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Exposure therapy in the treatment of PTSD among cocaine-dependent individuals: preliminary findings

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Abstract

Individuals ($n = 39$) participated in an outpatient, 16-session individual, manual-guided psychotherapy designed to treat concurrent PTSD and cocaine dependence. Therapy consisted of a combination of imaginal and in-vivo exposure therapy techniques to treat PTSD symptoms and cognitive-behavioral techniques to treat cocaine dependence. Although the dropout rate was high, treatment completers (i.e., patients who attended at least 10 sessions; $n = 15$) demonstrated significant reductions in all PTSD symptom clusters and cocaine use from baseline to end of treatment. Significant reductions in depressive symptomatology, as measured by the Beck Depression Inventory, and psychiatric and cocaine use severity, as measured by the Addiction Severity Index, were also observed. These improvements in PTSD symptoms and cocaine use were maintained over a 6-month follow-up period among completers. The average pre- to posttreatment effect size was 1.80 for PTSD symptoms and 1.26 for drug and alcohol use severity. Baseline comparisons between treatment completers and noncompleters revealed significantly higher avoidance symptoms, as measured by the Impact of Events Scale, and fewer years of education among treatment noncompleters as compared to completers. This study provides preliminary evidence to suggest that exposure therapy can be used safely and may be effective in the treatment of PTSD in some individuals with cocaine dependence. However, the study is limited by the uncontrolled nature of the study design, small number of subjects, and high dropout rate.

Keywords: PTSD; Posttraumatic stress disorder; Cocaine; Substance use; Exposure therapy

1. Introduction

Among psychosocial treatments for posttraumatic stress disorder (PTSD), exposure-based therapies have been extensively studied (Foa & Meadows, 1997; Foa & Rothbaum, 1998). Individuals with both civilian and combat-related PTSD have shown significant reductions in PTSD and associated symptoms (e.g., depression, social adjustment) when treated with exposure therapy (Hembree & Foa, 2000; Foa et al., 1999). In the consensus statement compiled by the International Consensus Group on Depression and Anxiety, exposure therapy was selected as the most appropriate form of psychotherapy to manage PTSD (Ballenger et al., 2000). The Expert Consensus Guidelines for the treatment of PTSD (Frances, 1999) recommend exposure therapy as the most effective and rapidly acting non-pharmacologic treatment for PTSD.

The clinical application and empirical investigation of the efficacy of exposure therapy, however, has been limited primarily to individuals without comorbid substance use disorders (SUDs). When asked to rate the appropriateness of various treatments for PTSD in the presence of a comorbid SUD, psychotherapy experts in the field (Foa et al., 1999) recommended exposure therapy as a second-line treatment of choice, following anxiety management techniques, cognitive therapy, and psychoeducation. The reluctance to use or systematically investigate exposure-based treatment of PTSD in SUD populations is based, in part, on the belief that exposure is too emotionally distressing for SUD patients, who are frail or vulnerable (cf., Abueg &

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Fairbank, 1992; cf. Triffleman, Carroll, & Kellogg, 1999) to tolerate exposure without relapse. In addition, it has been suggested that SUD patients may be too cognitively impaired for imagery procedures (Abueg & Fairbank, 1992; Pitman, Altman, Greenwald, Longpre, Macklin, et al., 1991). Several case studies investigating the use of exposure-based treatments in individuals with comorbid PTSD and a variety of SUDs have had mixed results (Keane & Kaloupek, 1982; Mueser & Butler, 1987; Pitman et al., 1991; Vaughan & Tarrier, 1992).

An estimated 30% to 60% of individuals with SUDs meet criteria for comorbid PTSD (Brown, Recupero, & Stout, 1995; Stewart, 1996). These estimates are significantly greater than estimates of PTSD in the general population (8% to 9%; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Hidalgo & Davidson, 2000). Cottler, Compton, Mager, Spitznagel, and Janca (1992) found that cocaine/opiate users had the highest risk for comorbid PTSD and that the rate of PTSD was ten times higher in this group when compared to individuals without cocaine dependence. Many investigators have noted that PTSD/SUD comorbidity is associated with a more severe symptom profile, higher rates of Axis I and II pathology, more severe social impairment, and poorer treatment course and outcome (Back et al., 2000; Brown et al., 1995; Najavits et al., 1998a; Ouimette, Finney, & Moos, 1999; Stewart, Conrod, Pihl, & Dongier, 1999).

