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TREATMENT OF POSTTRAUMATIC STRESS DISORDER IN U.S. COMBAT VETERANS: A META-ANALYTIC REVIEW[1]

JASON GOODSON AND AMY HELSTROM

Philadelphia VAMC
University of Pennsylvania

JACQUELINE M. HALPERN, MICHAEL P. FERENSCHAK, AND SETH J. GILLIHAN

University of Pennsylvania

MARK B. POWERS

Southern Methodist University

Summary.—Among U.S. veterans who have been exposed to combat-related trauma, significantly elevated rates of posttraumatic stress disorder (PTSD) are reported. Veterans with PTSD are treated for the disorder at Veterans Affairs (VA) hospitals through a variety of psychotherapeutic interventions. Given the significant impairment associated with PTSD, it is imperative to assess the typical treatment response associated with these interventions. 24 studies with a total sample size of 1,742 participants were quantitatively reviewed. Overall, analyses showed a medium between-groups effect size for active treatments compared to control conditions. Thus, the average VA-treated patient fared better than 66% of patients in control conditions. VA treatments incorporating exposure-based interventions showed the highest within-group effect size. Effect sizes were not moderated by treatment dose, sample size, or publication year. Findings are encouraging for treatment seekers for combat-related PTSD in VA settings.

Posttraumatic stress disorder (PTSD) includes distressing and debilitating symptoms comprising re-experiencing trauma, emotional numbing, and hyperarousal. According to recent estimates, as many as 25% of veterans returning from combat operations in Iraq will experience significant symptoms of PTSD (Milliken, Auchterlonie, & Hoge, 2007). In addition to the core symptoms of PTSD, trauma-related sequelae extend to a variety of physical health consequences, overall disability, and psychiatric comorbidity including depressive and substance use disorders (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Physical health problems associated with PTSD include chronic pain, back problems, and cardiovascular disease (Beckham, Crawford, Feldman, Kirby, Hertzberg, Davidson, *et al.*, 1997; Asmundson, Coons, Taylor, & Katz, 2002). In short, finding effective treatments for veterans reporting this spectrum of difficulties is critical.

[1]Address correspondence to Mark B. Powers, Ph.D., Department of Psychology, Southern Methodist University, 6116 North Central Expressway, Suite 1100, Dallas, TX 75206 or e-mail (mbpowers@smu.edu).

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Over the last three decades there have been systematic efforts to develop, test, and implement empirically supported treatments for PTSD. Rigorous meta-analyses have demonstrated that the effect of psychotherapy across various treatment modalities is generally effective in decreasing PTSD symptoms and improving psychological health (Sherman, 1998). For example, a meta-analysis of 61 medication and psychological treatment outcome trials suggested that psychotherapies, particularly behavior therapy and Eye Movement Desensitization and Reprocessing (EMDR), were effective interventions for PTSD (Van Etten & Taylor, 1998). Another meta-analysis of 38 randomized controlled trials of psychological treatments for PTSD also demonstrated the efficacy of cognitive-behavior therapy and EMDR (Bisson, Ehlers, Matthews, Pilling, Richards, & Turner, 2007). Although some of the studies in these meta-analyses included treatment outcome trials with combat veterans (Sherman, 1998), no meta-analysis has summarized treatment outcome trials among veterans with combat-related trauma. For this reason it cannot be known whether the effect sizes from existing meta-analyses are relevant to the treatment of PTSD in this population.

Existing reports have produced inconsistent findings on the effectiveness of PTSD treatment for veterans. Several treatment outcome studies have found moderate to large treatment effects with this population (Peniston, 1986; Cooper & Clum, 1989; Peniston & Kulkosky, 1991; Monson, Schnurr, Resick, Friedman, Young-Xu, & Stevens, 2006; Ready, Thomas, Worley, Backscheider, Harvey, Baltzell, *et al.*, 2008). For example, Monson, *et al.* reported a between-groups effect size (*ES*) of $d = 1.07$, with 50% of their sample showing a reliable change in their PTSD symptoms. However, meta-analyses of PTSD treatment studies that have included some studies with combat-related PTSD have raised concerns about the effectiveness of PTSD treatments among this population (Frueh, Grubaugh, Elhai, & Buckley, 2007). For example, moderator analyses in previous meta-analyses showed smaller effect sizes among combat veterans with PTSD than with civilian populations (Bradley, Greene, Russ, Dutra, & Westen, 2005; Bisson, *et al.*, 2007). Similarly, several studies of VA programs for PTSD treatment showed variable posttreatment gains and minimal to nonexistent gains at follow-up assessments (Hammarberg & Silver, 1994; Johnson, Rosenheck, Fontana, Lubin, Charney, & Southwick, 1996; Fontana & Rosenheck, 1997; Johnson, Lubin, & Corn, 1999; Johnson, Lubin, Rosenheck, Fontana, Charney, & Southwick, 1999).

Among several speculations that have been put forth to explain these findings, one is that the smaller treatment gains are an artifact of the Veterans Health Administration's (VHA) disability program; monetary incentives associated with the program are hypothesized to impede the

recovery process through secondary gains (rewarding veterans for continuing to report PTSD symptoms; Frueh, *et al.*, 2007). Some researchers have reported evidence in support of this hypothesis (Fontana & Rosenheck, 1997) while others have not (Jordan, Nunley, & Cook, 1992; Laffaye, Rosen, Schnurr, & Friedman, 2007).

It also has been suggested that the increased chronicity of disease and severity of symptoms associated with combat-related PTSD may adversely affect veterans' responses to treatment. Indeed, some studies have found that symptom severity is a marker of poorer outcome in psychotherapy (Durham, Allan, & Hackett, 1997; Westen & Morrison, 2001; Durham, Fisher, Dow, Sharp, Power, Swan, *et al.*, 2004). Disease chronicity may be particularly relevant to Vietnam veterans, many of whom have been living with persistent symptoms of PTSD for 40 years (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Additionally, a number of researchers have suggested that veterans with PTSD are more likely to present with comorbid physical and psychiatric conditions, which may pose additional treatment challenges. For example, combat-related PTSD is associated with significantly elevated incidence of substance use (73–84%), major depression (26–68%), dysthymia (21–34%), antisocial and borderline personality disorders (21–76%), and other anxiety disorders (Frueh, Turner, Beidel, Mirabella, & Jones, 1996). Similarly, PTSD and chronic pain-related problems co-occur at a rate as high as 80% among inpatient Vietnam veterans (White & Faustman, 1989).

It also has been suggested that treatment of veterans with combat-related PTSD may be hindered by factors specific to combat-related trauma. For instance, combat veterans may experience greater guilt and shame about their combat-related actions, rendering symptoms less amenable to treatment interventions (Foa & Meadows, 1997). Additionally, combat veterans with PTSD often have been exposed to a greater number of traumas than have civilians with PTSD, so their symptoms may be more resistant to treatment.

