Clinical Psychology and Psychotherapy
Clin. Psychol. Psychother. **5**, 126–144 (1998)

Comparative Efficacy of Treatments for Post-traumatic Stress Disorder: A Meta-Analysis

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A meta-analysis was conducted on 61 treatment outcome trials for post-traumatic stress disorder (PTSD). Conditions included drug therapies (TCAs, carbamazepine, MAOIs, SSRIs, and BDZs), psychological therapies (behaviour therapy, Eye-Movement Desensitization and Reprocessing (EMDR), relaxation training, hypnotherapy, and dynamic therapy), and control conditions (pill placebo, wait-list controls, supportive psychotherapies, and non-saccade EMDR control). Psychological therapies had significantly lower drop-out rates than pharmacotherapies (14% versus 32%), with attrition being uniformly low across all psychological therapies. In terms of symptom reduction, psychological therapies were more effective than drug therapies, and both were more effective than controls. Among the drug therapies, the SSRIs and carbamazepine had the greatest effect sizes, although the latter was based upon a single trial. Among the psychological therapies, behaviour therapy and EMDR were most effective, and generally equally so. The most effective psychological therapies and drug therapies were generally equally effective. Differences across treatment conditions were generally evident across symptom domains, with little matching of symptom domain to treatment type. However, SSRIs had some advantage over psychological therapies in treating depression. Follow-up results were not available for most treatments, but available data indicates that treatment effects for behaviour therapy and EMDR are maintained at 15-week follow-up.

INTRODUCTION

Post-traumatic stress disorder (PTSD) is characterized by three clusters of symptoms, which arise after the person is exposed to a traumatic stressor. The clusters are (1) recurrent reexperiencing of the traumatic event (e.g. flashbacks, nightmares, intrusive thoughts) (2) avoidance of trauma-related stimuli and numbing of general responsiveness, and (3) persistent hyperarousal (e.g. hypervigilance, exaggerated startle response: American Psychiatric Association (APA), 1994). PTSD is often chronic, and persists for at least 1 year after the trauma in approximately 50% of cases (Davidson *et al*., 1996). The most common precipitating events are combat trauma, physical and sexual assault, natural disasters, and motor vehicle accidents (Breslau *et al*., 1991; Davidson *et al*., 1991; Norris, 1992). Community-based studies indicate that PTSD has a lifetime prevalence of 1 to 14%, depending on

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Contract grant sponsor: Medical Research Council of Canada.
Contract grant sponsor: National Institute on Drug Abuse.

CCC 1063–3995/98/030126–19$17.50

diagnostic methods and type of population, and not surprisingly occurs at a much higher rate (3 to 58%) in people who are at risk for exposure to traumatic events (e.g. combat veterans, victims of natural disasters or criminal violence: APA, 1994).

Several forms of treatment have been applied to PTSD. Many treatments seem promising, although the literature currently provides no clear indication as to the method(s) of choice. Drug therapies used in treating PTSD include tricyclic antidepressants (TCAs), agents with anticonvulsant and mood-stabilizing properties (e.g. carbamazepine), benzodiazepines (BDZs), monoamine oxidase inhibitors (MAOIs), and serotonin specific reuptake inhibitors (SSRIs). Drug therapies are based on the assumption that exposure to trauma causes neurochemical aberrations in mechanisms controlling arousal and other aspects of emotional processing, and that medications correct these aberrations. Changes in the opioid, noradrenergic, dopaminergic, seratonergic, and hypothalamic–pituitary–adrenal axis systems have all been implicated in PTSD (van der Kolk, 1987; Friedman, 1991; Sutherland and Davidson, 1994). It is beyond the scope of the present article to offer a more detailed discussion of the neural structures, circuits, and neurotransmitters implicated in the various biochemical models of PTSD; see Sutherland and Davidson (1994) for details.

Behavioural therapies (e.g. imaginal exposure) and cognitive-behavioural therapies (e.g. stress inoculation training: Veronen *et al*., 1978) for PTSD were developed from conditioning and cognitive theories. In an early formulation, Mowrer's (1960) two-factor model was used to account for combat-related PTSD (Keane *et al*., 1985). According to this formulation, exposure to trauma produces a conditioned fear or anxiety response to trauma-related stimuli. Escape and avoidance of trauma-related stimuli are negatively reinforced (i.e. reinforced because they provide short-term relief from distress), and thereby prevent habituation from occurring.

Contemporary cognitive theories of PTSD are consistent with neo-conditioning models (Rescorla, 1988), and emphasize expectations and appraisals about the meaning of aversive experiences. Such models include the emotional processing model (Foa and Kozak, 1986; Foa *et al*., 1989) and similar approaches (e.g. Chemtob *et al*., 1988; Litz and Keane, 1989; Litz, 1992). These models propose that PTSD symptoms arise from a fear structure stored in long-term memory. The structure consists of a network of information about stimuli, their meanings, and responses to those stimuli (e.g. autonomic arousal, escape, avoidance). The traumatic experience is thought to be so intense that it causes fear-conditioning to a wide range of stimuli (e.g. sights, sounds, odours, and bodily sensations associated with the trauma). Such stimuli can serve as reminders of the trauma (retrieval cues), thus activating the fear structure and thereby producing hyperarousal and intrusive recollections of the trauma. Avoidance and numbing symptoms are thought to arise from mechanisms for deactivating the structure (Foa *et al*., 1992).

According to contemporary cognitive models, PTSD can be reduced by exposing the person to corrective information, which modifies the fear structure. Behavioural and cognitive-behavioural treatments are seen as effective means of producing this change. An important ingredient in these treatments is exposure to fear-evoking but objectively harmless stimuli. Some behavioral interventions also include cognitive restructuring, in which the meaning of the trauma is examined. Training in anxiety management skills is also provided (Foa *et al*., 1989). Throughout this article we will use the term 'behaviour therapy' to include behavioral and cognitive-behavioural treatments. We will examine these treatments as a class of interventions rather than evaluating specific types of treatment. This is because there are insufficient trials to separately examine each form of behavioural and cognitive-behavioural therapy. Our approach is similar to other meta-analytic efforts to evaluate the efficacy of broad classes of interventions (e.g. Lipsey and Wilson, 1993).

Eye-movement desensitization and reprocessing (EMDR) is a recent and controversial treatment that entails imaginal exposure to traumatic images while systematic saccadic eye movements are produced. Saccades are typically induced by tracking a therapist's finger as it is moved rapidly from side to side (Shapiro, 1991). Coping statements also are introduced while the scene is being imagined. Treatment typically takes one to four sessions. Recently, Shapiro (1995) suggested that eye movements in EMDR can be replaced with other lateral, stereotypic, motor movements. Shapiro (1995) postulated that exposure to trauma produces neuronal changes and disruption of a physiological balance between excitatory and inhibitory systems in the brain, which prevents appropriate processing of traumatic memories. EMDR purportedly restores this balance and reverses the neural pathology, and in so doing, allows appropriate reprocessing and integration of the traumatic memories (Shapiro, 1991, 1995). The theoretical underpinnings of EMDR have been criticized by several writers

(e.g. Lilienfeld, 1996; McNally, 1996). It may be that EMDR is an effective treatment, but not for the reasons proposed by Shapiro (1995).

Another therapy used for PTSD includes relaxation training, which is aimed at reducing hyperarousal (e.g. Vaughan *et al*., 1994). Other treatments include hypnotherapy and psychodynamic psychotherapy, which are aimed at uncovering and resolving unconscious conflicts arising from the traumatic events (e.g. Brom *et al*., 1989).

