Variants of Exposure and Response Prevention in the Treatment of Obsessive-Compulsive Disorder: A Meta-Analysis

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Consistent findings suggest that exposure and response prevention (ERP) procedures are highly effective in treating obsessive-compulsive disorder (OCD). However, the studies that have reported success with this intervention have employed numerous variations in treatment procedures. Four general variations have been (1) whether the exposure sessions were supervised by the therapist or conducted by the patient on his or her own, (2) whether in vivo or imaginal exposure was used, (3) whether exposure stimuli were presented, beginning with the least or the most anxiety-evoking, and (4) whether response prevention involved the complete or partial abstinence from ritualizing. Whereas a few authors have addressed the relative efficacy of these procedural variations within single studies, results have been largely equivocal. We employed meta-analytic methods to quantitatively examine the degree of symptom improvement associated with the aforementioned variations of ERP. A total of 38 trials from 24 controlled and uncontrolled studies were included in the meta-analysis. Effect sizes were calculated as the standardized within-group change from pre- to posttreatment, a procedure that varies from traditional meta-analytic methods and likely yielded inflated estimates of treatment efficacy. Our results suggested that therapist-supervised exposure was more effective than self-controlled exposure. Further, the addition of complete response prevention to exposure therapy was associated with better outcome than partial or no response prevention. In reducing symptoms of anxiety, the combination of in vivo and imaginal exposure was superior to in-vivo exposure alone. Findings are discussed in terms of advancing the effectiveness of ERP in the treatment of OCD.

Obsessive-compulsive disorder (OCD), once thought to be a rare and unmanageable condition, is now known to be the fourth most common psychiatric disorder after phobias, substance abuse, and major depression (Reiger, Narrow, & Raye, 1990). More importantly, OCD can now be fairly well-

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controlled with contemporary behavioral and pharmacological therapy. Meyer (1966) is credited with being the first to report successful behavioral treatment of OCD. He exposed patients directly to anxiety-evoking stimuli and then prevented them from carrying out their compulsive rituals. This treatment procedure has become known as exposure and response prevention (ERP). In the ensuing 30-year period, consistent findings in treatment outcome research have established ERP as a successful therapy for OCD. Stanley and Turner (1995), in reviewing this literature, concluded that 63% of OCD patients could be expected to show at least some favorable response to ERP. Equally impressive is that these positive results are achieved in an average of 14 sessions.

Exposure and response prevention are most often used in tandem for OCD patients. Exposure involves purposely evoking anxiety by direct confrontation with the situations that produce fear in the patient (e.g., touching a toilet seat) while demonstrating the nonoccurrence of the feared consequences. Response prevention involves refraining from ritualistic or otherwise compulsive behavior (e.g., no washing for the rest of the day). An obvious function of response prevention is to prolong exposure since ritualistic behavior is the method used by people with OCD to escape from anxiety. Often, a friend or family member may be involved with treatment to offer support and assistance. Importantly, the patient is typically given an active role in the process of planing a treatment strategy. For detailed descriptions of ERP procedures, see Riggs and Foa (1993) and Steketee (1993).

Despite the advances that have been made over the past 3 decades in demonstrating the efficacy of ERP, there has been tremendous variation in the treatment procedures used in these research studies. Further, there is little consensus regarding what are the optimal procedures for ERP, even when variations have been directly compared (Emmelkamp, 1982; Stanley & Turner, 1995). Previous outcome trials of ERP can be characterized as having four main dimensions of procedural variability, including: (a) who controls/ supervises the exposure, (b) the evocative medium, (c) the exposure strategy (gradual or flooding), and (d) the degree of response prevention.

Control of exposure. Whether exposure is supervised by the therapist in-session (therapist-controlled) or given as a homework assignment for the patient (self-controlled) has varied across studies. In a direct comparison, Emmelkamp and Kraanen (1977) found no differences in outcome between these two procedures following treatment. These investigators addressed this question with the intention of establishing a self-controlled procedure for treating OCD that would be both efficient and cost-effective.

Evocative medium. In therapy, it is often impossible to expose a person with OCD to the actual situations that evoke anxiety. Consider the man with obsessional thoughts about the death of a loved one. Although it would be impossible to re-create the actual situation, the event could be imagined with the aid of the therapist. Some ERP studies have used in-vivo exposure (exposure to real life objects) and some have employed imaginal exposure proce-

dures (imagining the anxiety-evoking situation). Still others have used both procedures together. Research studies comparing these techniques have generally not found significant differences in treatment efficacy (Foa, Steketee, & Grayson, 1985; Foa, Steketee, Turner, & Fischer, 1980; Rabavilas, Boulougouris, & Stefanis, 1976).

