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Evidence-Based Psychosocial Treatments for Child and Adolescent Obsessive–Compulsive Disorder

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Child and adolescent obsessive–compulsive disorder (OCD) is a chronic and debilitating condition associated with a wide range of impairments. This article briefly discusses the phenomenology of OCD, the theory underlying current treatment approaches, and the extant psychosocial treatment literature for child and adolescent OCD relative to the criteria for classification as an evidence-based intervention. Studies were evaluated for methodological rigor according to the classification system of Nathan and Gorman (2002) and then were assessed relative to the criteria for evidence-based treatments specified by Chambless et al. (1998), Chambless et al. (1996), and Chambless and Hollon (1998). Results from exposure-based cognitive behavioral therapy (CBT) trials with children and adolescents have been consistent, with remission rates of the disorder ranging from 40% to 85% across studies. Findings from this review indicate that individual exposure-based CBT for child and adolescent OCD can be considered as a *probably efficacious* treatment. CBT delivered in a family-focused individual or group format can be considered as a *possibly efficacious* treatment. Moderators, mediators, and predictors of treatment outcome are discussed, as are implications and generalizability of extant findings to real-world settings. We conclude with recommendations for best practice and future research directions.

Child and adolescent obsessive–compulsive disorder (OCD) is a chronic and debilitating condition that accrues significant concurrent and long-term risk to affected youth (Bolton, Luckie, & Steinberg, 1995; Hanna, 1995; Piacentini, Bergman, Keller, & McCracken, 2003; Pine, Cohen, Gurley, Brook, & Ma, 1998). More common than once thought, the disorder affects between 0.5% and 2% of children and adolescents (Flament et al., 1988; Heyman et al., 2003; Rapoport et al., 2000; Zohar, 1999), thus paralleling the prevalence rates reported within the adult population (Torres et al., 2006; Weissman et al., 1994). Growing awareness of the scope and impact of the disorder has been met with heightened research activity focused on identifying effective interventions, both psychosocial and psychopharmacological, for youth with OCD. Such work has generated an emerging evidence base and has spurred the publication of expert consensus guidelines (March, Frances, Kahn, & Carpenter, 1997) and practice parameters (American Academy of Child and

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Adolescent Psychiatry, 1998) for the treatment of this disorder. Both sets of guidelines recommend exposure-based cognitive behavioral therapy (CBT), either alone or in conjunction with a serotonin reuptake inhibitor (SRI) as a frontline intervention for youth with OCD. Although not empirically based, such guidelines undoubtedly mark a big step forward for enhancing treatment for youth with OCD; however, there is still much to be done to further understand and improve the available treatments for these youngsters.

In this article, we provide a brief discussion of the phenomenology of child and adolescent OCD and the theory underlying current treatment approaches. We then review the current state of the psychosocial treatment research literature, evaluating the specific studies comprising this literature base relative to the criteria for classification as an evidence-based intervention. We discuss mediators, moderators, and predictors of treatment outcome as well as the implications and clinical generalizability of findings to date. We conclude with a discussion of recommendations for best practice and future directions that stem from this body of work.

PHENOMENOLOGY

OCD is characterized by recurrent obsessions that stimulate anxiety or other distress and lead to compulsive behaviors (or avoidance) designed to reduce these noxious states. Historically, the prominent role of anxiety in the disorder has led to the classification of OCD with other anxiety disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (e.g., *DSM–IV–TR*; American Psychiatric Association, 2000). However, a growing body of evidence from the phenomenological, neurobiological, genetic, and treatment literatures has raised questions about this nosological classification and, as noted next, provides some justification for the consideration of OCD separate from the other anxiety disorders. Moreover, this evidence also has kindled recent calls for the placement of OCD in a newly created OC spectrum disorders category in *DSM–V* (Bartz & Hollander, 2006).