Finally, poorer outcomes among combat veterans with PTSD also may reflect a lack of evidence-based treatment programs in some VA settings (Turner, Beidel, & Frueh, 2005). The present review of literature found that the following interventions (among others) were being used for PTSD treatment: milieu therapy, skills building, EMDR, exposure, systematic desensitization, meaning-based interventions, stress management, supportive counseling, behavioral activation, long-term inpatient care, general psychiatric care, and interpersonal skills development. While a number of the above treatments may be effective interventions for veterans, the long list points to the need to evaluate how effective are currently delivered PTSD treatments for veterans. In light of this concern, consider-

able efforts are being made to disseminate evidence-based treatments for PTSD (i.e., Prolonged Exposure Therapy and Cognitive Processing Therapy) in VA hospitals. Both of these treatment rollout initiatives have standardized consultation and supervision to aid VA providers in the effective delivery of the primary treatment components. Early reports from these dissemination efforts have shown promising results for both Prolonged Exposure and Cognitive Processing therapies (Karlin, Ruzek, Chard, Eftekhari, Monson, Hembree, *et al.*, 2010).

Apparently, no systematic review and meta-analysis for combat-related PTSD in VA settings has been conducted. Such a study would provide a summary of research evidence and would provide an index of the VA's effectiveness in the treatment of combat-related PTSD. Thus the current study had two primary aims: (1) to estimate the effectiveness of VA treatments for combat-related PTSD and (2) to estimate the effectiveness of specific modalities of VA treatments for combat-related PTSD. It was hypothesized that: (1) VA treatments for combat-related PTSD would be demonstrated to be effective and (2) outcomes with evidence-based treatments for combat-related PTSD would be associated with larger effect sizes.

Method

Search Criteria

To conduct a thorough review and analysis of combat veteran PTSD outcome treatment literature, a broad search included PsycINFO, Medline, PILOTS, and Psychology and Behavioral Sciences Collections, using the terms "PTSD," "veterans," and "treatment." Also searched were bibliographies from obtained articles for additional references and authors were contacted for emerging publications. These initial search strategies produced 1,126 potential articles. Examination of the abstracts subsequently identified 22 relevant published articles and two emerging publications. Thus, a total of 24 studies was included in the current review and meta-analysis.

Study Selection

The following were criteria of inclusion: (1) at least one outcome variable was specific to PTSD symptoms, (2) adequate statistical data were present to calculate effect sizes, (3) participants were combat veterans with a diagnosis of PTSD, and (4) the treatment was carried out in a VA setting.

If procedures did not explicitly state that participants had been diagnosed with combat-related PTSD, studies were included only if all participants had served in official combat theaters (e.g., served in Vietnam during the official Vietnam era) and had diagnoses of PTSD. Studies of participants with "military-related PTSD" were included only if the ma-

jority of the participants (i.e., >75%) were combat veterans with PTSD. Studies which included combat veterans with subclinical PTSD (did not meet full diagnostic criteria or scored below cutoffs on screening measures) were not included.

In addition, both open and controlled trials were included in the current analyses. Several factors were considered in making this decision. First, there is a paucity of well-controlled treatment outcome trials within the VA (i.e., 10). Given that the majority of PTSD treatment studies in the VA are open trials, including only well-controlled studies would give an incomplete portrayal of PTSD treatment being provided in the VA. Also, including only well-controlled research trials may result in a bias against less established, but perhaps innovative and promising, new treatments.

Procedure

The coding of study characteristics and calculation of effect sizes were carried out in two parts. The first part was a systematic review of the literature and calculation of within- and between-groups effect sizes (*ES*). In this section study, characteristics of all 24 studies were coded and an overall within-group *ES* was calculated. In addition, studies were classified into categories, each of these categories was reviewed, and corresponding effect sizes were calculated. The categories included: (1) exposure-based studies, (2) other cognitive-behavioral studies, (3) inpatient studies, and (4) miscellaneous treatment. A liberal definition was used for inclusion as an exposure-based study intervention with a significant component of exposure therapy, which included EMDR studies and one systematic desensitization study. EMDR studies were included, given the large amount of exposure in this treatment protocol and the findings that eye movements do not add to treatment outcomes above and beyond the components of exposure (Sanderson & Carpenter, 1992; Renfrey & Spates, 1994). Likewise, the study of systematic desensitization was included because exposure is an integral part of this modality.

Controlled trials were separately meta-analyzed using comprehensive meta-analysis (Borenstein & Rothstein, 1999). Comprehensive meta-analysis is a program funded by the National Institutes of Health Small Business Innovation Research (SBIR) program. These analyses included between-groups *ES* calculations, a test of study heterogeneity, assessment of possible publication bias, and meta-regressions of potential effect-size moderators. Heterogeneity here refers to the variation in study outcomes between studies. The measure of heterogeneity is Cochran's Q, which is calculated as the weighted sum of squared differences between individual study effects and the pooled effect across studies, with the weights being those used in the pooling method (DerSimonian & Laird, 1986; Gavaghan, Moore, & McQuay, 2000; Higgins, Thompson, Deeks, & Altman, 2003). If

Cochran's Q is significant, then a random effects meta-analysis is suggested. However, many investigators consider the random effects approach to be preferred for all meta-analyses (DerSimonian & Laird 1986; Fleiss & Gross, 1991; Ades & Higgins, 2005). Meta-regression measures the potential moderating effect variables may have on effect sizes (Morton, Adams, Suttorp, & Shekelle, 2004). These analyses served as validity checks on the findings of the larger systematic review of the literature. Assessment of publication bias was completed because several authors suggest there may be a potential discrepancy between the number of published trials and the total number that are completed (Sterling, 1959; McNemar, 1960; Smart, 1964; Bakan, 1967). Therefore, any meta-analysis of published studies may be missing nonsignificant studies and thus overestimate the overall effect size. Rosenthal (1991) and others have called this confound the "File Drawer Problem." A conservative method of addressing this problem is to assume that the effect sizes of all current or future *unpublished* studies are equal to 0 and compute the number of such studies it would require to reduce the overall effect size to a nonsignificant level (Rosenthal & Rubin, 1988). This value may be referred to as the "fail-safe *N*."

Rosenthal (1991) suggested the following equation to compute a fail-safe *N*:

$$X = \frac{K(K\overline{Z}^2 - 2.706)}{2.706},$$

where K is the number of studies in the meta-analysis and $\overline{Z}$ is the mean Z obtained from the K studies. Rosenthal (1991) suggested that findings may be considered robust if the required number of studies (X) to reduce the overall effect size to a nonsignificant level exceeded $5K + 10$ which in this study would be 60.

All effect sizes were calculated using Cohen's *d*. Effect sizes were then interpreted with Cohen's convention of small (0.2), medium (0.5), and large (0.8) effects (Cohen, 1988). Within-group *ES* were calculated by subtracting the posttreatment mean from the pretreatment mean and dividing by the pooled standard deviation.

