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COGNITIVE BEHAVIORAL TREATMENT FOR YOUNG CHILDREN WITH OBSESSIVE COMPULSIVE DISORDER

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Abstract

Obsessive compulsive disorder (OCD) is a distressing and functionally impairing disorder that can emerge as early as age 4. Cognitive behavior therapy (CBT) for OCD in youth shows great promise for amelioration of symptoms and associated functional impairment. However, the empirical evidence base for the efficacy of CBT in youth has some significant limitations, particularly as related to treating the very young child with OCD. This paper includes a quantitative review of existing child CBT studies to evaluate evidence for the efficacy of CBT for OCD. It identifies gaps in the literature that when addressed would enhance the understanding of effective treatment in pediatric OCD. Finally, it presents a proposed research agenda for addressing the unique concerns of the young child with OCD.

Keywords

pediatric OCD; cognitive behavioral therapy; early childhood

Cognitive Behavioral Treatment for Young Children with Obsessive Compulsive Disorder

Obsessive Compulsive Disorder (OCD) is a serious and significant psychiatric disorder in childhood, affecting as many as 2–3% of children (e.g., Mullick & Goodman, 2005;Valleni-Basile et al., 1995). Point prevalence estimates indicate that, at any given moment, between 0.5 and 1% of the pediatric population suffers from OCD (Flament et al., 1988). For many children, the disorder severely impairs academic, social and family functioning (Flament et al., 1990;Leonard et al., 1993;Piacentini et al., 2003;Swedo et al., 1989). In addition, the vast majority of children with OCD also develop additional psychiatric disorders (e.g., 75–84% comorbidity reported by Geller et al., 1996).

OCD that emerges in early childhood (between ages 5–8) can be especially pernicious in its impact, disrupting functioning across many domains, and compounding its negative impact over time to derail normal development (Valderhaug & Ivarsson, 2005). In addition, an earlier

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age of onset and a longer duration of illness have both been associated with increased persistence of OCD symptoms (Stewart et al., 2004). Given that early childhood coincides with the beginning of formal schooling, OCD-related difficulties during this period may have a particularly devastating effect upon the establishment of strong academic functioning and peer relationships. Early, frequent intervention is warranted to enable the child to develop coping skills to minimize the chance that the child's anxiety will interfere with learning (Hirshfeld-Becker & Biederman, 2002). However, the current pediatric OCD treatment literature has not focused on treatment of this high-risk age group.

In considering this gap in the literature, one must take into account the type of treatment that would be appropriate for young children. The Expert Consensus Guidelines (March et al, 1997) and the AACAP Practice Parameters (King, Leonard, & March, 1998) for OCD both recommend starting treatment in children with cognitive behavior therapy (CBT), or CBT plus an SRI medication, depending on severity and comorbidity. Although many SRI treatment studies have demonstrated improvement in OCD symptoms in children and adolescents (Abramowitz, Whiteside, and Deacon, 2005), the FDA has approved few medications for children under the age of 8. The precise rates of adverse drug reactions and their relationship to treatment duration and moderator variables, such as age and gender, are poorly understood. For these reasons, it seems likely that a psychosocial treatment, rather than a medication or combined medication and psychotherapy treatment, would be the treatment of choice in younger child populations and therefore this paper will not review SRI-alone treatment studies.

The purpose of this paper is to present a quantitative review of the existing evidence of CBT efficacy in youth with OCD, with particular focus on how this literature might inform the clinician with a very young OCD patient. First, we identify gaps in the burgeoning literature that when addressed will secure a better understanding of how to treat the young OCD patient. Then, in the context of the theoretical and empirical supports reviewed, a research agenda directly tailored for very young children with OCD is proposed.

CBT for children and adolescents with OCD: Review of Findings

Research on CBT, and in particular, exposure with response prevention, has only recently been carefully reviewed and studied in children and adolescents (March, 1995;Piacentini, 1999). To identify CBT studies for the quantitative review, searches of major research literature databases relevant to OCD (i.e., Medline, PsychLit) were conducted using the keywords: [OCD or obsessive compulsive disorder] AND [treatment, CBT, family CBT, intervention, or trial]. Studies were limited to clients between 5 and 17 of age. Empirical papers and major OCD treatment reviews (e.g., Abramowitz et al., 2005) that were collected using these procedures were examined for other studies that had not been identified in the online literature search. Experts in OCD were also surveyed to ensure no studies, published or unpublished, were excluded. Studies identified for the review are listed in Table 1.