Despite the many treatments used for PTSD, none have been established as treatments of choice, and there has been only one previous attempt to quantify the relative efficacy of these interventions. Otto *et al*. (1996) reported an effect-size analysis for 14 treatment-outcome studies representing 20 trials of drug therapy or psychological therapy. Studies were published or presented between 1991 and 1994. Only randomized controlled trials were included. On measures of PTSD symptoms, general anxiety, and depression, the drug therapies with the largest effect sizes were fluoxetine and amitriptyline. Behavioural therapies tended to have larger effect sizes than these drug therapies, and were associated with less attrition. Follow-up data were not examined, and no trials of EMDR were included.

These findings were based on a small number of trials, and so the results should be regarded with caution. Moreover, there are several major methodological concerns with Otto *et al*.'s (1996) study. They computed each effect size by subtracting the mean of the posttreatment treatment group from the mean of the posttreatment control group, and then dividing by the standard deviation of the control group. The problem with this approach is that it ignores pretreatment differences between treatment and control groups. The trials typically consisted of small numbers of participants (e.g. *N*s of 8 to 16). With such small samples it is likely that random assignment of participants to treatment versus control groups would often fail to equate groups on pretreatment severity. This means that some of the effect sizes may actually represent pretreatment differences rather than differences in the efficacy of treatment and control conditions. Moreover, Otto *et al*. compared treatments against different types of controls. Drug therapies were compared to pill placebo whereas psychological therapies were typically compared to waiting-list controls. Thus, the comparison of treatments was confounded by the use of different types of controls.

A further concern with the Otto *et al*. (1996) study is that they computed effect sizes across scales, thus combining data from self-report and observer-rated scales. Observer-rated scales typically yield larger effect sizes than self-report scales (e.g. Lambert *et al*., 1986; Taylor, 1995). If observer-rated scales were more likely to be used in studies of some treatments (e.g. drug therapies) than in others (e.g. behaviour therapy), then the comparison between treatments will be confounded by differences in assessment method.

Otto *et al*. (1996) did not include uncontrolled trials, and thereby excluded many studies from their analysis. For a given type of treatment (e.g. behaviour therapy), the effect sizes of these trials can be compared with those of controlled trials in order to determine the comparability of controlled and uncontrolled trials. If the mean effect sizes for controlled and uncontrolled trials do not differ, then uncontrolled trials can be included, thereby increasing statistical power (Hunter and Schmidt, 1990).

The purpose of the present study was to further investigate the comparative efficacy of PTSD treatments, using a broader range of treatments than those examined by Otto *et al*. (1996). We also intended to circumvent the methodological concerns inherent in the latter study. We used meta-analysis to empirically evaluate the relative efficacy of treatments for PTSD. Our aims were (1) to identify which classes of treatment are more effective than wait-list controls and placebo; (2) to determine whether some classes of treatment are more effective than others; and (3) to determine whether treatment gains are maintained at follow-up.

METHOD

Inclusion and Exclusion Criteria

English-language articles published, unpublished, or presented at conferences from 1984 to 1996 were located from Medline, the PILOTS Database, Psychological Abstracts, Current Contents, conference programs, recent journal issues, and secondary sources (e.g. narrative reviews, book chapters), and by contacting PTSD researchers. Articles were included if the following criteria were met: (1) all participants were diagnosed with PTSD according to DSM III, DSM III-R, or DSM-IV criteria, as assessed by structured or unstructured clinical interviews. (2) Five or more participants were included in each trial. (3) Sufficient information was provided to compute effect sizes (or necessary additional data was supplied by the authors). (4) Outcome was presented in terms of self-report or observer-rated measures for one or more of the following variables:

intrusions, avoidance, total PTSD severity, depression, and anxiety. These variables were selected because they are the ones most commonly used to assess outcome in treatments of PTSD. (5) The outcome measures had acceptable levels of reliability and validity, as reported in the outcome study or in previous reports.

A total of 41 studies were located, yielding 68 treatment-outcome trials. Three trials from three different studies were of various inpatient treatments that were sufficiently heterogeneous and/or poorly described so as to prevent interpretation of the data as a distinct treatment class. These were therefore excluded from the analysis, leaving 65 trials. In 61 of the these trials, most participants had chronic PTSD, and four trials included only people with acute PTSD. Chronic PTSD is defined as duration of symptoms of 3 months or longer (APA, 1994). The four trials based on acute PTSD included two behaviour therapy trials, an assessment-only trial, and a trial of relaxation training. The mean duration between the trauma and commencement of each outcome trial ranged from 3 to 12 weeks in the studies of acute PTSD, compared to approximately 6 years in studies of chronic PTSD. Compared to the effect sizes for chronic PTSD, the effect sizes for the four trials of acute PTSD were statistical outliers. The large effect sizes for acute PTSD may reflect spontaneous remission, which is more common in acute than chronic PTSD (Foa, 1994; Rothbaum *et al*., 1992). Thus, our meta-analysis consisted of 61 trials from 39 studies of chronic PTSD. These are listed in Table 1. All treatments were provided in individual format with the exception of one behaviour therapy trial, which used a combination of group and individual treatment (Frueh *et al*., 1996). An appendix listing the studies that were excluded, and reasons for exclusion, is available on request.

Of the 61 trials included in the meta-analysis, 36 were from studies in which two or more conditions (e.g. TCA versus placebo) were compared. Five studies used crossover designs, where participants completed one condition followed by a waiting period, and then completed another condition (Shestatzky *et al*., 1988; Reist *et al*., 1989; Braun *et al*., 1990; Pitman *et al*., 1996a; Rothbaum *et al*., 1996). To avoid the problem of confounding within- and between-subject variance, and to avoid possible problems of carry-over effects from one treatment to another, we included only the first active treatment trial (i.e. drug or psychological treatment) from each crossover study. To illustrate, Shestatzky *et al*. (1988) used a cross-over design with one condition consisting of phenelzine followed by pill placebo. Here, we included only the phenelzine condition.

Of the trials included in the meta-analysis, six were TCA treatments, which included desipramine (n (number of trials) = 2; mean dose (i.e. mean dose at the end of treatment) = 200 mg/day), imipramine ($n = 2$; mean dose = 242 mg/day), amitriptyline ($n = 1$; mean dose = 175 mg/day), and trazadone ($n = 1$; mean dose = 300 mg/day). Although carbamazepine is structurally similar to TCAs, we classified it separately because it appears to have different pharmacologic properties to conventional TCAs. In addition to its anti-seizure effects, it is thought to reduce problems of impulse control (Coccaro and Siever, 1995), which in turn raises the question of whether it plays an important role in the reduction of unwanted, intrusive, trauma-related thoughts (Lipper, 1990). One trial of carbamazepine that was suitable for inclusion was located (mean dose = 661 mg/day).

Seven MAOI treatments were included, consisting of phenelzine ($n = 6$; mean dose = 60 mg/day) and brofaromine ($n = 1$; mean dose = 150 mg/day). BDZ treatment consisted of a single trial of alprazolam (mean dose = 3.75 mg/day). Four SSRIs trials included fluoxetine ($n = 2$; mean dose = 60 mg/day), fluvoxamine ($n = 1$; mean dose = 150 mg/day), and sertraline ($n = 1$; mean dose = 105 mg/day). All patients in all drug trials were on medication when assessed at posttreatment.

Thirteen behavioural therapy trials were included. They generally entailed some type of exposure therapy ($n = 11$), with some of these using imaginal exposure ($n = 4$) and others using both imaginal and *in-vivo* exposure ($n = 7$). Some behavioural therapies also included stress-inoculation training (SIT: $n = 3$). As mentioned earlier, we examined behavioural therapies as a group (which included cognitive-behavioural treatments), rather than separately examining each 'type' of behavioural intervention. This was because there were insufficient trials to conduct a more fine-grained analysis. Thus, our meta-analysis was directed toward examining behaviour therapy as a class of interventions, which is similar to the way in which other meta-analyses have examined classes or groups of interventions (see, for example, Lipsey and Wilson's (1993) meta-analysis of very broad classes of psychological and educational interventions).

