Journal of Consulting and Clinical Psychology
2005, Vol. 73, No. 5, 953–964

0022-006X/05/$12.00 DOI: 10.1037/0022-006X.73.5.953

Randomized Trial of Prolonged Exposure for Posttraumatic Stress Disorder With and Without Cognitive Restructuring: Outcome at Academic and Community Clinics

Edna B. Foa, Elizabeth A. Hembree, Shawn P. Cahill, Sheila A. M. Rauch, and David S. Riggs
University of Pennsylvania

Norah C. Feeny
Case Western Reserve University

Elna Yadin
University of Pennsylvania

Female assault survivors ($N = 171$) with chronic posttraumatic stress disorder (PTSD) were randomly assigned to prolonged exposure (PE) alone, PE plus cognitive restructuring (PE/CR), or wait-list (WL). Treatment, which consisted of 9–12 sessions, was conducted at an academic treatment center or at a community clinic for rape survivors. Evaluations were conducted before and after therapy and at 3-, 6-, and 12-month follow-ups. Both treatments reduced PTSD and depression in intent-to-treat and completer samples compared with the WL condition; social functioning improved in the completer sample. The addition of CR did not enhance treatment outcome. No site differences were found: Treatment in the hands of counselors with minimal cognitive–behavioral therapy (CBT) experience was as efficacious as that of CBT experts. Treatment gains were maintained at follow-up, although a minority of patients received additional treatment.

Keywords: exposure therapy, posttraumatic stress disorder, cognitive restructuring, rape

Despite the progress that has been made in the development of efficacious psychosocial treatments for chronic posttraumatic stress disorder (PTSD; Foa, Keane, & Friedman, 2000), on the average, treated patients remain somewhat symptomatic (Cahill & Foa, 2004). For example, the mean posttreatment score of the Clinicians Administered PTSD Scale (Blake et al., 1990) was greater than 30 (a score of 20 defines remission) in the studies by Marks, Lovell, Noshirvani, Livanou, and Thrasher (1998) and Bryant, Moulds, Guthrie, Dang, and Nixon (2003). Moreover, the treatments developed in academic clinical centers are not widely used by clinicians in the community who treat patients with trauma-related disturbances (Becker, Zayfert, & Anderson, 2004). In the current study, we address these issues by examining two strategies for improving outcome and by comparing outcome in an academic center and a community clinic.

The wide range of PTSD symptoms has led some experts to suggest that treatment programs with multiple techniques will be more efficacious than any single approach (e.g., Kilpatrick, Veronen, & Resick, 1982). Accordingly, most cognitive–behavioral therapy (CBT) programs for PTSD include several techniques (e.g., Blanchard et al., 2003). Foa, Dancu, et al. (1999) examined the hypothesis that prolonged exposure (PE) combined with stress inoculation training (SIT) would be superior to either PE or SIT alone. Contrary to the predictions, the three treatments performed equally well on most measures. PE alone, which focuses on exposure to trauma-related memories and situations, was superior on anxiety and global social adjustment and yielded larger effect sizes on severity of PTSD, depression, and anxiety at posttreatment and follow-up. The authors suggested that the combined treatment put an excessive demand on patients (SIT alone involved seven techniques) and thus attenuated its potential superiority. The first aim of this study was to compare the efficacy of PE alone with a program that combined PE plus cognitive restructuring (PE/CR), a potent technique for ameliorating anxiety disorders.

Customarily, CBT programs used in research include a fixed number of treatment sessions. In typical clinical practice, however, treatment duration is determined by the patient's response to

Edna B. Foa, Elizabeth A. Hembree, Shawn P. Cahill, Sheila A. M. Rauch, David S. Riggs, and Elna Yadin, Department of Psychiatry, University of Pennsylvania; Norah C. Feeny, Department of Psychiatry, Case Western Reserve University.

Sheila A. M. Rauch is now at the Psychiatry Service, Veterans Affairs Medical Center, Ann Arbor, Michigan.

This study was supported by National Institute of Mental Health Grant MH42178 awarded to Edna B. Foa. We wish to acknowledge our colleague Constance V. Dancu, who supervised the therapists of Women Organized Against Rape throughout the study. We thank David M. Clark, who provided training and consultation in cognitive restructuring. We also thank the study therapists: Lee Fitzgibbons, Lisa Jaycox, Kelly Johnson, Ann Gaulin, Deborah Callahan, Latonda Redick, Jane Folk, Katherine Rehm, and Mary Long.

Correspondence concerning this article should be addressed to Edna B. Foa, Center for the Treatment and Study of Anxiety, Department of Psychiatry, University of Pennsylvania, 3535 Market Street, Suite 600N, Philadelphia, PA 19104. E-mail: foa@mail.med.upenn.edu

treatment. The second aim of the study was to examine whether additional sessions would enhance outcome for patients who did not reach an excellent response after eight sessions.

The vast majority of knowledge about the efficacy of CBT for PTSD has been derived from studies conducted in academic centers, but CBT has not been commonly used in community clinics that specialize in treating trauma survivors, such as rape counseling centers. Becker et al. (2004) found that most community therapists do not use imaginal exposure with PTSD sufferers primarily because they lack training. Thus, the third aim of the study was to provide training in the use of PE and CR to rape counselors and to compare the outcome of patients treated by these counselors with those treated in the academic center where the PE protocol was developed.

To achieve the aims of the study, we enrolled women with chronic PTSD resulting from rape, nonsexual assault, or childhood sexual abuse in a treatment outcome study. Enrollment was either through the Center for the Treatment and Study of Anxiety (CTSA), an academic center, or through Women Organized Against Rape (WOAR), a Philadelphia community clinic for rape survivors where therapists had no prior experience with CBT. Participants at each site were randomly assigned to PE alone, PE/CR, or wait-list (WL) control. Participants in active treatment who, at the end of eight sessions, reached at least 70% improvement in self-reported PTSD symptoms completed treatment after Session 9. The rest were offered up to 12 sessions. We hypothesized the following: (a) There would be greater reduction in PTSD, depression, and social and work dysfunction in PE and PE/CR than in WL; (b) PE/CR would be superior to PE alone on all four measures; (c) Outcome at the CTSA would be superior to that at WOAR on all four measures; (d) Participants who failed to achieve excellent response on self-reported PTSD at Session 8 would further improve after additional sessions; and (e) Participants in active treatment would maintain their gains at follow-up on all four measures.

Method

Participants

Participants were referred by police departments, victim advocacy workers, and other professionals, or they were recruited through advertisements in city newspapers and flyers. Recruitment occurred between January 1995 and September 2000. Eligible participants were adult women with a primary diagnosis of PTSD related to a sexual or nonsexual assault that occurred at least 3 months prior to the evaluation or to childhood sexual abuse (i.e., the index trauma). Exclusion criteria were as follows: being in an abusive relationship; current diagnosis of organic mental disorder, schizophrenia, or psychotic disorder; unmedicated, symptomatic bipolar disorder; substance dependence; and illiteracy in English. Women deemed at high risk for suicidal behavior (i.e., with intent or plan or both) or with recent history of serious self-injurious behavior (i.e., cutting) were also excluded. Women taking psychiatric medication (e.g., antidepressants) were required to have been on a stable dose for at least 3 months prior to entry, and they were asked to maintain this regimen during treatment.

Figure 1 summarizes participants' flow from the intake evaluation to treatment completion. A total of 285 women were evaluated. Fifty-six did not meet study criteria, 8 were eligible but not interested in the study, and 11 were lost to contact after evaluation but prior to consenting. Of the 210 eligible women who signed consent, 20 withdrew before being assigned a treatment condition, and 11 were removed from the study after randomization. Thus, our intent-to-treat sample consisted of 179 women who signed consent, were randomized to a condition, and were not removed by the investigators. Twenty-six were assigned to WL, 74 to PE/CR, and 79 to PE. A total of 105 were treated at the CTSA, and 74 were treated at WOAR.

