The Effectiveness of Treatment for Pediatric Obsessive-Compulsive Disorder: A Meta-Analysis

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The last decade has seen a noticeable increase in the number of treatment outcome studies for pediatric obsessive-compulsive disorder (OCD). The present article describes a meta-analysis of this literature with the aim of quantifying treatment effects and examining the extent to which various patient or treatment variables are related to outcome. Results showed that pharmacotherapy with serotonergic antidepressants and cognitive-behavioral therapy involving exposure and response prevention are each effective in reducing OCD symptoms. Cognitive-behavioral therapy produced larger effect sizes and greater rates of clinically significant improvement compared to medication, although there were methodological differences between medication and psychotherapy studies.

OBSESSIVE-COMPULSIVE DISORDER (OCD) is an anxiety disorder that involves (a) intrusive unwanted thoughts, ideas, or images that evoke anxiety (obsessions), and (b) behavioral or mental rituals performed to neutralize this distress (compulsions). The illness affects between 2% and 3% of adults and approximately 1% of children and adolescents (Flament et al., 1988). Untreated symptoms typically persist and as many as 50% of adult OCD cases develop during childhood (Karno & Golding, 1991; Rasmussen & Eisen, 1990). Moreover, sufferers usually experience impairment in social, academic, or family functioning. Considering its prevalence and associated personal costs, OCD is clearly a significant public health concern. Given the fact that childhood onset predicts adult morbidity, identifying effective interventions for this disorder in pediatric populations is imperative.

Research supports the efficacy of two forms of treatment for pediatric OCD: psychotropic medications and psychotherapy. Medication is the most widely available, and therefore the most commonly employed, treatment. Serotonin reuptake inhibitors (SRIs; e.g., fluoxetine) are the most well-studied agents and are thought to be effective through their action on serotonin neurotransmission. Other medications that selectively effect norepinephrine (e.g., desipramine) have not been found effective for pediatric OCD (e.g., Leonard et al., 1989). Due to the potential for relapse upon discontinuation, SRI therapy is usually long term (Leonard et al., 1989).

Cognitive-behavioral therapy involving exposure and response prevention (ERP) is the most well-studied psychological treatment for OCD (e.g., March & Leonard, 1996) and is considered the treatment of choice for children and adolescents (Expert Consensus Guidelines; March, Frances, Carpenter, & Kahn, 1997). Conceptualized based on the functional relationship between obsessions and compulsions, ERP aims to weaken associations between obsessions and increased anxiety, and between compulsions and anxiety relief. Treatment involves repeated and prolonged confrontation with stimuli that evoke obsessional fear (e.g., the number 13) while simultaneously refraining from compulsive behaviors (e.g., counting; March & Mulle, 1998). As obsessional fear dissipates without rituals, the patient learns that compulsive rituals are not necessary to prevent disaster or reduce anxiety. An important shortcoming of ERP is the lack of therapists trained in its use.

Several narrative reviews of the pediatric OCD treatment literature have been published (e.g., Grados, Scahill, & Riddle, 1999; Rapoport & Inoff-Germain, 2000). While informative, such reviews do not quantify the effects of treatment across studies. In contrast, meta-analysis is an approach to literature review in which results are aggregated to quantitatively determine the magnitude of treatment effectiveness. A number of meta-analyses of the OCD treatment literature have been published (e.g., Abramowitz, 1997; van Balkom et al., 1994), yet these have focused exclusively on the treatment of adults. Thus, the purpose of the present meta-

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analysis was to evaluate the effects of treatment for pediatric OCD as reported in the literature. Specifically, we examined the effects of SRI medication and ERP, as well as the extent to which these treatments produce clinically significant improvement in OCD symptoms.

Method

STUDIES

Pediatric OCD treatment studies were identified through searches of the PsycINFO and MedLine databases (using the following keywords: *OCD, medication, cognitive behavior therapy, behavior therapy, exposure*), reference lists from research articles and other materials on childhood OCD, and an issue-by-issue examination of relevant journals between 1970 and December 2004.[1] As in previous OCD treatment reviews, only published research was included. Two broad criteria were initially used to select studies. First, only investigations in which samples were exclusively children and/or adolescents (age < 18) with a primary diagnosis of OCD (as indicated in study inclusion criteria) were considered. Studies in which patients had concurrent comorbid diagnoses were included as long as OCD was the primary disorder. Second, only studies that reported outcome on at least one measure of OCD were included.

