# Cognitive Behavioral Treatment for Young Children With Obsessive-Compulsive Disorder

Jennifer B. Freeman, Molly L. Choate-Summers, Phoebe S. Moore, Abbe M. Garcia, Jeffrey J. Sapyta, Henrietta L. Leonard, and Martin E. Franklin

*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 report 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.*

**Key Words:** Cognitive behavioral therapy, early childhood, pediatric OCD

Obsessive-Compulsive Disorder (OCD) is a serious and significant psychiatric disorder in childhood, affecting as many as 2%–3% of children (e.g., Mullick and Goodman 2005; Valleni-Basile et al 1995). Point prevalence estimates indicate that, at any given moment, between .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 and 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 and Ivarsson 2005). In addition, an earlier 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 might 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 and 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 American Academy of Child & Adolescent Psychiatry Practice Parameters (King et al 1998) for OCD both recommend starting treatment in children with cognitive behavior therapy (CBT) or CBT plus a serotonin reuptake inhibitor (SRI) medication, depending on severity and comorbidity. Although many SRI treatment studies have demonstrated improvement in OCD symptoms in children and adolescents (Abramowitz et al 2005), the Food and Drug Administration 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 report will not review SRI-alone treatment studies.

The purpose of this article 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 with the key words: [OCD or obsessive compulsive disorder] AND [treatment, CBT, family CBT, intervention, or trial]. Studies were limited to clients between 5 and 17 years of age. Empirical articles and major OCD treatment reviews (e.g., Abramowitz et al 2005) that were collected with 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 that 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

From the Bradley/Hasbro Research Center (JBF, MLC-S, AMG, HLL), Brown Medical School, Providence, Rhode Island; Duke University Medical Center (PSM, JJS), Durham, North Carolina; and the University of Pennsylvania School of Medicine (MEF), Philadelphia, Pennsylvania.

Address reprint requests to Jennifer B. Freeman, Ph.D., Pediatric Anxiety Research Clinic, Bradley/Hasbro Research Center, 593 Eddy St. Coro West 2nd Floor, Providence, RI 02903; E-mail: jfreeman@lifespan.org.

0006-3223/07/$32.00
doi:10.1016/j.biopsych.2006.12.015

**Table 1.** Table of CBT Studies for Pediatric OCD

| Individual CBT | | | Group CBT | | | Family CBT | | | CBT Efficacy | | | Combination CBT & Meds | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| [a]Piacentini et al (2002) OPEN | | | [b]Asbahr et al (2005) RCT | | | [b]Barrett et al (2004) RCT | | | [b]Asbahr et al (2005) RCT | | | POTS (2004) RCT | | |
| Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds |
| 5–17 (11.8) | 42 | 52% | 9–17 (13.7) | 20 | 0% | 7–17 (11.8) | 53 | 23% | 9–17 (13.7) | 20 | 0% | 7–17 (11.7) | 28 | 0 |
| [a]Franklin et al (1998) OPEN | | | [b]Barrett et al (2004) RCT | | | [b]Martin & Thienemann (2005) OPEN | | | [a]POTS (2004) RCT | | | [a]Wever & Rey (1997) OPEN | | |
| Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds |
| 10–17 (14.1) | 14 | 57% | 7–17 (12.9) | 29 | 31% | 8–14 (11.3) | 14 | 64% | 7–17 (11.4) | 28 | 0% | 7–19 (13.7) | 57 | 100% |
| [a]Scahill et al. (1996) OPEN | | | [b]Martin & Thienemann (2005) OPEN | | | Waters et al (2001) OPEN | | | [a]de Haan et al (1998) RCT | | | | | |
| Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | | | |
| 10–15 (13) | 7 | 71% | 8–14 (11.3) | 14 | 64% | 10–14 | 7 | NA | 8–18 (13.3) | 12 | 0% | | | |
| [a]March, Mulle & Herbel (1994) OPEN | | | Himle et al (2003) OPEN | | | | | | [a]Benazon et al (2002) OPEN | | | | | |
| Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | | | | Age (avg.) | *n* | % on meds | | | |
| 8–18 (14.3) | 15 | 93% | 12.17 (14.6) | 19 | 68.4% | | | | 8–17 (NA) | 16 | 0% | | | |
| [a]Bolton et al (1983) OPEN | | | [a]Thienemann et al (2001) OPEN | | | | | | | | | | | |
| Age (avg.) | *n* | % on meds | Age (avg.) | *n* | % on meds | | | | | | | | | |
| 12–18 (14.1) | 15 | 33% | 13–17 (15.2) | 18 | 83% | | | | | | | | | |
| | | | Fischer et al (1998) OPEN | | | | | | | | | | | |
| | | | Age (avg.) | *n* | % on meds | | | | | | | | | |
| | | | 12–17 (14.5) | 15 | 67% | | | | | | | | | |