EMDR therapies were also examined as a class of therapies, consisting of 11 trials. Although EMDR has been modified since it was first described by Shapiro (1989), the initially proposed elements of

Table 1. Trials included in meta-analysis

Author(s)	Condition	*N* completers	% dropout	Trial duration (weeks)	Self-report measures	Observer-rated measures
Baker *et al*. (1995)	Brofaromine 150 mg	56	5	12	DTS, IES,	CAPS
	Pill placebo	58	2	12	DTS, IES	CAPS
Braun *et al*. (1990)	Alprazolam 3.75 mg	10	38	5	IES	HAM-A, HAM-D, PTSD Scale
Brom *et al*. (1989)*†	Exposure therapy	29	11	15	IES, STAI-T	—
	Hypnotherapy	29	11	14	IES, STAI-T	—
	Dynamic psychotherapy	31	11	19	IES, STAI-T	—
	Waiting-list control	23	11	16	IES, STAI-T	—
Burstein (1984)	Imipramine 260 mg	10	33	3	IES	—
Carlson *et al*. (in press)*†	EMDR	10	0	12	BDI, IES, MISS, STAI-T	CAPS
	Relaxation training	13	8	12	BDI, IES, MISS, STAI-T	CAPS
	Supportive psychotherapy	12	0	6	BDI, IES, MISS, STAI-T	CAPS
Cooper and Clum (1989)	Exposure therapy	8	27	10	BDI, STAI-T	—
	Supportive psychotherapy	8	27	10	BDI, STAI-T	—
Davidson (1987)	Phenelzine 52.5 mg	7	36	6	IES	HAM-A
Davidson *et al*. (1990)	Amitriptyline 175 mg	17	29	8	IES	HAM-A, HAM-D, SI-PTSD
	Pill placebo	16	29	8	IES	HAM-A, HAM-D, SI-PTSD
Devilly and Spence (1996)	EMDR	19	—	2	BDI, MISS, STAI-T	—
	Supportive psychotherapy	16	—	2	BDI, MISS, STAI-T	—
	No-saccade control	16	—	2	BDI, MISS, STAI-T	—
Foa *et al*. (1996)*†	SIT	19	—	9	BDI, STAI-T	PSS-I
	Exposure therapy	22	—	9	BDI, STAI-T	PSS-I
	SIT + exposure therapy	22	—	9	BDI, STAI-T	PSS-I
	Waiting-list control	15	—	9	BDI, STAI-T	PSS-I
Foa *et al*. (1991)*†	Exposure therapy	10	29	5	BDI, RAST, STAI-T	PSS-I
	SIT	14	18	5	BDI, RAST, STAI-T	PSS-I
	Supportive psychotherapy	11	21	5	BDI, RAST, STAI-T	PSS-I
	Waiting-list control	10	0	5	BDI, RAST, STAI-T	PSS-I
Forbes *et al*. (1994)*†	EMDR	8	—	4	BDI, IES	SI-PTSD
Frueh *et al*. (1996)	Trauma mgt therapy	15	27	17	—	CAPS, HAM-A
Hertzberg *et al*. (1996)	Trazadone 150 mg	6	0	16	DTS	CAPS
Hickling and Blanchard (1997)*	Exposure therapy	8	17	10	—	CAPS
Jensen (1994)†	EMDR	13	14	3	—	SI-PTSD
Kauffman *et al*. (1987)	Desipramine 200 mg	8	—	4	BDI	HAM-A, HAM-D
Keane *et al*. (1989)	Flooding	11	0	14	BDI, STAI-T	PTSD Symptom Checklist
	Waiting-list control	13	0	20	BDI, STAI-T	PTSD Symptom Checklist
Kosten *et al*. (1991)	Imipramine 225 mg	23	48	6	Covi Anx, IES, Raskin Dep	HAM-D
	Phenelzine 68 mg	19	48	6	Covi Anx, IES, Raskin Dep	HAM-D
	Pill placebo	18	48	6	Covi Anx, IES, Raskin Dep	HAM-D

Lazrove *et al*. (1996)*†	EMDR	8	12	1	BDI, IES	CAPS
Lerer *et al*. (1987)	Phenelzine 60 mg	6	12	6	—	HAM-A, HAM-D, PTSD Scale
	Phenelzine 60 mg	7	36	11	—	HAM-A, HAM-D, PTSD Scale
	Phenelzine 60 mg	9	64	16	—	HAM-A, HAM-D, PTSD Scale
Lipper (1990)	Carbamazepine 661 mg	10	9	5	BDI, IES, IPAT, PTSD Index	HAM-A, HAM-D, PTSD Sx cklst
Marcus *et al*. (1996)*†	EMDR	67	1	3	BDI, IES, M-PTSD	—
Marmar *et al*. (1996)	Fluvoxamine 150 mg	10	9	10	IES, SCL Anx & Dep, SRRS	—
Mollica *et al*. (1990)	Supportive psychotherapy	26	34	24	—	Hopkins Anx & Dep Scales
Montgomery and Ayllon (1994)	EMDR	6	—	2	BDI	—
Nagy *et al*. (1993)	Fluoxetine 80 mg	10	63	10	IES	CAPS, HAM-A, HAM-D
Pitman *et al*. (1996a)*†	Flooding	20	0	6	IES, MISS, PTSD Sev Scale	—
Pitman *et al*. (1996b)*†	EMDR	17	30	6	IES, MISS	CAPS
Reist *et al*. (1989)	Desipramine 200 mg	18	22	4	BDI, IES	HAM-A, HAM-D
Richards *et al*. (1994)	Exposure therapy	13	7	8	BDI, IES, PTSD Checklist	—
Rothbaum (in press)*†	EMDR	10	14	3	BDI, IES, RAST, STAI-T	PSS-I
	Waiting-list control	8	14	3	BDI, IES, RAST, STAI-T	PSS-I
Rothbaum *et al*. (1996)	Sertraline 105 mg	5	—	12	IES	CAPS
Scheck *et al*. (in press)*†	EMDR	26	30	2	BDI, IES, PENN, STAI-T	—
Shestatzky *et al*. (1988)	Phenelzine 60 mg	10	54	4	IES	PTSD Scale, HAM-A, HAM-D
Vaughan and Tarrier (1992)	Image habituation	10	—	10	BDI, IES	—
Wilson *et al*. (1995, 1997)*†	EMDR	32	—	3	IES, STAI-T	—
van der Kolk *et al*. (1994)	Fluoxetine 40 mg	21	36	5	—	CAPS
	Pill placebo	27	13	5	—	CAPS

Doses for drug trials refer to the mean dose attained by the end of treatment. '—' refers to data not reported (for % dropouts) or not used (for measures).
*Psychotherapy trial reporting treatment fidelity check.
†Psychotherapy trial reporting level of therapist training.
BDI, Beck Depression Inventory; CAPS, Clinician Administered PTSD Scale; Covi Anx, Covi Anxiety Inventory; DTS, Davidson Self-Rating Trauma Scale; EMDR, Eye Movement Desensitization and Reprocessing; HAM-A, Hamilton Anxiety Scale; HAM-D, Hamilton Depression Scale; IES, Impact of Event Scale; IPAT, IPAT Anxiety Scale; MISS, Mississippi Scale for PTSD; M-PTSD, Modified PTSD Scale; PE, Prolonged Exposure; PENN, Penn Inventory for PTSD; PSS-I, PTSD Symptom Scale—Interview Form; Raskin, Raskin Depression Scale; RAST, Rape Aftermath Symptom Test; SCL Anx & Dep, Anxiety and depression scales from the SCL-90-R; SIT, Stress Innoculation Training; SI-PTSD, Structured Interview for PTSD; SRRS, Stress Response Rating Scale; STAI-T, Trait version of State-Trait Anxiety Inventory.

treatment have remained unchanged: i.e. imaginal exposure with concomitant lateralized movements, along with coping statements (Shapiro, 1995; personal communication, 1 November 1996). Relaxation therapy included biofeedback-guided relaxation ($n = 1$). Other treatment trials included hypnotherapy ($n = 1$) and psychodynamic therapy ($n = 1$).