Qualitative Review

Upon inspecting the studies listed in Table 1, three major gaps with particular relevance for young child OCD are evident: age of participants, the role of medication in the context of CBT, and the use of family-based models of treatment of OCD. With regard to age, no treatment studies have examined treatment of children with OCD who are younger than 7 years old. As seen in Table 1, research has focused on treatment of children between the ages of 7 and 18, with the average age participants being 13. Although they provide a reasonable starting place, findings from studies of CBT for older children and adolescents may not generalize to young children due to differences in developmental level, which can impact symptom expression and ability to benefit from CBT interventions. Due to their level of cognitive development, young children with OCD, unlike older children or adults, may not understand or be able to identify

the connection between obsessional thoughts and subsequent compulsions, or to verbally express this pattern to others. Therefore, obsessional thoughts may be a less prominent feature in the symptom picture of young children with OCD.

A second unresolved question in the literature on the treatment of pediatric OCD involves the combination of psychotherapy and medication in treatment. As noted in Table 1, only 4 CBT outcome studies examine the efficacy of CBT without concomitant medication treatment. The large majority of CBT studies included some percentage of patients receiving concurrent stable SRI treatment for OCD. This gap is particularly salient for young child OCD, as young children are less likely to be prescribed medications concurrently to their treatment.

As shown in the Table 1, studies examining a family-based treatment approach to pediatric OCD are few, and none have included young children. This situation is particularly problematic for making empirically-informed decisions about the treatment of young children with OCD because it is likely that a family component would be needed for this young age group. Early-onset OCD is influenced by a familial history of OCD (Hanna et al, 2005). Families affect and are affected by OCD through their accommodation of, and participation in, rituals and avoidance behaviors (Lenane, 1989,1991;Pollack & Carter, 1999;Steketee, 1997). In addition, other family behavior patterns are likely to affect OCD symptoms in children. Parents of children with OCD show poor problem-solving skills, decreased confidence in the affected child, increased levels of expressed emotion (criticism and emotional overinvolvement) and increases in parental catastrophizing behavior (Barrett et al., 2002;Leonard et al., 1993;Moore et al., 2004). Higher familial dysfunction has also been found to be a predictor of poorer long-term treatment outcome (Barrett et al., 2005). These findings are particularly relevant to young child OCD, as young children may be particularly vulnerable to family influences.

Those studies that have tested family-based CBT approaches in older children and adolescents have found promising results. Barrett, Healy-Farrell, and March (2004) published a randomized control trial comparing family-based individual CBT for OCD to family-based group CBT to waitlist controls. They found statistically and clinically significant reductions in OCD symptoms in both family-based treatment conditions, which were maintained at 18-month follow-up (Barrett et al, 2005). Two open treatment studies that examined family-based treatments also observed significant reductions in OCD symptoms (Martin & Thienemann, 2005;Waters, Barrett, & March, 2001).

In spite of these gaps in the extant literature, we move next to a quantitative review of this literature as a means of evaluating the strength of the foundation upon which treatments for young children with OCD might be developed.

Quantitative Review

To be included in the quantitative review certain general study criteria were required. All studies were required to be written or have a translation available in English. Both published (e.g., peer and non-peer reviewed journals) and non-published (e.g., theses, conference presentations) studies could be included in the review. In the event of multiple studies reporting on the same sample and dependent variables, the one with the largest sample size was included.

All studies were required to have a minimum of 10 subjects per cell and enough information available to complete a within-group CBT effect size. In cases where a trial had both CBT and non-CBT treatment arms, only the CBT treatment arms were coded. Since most of the trials in pediatric OCD involved CBT-only formats, this was the focus for this review, and treatment arms where the experimenter assigned combined treatment (i.e., CBT and medication) were not coded because our statistical method does not allow for inclusion of more than one cell per

study. For these reasons, some studies identified in Table 1 were not included in the effect size calculations.[1] Studies included in the effect size calculations are listed in Table 2.