Grunes et al (2001) included adults also (average age was 28), so it was excluded from this review of cognitive behavior therapy (CBT) for pediatric obsessive-compulsive disorder (OCD). Piacentini et al, Franklin et al, and Scahill et al studies reported results split into CBT with medication versus CBT without medication. Meds, medication; POTS, Pediatric OCD Treatment Study; RCT, randomized controlled trials.

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

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

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 of participants being 13. Although they provide a reasonable starting place, findings from studies of CBT for older children and adolescents might not generalize to young children, owing to differences in developmental level, which can impact symptom expression and ability to benefit from CBT interventions. Because of their level of cognitive development, young children with OCD, unlike older children or adults, might 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 might 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 four 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, because 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 and 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

**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 | .98 |
| March et al (1994) | Open Individual CBT | CY-BOCS | 15 | .95 |
| Himle et al (2003) | Open Group CBT | CY-BOCS | 19 | .78 |
| Fischer et al (1998) | Open Group CBT | CY-BOCS | 15 | .54 |

Due to two family CBT arms in Barrett et al, a weighted average of effect size and *n* were used in computation. CY-BOCS, Children's Yale-Brown Obsessive Compulsive Scale; other abbreviations as in Table 1.

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, because young children might 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 et al (2004) published a randomized controlled trial comparing family-based individual CBT for OCD with family-based group CBT to waitlist control subjects. 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 and Thienemann 2005; Waters et al 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 in 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/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. Because most of the trials in pediatric OCD involved CBT-only formats, this was the focus for this review. 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/study. For these reasons, some studies identified in Table 1 were not included in the effect size calculations (Scahill et al [1996] and Waters et al [2001] had fewer than 10 subjects/cell. Abash et al [2005] was excluded, because there was not enough posttreatment information available). 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 SD (Rosenthal 1994). When possible, calculation of the effect size was done directly with means, SDs, 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 SDs, 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 and 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 makes them more comparable (Lipsey and Wilson 2001).

### General Effects Analyses

Effect sizes were combined as described previously to generate one effect size/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 controlled trials generally had larger effects than open trials. All three randomized

**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) | .76 (.34–1.17) |
| Family CBT | 2 (40) | 1.88 (.15–3.63) |

CI, confidence interval; other abbreviations as in Table 1.

controlled trials (RCTs) reviewed were in the top 25% by effect size. This finding might be attributed to the ability of RCTs to control for confounding factors that might attenuate true treatment effects. In addition, effect sizes obtained from RCTs are likely more reliable than those obtained in less rigorously controlled designs (Heinsman and Shadish 1996; Shadish and Ragsdale 1996).

The general effect of CBT on OCD symptoms was determined by generating random effects models with method of moments. Establishing our null model (i.e., no moderators), the overall effect size including all 12 studies was 1.55 (95% confidence interval 1.12–1.97), indicating a large effect. Owing 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). A robust effect with the number of studies included is 70. On the basis of 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 on the basis of treatment modality into: individual CBT, group CBT, and family-based CBT. This breakdown 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 seem 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 might be contaminated by medication effects. Because most of the open CBT studies included some percentage of children receiving 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 might encounter.