Exposure strategy. Across the treatment literature, some investigators have opted to have patients begin exposure with the most anxiety-evoking stimulus (flooding). In other studies, a gradual progression to increasingly more anxiety-evoking stimuli (gradual exposure) was used. Boersma, den Hengst, Dekker, and Emmelkamp (1976) conducted a direct comparison of these two procedures and reported that gradual exposure and flooding were not significantly different in overall effectiveness.

Degree of response prevention. The degree to which participants in OCD treatment studies have been instructed to abstain from ritualizing varies from study to study. Some research has employed complete response prevention, while other studies have used a gradual or partial method. Additional treatment trials excluded response prevention altogether. Complete response prevention in combination with exposure appears to yield superior OCD symptom reduction (Foa, Steketee, Grayson, Turner, & Lattimer, 1984; Foa, Steketee, & Milby, 1980).

Even though ERP is a well-established and clearly efficacious procedure for treating OCD, the general lack of definitive conclusions regarding the relative efficacy of variants of ERP is somewhat disappointing. One explanation for these generally null conclusions might be that they are based largely on single studies, many of which included small sample sizes. Thus, it seemed desirable to use meta-analytic methods to investigate the effects of these procedural variations by aggregating the results of many ERP studies. An advantage of meta-analysis is that it involves converting the individual results of primary studies into standardized effect sizes that can be compared across treatment trials. These techniques provide a powerful method to infer differences between variants of ERP.

Method

Studies

OCD treatment studies were identified through searches of the following media: PsycLit and MedLine electronic databases, reference lists from publications concerning OCD, and an issue-by-issue examination of relevant journals published through 1995.[1] As in all previous OCD treatment reviews,

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[1] The following journals were searched: *American Journal of Psychiatry, Archives of General Psychiatry, Behavior Therapy, Behaviour Research and Therapy, British Journal of Clinical Psychology, British Journal of Psychiatry, Journal of Anxiety Disorders, Journal of Behavior Therapy and Experimental Psychiatry, Journal of Clinical Psychiatry, Journal of Consulting and Clinical Psychology.*

only published research was considered for inclusion. Three inclusion criteria were adopted. First, inclusion was limited to studies with a treatment condition in which some form of confrontation with anxiety-evoking stimuli (exposure) or a plan for abstinence from rituals (response prevention) was implemented. Treatments that combined exposure procedures with other psychological therapies, such as cognitive restructuring or self-instructional training, were included. However, treatment groups that received an active medication or placebo in combination with exposure were withheld.[2] Second, only investigations of adult samples with the primary diagnosis of OCD, or the former label "obsessive-compulsive neurosis," were included. Studies in which patients had concurrent diagnoses with active phases of other disorders (e.g., psychotic disorders) were excluded. This criterion was used because most of the studies considered for review limited their patient samples similarly. Third, only reports that provided sufficient statistical data to allow for computation of effect sizes at posttest and/or follow-up assessments were used. In order to rule out carry-over effects, studies using crossover designs were included only if outcomes were reported for each group separately before the crossover point. In these cases, effect sizes were calculated using the outcomes before the crossover.

Twenty-eight studies were identified in the literature search. Out of these, 3 were excluded because of insufficient information for calculating effect size and 1 was excluded because specific diagnostic criteria were not used. One additional study (Steketee, Foa, & Grayson, 1982) was removed because it contained data reported in a later study by Foa et al. (1984). Thus, 24 studies, with 38 ERP treatment groups, were included in the review. The year of publication ranged from 1975 to 1995. Descriptive statistics pertaining to the 38 treatment groups can be found in Table 1. A complete table of the effect sizes and treatment characteristics for each treatment group is contained in the Appendix.

Treatment

Variants of ERP. All subjects in this review received some form of exposure therapy. Variations in the treatment procedures along the four main dimensions discussed above were coded (control of exposure, evocative medium, exposure strategy and degree of response prevention). Table 2 provides these results, indicating how often each ERP variant was used.

Additional treatment variables. Treatment was conducted on an outpatient basis in 37 of the 38 trials (97.4%). Only one treatment group included inpatients. Patients were treated individually in 36 of the 38 trials

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[2] The decision to exclude trials in which ERP was combined with medication or pill placebo was based upon a quantitative review of the combination treatment studies which suggested that the effectiveness of combined ERP and medication treatments may depend more on whether active medication or placebo was received rather than on the type, or variant, of psychological intervention delivered (Abramowitz & Houts, 1995).