Control groups included pill placebo ($n = 4$), wait-list control ($n = 5$), a non-saccade control for EMDR studies ($n = 1$), and supportive psychotherapy ($n = 5$). The non-saccade condition is an EMDR control in that it includes no eye saccades nor any other oscillating stimulation. The supportive psychotherapy condition included three trials where participants received standard supportive therapy at a VA medical centre, one trial described as a supportive control in which subjects received general therapist support and some teaching in general problem-solving, and one trial in which participants met weekly with a social support service team. Four of the five supportive psychotherapy trials were described by their authors as control conditions. These trials can therefore be regarded as attention placebos.

Statistical Procedures

Effect sizes were calculated according to Cohen's (1988) *d* statistic. For each trial the magnitude of change from pre- to posttreatment was defined as $(M_{pre} - M_{post})/SD_{pooled}$, where $SD_{pooled} = \sqrt{[(SD^2_{pre} + SD^2_{post})/2]}$. The magnitude of change from pre-treatment to follow-up was defined by replacing M_{post} with $M_{follow\text{-}up}$, and SD_{post} with $SD_{follow\text{-}up}$. For the outcome measures used in the present study, positive effect sizes represent improvements in PTSD and other symptoms (i.e. reductions in problem severity), whereas negative effect sizes indicate a worsening of symptoms. Effect sizes were based on completer analyses rather than end-point or intent-to-treat analyses. In other words, effect sizes were based on pre- and posttreatment data for participants completing each trial. This was because most trials only reported data for treatment completers.

There are a number of different formulae for computing effect sizes (for examples, see Smith *et al*., 1980; Wolf, 1986) and none has been established as a gold standard. We selected the above-mentioned effect size because the same or very similar effect size formulae are commonly used (e.g. Christensen *et al*., 1987; Taylor, 1996; Abramowitz, 1997a,b) and because it provides an effect size for each trial, rather than an effect size defined as the posttreatment difference between a treatment and control trial. Thus, we were able to include uncontrolled studies in the meta-analysis, thereby increasing the number of trials and statistical power to detect differences between treatments. (Note also that we were able to determine whether the effect sizes of controlled studies differed from those of uncontrolled studies). The different formulae for effect sizes can differ in the magnitudes of obtained effects, and the effect size used in the present study tends to be larger than effect sizes computed according to other methods (Abramowitz, 1997a). Accordingly, one should not interpret in isolation an effect size for a given treatment. The meaning of the effect size is determined by comparing it to the effect sizes for other treatments.

Regardless of the method of computing effect sizes, a further concern that we will examine is the possibility of inflated effect sizes due to the 'file drawer' problem (Rosenthal, 1979). We use the procedures outlined by Hunter and Schmidt (1990, pp. 512–513) to determine whether this was a problem for the treatments examined in the present study. The file drawer problem is a publication bias in which studies obtaining significant findings and large effect sizes are published, whereas findings obtaining null results are unpublished. To determine the likelihood of this bias, the fail-safe *N* is computed (Orwin, 1983), which is the number of unpublished trials obtaining zero effect sizes that are required to reduce an obtained mean effect size to a trivial level.

If a large number of unpublished trials are required, then it is unlikely that the obtained effect is biased by the file drawer problem. The number of unpublished or unobtained trials obtaining zero effect sizes is defined by $k[(d_k/d_c) - 1]$, where k = the number of obtained trials, d_k = mean obtained effect size, d_c = the trivial value to which the obtained effect would be reduced. Note that d_c cannot equal 0, because d_k/d_c would be undefined. Orwin (1983) suggested that a small effect size (i.e. 0.200) would qualify as a trivial value. However, such an effect size is considered non-trivial by some meta-analysts (e.g. Lipsey and Wilson, 1993). Accordingly, we defined a trivial effect size as 0.050.

Assessment

Previous meta-analyses have shown that interviewer-rated scales consistently yield larger effect-sizes than self-report scales (e.g. Lambert

et al., 1986; Taylor, 1995). This may reflect the greater sensitivity of interviewer-rated scales, or it may be an artifact reflecting interviewer bias (i.e. the interviewer typically knows whether or not the assessment is a pre- or posttreatment evaluation, and therefore may be biased by expecting comparatively lower symptom scores at the posttreatment assessment). Systematic bias in computing effect sizes can occur if some types of treatments are evaluated with interviewer-rated measures (e.g. drug therapies), while others are evaluated with self-report measures (e.g. psychological therapies). Thus, we calculated treatment effect sizes separately for interviewer-rated and self-rated outcome variables (see Table 2).

The outcome measures used in each study are presented in Table 1. As the table shows, scores for self-reported intrusions, avoidance and total PTSD symptoms were obtained from the Impact of Event Scale (Horowitz *et al*., 1979), the Mississippi Scale for Combat-related PTSD (Keane *et al*., 1988), and various DSM-tailored measures such as the PTSD Symptom Checklist (Richards *et al*., 1994), PTSD Index (Lipper, 1990), and Modified PTSD Scale (Saunders *et al*., 1990). Self-reported depression was typically assessed by the Beck Depression Inventory (Beck *et al*., 1979), and self-reported anxiety was typically assessed by the State-Trait Anxiety Inventory (Spielberger *et al*., 1970). Scores from other measures were also included in each symptom domain if the alternative measure adequately represented the domain. For example, single flashback or nightmare scores were not included in the intrusion score as they represent only a portion of the domain of intrusion symptoms.

The table also shows that scores for observer-rated intrusions, avoidance, and total PTSD symptoms were typically obtained from the Clinician Administered PTSD Scale (Blake *et al*., 1995) or from the Structured Interview for PTSD (Davidson *et al*., 1989). Observer-rated depression was obtained from the Hamilton Rating Scale for Depression (Hamilton, 1960), and observer-rated anxiety was typically assessed by from the Hamilton Rating Scale for Anxiety (Hamilton, 1959).

RESULTS

Preliminary Analyses

A series of preliminary analyses were conducted to determine whether it was necessary to match treatment conditions on important variables (e.g. treatment duration) before comparing effect sizes for treatment outcome. In the aggregate, treatment and control conditions (as listed in Table 2) did not differ in the mean duration of their trials, $F(13,\ 47) = 1.86,\ p > 0.05$ ($M = 8.2$ weeks, $SD = 4.7$ weeks). Note, however, that EMDR trials tended to be shorter than those of behaviour therapy ($M = 3.7$ versus 10.1 weeks, respectively; $t(22) = 4.66,\ p < 0.001$), and consisted of fewer treatment sessions ($M = 4.6$ versus 14.8, respectively; $t(22) = 5.51,\ p < 0.001$). We will return to consider these differences later in this article.

