiding trauma-related reminders for many years understandably may experience an increase in symptoms when they enter treatment and no longer avoid troubling material that needs to be processed to promote recovery. Moreover, trauma-focused treatments can have important consequences, such as reducing suicidal ideation (Gradus, Suvak, Wisco, Marx, & Resick, 2013), depression (Resick et al., 2002), and symptoms of "complex PTSD" (Resick, Nishith, & Griffin, 2003).

It is important to note that clinicians in the studies that have been used to examine the potential impact of exacerbations had received supervised training in trauma-focused treatments. While not all were experts, they were prepared to address traumatic memories and emotionally-laded materials with their patients. Future research should examine the potential for exacerbation when these treatments are delivered by less experienced clinicians. Further, clinicians who wish to learn and offer trauma-focused treatments should strongly consider receiving consultation on their early cases. Indeed, receiving training in empirically supported treatments helps to decrease clinician beliefs that CPE or PE will be potentially harmful (Rosen et al., 2014).

Some limitations should be borne in mind when interpreting these results. First, our analyses were of necessity limited to participants who completed several treatment sessions so as to allow for our calculating the presence or absence of symptom exacerbations. It is possible that those who dropped out early had symptom exacerbations that were not recorded. Some of the participants who left treatment early may have even done so in anticipation of trauma-focused work. In particular for PE, where exposure begins in session 3, it is possible that we were not able to detect some exacerbations due to early dropout. However, only two patients in the PE condition dropped out after session 3, when imaginal exposure began, and thus the existing data still captures most of the data regarding exacerbations after exposure or written accounts began in PE and CPT. It is also possible that some exacerbations were missed, either because they happened at a session when measures were not administered or in the gap between the end of treatment and the 2-week-post-treatment follow-up, or because participants did not attend a session at those times. Future research should administer measures at every session when possible to best assess symptom exacerbation, including early in treatment, and directly following the end of treatment. Sample sizes were small for some of our analyses, limiting our ability to test some interactions and detect small effects. In particular, CPT-C sample sizes are smaller, particularly for those who experienced exacerbations, and results should be seen as preliminary. Both Foa et al. (2002) and our study found somewhat higher rates of exacerbations at session 4 when exposure was introduced. Though these differences were non-significant, they may have been significant with a larger sample. Likewise, future research with larger sample sizes could examine time-by-treatment effects on the occurrence of exacerbations; there appear to be some differences across treatments at sessions other than the fourth, but sample sizes are too small to systematically analyze or interpret these differences. It will also be useful to examine other predictors of symptom increases, and to differentiate when symptom increases indicate necessary cognitive/emotional processing as opposed to external adverse events.

This particular sample excluded those with active substance dependence. Given that recent studies have indicated that individuals can benefit from trauma-focused treatment while receiving treatment for substance use disorder (Foa et al., 2013), the impact of current substance use disorder on symptom exacerbations during PTSD treatment will be important to investigate. Conversely, it would be useful for future studies to examine whether symptom exacerbations affect substance use, depression (only examined in one study: Keller et al., 2014), or other outcomes beyond PTSD. Finally, similar to the study conducted by Foa et al. (2002), this study was limited to mostly white female assault survivors, and may not be generalizable to other populations. An earlier study based on the same RCTs found that African-American women were less likely to complete treatment, so it is unclear how much the current results might apply in this population (Lester et al., 2010).

Despite these limitations, the current study provides some important information for clinicians and researchers interested in trauma treatment. In three separate trauma-focused treatments, a

minority of patients experienced symptom exacerbations. Although those who experienced exacerbations had somewhat slower rates of recovery, they still completed treatment, and most importantly, they still experienced large, clinically- and statistically-significant improvements from pre- to post-treatment. Particularly given that a large proportion of patients in this sample experienced multiple or repeated trauma, including childhood sexual and physical abuse, these findings should provide reassurance that patients are very unlikely to experience lasting negative effects from trauma-focused treatments, even if they experience short-term exacerbations.

Conflicts of interest

None.

Financial support

None.

Acknowledgments

This manuscript was funded by NIMH grants #1-R01-MH51509 and 2-R01-MH51509 awarded to Patricia Resick. It is partially the result of work supported with resources and the use of facilities at the Clement J. Zablocki VAMC, Milwaukee, WI.