Between-groups effect sizes for each study were computed using Cohen's *d* (Rosenthal, 1991). When the necessary data were available, Cohen's *d* was computed with a pooled standard deviation using the following formula:

$$d = \frac{mean_1 - mean_2}{\sqrt{(SD_1^2 - SD_2^2)/2}} \quad .$$

If these data were not provided, Cohen's *d* was computed using conversion equations for significance tests (e.g., *t*, *F*; see Rosenthal, 1991). When studies reported more than one PTSD measure, the *ES* for each measure

was calculated, averaged across measures, and reported as a single combined *ES* for that particular study. When studies reported *ES*, these values were reported here, even when raw data were not made available. When studies used variables that comprised lists of self-report single-item symptoms, only those items that represented official DSM–IV diagnostic symptoms for PTSD were included in the *ES* calculations. For instance, if a study reported participants' ratings of anger, sleep, nightmares, depressed mood, feeling worthless, and psychological distress, effect sizes were calculated for anger, sleep, and nightmares. All effect sizes were corrected for small sample sizes according to Hedges and Olkin (1985). Therefore, a smaller sample size reduces the estimated effect size, helping control for different sample sizes across studies. These controlled effect sizes may then be conservatively interpreted with Cohen's (1988) convention of small (0.2), medium (0.5), and large (0.8) effects. The overall mean effect size for all of the studies combined was computed using the following formula:

$$\bar{d} = \frac{\sum w_j d_j}{\sum w_j},$$

where w_j is the weight for each study and d_j is the effect size for each study. Effect sizes were calculated with random effects models rather than fixed effects. The fixed effects analysis estimates the exact overall effect size based on the studies included—assuming this represents the entire population of studies. The random effects analysis estimates the overall effect size assuming the studies included are only a sample of the entire population of studies. Thus, the random effects analysis is preferred when attempting to generalize findings.

Results

A total of 24 studies was identified and included in the review. A brief summary of each study follows, including outcomes at posttreatment (and at follow-up if provided).

Albrecht (2007) administered Prolonged Exposure Therapy to examine if benchmarks derived from research could be attained in a typical outpatient veteran sample. Eighteen of 23 patients (17 male, 1 female) completed at least five imaginal exposure sessions, each lasting 30–45 min. Results indicated that those who completed treatment had a significant improvement in their PTSD symptoms.

Bolton, Lambert, Wolf, Raja, Varra, and Fisher (2004) gathered data from 197 male veterans with chronic and severe PTSD who agreed to complete assessments while enrolled in one of three groups: Understanding PTSD ($n = 105$), Stress Management ($n = 62$), and Anger Management ($n = 30$). Data were collected at Week 1 and Week 12 from each group. Re-

sults indicated a modest improvement in distress across groups, with more improvement rated on behavioral change and life satisfaction than core PTSD symptoms.

Bormann, Thorpe, and Wetherell (2008) randomly assigned 33 male veterans (29 completed) to a group-based spirituality intervention ($n = 14$) or a delayed treatment control condition ($n = 15$). Results demonstrated that the intervention led to increased quality of life and reduced PTSD symptom severity and psychological distress.

Carlson, Chemtob, Rusnak, Hedlund, and Muraoka (1998) randomly assigned 35 male combat veterans to one of three conditions: 12 sessions of EMDR ($n = 10$), 12 sessions of biofeedback-assisted relaxation ($n = 13$), or routine clinical care ($n = 12$). Results showed that EMDR had a statistically significant effect and that these gains were generally maintained at 3-mo. follow-up. Many of the effects were maintained at a 9-mo. follow-up, as well.

Cooper and Clum (1989) assigned 14 male Vietnam veterans with PTSD to a standard treatment control group or standard treatment plus imaginal flooding group. Participants received up to 14 sessions of flooding with a maximum of 1.5 hours per session and were assessed prior to treatment, after treatment, and at a 3-mo. follow-up. Results indicated that flooding increased the effectiveness of standard treatment, especially concerning re-experiencing symptoms and sleep disturbances. However, flooding was not shown to have any effect on depression, trait anxiety, or proneness to violence.

Donovan, Padin-Rivera, and Kowaliw (2001) studied 46 male Vietnam veterans who participated in a 12-wk. partial hospitalization treatment that addressed both PTSD and substance abuse. Results suggested that this integrative treatment approach was effective in reducing overall PTSD symptoms and addictive behavior at the end of treatment and during the follow-ups (6 and 12 mo.).

Dunn, Rehm, Schillaci, Souchek, Mehta, Ashton, *et al.* (2007) randomly chosen 101 male veterans with chronic combat-related PTSD and depressive disorder to an evidence-based depression treatment (self-management therapy; $n = 51$) or an active-control therapy ($n = 50$). Self-management therapy produced modestly greater clinical improvement than did the active-control therapy; however, these gains disappeared at follow-up (3, 6, and 12 mo.).

Fontana and Rosenheck (1997) compared three models of inpatient treatment for PTSD at the Department of Veterans Affairs: long-stay specialized inpatient PTSD units, short-stay specialized evaluation and brief-treatment PTSD units, and nonspecialized general psychiatric units. Data were collected from 785 male Vietnam veterans across 10 programs. All

models demonstrated improvement at the time of discharge; however, following discharge, symptoms regressed toward admission levels, especially in those who had been treated in long-stay PTSD units. Those in the short-stay and general psychiatric units showed significantly more improvement than did participants in long-stay units during follow-up (4, 8, and 12 mo.).

Glynn, Eth, Randolph, Foy, Urbaitis, Boxer, *et al.* (1999) examined a family-based skills-building intervention in 42 male veterans with chronic combat-related PTSD. The participants, along with a family member, were randomly assigned to one of three conditions: wait list ($n = 13$), 18 sessions of twice-weekly exposure therapy ($n = 12$), or 18 sessions of twice-weekly exposure therapy followed by 16 sessions of behavioral family therapy ($n = 17$). Results indicated that participation in exposure therapy reduced hyperarousal and re-experiencing symptoms; these gains were maintained at 6-mo. follow-up. Participation in family therapy had no extra effect on the veterans' PTSD symptoms.

Goodson (2008) reported on Prolonged Exposure (PE) with 25 veterans (3 females) at Behavioral Health outpatient clinics at the Philadelphia VA Medical Center. Results indicated significant reductions in PTSD Checklist–Military Version scores.

Hammarberg and Silver (1994) examined 39 male Vietnam veterans diagnosed with PTSD who were newly admitted into a comprehensive 90-day inpatient treatment program. In addition, the authors tracked two control groups: one of veterans previously diagnosed with and treated for PTSD ($n = 26$) and one of Vietnam veterans without PTSD and nonveterans ($n = 16$). Forty-eight percent of the completers showed significant reductions in PTSD symptoms as measured by the the Penn Inventory for PTSD, while the remaining showed no improvement (39%) or had a worsening of symptoms (11%). At the 1-yr. follow-up, those participants who benefited from treatment had not maintained their gains (i.e., scores on the Penn Inventory for PTSD returned to pretreatment levels). Jakupcak, Roberts, Martell, Mulick, Michael, Reed, *et al.* (2006) enrolled 11 veterans (9 completed; 1 female) seeking treatment at the VA outpatient PTSD clinic into the study protocol, which consisted of 16 weekly individual sessions of behavioral activation. Results showed a significant improvement in PTSD symptom severity.