The effect sizes of studies that included a control group were compared to effect sizes of uncontrolled studies. There was no significant difference for either self-report, $F(1,\ 40) = 2.77$, $p > 0.1$, or observer-rated measures of total PTSD symptoms, $F(1,\ 32) = 0.62,\ p > 0.1$. Among the psychological therapies, 75% reported the level of therapist training. Studies were coded as having adequate therapist training if they specifically reported adequate years of therapist experience (e.g. over 5 years) or formal training with a senior colleague experienced in the treatment modality. In the aggregate (i.e. across treatment conditions), effect sizes did not significantly differ on either self-report or observer-rated measures for trials that reported therapist training versus those that did not report on this variable, ps > 0.1.

Three types of trauma were classified: combat-related, rape or assault-related, and a category reflecting a mix of various trauma or another trauma. Across the 59 trials that reported trauma type, 51% involved combat-related trauma only, 19% rape or assault-related trauma only, and 30% a mix of trauma or other trauma. Within each treatment condition (for conditions with three or more trials), mean effect sizes did not significantly differ across trauma types, ps > 0.1.

We intended to examine the relationship between degree of psychiatric comorbidity and effect size. However, there were insufficient data for such analysis. Of the 61 trials, only 21% reported a quantifiable level of comorbid anxiety disorders apart from PTSD. A total of 33% of trials reported comorbidity data on mood disorders, and 16% of trials reported comorbidity data for substance use disorders. Thus, we did not examine the relationship between effect size and comorbidity.

Follow-up data were not reported for drug therapies. For the control conditions, follow-up data were reported for only one supportive therapy trial. For several psychological therapies, follow-up

data were available only on few outcome measures. Only behaviour therapy and EMDR provided sufficient follow-up data. Analysis of follow-up data was therefore restricted to these two treatment conditions. Across these conditions, the duration between posttreatment and follow-up did not differ significantly, $F(1, 17) = 1.68$, $p > 0.1$ (grand $M = 15$ weeks; behaviour therapy: $M = 18$ weeks, SD = 12; EMDR: $M = 12$ weeks, SD = 6).

Comparison of Treatments

Data Analysis

Rather than conducting multiple comparisons (e.g. *t*-tests) between the 14 treatment conditions, the simplest and most efficient method of evaluating the comparative efficacy of treatments is to compute confidence intervals. Given the small number of trials in each condition for each outcome measure, we chose to use 90th percentile confidence intervals. Confidence intervals have methodological advantages over the conventional use of *p*-values (see Cohen, 1990). Accordingly, 90% confidence intervals were calculated for all conditions across all measures. Conditions with non-overlapping confidence intervals differ significantly at $p < 0.10$. Some treatment conditions consisted of only a single trial, in which case it is not possible to compute confidence intervals. However, the effect sizes of these trials could be compared to the confidence intervals of other trials, to determine whether the obtained effect size fell within the confidence interval. If the effect size falls within the interval, then the two conditions do not significantly differ.

For each dependent measure, comparisons were as follows: (1) general comparisons between the overall drug therapy, psychological therapy, and control groups; (2) comparison of treatments to controls and to one another (e.g. SSRIs versus controls; SSRIs versus TCAs); (3) comparisons across treatment types (e.g. SSRIs versus EMDR); (4) where relevant, comparisons between control conditions (e.g. WLCs and pill placebo versus supportive psychotherapy). Following the recommendations of Hunter and Schmidt (1990), Wolf (1986), and others, we made these comparisons by computing weighted means. That is, means were computed by weighting the effect size of each trial by the number of participants completing that trial. This procedure gives greater weighting to the effect sizes of larger trials, which are likely to be more reliable estimates of treatment efficacy than the effect sizes of small trials (Hunter and Schmidt, 1990). Confidence intervals were computed around weighted means.

Attrition

Table 2 shows the proportions of dropouts for each treatment condition. With regard to the three classes of treatment conditions (i.e. drugs treatments, psychological therapies, and control conditions), the table shows that attrition was significantly greater (i.e. confidence intervals did not overlap) for drug therapies ($M = 31.9\%$) compared to psychological therapies (14.0%) and controls (16.6%). Within the drug therapy conditions, the dropout rates tended not to differ from one another. No differences in dropout rates were observed within the psychological therapy conditions or the control conditions, or between the psychological therapies and controls.

Effect Sizes at Posttreatment: PTSD Symptoms

Intrusions (Self-report)

Table 2 shows that for the three classes of treatment conditions (drugs treatments, psychological therapies, and control conditions), drug therapies and psychological therapies were generally equally effective (i.e. their confidence intervals overlapped), and only psychological therapies were significantly superior to controls. Among the drug therapies, all treatments were superior to WLCs. Both SSRIs and carbamazepine were superior to supportive therapy controls. Only the effect size for the single carbamazepine was significantly greater than that of pill placebo. SSRIs and carbamazepine had comparable effect sizes, and both were superior to all other drug therapies.

All psychological therapies were significantly superior to WLCs, and none were significantly better than pill placebo. Behaviour therapy was as effective as other psychological therapies, and EMDR was superior to relaxation and dynamic therapy. Across drug therapies and psychological therapies, SSRIs were superior to all psychological therapies except EMDR and behaviour therapy. Carbamazepine was similarly superior to all psychological therapies except behaviour therapy.

Intrusions (Observer-rated)

Only some drug therapies (one TCA trial, SSRIs, and one BDZ trial), two of the psychological therapies (behaviour therapy and EMDR), and two control conditions (WLCs and one supportive psychotherapy) reported results for observer-rated

Table 2. Dropout proportions and pre–post effect sizes for measures of PTSD symptoms

Condition	No. trials	% dropout		Intrusions				Avoidance				Total severity of PTSD symptoms			
				Self-report		Observer-rated		Self-report		Observer-rated		Self-report		Observer-rated	
		M	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI
TCA	6	26.4	14.4–38.4	0.64	0.30–0.98	0.46	—	0.35	0.22–0.48	0.55	—	0.54	0.34–0.74	0.86	0.75–0.97
Carbmz	1	9.0	—	1.53	—	—	—	0.52	—	—	—	0.93	—	1.45	—
MAOI	7	36.4	24.7–48.2	0.64	0.27–1.01	—	—	0.40	−0.21–1.01	—	—	0.61	0.38–0.84	0.92	0.73–1.11
SSRI	4	36.0	6.5–65.5	1.71	1.08–2.34	1.28	0.90–1.66	0.92	0.73–1.11	1.37	1.05–1.69	1.38	1.02–1.74	1.43	1.19–1.67
BDZ	1	38.0	—	0.51	—	0.66	—	0.16	—	0.32	—	0.49	—	0.54	—
Drug Txs (overall)	19	31.9	25.4–38.4	0.86	0.63–1.09	1.01	0.71–1.31	0.45	0.31–0.59	1.00	0.64–1.36	0.69	0.55–0.83	1.05	0.91–1.19
Behav Tx	13	15.1	9.8–20.4	1.12	0.49–1.75	1.76	−0.05–3.57	1.12	0.61–1.63	1.45	−0.10–3.00	1.27	0.80–1.74	1.89	1.66–2.12
EMDR	11	14.4	7.8–21.0	1.12	0.72–1.52	1.39	0.99–1.79	1.27	0.74–1.80	2.01	1.25–2.77	1.24	0.99–1.49	0.69	−0.06–1.44
Relaxat'n	1	8.0	—	0.54	—	—	—	0.46	—	—	—	0.45	—	—	—
Hypnosis	1	11.0	—	1.06	—	—	—	0.80	—	—	—	0.94	—	—	—
Dynamic	1	11.0	—	0.70	—	—	—	0.64	—	—	—	0.90	—	—	—
Psych Tx (overall)	27	14.0	10.8–17.2	1.02	0.80–1.24	1.57	1.12–2.02	1.03	0.77–1.29	1.74	1.23–2.25	1.17	0.99–1.35	1.51	1.17–1.85
Pill Plac	4	23.0	6.6–39.4	0.48	−0.17–1.13	—	—	0.07	0.05–0.09	—	—	0.51	0.29–0.73	0.77	0.63–0.91
WLC	5	6.2	0.2–12.2	0.32	0.28–0.36	0.74	0.72–0.76	0.21	0.14–0.28	0.22	−0.65–1.09	0.44	0.28–0.60	0.75	0.67–0.83
Sup Psych	5	20.5	8.5–32.5	0.95	—	0.53	—	0.77	—	0.09	—	0.34	0.01–0.67	0.92	—
No Sacc	1	—	—	—	—	—	—	—	—	—	—	0.22	—	—	—
Controls (overall)	15	16.6	10.5–22.7	0.49	0.29–0.69	0.66	0.54–0.78	0.23	0.06–0.46	0.17	−0.18–0.52	0.43	0.33–0.53	0.77	0.71–0.83