Treatment completers were 121 participants (44 in PE/CR and 52 in PE) who attended at least eight therapy sessions and completed a posttreatment assessment. Twenty-five participants in the WL condition completed a posttreatment assessment. The overall dropout rate was 32.4% and was lower for WL (3.8%) than PE/CR (40.5%), $\chi^2(1, N = 100) = 12.1, p < .001$, and PE (34.2%), $\chi^2(1, N = 105) = 9.2, p < .01$. Dropout rates did not differ between the two treatments, $\chi^2(1, N = 153) < 1$, *ns*, or across sites (CTSA = 33.3% and WOAR = 31.1%), $\chi^2(1, N = 179) < 1$, *ns*.

Demographic information for the intent-to-treat sample is summarized in Table 1. Participants had a mean age of 31 years and were predominately Caucasian or African American, single, with at least some college education. Nearly half of the participants reported annual incomes of $15,000 or less, and one third were not working or on disability. Sexual assault during adulthood was the most prevalent index trauma, defined as the one experienced by the patient as currently most distressing or most frequently reexperienced or both. The average time since the index trauma was 9 years. Almost all participants either witnessed or directly experienced at least one traumatic event in addition to the index trauma, and more than 80% directly experienced at least one additional incident of interpersonal violence. Psychiatric comorbidity was common, with 67% of the sample having at least one comorbid Axis I disorder. The most common comorbid conditions were as follows: major depression (41.2%), social anxiety disorder (20.4%), specific phobias (20.4%), generalized anxiety disorder (13.9%), and panic disorder (11.9%). All other disorders were present at rates of 6% or less.

Significant site differences were found on five demographic variables: age, index trauma, relationship status, employment, and overall comorbidity, although sites did not differ on any specific disorder. There was also a trend for a difference in ethnicity (see Table 1).

Completers differed from noncompleters on level of education, $\chi^2(4, N = 177) = 11.8, p < .05$, being more likely to have a bachelor's degree or higher (34% vs. 12%) and less likely to have not completed high school (8% vs. 17%). There were trends for completers to be older ($M = 32.2$, $SD = 9.7$) than noncompleters ($M = 29.3$, $SD = 10.0$), $t(176) = 1.9, p = .064$, and to be employed full time (43% vs. 33%) or to be students (22% vs. 14%) rather than unemployed (17% vs. 35%), $\chi^2(4, N = 176) = 7.9, p < .096$. There was a trend for completion rates to differ across traumas, $\chi^2(2, N = 179) = 4.6, p = .099$, with 63% of survivors of adult rape, 76% of nonsexual assault, and 81% of childhood sexual abuse completing treatment. Notably, comorbidity, exposure to additional trauma, or direct experience of additional interpersonal violence was not associated with dropout status (all χ^2 values < 1, *ns*).

Twelve serious adverse events led to termination in the study, six of which are included in the postrandomization removal category in Figure 1 (4 participants reassaulted, 1 developing a life threatening illness, and 1 death). The remaining six serious adverse events were classified as dropouts (4 had severe depression and suicidal ideation that required immediate intervention, 2 of which were hospitalized, and 2 exhibited extreme dissociative symptoms).

Measures

Diagnostic Interview

The Structured Clinical Interview for *DSM–IV* Axis I Disorders With Psychotic Screen (First, Spitzer, Gibbon, & Williams, 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

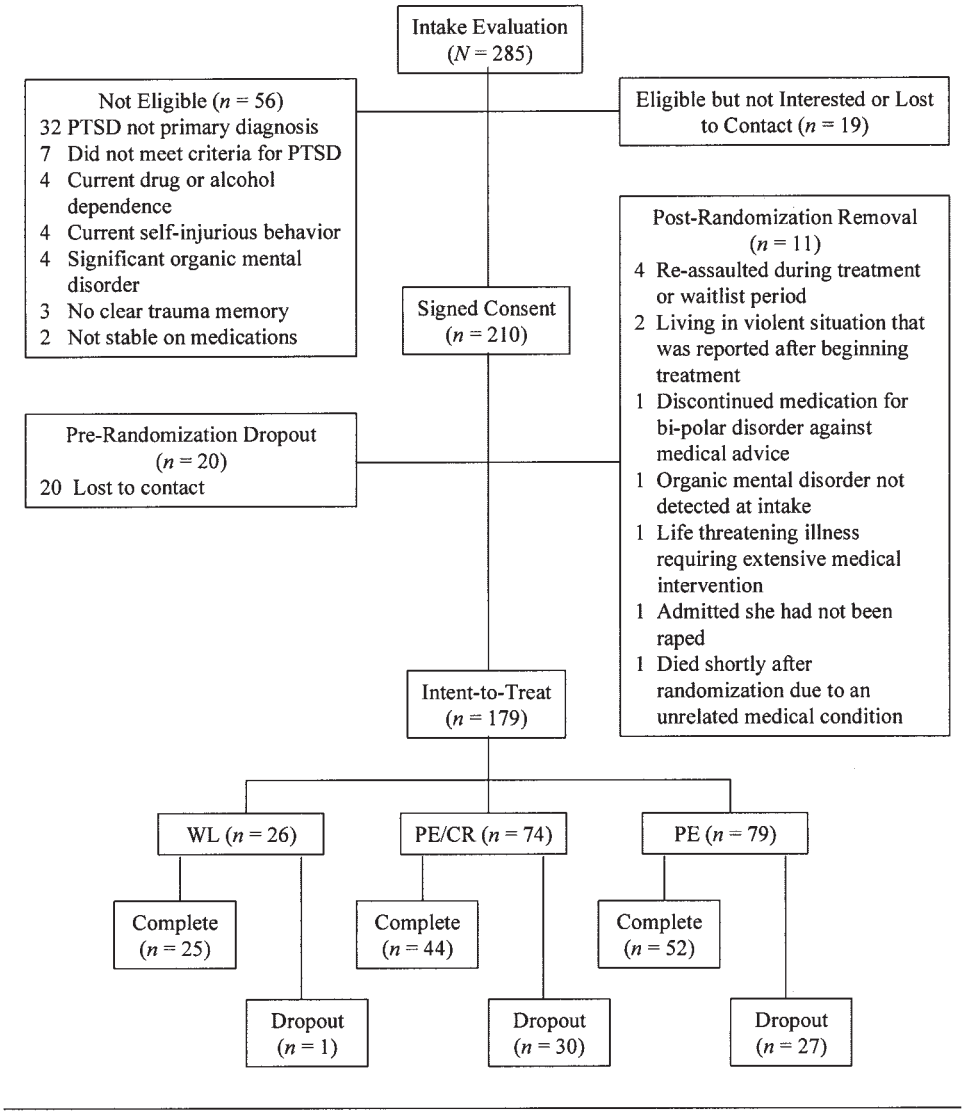

Figure 1. Flow of participants from intake evaluation through completion of treatment. PTSD = posttraumatic stress disorder; WL = wait-list; PE/CR = prolonged exposure plus cognitive restructuring; PE = prolonged exposure.

study, it was used to assess comorbid conditions and some exclusion criteria.

Primary Outcome Measure

The PTSD Symptom Scale—Interview (PSS–I; Foa, Riggs, Dancu, & Rothbaum, 1993) is a semistructured interview that consists of 17 items corresponding to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) PTSD symptoms. Items are rated on 0–3 scales for combined frequency and severity in the past 2 weeks (0 = *not at all*, 3 = *5 or more times per week/very much*). Interrater reliability for PTSD diagnosis (κ = .91) and overall severity (r = .97) are excellent (Foa et al., 1993). Of the audiotaped PSS–I interviews in the current study, 5% were randomly selected for rating by a second evaluator. The interrater reliability was .94.