References

Aderka, I. M., Appelbaum-Namdar, E., Shafran, N., & Gilboa-Schechtman, E. (2011). Sudden gains in prolonged exposure for children and adolescents with post-traumatic stress disorder. *Journal of Consulting and Clinical Psychology, 79*, 441–446.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Becker, C. B., Zayfert, C., & Anderson, E. (2004). A survey of psychologists' attitudes towards and utilization of exposure therapy for PTSD. *Behaviour Research and Therapy, 42*, 277–292.

Berger, A. M., Knutson, J. F., Mehm, J. G., & Perkins, K. A. (1988). The self-report of punitive childhood experiences of young adults and adolescents. *Child Abuse & Neglect, 12*, 251–262.

Blake, D. D., Weather, F. W., Nagy, L. M., Kaloupek, D. G., Klauminzer, G., Charney, D. S., et al. (1990). A clinician rating scale for assessing current and lifetime PTSD: the CAPS-1. *The Behavior Therapist, 18*, 187–188.

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., et al. (1995). The development of a clinician- administered PTSD scale. *Journal of Traumatic Stress, 8*(1), 75–90.

Cahill, S. P., & Frueh, B. C. (1997). Flooding versus eye movement desensitization and reprocessing therapy: relative efficacy has yet to be investigated—comment on Pitman et al. (1996). *Comprehensive Psychiatry, 38*, 300–303.

Chard, K. M., Ricksecker, E. G., Healy, E. T., Karlin, B. E., & Resick, P. A. (2012). Dissemination and experience with cognitive processing therapy. *Journal of Rehabilitation Research and Development, 49*, 667–678.

Cloitre, M., Koenen, K. C., Cohen, L. R., & Han, H. (2002). Skills training in affective and interpersonal regulation followed by exposure: a phase-based treatment for PTSD related to childhood abuse. *Journal of Consulting and Clinical Psychology, 70*, 1067–1074.

Cook, J. M., O'Donnell, C., Dinnen, S., Bernardy, N., Rosenheck, R., & Hoff, R. (2013). A formative evaluation of two evidence-based psychotherapies for PTSD in VA residential treatment programs. *Journal of Traumatic Stress, 26*, 56–63.

Cook, J. M., Schnurr, P. P., & Foa, E. B. (2004). Bridging the gap between post-traumatic stress disorder research and clinical practice: the example of exposure therapy. *Psychotherapy: Theory, Research, Practice, Training, 41*, 374–387.

Devilly, G. J., & Foa, E. B. (2001). The investigation of exposure and cognitive therapy: comment on Tarrier et al. (1999). *Journal of Consulting and Clinical Psychology, 69*, 114–116.

Devilly, G. J., & Huther, A. (2008). Perceived distress and endorsement for cognitive or exposure-based treatments following trauma. *Australian Psychologist, 43*, 7–14.

Doane, L. S., Feeny, N. C., & Zoellner, L. A. (2010). A preliminary investigation of sudden gains in exposure therapy for PTSD. *Behaviour Research and Therapy, 48*, 555–560.

Ehlers, A., Hackman, A., Grey, N., Wild, J., Liness, S., Albert, I., et al. (2014). A randomized controlled trial of 7-day intensive and standard weekly cognitive therapy for PTSD and emotion-focused supportive therapy. *American Journal of Psychiatry, 171*, 294–304.

Feeny, N. C., Hembree, E. A., & Zoellner, L. A. (2003). Myths regarding exposure therapy for PTSD. *Cognitive and Behavioral Practice, 10*, 85–90.

Feindler, E. L., Rathus, J. H., & Silver, L. B. (2003). *Assessment of family violence: A handbook for researchers and practitioners*. Washington, DC: American Psychological Association.

Foa, E. B. (1995). *Posttraumatic stress diagnostic scale* (manual). Minneapolis, MN: National Computer Systems.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: the posttraumatic diagnostic scale. *Psychological Assessment, 9*, 445–451.

Foa, E. B., Hearst, D. E., Dancu, C. V., Hembree, E., & Jaycox, L. H. (1994). *Prolonged exposure (PE) manual*. Unpublished manuscript. Medical College of Pennsylvania, Eastern Pennsylvania Psychiatric Institute.

Foa, E. B., Keane, T. M., Friedman, M. J., & Cohen, J. A. (Eds.). (2009). *Effective treatments for PTSD: Practice guidelines from the international society for traumatic stress studies* (2nd ed.). New York, NY: Guilford Press.

Foa, E. B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress, 6*, 459–473.

Foa, E. B., Yusko, D. A., McLean, C. P., Suvak, M. K., Bux, D. A., Oslin, D., et al. (2013). Concurrent naltrexone and prolonged exposure therapy for patients with comorbid alcohol dependence and PTSD. *Journal of the American Medical Association, 310*, 488–495.