TABLE 1
CHARACTERISTICS OF THE 38 GROUPS TREATED BY EXPOSURE
WITH RESPONSE PREVENTION (ERP)

Characteristic	M^a	Range
Number of completers at posttreatment	15.58	4–43
Number of completers at follow-up	12.57	4–37
Mean age of participants (years)	34.07	29–41
Percent of male subjects	40.33	17–67
Pretreatment OCD symptom duration (years)	10.28	3–19
Length of session (minutes)	88.97	30–120
Number of sessions per week	2.80	1–5
Total number of sessions	14.00	4–24
Length of treatment (weeks)	7.54	2–28
Length of follow-up (weeks)	18.00	1–50
Percent of patients who discontinued treatment	9.06	0–29

[a] Each mean is based on at least 31 of the 38 ERP treatment groups.

(94.7%) and in group therapy in 2 instances. Other variables not directly related to ERP procedures were also varied by investigators across treatment groups. In 10 groups (26.3%), the patients' spouse, family, or friends played an active role by providing support or supervision with ERP procedures. The remaining 28 groups (73.6%) either specified that no family involvement took place or did not refer to this aspect of treatment. Cognitive restructuring pro-

TABLE 2
NUMBER AND PERCENT OF TREATMENT TRIALS USING EACH VARIANT OF ERP

ERP Dimension and Variants	Number of Trials	Percent
Control of exposure		
Therapist-controlled	26	68.4
Self-controlled	10	26.3
Not specified	2	5.3
Evocative medium		
In vivo	24	63.2
In imagination	1	2.6
In vivo and in imagination	6	15.8
Not specified	7	18.4
Exposure strategy		
Gradual exposure	29	76.3
Flooding	3	7.9
Not specified	6	15.8
Degree of response prevention		
Total RP	12	31.6
Partial RP	21	55.3
No RP	5	13.2

cedures, such as Rational Emotive Behavior Therapy (REBT), were combined with ERP in 5 of the 38 treatment groups (13.2%). The remaining 33 groups (86.8%) did not specify that cognitive restructuring was added to ERP.

The following additional variables were also coded from each treatment trial: year of publication, number of subjects in the treatment group, number of weeks of treatment, number of sessions per week, total number of sessions, length of sessions, and the percent of participants who did not complete the trial.

Estimating Treatment Effects

Of the 24 studies that were chosen for review, only one included comparisons between ERP groups and a control group (Fals-Stewart, Marks, & Schafer, 1993). Thus, in order to incorporate the entire sample of ERP research, outcome was assessed as the improvement from pre- to posttreatment or follow-up, rather than as the difference between treatment and control. Outcome that is assessed using this formula represents within-group changes during treatment but does not take into account the effects of nonspecific factors, maturation, history, regression to the mean, or other threats to internal validity.

In order to compare the effects of ERP across different measures of outcome, improvement from pretest to posttest was represented in terms of Cohen's (1977) d, a standardized measure of effect size. As used in this study, Cohen's d is defined as:

$$ d = \frac{M_{pre} - M_{post}}{SD_{pooled}} $$

where M_{pre} and M_{post} represent a treatment group's pretest and posttest means on a given outcome measure and SD_{pooled} is the pooled standard deviation. When the relevant means and standard deviations were not reported, effect sizes were calculated using the results of t or F tests. In two studies (Emmelkamp, van Linden, van den Heuvell, Ruphan, & Sanderman, 1989; Emmelkamp, de Haan, & Hoogduin, 1990) the outcome data for different treatment groups were averaged together because the authors found no differences between the treatments. In these cases, the same pre- and posttreatment means and standard deviations were used in computing effect sizes for these treatments.

Outcome data are likely to yield varying results depending on whether they are obtained by clinician ratings or patients' self-ratings. In this review, clinicians' ratings of improvement were reliably larger than patients' self-ratings across all symptoms, paired $t(17) = 4.92, p < .001$. In order to avoid measurement bias, effect sizes were calculated separately for these different types of measures. Similarly, separate effect sizes were obtained for measures of OCD, depression, and general anxiety. Effect sizes were calculated

TABLE 3
MEAN EFFECT SIZES FOR EXPOSURE WITH RESPONSE PREVENTION (ERP)
AT POSTTEST AND FOLLOW-UP

	Effect size[a]					
	Patient self-ratings			Clinician ratings		
Symptoms	N	M	SD	N	M	SD
Pretest-Posttest						
Obsessive-compulsive	29	1.16	0.78	12	1.41	0.50
General anxiety	5	1.64	1.01	5	1.64	0.37
Depression	23	0.76	0.39	1	0.96	–
Pretest-Follow-up						
Obsessive-compulsive	15	1.10	0.48	9	1.57	0.46
General anxiety	6	2.05	1.74	4	2.25	0.41
Depression	17	0.85	0.47	–	–	–

[a] Mean effect sizes are weighted by the sample size.

only from outcome measures with known psychometric properties (e.g., Maudsley Obsessional Compulsive Inventory; Hodgson & Rachman, 1977). If multiple measures were used to evaluate the same type of outcome (e.g., two different clinician-rated measures of OCD symptoms), the mean effect size from those measures was used for that particular treatment group.