Secondary Outcome Measures

Beck Depression Inventory (BDI). The BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item self-report inventory measuring depression severity. Split-half reliability was .93. Correlations with clinician ratings ranged from .62 to .66.

Social Adjustment Scale (SAS). The SAS (Weissman & Paykel, 1974) is a semistructured interview assessing functioning in eight specific areas on separate 7-point scales, with higher scores indicating more severe maladjustment. We used only the Social and Work scales.

PTSD Symptom Scale—Self-Report (PSS–SR). The PSS–SR (Foa et al., 1993) is a self-report version of the PSS–I. It is both internally consistent (α = .91) and stable over a period of 1 month (r = .74). Symptoms were rated for frequency/severity in the past week.

Table 1
Demographics of the Intent-to-Treat Sample (Total and at Each Site)

Variable	Total	CTSA	WOAR	Statistic
Age (years)				
M (*SD*)	31.3 (9.8)	33.4 (10.2)	28.2 (8.5)	$t(170.5) = 3.7$,[a] $p < .001$
N	178	105	73	
Years since index trauma				
M (*SD*)	9.0 (11.3)	8.8 (10.2)	9.3 (12.9)	$t(135) < 1.0$, *ns*
N	137	81	56	
Index trauma, *n* (%)				
Sexual assault	123 (68.7)	63 (60.0)	60 (81.1)	$\chi^2(2, N = 179) = 17.2$, $p < .001$
Nonsexual assault	25 (14.0)	24 (22.9)	1 (1.4)	
Childhood sex abuse	31 (17.3)	18 (17.1)	13 (17.6)	
N	179	105	74	
Witnessed or experienced other (nonindex) traumatic event, *n* (%)	171 (96.6)	98 (95.1)	73 (98.6)	Fisher's Exact Test, *ns*
N	177	103	74	
Experienced other interpersonal violence, *n* (%)	145 (82.9)	82 (80.4)	63 (86.3)	$\chi^2(1, N = 177) = 1.0$, *ns*
N	175	102	73	
Ethnicity, *n* (%)				
African American	78 (43.6)	40 (38.1)	38 (51.4)	$\chi^2(2, N = 179) = 5.1$, $p = .08$
Caucasian	88 (49.2)	59 (56)	29 (39.2)	
Other	13 (7.3)	6 (5.7)	7 (9.5)	
N	179	105	74	
Relationship, *n* (%)				
Single	109 (61.6)	52 (50.0)	57 (78.1)	$\chi^2(2, N = 177) = 16.6$, $p < .001$
Married/cohabiting	38 (21.5)	26 (25.0)	12 (16.4)	
Divorced/separated	30 (16.9)	26 (25.0)	4 (5.5)	
N	177	104	73	
Employment, *n* (%)				
Not working	40 (22.7)	26 (25.0)	14 (19.4)	$\chi^2(4, N = 176) = 12.1$, $p < .05$
Part time	19 (10.8)	9 (8.7)	10 (13.9)	
Full time	70 (39.8)	45 (43.3)	25 (34.7)	
Disability	13 (7.4)	11 (10.6)	2 (2.8)	
Student	34 (19.3)	13 (12.5)	21 (29.2)	
N	176	104	72	
Education, *n* (%)				
Some high school	19 (10.7)	13 (12.5)	6 (8.2)	$\chi^2(4, N = 177) = 4.9$, *ns*
High school/GED	34 (19.2)	22 (21.2)	12 (16.4)	
AA or some college	77 (43.5)	40 (38.5)	37 (50.7)	
BA/BS	25 (14.1)	13 (12.5)	12 (16.4)	
Greater than BA/BS	22 (12.4)	16 (15.4)	6 (8.2)	
N	177	104	73	
Income, *n* (%)				
Less than or equal to 15,000	82 (47.4)	48 (47.5)	34 (47.2)	$\chi^2(3, N = 173) = 2.1$, *ns*
15,001–30,000	42 (24.3)	24 (23.8)	18 (25.0)	
30,001–50,000	28 (16.2)	19 (18.8)	9 (12.5)	
Greater than 50,001	21 (12.1)	10 (9.9)	11 (15.3)	
N	173	101	72	
Any current comorbid Axis I condition, *n* (%)	103 (67.3)	55 (61.1)	48 (76.2)	$\chi^2(1, N = 153) = 3.8$, $p = .05$
N	153	90	63	

Note. CTSA = participants treated at the university-based Center for the Treatment and Study of Anxiety; WOAR = participants treated at the community-based Women Organized Against Rape; GED = general equivalency diploma; AA = Associate of Arts degree; BA = Bachelor of Arts degree; BS = Bachelor of Science degree.
[a] Degree of freedom adjusted because of unequal variances.

Procedure

Evaluations

Independent evaluations were conducted at pretreatment and posttreatment and 3-, 6-, and 12-month posttreatment. All evaluations were conducted by trained doctoral or master's level CTSA clinicians who were blind to study condition. The same evaluators conducted assessments for both the CTSA and the WOAR participants. Participants were evaluated at their treatment site. The Structured Clinical Interview for *DSM–IV* Axis I Disorders With Psychotic Screen was administered only at pretreatment; PSS–I and SAS were administered at all evaluations. At each assessment point, participants completed the PSS–SR and BDI. Participants were instructed by their therapists and the evaluators to not reveal any information that might unblind the evaluator to treatment condition. After the

9-week WL period, WL participants were offered treatment with PE or PE/CR (randomly determined), but their treatment data are not included in the current analyses. WL participants did not participate in the follow-up assessments. PE and PE/CR participants completed the PSS–SR prior to each even numbered therapy session to monitor treatment progress and to determine treatment termination.

Randomization Procedure

The study statistician assigned participants who provided informed consent to one of the three conditions using a weighted randomization procedure such that participants were assigned to one of the active treatment conditions at a greater rate than to WL. Therapists made contact with the participants and arranged initial therapy appointments with those assigned to active treatment, and they also informed them of the specific treatment condition at the first session. WL participants were informed by phone that they had been assigned to the WL condition.

Treatments

Female therapists delivered all treatments in individual sessions that lasted 90–120 min. Five clinicians with doctoral degrees in clinical psychology administered the treatments at the CTSA; six clinicians with master's degrees in counseling or social work administered the treatments at WOAR. In the startup phase of the study, all CTSA and WOAR therapists were trained together in a 5-day workshop led by Edna B. Foa and Constance V. Dancu (PE) and a second 5-day workshop led by David M. Clark (CR). Therapists were trained to use manuals that described the procedures for each session in great detail. All therapists received ongoing supervision at the therapists' site throughout the study from Edna B. Foa, Constance V. Dancu, and Elizabeth A. Hembree.

Treatment sessions were scheduled once a week. PSS–SR scores obtained at Session 8 were compared with pretreatment scores. Participants who showed at least a 70% reduction in PSS–SR scores ended treatment after Session 9. The remaining participants were offered three extension sessions. All sessions were videotaped (for supervision and treatment fidelity ratings) and audiotaped (to be reviewed by the participants for homework). Each session began with homework review and ended with homework assignment. Below is a short description of the treatments. For more detailed descriptions, see Foa and Rothbaum (1998).

PE. Session 1 included presentation of treatment rationale and program overview, information gathering, and breathing retraining. Session 2 included education about common reactions to trauma, rationale for in vivo exposure, construction of an in vivo exposure hierarchy, and initiation of in vivo homework. The hierarchy included safe or low-risk activities and situations that were avoided because of their association with the trauma. Throughout the treatment, participants were assigned homework to confront items on the hierarchy in a gradual fashion, working up to the most anxiety-arousing situations.

Session 3 included presentation of the rationale for confronting the trauma memory in imagination and initiation of imaginal exposure. In this procedure, participants were asked to close their eyes, visualize the assault, and recount it aloud in the present tense for 45–60 min. The memory recounting was repeated if necessary to allow total reliving of 45–60 min. The exposure was audiotaped; participants were instructed to listen daily to the tape.