The goal of the present study was a preliminary, uncontrolled evaluation of the safety, efficacy, and tolerability of exposure therapy in the treatment of PTSD among a sample of individuals with comorbid PTSD and cocaine dependence. Patient retention and improvement in PTSD severity, cocaine use, and associated features during treatment and at 6 months follow-up were examined. Baseline characteristics of individuals who completed treatment (i.e., attended ten or more sessions) were compared with individuals who did not complete treatment.

2. Methods

2.1. Participants

Participants were 39 treatment-seeking individuals (32 women, 7 men) enrolled in an outpatient psychotherapy development trial designed for the concurrent treatment of PTSD and cocaine dependence. All patients met the *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition (DSM-IV) criteria for current PTSD and cocaine dependence, and attended at least one therapy session. Completion of ten therapy sessions involved participation in at least three exposure-based therapy sessions. Based on the clinical experience of our group, three exposure-based sessions were considered to be the minimum amount of exposure necessary for therapeutic effect. Therefore, individuals who completed ten or more sessions were defined as therapy completers (n = 15) and the others as noncompleters (n = 24). Exclusionary criteria included psychosis, dissociative identity disorder, dementia, illiteracy, and suicidal or homicidal ideation. Individuals who were medically unstable were also excluded from the study. Participants could be dependent on another substance, but had to identify cocaine as their drug of choice.

2.2. Measures

2.2.1. Substance use assessments

Diagnoses of substance use disorders were made at baseline using the Structured Clinical Interview for the DSM-IV (SCID) (First, Spitzer, Gibbon, & Williams, 1996). The Addiction Severity Index (ASI) (McLellan, Parikh, & Bragg, 1990) was used to measure severity of substance use and impairment in associated areas (e.g., family, medical) at baseline and treatment termination. Urine drug screen (UDS) tests for cocaine, marijuana, and opiates were performed weekly to assess drug use.

2.2.2. Trauma and PTSD assessments

The National Women's Study (NWS) PTSD Module (Kilpatrick, Resnick, Saunders, & Best, 1989), a structured clinical interview, was employed to screen for Criterion A events and assess PTSD. Reliability and validity of the NWS are adequate (Resnick, Kilpatrick, Dansky, & Best, 1993). The Clinician-Administered PTSD Scale (CAPS) (Blake et al., 1995), a 30-item structured interview, measured frequency, and intensity of Criterion B, C and D PTSD symptoms. Research has demonstrated adequate reliability and validity for the CAPS (Blake et al., 1995). Two self-report instruments, the Impact of Events Scale (IES) (Horowitz, Wilner, & Alvarez, 1979) and the Mississippi Scale for PTSD (MISS) (Keane, Caddell, & Taylor, 1997), were also administered weekly to measure PTSD symptoms. IES subscales for intrusion, avoidance, and a total score were computed. Depressive symptomatology was assessed with the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

2.3. Procedure

2.3.1. Evaluations

Eligible participants read and signed an IRB-approved consent form and were interviewed by a trained clinician. The NWS was administered at baseline. The CAPS and ASI were administered at baseline, weeks 4, 8, 12, and 16, and at 6 months follow-up. The IES, MISS, and TLFB were administered weekly and at follow-up. UDS samples were collected at each treatment session. Attendance was reinforced with gift certificates worth $10 given at the end of completing two consecutive scheduled therapy sessions.

2.3.2. Treatment

The treatment protocol was adopted from Foa and colleagues (1999). Patients attended one to two individual, 90-minute psychotherapy sessions per week, depending on feasibility and patient/therapist preference. The treatment (described in detail in Back, Dansky, Carroll, Foa, & Brady, 2001) consisted of combined imaginal and in-vivo exposure therapy and cognitive-behavioral relapse prevention techniques. The treatment was delivered by six different therapists with diverse credentials (e.g., M.A., M.D., M.S.W., Ph.D.) and background training in the areas of PTSD and addiction. Therapists were required to undergo intensive training for the study, including videotaped sessions of the full treatment with at least one patient, watching videotapes of other therapists conducting the treatment, and weekly group supervision with other therapists currently conducting the treatment. Areas of knowledge and skills that may be helpful for clinicians conducting this type of treatment are reviewed in Back et al. (2001), although these recommendations are speculative in nature.