Several lines of family, genetic, and neuropsychopharmacological research point to OCD as a complex neurobehavioral illness that may be distinct from other forms of anxiety (MacMaster et al., 2006; Nestadt et al., 2001; Szeszko et al., 2004). Richter, Summerfeldt, Antony, and Swinson (2003) found adult patients with OCD to have higher lifetime rates of any coexisting spectrum disorder (including Tourette disorder, body dysmorphic disorder, and some impulse control disorders, among others) than patients with either social anxiety disorder or panic disorder. In addition, the Johns Hopkins OCD Family Study (Nestadt et al., 2001) found higher rates of anxiety and depressive disorder in the relatives of OCD cases versus relatives of controls. By contrast, panic disorder, separation anxiety disorder, and recurrent major depressive disorder occurred more frequently in case relatives with OCD than those without OCD. This finding suggests that anxiety disorders co-occurring with OCD, with the possible exception of generalized anxiety disorder and agoraphobia, may emerge as a consequence of OCD rather than from shared etiology.

OCD also has been distinguished from other anxiety disorders on the basis of pathophysiology (Bartz & Hollander, 2006). Prevailing theories underscore the role of dysfunction in the frontal cortical-striatal-thalamocortical networks that govern complex motor programs, response inhibition, and affect integration (Mataix-Cols & van den Heuvel, 2006; McCracken, 2005). Whereas neuroimaging data implicate dysfunction in frontal-striatal circuitry involving the orbital frontal cortex, caudate nucleus, thalamus, and anterior cingulate gyrus in the pathophysiology of OCD (e.g., Saxena & Rauch, 2000), fear circuitry involving the amygdala, hippocampus, and certain prefrontal cortical structures are thought to be operative for most anxiety disorders (e.g., Kent & Rauch, 2004; Mataix-Cols & van den Heuvel, 2006). Finally, neuropsychopharmacological models point to the unique characteristics of OCD (Rosenberg, Russell, & Fougere, 2005). In particular, although OCD and the other anxiety disorders share treatment responsiveness to SRI medication, the selective efficacy of other medication classes (e.g., benzodiazepines and norepinephrine reuptake inhibitors) for all of the anxiety disorders except OCD raises further questions regarding the shared etiology of these disorders (Bartz & Hollander, 2006).

A thorough review of the aforementioned literature is beyond the scope of this article (see McCracken, 2005; Rosenberg et al., 2005). However, taken together, findings from multiple strands of research provide compelling evidence that OCD is distinct from other anxiety disorders, and they argue for an examination of psychosocial treatments that is separate from these other conditions. Certainly, this line of approach is not new. Indeed, the value of considering OCD as distinct from other forms of anxiety has been underscored by existing psychosocial and psychopharmacological treatment studies that have considered OCD separately, while grouping other youth anxiety disorders (e.g., social anxiety disorder, separation anxiety disorder, and generalized anxiety disorder) together within the same trial (e.g., Barrett, Dadds, & Rapee, 1996; Kendall et al., 1997; The Research Units on Pediatric Psychopharmacology Anxiety Study Group, 2001).

THEORETICAL FOUNDATION FOR TREATMENT

A number of theories have been put forward to account for the development and maintenance of OCD. Early behavioural theories (Dollard & Miller, 1950) emphasized fear and avoidance conditioning models wherein neutral stimuli are paired with naturally anxiety provoking events and subsequently come to elicit distress themselves. Fear is then maintained by the negative reinforcement provided by escape or avoidance behaviors (i.e., compulsions). Cognitive behavioral accounts have expanded on these ideas to emphasize faulty cognitive appraisals and, in particular, tendencies toward distorted risk appraisal, inflated sense of responsibility for harm, excessive self-doubt, and thought-action fusion (i.e., thinking of a harmful act is the same as actually doing it) that appear to be central to OCD (Salkovskis, 1996). These theoretical models have provided the foundation for the bulk of psychosocial treatment development and research for both youth and adults with OCD and as such, have argued for primarily cognitive behavioural intervention approaches. Indeed, the vast majority of extant psychosocial treatment literature has employed some variant of CBT.