Jensen (1994) randomly assigned 25 male Vietnam veterans with PTSD to an EMDR condition or a delayed-treatment control group. Overall, Jensen found that EMDR had little effect on PTSD symptoms, although it was effective in reducing in-session subjective anxiety.

Johnson, *et al.* (1999) examined 11 male veterans with PTSD over the course of a 4-mo. inpatient treatment program. The authors concluded

that those veterans who were more symptomatic at the time of their admission fared worse in the inpatient program as measured by the Mississippi PTSD scale.

Johnson, Lubin, Rosenheck, *et al.* (1999) compared the treatment outcome for two samples in an inpatient program at discharge and 4-, 8-, and 12-mo. follow-up. The authors first examined a mixture of Vietnam combat veterans with PTSD and general psychiatric patients ($n = 42$), and then they examined the same program using only male Vietnam combat veterans with PTSD ($n = 33$). Results indicated that veterans showed no improvement in condition at the 12-mo. follow-up and that no differences in outcome were found between the homogeneous or heterogeneous treatment environments.

Johnson, *et al.* (1996) examined the outcome of a 4-mo. intensive inpatient program for combat-related PTSD among 51 male Vietnam veterans. Participants were assessed at admission, discharge, and 6, 12, and 18 months after discharge. Results indicated an increase in symptoms from admission to follow-up and a decrease in violent behaviors and legal problems. Interpersonal relationships and overall morale were improved at discharge but returned to pretreatment levels 18 mo. after discharge.

Keane, Fairbank, Caddell, and Zimering (1989) randomly assigned 24 male Vietnam veterans with PTSD to either a group receiving 14 to 16 sessions of implosive (flooding) therapy or to a wait-list control group. Participants were assessed at pretreatment, posttreatment, and 6-mo. follow-up. Results indicated those receiving implosive therapy showed significant improvement across measures, as compared to the wait-list controls. Improvement in the implosive therapy was noted most on the re-experiencing symptoms of PTSD, anxiety, and depression.

Monson, *et al.* (2006) conducted a wait-list controlled trial of cognitive processing therapy with 60 veterans (54 males, 6 females) with chronic military-related PTSD. Participants were assessed at pretreatment, midtreatment, posttreatment, and 1-mo. follow-up. Results indicated significant improvements in PTSD and comorbid symptoms in the therapy condition, compared to the wait-list control.

Peniston (1986) randomly assigned 16 male Vietnam combat veterans with chronic PTSD to groups receiving either a modified version of EMG biofeedback-induced desensitization procedure or no treatment. Patients in the biofeedback condition significantly reduced their muscle tension and continued to show improvement in functioning over the 24-mo. follow-up period, as compared to no improvements in the no-treatment condition.

Rauch, Defever, Favorite, Duroe, Garrity, Martis, *et al.* (2009) conducted PE therapy with 10 veterans (8 men) with chronic PTSD. The mean

number of PE sessions was 12.7 with a range of 7 through 21. Results showed significant reductions in scores on the Posttraumatic Diagnostic Scale and the Beck Depression Inventory–II at posttreatment.

Ready, *et al.* (2008) examined group-based exposure therapy with 102 veterans (1 female) with PTSD. Participants attended 3 hr. of group therapy twice weekly for 16 to 18 wk. Results demonstrated that the intervention produced clinically significant and lasting reductions in PTSD symptoms for most patients at posttreatment and at the 6-mo. follow-up.

Russell, Silver, Rogers, and Darnell (2007) examined the effectiveness of EMDR with 72 veterans with PTSD (sex is not reported). Results indicated a significant improvement in PTSD symptoms at post-treatment.

Silver, Brooks, and Obenchain (1995) reported data on the effectiveness of an inpatient program for 100 male Vietnam veterans with PTSD. Participants received either standard programmatic treatment (control; $n = 55$), programmatic treatment plus EMDR ($n = 13$), programmatic treatment plus relaxation ($n = 9$), or programmatic treatment plus biofeedback ($n = 6$). Results of the standard programmatic treatment alone suggested significant improvements in PTSD symptoms at post-treatment. Further, those participants who received additional treatment interventions tended to show larger treatment gains than control participants.

Turner, *et al.* (2005) developed a multicomponent treatment program called trauma management therapy to enhance the effects of exposure for the treatment of PTSD. Participants were 15 male Vietnam combat veterans with PTSD. Results indicated that there was significant improvement in PTSD symptoms. See Table 1 for a summary of the characteristics for all 24 studies. Several of the studies included more than one experimental condition, which made a total of 32 conditions. Ten (42%) of the 24 studies were controlled trials with random or quasi-random assignment to some form of a control or comparison condition, while the remaining 14 (63%) were open-trial studies with no control group. The total number of participants across the 24 studies was $N = 1{,}742$ (range = 10–785) with a mean of 69.7 ($SD = 15.5$) per study. Within-group effect sizes were calculated across 24 study conditions. The mean weighted effect size from this calculation was $d = 0.43$. An effect size of this magnitude may be interpreted as a medium treatment effect (Cohen, 1988). A between-groups effect size was calculated across the 10 controlled studies. The mean weighted *ES* for this calculation was $d = 0.49$, which also is suggestive of a medium magnitude of effect (Cohen, 1988). As mentioned above, the 24 studies were classified into four general categories based on similar intervention components. Of the 24 studies, 12 (50%) were exposure-based studies, eight (33%) were studies carried out in PTSD inpatient treatment programs, two (8%) were other Cognitive Behavior Therapy studies (cognitive processing therapy

TABLE 1
Study Characteristics and Within- and Between-group Effect Sizes for PTSD Treatments