Depending on their level of avoidance and subjective distress, patients received six to nine sessions of imaginal exposure in which they narrated their traumatic event, including thoughts, emotions, and physiological sensations associated with the memory. For individuals with multiple traumas, an "index" trauma (i.e., that trauma which causes the most distress or impairment in life) was identified and used during imaginal exposure. Beginning at session 6, patients were required to complete in-vivo exposure (i.e., confront safe, yet fear-inducing, situations associated with the event) outside of therapy as homework exercises. Imaginal exposure began at session 7 for all patients.

2.3.3. Statistical analysis

Comparisons were made to identify differences among treatment completers and noncompleters. Then, using data from treatment completers only, pre- and posttreatment comparisons were made on PTSD and substance use outcome variables. The pretreatment to follow-up analyses included data from patients who completed the treatment and the 6-month follow-up visit. This is reflected in the different pretreatment baseline scores. Categorical data comparisons were made using chi-square analyses. Comparisons of continuous data were made using independent and paired *t*-tests or analyses of variance (ANOVA) tests. The treatment effect size was calculated using Glass's delta. Given the exploratory nature of the study and the small sample size, Type I error was not controlled for and alpha was set at .05.

3. Results

3.1. Preliminary analyses

3.1.1. Comparison of treatment completers and noncompleters

Table 1 displays subject characteristics. As can be seen, treatment completers had significantly more years of education as compared to noncompleters. No other significant differences between completers and noncompleters on sociodemographic or psychiatric variables were found. All subjects met criteria for at least one other psychiatric disorder. There were no significant between-group differences for any baseline substance use measures. Group comparisons of trauma histories revealed no significant differences. The majority of individuals reported experiencing rape (74.4%), aggravated assault (89.0%), and other physical assault (94.9%) during their lifetime.

Group comparisons were conducted on baseline IES, CAPS, MISS, and BDI scores. Noncompleters reported significantly higher levels of baseline avoidance as measured by the IES in comparison to completers (27.91 vs.

Table 1
Subject characteristics

	Completers (n = 15)	Noncompleters (n = 24)	Total (n = 39)
Background characteristics			
Age, yrs	33.5 ± 4.5	32.2 ± 4.6	33.7 ± 4.5
Gender, % female	86.7	79.2	82.1
Race, % Caucasian	53.3	45.8	48.7
African American	46.7	50.0	48.7
Hispanic	0.0	4.2	2.6
Education, yrs[a]	13.2 ± 2.6	11.8 ± 1.5	12.3 ± 2.1
Employment, % full-time	46.7	54.2	51.3
Marital status, % married	0.0	12.5	7.7
Separated	20.0	33.3	28.2
Divorced	33.3	8.3	17.9
Single, never married	46.7	45.8	46.2
Psychiatric comorbidity			
Any Axis I disorder	100.0	100.0	100.0
Any anxiety disorder	57.1	45.5	52.0
Any affective disorder	78.6	54.5	68.0

[a] Significant difference between groups in number of years of education ($t = -2.09$, df = 37, $p < 0.05$).

22.20; $t = 2.32$, df = 1, $p < 0.05$). There were no other significant differences on baseline PTSD ratings.

3.1.2. Treatment adherence

Subjects were recruited from local substance abuse treatment programs. One hundred forty-five patients were screened for study participation. Patients were excluded because they did not meet criteria for PTSD ($n = 74$) or they failed to consistently attend pre-study assessment visits ($n = 32$).

3.1.3. Patient retention

Twenty-seven individuals (69.2%) attended at least one quarter and 19 individuals (48.7%) attended at least half of the therapy sessions. Fifteen individuals (38.5%) attended at least 10 of 16 therapy sessions and were considered treatment completers. The average number of sessions attended was 14.7 ($SD = 1.9$) for treatment completers and 4.1 ($SD = 2.6$) for treatment noncompleters. The majority (75%; 18/24) of individuals who did not complete treatment dropped out before exposure therapy was initiated. The reasons for dropout included transportation difficulty ($n = 6$), entering inpatient treatment ($n = 2$), relocation ($n = 3$), and scheduling difficulties ($n = 4$). We were unable to contact the remaining subjects ($n = 9$) to determine the reason for dropout. Six-month follow-up data was obtained on seven treatment completers and six noncompleters. None of the six treatment noncompleters who were interviewed for 6-month follow-up reported that the exposure therapy was the reason for dropout.