Results

How Effective Is ERP?

Table 3 presents the posttest and follow-up effect sizes for self- and clinician-rated outcome measures of OCD, general anxiety, and depression. The large mean effect sizes on measures of OCD at posttest suggest that, on average, patients treated with ERP experienced a substantial decline in symptoms of OCD.[3] At follow-up, self-ratings indicated that patients maintained their treatment gains; clinician ratings suggest that further improvement took place following the termination of treatment.

As shown in Table 3, the effect sizes derived from self- and clinician-rated posttest measures of general anxiety were also profound. These results suggest that patients also experienced reductions in generalized anxiety symptoms. Follow-up effect sizes indicate that declines in these symptoms continued after the cessation of therapy.

Effect sizes derived from measures of depression indicate that depressive

[3] The calculation of effect sizes as the difference between a treatment group's pretest and posttest may account for the large magnitude of these treatment effects. These results may not be comparable to meta-analyses in which effect size is calculated as the difference between a treatment and a control group at posttest.

symptoms declined from pre- to posttreatment. These effect sizes, however, were not as large as those derived from measures of OCD or general anxiety. Patients' self-ratings suggest that at follow-up, treatment gains were maintained, yet additional symptom improvement was minimal. Clinicians' assessment of depression was reported by only one study at posttest and by none at follow-up.

Are Variants of ERP Differentially Effective?

To determine whether variations along the four dimensions of ERP were differentially effective in reducing OCD symptoms, differences between mean effect sizes for each dimension were tested for significance using the weighted least squares analysis of variance (ANOVA) procedure. The weighted mean effect size for each ERP variation and the F test of their differences appear in Table 4. Only self-ratings of OCD symptoms at posttest and follow-up provided enough data to produce meaningful results, thus Table 4 reports data from only these sources.

A significant effect for the control of exposure was found at both posttest, $F(1, 21) = 13.91, p < .001$, and at follow-up, $F(1, 12) = 11.95, p < .005$, for OCD symptoms. The mean effect sizes imply that the reduction in OCD symptoms was significantly larger when exposure was conducted in session and under the control of the therapist as compared to when patients were

TABLE 4
EFFECTS OF ERP VARIANTS ON PATIENT'S SELF-RATINGS OF OCD SYMPTOMS
AT POSTTEST AND FOLLOW-UP

ERP Dimensions/Variants	Posttest				Follow-up			
		Effect size[ab]				Effect size[ab]		
	N	M	(SD)	F	N	M	(SD)	F
Control of exposure								
Therapist-controlled	19	1.58	(.79)	8.20**	9	1.47	(.44)	10.55***
Self-controlled	10	0.81	(.41)		6	0.84	(.31)	
Evocative medium								
In vivo	19	1.27	(.91)	0.24	10	1.19	(.46)	3.58
In vivo and imagination	6	1.46	(.55)		4	1.54	(.43)	
Exposure strategy								
Gradual exposure	25	1.33	(.69)	0.63	c			
Flooding	3	1.73	(1.19)					
Response prevention totality								
Total RP	11	1.67	(.91)	4.09*	7	1.46	(.41)	4.41*
Partial RP	17	1.11	(.70)		11	1.09	(.36)	

[a] Mean effect sizes calculated from self-rated measures of OCD symptoms. [b] Mean effect sizes are weighted by sample size. [c] Not enough data for an F test.
* $p < .05$. ** $p < .01$. *** $p < .005$.

expected to conduct exposure on their own for homework assignments. Variations in degree of response prevention also produced significant differences at both posttest, $F(1, 24) = 3.93$, $p < .05$, and follow-up, $F(1, 16) = 8.73$, $p < .01$. Greater improvement occurred for patients who completely abstained from compulsive rituals during the treatment period, in contrast to those who were not told to stop ritualizing. Presentation of exposure stimuli in vivo was as effective as imagining anxiety-evoking situations at post-treatment, $F(1, 21) = 0.03$, ns, and at follow-up, $F(1, 9) = 1.25$, ns. Also, there was no significant difference between treatments that included gradual exposure and those that included flooding, $F(1, 24) = 1.30$, ns.