More broadly, the role of medication in CBT is important when considering the theoretical basis for CBT versus medication treatment of OCD. The CBT models for child/adolescent OCD primarily focus on providing skills to facilitate exposure with response (ritual) prevention (EX/RP; March and 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 and Kozak 1986; Francis and Gragg 1996). Foa and Kozak (1986) suggest that 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. It is possible that, alternatively, 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 might be different as compared with the learning context once the medication has been discontinued (see Smits et al 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 after treatment discontinuation for patients who received combined treatment as compared with CBT monotherapy (for a review see Foa et al 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, although combined treatment proved superior to CBT alone or medication alone, the remission rate (defined as a Children's Yale-Brown Obsessive Compulsive Scale 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 might 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 and Barlow 2002, for a review).

Although the literature on the treatment of pediatric OCD is clearly expanding, only four of the published CBT intervention studies for pediatric OCD were RCTs, whereas 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 et al 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 might 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 and 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 POTS (2004) report, 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 2007).

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 et al 2001), motivation (de Haan et al 1997), symptom severity (de Araujo et al 1995; Mataix-Cols et al 2002), insight and overvalued ideation (Foa et al 1999; Neziroglu et al 2001), and expressed emotion in the family (Chambless and Steketee 1999). Although these variables might 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 might 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 might be spent unnecessarily in areas that are not actively contributing to treatment, diluting or limiting the optimal efficacy of CBT (Kazdin and 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 article 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 seem to be the most promising.

There are specific gaps in the current research that must be addressed 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. Moreover, 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. An 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 is needed 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. Although there is both clinical and research evidence that some children as young as 5 can participate in cognitive tasks, including cognitive training (Grave and 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 treatment model 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 might 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. Because 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 might offer economic benefits of increased productivity, along with enhanced life quality, into adolescence and adulthood.

*This work was supported, in part, by a National Institute of Mental Health grant (R21MH60669) to HLL. We wish to acknowledge the administrative and research assistance by Noah Berman, Alexa Ogata, Chelsea Ale, Janet Ng, and Marsha Spirito.*

Abramowitz JS, Franklin ME, Street GP, Kozak MJ, Foa EB (2000): Effects of comorbid depression on response to treatment for obsessive-compulsive disorder. *Behav Ther* 31:517–528.

Abramowitz JS, Franklin ME, Zoellner LA, DiBernardo CL (2002): Treatment compliance and outcome in obsessive-compulsive disorder. *Behav Mod* 26:447–463.

Abramowitz J, Whiteside S, Deacon B (2005): The effectiveness of treatment for pediatric obsessive-compulsive disorder: A meta-analysis. *Behav Ther* 36:55–63.

Asbahr FR, Castillo AR, Ito LM, Oliveira Latorre MDRD, Moreira MN, Lotufo-Neto F (2005): Group cognitive-behavioral therapy versus sertraline for the treatment of children and adolescents with obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* 44:1128–1136.

Barrett P, Farrell L, Dadds M, Boulter N (2005): Cognitive-behavioral family treatment of childhood obsessive-compulsive disorder: Long-term follow-up and predictors of outcome. *J Am Acad Child Adolesc Psychiatry* 44:1005–1014.

Barrett P, Healy-Farrell L, March JS (2004): Cognitive-behavioral family treatment of childhood obsessive-compulsive disorder: A controlled trial. *J Am Acad Child Adolesc Psychiatry* 43:46–62.

Barrett P, Shortt A, Healy L (2002): Do parent and child behaviors differentiate families whose children have obsessive-compulsive disorder from other clinic and non-clinic families? *J Child Psychol Psychiatry* 43:597–607.

Benazon NR, Ager J, Rosenberg DR (2002): Cognitive behavior therapy in treatment-naïve children and adolescent with obsessive compulsive disorder: An open trial. *Behav Res Therapy* 40:529.

Bolton D, Collins S, Steinberg D (1983): The treatment of obsessive-compulsive disorder in adolescence: A report of fifteen cases. *Br J Psychiatry* 142:456–464.