Foa, E. B., Zoellner, L. A., Feeny, N. C., Hembree, E. A., & Alvarez-Conrad, J. (2002). Does imaginal exposure exacerbate PTSD symptoms? *Journal of Consulting and Clinical Psychology, 70*, 1022–1028.

Gradus, J. L., Suvak, M. K., Wisco, B. E., Marx, B. P., & Resick, P. A. (2013). Treatment of posttraumatic stress disorder reduces suicidal ideation. *Depression and Anxiety, 30*, 1046–1053.

Gutner, C. A., Gallagher, M. W., Baker, A. S., Sloan, D. M., & Resick, P. A. (2015). Time course of treatment dropout in cognitive-behavioral therapies for post-traumatic stress disorder. *Psychological Trauma: Theory, Research, Practice, and Policy*. http://dx.doi.org/10.1037/tra0000062 (in press), Online First Publication, June 22, 2015.

Hamblen, J. L., Bernardy, N. C., Sherrieb, K., Norris, F. H., Cook, J. M., Louis, C. A., et al. (2015). VA PTSD clinic director perspectives: how perceptions of readiness influence delivery of evidence-based PTSD treatment. *Professional Psychology: Research and Practice, 46*, 90–96.

Hayes, A. M., Feldman, G. C., Beevers, C. G., Laurenceau, J., Cardaciotto, L., & Lewis-Smith, J. (2007a). Discontinuities and cognitive changes in an exposure-based cognitive therapy for depression. *Journal of Consulting and Clinical Psychology, 75*, 409–421.

Hayes, A. M., Laurenceau, J., Feldman, G., Strauss, J. L., & Cardociotto, L. (2007b). Change is not always linear: the study of nonlinear and discontinuous patterns of change in psychotherapy. *Clinical Psychology Review, 27*, 715–723.

Hembree, E. A., Foa, E. B., Dorfan, N. M., Street, G. P., Kowalski, J., & Tu, X. (2003). Do patients drop out prematurely from exposure therapy for PTSD? *Journal of Traumatic Stress, 16*, 555–562.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: a statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59*, 12–19.

Jayawickreme, N., Cahill, S. P., Riggs, D. S., Rauch, S. A. M., Resick, P. A., Rothbaum, B. O., et al. (2013). Primum non nocere (first do no harm): symptom worsening and improvement in female assault victims after prolonged exposure for PTSD. *Depression and Anxiety, 00*, 1–8.

Karlin, B. E., Ruzek, J. I., Chard, K. M., Eftekhari, A., Monson, C. M., Hembree, E. A., et al. (2010). Dissemination of evidence-based psychological treatments for posttraumatic stress disorder in the Veterans Health Administration. *Journal of Traumatic Stress, 23*, 663–673.

Keller, S. M., Feeny, N. C., & Zoellner, L. A. (2014). Depression sudden gains and transient depression spikes during treatment for PTSD. *Journal of Consulting and Clinical Psychology, 82*, 102–111.

Kelly, K. A., Rizvi, S. L., Monson, C. M., & Resick, P. A. (2009). The impact of sudden gains in cognitive therapy for posttraumatic stress disorder. *Journal of Traumatic Stress, 22*, 287–293.

Killeen, T., Hien, D., Campbell, A., Brown, C., Hansen, C., Jiang, H., et al. (2008). Adverse events in an integrated trauma-focused intervention for women in community substance abuse treatment. *Journal of Substance Abuse Treatment, 35*, 304–311.

Kilpatrick, D. G., & Best, C. L. (1984). Some cautionary remarks on treating sexual assault victims with implosion. *Behavior Therapy, 15*, 421–423.

Lester, K., Artz, C., Resick, P., & Young-Xu, Y. (2010). Impact of race on early treatment termination and outcomes in posttraumatic stress disorder treatment. *Journal of Consulting and Clinical Psychology, 78*, 480–489.

Lilienfeld, S. O. (2007). Psychological treatments that cause harm. *Perspectives on Psychological Science, 2*, 53–70.

Mott, J. M., Sutherland, R. J., Williams, W., Lanier, S. H., Ready, D. J., & Teng, E. J. (2013). Patient perspectives on the effectiveness and tolerability of group-based exposure therapy for posttraumatic stress disorder: preliminary self-report findings from 20 veterans. *Psychological Trauma: Theory, Research, Practice, and Policy, 5*, 453–461.