3.1.4. Treatment outcome

For pre- to posttreatment outcome analyses, only data for the 15 treatment completers was used.

3.1.4.1. PTSD symptom reduction. As can be seen in Table 2, intrusive, avoidant, and hyperarousal symptoms, as measured by the IES and/or the CAPS, decreased significantly from pre- to posttreatment. CAPS total scores, and the MISS total and BDI scores were also significantly lower at posttreatment and at 6-month follow-up. There were no differences in any PTSD scale scores in the treatment noncompleters at 6-month follow-up. Fig. 1 illustrates the change over time in PTSD symptoms as measured by the IES.

3.1.4.2. Substance use reduction. All ASI subscale scores decreased from pre- to posttreatment. Significant reductions were found in ASI drug, alcohol, and psychiatric subscale scores (see Table 2). From baseline to 6-month follow-up, significant reductions were found in ASI drug, alcohol, and employment subscale scores. The psychiatric subscale score also showed substantial improvement (i.e., reduction of approximately 50%) from pretreatment to follow-up, although this difference did not reach statistical significance. In addition, patients reported experiencing drug-related

Table 2
Treatment outcome

	Pre- to Posttreatment[a]		Pretreatment to Follow-Up[b]	
	M(*SD*)	M(*SD*)	M(*SD*)	M(*SD*)
IES				
Intrusion	19.5 (13.0)	9.1 (7.1)*	22.0 (13.5)	9.4 (6.2)
Avoidance	20.1 (9.1)	14.6 (8.2)	22.4 (7.7)	9.0 (7.3)
Total	39.6 (21.4)	23.8 (13.7)	44.4 (18.3)	18.4 (12.2)
CAPS				
Intrusion	9.4 (6.3)	3.2 (6.7)**	9.0 (4.9)	8.4 (8.5)
Avoidance	19.7 (10.1)	5.8 (8.9)**	19.6 (7.0)	10.1 (11.2)
Hyperarousal	16.6 (7.9)	8.7 (11.6)*	16.9 (6.9)	5.4 (8.1)*
Total	45.2 (19.8)	15.8 (23.0)***	45.4 (12.2)	24.0 (24.2)[c]
MISS				
Total	111.7 (21.9)	83.7 (24.8)*	122.8 (13.6)	66.3 (15.8)*
BDI	12.1 (8.0)	5.7 (7.4)*	11.0 (1.7)	2.3 (2.1)*
ASI				
Family	0.28 (0.19)	0.18 (0.16)	0.31 (0.09)	0.24 (0.04)
Medical	0.35 (0.37)	0.26 (0.34)	0.05 (0.01)	0.17 (0.19)
Employment	0.61 (0.37)	0.57 (0.38)	0.59 (0.35)	0.34 (0.34)*
Psychiatric	0.46 (0.10)	0.19 (0.17)***	0.45 (0.09)	0.23 (0.25)
Legal	0.13 (0.17)	0.07 (0.07)	0.16 (0.22)	0.06 (0.13)
Drug	0.20 (0.08)	0.08 (0.07)***	0.15 (0.06)	0.04 (0.05)*
Alcohol	0.27 (0.22)	0.11 (0.16)***	0.29 (0.20)	0.09 (0.08)*

[a] $n = 15$.
[b] $n = 7$.
[c] Marginally significant difference between CAPS total at pretreatment and follow-up ($t = 2.28$, df = 6, $p = 0.06$).
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