Sessions 4–9 (or 12) were conducted in a similar fashion: Therapists reviewed homework, conducted imaginal exposure to trauma memory for 30–45 min, discussed the imaginal exposure, and assigned in vivo and imaginal exposure homework. In addition, in the final session, participants summarized what they had learned in treatment, and they discussed their progress. Therapists and participants also discussed future plans and what to do if their symptoms increased.

PE/CR. The procedure in the PE/CR treatment was identical to PE alone with two exceptions. First, Session 3 was devoted to presenting the idea that posttrauma symptoms are maintained in part by trauma-related thoughts and beliefs and to practicing CR. Specifically, participants were taught to identify and challenge erroneous and unhelpful beliefs and instructed to record and challenge them for homework using a daily diary. Imaginal exposure was introduced in Session 4, following a review of the preceding week's diaries. Second, all subsequent sessions included 30–45 min of imaginal exposure followed by 15–25 min of CR. Participants in PE/CR were given the same amount of exposure homework as those in PE, and they also practiced CR using their diaries.

WL. Participants were assigned a therapist who informed them that they would receive treatment in 9 weeks. Therapists encouraged the WL participants to call at anytime if they were having problems. In addition, the therapists called the WL participants half way through the waiting period to check in with them and determine their state.

Results

Preliminary Analyses

We first examined possible pretreatment differences on PSS–I, BDI, SAS—Work (SAS–W), and SAS—Social (SAS–S) scores across treatment groups and sites using a series of separate single factor analyses of variance (ANOVAs). No significant differences between sites emerged (all F values < 1, *ns*). A main effect for treatment condition emerged on the PSS–I, $F(2, 176) = 3.4$, $p < .05$. Pairwise comparison with t tests for independent samples revealed lower scores for PE/CR ($M = 31.1$, $SD = 8.1$) than PE ($M = 34.0$, $SD = 5.9$), $t(132.1) = 2.5$, $p < .05$, degrees of freedom (*df*) adjusted because of unequal variances. WL ($M = 33.3$, $SD = 6.2$) did not differ from PE/CR, $t(98) = 1.3$, *ns*, or PE, $t(103) < 1.0$, *ns*. These analyses were repeated on the completer sample; no differences emerged, largest $F(2, 116) = 1.4$, *ns*.

Treatment Adherence

Adherence to treatment protocol was monitored during weekly supervision meetings. Using adherence manuals, we randomly selected and rated videotapes of 141 therapy sessions (11.5% of 1,227 sessions) 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 completed 97% of the components prescribed in the protocol. Seventeen protocol violations were observed in the 141 sessions; 24% of these were observed in the CTSA sessions and 76% in the WOAR sessions.

Acute Treatment Outcome

Intent-to-Treat Analyses

We conducted separate Group (WL vs. PE/CR vs. PE) × Site (CTSA vs. WOAR) × Time (pre- vs. posttreatment) mixed facto-

rial ANOVAs on the PSS–I,[1] BDI, SAS–W, and SAS–S scores for the intent-to-treat sample, substituting pretreatment scores for missing posttreatment scores. Time was the sole within-group factor. Descriptive statistics (*M, SD*, *n*) for each measure at pre- and posttreatment are presented in Table 2. A significant main effect for group was detected on the PSS–I, $F(2, 173) = 4.0$, $p < .05$, but not on any other measure, largest $F(2, 145) = 1.1$, *ns*. There was a significant main effect of time on the PSS–I, $F(1, 173) = 134.7$, $p < .001$; BDI, $F(1, 167) = 72.4$, $p < .001$; SAS–W, $F(1, 145) = 7.2$, $p < .01$; and SAS–S, $F(1, 171) = 17.5$, $p < .001$; and Group × Time interactions on the PSS–I, $F(2, 173) = 5.6$, $p < .01$; and BDI, $F(2, 167) = 6.9$, $p < .001$; with a trend toward an interaction on the SAS–W, $F(2, 145) = 2.9$, $p = .059$; but not SAS–S, $F(2, 171) = 1.8$, *ns*. No main effects of site, largest $F(1, 173) = 3.0$, *ns*, and no interactions involving site emerged, largest $F(2, 173) = 1.7$, *ns*.[2]

Using *t* tests for independent samples, we examined the Group × Time interactions via pairwise comparisons among groups at posttreatment. PSS–I scores were higher in the WL group than in PE/CR group, $t(60.6) = 4.1$, $p < .001$, *df* adjusted because of unequal variances; and PE group, $t(65.2) = 3.6$, $p < .001$, *df* adjusted because of unequal variances. The PE/CR and PE groups did not differ from one another, $t(151) < 1.0$, *ns*. BDI scores were higher in the WL group than in the PE/CR, $t(92) = 2.4$, $p < .05$, and PE groups, $t(99) = 2.0$, $p < .05$; the PE/CR and PE groups did not differ, $t(141) < 1.0$, *ns*.

Completer Analyses

We repeated the above analyses using data from participants who completed the trial. Descriptive statistics (*M, SD*, *n*) for each measure at pre- and posttreatment assessments are presented in the Table 3. The ANOVAs revealed a main effect of group on the PSS–I, $F(2, 115) = 16.9$, $p < .001$, and BDI, $F(1, 111) = 4.7$, $p < .05$, but not the SAS–W, $F(2, 101) = 2.3$, *ns*, or SAS–S, $F(2, 113) = 1.5$, *ns*. There was a main effect of time on the PSS–I, $F(1, 115) = 415.7$, $p < .001$; BDI, $F(1, 111) = 162.8$, $p < .001$; SAS–W, $F(1, 101) = 9.7$, $p < .01$; and SAS–S, $F(1, 113) = 35.2$, $p < .001$; and Group × Time interactions on the PSS–I, $F(2, 115) = 32.5$, $p < .001$; BDI, $F(2, 111) = 22.1$, $p < .001$; SAS–W, $F(2, 101) = 3.6$, $p < .05$; and SAS–S, $F(2, 113) = 3.8$, $p < .05$. No main effect of site, largest $F(1, 115) = 3.3$, *ns*, and no interactions involving site emerged, largest $F(2, 115) = 2.3$, *ns*.

Using *t* tests for independent samples, we examined the Group × Time interactions via pairwise comparisons among groups at posttreatment. PSS–I scores were higher in the WL group than in the PE/CR, $t(67) = 7.3$, $p < .001$, and PE groups, $t(75) = 7.9$, $p < .001$. The PE/CR and PE groups did not differ from one another, $t(95) < 1.0$, *ns*. BDI scores were higher in the WL group than in the PE/CR group, $t(63) = 4.1$, $p < .001$, and PE group, $t(29.6) = 5.6$, $p < .001$, *df* adjusted because of unequal variances. The PE/CR and PE groups did not differ from one another, $t(93) < 1.0$, *ns*. SAS–W scores were higher in the WL group than in the PE/CR group, $t(57) = 2.8$, $p < .01$, and PE group, $t(65) = 3.2$, $p < .01$. The PE/CR and PE groups did not differ from one another, $t(86) < 1.0$, *ns*. SAS–S scores were higher in the WL group than in the PE/CR group, $t(66) = 2.8$, $p < .01$, and PE group, $t(73) = 2.3$, $p < .05$. The PE/CR and PE groups did not differ from one another, $t(93) < 1.0$, *ns*.