Study	Condition	*N*	Within-group Effect Size		Between-groups Effect Size	
			Posttreatment	Follow-up[a]	Posttreatment	Follow-up[a]
Albrecht (2007)	Prolonged Exposure	18	1.8			
Bolton, *et al.* (2004)	Psychoeducation & skills building	105	0.09			
Bormann, *et al.* (2008)	Spirituality	14	0.67			
	Delayed control	15	0.31			
Carlson, *et al.* (1998)	EMDR	10	1.16	0.89 (12)	0.56	.91 (9)
	Relaxation	13	0.45	0.52 (3)	−0.14	
	Routine care	12	0.62			
Cooper & Clum (1989)	Standard treatment + Flooding	7	1.6		0.95	.51 (3)
	Standard treatment	7	0.15			
Dunn, *et al.* (2007)	Self-management	51	0.06	0.25 (12)	0.20	.24 (12)
	Active control	50	−0.15	0.05 (12)		
Donovan, *et al.* (2001)	Inpatient PTSD program	46	0.67	0.63 (12)		
Fontana & Rosenheck (1997)	Long-stay PTSD unit	333	0.30			
	Short-stay PTSD unit	222	0.56			
	General psychiatric unit	230	0.57			
Goodson (2008)	Prolonged Exposure	25	1.4			
Glynn, *et al.* (1999)	Exposure	12				
	Exposure + behavioral family therapy	17	0.50		0.80	
	Wait-list control	13	0.52		0.55	
Hammarburg & Silver (1994)	Inpatient PTSD program	23	0.74			
Jakupcak, *et al.* (2006)	Behavioral activation	11	0.48			
Jensen (1994)	EMDR vs delayed control	25	−0.58		0.05	
Johnson, Lubin, & Corn (1999)	Inpatient PTSD program	11	−0.46			
Johnson, Lubin, Rosenheck, *et al.* (1999)	Inpatient–vets only	33	0.11	−0.07 (12)		
	Inpatient–mixed	42	0.36	−0.37 (12)		
Johnson, *et al.* (1996)	Inpatient PTSD program	51	−0.20	−0.33 (18)		

(continued on next page)

Note.—Studies listed in bold are controlled trials. EMDR = Eye Movement and Desensitization and Reprocessing; EMG – BAD = Electromyography Biofeedback Assisted Desensitization; Cohen (1988) *ES* interpretation: small (.2), medium (.5), large (.8). [a]Numbers in parentheses represent the number of months at follow-up.

TABLE 1 (cont'd)
Study Characteristics and Within- and Between-group Effect Sizes for PTSD Treatments

Study	Condition	*N*	Within-group Effect Size		Between-groups Effect Size	
			Posttreatment	Follow-up[a]	Posttreatment	Follow-up[a]
Keane, *et al.* (1989)	Implosive flooding-Wait-list control	24	0.59		0.23	
Monson, *et al.* (2006)	Cognitive Processing TherapyWait-list control	60			1.07	0.79 (1)
Peniston (1986)	EMG –BAD No treatment	16				1.58 (24)
Rauch, *et al.* (2009)	Prolonged Exposure	10	2.19			
Ready, *et al.* (2008)	Group exposure	102	0.98			
Russell, *et al.* (2007)	EMDR	72	2.2			
Scurfield, *et al.* (1990)	Inpatient PTSD program	86	0.25			
Silver, *et al.* (1995)	Inpatient + EMDR	13			0.46	
	Inpatient + relaxation	9			0.27	
	Inpatient + biofeedback	6			0.01	
	Inpatient	55				
Turner, *et al.* (2005)	Trauma management therapy	11	0.81			
Effect size			0.43		0.49	

Note.—Studies listed in bold are controlled trials. EMDR = Eye Movement and Desensitization and Reprocessing; EMG–BAD = Electromyography Biofeedback Assisted Desensitization; Cohen (1988) *ES* interpretation: small (.2), medium (.5), large (.8). [a]Numbers in parentheses represent the number of months at follow-up.

and trauma management therapy), and three (13%) were classified as miscellaneous treatment studies (spirituality-based intervention, behavioral activation intervention, and skills-based group intervention).

Exposure-based studies.—Twelve of the 24 (50%) studies included significant intervention components of exposure (Peniston, 1986; Cooper & Clum, 1989; Keane, *et al.*, 1989; Jensen, 1994; Silver, *et al.*, 1995; Carlson, *et al.*, 1998; Glynn, *et al.*, 1999; Albrecht, 2007; Russell, *et al.*, 2007; Goodson, 2008; Ready, *et al.*, 2008; Rauch, *et al.*, 2009). See Table 2 for characteristics of studies and effect sizes. Seven of the 12 studies (58%) were controlled trials, while five (42%) were open trials. Of the 12 exposure-based studies, four were EMDR, three were Prolonged Exposure, two were implosive therapy, one was a group-based exposure study, one was an exposure study with an augmentation of behavioral family therapy, and one was based on a systematic desensitization paradigm. The mean number

of participants across all 12 of these studies was 21.7 (*SD* = 24.0). The mean number of sessions across the exposure studies was 18.7 (*SD* = 14.1). Two studies did not report adequate statistical data to calculate within-group *ES* (Peniston, 1986; Silver, *et al.*, 1995) and one study incorporated two different exposure conditions (Glynn, *et al.*, 1999). As such, the within-group calculations for the exposure studies are based on 11 studies with a total of 12 conditions. As shown in Table 2, the mean within-group *ES* across all exposure conditions was 1.10 (range = −0.58 to 2.20). An effect size of this magnitude may be interpreted as a large treatment effect (Cohen, 1988). A between-groups *ES* was calculated for the seven controlled studies; the resulting *ES* of $d = 0.64$ may be interpreted as a medium treatment effect (Cohen, 1988).

Inpatient studies.—Seven of the 24 studies (29%) were carried out on inpatient PTSD treatment units (Hammarberg & Silver, 1994; Johnson, *et al.*, 1996; Fontana & Rosenheck, 1997; Johnson, *et al.*, 1999; Johnson, Lu-

TABLE 2
CHARACTERISTICS AND WITHIN- AND BETWEEN-GROUP PTSD EFFECT SIZES FOR EXPOSURE-BASED STUDIES

Study	Condition	*N*	Within-group Effect Size		Between-groups Effect Size	
			Posttreatment	Follow-up[a]	Posttreatment	Follow-up[a]
Albrecht (2007)	Prolonged Exposure	18	1.8			
Cooper & Clum (1989)	Standard treatment + Flooding	7	1.6		0.95	0.51 (3)
	Standard treatment	7	0.15			
Glynn, *et al.* (1999)	Exposure	12	0.50		0.80	
	Exposure + family behavioral therapy	17	0.52		0.55	
	Wait-list control	13				
Goodson (2008)	Prolonged Exposure	25	1.4			
Jensen (1994)	EMDR Delayed control	25	−0.58		0.05	
Keane, *et al.* (1989)	Implosive flooding-Wait-list control	24	0.59		0.23	
Rauch, *et al.* (2009)	Prolonged Exposure	10	2.19			
Ready, *et al.* (2008)	Group exposure	102	0.98			
Russell, *et al.* (2007)	EMDR	72	2.2			
Effect size			1.1		0.64	

Note.—Studies listed in bold are controlled trials. EMDR = Eye Movement and Desensitization and Reprocessing; Implosive = implosive flooding; *ES* = effect size; *ES* interpretation: small (.2), medium (.5), large (.8) (Cohen, 1988). [a]Numbers in parentheses represent the number of months at follow-up.

bin, Rosenheck, *et al.*, 1999; Donovan, *et al.*, 2001; Dunn, *et al.*, 2007). See Table 3 for study characteristics and effect sizes for individual studies. Of the seven studies, one was a controlled trial (Dunn, *et al.*, 2007) and the remainder were open trials. The mean number of participants in these studies was 106 ($SD = 10.5$) and the mean length of stay was 12.2 wk. ($SD = 4.2$) with a range of 4 to 16 wk. All seven studies were carried out in multicomponent and multidisciplinary PTSD treatment programs. The seven studies included a total of 11 different conditions. As illustrated in Table 3, the mean *ES* across all conditions was $d = 0.19$ (range = −0.54 to 0.74) which may be interpreted as a small treatment effect (Cohen, 1988). A between-groups *ES* was calculated for the one controlled trial, resulting in an effect size of $d = 0.20$, which is viewed as a small treatment effect (Cohen, 1988).