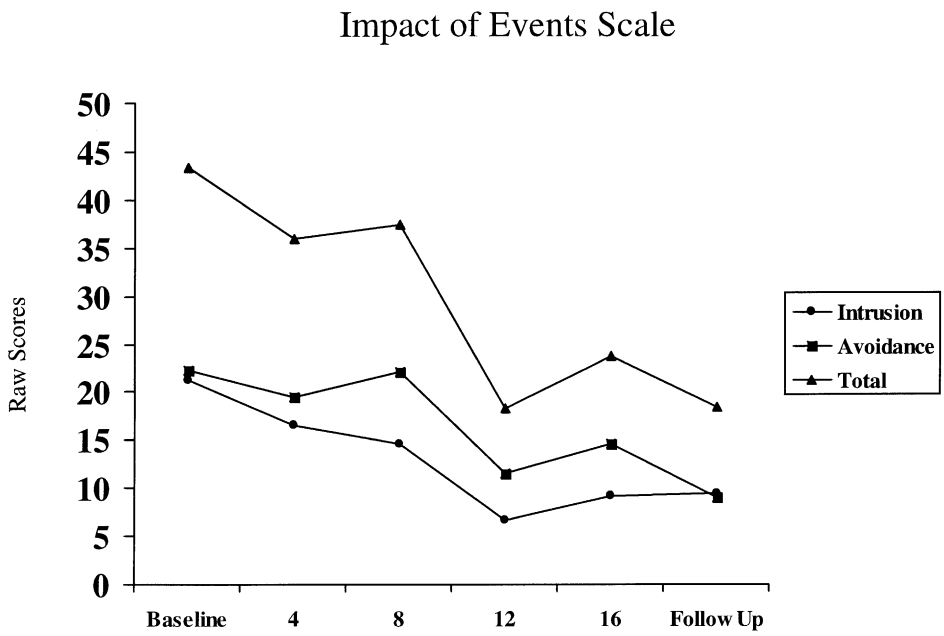

Fig. 1. Change over time in PTSD symptoms as measured by the IES.

problems on fewer days at posttreatment in comparison to pretreatment (14.08 vs. 3.62, $t = 3.06$, df = 12, $p < 0.01$). There were no differences in ASI scores between baseline and 6-month follow-up in treatment noncompleters.

Examination of UDS data revealed a decrease in the percentage of positive UDS tests for any drug of abuse over the course of treatment. Differences, however, were not statistically significant. The already low baseline percentage of positive UDS tests (12.8%) at baseline may have created a ceiling effect. The percent of UDS tests that were negative for cocaine ranged from 76.9% at week 12 to 100% at week 16. The average percent negative for cocaine during the first half of treatment was 87.8%; during the last half of treatment, it was 90.3%.

3.1.5. Effect size

The treatment effect size (ES) was calculated by using Glass's delta: the mean change from baseline to treatment termination divided by the *sd* of that change (>1.0 is clinically significant). For the PTSD posttreatment ratings, the ESs were: 1.48 for the CAPS total, 1.28 for the MISS total, and 0.74 for the IES total score. At 6-month follow-up, the ESs were: 1.75 for the CAPS total, 4.15 for the MISS total, and 1.42 for the IES total. For the posttreatment ASI drug and alcohol composite scores, the ESs were 1.50 and 0.74. At 6-month follow-up, the ESs were 1.83 for the drug composite and 1.00 for the alcohol composite scores. In addition, the ASI psychiatric composite score ES was 2.70 at posttreatment and 2.40 at 6-month follow-up. The average treatment effect size was 1.80 for PTSD symptoms, 1.65 for drug use severity, and 1.26 for drug and alcohol use severity.

4. Discussion

In this study, the safety and tolerability of exposure therapy in treating PTSD in cocaine-dependent individuals was investigated. This study constitutes a preliminary investigation of exposure treatment in this population and caution in interpreting the findings is warranted. The findings revealed significant improvement during treatment that was maintained over a 6-month follow-up period for treatment completers. Although there was no control group, these results suggest that some individuals with PTSD and comorbid cocaine dependence can be successfully treated with exposure therapy and that exposure therapy does not necessarily increase the risk for relapse. While the dropout rate in the study was high, it was lower than those seen in other studies of psychotherapy treatments of cocaine dependence (Crits-Christoph et al., 1999). In addition, the timing of dropout in the current study is not consistent with the notion that the exposure treatment was the precipitant of relapse or dropout. The lack of increase in positive UDS tests during or after exposure therapy also argues against the belief that exposure therapy precipitated relapse to drug use.

Baseline comparisons of sociodemographic, PTSD symptomatology, and cocaine use severity revealed only two significant differences between individuals who completed treatment and those who did not complete treatment. In comparison to treatment completers, noncompleters had significantly less education and reported significantly higher levels of avoidance on the IES. Although a significant difference in avoidance on the CAPS was not observed, the self-reported avoidance may reflect a stronger subjective

tendency or desire to avoid confronting the trauma, which might lead to an increased risk for attrition from exposure-based treatment. Similar to the findings of McMahon, Kouzekanani, and Malow (1999), there were no significant differences between groups in baseline cocaine use.