Chambless D, Steketee G (1999): Expressed emotion and behavior therapy outcome: A prospective study with obsessive-compulsive and agoraphobic outpatients. *J Consult Clin Psychol* 67:658–665.

de Araujo LA, Ito LM, Marks IM, Deale A (1995): Does imagined exposure to the consequences of not ritualizing enhance live exposure for OCD? A controlled study. I. Main outcome. *Br J Psychiatry* 167:65–70.

de Haan E, Hoogduin KAL, Buitelaar JK, Keijsers GPJ (1998): Behavior therapy versus clomipramine for the treatment of obsessive-compulsive disorder in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 37: 1022–1029.

de Haan E, Van Oppen P, Van Balkom AJ, Spinhoven P, Hoogduin KA, Van Dyck R, et al (1997): Prediction of outcome and early vs. late improvement in OCD patients treated with cognitive behaviour therapy and pharmacotherapy. *Acta Psychiatr Scand* 96:354–361.

Fischer DJ, Himle JA, Hanna GL (1998): Group behavioral therapy for adolescents with obsessive-compulsive disorder: Preliminary outcomes. *Res Social Work Practice* 8:629–636.

Flament MF, Koby E, Rapoport J, Berg C, Zahn T, Cox C, et al (1990): Childhood obsessive-compulsive disorder: A prospective follow-up study. *J Child Psychol Psychiatry* 31:363–380.

Flament MF, Whitaker A, Rapoport J, Davies M, Berg C, Kalikow K, et al (1988). Obsessive compulsive disorder in adolescence: An epidemiological study. *J Am Acad Child Adolesc Psychiatry* 27:764–771.

Foa EB, Abramowitz JS, Franklin ME, Kozak MJ (1999): Feared consequences, fixity of belief, and treatment outcome in OCD. *Behav Ther* 30:717–724.

Foa EB, Franklin ME, Moser J (2002): Context in the clinic: How well do CBT and medications work in combination? *Biol Psychiatry* 51:989–997.

Foa EB, Kozak MJ (1986): Emotional processing of fear: Exposure to corrective information. *Psychol Bull* 99:20–35.

Francis G, Gragg R (1996): *Childhood Obsessive Compulsive Disorder*. Thousand Oaks, California: Sage Publications.

Franklin ME, Kozak MJ, Cashman LA, Coles ME, Rheingold AA, Foa EB (1998): Cognitive-behavioral treatment of pediatric obsessive-compulsive disorder: An open clinical trial. *J Am Acad Child Adolesc Psychiatry* 37:412–419.

Freeman J, Garcia A, Fucci C, Karitani M, Miller L, Leonard HL (2003): Family-based treatment of early-onset obsessive-compulsive disorder. *J Child Adolesc Psychopharmacol* 13:S71–S80.

Geller D, Biederman J, Griffin S, Jones J, Lefkowitz T (1996): Comorbidity of juvenile obsessive-compulsive disorder with disruptive behavior disorders. *J Am Acad Child Adolesc Psychiatry* 35:1637–1646.

Grave J, Blissett J (2004): Is cognitive behavior therapy developmentally appropriate for young children? A critical review of the evidence. *Clin Psychol Rev* 24:399–420.

Hanna GL, Fischer D, Chadha K, Himle J, Van Etten M (2005): Familial and sporadic subtypes of early-onset obsessive-compulsive disorder. *Biol Psychiatry* 57:895–900.

Heinsman DT, Shadish WR (1996): Assignment methods in experimentation: When do nonrandomized experiments approximate answers from randomized experiments. *Psychol Methods* 1:154–169.

Himle JA, Fischer DJ, Van Etten ML, Janeck AS, Hanna GL (2003): Group behavioral therapy for adolescents with tic-related and non-tic-related obsessive-compulsive disorder. *Depress Anxiety* 17:73–77.

Hirshfeld-Becker DR, Biederman J (2002): Rationale and principles for early intervention with young children at risk for anxiety disorders. *Clin Child Family Psychol Rev* 5:161–172.