A similar ANOVA was performed to examine whether the variants of ERP had any effects on general anxiety. The results of this ANOVA are summarized in Table 5. Once again, only self-rated measures provided a sufficient number of effect sizes for the calculation. As can be seen in Table 5, posttest and follow-up reductions in anxiety symptoms were significantly greater when therapist-controlled, as opposed to self-controlled, exposure procedures were implemented, posttest $F(1, 6) = 7.43$, $p < .05$; follow-up $F(1, 4) = 8.00$, $p < .05$. In contrast to the findings with OCD symptoms, an effect for evocative medium was found with general anxiety symptoms. Treatments that included both in vivo and imaginal exposure procedures were more effective than in-vivo exposure alone in reducing general anxiety symptoms at posttest, $F(1, 5) = 13.30$, $p < .01$. Sufficient data for a follow-up analysis were not available.

To examine the effects of variants of ERP on symptoms of depression, a third ANOVA was performed with effect sizes derived from self-rated measures of depression as the dependent variable. At posttest as well as at follow-up, no reliable differences in effect size were observed between the variants

TABLE 5
EFFECTS OF ERP VARIANTS ON PATIENT'S SELF-RATINGS OF GENERAL ANXIETY SYMPTOMS
AT POSTTEST AND FOLLOW-UP

		Posttest				Follow-up		
		Effect size[a,b]				Effect size[a,b]		
ERP Dimensions/Variants	N	M	(SD)	F	N	M	(SD)	F
Control of exposure								
Therapist-controlled	4	2.46	(1.25)	7.43*	2	3.65	(1.89)	8.00*
Self-controlled	3	0.86	(.03)		3	1.01	(.19)	
Evocative medium								
In vivo	4	0.91	(.15)	13.30**	4	1.08	(.26)	—[c]
In vivo and imagination	2	2.76	(1.20)					

[a] Mean effect sizes calculated from self-rated measures of general anxiety symptoms. [b] Mean effect sizes are weighted by sample size. [c] Not enough data for an F test.
* $p < .05$. ** $p < .01$.

on the control of exposure, form of exposure stimuli, and totality of response prevention dimensions. Insufficient data were available for an analysis of the exposure strategy dimension. Thus, variations in ERP procedures were not significantly different in reducing depressive symptoms in OCD patients.

Is There a Dose-Effect Relationship With ERP?

Several variables relating to temporal aspects of therapy were coded from the descriptions of the 38 treatments. These included the number of weeks in treatment, the number of treatment sessions per week, the total number of treatment sessions, and the length of the sessions. Because exposure was sometimes assigned as homework, the number of sessions does not accurately represent the amount of time spent in exposure to anxiety-evoking stimuli. Thus, the number of hours of therapist-controlled exposure was also coded. These variables represent the intensity of the treatment procedures or "dose" of ERP. To examine relationships between these variables and symptom reduction, correlation coefficients were computed with the effect sizes for OCD, general anxiety, and depression symptoms. Because of the small number of outcome measures used in the studies, these analyses were conducted only on effect sizes derived from self-rated measures. Significant findings are summarized below.

Length of sessions was the only variable that correlated significantly with effect sizes on measures of OCD. This relationship was found at both post-test, $r(24) = .47, p < .05$, and at follow-up, $r(12) = .64, p < .05$. Reductions in OCD symptoms were larger when individual therapy sessions lasted longer. Improvement on general anxiety symptoms was significantly correlated with the total number of sessions at posttest, $r(10) = .84, p < .005$, and follow-up, $r(6) = .95, p < .005$. Larger reductions in general anxiety symptoms occurred with treatments that included more therapy sessions. In addition, a strong relationship between anxiety effect sizes and the number of hours of in-session, therapist-controlled exposure was observed at posttest, $r(8) = .91, p < .005$. More therapist-controlled exposure was associated with greater improvement in anxiety symptoms. There were no significant correlations between treatment variables and effect sizes on measures of depression.

Are Characteristics of the Patients Related to Outcome?

Correlation coefficients were calculated to determine the relationships between effect size and the following patient variables: age, percent of the participants who were male, and duration of OCD symptoms prior to treatment. Effect size was weighted by the sample size. At posttest, the duration of OCD symptoms prior to therapy was significantly, negatively related to the effect size on measures of depression, $r(23) = -.45, p < .05$. This suggests that patients who suffer from OCD longer before receiving treatment may show less reduction in depression. No significant relationships were observed between patient variables and effect size at follow-up.