Effect Sizes

We computed within-group effect sizes comparing pre- and posttreatment according to the formula $ES = (M_{pre} - M_{post})/[(SD^2_{pre} + SD^2_{post})/2]^{one\ half}$. Effect sizes in the intent-to-treat sample for WL were 0.80 for the PSS–I, 0.25 for the BDI, −0.07 for the SAS–W, and 0.18 for the SAS–S. The corresponding effect sizes for PE/CR were as follows: 1.30, 0.86, 0.38, and 0.63, and for PE were 1.37, 0.96, 0.46, and 0.52. Effect sizes in the completer sample for WL were 0.86 for the PSS–I, 0.25 for the BDI, −0.16 for the SAS–W, and 0.18 for the SAS–S. The corresponding effect sizes for PE/CR were 2.39, 1.36, 0.48, and 0.93, and for PE were 3.31, 2.14, 0.73, and 0.86.

Length of Treatment and Outcome

Of the 96 participants who completed active treatment, 40 terminated treatment at Session 8 or 9 (short treatment), and 56 received between 10 and 12 sessions (long treatment). Participants in the shorter treatment group terminated because they achieved the criterion for early termination ($n = 27$) or declined the additional sessions ($n = 13$). We conducted a Site (CTSA vs. WOAR) × Treatment Length (short vs. long) × Time (pretreatment, Session 8, posttreatment) mixed factorial ANOVA on PSS–SR scores, with time as a within-subjects factor. Main effects emerged for treatment length, $F(1, 91) = 17.9$, $p < .001$, and time, $F(2, 182) = 274.1$, $p < .001$, modified by a Treatment Length × Time interaction, $F(2, 182) = 18.6$, $p < .001$. No significant main effect of site or interactions with site emerged, all F values < 1, *ns*.

Paired sample *t* tests were used to assess change in PSS–SR scores over time within each group. In the short treatment group, there was a significant reduction from pretreatment ($M = 32.1$, $SD = 9.2$) to Session 8 ($M = 9.6$, $SD = 7.8$), $t(39) = 15.6$, $p < .001$, followed by further reduction from Session 8 to posttreatment ($M = 5.5$, $SD = 7.1$), $t(39) = 6.1$, $p < .001$. The same pattern emerged in the long treatment group: a significant reduction from pretreatment ($M = 32.9$, $SD = 8.3$) to Session 8 ($M = 22.7$, $SD = 9.9$), $t(54) = 7.4$, $p < .001$, followed by further reduction from Session 8 to posttreatment ($M = 13.1$, $SD = 11.7$),

[1] Because of the pretreatment difference between PE/CR and PE groups on the PSS–I in the intent-to-treat sample, we also analyzed these posttreatment scores using analysis of covariance (ANCOVA), with pretreatment scores as the covariate. The ANCOVA results indicated a main effect for group, no main effect of site, and no Group × Site interaction. Pairwise comparisons on posttreatment PSS–I scores, using ANCOVA, indicated that WL had higher scores than PE/CR and PE groups, whereas PE/CR and PE did not differ. In response to editorial feedback, we also used ANCOVA to investigate posttreatment differences across the three *DSM–IV* PTSD symptom clusters (reexperiencing, avoidance/numbing, and hyperarousal). For each cluster, there was main effect for group, no main effect for site, and no Group × Site interaction. Pairwise comparisons on posttreatment cluster scores, using ANCOVA, indicated that WL had higher scores on each symptom cluster than PE/CR and PE, whereas PE/CR and PE did not differ. This was true for both the intent-to-treat and the completer samples.

[2] In response to reviewer feedback, we computed partial h^2 values to determine the amount of variance accounted for by site differences. Across all four measures in the intent-to-treat and completer samples, the largest value was .04 for all site main effects and all interactions involving site.

Table 2
Means (and Standard Deviations) for Intent-to-Treat Sample by Condition and Treatment Site

	Group					
	PE		PE/CR		WL	
Site	Pretreatment	Posttreatment	Pretreatment	Posttreatment	Pretreatment	Posttreatment
PTSD Symptom Scale Interview						
CTSA						
M (*SD*)	35.1 (5.7)	19.0 (14.8)	30.0 (8.8)	17.1 (13.7)	35.5 (5.8)	29.4 (8.9)
n	47	47	43	43	15	15
WOAR						
M (*SD*)	32.3 (5.8)	16.2 (14.1)	32.6 (7.0)	16.3 (12.8)	30.3 (5.8)	23.2 (9.7)
n	32	32	31	31	11	11
Total						
M (*SD*)	34.0 (5.9)	17.9 (14.5)	31.1 (8.1)	16.8 (13.2)	33.3 (6.2)	26.8 (9.6)
N	79	79	74	74	26	26
Beck Depression Inventory						
CTSA						
M (*SD*)	26.1 (10.8)	16.2 (13.4)	23.2 (10.2)	14.7 (14.2)	26.0 (7.7)	22.7 (8.8)
n	47	47	43	43	13	13
WOAR						
M (*SD*)	26.1 (9.7)	12.4 (14.1)	23.8 (7.8)	12.4 (10.8)	20.0 (12.9)	18.6 (13.0)
n	32	32	29	29	9	9
Total						
M (*SD*)	26.1 (9.9)	14.6 (13.8)	23.4 (9.3)	13.8 (12.9)	23.6 (10.3)	21.0 (10.7)
n	79	79	72	72	22	22
Social Adjustment Scale—Work						
CTSA						
M (*SD*)	3.3 (1.1)	2.8 (1.4)	3.2 (1.5)	2.6 (1.4)	3.6 (1.7)	3.9 (1.5)
n	40	40	33	33	11	11
WOAR						
M (*SD*)	3.7 (1.8)	2.9 (1.5)	3.2 (1.0)	2.8 (1.4)	3.0 (1.1)	3.0 (0.9)
n	29	29	29	29	9	9
Total						
M (*SD*)	3.4 (1.2)	2.8 (1.4)	3.2 (1.2)	2.7 (1.4)	3.4 (1.5)	3.5 (1.3)
N	69	69	62	62	20	20
Social Adjustment Scale—Social						
CTSA						
M (*SD*)	4.1 (1.0)	3.6 (1.3)	3.9 (1.0)	3.3 (1.2)	3.9 (1.0)	3.7 (1.1)
n	47	47	43	43	14	14
WOAR						
M (*SD*)	4.2 (1.0)	3.4 (1.3)	4.1 (0.9)	3.3 (1.3)	4.0 (1.5)	3.8 (1.1)
n	31	31	31	31	11	11
Total						
M (*SD*)	4.1 (1.0)	3.5 (1.3)	4.0 (1.0)	3.3 (1.2)	4.0 (1.2)	3.8 (1.1)
N	78	78	74	74	25	25

Note. PE = prolonged exposure; PE/CR = prolonged exposure plus cognitive restructuring; WL = wait-list; PTSD = posttraumatic stress disorder; CTSA = participants treated at the university-based Center for the Treatment and Study of Anxiety; WOAR = participants treated at the community-based Women Organized Against Rape.

$t(54) = 7.5$, $p < .001$. Simple between-group effects were tested with *t* tests for independent samples. The groups did not differ at pretreatment, $t(93) < 1.0$, *ns*; participants who completed treatment by Session 9 had lower scores at Session 8, $t(92.4) = 7.2$, $p < .001$, *df* adjusted because of unequal variances; and at posttreatment, $t(90.2) = 3.9$, $p < .001$, *df* adjusted because of unequal variances. Participants in the short treatment did not differ from participants who received additional sessions on any of the demographic variables presented in Table 1, largest $t(94) = 1.1$, *ns*; largest $\chi^2(4, N = 95) = 4.2$, *ns*.

Follow-Up Outcome

We conducted separate Group (PE vs. PE/CR) × Site (CTSA vs. WOAR) × Time (posttreatment, 3-, 6-, and 12-month follow-ups) mixed factorial ANOVAs on the PSS–I, BDI, SAS–W, and SAS–S scores. Only treatment completers with PSS–I data from at least one follow-up assessment were included (total $N = 89$). For each measure, we estimated missing data at the follow-up assessments using either (a) the mean of the preceding and following assessment scores, if available, or (b) the last observation carried forward. Descriptive statistics (*M, SD*, *n*) for each measure at each follow-up assessment point are presented in Table 4.