Twenty-two studies met the initial inclusion criteria. Two included sample sizes of four patients or less and thus were excluded due to this small *N*. An additional study by Leonard et al. (1991) was excluded because it examined the effects of substituting one medication (desipramine) during long-term treatment with another (clomipramine). Ten of the 11 psychotherapy trials examined ERP. The remaining study, which evaluated inpatient psychodynamic therapy over an unspecified period of time, was excluded to reduce the heterogeneity of the psychotherapy treatments under review. The remaining 18 studies included 28 treatment and control groups[2]: 11 groups received pharmacotherapy, 10 received psychotherapy, and 7 had received a placebo. Publication year ranged from 1983 to 2004.

The 18 studies examined either SRI medication or ERP; none systematically examined the effects of combining these two interventions. Nevertheless, patients in many ERP studies were concurrently using medication; thus, we coded this information. Studies of SRIs examined a total of six different agents. Four treatment groups received clomipramine and two additional groups received fluoxetine. Paroxetine, sertraline, and fluvoxamine each appeared only once. This allowed us to examine the effects of clomipramine separately from other serotonergic medications. Only one treatment group received a nonserotonergic medication: Leonard et al. (1989) compared desipramine to clomipramine. To reduce the heterogeneity of the pharmacological treatments under review, we excluded the despiramine group from further analyses. One additional study (Wever & Rey, 1997) included a group of patients that received different SRIs. The six studies that included placebo groups allowed for the examination of changes in symptoms with the administration of a placebo.

With regard to ERP, some studies described an intensive treatment regimen in which patients met daily with the therapist to do exposure practices; whereas in others, sessions were held once weekly and therapy time was spent planning exposure assignments to be performed for homework. Regardless, all ERP protocols incorporated homework as well as parental assistance with treatment. For example, parents were instructed in how to empathetically encourage and reinforce their children in performing treatment exercises and resisting compulsive urges.

Descriptive characteristics of the 18 studies are displayed in Table 1. Although all patients in these studies met *DSM* criteria for OCD, the samples were somewhat heterogeneous with respect to other potentially meaningful variables (e.g., comorbidity). As can be seen, the typical patient was an adolescent male experiencing moderate OCD symptoms

TABLE 1 Characteristics of Pediatric OCD Treatment Studies

Study Characteristic	*M*[a]	Median	Range
Patients per treatment group	30.0	16.5	11–94
Percentage attrition (posttest)	12.9	5.6	0–38
% male patients	58.3	53.2	50–71
Patient age (years)	13.7	13.8	13–15
Initial Y-BOCS/CY-BOCS score	23.4	22.8	22.3–24.5
Duration of OCD (years)	4.2	4.9	2.5–5.2
Length of treatment (weeks)	11.6	12.0	10–14
Number of professional contacts	12.4	12	7–17
% with psychiatric comorbidity	25.3	19.5	10–56
% comorbid tics	7.3	5.0	2–14

Note. OCD = obsessive-compulsive disorder; Y-BOCS = Yale-Brown Obsessive-Compulsive Scale; CY-BOCS = Child Yale-Brown Obsessive-Compulsive Scale.

[a] Each mean is based on at least 11 of the 18 studies.

[1] The issue-by-issue search was conducted for the following journals: *American Journal of Psychiatry, Archives of General Psychiatry, Behavior Therapy, Behaviour Research and Therapy, British Journal of Psychiatry, Journal of the American Academy of Child and Adolescent Psychiatry, Journal of Anxiety Disorders, Journal of Behaviour Therapy and Experimental Psychiatry, Journal of Consulting and Clinical Psychology, Journal of Nervous and Mental Disease*, and *Psychopharmacology Bulletin*.

[2] A table of individual effect sizes derived from each study included in the meta-analysis is provided in the Appendix.