Olatunji, B. O., Deacon, B. J., & Abramowitz, J. S. (2009). The cruelest cure? Ethical issues in the implementation of exposure-based treatments. *Cognitive and Behavioral Practice, 16*, 172–180.

Pitman, R. K., Altman, B., Greenwald, E., Longpre, R. E., Macklin, M. L., Poire, R. E.,

et al. (1991). Psychiatric complications during flooding therapy for posttraumatic stress disorder. *Journal of Clinical Psychiatry, 52*, 17–20.
Pitman, R. K., Orr, S. P., Altman, B., Longpre, R., Poire, R. E., & Macklin, M. L. (1996a). Emotional processing during eye movement desensitization and reprocessing therapy of Vietnam veterans with chronic posttraumatic stress disorder. *Comprehensive Psychiatry, 37*, 419–429.
Pitman, R. K., Orr, S. P., Altman, B., Longpre, R., Poire, R. E., Macklin, M. L., et al. (1996b). Emotional processing and outcome of imaginal flooding therapy in Vietnam veterans with chronic posttraumatic stress disorder. *Comprehensive Psychiatry, 37*, 409–418.
Resick, P. A. (2001). *Cognitive processing Therapy: Generic manual*. Unpublished instrument. University of Missouri—St. Louis.
Resick, P. A., Galovski, T. E., Uhlmansiek, M. O., Scher, C. D., Clum, G. A., & Young-Xu, Y. (2008). A randomized clinical trial to dismantle components of Cognitive Processing Therapy for Posttraumatic Stress Disorder in female victims of interpersonal violence. *Journal of Consulting and Clinical Psychology, 76*, 243–258.
Resick, P. A., Nishith, P., & Griffin, M. G. (2003). How well does cognitive-behavioral therapy treat symptoms of complex PTSD?: an examination of child sexual abuse survivors within a clinical trial. *CNS Spectrums, 8*, 340–355.
Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of Cognitive Processing Therapy with Prolonged Exposure and a waiting condition for the treatment of chronic Posttraumatic Stress Disorder in female rape victims. *Journal of Consulting and Clinical Psychology, 70*, 867–879.
Resick, P. A., & Schnicke, M. K. (1993). *Cognitive processing therapy for rape victims: A treatment manual*. Newbury Park, CA: Sage.
Resick, P. A., Suvak, M. K., & Wells, S. Y. (2014). The impact of childhood abuse among women with assault-related PTSD receiving short-term CBT. *Journal of Traumatic Stress, 27*(5), 558–567.
Rosen, C. S., Chow, H. C., Finney, J. F., Greenbaum, M. A., Moos, R. H., Sheikh, J. I., et al. (2004). VA practice patterns and practice guidelines for treating posttraumatic stress disorder. *Journal of Traumatic Stress, 17*, 213–222.
Rosen, C. S., Ruzek, J., Crowley, J., Eftkehari, A., Kuhn, E., & Karlin, B. (2014, November). Use of prolonged exposure therapy after completion of training. In C. S. Rosen (Ed.), *Training VA clinicians in prolonged exposure therapy: Clinician attitudes, treatment adoption, and patient outcomes. Symposium presented at the 30thAnnual Meeting of the International Society for Traumatic Stress Studies, Miami, FL*.
Rowan, A. B., Foy, D. W., Rodriguez, N., & Ryan, S. (1994). Posttraumatic stress disorder in a clinical sample of adults sexually abused as children. *Child Abuse & Neglect, 18*, 51–61.
Shiner, B., D'Avolio, L. W., Nguyen, T. M., Zayed, M. H., Young-Xu, Y., Desai, R. A., et al. (2013). Measuring use of evidence based psychotherapy for posttraumatic stress disorder. *Administration and Policy in Mental Health, 40*, 311–318.
Stein, N. R., Dickstein, B. D., Schuster, J., Litz, B. T., & Resick, P. A. (2012). Trajectories of response to treatment for posttraumatic stress disorder. *Behavior Therapy, 43*, 790–800.
Tarrier, N., Pilgrim, H., Sommerfield, C., Faragher, B., Reynolds, M., Graham, E., et al. (1999). A randomized trial of cognitive therapy and imaginal exposure in the treatment of chronic posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 67*, 13–18.
Weathers, F. W., Keane, T. M., & Davidson, J. R. T. (2001). Clinician-administered PTSD scale: a review of the first ten years of research. *Depression and Anxiety, 13*, 132–156.