Other studies involving PTSD treatments reveal mixed results with regard to differences in treatment completers and noncompleters. Marks, Lovell, Noshirvani, Livanou, and Thrasher (1998) investigated differences in demographic, trauma history, and psychiatric severity among individuals with PTSD (14% met criteria for alcohol abuse or dependence). Treatment completers (defined as attending ten sessions of exposure and/or cognitive restructuring therapy during a mean of 16 weeks) had more past psychological treatment and more severe CAPS scores than noncompleters. Selection criteria were applied, however, to exclude patients with severe substance use (e.g., ingestion of 30 or more alcohol units a week). Another study involving the treatment of women with PTSD and comorbid substance dependence (24% cocaine dependent) found similar results (Najavits, Weiss, & Shaw, 1998b). In comparison to treatment dropouts ($n = 10$), treatment completers ($n = 17$) had more severe global symptom profiles (e.g., PTSD symptoms, interpersonal sensitivity, social adjustment) (Najavits et al., 1998b). In addition, completers were more engaged in the treatment, showed greater satisfaction with the treatment, and attended more self-help groups than did noncompleters. Differences in subject characteristics (e.g., gender) and comorbidity may help explain the discrepancy. Further research is needed to help determine which individuals are at risk for attrition, and conversely, which are most likely to do well with exposure-based treatments.

Individuals who completed the treatment demonstrated significant reductions in intrusion, avoidance, and hyperarousal symptoms from baseline to end of treatment. Comparisons between baseline and follow-up assessment periods also revealed improvement in PTSD symptoms, particularly hyperarousal symptoms. In contrast, there was no difference in PTSD symptoms in the treatment noncompleters interviewed at 6-month follow-up. Among a sample of combat veterans with PTSD and comorbid substance use disorder, individuals reported that cocaine made their PTSD symptoms (particularly hyperarousal symptoms) worse (Bremner, Southwick, Darnell, & Charney, 1996). As such, the hyperarousal symptoms may be more likely than intrusive or avoidance symptoms to improve with reduced cocaine use. It is possible that the improvement in PTSD hyperarousal symptoms may be primarily due to cocaine abstinence, rather than to the exposure, or to the combination of both.

The average pre- to posttreatment effect size for PTSD symptoms across all measures and times for completers was 1.80. This finding is similar to previous reports in the literature involving individuals with PTSD and little or no comorbid SUDs. Van Etten and Taylor (1998) conducted a meta-analysis that involved 61 controlled pharmacologic and psychosocial interventions. In their analysis of controlled behavioral treatments for PTSD, all of which included some form of exposure therapy alone or in conjunction with stress-inoculation training, an ES of 1.93 for observer-reported and 1.63 for self-reported PTSD symptom improvement at follow-up was reported (Van Etten & Taylor, 1998). The different rates of SUD comorbidity in the samples used in the meta-analysis, however, did not appear to be controlled for. In a study involving prolonged exposure therapy among individuals with PTSD and no comorbid SUDs, Foa and colleagues (1999) noted an ES of 1.92 for observer-rated PTSD symptoms measured by the Posttraumatic Stress Disorder Symptom Scale. Finally, among a sample of individuals with PTSD (14% of whom met criteria for comorbid alcohol abuse or dependence) treated with exposure therapy, rates of improvement similar to those of the current study were observed (Marks et al., 1998). The ES for the CAPS was 1.30 for pre- to posttreatment, 1.50 pretreatment to 1-month follow-up, and 1.40 from pretreatment to 3 months follow-up. The reported ES for the CAPS at 6-month follow-up and the IES ES scores were, however, higher than those observed in the current study. Differences between comorbid SUDs in the Marks and colleagues (1998) sample and the present investigation may help explain the discrepancy. Consistent with previous investigations (Van Etten & Taylor, 1998), the findings of the current study revealed that observer-rated PTSD measures yielded larger ESs than self-report rated measures, with the exception of the MISS at follow-up.