Other cognitive-behavioral studies.—Two of the 24 studies (8%) were classified as other cognitive-behavioral studies. These two studies included a controlled trial of cognitive processing therapy and an open trial of

TABLE 3
Characteristics and Within- and Between-group PTSD Effect Sizes for Inpatient Studies

Study	Condition	*N*	Within-group Effect Size		Between-group Effect Size	
			Posttreatment	Follow-up[a]	Posttreatment	Follow-up[a]
Dunn, *et al.* (2007)	Self-management	51	0.06	0.25 (12)	0.20	0.24 (12)
	Active control	50	−0.15	0.05 (12)		
Donovan, *et al.*(2001)	Inpatient PTSD program	46	0.67	0.63 (12)		
Fontana & Rosenheck (1997)	Long-stay PTSD unit	333	0.30			
	Short-stay PTSD unit	222	0.56			
	General psychiatric unit	230	0.57			
Hammarburg & Silver (1994)	Inpatient PTSD program	23	0.74			
Johnson, Lubin, & Corn (1999)	Inpatient PTSD program	11	−0.46			
Johnson, Lubin, Rosenheck, *et al.* (1999)	Inpatient–veterans only	33	0.11	−0.07 (12)		
	Inpatient–mixed	42	0.36	−0.37 (12)		
Johnson, *et al.* (1996)	Inpatient PTSD program	51	−0.20	−0.33 (18)		
Scurfield, *et al.* (1990)	Inpatient PTSD program	86	0.25			
Effect size			1.1		0.64	

Note.—Studies listed in bold are controlled trials. Effect size interpretation: small (0.2), medium (0.5), large (0.8) (Cohen, 1988). [a]Numbers in parentheses represent the number of months at follow-up.

trauma management therapy. The cognitive processing therapy study did not report adequate statistical data for calculation of a within-group *ES*. However, a between-group effect size calculation gave a value of $d = 1.07$, which is viewed as a large treatment effect (Cohen, 1988). The within-group *ES* for the trauma management therapy study also was large ($d = 0.81$; Cohen, 1988).

Miscellaneous treatment studies.—The remaining three studies were combined into a miscellaneous category, which included a controlled spirituality-based intervention (Bormann, *et al.*, 2008), an open behavioral activation study (Jakupcak, *et al.*, 2006), and an open skills-based group intervention (Bolton, *et al.*, 2004). Two of these studies reported adequate statistical data for calculation of within-group *ES* including the behavioral activation study ($d = 0.48$) and the spirituality-based intervention ($d = 0.67$). The between-groups *ES* for the spirituality-based intervention was $d = 0.56$. These are considered medium effect sizes according to Cohen (1988).

Meta-analytic Review

To validate the above findings further, additional analyses were carried out with controlled studies using comprehensive meta-analysis (Borenstein & Rothstein, 1999), a program funded by the National Institutes of Health's Small Business Innovation Research (SBIR) program. Table 4 provides a summary of the 10 controlled trials. The different types of intervention included seven (70%) exposure-based studies (i.e., implosive flooding or imaginal flooding, three EMDR studies, systematic desensitization, and an exposure study with two groups including exposure and exposure plus behavioral family therapy), one (10%) cognitive processing study, one (10%) spirituality-based intervention, and one (10%) skills-based intervention study. Control conditions were classified as treatment as usual or wait list. Six of the 10 studies utilized a wait-list control and four utilized treatment as usual. Consistent with prediction, results of a heterogeneity analysis were significant ($Q_9 = 6.27$, $p < .001$) and suggested that the random effects analyses were most appropriate for this study. As shown in Table 4, the mean between-groups medium *ES* for the 10 controlled studies was $d = 0.49$ ($SE = 0.11$, $p < .001$; 95%$CI = 0.27, 0.71$). Thus, the average participant receiving active treatment fared better than 69% of the control participants at posttreatment. Fig. 1 shows the overall between-group meta-analysis. For each study, the hash mark represents the effect size for that study and the horizontal line represents the 95% confidence interval. The middle line represents an effect size of "0" (i.e., no effect). Effect sizes to the right of this line show that the effect size favors the active treatment, while effect sizes to the left of this line show that the effect size favors the control condition. The confidence intervals can then be assessed

TABLE 4
Characteristics and Within- and Between-group PTSD Effect Sizes for Controlled Trials

Study	Condition	*N*	Within-group Effect Size		Between-groups Effect Size	
			Posttreatment	Follow-up[a]	Posttreatment	Follow-up[a]
Bormann, *et al.* (2008)	Spirituality	14	0.67			
	Delayed control	15	0.31			
Carlson, *et al.* (1998)	EMDR	10	1.16	0.89 (12)	0.56	0.91 (9)
	Relaxation	13	0.45	0.52 (3)	−0.14	
	Routine care	12	0.62			
Cooper & Clum (1989)	Standard treatment + Flooding	7	1.6		0.95	0.51 (3)
	Standard treatment	7	0.15			
Dunn, *et al.* (2007)	Self-management	51	0.06	0.25 (12)	0.20	0.24 (12)
	Active control	50	−0.15	0.05 (12)		
Glynn, *et al.* (1999)	Exposure	12	0.50		0.80	
	Exposure + family behavioral therapy	17	0.52		0.55	
	Wait list control	13				
Jensen (1994)	EMDR delayed control	25	−0.58		0.05	
Keane, *et al.* (1989)	Implosive flooding					
	Wait-list control	24	0.59		0.23	
Monson, *et al.* (2006)	Cognitive processing therapy	60			1.07	0.79 (1)
	Wait-list control					
Peniston (1986)	EMG–BAD	16				1.58 (24)
	No treatment					
Silver, *et al.* (1995)	Inpatient + EMDR	13				
	Inpatient + relaxation	9			0.46	
	Inpatient + biofeedback	6			0.27	
	Inpatient alone	55			0.01	
Effect size					0.49	

Note.—Studies listed in bold are controlled trials. EMDR = Eye Movement and Desensitization and Reprocessing; EMG–BAD = Electromyography Biofeedback Assisted Desensitization. Effect size interpretation: small (.2), medium (.5), large (.8) (Cohen, 1988). [a]Numbers in parentheses represent the number of months at follow-up.