Discussion

Thirty-eight trials in which ERP was used in the treatment of OCD were quantitatively reviewed. Consistent with the conclusions of other literature reviews (e.g., van Balkom et al., 1994; Christensen, Hadzi-Pavlovic, Andrews, & Mattick, 1987; Stanley & Turner, 1995) and treatment outcome studies (e.g., Foa & Goldstein, 1978), the findings of this review suggest that behavior therapy by ERP produces considerable reductions in OCD symptoms at the end of treatment. Further, effect sizes at follow-up suggest that improvement was maintained for at least 18 weeks on average.

Posttest and follow-up effect sizes for measures of anxiety were even larger than those for OCD symptoms. Such a finding is not surprising considering a behavioral mechanism of ERP. Foa and Kozak (1986) argued that the therapeutic goal of exposure procedures is to modify the emotional response to feared stimuli. Since obsessions produce anxiety (Hodgson & Rachman, 1972), exposure is aimed at reducing the fear associated with obsessional phenomena (Emmelkamp & Geisselbach, 1981). However, obsessions are a symptom of OCD; and while ERP aids in the reduction of fear, the intervention may not prevent the obsessional phenomena from occurring. Obsessional symptoms may, therefore, account for a decrease in levels of anxiety that exceeds the decrease in OCD symptoms. Unfortunately, not enough data were available for a quantitative analysis of obsessional and compulsive symptoms separately.

Effect sizes with ERP treatment were somewhat smaller for measures of depression than for measures of general anxiety or OCD. Because many ERP treatment studies excluded patients with high levels of depression, one limitation of the present work is that our results likely underrepresent the levels of depression typically observed in OCD patients. Thus, the smaller effect sizes for measures of depression could be explained either by depression's resistance to ERP procedures, or to low pretest depression levels.

The main focus of this review was on the differences in outcome across variations in ERP procedures. Although a few studies have directly compared these variants, the results have been largely equivocal. This meta-analysis, however, allows conclusions to be drawn based on an aggregated sample of 24 outcome studies rather than based on the findings of a single trial. There were four dimensions upon which ERP procedures varied throughout the OCD treatment literature: (1) whether exposure sessions were supervised by the therapist or conducted by the patient on his or her own, (2) whether the evocative medium was real-life in-vivo presentation of stimuli, or imagined situations, (3) whether gradual exposure or flooding procedures were used, and (4) the totality of response prevention.

Therapist-controlled exposure was associated with greater improvement compared to self-controlled exposure on symptoms of OCD and general anxiety. It is likely that a therapist's presence adds nonspecific effects to treat-

ment in that coaching and support from a caring individual may put a person more at ease during exposure. Additionally, the integrity of treatment is presumably maintained by a therapist to assess anxiety levels and ensure that exposure continues until the individual's anxiety decreases. Because of the subjective aversiveness of confronting one's own fears, individuals doing exposure on their own might be inclined to discontinue exposure assignments prematurely—before extinction of anxiety has taken place. Additionally, the therapist's help in maintaining focused attention on the anxiety-provoking situation might add to ERP's effectiveness (Grayson, Foa, & Steketee, 1982, 1986). It is important to note that the results of this review do not imply that self-controlled exposure should be avoided during therapy. Emmelkamp and Kraanen (1977) found that treatment gains were better maintained at followup when patients treated with supervised exposure were also taught self-controlled exposure procedures.

Symptoms of OCD were reduced equally well when exposure was presented only in vivo and when in vivo was combined with imaginal procedures. However, the combination of in vivo and imaginal procedures was significantly more effective in reducing posttreatment levels of general anxiety. This is concordant with the work of Lang (1977) and Foa and Kozak (1986), who proposed that the intentional activation of fear was a necessary condition for therapeutic anxiety reduction to take place. Lang suggested that the closer the match between exposure stimuli and the patient's actual fear model, the more effective treatment would be. For OCD patients, fears of tangible objects (e.g., knives) are often combined with fears of possible disasters (e.g., "I might harm someone I love"). Thus, Lang's matching hypothesis would predict that the anxiety in OCD would be most effectively reduced with the combination of in-vivo exposure to tangible stimuli and imaginal exposure to the consequences of anxiety-evoking fantasies. ·

Treatment programs that employed complete response prevention were associated with better outcomes than those that involved only partial or no response prevention. This finding, supported by previous research (Foa, Steketee, & Milby, 1980; Foa et al., 1984), suggests the importance of consistently blocking rituals during treatment. For example, someone who engages in compulsive showering might be urged to completely abstain from washing except for one 10 min shower every other day. Stricter and more discriminant response prevention procedures also relieve the patient from the responsibility of having to decide in which situations it is acceptable to ritualize (Steketee, 1993).