Hunter JE, Schmidt FL (1994): Estimation of sampling error variance in the meta-analysis of correlations: Use of average correlation in the homogenous case. *J Applied Psychol* 79:171–177.

Kazdin AE, Nock MK (2003): Delineating mechanisms of change in child and adolescent therapy: Methodological issues and research recommendations. *J Child Psychol Psychiatry* 44:1116–1129.

King R, Leonard H, March J (1998): Practice parameters for the assessment and treatment of children and adolescents with obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* Special issue: Practice parameters 37:27S–45S.

Larzelere RE, Kuhn BR, Johnson B (2004): The intervention selection bias: An under recognized confound in intervention research. *Psychol Bull* 130: 289–303.

Lenane M (1989): Families and obsessive-compulsive disorder. In: Rapoport JL, editor. *Obsessive-Compulsive Disorder in Children and Adolescents*. Washington, DC: American Psychiatric Association Press, 237–249.

Lenane M (1991): Family therapy for children with obsessive-compulsive disorder. In: Pato MT, Zohar M, editors. *Current Treatments of Obsessive Compulsive Disorder-Clinical Practice, vol. 18*. Washington, DC: American Psychiatric Association Press, 103–113.

Leonard HL, Swedo SE, Lenane MC, Rettew DC, Hamburger SD, Bartko J, Rapoport JL (1993): A 2- to 7-year follow-up study of 54 obsessive compulsive children and adolescents. *Arch Gen Psychiatry* 50:429–439.

Lipsey MW, Wilson DB (2001): *Practical Meta-Analysis*. Thousand Oaks, California: Sage Publications.

March J (1995): Cognitive-behavioral psychotherapy for children and adolescents with OCD: A review and recommendations for treatment. *J Am Acad Child Adolesc Psychiatry* 34:7–18.

March J, Frances A, Kahn D, Carpenter D (1997): The Expert Consensus Guideline Series: treatment of obsessive-compulsive disorder. *J Clin Psychiatry* 58:4–6.

March J, Franklin ME, Leonard HL, Garcia AM, Moore PS, Freeman JB, Foa E (2007): Tics moderate the outcome of treatment with medication but not cognitive-behavioral therapy in pediatric obsessive-compulsive disorder. *Biol Psychiatry* 61:344–347.

March J, Mulle K (1998): *OCD in Children and Adolescents: A Cognitive-Behavioral Treatment Manual*. New York: Guilford Press.

March J, Mulle K, Herbel B (1994): Behavioral psychotherapy for children and adolescents with obsessive-compulsive disorder: An open trial of a new protocol-driven treatment package. *J Am Acad Child Adolesc Psychiatry* 33:333–341.

Martin JL, Thienemann M (2005): Group cognitive-behavior therapy with family involvement for middle-school-age children with obsessive-compulsive disorder: A pilot study. *Child Psychiatry Hum Dev* 36:113–127.

Mataix-Cols D, Marks I, Greist JH, Kobak K (2002): Obsessive-compulsive symptom dimensions as predictors of compliance with and response to behavior therapy: Results from a controlled trial. *Psychother Psychosom* 71:255–262.

Moore P, Whaley SE, Sigman M (2004): Interactions between mothers and children: Impacts of maternal and child anxiety. *J Abnorm Psychol* 113:471–476.

Mosteller F, Colditz GA (1996): Understanding research synthesis (meta-analysis). *Annu Rev Public Health* 17:1–23.

Mullick M, Goodman R (2005): The prevalence of psychiatric disorders in 5–10 year olds in rural, urban and slum areas in Bangladesh. *Soc Psychiatry Psychiatr Epidemiol* 40:663–671.

Neziroglu F, Stevens K, McKay D, Yaryura-Tobias JA (2001): Predictive validity of the Overvalued Ideas Scale: Outcome in obsessive-compulsive and body dysmorphic disorders. *Behav Res Ther* 39:745–756.