TABLE 2 Characteristics of Measures Used to Derive Effect Sizes from Pediatric OCD Treatment Studies

Instrument	Symptom[a]	Type[b]	% Studies
Y-BOCS or CY-BOCS	OCD	I	78
NIMH Global OCD Scale	OCD	I	56
Hamilton Depression Rating Scale	D	I	22
Leyton Obsessional Inventory–Child Version	OCD	SR	22
Children's Depression Inventory	D	SR	11
Hamilton Anxiety Rating Scale	A	I	11
NIMH Anxiety Rating Scale	A	I	11
NIMH Depression Rating Scale	D	I	11
Brief Psychiatric Rating Scale–Depression subscale	D	I	6
Children's Depression Scale	D	SR	6
Comprehensive Psychiatric Rating Scale–OCD subscale	OCD	I	6
Global OCD severity rating	OCD	I	6
Multidimensional Anxiety Scale for Children	A	SR	6
Obsessive-Compulsive Rating Scale	OCD	I	6
Revised Children's Manifest Anxiety Scale	A	SR	6
Self-Rated Depression Scale	D	SR	6

Note. OCD = obsessive-compulsive disorder; Y-BOCS = Yale-Brown Obsessive-Compulsive Scale; CY-BOCS = Child/Yale-Brown Obsessive-Compulsive Scale; NIMH = National Institute of Mental Health.
[a] A = anxiety symptoms, D = depressive symptoms.
[b] I = interview, SR = self-report.

as measured by the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) or the child version of this instrument (CY-BOCS).[3]

Treatment outcome was measured in the 18 studies by a variety of interview and self-report instruments that assessed signs and symptoms of OCD, depression, and anxiety. We derived effect sizes only using data based on assessment instruments of known reliability and validity. The lone exception occurred in a study by Bolton, Collins, and Steinberg (1983) in which the only assessment of OCD was a scale in which symptom severity was rated from 0 (*no symptoms*) to 3 (*severe*). Table 2 displays characteristics of the measures from which we derived effect sizes. As can be seen, the Y-BOCS/CY-BOCS was the most commonly used instrument.

ESTIMATING TREATMENT EFFECTS

Seven of the 18 studies included comparisons between active treatments and a placebo, and 3 included comparisons between different active treatments. The most common research design, however, was a repeated-measures design in which a single group was assessed at pre- and posttest. To ensure that effect sizes derived from studies using different research designs were on a common metric, we followed the suggestion of Morris and DeShon (2002; personal communication with Scott B. Morris, October 1, 2003) and calculated each effect size as the difference from pre- to posttest (or follow-up) divided by the pretest standard deviation. By doing this for each treatment group within a study (and placebo groups in the available placebo-controlled studies), we were able to compare the effects of treatments across different measures of outcome regardless of the experimental design. Importantly, effect sizes computed in this manner merely reflect within-group change and do not partial out effects of nonspecific factors such as the passage of time; therefore, they may overestimate the actual effectiveness of specific treatment procedures themselves (Morris & DeShon, 2002).

For most outcome measures, higher scores indicated greater severity. For the few cases in which lower numbers represented more severity, the sign of the effect size was adjusted so that positive effect sizes always indicated that the treatment group improved from pre- to posttest. Thus, an effect size of 1.50 indicated that the posttest mean score was one-and-a-half standard deviations lower than the pretest score. Most effect sizes were calculated from means and standard deviations reported in the research articles. However, when this information was unavailable we employed available methods (e.g., Morris & DeShon, 2002; Ray & Shadish, 1996) for computing (or estimating) effect sizes from other data. Two studies did not report enough information from which to estimate effect sizes, yet we obtained the necessary information by contacting the study authors. We also applied Hedges' (1982) small sample correction to all effect sizes.

PRELIMINARY CONSIDERATIONS

The effects of treatment in each study were typically assessed by multiple outcome measures (M = 3.9, range = 1 to 9). We averaged all effect sizes derived from the same treatment group, yet also calculated separate mean effect sizes for measures of OCD, depression, and anxiety. These additional means were subsequently used in analyses in which we assessed the effects of treatment on particular kinds of symptoms.

Because only published research was included, we examined the likelihood that our effect sizes were inflated due to a publication bias. Such a bias may occur if studies reporting significant findings (large effect sizes) are published, whereas those obtaining null results (small effect sizes) are not. We computed the fail safe N (Orwin, 1983) to deter-

[3] The main difference between the Y-BOCS and CY-BOCS is the substitution of simpler language for the various probe items.

mine the number of unpublished trials obtaining small effect sizes required to reduce our overall mean effect size for all active treatments combined ($d = 1.16$) to a trivial level. This analysis indicated that over 50 unpublished studies with effect sizes of zero would be needed to reduce our effect size to a trivial level (e.g., < 0.30) and overturn our conclusion that treatment produced at least a small to moderate effect. Given the relative shortage of research on pediatric OCD and the challenges in recruiting and retaining study samples, it is highly unlikely that this many trials would exist unpublished. Thus, we concluded that our results were unlikely to have been affected by publication bias.