if they are significant. Confidence intervals that overlap with the "0" line are considered nonsignificant. In addition, any confidence intervals that do not overlap with confidence intervals from other studies show these two effect sizes are significantly different. Finally, the diamond shape at the bottom shows the overall combined effect size and the width repre-

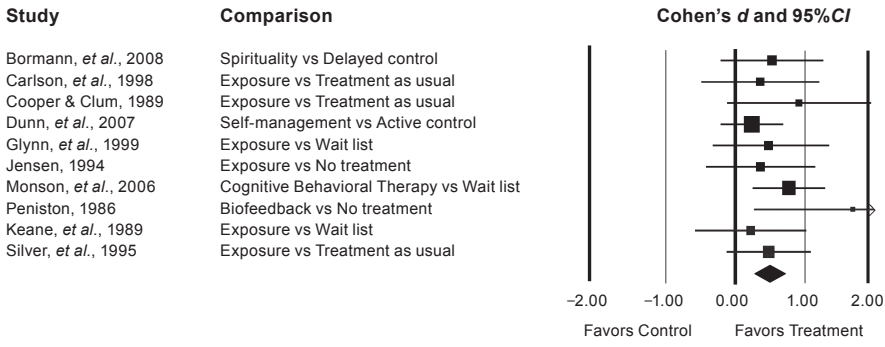

FIG. 1. Forest plot of treatment vs control effect sizes (Cohen's *d*) at posttreatment

sents the 95% confidence interval for this combined effect for all studies. If the diamond does not overlap with the "0" line, then the overall effect size is considered significant.

Three studies reported sufficient follow-up data for analysis. As shown in Table 4, the mean small-to-medium *ES* decreased to $d = 0.39$ ($SE = 0.17$, $p = .02$; 95%$CI = 0.06, 0.72$) at follow-up (Cohen, 1988).

Meta-regression analyses were carried out to assess whether the following variables were significantly associated with *ES*: quality of study, treatment dose, sample size, and year of publication. More specifically, examination of moderators was completed using unrestricted maximum likelihood meta regressions. These potential moderator variables were chosen based on suggestions in the literature and by previous PTSD meta-analyses (Thompson & Higgins, 2002; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). Meta-regression allows the effect of continuous, as well as categorical, characteristics of studies to be investigated as potential effect size moderators (Thompson & Higgins, 2002). Meta-regressions are similar to simple regressions. In the case of a meta-analysis, the putative moderator (e.g., dose) is regressed on the outcome variable (effect size). The potential moderators are characteristics of studies that might influence the size of intervention effect (effect size). The regression coefficient obtained from a meta-regression analysis will describe how the outcome variable (effect size) changes with each unit increase in the moderator (the potential effect modifier). The statistical significance of the regression coefficient is a test of whether there is a linear relationship between effect size and the moderator variable (Thompson & Higgins, 2002). The quality of the included studies was rated using a modified version of Jadad, Moore, Carroll, Jenkinson, Reynolds, Gavaghan, *et al.*'s quality assessment guidelines (1996). Jadad, *et al.* included three criteria for the quality assessment: random assignment to condition, double-blinding, and description

of withdrawals and dropouts. Given that this meta-analysis reviews psychological treatments, the second criterion could not be applied here because patients cannot remain blind to their psychological treatment condition; therefore this criterion was modified to require that the evaluators were blind to the therapeutic condition (i.e., single blind).

These meta-regression analyses revealed a significant effect for quality of study ($\beta = -0.11$, $p = .0003$), suggesting that higher quality studies were more likely to have smaller effect sizes. Tests were not significant for treatment dose ($\beta = -0.003$, $p = .61$), sample size ($\beta = -0.11$, $p = .15$), or year of publication ($\beta = -0.01$, $p = .13$). Finally, a failsafe *N* analysis revealed that 66 studies with $ES = 0$ would be needed to render the reported effect not significant. This result suggests that the results of this meta-analysis are robust.

Discussion

The current review and meta-analyses were conducted to investigate the effectiveness of psychological treatment at VA hospitals for combat veterans with PTSD. This investigation also provides a comparison of different treatment modalities used with this population. The review and meta-analysis of 24 treatment outcome studies reported here provide an estimate of the effectiveness of VA treatments for combat-related PTSD. Consistent with the hypothesis, VA treatments were moderately effective in reducing PTSD symptoms in combat veterans. In particular, across all 24 studies analyzed the within-group *ES* was $d = 0.43$, while across the 10 controlled studies, the between-groups *ES* was $d = 0.48$. Effect sizes of this magnitude, which fall in the conventionally defined "medium" range (Cohen, 1988), would suggest that well over half of the combat veterans receiving treatment at VA settings would show improvement following their treatment. It is worth noting that this overall *ES* is based on results from studies of empirically supported treatments, nonempirically supported treatments, as well as exploratory interventions (e.g., spirituality and behavioral activation for PTSD).

The second purpose of the present study was to provide a comparison of the different treatments used in VA settings for combat-related PTSD. Those treatment interventions with empirical support were hypothesized to be more effective than those without. Given the small number of studies available for this review, especially involving controlled trials, the conclusions should be considered tentative. Further caution is warranted as no statistical tests of significance were carried out to compare effect sizes among the differing modalities. With these caveats, it appears as if the hypothesis was supported. That is, treatments with accumulated empirical evidence or based on empirically derived models generated larger and more robust effect sizes than those without. Specifically, the mean within-

and between-group *ES* for exposure-based studies was $d = 1.10$ (large) and 0.64 (medium), respectively. Moreover, the effect size for cognitive processing therapy was $d = 1.00$ (large), while that for trauma-focused therapy (with primary cognitive-behavioral therapy components) was $d = 0.81$ (large). These findings stand in contrast to the small effect size for inpatient programs ($d = 0.19$) and the medium effect size across all 24 studies examined ($d = 0.43$).

Perhaps the clearest finding from this review is that treatments with significant exposure-based components were effective in treating combat-related PTSD, and as a broad category, appear to have the most empirical support. This finding is particularly impressive given that exposure-based interventions were tested most often with methodologically rigorous randomized controlled designs. Importantly, existing research has revealed hesitance to use exposure-based interventions for veterans with combat-related PTSD (Boudewyns & Shipley, 1983; Fontana & Rosenheck, 1993), which suggests that these interventions may be underutilized.