Effect size calculation

Effect sizes were calculated within each one-sample CBT treatment group. Similar to the two-sample Cohen's *d*, the effect size is an estimate of the true difference between pretreatment and posttreatment outcome divided by the standard deviation (Rosenthal, 1994). When possible, calculation of the effect size was done directly using means, standard deviations, and sample size of the treatment group. When this was not possible, the effect size was coded from data that best approximated direct computation of means and standard deviations, according to Lipsey and Wilson (2001). In order of priority, effect sizes were calculated by algebraically equivalent formulas (i.e., t-test or F-test ratios), probability value for t- or F-tests (i.e., "$p<.05$" was coded as exactly .05), mean difference scores, or approximations based on dichotomous data (e.g., percent diagnosed as depressed). Random effects models were chosen over fixed effects models for this analysis of effect sizes. Because fixed effects models have larger Type I error rates and are less generalizable than random effects models, random effects models have been recommended as the preferred strategy (Mosteller & Colditz, 1996).

Effect Size Adjustments

Before formal analyses were conducted, each effect size was adjusted for small sample bias. Standardized mean difference effect size types have a consistent upward bias when based on small samples (Rosenthal, 1994). Hunter and Schmidt (1994) adjustments were not implemented, because too few studies provided enough information to code sample-specific reliability and range restriction estimates. In cases where only few studies have the necessary information, some have argued that leaving them all unadjusted make them more comparable (Lipsey & Wilson, 2001).

General Effects Analyses

Effect sizes were combined as described previously to generate one effect size per study. The studies included with their respective effect sizes are provided in Table 2. One study (i.e., Barrett et al 2004) included more than one CBT treatment arm with no statistics available describing the overall CBT effect. For this reason, the effect size and representative N for this study was averaged in order for one effect size to represent each study. Inspection of the studies indicates that all studies were effective in reducing OCD symptoms, and randomized control trials generally had larger effects than open trials. All three RCTs reviewed were in the top 25% by effect size. This finding may be attributed to the ability of RCTs to control for confounding factors that may attenuate true treatment effects. In addition effect sizes obtained from RCT are likely more reliable than those obtained in less rigorously controlled designs (Heinsman & Shadish, 1996;Shadish & Ragsdale, 1996).

The general effect of CBT on OCD symptoms was determined by generating random effects models using method of moments. Establishing our null model (i.e., no moderators), the overall effect size including all 12 studies was 1.55 (95% CI 1.12–1.97), indicating a large effect. Due to the possibility of undiscovered studies biasing the estimate of treatment effect upward, Rosenthal's file-drawer method was used to determine the number of unpublished studies it would take with an average effect size of zero for the overall z score to no longer be significant (Rosenthal, 1979). A fail-safe number of $5k + 10$ is considered to provide evidence of a robust effect, where k equals the number of studies included in the meta-analysis (Rosenthal, 1991).

[1]Scahill et al (1996) and Waters et al (2001) had fewer than 10 subjects per cell. Asbahr et al (2005) was excluded as there was not enough posttreatment information available.

A robust effect with the number of studies included is 70. Based on our findings, the number of null studies it would take to lower the overall z score to below 1.96 is 499 studies, which is well over the criteria for being considered a robust effect.

These studies were also broken down based on treatment modality into: individual CBT, group CBT, and family-based CBT, this break down is displayed in Table 3. Studies that included a family component delivered in a group format were coded in the family-based modality. Although the paucity of studies prohibits us from comparing these sufficiently, individual and family-based CBT trials appear to be the most promising.

Future Directions to Solidify the Empirical Base

Although this review of the existing literature indicates great promise for CBT as a treatment for pediatric OCD, there are notable issues in the literature that could shape future research. As can be noted from the quantitative review, there is a dearth of studies examining CBT's true effect, as opposed to CBT delivered in the context of other treatment, such as medication. Unfortunately, the majority of studies on which the supportive evidence of CBT is based may be contaminated by medication effects. Because most of the open CBT studies included some percentage of children on medication, it is difficult to determine the effect of medication status on CBT, and effect sizes of CBT are in some ways contaminated by the medication treatment. Only two of the open CBT studies published the mean results of their outcome data according to medication presence or absence. Conversely, allowing concurrent stable SRI treatment increases the applicability of the treatment studies by approximating more closely what some practitioners of CBT in the community may encounter.