Significant decrease in substance use severity during the treatment and follow-up periods was also found for completers. In comparison to baseline, the ASI drug and alcohol composite scores were significantly lower at the end of treatment and 6-month follow-up for treatment completers. In contrast, there was no change in ASI scores at 6 months in the few treatment noncompleters we were able to interview. In addition, the percent of cocaine-positive UDS tests showed some decrease over the course of treatment for completers.

The continued decrease in substance use severity is particularly important as it relates to the use of exposure-based therapy in the treatment of PTSD for individuals with comorbid SUDs. In this study, exposure was initiated at session 7, midway through the protocol. Comparisons between the percent positive UDS tests during relapse prevention and exposure-based therapeutic sessions indicate that patients' cocaine use did not increase during the exposure therapy. The 6-month follow-up data suggest continued improvement in cocaine and alcohol use for completers.

The retention rate for the current study was similar to the rate reported by Najavits et al. (1998b), who found that 63% of individuals with PTSD and comorbid SUDs attended six or more therapy sessions. In our study, 69.2% of the sample attended 25% of the therapy sessions. While the treatment completion rate of 38.5% in the current study was low, it

was higher than the 28.1% treatment completion rate reported by the Collaborative Cocaine Treatment Study (Crits-Christoph et al., 1999), which consisted of 487 cocaine-dependent outpatients who received various psychosocial interventions. Furthermore, the timing of attrition in the present investigation is important to consider as it relates to the use of exposure therapy. The majority (75%) of individuals who did not complete treatment dropped out prior to the initial session of exposure therapy. As such, for the majority of patients the decision to discontinue therapy was not a direct result of exposure therapy. However, the fact that six individuals in the study dropped out after the initiation of exposure cannot be overlooked. It is not known whether these patients dropped out because of the exposure, or if the patients who dropped out prior to the initiation of exposure did so because of worries or concerns about the procedure. However, no patients who dropped out of the study (before or after exposure) reported this as the reason for dropout.

Two additional areas of improvement were also noted in the findings. Significant improvement from pre- to posttreatment in depression was found, with the end state scores being similar to those noted by Foa et al. (1999) in a group of women with PTSD and no comorbid substance use disorders who received exposure therapy. In addition, a delayed improvement in occupational impairment, as evidenced by significantly lower scores on the ASI employment subscale score from pretreatment to follow-up was observed. ASI scores associated with other areas of functioning (e.g., family, social, legal impairment) decreased over time but did not reach statistical significance. These areas may be more resistant to change or may require more specifically tailored interventions. A recent investigation comparing women with PTSD and either alcohol or cocaine dependence (Brady, Back, Sonne, Dansky, & Diaz-Zuniga, 2000) reported that cocaine-dependent women suffered more social and occupational impairment than women with alcohol-dependence. Hence, modalities that meet unique, substance-specific treatment needs may be in order.

Several important limitations of the current study should be noted. This study was a psychotherapy developmental trial and, as such did not include a control group. Thus, all findings are tentative, as it cannot be determined whether the observed improvements in PTSD and cocaine use were due to the treatment or another factor or set of factors (e.g., natural course of disorders, passage of time). The high dropout rate and lack of posttreatment and follow-up data on those patients who did drop out is also a drawback, as the patients who completed treatment were a self-selected group. Randomized control studies and comparative treatment outcome studies are needed. In addition, the current study included only cocaine-dependent individuals, and the findings may not be generalizable to individuals with PTSD and other types of comorbid SUDs. Finally, the small sample size and potential for inflation in Type I error must be considered.

In summary, the findings from this study are encouraging and provide preliminary support for the use of exposure-based therapies among some individuals with PTSD and comorbid cocaine dependence. When exposure therapy is provided to treat PTSD symptoms in conjunction with sufficient attention to the cocaine use disorder, it appears that both disorders can experience substantial improvement in some individuals. Caution is warranted given the early stage of treatment development and the methodological limitations of the study. Not all cocaine-dependent patients with PTSD will benefit from exposure therapy and careful consideration prior to beginning exposure, as with any other form of psychosocial or pharmacologic intervention, is warranted (see accompanying article for suggested criteria). Further investigation of the use of exposure-based psychotherapy, including predictors of success and contraindications to use, would be of great benefit in improving treatment options for the substantial subgroup of substance-dependent individuals who have comorbid PTSD.

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