Current psychosocial treatments for OCD and the other anxiety disorders are largely similar in terms of their reliance on exposure and cognitive restructuring. At the same time, the relative emphasis on techniques is distinct for OCD, with exposure and response prevention (ERP) representing a cornerstone of effective treatment. ERP involves exposing patients to stimuli that trigger obsessive fears while encouraging them to resist engaging in compulsive behaviors invoked to reduce the obsession-triggered distress (Foa & Kozak, 1986; Meyer, 1966). The most commonly proposed mechanism for ERP efficacy is that, over repeated exposures, obsession-triggered anxiety dissipates through the process of autonomic habituation. In addition, as the individual's fears dissipate, she or he comes to learn that the feared consequences of not ritualizing will not materialize. As noted, cognitive factors such as inflated sense of responsibility for harm, excessive self-doubt, and thought-action fusion have been implicated as important etiological and maintaining factors for OCD in adults (Salkovkis, 1996). The extent to which these factors are specific to, or even applicable to, child and adolescent OCD remains unclear (Barrett & Healy, 2003; Barrett & Healy-Farrell, 2003). Despite this limited empirical support, cognitive techniques directly addressing obsessional beliefs and/or aimed at enhancing compliance with ERP are included routinely in interventions for young people with OCD (e.g., Kearney & Silverman, 1990; see Piacentini, March, & Franklin, 2006; Soechting & March, 2002).

REVIEW OF THE PSYCHOSOCIAL TREATMENT LITERATURE FOR CHILD AND ADOLESCENT OCD

In an effort to present a thorough account of the current state of the psychosocial treatment literature for child and adolescent OCD, this review begins with a narrative description of the specific studies composing this literature. Where possible and to the extent feasible based on the actual source articles, we provide information on sample demographics that may influence generalization of findings to diverse populations. As noted, all of the child and adolescent OCD psychosocial treatments tested to date have been based on exposure plus response prevention delivered in either individual or group format and in the presence or absence of an adjunctive family-based (typically, parent) intervention. In addition, almost all have included some form of cognitive intervention either to address obsessional thinking directly or to enhance compliance with ERP. Relative to the other child and adolescent treatment research areas that are covered in this special issue, the number of controlled youth OCD psychosocial trials published to date is small ($N = 4$). Because we wish to provide an overview of the status of the extant literature while also offering conclusions regarding the efficacy of various treatment approaches, we have therefore also included uncontrolled trials in our review (e.g., Abramowitz, Whiteside, & Deacon, 2005).

At the same time, to reflect the effects of including less methodologically rigorous trials, each study has been classified using the scheme developed by Nathan and Gorman (2002). According to this scheme, Type 1 studies describe the most rigorous scientific evaluations, involving randomized, prospective clinical trials, including comparison groups, blinded assessments, inclusion and exclusion criteria, state-of-the-art diagnostic methods, adequate sample size, and clearly described statistical methods. Type 2 studies are clinical trials in which an intervention is made but some aspects of the Type 1 study requirement are missing—for example, a trial in which two treatments are compared but assignment is not randomized. Type 3 studies are clearly methodologically limited, in that they generally are open trials aimed at obtaining pilot data. These studies are largely subject to observer bias. Type 4 and 5 studies include reviews with (Type 4) or without (Type 5) secondary data analysis, whereas Type 6 classification refers to reports of marginal value such as single case reports, essays, and opinion papers. Only studies meeting the criteria for a Type 1, Type 2, or Type 3 study were included in the present review. Initial Nathan and Gorman (2002) classification of studies for this review was conducted by the first author. These classifications then were reviewed by the remaining authors and discussed and

refined by all authors using a consensus approach. Because rates of agreement were not tracked formally, specific reliability estimates for this classification approach are not available.