Effect size = $(M_{pre} - M_{post})/SD_{pooled}$, where $SD_{pooled} = \sqrt{[(SD^2_{pre} + SD^2_{post})/2]}$. All means are weighted by sample size. See text for details. 90%CI = 90th percentile confidence interval arounded weighted mean. Note that '—' refers to data missing or not reported. For the 90%CIs '—' appears when there was only one effect size. Within each row, total number of trials may differ across outcome domains (intrusions, avoidance, and global severity) because some trials did not assess all domains.

BDZ, benzodiazepines; Behav Tx, behaviour therapy; Carbmz, carbamazepine; Dynamic, psychodynamic psychotherapy; EMDR, eye movement desensitization and reprocessing; MAOI, monoamine oxidase inhibitors; No Sacc, no saccade control (control for EMDR); Pill Plac, pill placebo; Relaxat'n, relaxation training; SSRI, selective serotonin reuptake inhibitors; Sup Psych, supportive psychotherapy; TCA, tricyclic antidepressants; WLC, waiting-list control.

measures. Among these trials, the drug therapies and the psychological therapies were generally equally effective, although there was a trend for psychological therapies to have larger effect sizes (Table 2). Drug and psychological therapies were more effective than controls. Within the drug therapies, only SSRIs were more effective than all controls, and SSRIs were also more effective than all other drug therapies. Within the psychological therapies, EMDR and behaviour therapy demonstrated comparable effect sizes, but only EMDR was significantly more effective than all controls. EMDR was more effective than the most effective drug therapy, the SSRIs.

Avoidance (Self-report)

Table 2 shows that psychological therapies were more effective than both drug therapies and control conditions. All drug therapies were more effective than pill placebo and WLCs, and none were more effective than the single supportive therapy control. SSRIs were significantly more effective than all other drug therapies. All psychological therapies were more effective than pill placebo and WLCs, but again, not more effective than the single supportive psychotherapy trial. Among the psychological therapies, behaviour therapy and EMDR were equally effective, but only EMDR was superior to relaxation and dynamic therapy. SSRIs, EMDR, behaviour therapy and hypnotherapy did not differ in effect sizes.

Avoidance (Observer-rated)

Only some of the drug therapies (one TCA trial, SSRIs, and one BDZ trial), two of the psychological therapies (behaviour therapy and EMDR), and two control conditions (WLCs and one supportive psychotherapy) reported on avoidance. Drug therapies and psychological therapies were more effective than controls, and were equally effective to one another (Table 2). However, there was a trend for psychological therapies to have a larger effect size. Among the drug therapies, all were significantly more effective than supportive psychotherapy, and there was a trend for SSRIs to also be more effective than WLCs. Among the psychological therapies, EMDR and behaviour therapy were equally effective, but only EMDR was more effective than the control conditions. SSRIs, behaviour therapy, and EMDR were all equally effective.

Total PTSD Symptoms (Self-report)

As seen in Table 2, psychological therapies were more effective than drug therapies, and both were more effective than controls. Among the drug therapies, only the SSRIs and carbamazepine were more effective than all control conditions. The SSRIs had a significantly greater mean effect size than all other drug therapies. Among the psychological therapies, all but relaxation were significantly more effective than all control groups. EMDR and behaviour therapy were equally effective, but only EMDR was significantly superior to all other psychological therapies. Behaviour therapy and EMDR were equally effective as the SSRIs.

Total PTSD Symptoms (Observer-rated)

As seen in Table 2, outcome data for total PTSD symptoms were reported by all drug therapies and all controls except the non-saccade control. However, these data were reported by only two psychological therapies (behaviour therapy and EMDR). Psychological therapies and drug therapies were equally effective, although there was a trend for psychological therapies to have larger effect sizes. Drug and psychological therapies were more effective than controls. Among the drug therapies, only the SSRIs and carbamazepine were more effective than all controls, and both were also more effective than all other drug therapies. Among the psychological therapies, only behaviour therapy was more effective than all controls, and behaviour therapy was also more effective than EMDR. The best psychological therapy, behaviour therapy, was significantly more effective than carbamazepine, and there was a trend for behaviour therapy to be more effective than SSRIs as well.

Effect Sizes at Posttreatment: Anxiety and Depression

Anxiety (Self-report)

Psychological therapies were more effective than drug therapies, and both were more effective than controls (Table 3). All psychological therapies were more effective than all controls. All drug therapies except TCAs were superior to all controls. The SSRI trial tended to be superior to all other drug therapies. Within the psychological therapies, the largest effect sizes were observed in the EMDR and behaviour therapy conditions. Of these conditions, only behaviour therapy was more effective than relaxation therapy. Across drug therapies and psychotherapies, SSRIs and behaviour therapy were equal in efficacy. The SSRI trial was comparable to behaviour therapy, but more effective than EMDR and other psychological therapies. EMDR

Table 3. Pre–post effect sizes for measures of anxiety and depression

Condition	Anxiety				Depression			
	Self-report		Observer-rated		Self-report		Observer-rated	
	M	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI
TCA	0.44	−0.08–0.96	0.54	0.13–0.95	0.44	0.09–0.79	0.85	0.53–1.17
Carbmz	0.47	—	1.73	—	0.48	—	1.25	—
MAOI	0.65	—	0.92	0.44–1.40	0.98	—	0.43	0.28–0.58
SSRI	1.24	—	1.20	—	1.41	—	1.38	—
BDZ	—	—	0.72	—	—	—	0.11	—
Drug Txs (overall)	0.61	0.39–0.83	0.64	0.61–1.09	0.65	0.39–0.91	0.72	0.55–0.89
Behav Tx	1.12	0.84–1.40	1.47	—	0.97	0.80–1.14	—	—
EMDR	0.95	0.69–1.21	—	—	1.05	0.81–1.29	—	—
Relaxat'n	0.83	—	—	—	0.67	—	—	—
Hypnosis	0.95	—	—	—	—	—	—	—
Dynamic	1.07	—	—	—	—	—	—	—
Psych Tx (overall)	1.04	0.89–1.19	1.47	—	1.00	0.87–1.13	—	—
Pill Plac	0.03	—	0.38	—	0.24	—	0.36	0.19–0.53
WLC	0.25	0.14–0.36	—	—	0.25	0.12–0.42	—	—
Sup Psych	0.25	0.04–0.46	—	—	0.22	0.07–0.37	—	—
No Sacc	0.06	—	—	—	0.14	—	—	—
Controls (overall)	0.17	0.06–0.28	0.38	—	0.23	0.16–0.30	0.36	0.19–0.53

See footnote to Table 2 for a definition of statistics and acronyms.

yielded a larger effect size than the non-saccade control.