More broadly, the role of medication in CBT is important when considering the theoretical basis for CBT versus medication treatment of OCD. CBT models for child/adolescent OCD primarily focus on providing skills to facilitate exposure with response (ritual) prevention (EX/RP; March & Mulle, 1998). The theory behind EX/RP is that, as a patient is exposed to the feared situation, prevention of the response (i.e., the ritual or avoidance behavior) results in anxiety reduction over time. The effectiveness of EX/RP is most often attributed to the concepts of habituation and extinction. Patients gradually learn that their anxious response decreases over time and that, with prolonged exposure to the stimulus, anxiety can be reduced without performing compulsions (Foa & Kozak, 1986;Francis & Gragg, 1996). Foa and Kozak (1986) suggest exposure tasks disintegrate the fear structure into elements of stimulus, response, and meaning, resulting in reduced anxiety. When CBT is conducted in the context of medication treatment, it is possible that medication enhances the ability to access fear structures, "turning down the volume" of anxiety, allowing a child to participate in exposure exercises and attendance habituation. Alternately, it is possible that medication interferes with the ability to adequately access fear structures, impacting long-term maintenance of treatment gains. Studies on the nature of learning support the latter hypothesis in the sense that the context in which the learning takes place while taking medication may be different as compared to the learning context once the medication has been discontinued (see Smits, O'Cleirigh, & Otto, 2006). Generally speaking adult studies on combined treatment for anxiety disorders have not indicated that combined treatment is associated with attenuated acute outcomes, although there is some evidence in panic disorder for an increased risk of relapse following treatment discontinuation for patients who received combined treatment as compared to CBT monotherapy (for a review see Foa, Franklin, & Moser, 2002).

As noted in the Table 1, only one RCT has systematically examined the relative efficacy of medication, CBT, and their combination. In their study, the Pediatric OCD Treatment Study (POTS) team found that, while combined treatment proved superior to CBT alone or medication alone, the remission rate (defined as a CY-BOCS score of 10 or below) for

combined treatment did not differ from that of CBT alone, but did differ from medication alone and from placebo (POTS, 2004). Further research is required to clarify what role, if any, medication may play in the context of CBT learning. Follow-up studies of treatment gains are particularly relevant. For example, adult OCD studies of CBT combined with medication have found little to no benefit over follow-up of combined treatment to CBT alone (see Steketee & Barlow, 2002, for a review).

While the literature on the treatment of pediatric OCD is clearly expanding, only 4 of the published CBT intervention studies for pediatric OCD were RCTs, while the remaining studies were open trials. Those studies that do not have random assignment are more susceptible to threats to internal validity, thus lowering the ability to make causal inferences regarding CBT's effect on treatment outcome. For example, selection bias, or pre-existing differences among groups, is one such threat to internal validity that is best controlled by random assignment (Larzelere, Kuhn & Johnson, 2004). Similarly, uncontrolled trials do not control for variables such as length of treatment or nonspecific factors such as the positive benefits associated with engaging in treatment that may account for observed treatment effects. In addition, the effect size calculations from uncontrolled trials are different from controlled trials, limiting the ability to make inferences regarding treatment effects across studies (Heinsman & Shadish, 1996).

Despite the increase of CBT treatment studies for OCD, few studies report analyses of mediators or moderators of treatment change, thus limiting the understanding of how and for whom CBT works with pediatric OCD. Studies that have attempted to identify moderators of treatment have been limited by inadequate power. For example, Piacentini et al. (2002) found that baseline severity of obsessions and OCD-related academic difficulties were associated with poorer treatment outcome in their relatively small treatment sample of 42 children. Interestingly, the researchers did not find that age, gender, medication status, or comorbid symptomatology impacted treatment outcome (Piacentini et al., 2002). In the Pediatric OCD Treatment Study (POTS, 2004) comparing the relative efficacy of CBT and sertraline monotherapies to their combination, comorbid tic disorder was associated with poorer outcome in the sertraline monotherapy but was not associated with outcome in either CBT containing condition (March et al, this issue).

The adult OCD literature has identified several additional possible moderators that could inform research in pediatric OCD. However, results have often been equivocal or contradictory regarding depression (Abramowitz et al, 2000;Mataix-Cols et al, 2002), specific personality traits (Steketee, Chambless, & Tran, 2001), motivation (deHaan et al, 1997), symptom severity (De Araujo, Ito, & Marks, 1996; Mataix-Cols et al, 2002), insight and overvalued ideation (Foa et al, 1999;Neziroglu et al, 2001), and expressed emotion in the family (Chambless & Steketee, 1999). Although these variables may be useful in identifying future directions in understanding moderators of treatment in pediatric OCD, at this point, we do not know which characteristics relate to CBT outcome.