The Group × Site × Time Analyses on the PSS–I and SAS–W scores revealed no main effects or interactions, all Fs < 2.2, *ns*. On the BDI, there was a main effect for site, $F(1, 81) = 4.2$, $p < .05$: On average across assessments, women treated at WOAR ($M = 5.9$, $SE = 1.3$) had lower BDI scores than women treated at the CTSA ($M = 9.5$, $SE = 1.1$). Analysis of the SAS–S revealed a

Table 3
Means (and Standard Deviations) for Completer Sample by Condition and Treatment Site

	Group					
	PE		PE/CR		WL	
Site	Pretreatment	Posttreatment	Pretreatment	Posttreatment	Pretreatment	Posttreatment
PTSD Symptom Scale Interview						
CTSA						
M (*SD*)	34.8 (5.4)	10.4 (9.7)	29.3 (9.4)	9.76 (10.6)	35.2 (5.89)	28.6 (8.7)
n	30	30	26	26	14	14
WOAR						
M (*SD*)	31.1 (5.6)	7.9 (6.3)	33.2 (7.9)	8.7 (6.9)	30.3 (5.8)	23.2 (9.7)
n	22	22	18	18	11	11
Total						
M (*SD*)	33.2 (5.7)	9.3 (8.4)	30.9 (9.0)	9.3 (9.2)	33.0 (6.2)	26.2 (9.4)
N	52	52	44	44	25	25
Beck Depression Inventory						
CTSA						
M (*SD*)	24.6 (9.6)	8.9 (8.5)	23.3 (9.3)	9.6 (13.2)	26.0 (7.7)	22.7 (8.8)
n	30	30	26	26	13	13
WOAR						
M (*SD*)	24.2 (8.0)	4.4 (4.1)	22.9 (8.9)	7.8 (9.4)	20.0 (12.9)	18.6 (13.0)
n	22	22	17	17	9	9
Total						
M (*SD*)	24.4 (8.9)	7.0 (7.3)	23.1 (9.1)	8.8 (11.7)	23.6 (10.3)	21.0 (10.7)
N	52	52	43	43	22	22
Social Adjustment Scale—Work						
CTSA						
M (*SD*)	3.0 (1.0)	2.4 (1.2)	3.1 (1.4)	2.3 (1.1)	3.4 (1.6)	3.7 (1.3)
n	28	28	22	22	10	10
WOAR						
M (*SD*)	3.4 (1.1)	2.4 (1.2)	3.0 (1.0)	2.4 (1.5)	3.0 (1.1)	3.0 (0.9)
n	20	20	18	18	9	9
Total						
M (*SD*)	3.2 (1.0)	2.4 (1.2)	3.0 (1.2)	2.4 (1.3)	3.2 (1.4)	3.4 (1.2)
N	48	48	40	40	19	19
Social Adjustment Scale—Social						
CTSA						
M (*SD*)	4.0 (0.9)	3.2 (1.2)	3.7 (1.0)	2.9 (1.2)	3.8 (1.0)	3.6 (1.0)
n	30	30	26	26	13	13
WOAR						
M (*SD*)	4.0 (1.0)	2.9 (1.2)	4.2 (0.8)	2.9 (1.2)	4.0 (1.5)	3.8 (1.1)
n	21	21	18	18	11	11
Total						
M (*SD*)	4.0 (0.9)	3.1 (1.2)	3.9 (1.0)	2.9 (1.2)	3.9 (1.2)	3.7 (1.0)
N	51	51	44	44	24	24

Note. PE = prolonged exposure; PE/CR = prolonged exposure plus cognitive restructuring; WL = wait-list; PTSD = posttraumatic stress disorder; CTSA = participants treated at the university-based Center for the Treatment and Study of Anxiety; WOAR = participants treated at the community-based Women Organized Against Rape.

main effect for site, $F(1, 83) = 4.8$, $p < .05$, and time, $F(3, 249) = 6.6$, $p < .01$. Women treated at WOAR had significantly lower (better functioning) SAS–S scores ($M = 2.3$, $SE = 0.2$) than women treated at CTSA ($M = 2.9$, $SE = 0.1$). Paired-sample t tests comparing SAS–S scores at consecutive assessments revealed a significant decrease from posttreatment ($M = 2.9$, $SD = 1.1$) to the 3-month follow-up ($M = 2.6$, $SD = 1.3$), $t(86) = 3.3$, $p < .01$. No changes emerged from 3 to 6 months, $t(86) = 0.2$, *ns*, or from 6 to 12 months, $t(86) = 0.5$, *ns*.

A total of 33 participants received at least three sessions of therapy during follow-up, 6 of whom saw their study PTSD therapist (median number of session was 10); 14 had received PE, and 19 received PE/CR, $\chi^2(1, N = 88) = 2.1$, $p = .15$. Participants receiving additional therapy were more symptomatic on the PSS–I at posttreatment ($M = 14.1$, $SD = 10.8$; versus $M = 6.0$, $SD = 4.8$), $t(39.8) = -4.1$, $p < .0005$, and at last follow-up ($M = 12.6.$, $SD = 10.4$; versus $M = 6.0$, $SD = 4.8$), $t(46.7) = -3.5$, $p < .005$, than those who did not receive treatment.

To examine possible differences within the sample, we divided participants into three groups on the basis of their PSS–I scores from posttreatment and the last available follow-up: (a) those with reliable worsening of PTSD symptoms, (b) those with reliable improvement on PTSD symptoms, and (c) those with stable PTSD symptoms. Reliable worsening or improvement was set at ± 7.53 points on the PSS–I (see Devilly & Foa, 2001, for the calculation of this figure).

Table 4
Follow-Up Means (and Standard Deviations) for Completer Sample by Condition and Treatment Site

	Group					
	PE			PE/CR		
Site	3 months	6 months	12 months	3 months	6 months	12 months
PTSD Symptom Scale Interview						
CTSA						
M (*SD*)	9.1 (8.4)	10.7 (9.8)	11.0 (9.7)	8.2 (9.1)	8.7 (10.1)	8.4 (9.8)
n	28	28	28	25	25	25
WOAR						
M (*SD*)	6.4 (6.0)	6.2 (6.5)	5.7 (5.8)	7.1 (5.4)	7.9 (7.9)	6.4 (7.3)
n	19	19	19	17	17	17
Total						
M (*SD*)	8.0 (7.6)	8.9 (8.8)	8.9 (8.7)	7.7 (7.8)	8.4 (9.1)	7.6 (8.8)
N	47	47	47	42	42	42
Beck Depression Inventory						
CTSA						
M (*SD*)	10.4 (8.6)	9.9 (7.9)	8.7 (7.5)	9.0 (11.5)	7.7 (10.1)	11.1 (12.3)
n	28	28	28	23	23	23
WOAR						
M (*SD*)	5.3 (3.6)	6.6 (4.2)	5.5 (3.4)	6.7 (6.1)	7.5 (8.4)	5.5 (7.0)
n	18	18	18	16	16	16
Total						
M (*SD*)	8.4 (7.5)	8.6 (6.9)	7.5 (6.4)	8.0 (9.6)	7.6 (9.3)	8.8 (10.7)
N	46	46	46	39	39	39
Social Adjustment Scale—Work						
CTSA						
M (*SD*)	2.2 (1.2)	2.2 (1.2)	2.2 (1.1)	2.3 (0.8)	2.0 (0.9)	1.9 (1.0)
n	25	25	25	21	21	21
WOAR						
M (*SD*)	1.8 (0.5)	2.0 (0.7)	1.7 (0.9)	2.0 (1.4)	2.1 (1.1)	2.1 (1.1)
n	16	16	16	16	16	16
Total						
M (*SD*)	2.0 (1.0)	2.1 (1.1)	2.0 (1.0)	2.2 (1.1)	2.0 (1.0)	2.0 (1.1)
N	41	41	41	37	37	37
Social Adjustment Scale—Social						
CTSA						
M (*SD*)	2.8 (1.4)	2.8 (1.4)	2.9 (1.2)	2.8 (1.2)	2.6 (1.5)	2.8 (1.6)
n	27	27	27	25	25	25
WOAR						
M (*SD*)	2.0 (1.0)	2.2 (1.0)	1.9 (0.8)	2.5 (1.1)	2.5 (1.2)	2.2 (1.1)
n	18	18	18	17	17	17
Total						
M (*SD*)	2.5 (1.3)	2.6 (1.2)	2.5 (1.1)	2.7 (1.2)	2.6 (1.4)	2.5 (1.4)
N	45	45	45	42	42	42

Note. PE = prolonged exposure; PE/CR = prolonged exposure plus cognitive restructuring; PTSD = posttraumatic stress disorder; CTSA = participants treated at the university-based Center for the Treatment and Study of Anxiety; WOAR = participants treated at the community-based Women Organized Against Rape.