The present analyses also suggest that larger reductions of OCD and general anxiety symptoms occurred when therapy involved more sessions of longer length, and when the therapist controlled exposure. An area of study for further investigation is the impact of patient variables on treatment outcome. In this review, none of the patient variables were significantly related to outcome. Since OCD occurs with such diverse symptoms, this is a direction that appears promising for investigators intent on developing treatment

procedures that are maximally effective for different types of rituals and obsessional fears. Additionally, there remains some question about the effects of depression on treatment.

It is important to note that the effect sizes used in this meta-analysis were calculated by subtracting a treatment group's posttest (or follow-up) mean from it's pretest mean and dividing by the pooled within-group standard deviation. This represents a standardized measure of improvement from pre- to posttest on a group-by-group basis. Lipsey and Wilson (1993) have cautioned that pre-post effect sizes might overestimate actual treatment effects since within-group variability is likely to be smaller, and threats to internal validity are not controlled. To avoid this potential problem, effect sizes are often calculated by subtracting a treatment group's posttest mean from a control group's posttest mean. However, because very few of the studies included in the present review used control groups, the only way to obtain a sufficient number of effect sizes for meaningful analyses was to calculate effect sizes using pre- and posttest data. Thus, the effect sizes in the present research would be expected to be larger than conventional meta-analyses that use comparisons between treatment and control groups.

Although the major variables related to ERP procedures were coded in this review, it is possible that additional factors contributed to the pattern of results found here. For example, the use of cognitive restructuring techniques, such as REBT (Ellis, 1994) are commonly employed in clinical practice to help the patient over initial anxiety about exposure sessions. While only two treatment groups included in the present review specified the use of such a procedure (Emmelkamp & Beens, 1991; Emmelkamp, Visser, & Hoekstra, 1988), it is likely that cognitive techniques were employed more often, even if used informally.

Assessment tools represent another possible source of variability. Although standardized effect sizes were used to compare results across different measures of symptoms, the outcome measures from which effect sizes were derived might have varied in their sensitivity. To address this possible confound, only measures with known reliability, validity, and acceptance in the field were used in calculating effect sizes. Further, patients' self-ratings of outcome were analyzed separately from ratings made by clinicians.

In summary, while previous literature reviews have not found significant differences in the treatment effectiveness of various ERP procedures, the present research has identified such differences using meta-analytic procedures. Specifically, therapist-controlled exposure was associated with better outcome than self-controlled exposure. Using in-vivo and imaginal exposure together was superior to using in vivo alone. Combining complete response prevention with exposure was associated with better treatment gains than when partial response prevention was used. Also, therapy sessions that lasted longer (probably until habituation to exposure stimuli has occurred) were predictive of more improvement in symptoms.

Appendix

MEAN EFFECT SIZES AND CHARACTERISTICS FOR EACH ERP TREATMENT GROUP

Study	N	No. of Sess.	Control	Evoc. Med.	Exposure Strategy	Deg. of RP	Effect Size (follow-up in italics)		
							OCD	Anx.	Dep.
de Araujo, Ito, Marks, & Deale (1995)	23	9	therapist	in vivo	gradual	none	1.88 *1.97*	– –	0.65 *0.68*
de Araujo et al. (1995)	23	9	therapist	both	gradual	none	2.44 *2.44*	– –	1.00 *1.22*
Baxter et al. (1992)	9	15	therapist	–	–	partial	1.38	0.74	0.96
Boersma, den Hengst, Dekker, & Emmelkamp (1976)	7	15	therapist	in vivo	gradual	partial	3.20	–	1.43
Boersma et al. (1976)	6	15	therapist	in vivo	flooding	total	3.91	–	1.43
Emmelkamp, van der Helm, van Zanten, & Plochg (1980)*	7	15	therapist	both	gradual	total	1.88 *2.06*	– –	0.94 *0.88*
Emmelkamp et al. (1980)	8	15	therapist	in vivo	gradual	total	1.88 *2.06*	– –	0.94 *0.88*
Emmelkamp, Visser, & Hoekstra (1988)	9	14	self	in vivo	gradual	partial	.75 *1.05*	– –	.13 *.00*
Emmelkamp, van Linden, van den Heuvelt, Ruphan, & Sanderman (1989)	7	10	self	in vivo	gradual	partial	1.60 *1.26*	– –	1.13 *0.95*
Emmelkamp et al. (1989)	7	10	therapist	in vivo	gradual	partial	1.60 *1.26*	– –	1.13 *0.95*
Emmelkamp, de Haan, & Hoogduin (1990)	25	8	self	in vivo	gradual	partial	0.45 *0.50*	1.32 *1.48*	– –
Emmelkamp et al. (1990)**	25	8	self	in vivo	gradual	partial	0.45 *0.50*	1.32 *1.48*	– –
Emmelkamp & Beens (1991)***	10	12	therapist	in vivo	gradual	partial	0.73 *1.23*	1.50 *2.25*	– –
Emmelkamp & Beens (1991)	11	12	self	in vivo	gradual	partial	0.56 *0.76*	1.23 *1.94*	– –
Emmelkamp & Kraanen (1977)	7	10	self	in vivo	gradual	partial	1.11	–	0.44
Emmelkamp & Kraanen (1977)	7	10	therapist	in vivo	gradual	partial	1.11	–	0.44