Similarly, pediatric OCD studies suffer from a lack of mediator analyses to determine the active therapeutic ingredient. Although the adult literature has some evidence that treatment compliance may be a mediator of treatment outcome (Abramowitz et al, 2002), many factors could account for the treatment's efficacy. Psychoeducation, cognitive training, exposure, or other nonspecific or unidentified therapeutic ingredients are all potential causes of therapeutic effect. Without identifying the active components of treatment, time and effort may be spent unnecessarily in areas that are not actively contributing treatment, diluting or limiting the optimal efficacy of CBT (Kazdin & Nock, 2003). Overall, we do not know which aspects of which treatment works for which child under what conditions. This gap in knowledge of treatment mechanisms is not limited to pediatric OCD, but is especially relevant to this review (Kazdin and Nock, 2003).

Summary and Conclusions

This paper has presented a critical review of the extant evidence of CBT efficacy in youth with OCD, with a specific focus on how this literature might inform the clinician with a very young OCD patient. A meta-analysis of the existing literature indicates great promise for CBT as a treatment for children and adolescents with OCD, with preliminary evidence suggesting that individual and family-based CBT trials appear to be the most promising.

There are specific gaps in the current research that must be addressed in order to generalize the knowledge base to the population of very young children with OCD. Family treatment, which is particularly important for young children with OCD, needs more research. In addition, most of the current studies are confounded by concomitant medications, making the findings regarding CBT less applicable to young children. Third, no controlled treatment research has been published with this young age group and little research has examined age as a moderator of treatment outcome. Developmentally appropriate treatments tailored to young children's specific concerns are needed.

Future research to address these gaps in the literature is recommended. For example, it is recommended that outcome studies report treatment results that are divided into results for those children who were taking a stable dose of medication and those children who only received CBT. Presentation of results in this manner would help to clarify the role of combined medication and psychotherapy in the treatment of OCD. Additionally, although preliminary evidence suggests that a family-based, developmentally tailored CBT model is acceptable to families of young children with OCD (Freeman et al., 2003), more research is needed to test the efficacy of this treatment model. A randomized controlled trial (RCT) of family-based CBT for young children (ages 5–8) with OCD is needed in which the effect of CBT on symptom reduction, functional impairment, and quality of life is evaluated. Additionally, research evaluating potential moderators, such as baseline characteristics of the child/family, comorbidity, parent psychopathology, and family functioning, and mediators of treatment response, such as compliance and family accommodation.

In addition, more knowledge is needed in the field of early childhood OCD. For example, the developmental variability in this age range is not well understood. While there is both clinical and research evidence that some children as young as five can participate in cognitive tasks, including cognitive training (Grave & Blissett, 2004), the factors that would identify these children from other young children who would have difficulty understanding the treatment model are not well understood. These factors would likely include cognitive, developmental, and environmental factors (including exposure to formal schooling and family interaction style). Further research delineating these factors would be helpful in determining the extent to which the therapist can expect children to understand the treatmentmodel versus relying on parent training and implementation of behavioral principles consistent with the model.

Finally, when working with young children with OCD, it is possible that an early intervention model may be required, rather than a treatment model. Future research could address the treatment of sub-syndromal children and application of these CBT principles to nonclinical OCD symptoms in a model of secondary prevention of the development of OCD. As earlier age of onset and longer duration of OCD symptoms predict higher rates of persistence of full-blown OCD at follow-up (Stewart et al., 2004), early and frequent intervention is needed. Intervening early with young children offers a unique opportunity to prevent the development of long-standing problematic behaviors. Such intervention increases the probability of keeping OCD-affected youngsters on track with developmental milestones and thus may offer economic benefits of increased productivity, along with enhanced life quality, into adolescence and adulthood.