The vast majority of participants (79.8%) showed stable PSS–I scores during follow-up (posttreatment PSS–I, $M = 7.1$, $SD = 5.9$; last available PSS–I, $M = 6.6$, $SD = 7.1$). A small group of participants (12.4%) showed reliable improvement during follow-up (posttreatment PSS–I, $M = 22.4$, $SD = 11.5$; last available PSS–I, $M = 10.5$, $SD = 11.1$), and an even smaller group (7.9%) showed reliable symptom worsening (posttreatment PSS–I, $M = 7.3$, $SD = 7.8$; last available PSS–I, $M = 22.1$, $SD = 7.4$). A Group (improvement vs. no change vs. worsening) × Site (CTSA vs. WOAR) chi-square failed to detect site differences, $\chi^2(2, N = 89) = 2.2$, *ns*. A Group (improvement vs. no change vs. exacerbation) × Treatment (PE vs. PE/CR) chi-square also failed to detect differences between the two treatments, $\chi^2(2, N = 89) = 3.4$, *ns*. A series of exploratory analyses examined differences among the three groups on pre- and posttreatment symptom severity scores, change in PTSD during treatment, type of trauma (child sexual abuse vs. adult sexual assault vs. adult nonsexual assault) and demographic variables. Participants who showed improvement were more symptomatic at posttreatment than either of the other groups, comparison with worse, $t(16) = 3.03$, $p < .01$; stable, $t(80) = -4.3$, $p < .01$. Relapsers and stable participants did not differ, $t(76) = -0.01$, *ns*. No other predictors of relapse were found.

Discussion

Consistent with our first hypothesis, treatment with PE alone and PE/CR was superior to WL in reducing PTSD and depression. These results were obtained in both intent-to-treat and completer samples. In addition, participants who completed treatment showed significant improvement in work and social functioning. Inconsistent with our second hypothesis, the addition of CR to PE did not enhance treatment outcome. Our third hypothesis that CBT experts would achieve better outcome than master's level counselors was also not supported. In fact, participants treated at WOAR had better outcome on depression and social functioning at follow-up.

In the current study, participants who achieved at least a 70% reduction in self-reported PTSD severity by Session 8 terminated treatment at Session 9, whereas the others were offered up to a total of 12 sessions. Among treatment completers, 42% completed treatment by Session 9 and 58% received additional treatment. These results suggest that, although most patients show improvement within the first eight sessions of PE, the majority need additional sessions to reach excellent response. Consistent with our fourth hypothesis, PTSD severity decreased between Session 8 and the final session for those who received extension sessions, but they remained more symptomatic than those who ended treatment by Session 9. Consistent with our fifth hypothesis, overall, treatment gains were maintained at follow-up on measures of PTSD, depression, and work functioning. Further improvement in social functioning was observed during the first 3 months, and this improvement was maintained. Among participants who returned for follow-up assessment, 80% maintained their gains, 12% showed further improvement, and 8% showed some relapse. Notably, a significant minority of participants received additional treatment during the follow-up period, although few received the additional treatment from their PTSD therapist. An interesting finding is that, on the average, participants who received treatment at follow-up did not do as well as those who did not receive treatment either at posttreatment or at follow-up. This finding suggests the importance of achieving excellent response during acute treatment. However, because most of the participants who received the additional treatment had shown substantial improvement on PTSD symptoms at the end of treatment and did not seek the additional treatment from their study PTSD therapist, it is possible that they sought treatment for other problems, perhaps as a result of the improvement they experienced on their PTSD symptoms.

Our finding that adding CR did not improve the outcome of PE is consistent with results reported by Marks et al. (1998) and Paunovic and Ost (2001). Similarly, Foa, Dancu, et al. (1999) found that the combination of PE and SIT was not superior to either individual treatment. Thus, combining separately efficacious treatments (e.g., PE/CR, PE/SIT) does not seem to enhance outcome for PTSD. An exception is Bryant et al.'s (2003) finding that adding CR improved outcome relative to exposure therapy. However, Bryant et al.'s study included only imaginal exposure, whereas the current study—as well as the studies by Marks et al. and Paunovic and Ost—included both imaginal and in vivo exposure. Perhaps the efficacy of imaginal exposure to trauma memories can be augmented adding either in vivo exposure or CR. Despite the growing body of evidence that adding treatment components to imaginal plus in vivo exposure does not improve outcome, most treatment programs for PTSD include multiple components on top of a strong exposure base (e.g., Blanchard et al., 2003) that may be unnecessary.

Two explanations have been offered for the failure to augment the outcome of exposure therapy with elements of SIT or CR. First, for study purposes, programs that combine two separately efficacious treatments have been administered in the same number and duration of sessions used for the individual treatments (e.g., Foa, Dancu, et al., 1999; Marks et al., 1998). Thus, participants in the combined treatment may not have received the full dose of the individual treatments. Alternatively, it is possible that all efficacious treatments modify the same dysfunctional cognitions underlying PTSD (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999). CR was explicitly designed to help patients modify beliefs that are thought to maintain their symptoms (Beck & Emory, 1985). Foa and Kozak (1986) proposed that exposure therapy reduces pathological anxiety by modifying erroneous cognitions via disconfirming information embedded in the exposure exercises. Support for the hypothesis that exposure therapy changes pathogenic cognitions comes from several treatment studies on anxiety disorders, including PTSD (Foa & Rauch, 2004), panic disorder/agoraphobia (Williams & Rappaport, 1983), and social phobia (Hope, Heimberg, & Bruch, 1995). Notably, there is considerable overlap between exposure therapy and cognitive therapy for PTSD. Both approaches include similar elements, although programs may differ in how much emphasis they place on exposure versus cognitive therapy, how exposure and cognitive therapy are conducted, and even in what they label as exposure or cognitive therapy.

The current study demonstrated that both PE alone and combined with CR could be successfully disseminated to community-based master's level clinicians. Becker et al.'s (2004) finding that many community clinicians do not use exposure therapy for PTSD because they lack appropriate training suggests a need to devise effective and efficient training programs to educate clinicians. Less complex programs may be more easily disseminated. In our study, WOAR therapists were provided 5 days of training in PE and 5 additional days of training in CR. Thus, the combined treatment doubled the length of training without yielding significant benefit. The reduced training requirement for PE alone may make this program more easily disseminated. This is consistent with our experiences in successfully disseminating PE, via 5-day workshops, to therapists in Israel who had no prior CBT experience (Cahill, Hembree, & Foa, in press).