A comparison of the present findings with those from other meta-analyses (Bradley, *et al.*, 2005; Bisson, *et al.*, 2007) suggests that the PTSD treatment effects may be smaller for studies of veterans with combat-related PTSD compared to those found in other meta-analyses of civilians or both civilian and veteran populations. For example, Bradley, *et al.*'s meta-analysis review (2005) included four active treatment categories (exposure, cognitive behavior therapy, exposure plus cognitive therapy, and EMDR) and found a large mean within-group effect size of $d = 1.43$. Given that this meta-analysis primarily reviewed treatment studies with empirically supported components, the most meaningful comparison in the present study would be with studies of empirically supported components: exposure, cognitive processing, and trauma-management therapy. Interestingly, the large within-group effect size obtained from these studies was $d = 1.10$ and of a similar magnitude as that from the Bradley, *et al.* meta-analysis. This finding suggests that, for pre-post treatment changes among combat veterans in PTSD treatment, outcomes are similar to those of civilians in PTSD treatment interventions, with perhaps somewhat attenuated treatment effects.

A similar pattern was obtained for between-groups effect sizes. The mean between-groups effect size for the Bradley, *et al.* (2005) meta-analysis was $d = 1.04$ (large), while for the present study it was $d = 0.68$—again, a smaller *ES* by about one-third of a *SD* for the VA PTSD treatment. Similar differences in *ES* emerged in a comparison of the present findings with those of the Bisson, *et al.* (2007) meta-analysis. The authors reported a mean *ES* across five categories of treatment studies (trauma-focused Cognitive Behavioral Therapy, EMDR, Group Cognitive Behavioral Therapy,

and other treatments) of $d = 1.0$ (large). After matching on similar studies with combat veterans (to the extent possible), a between-groups effect size of $d = 0.60$ (medium) was obtained. Again, it appears that combat veterans respond somewhat less well than do civilians in well-controlled clinical trials. The lower effect sizes among veterans may be accounted for partly by the inclusion in the current meta-analysis of studies in which PTSD was not the primary target of the treatment (e.g., Bolton, *et al.*, 2004, post-treatment $d = 0.09$). This observation underscores the importance of delivering empirically supported interventions that target patients' PTSD. Additional work is necessary to provide more definitive information about the comparability of treatment effect sizes in veteran and civilian populations.

The current findings also underscore the need to develop more effective inpatient treatment programs for combat-related PTSD. The overall effect size for inpatient treatment studies was $d = 0.19$, which indicates a small treatment effect (Cohen, 1988). This *ES* corresponds roughly to a 3-point reduction on the 136-point Clinician-Administered PTSD Scale (CAPS). There may be several reasons for the smaller effect sizes for inpatient PTSD programs. First, this finding could represent a lack of reporting of outcome data by inpatient/residential programs. Indeed, early reports from inpatient/residential programs implementing Prolonged Exposure seem to be showing positive outcomes for those veterans who underwent the therapy in these settings (Reeder, 2008, unpublished data). Likewise, inpatient/residential programs with established Prolonged Exposure, Cognitive Behavioral Therapy, or EMDR components may have little incentive to publish outcome findings. Alternatively, it could be that those veterans in need of inpatient or residential treatment programs represent a more severe sub-population of combat-related PTSD veterans, both with respect to PTSD symptomatology as well as psychosocial stressors.

It also may be that inpatient treatment interventions are less effective than those delivered on an outpatient basis. In particular, the inpatient programs in this review included multicomponent treatment programs. For example, the inpatient treatment program used by Johnson, *et al.* (1996) included relaxation and anger management training, creative arts therapy, review of trauma (both individually and in groups), cognitive restructuring, and plans for reintegration into the community. While a comprehensive treatment approach may have intuitive appeal, it could also dilute the most effective treatment components. It may also be speculated that inpatient or residential treatment programs, which include an abundance of safety and support, are not an ideal setting for effectively treating PTSD. For instance, Foa and Kozak suggested in 1986 that successful treatment of chronic PTSD requires both activation of the fear structure and availability of corrective information. Perhaps perception of safety within

inpatient or residential treatment settings does not allow adequate activation of the fear structure. Whatever the reason, it is evident that outcomes from inpatient and/or residential treatment programs included in the current study were marginally effective in treating combat-related PTSD.

Meta-regression analyses showed that effect sizes were not moderated by treatment dose (hours of treatment), sample size, or publication year. This result suggests that the findings may be considered particularly robust and consistent over time. However, study quality ratings were associated with effect sizes. More specifically, higher quality studies showed smaller effect sizes. At first this finding may appear counterintuitive. However, higher quality studies consistently are associated with smaller effect sizes in the literature (Chalmers, Celano, Sacks, & Smith, 1983; Schulz, Chalmers, Hayes, & Altman, 1995; Moher, Pham, Jones, Cook, Jadad, Moher, *et al.*, 1998). Lower quality studies are by definition more vulnerable to threats to internal validity (Schulz, 1995; Jadad, *et al.*, 1996; Juni, Witschi, Bloch, & Egger, 1999). For example, without adequate randomization and independent evaluators, treatment effects may be magnified artificially due to individual differences, demand characteristics, and experimenter effects. Thus, this finding is common in meta-analyses but still concerning and suggests the need for continued quality research in VA settings.

One of the limitations of the current study was the lack of reported intent-to-treat analyses. Of the 24 studies included, only three reported data on intent-to-treat analyses (Monson, *et al.*, 2006; Dunn, *et al.*, 2007; Ready, *et al.*, 2008). As a rule, fewer than 10 studies for meta-analysis renders suspect the reliability of resulting effect sizes (Rosenthal, 1991). To facilitate comparison across all studies, effect sizes were calculated based on completer analyses. However, completer analyses may overestimate the actual size of the treatment effects. Although this is common in most meta-analyses, caution is merited in the interpretation of these results until more data with intent-to-treat analyses are available. Of the 24 studies included, only three (Monson, *et al.*, 2006; Dunn, *et al.*, 2007; Ready, *et al.*, 2008) reported data on intent-to-treat analyses. To facilitate comparison across all studies, we reported effect sizes based only on completer analyses. This is a limitation of the current study as the use of completer analyses only may overestimate the actual size of the treatment effect and may provide a less realistic picture of the actual treatment effects.

Conclusions and Directions for Study

Overall, one may infer from findings of the current review that combat-related PTSD is amenable to treatment interventions and can be treated effectively in VA settings. Also, evidence-based treatments, especially exposure, appear to be most effective in treating combat-related PTSD.

Studies of inpatient programs for PTSD also were found to be associated with smaller effect sizes.

As with all reviews and meta-analyses, results are based on decisions made about inclusion of each study and classifications for comparisons. For the current study, EMDR and systematic desensitization were classified as exposure-based treatment studies. Previous meta-analyses have tended to include these studies in separate categories. The present classification system may represent a more liberal definition of exposure, which must be taken into account when considering the present findings.

From the current results, several areas of future research may be worth pursuing. First, obtaining outcome data from inpatient and/or residential programs utilizing evidence-based treatments should be encouraged. Second, some treatments appear promising but are not yet widely validated in the VA setting (e.g., spirituality based interventions, trauma-management therapy) and may warrant additional research trials. Additionally, while these results suggest that VA treatments are largely effective, it may be beneficial to assess institutional factors that account for the apparently smaller treatment gains among combat-related PTSD veterans than those for civilian outcomes.

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