The precision with which a given study estimates treatment effects is influenced by the study's methodology. More rigorously conducted research (i.e., larger sample sizes and greater experimental control) yields more precise estimates of treatment effects. Previous authors (e.g., Hedges & Olkin, 1985; Morris & DeShon, 2002) have suggested that allowing more precise estimates to have a stronger influence on the mean effect size will increase the accuracy of meta-analytic results. To address this issue, all mean effect sizes reported below are based on weighted least squares analyses in which individual effect sizes were weighted by the reciprocal of the sampling variance (which takes into account sample size and design; see Morris & DeShon, 2002, p. 117, Table 2).

Results

The overall weighted mean effect size across the 28 groups at posttest was 0.99 (95% confidence interval = 0.88, 1.10). This indicates that, on average, symptoms reported by young people with OCD improved substantially from pre- to posttest. Follow-up effect sizes could be obtained from only five studies, for a mean weighted effect size of 2.20 (95% confidence interval = 1.42, 2.97). Given the small number of observations and the fact that within these studies, posttest and follow-up effect sizes were not significantly different from each other, paired $t(4) = 1.60$, $p > .10$, and strongly correlated, $r(5) = .97$, $p < .001$, the results presented in the remainder of the review are based on posttest data only.

A stem-and-leaf plot of posttest effect sizes (Figure 1 was constructed to examine the homogeneity of these observations. We found that sampling variance accounted for only a small proportion of the observed variability in effect sizes, $r(25) = .20$, $p = .10$, suggesting substantial variability in effect sizes that was not attributed to sampling error (Hunter & Schmidt, 1990). Hedges' test of homogeneity ($Q = 73.58$) further revealed evidence of heterogeneity among the effect sizes, warranting a search for possible moderator variables.

2.6	01
2.5	
2.4	
2.3	
2.2	
2.1	
2.0	
1.9	247
1.8	
1.7	2
1.6	
1.5	37
1.4	9
1.3	30
1.2	
1.1	
1.0	5
0.9	018
0.8	
0.7	48
0.6	4
0.5	3789
0.4	069
0.3	00
0.2	
0.1	2
0.0	

FIGURE 1 Stem and leaf plot of posttest effect sizes. Digits to the left (stems) of the vertical line are read as the ones and tenths place of each effect size. Numbers to the right (leafs) of the vertical line are the hundredths place for each effect size. Multiple leafs indicate that there were multiple effect sizes with the same stem (e.g., 1.92, 1.94, 1.97).

TYPE OF TREATMENT

Table 3 presents the effects of different types of treatment as well as the effects of pill placebo on OCD symptoms. As can be seen, each form of treatment (including placebo) led to reliable improvement from pretest to posttest. The variation in effect sizes between SRI medication, ERP, and placebo was significant, $F(2, 27) = 31.66$, $p < .01$. Post-hoc (LSD) tests indicated that ERP was more effective than SRI medication, which was more effective than placebo (ps $< .01$). Among the seven placebo-controlled studies, effect sizes for SRI medication (weighted $M = 1.04$, $SD = 0.26$) were significantly greater than placebo effect sizes (weighted $M = 0.48$, $SD = 0.09$), paired $t(6) = 4.94$, $p < .01$. As some researchers have asserted that clomipramine is superior to other SRIs in the treatment of adults with OCD (e.g., Greist et al., 1995), we examined this issue in our data. However, the mean effect size for clomipramine and the combined mean for other SRIs were not significantly different, $t(8) = 0.94$, $p = .38$.

TABLE 3 Effects of Different Treatment for Pediatric OCD

	Effect Size					
	OCD Symptoms			Anxiety and Depression		
	N	*M*	95% CI	*N*	*M*	95% CI
SRI medication	11	1.13**	0.82, 1.25	7	0.33	−0.03, 0.68
Clomipramine	4	1.10*	0.30, 1.89	3	0.57*	0.15, 0.98
Other SRIs	6	1.01*	0.81, 1.22	4	0.23	−0.45, 0.92
ERP	10	1.98**	1.40, 2.56	4	0.48	−0.02, 1.00
Placebo	7	0.48**	0.40, 0.55	4	0.06	−0.04, 0.17

Note. Means and confidence intervals (CI) are based on weighted least squares analyses in which effect sizes were weighted by the inverse of the sampling variance estimate.
* $p < .05$, ** $p < .01$, effect sizes significantly different from zero.