Anxiety (Observer-rated)

As seen in Table 3, among the psychological therapies, only one trial of behaviour therapy reported on this measure, and only one pill placebo trial was available among controls. The behaviour therapy trial demonstrated a significantly greater effectiveness than the drug therapies, and both were more effective than pill placebo. Among the drug therapies, the carbamazepine trial and SSRI trial were more effective than all other drug therapies.

Depression (Self-report)

Table 3 shows that the drug therapies and psychological therapies were generally comparable in efficacy, although there was a trend for psychological therapies to have larger effect sizes. Both were more effective than the control conditions. All drug therapies except TCAs were superior to all controls. The one SSRI trial tended to be more effective than all other drug therapies. All psychological therapies were more effective than all control conditions. Behaviour therapy and EMDR were equally effective, and both were more effective than the relaxation condition. The single SSRI trial was significantly more effective than the best psychological therapies, EMDR and behaviour therapy.

Depression (Observer-rated)

No psychological therapies reported observer-rated depression, and only two pill placebo trials reported outcomes among the control conditions. All drug therapies except the BDZ trial were more effective than pill placebo. Among the drug therapies, the SSRIs were more effective than carbamazepine, and both SSRIs and carbamazepine had larger effect sizes than MAOIs and TCAs.

Posttreatment: Summary of Results

Psychological therapies tended to be more effective than drug therapies, and both tended to be more effective than controls. Of the drug therapies, the SSRIs and carbamazepine were the most effective, although the carbamazepine condition was based on only a single trial. SSRIs tended to be more effective in treating intrusions than avoidance symptoms according to self-report but not observer-rated measures. Other drug therapies generally demonstrated small effect sizes in relation

to control conditions, and affected only a few of the symptom domains. Of the psychological therapies, behaviour therapy and EMDR were the most effective, with the two being generally equally efficacious, although behaviour therapy was significantly more effective than all treatments, including EMDR, SSRIs, and carbamazepine, on observer-rated total PTSD symptoms. No differences in comparative treatment efficacy were discernible between behaviour therapy and EMDR across the specific symptom domains of PTSD.

The less effective psychological therapies demonstrated moderate yet consistent effect sizes across symptom domains in relation to control conditions, with relaxation being the least effective, followed by dynamic therapy and lastly, hypnotherapy. However, these conditions were all based upon single trials, and therefore should be interpreted with caution. Supportive psychotherapies tended to demonstrate efficacy comparable to the less effective psychological therapies and drug therapies.

The best psychological therapies, behaviour therapy and EMDR, tended to be as effective as the best drug therapies, SSRIs and carbamazepine, across PTSD outcomes domains. However, as noted above, behaviour therapy was more effective than all treatments on observer-rated total PTSD symptoms. SSRIs may have been more effective than behaviour therapy and EMDR in treating depression, but this was based upon a single SSRI trial, and results were only available for self-reported depression.

Effect Sizes at Follow-up

No drug therapy, pill placebo, or wait-list control results were available for follow-up, and only single trials of other psychological therapies and the supportive psychotherapy trial provided follow-up data, and only across some symptom measures. Therefore the only conditions which provided adequate trials for calculating follow-up effect sizes across most symptom domains were behaviour therapy and EMDR. Table 4 shows the follow-up effect sizes for PTSD symptoms, and Table 5 shows the results for measures of anxiety and depression. As noted in the section titled 'Preliminary analyses', the mean duration between post-treatment and follow-up did not differ significantly across conditions (mean follow-up duration = 15 weeks).

Across all self-report and observer-rated measures of PTSD symptoms, depression and anxiety, both behaviour therapy and EMDR demonstrated a maintenance of treatment effects at follow-up, with no differences between the two conditions at follow-up on any measures. Differences in effect size from posttreatment to follow-up were nonsignificant for all measures across both conditions, except that EMDR demonstrated a significant increase in effect size for observer-rated total PTSD symptoms at follow-up, making it equal to behaviour therapy, whereas EMDR was less effective than behaviour therapy for this measure at posttreatment.

Table 4. Effect sizes at follow-up (i.e. symptom reductions from pretreatment to 15 week follow-up) for PTSD symptoms

Condition	No. trials	Intrusions				Avoidance				Total severity of PTSD symptoms			
		Self-report		Observer-rated		Self-report		Observer-rated		Self-report		Observer-rated	
		M	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI	*M*	90%CI
Behav Tx	5	1.56	0.81–2.29	1.47	0.60–2.34	1.44	0.47–2.41	1.32	0.71–1.93	1.63	1.10–2.16	1.93	1.67–2.19
EMDR	6	1.75	1.46–2.04	2.07	1.77–2.37	1.89	1.08–2.70	2.34	1.76–2.92	1.33	0.89–1.77	2.27	1.78–2.76

See footnote to Table 2 for a definition of statistics and acronyms.

Table 5. Effect sizes at follow-up for measures of anxiety and depression

Condition	No. trials	Anxiety Self-report		Depression Self-report	
		M	90%CI	*M*	90%CI
Behav Tx	9	0.99	0.66–1.32	0.93	0.76–1.10
EMDR	5	0.90	0.64–1.16	0.91	0.46–1.36

See footnote to Table 2 for a definition of statistics and acronyms.

Fail-Safe N

Recall that fail-safe *N* is defined as the number of unpublished null trials (i.e. those obtaining zero effect sizes) required to reduce an obtained mean effect size to a trivial magnitude (0.050). An average of self-reported and observer-rated total PTSD symptom effect sizes were used for d_k (mean obtained effect size) in calculating fail-safe *N* calculations. For the posttreatment data, the necessary number of unpublished null trials in each active treatment condition were as follows: TCA (41), carbamazapine (23), MAOI (66), SSRI (95), BDZ (10), behaviour therapy (220), EMDR (139), relaxation therapy (8), hypnotherapy (18), and dynamic therapy (17). These results suggest that for most conditions, there would need to be a large number of unpublished null trials to reduce obtained mean effect sizes to trivial levels. It seems unlikely, especially for SSRIs, EMDR, and behaviour therapy, that there would be so many unpublished trials finding zero effects for these treatments, and so we conclude that these findings were unlikely to have been biased by the 'file-drawer' problem. However, significantly fewer unpublished null trials were indicated to suggest bias in results for the carbamazepine, BDZ, relaxation, hypnotherapy, and dynamic therapy conditions. Therefore these conditions should be viewed cautiously as they may be subject to the file-drawer bias.

DISCUSSION

The purpose of the present study was to empirically evaluate the relative efficacy of various treatments for PTSD. We found that behaviour therapy and EMDR were the most effective psychological therapies for PTSD. Effect sizes for these therapies were large relative to control conditions, indicating strong treatment effects, and dropout rates were low, indicating good treatment acceptance. The SSRIs and carbamazepine were the most effective drug therapies, with similarly large effect sizes, but dropout was fairly high (Table 2). Even SSRIs, characteristically more acceptable to patients due to a more tolerable side-effect profile, were tolerated poorly by the PTSD patients. An average of 36% of patients treated with SSRIs discontinued treatment prematurely. It may be that PTSD patients, characteristically hyperaroused, may be particularly sensitive to side-effects occasionally associated with SSRIs (e.g. agitation), and may discontinue secondary to these effects.

Although dropout rates were high for drug therapies, the SSRIs demonstrated the greatest treatment efficacy of all drug therapies (other than carbamazepine, for which there was only one trial), with large effect sizes apparent across all symptom domains. Effects were perhaps stronger for intrusive symptoms and depressive symptoms than for avoidance symptoms, but all effect sizes were large relative to control conditions. However, posttreatment assessments for drug therapies occurred prior to medication discontinuation, so it is not clear whether treatment effects maintain when medications are withdrawn.