(continued)

APPENDIX (*Continued*)

Study	N	No. of Sess.	Control	Evoc. Med.	Exposure Strategy	Deg. of RP	Effect Size (follow-up in italics)		
							OCD	Anx.	Dep.
Emmelkamp & de Lange (1983)	6	13	therapist	in vivo	gradual	total	1.18	–	0.97
Emmelkamp & de Lange (1983)	6	13	self	in vivo	gradual	total	1.33	–	1.85
Fals-Stewart, Marks, & Schafer (1993)	31	24	therapist	both	gradual	partial	1.27 *1.14*	– *–*	0.49 *0.38*
Fals-Stewart et al. (1993)***	30	24	therapist	in vivo	gradual	partial	1.58 *1.27*	– *–*	0.45 *0.41*
Foa, Steketee, & Milby (1980)	4	14	therapist	in vivo	gradual	none	–	1.57	–
Foa et al. (1980)	4	14	–	–	–	total	–	0.71	–
Foa, Steketee, Grayson, Turner, & Lattimer (1984)	12	15	therapist	in vivo	gradual	none	1.97 *1.04*	1.69 *–*	0.91 *0.92*
Foa et al. (1984)	9	15	–	–	–	total	1.44 *1.18*	0.83 *–*	0.67 *0.74*
Foa et al. (1984)	11	15	therapist	in vivo	gradual	total	2.48 *1.06*	0.80 *–*	1.00 *0.48*
Foa, Steketee, & Grayson (1985)	10	12	therapist	imaginal	gradual	total	0.67 *1.06*	– *–*	– *–*
Foa et al. (1985)	9	15	therapist	in vivo	gradual	total	0.73 *1.30*	– *–*	– *–*
Foa & Goldstein (1978)	21	14	therapist	both	gradual	total	1.23 *1.29*	– *–*	– *–*
Hackman & McLean (1977)	5	–	therapist	in vivo	flooding	partial	0.38	–	–
van den Hout, Emmelkamp, Kraaykamp, & Grietz (1988)	43	20	therapist	in vivo	gradual	partial	1.58	–	–
Keijsers, Hoogduin, & Schaap (1994)	40	18	self	in vivo	gradual	partial	0.38	–	–
Kozak, Foa, & Steketee (1988)	14	15	therapist	both	gradual	total	1.67	–	–
Krone, Himle, & Nesse (1991)****	36	7	therapist	–	–	partial	0.84 *1.53*	– *–*	0.50 *0.86*
Mehta (1990)	15	24	therapist	–	–	partial	1.27 *0.95*	2.35 *3.53*	1.14 *1.33*

(*continued*)

APPENDIX (Continued)

Study	N	No. of Sess.	Control	Evoc. Med.	Exposure Strategy	Deg. of RP	Effect Size (follow-up in italics)		
							OCD	Anx.	Dep.
Mehta (1990)**	15	24	therapist	–	–	partial	1.62	4.00	1.75
							1.75	5.21	2.22
van Oppen et al. (1995)	29	16	self	in vivo	gradual	partial	0.75	–	0.44
Rabavilas, Boulougouris, & Stefanis (1976)	12	13	therapist	both	flooding	none	0.90	–	–
Thornicroft, Colson, & Marks (1991)	39	16	self	–	gradual	partial	0.68	–	0.62
							0.95	–	0.99

Note: – = could not be determined; Control = control over exposure; RP = response prevention; * self-instructional training added to ERP; ** Spouses/family members were instructed to assist with treatment; *** REBT added to ERP; **** Group treatment.

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Note. Studies that are included in the review are preceded with an asterisk.

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