Researchers used a variety of measures to assess anxiety and depressive symptoms in the studies under review, likely because these represented secondary outcome variables. Eight of the 28 treatment groups (32%) were assessed using measures of anxiety and 14 groups (56%) were assessed with measures of depression. Analyses revealed that for the 7 groups assessed with both anxiety and depression measures, there were no differences in the effect sizes derived from each type of measure, paired $t(6) = -0.12$, $p = .91$. Therefore, we computed an average effect size derived from measures of anxiety and depression for groups receiving each treatment. As Table 3 indicates, only clomipramine was associated with reliable improvement in these symptoms. The small number of observations precluded further analyses of these effect sizes.

CLINICAL SIGNIFICANCE AND RELIABILITY OF TREATMENT EFFECTS

While we found that young people with OCD improve following treatment with ERP or SRIs, these results do not speak to the extent to which treated patients experience clinically meaningful improvement. The effect size statistic describes the magnitude of pre- to posttest change, but not patients' level of posttest symptomatology relative to individuals without OCD (even treatments with large effect sizes may leave patients with residual symptoms). Information about end-state functioning is particularly relevant to patients and clinicians interested in the extent to which pediatric OCD patients "recover" as a result of treatment.

Therefore, to examine the clinical significance of pediatric OCD outcomes we employed the procedures described by Jacobson and Truax (1991) and Trull, Nietzel, and Main (1988) for comparing the posttest scores of treated patients to scores of nonpatients. This requires using a particular symptom measure that is (a) widely employed in treatment outcome research and (b) studied in nonpatient samples. We found that the Y-BOCS met the first criteria nicely: 78% of the studies under review included this measure. However, no normative data on the Y-BOCS has been reported for pediatric populations. Therefore, given that the Y-BOCS is routinely adapted for children in clinical settings (e.g., Franklin et al., 1998) and contains identical items when administered with children (i.e., C/YBOCS), we performed analyses of clinical significance based on adult norms for the Y-BOCS published by Steketee, Frost, and Bogert (1996; $M = 7.2$, $SD = 4.5$).

Table 4 presents the weighted mean pre- and posttest Y-BOCS scores for patients receiving ERP, SRIs, and placebo. As can be seen, pretest OCD severity was at the high end of the moderate range and low end of the severe range; the variability in scores across treatment types was not significant, $F(2, 18) = 0.32$, $p = .73$. Following treatment, patients receiving SRIs were functioning in the lower end of the moderate range, whereas those treated with ERP were functioning in the mild range of symptoms. Patients receiving a placebo remained within the moderate range. Significant differences in posttest Y-BOCS scores among the three treatments were observed even when controlling for pretest scores, $F(2, 18) = 15.62$, $p = .001$. Post-hoc tests revealed that the ERP groups had significantly lower Y-BOCS scores than did the SRI groups, which had significantly lower scores than did the placebo groups ($ps < .01$).

TABLE 4 Pre- and Posttest Yale-Brown Obsessive Compulsive Scale (Y-BOCS) Scores for Pediatric OCD Patients and Number of Studies in Which Patients Met Criteria for a Clinically Significant Response by Treatment Type

	Y-BOCS[a]		Clinical Significance
Treatment	*M*	SD	*n* (%)
Serotonin reuptake inhibitors ($n = 8$)			3 (38)
Pretest	24.5	2.3	
Posttest	17.1	2.0	
Exposure and response prevention ($n = 8$)			5 (63)
Pretest	23.9	1.7	
Posttest	11.1	3.3	
Placebo ($n = 6$)			0 (0)
Pretest	23.7	2.2	
Posttest	20.4	2.7	

[a] Y-BOCS scores from 0 to 7 indicate subclinical OCD; from 8 to 15 indicate mild OCD; from 16 to 23 indicate moderate OCD; from 24 to 31 indicate severe OCD; and from 32 to 40 indicate extremely severe OCD (Goodman et al., 1989). Mean scores computed by weighting individual observations by the inverse of the sampling variance estimate.