Piacentini J (1999): Cognitive behavioral therapy of childhood OCD. *Child Adolesc Psychiatr Clin N Am* 8:599–616.

Piacentini J, Bergman L, Jacobs C, McCracken JT, Kretchman J (2002): Open trial of cognitive behavior therapy for childhood obsessive-compulsive disorder. *J Anx Disord* 16:207–219.

Piacentini J, Bergman R, Keller M. (2003): Functional impairment in children and adolescents with obsessive-compulsive disorder. *J Child Adolesc Psychopharmacol* 13:S61–S69.

Pollack RA, Carter AS (1999): The familial and developmental context of obsessive-compulsive disorder. In: King RA, Scahill L, editors. *Obsessive Compulsive Disorder: Child and Adolescent Psychiatric Clinics of North America, vol. 8*. Philadelphia: W.B. Saunders 461–479.

POTS (2004): Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder. *JAMA* 292:1969–1976.

Rosenthal R (1979): The file drawer problem and tolerance for null results. *Psychol Bull* 86:638–641.

Rosenthal R (1991): Meta-analysis: A review. *Psychosom Med* 53:247–271.

Rosenthal R (1994): Science and ethics in conducting, analyzing, and reporting psychological research. *Psychol Science* 5:127–134.

Scahill L, Vitulano LA, Brenner EM, Lynch KA, King RA (1996): Behavioral therapy in children and adolescents with obsessive-compulsive disorder: A pilot study. *J Child Adolesc Psychopharmacol* 6:191–202.

Shadish WR, Ragsdale K (1996): Random versus nonrandom assignment in psychotherapy experiments: Do you get the same answer? *J Consult Clin Psychol* 55:22–28.

Smits J, O' Cleirigh C, Otto M (2006): Combining cognitive-behavioral therapy and pharmacotherapy for the treatment of panic disorder. *J Cognitive Psychother* 20:75–84.

Steketee G (1997): Disability and family burden in obsessive-compulsive disorder. *Can J Psychiatry* 42:919–928.

Steketee G, Barlow DH (2002): Obsessive compulsive disorder. In: Barlow DH, editor. *Anxiety and Its Disorders: The Nature and Treatment of Anxiety and Panic, 2nd ed*. New York: Guilford Press, 516–550.

Steketee G, Chambless DL, Tran GQ (2001): Effects of Axis I and II comorbidity on behavior therapy outcome for obsessive-compulsive disorder and agoraphobia. *Compr Psychiatry* 42:76–86.

Stewart S, Geller D, Jenike M, Shaw D, Mullin B, Faraone S (2004): Long-term outcome of pediatric obsessive-compulsive disorder: A meta-analysis and qualitative review of the literature. *Acta Psychiatr Scand* 110:4–13.

Swedo S, Rapoport J, Leonard H, Lenane M, Cheslow D (1989): Obsessive compulsive disorders in children and adolescents: Clinical phenomenology of 70 consecutive cases. *Arch Gen Psychiatry* 46:335–343.

Thienemann M, Martin J, Cregger B, Thompson HB, Dyer-Friedman J (2001): Manual-driven group cognitive-behavioral therapy for adolescents with obsessive-compulsive disorder: A pilot study. *J Am Acad Child Adolesc Psychiatry* 40:1254–1260.

Valderhaug R, Ivarsson T (2005): Functional impairment in clinical sample of Norwegian and Swedish children and adolescents with obsessive-compulsive disorder. *Eur Child Adolesc Psychiatry* 14:164–173.

Valleni-Basile L, Garrison C, Jackson K (1995): Family and psychosocial predictors of obsessive compulsive disorder in a community sample of young adolescents. *J Child Fam Stud* 4:193–206.

Waters T, Barrett P, March J (2001): Cognitive-behavioral family treatment of childhood obsessive-compulsive disorder: Preliminary findings. *Am J Psychother* 55:372–387.

Wever C, Rey JM (1997): Juvenile obsessive-compulsive disorder. *Aust N Z J Psychiatry* 31:105–113.