In the current study, treatment targeted at PTSD also reduced depression and improved work and social functioning, especially among treatment completers. Other studies of PE targeting PTSD have found it to reduce depression (Foa, Dancu, et al., 1999; Foa, Rothbaum, Riggs, & Murdock, 1991; Resick, Nishith, Weaver, Astin, & Feurer, 2002), anxiety (Foa, Dancu, et al., 1999; Foa et al., 1991), anger (Cahill, Rauch, Hembree, & Foa, 2003), and guilt (Resick et al., 2002). Studies of other CBT programs for PTSD have found improvements on a variety of outcome measures such as depression and guilt (e.g., Resick et al.'s, 2002, cognitive processing therapy); anger, dissociation, alexithymia, and emotional regulation (Cloitre, Koenen, Cohen, & Han's, 2002, skills training in affective and interpersonal regulation/PE combination); and comorbid generalized anxiety disorder (Blanchard et al., 2003). Perhaps CBT programs affect an etiological or maintaining

variable common across anxiety and mood disorders such as negative cognitions about self and world, intolerance for negative affect, or affect regulation deficits.

Several caveats should be recognized. First, the current study did not include an active control group. Therefore, we cannot ascertain how much of the improvement can be attributed to the specific elements of the PE programs compared with the common factors of psychotherapy. Although the current results demonstrate that community therapists with little prior CBT experience can successfully deliver PE and PE/CR, it is unclear whether such success would have been achieved without ongoing supervision from the CTSA experts. The ongoing supervision limits the generalizability of our findings. We are currently testing how well WOAR therapists maintain their competence delivering PE without CTSA supervision and whether a more streamlined training and supervision model yields similar results at another community site.

The accumulating evidence that combining separately efficacious psychological treatments does not yield better outcome for PTSD suggests that alternative models need to be used for enhancing treatment efficacy. Perhaps a better strategy is to translate principles emerging from basic experimental research on topics such as extinction, cognitive biases, and memory into new treatment procedures (Foa & Kozak, 1997). One promising example of this strategy is the augmentation of exposure with the medication d-cycloserine in acrophobia (Ressler et al., 2004), a medication shown in animals to enhance extinction of conditioned fear.

The current results encourage the dissemination of CBT for PTSD. However, as noted above, few trauma therapists use CBT, largely because of lack of training (Becker et al., 2004). In the current study, we successfully disseminated two empirically supported treatment programs. However, developing strategies to do this in an efficient manner that promotes their acceptability to therapists and ensures therapists' adherence to treatment protocols remains a challenge. We need to develop and test various dissemination models to ensure that people suffering from PTSD can readily benefit from efficacious treatments.

References

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

Beck, A. T., & Emory, G. (1985). *Anxiety disorders and phobias: A cognitive perspective.* New York: Basic Books.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4,* 561–571.

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

Blake, D. D., Weather, F. W., Nagy, L. M., Kaloupek, D. G., Kaulminzer, G., Charney, D. S., et al. (1990). A clinician rating scale for assessing current and lifetime PTSD: The CAPS 1. *Behavior Therapist, 13,* 187–188.

Blanchard, E. B., Hickling, E. J., Devineni, T., Veazey, K. C. H., Galovski, T. E., Mundy, E., et al. (2003). A controlled evaluation of cognitive–behavioral therapy for posttraumatic stress in motor vehicle accident survivors. *Behaviour Research & Therapy, 41,* 79–96.

Bryant, R. A., Moulds, M. L., Guthrie, R. M., Dang, S. T., & Nixon, R. D. (2003). Imaginal exposure alone and imaginal exposure with cognitive restructuring in treatment of posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 71,* 706–712.

Cahill, S. P., & Foa, E. B. (2004). A glass half empty or half full? Where we are and directions for future research in the treatment of PTSD. In S. Taylor (Ed.), *Advances in the treatment of posttraumatic stress disorder: Cognitive–behavioral perspectives* (pp. 267–313). New York: Springer.

Cahill, S. P., Hembree, E. A., & Foa, E. B. (in press). Dissemination of prolonged exposure therapy for posttraumatic stress disorder: Successes and challenges. In Y. Neria, R. Gross, R. Marshall, & E. Susser (Eds.), *9/11: Public health in the wake of terrorist attacks.* Cambridge, England: Cambridge University Press.

Cahill, S. P., Rauch, S. A., Hembree, E. A., & Foa, E. B. (2003). Effect of cognitive–behavioral treatments for PTSD on anger. *Journal of Cognitive Psychotherapy, 17,* 113–131.

Cloitre, M., Koenen, K., 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.

Devilly, G. J., & Foa, E. B. (2001). Comments on Tarrier et al.'s study and the investigation of exposure and cognitive therapy. *Journal of Consulting and Clinical Psychology, 69,* 114–116.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (1995). *Structured Clinical Interview for DSM–IV Axis I Disorders—Patient Edition (SCID–I/P, Version 2).* New York: New York State Psychiatric Institute, Biometrics Research Department.

Foa, E. B., Dancu, C. V., Hembree, E. A., Jaycox, L. H., Meadows, E. A., & Street, G. P. (1999). A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *Journal of Consulting and Clinical Psychology, 67,* 194–200.

Foa, E. B., Ehlers, A., Clark, D. M., Tolin, D. F., & Orsillo, S. M. (1999). The Posttraumatic Cognitions Inventory (PTCI): Development and validation. *Psychological Assessment, 11,* 303–314.

Foa, E. B., Keane, T. M., & Friedman, M. J. (2000). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies.* New York: Guilford Press.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99,* 20–35.

Foa, E. B., & Kozak, M. J. (1997). Beyond the efficacy ceiling? Cognitive–behavior therapy in search of a theory. *Behavior Therapy, 28,* 601–611.

Foa, E. B., & Rauch, S. A. M. (2004). Cognitive changes during prolonged exposure alone versus prolonged exposure and cognitive restructuring in female assault survivors with PTSD. *Journal of Consulting and Clinical Psychology, 72,* 879–884.

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

Foa, E. B., & Rothbaum, B. O. (1998). *Treating the trauma of rape: Cognitive–behavioral therapy for PTSD.* New York: Guilford Press.

Foa, E. B., Rothbaum, R. O., Riggs, D. S., & Murdock, T. B. (1991). Treatment of posttraumatic stress disorder in rape victims: A comparison between cognitive–behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology, 59,* 715–723.

Hope, D. A., Heimberg, R. G., & Bruch, M. A. (1995). Dismantling cognitive–behavioral group therapy for social phobia. *Behaviour Research & Therapy, 33,* 637–650.

Kilpatrick, D. G., Veronen, L. J., & Resick, P. A. (1982). Psychological sequelae to rape: Assessment and treatment strategies. In D. M. Dolays & R. L. Meredith (Eds.), *Behavioral medicine: Assessment and treatment strategies* (pp. 473–497). New York: Plenum Press.

Marks, I., Lovell, K., Noshirvani, H., Livanou, M., & Thrasher, S. (1998). Treatment of posttraumatic stress disorder by exposure

and/or cognitive restructuring. *Archives of General Psychiatry, 55,* 317–325.

Paunovic, N., & Ost, L. G. (2001). Cognitive–behavior therapy vs exposure therapy in the treatment of PTSD in refugees. *Behaviour Research & Therapy, 39,* 1183–1197.

Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feurer, 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.

Ressler, K. J., Rothbaum, B. O., Tannenbaum, L., Anderson, P., Graap, K., Zimand, E., et al. (2004). Cognitive enhancers as adjuncts to psychotherapy: Use of d-cycloserine in phobic individuals to facilitate extinction of fear. *Archives of General Psychiatry, 61,* 1136–1144.

Weissman, M. M., & Paykel, E. S. (1974). *The depressed woman: A study of social relations.* Chicago: University of Chicago Press.

Williams, S. L., & Rappaport, A. (1983). Cognitive treatment in the natural environment for agoraphobics. *Behavior Therapy, 14,* 299–313.

Received October 28, 2004
Revision received March 29, 2005
Accepted May 4, 2005 ■