Jacobson and Truax (1991) proposed that improvement is clinically significant if posttest functioning falls statistically within the normative (as opposed to the dysfunctional) population distribution. To address this issue, we computed the cut score (Jacobson & Truax, 1991, p. 13) for each individual treatment group, below which a mean Y-BOCS score would be considered more likely to be drawn from the normative population. As suggested by Trull et al. (1988), we examined whether the change observed in each group was clinically significant and reliable. The right two columns of Table 4 show the number of SRI, ERP, and placebo groups that attained posttest mean Y-BOCS scores below the cut score for their group (i.e., clinically significant improvement) reliable change. Fisher's exact test indicated that more ERP groups attained clinically significant improvement compared to the number of placebo groups ($p = .02$), but not SRI groups ($p = .10$). The frequency of SRI and placebo groups that achieved clinically significant improvement did not differ ($p = .27$). An equal number of SRI and ERP groups underwent reliable change, and this was more frequent than among placebo groups (ps = .01).

Discussion

The present study represents the first meta-analysis of the pediatric OCD treatment literature to date. In accord with previous summary reviews, our analyses indicate that SRI medication and ERP are effective in reducing pediatric OCD symptoms. Some of our findings suggest that ERP is superior to SRI medication, yet others indicate no differences in the effectiveness of these treatments. Specifically, ERP was associated with larger effect sizes on OCD measures and fewer residual symptoms compared to SRIs. However, there were no differences between these treatments on measures of anxiety and depression, or in the rates of clinically significant and reliable change. Thus, our meta-analytic findings generally support the clinical recommendations of the OCD Expert Consensus Guidelines (March, Francis, et al., 1997) that ERP is the first-line treatment approach for children and adolescents with this disorder.

In the majority of studies of ERP, patients on average evidenced clinically significant and reliable improvement in OCD symptoms: on average, patients' posttest Y-BOCS scores were within the mild range of symptom severity. It is noteworthy that youngsters who complete a trial of ERP on average experience mild residual symptoms at posttest; thus, ERP does not cure OCD. However, given that obsessions and compulsions occur normally in the general population (Gibbs, 1996), it may be unrealistic to expect that treatment could completely ameliorate these symptoms. Given the variability in outcome, future studies should examine possible predictors of response such as insight, family involvement in treatment, and treatment expectancy. Although these and other variables were coded from studies in the present review, there was insufficient power to conduct meaningful analyses of their relationship to effect size.

The existing literature suggests that serotonergic medication also reduces pediatric OCD symptoms. However, at posttest, the average child treated with SRIs still evidenced moderate symptom severity on the Y-BOCS. Moreover, the degree of residual symptoms with SRIs was sufficient to meet OCD severity criteria for entry into most medication trials (Y-BOCS > 16). Thus, SRIs represent a viable treatment strategy when ERP is unavailable or in cases of ERP refusal or failure. One drawback of pharmacotherapy is that symptoms typically return when medication is discontinued (e.g., Thomsen, 2000); however, the lack of follow-up results in pediatric OCD medication trials prevents definitive conclusions about long-term effects. Our meta-analytic data suggest that clomipramine is not more effective than other SRI medications. However, to date, no direct comparisons between clomipramine and more selective SRIs have been conducted and, thus, final conclusions await further research.

Although our findings suggest that treatment for pediatric OCD is effective, the existing literature contains mainly uncontrolled trials. Such studies, while informative, do not control for the effects of time or other nonspecific factors that might account for improvement. The medication literature is far ahead of psychotherapy research in this area: There are seven placebo-controlled medication trials and one controlled ERP study. Comparisons between ERP and credible control conditions (e.g., anxiety management training) are necessary to determine convincingly the extent to which ERP procedures are "active ingredients" in therapy over and above aspects of the structure or process of treatment ("common factors") that are present in most therapies.

The dearth of controlled studies is also relevant to our meta-analytic procedure in that in order to obtain a sufficient number of observations for meaningful analyses it was necessary to compute effect sizes that merely standardize the pre- to posttest improvement on a group-by-group basis. Because such "uncontrolled" effect sizes do not partial out the effects of nonspecific factors (e.g., time), they may overestimate therapeutic effects. Difficulty also arises in comparing mean effect sizes

across different treatments because the amount of change attributable to nonspecific factors may vary systematically with the type of treatment. For example, children with a good prognosis may be selectively referred for particular interventions. To more rigorously assess the relative efficacy of these treatments, it is necessary to directly compare them within a single study. Only two such studies exist, and their results are equivocal.