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Table 1

Table of CBT Studies for Pediatric OCD

Individual CBT

[a] Piacentini et al (2002) OPEN

Age (avg.)	*n*	% on meds
5–17 (11.8)	42	52%

[a] Franklin et al (1998) OPEN

Age (avg.)	*n*	% on meds
10–17 (14.1)	14	57%

[a] Scahill et al. (1996) OPEN

Age (avg.)	*n*	% on meds
10–15 (13)	7	71%

[a] March, Mulle & Herbel (1994) OPEN

Age (avg.)	*n*	% on meds
8–18 (14.3)	15	93%

[a] Bolton et al (1983) OPEN

Age (avg.)	*n*	% on meds
12–18 (14.1)	15	33%

Group CBT

[b] Asbahr et al (2005) RCT

Age (avg.)	*n*	% on meds
9–17 (13.7)	20	0%

[b] Barrett, Healy-Farrell, & March (2004) RCT

Age (avg.)	*n*	% on meds
7–17 (12.9)	29	31%

[b] Martin & Thienemann (2005) OPEN

Age (avg.)	*n*	% on meds
8–14 (11.3)	14	64%

Himle et al (2003) OPEN

Age (avg.)	*n*	% on meds
12.17 (14.6)	19	68.4 %

[a] Thienemann et al (2001) OPEN

Age (avg.)	*n*	% on meds
13–17 (15.2)	18	83%

Fischer, Himle, & Hanna (1998) OPEN

Age (avg.)	*n*	% on meds
12–17 (14.5)	15	67%

Family CBT

[b] Barrett, Healy-Farrell, & March (2004) RCT

Age (avg.)	*n*	% on meds
7–17 (11.8)	53	23%

[b] Martin & Thienemann (2005) OPEN

Age (avg.)	*n*	% on meds
8–14 (11.3)	14	64%

Waters, Barrett, & March (2001) OPEN

Age (avg.)	*n*	% on meds
10–14	7	N/A

CBT Efficacy

[b] Asbahr et al (2005) RCT

Age (avg.)	*n*	% on meds
9–17 (13.7)	20	0%

[a] POTS (2004) RCT

Age (avg.)	*n*	% on meds
7–17 (11.4)	28	0%

[a] de Haan et al (1998) RCT

Age (avg.)	*n*	% on meds
8–18 (13.3)	12	0%

[a] Benazon et al (2002) OPEN

Age (avg.)	*n*	% on meds
8–17 (N/A)	16	0%

Combinat

POTS

Age (avg.)
7–17 (11.7)

[a] Wever &

Age (avg.)
7–19 (13.7)

[a] Indicates cell was included CBT effect size calculations in Abramowitz et al. (2005)

[b] Indicates cell subjects included in multiple columns (e.g., family CBT in group format)

Shaded cell indicates study reported results split into CBT with medication versus CBT without medication.

N/A indicates study did not report the information.

Note: Grunes, Neziroglu, & McKay (2001) included adults also (average age was 28), so was excluded from this review of CBT for pediatric OCD.

Table 2

Description and overall effect of CBT trials included in the meta-analysis

Study	CBT Modality	Primary Outcome Measure	N	Effect Size
Barrett et al (2004)	RCT Family CBT	CY-BOCS	(26)	2.76
POTS (2004)	RCT Individual CBT	CY-BOCS	28	2.53
Bolton (1983)	Open Individual CBT	Severity Rating	15	2.45
de Haan et al (1998)	RCT Individual CBT	CY-BOCS	12	1.94
Piacentini et al (2002)	Open Individual CBT	NIMHOCS	42	1.72
Benazon et al (2002)	Open Individual CBT	CY-BOCS	16	1.58
Franklin et al (1998)	Open Individual CBT	CY-BOCS	14	1.13
Thienemann et al (2001)	Open Group CBT	CY-BOCS	18	1.09
Martin & Thienemann (2005)	Open Family CBT	CY-BOCS	14	0.98
March et al (1994)	Open Individual CBT	CY-BOCS	15	0.95
Himle et al (2003)	Open Group CBT	CY-BOCS	19	0.78
Fischer et al (1998)	Open Group CBT	CY-BOCS	15	0.54

Note: Due to two family CBT arms in Barrett et al, a weighted average of effect size and N were used in computation.

Table 3

Effects of CBT treatment on pediatric OCD both overall and broken down by modality

Study Type	Studies (Participants)	Mean effect size (95% CI)
CBT (ALL)	12 (231)	1.55 (1.12–1.97)
Individual CBT	7 (142)	1.77 (1.33 – 2.21)
Group CBT	3 (49)	0.76 (.34–1.17)
Family CBT	2 (40)	1.88 (0.15–3.63)