These results suggest that although SSRIs may not be the treatment of choice for PTSD, given the higher dropout rate, they may still be an acceptable and useful treatment if the patient can be retained in treatment, and may be particularly useful for patients whose intrusive and depressive symptoms predominate their experience. Unfortunately, follow-up data were unavailable for these trials, so it is unclear as to whether effects were maintained over time, or even immediately after medication discontinuation. Further research is warranted to investigate the maintenance of SSRI effects in PTSD patients. Carbamazepine also appeared to be an effective treatment for PTSD, but results were based upon a single trial and should therefore be interpreted with caution.

Only one BDZ trial was available for this meta-analysis. This study used alprazolam at a moderately high dose (1.5 to 6 mg/day) and generally failed to demonstrate treatment efficacy (Braun *et al*., 1990). Effect sizes were small if not trivial relative to control conditions. Although not included in our meta-analysis due to failure to meet our 100% PTSD diagnosis rule, another study also supports the inefficacy of BDZs for PTSD. Gelpin *et al*. (1996) reported that clonazepam and alprazolam failed to benefit acute trauma survivors' PTSD and anxiety symptoms over controls. In another study not included in the meta-analysis due to lack of a standard posttreatment assessment time across patients, alprazolam did not alter auditory startle response in PTSD patients (Bloch *et al*., 1996). This is enlightening given the high frequency of BDZ prescriptions by general practitioners for anxiety disorders. If medications are to be prescribed for PTSD treatment, it appears as though SSRIs are a more effective treatment than BDZs. The more narrow side-effect profile, non-addictiveness, and greater overdose threshold of SSRIs over BDZs also support this point (Canadian Pharmaceutical Association, 1996).

As noted, behaviour therapy and EMDR were the most effective psychological therapies, and both were as effective as SSRIs. Effect sizes were large across all PTSD symptom domains for these treatments in relation to controls, and treatments were generally statistically comparable in efficacy, with some minor exceptions. For example, behaviour therapy was more effective than EMDR and SSRIs on observer-rated total PTSD symptoms at posttreatment. However, by follow-up, the differences between behaviour therapy and EMDR were nonsignificant (SSRIs did not report follow-up data). Because attrition from posttreatment to follow-up was comparable across EMDR and behaviour therapy conditions, the 'catching up' of EMDR to behaviour therapy cannot be attributed to differential attrition of poor responders. No differences in treatment efficacy between behaviour therapy and EMDR were noted across the specific PTSD symptom domains. Both treatments maintained effects at follow-up, and were equally effective on all measures at follow-up.

The efficacy of EMDR in PTSD treatment is a controversial finding in light of recent discussions in the literature critically questioning the validity of EMDR as a treatment for anxiety disorders (Herbert and Mueser, 1992; Lohr *et al*., 1995; Lilienfeld, 1996). The results of the present study suggest that EMDR is effective for PTSD, and that it is more efficient than other treatments. Despite its apparent efficacy, what works in EMDR and the mechanism for how it works remains unclear. That is, we know little about the active ingredients in EMDR and the mechanisms by which these ingredients result in decreased PTSD symptoms. EMDR theory suggests that eye movements or other oscillatory movements during trauma imagining somehow result in brain alterations which then allow more appropriate processing of the trauma, thereby reducing PTSD symptoms. This still does not inform us of what changes occur in what part of the brain, how oscillatory movements are involved in those changes, how that leads to 'reprocessing' of the trauma, and how such reprocessing results in decreased PTSD symptoms. Some might argue that EMDR works through exposure and desensitization, similar to behaviour therapy. However, this is unlikely to be the case given that EMDR provides significantly less trauma exposure than behaviour therapy and is demonstrating comparable effects, which suggests that another treatment component specific to EMDR is active. Expectancy may play a role, but this has not been thoroughly examined, and is inconsistent with our finding that the effect sizes of EMDR tended to be larger than those of control conditions, such as pill placebo and supportive psychotherapy.

Regarding the question of active ingredients in EMDR, even the utility of the actual eye-movements in EMDR is unclear, since other stimuli are used clinically. Only one non-saccade trial was available for comparison with the EMDR trials in our meta-analysis (Devilly and Spence, 1996), therefore permitting little comment on this issue. When all eye-movement conditions in the meta-analysis were compared to this one fixed-eye condition, EMDR was more effective than the fixed-eye control across measures. However, when the fixed-eye control was compared to the EMDR condition within the same study (i.e. Devilly and Spence, 1996), the fixed-eye condition was comparable to EMDR across measures. However, that study may have been compromised by insufficient treatment duration because only two EMDR sessions for multiply traumatized sessions were used. Thus, there may not have been sufficient time to treat each traumatic memory.

Pitman and colleagues (1996b) also included a fixed-eye control (that included hand-tapping) in their EMDR study. This was not included in the meta-analysis because it was tested within a crossover design for which we already included the EMDR treatment phase. Pitman and colleagues report that the fixed-eye condition performed as well if not better than the EMDR trial. Another study not included in the meta-analysis (due to the fact that not all patients had full PTSD) reported that a fixed-eye control demonstrated large treatment effect sizes comparable to that of the EMDR condition, although the eye movement condition was more efficient (Renfrey and Spates, 1994). This study also demonstrated that a condition using light bars versus a therapist's finger to stimulate eye-movements was also comparable to the other conditions, suggesting that alternate means of eye-movement production may be comparable to the procedures originally described for EMDR. Clarification of the mechanisms by which symptoms change and the active ingredients in EMDR is now critical given its apparent efficacy. Without such clarification, the acceptability of EMDR within the professional community is likely to remain controversial.

Recent efforts in treatment outcome research have been made to examine both separate and combined treatments for various disorders. In a recent meta-analysis of treatments for panic disorder, Gould *et al*. (1995) reported that the combination of

benzodiazepine treatment with behaviour therapy is less effective than behaviour therapy alone. Future research is needed to determine whether a similar finding holds for PTSD. That is, whether combined benzodiazepine therapy and psychological therapy is less effective than the latter alone. Another direction for future research suggested by the results of this meta-analysis is the study of the effectiveness of combined psychological therapies (e.g. combined behaviour therapy and EMDR) versus individual psychological therapies. Finally, we focused our analysis on studies of fully diagnosed chronic PTSD in adults. Future studies examining the comparability of treatment efficacy in subclinical PTSD and full PTSD in children would also enhance our understanding of the treatment of psychological trauma.

A limitation of this paper is the relatively low number of trials available per treatment condition, and our corresponding use of 90% rather than 95% confidence intervals. Yet despite the small number of trials, the number of trials was sufficient to detect differences in effect sizes across various treatment conditions. Even so, more research trials on PTSD treatment outcome would likely increase the power of the meta-analysis and would also permit more fine-grained analyses. Future research should address this issue. Another limitation that was true for our study and is also common to many meta-analyses is that we were only able to base our results on completer-analyses. It remains to be seen whether our conclusions hold up for intent-to-treat analyses. Although effect sizes should be smaller for intent-to-treat analyses, we expect that most of these findings would maintain. One exception is that the effect sizes for drug treatments may diminish more than those for psychological therapies, because the former had more dropouts, and dropouts may have poorer treatment response.

To conclude, this meta-analysis provides new information about the relative efficacy of treatments for PTSD. Our results support the use of behaviour therapy, EMDR, and SSRIs. It remains to be seen whether the efficacy of treatment can be improved by using these interventions in combination.

ACKNOWLEDGEMENTS

This research was supported in part by a grant from the Medical Research Council of Canada and a training grant from the National Institute on Drug Abuse. Thanks to Drs Baker, Blanchard, Burstein, Carlson, Devilly, Foa, Frueh, Lazrove, Lipper, Lohr, Marcus, Pitman, Rothbaum, Scheck, Shapiro and Wilson for providing unpublished data.

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