A merit of using uncontrolled effect sizes in meta-analytic research is that response in control (placebo) groups can be gauged (Morris & DeShon, 2002). The need for additional controlled trials is underscored by our meta-analytic finding of a small to moderate placebo response in existing studies. This suggests that nonspecific factors indeed contribute to the effects of pharmacotherapy for pediatric OCD. Our finding of a placebo response in pediatric OCD parallels that found in a recent meta-analysis of medication studies in adult OCD (Ackerman & Greenland, 2002). Such nonspecific factors likely also contribute somewhat to the effects of ERP also, yet this could not be assessed because of the lack of controlled psychotherapy studies.

Although we did not conduct statistical comparisons, the effect sizes derived from measures of OCD generally appeared larger than those derived from measures of general anxiety and depression. Examination of the studies under review revealed that almost all measures of OCD symptoms were interviewer-rated, whereas most measures of general anxiety and depression were self-report instruments. Given this confound, it is difficult to know whether the smaller effect sizes for anxiety and depression occurred because these symptoms are not the specified targets of treatment, or because of differences in research methodology. Future studies should employ a multitrait, multimethod approach to assessing treatment response.

Overall, our results are encouraging and suggest that two forms of treatment, ERP and medication with SRIs, are effective in reducing obsessions and compulsions in pediatric samples. As noted above, SRI pharmacotherapy is the most widely available, and hence the most widely employed, treatment for OCD. Indeed, most OCD patients, before they obtain ERP, have already undergone trials of SRIs. Therefore, future studies should consider this reality in order to maximize generalizability of results to typical clinical service settings. For example, as opposed to studying the effects of ERP, SRIs, or combined therapy begun simultaneously, it would be useful to know the relative efficacy of different methods of starting and sequencing these two treatments. Is it best to start medication first to promote compliance with ERP? Could medication subsequently be phased out as the effects of ERP begin? Future studies should also address whether ERP can be used to help youngsters with OCD discontinue costly medications without relapse.

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Appendix

Pediatric OCD Treatment Studies Reviewed in the Meta-Analysis

Study	Treatment	Group *n*	Weeks of Treatment	SVE	Effect Size: Anxiety/ Depression	Effect Size: OCD Symptoms[a]
Bolton et al. (1983)	ERP	68	15	0.34		1.97 (2.02)
Flament et al. (1985)	Clomipramine	9	5	0.67	0.27	0.89
	Placebo	10	5	0.58	−0.03	0.25
Leonard et al. (1989)	Clomipramine	25	5	0.19	0.62	0.73
DeVeaugh-Geiss et al. (1992)	Clomipramine	31	8	0.15		1.57
	Placebo	29	8	0.16		0.30
Riddle et al. (1992)	Fluoxetine	7	8	1.00	0.41	1.31
	Placebo	7	8	1.00	0.29	0.51
March et al. (1994)	ERP	15	32	0.34		1.68 (1.68)
Franklin et al. (1998)	ERP	14	10	0.37	0.20	1.71 (1.64)
Scahill et al. (1996)	ERP	7	15	1.00		1.92
Wever & Ray (1997)	ERP	57	4	0.08		2.60 (2.68)
	Various SRIs	12	24	0.45		1.05 (1.45)
March et al. (1998)	Sertraline	92	12	0.05		0.98
	Placebo	95	12	0.04		0.53
de Haan et al. (1998)	ERP	12	12	0.47	0.36	0.99
	Clomipramine	10	12	0.58	0.66	0.54
Rosenberg et al. (1999)	Paroxetine	20	12	0.24	0.72	1.58
Riddle et al. (2001)	Fluvoxamine	57	10	0.08	−0.08	0.82
	Placebo	63	10	0.07	0.06	0.50
Thienemann et al. (2001)	ERP	18	14	0.30	0.36	0.95
Piacentini et al. (2002)	ERP	42	13	0.11		1.72
Benazon et al. (2002)	ERP	16	12	0.31	0.92	2.44
Liebowitz et al. (2002)	Fluoxetine	21	16	0.23	0.67	1.13
	Placebo	22	16	0.21	0.07	0.49
POTS (2004)	ERP	28	12	0.16		2.61
	Sertraline	28	12	0.16		1.49
	Placebo	28	12	0.16		0.74

Note. SVE = sampling variance estimate; SRI = serotonin reuptake inhibitor medication; ERP = exposure and response prevention; POTS = Pediatric OCD Treatment Study Team.

[a] Numbers in parentheses represent follow-up effect sizes.