In addition to using the Nathan and Gorman (2002) criteria, the published interventions for child and adolescent OCD also are evaluated relative to criteria for *well-established, probably efficacious, possibly efficacious*, and *experimental* treatments (based on criteria suggested by Chambless et al., 1998; Chambless et al., 1996; and Chambless & Hollon, 1998). According to this system, designation as well-established requires (a) at least two ''good'' group-design studies from different investigative teams showing the treatment to be superior to pill placebo or alternate treatment or equivalent to an already established treatment in studies with adequate statistical power, and (b) treatment manuals, clearly specified patient populations, psychometrically sound assessment measures, and appropriate statistical analyses. Probably efficacious denotes interventions (a) shown as more effective than no-treatment (or wait-list) control in two ''good'' studies, or (b) with one or more ''good'' studies meeting the requirements for well-established treatment classification except for having been done in separate research settings or by separate research teams. Designation as possibly efficacious requires the presence of at least one ''good'' study demonstrating the intervention to be efficacious in the absence of evidence to the contrary. Finally, treatments that have yet to be evaluated in methodologically rigorous trials are deemed experimental treatments. For purposes of this review, a ''good'' group design experiment was operationally defined as one that met the criteria for Type 1 classification as set forward by Nathan and Gorman.

SUMMARY OF EVIDENCE-BASED PSYCHOSOCIAL INTERVENTION STUDIES FOR CHILD AND ADOLESCENT OCD

Potential studies for this review were identified through a number of sources, including searches of the PsycINFO and Medline databases (keywords: *OCD* or *obsessive, exposure* or *behavior therapy* or *cognitive-behavior therapy*, and *child* or *adolescent* or *pediatric obsessive–compulsive disorder*), examination of review articles and past treatment studies on this topic, and consultation with investigators working in this area. Only studies published since 1994, the date of the first published child OCD treatment study utilizing standardized treatment protocols (as noted in March 1995), were considered. Our search produced 50 peer-reviewed articles, 21 of which were potential Type 1, 2, or 3 psychosocial treatment studies written in English, involving children and adolescents with OCD, and including more than 1 participant. Three studies were classified as Type 1 (Barrett, Healy-Farrell, & March, 2004; Grunes, Neziroglu, & McKay, 2001; and Pediatric OCD Treatment Study [POTS] Team, 2004). Four were identified as Type 2 (Asbahr et al., 2005; de Haan, Hoogduin, Buitelaar, & Keijsers, 1998; Franklin et al., 1998; Storch et al., 2007). Fourteen were identified as Type 3, 10 of which examined individual CBT (ICBT; Benazon, Ager, & Rosenberg, 2002; Knox, Albano, & Barlow, 1996; March, Mulle & Herbel, 1994; Piacentini, Gitow, Jaffer, Graae, & Whitaker, 1994; Piacentini, Bergman, Jacobs, McCracken, & Kretchman, 2002; Scahill, Vitulano, Brenner, Lynch, & King, 1996; Storch et al., 2006; Valderhaug, Larsson, Göttestam, & Piacentini, 2007; Waters, Barrett & March, 2001; Wever & Rey, 1997), and 4 of which centered on group-administered CBT (Fischer, Himle, & Hanna, 1998; Himle, Fischer, Van Etten, Janeck, & Hanna, 2003; Martin & Thienemann, 2005; Thienemann, Martin, Cregger, Thompson, & Dyer-Friedman, 2001).

After careful examination, 2 of the 3 Type 1, all 4 of the Type 2, and 10 of the 14 Type 3 (7 individual and 3 group treatment) studies were retained for inclusion in this review. Before describing the studies retained for review, a note about the excluded studies is warranted. Briefly, Grunes et al. (2001), a comparison of ERP alone to ERP plus a family component, included only 6 individuals younger than 18 in a total sample of 28 individuals and did not report treatment response for these youths separately. Given that it was not possible to draw conclusions about the efficacy of the study treatments for childhood OCD from this investigation, this Type 1 study was not considered for further review. The primary reason for excluding three of the Type 3 ICBT trials (Knox et al., 1996; Piacentini et al., 1994; Wever & Rey, 1997) was small sample size (all Ns < 4). Notably, although Wever and Rey treated 57 children and adolescents in their study comparing CBT, medication, and combined treatment, only 3 of these youngsters received CBT only, thereby precluding interpretation of study findings. The fourth excluded Type 3 study, Fischer et al. (1998), served as an interim report of the Himle et al. (2003) group CBT (GCBT) study described later in this article and was eliminated to avoid inflating the literature through double counting of study participants.

For the studies retained for inclusion in this review, within each study category, we made a further distinction between individual-child and family-focused CBT interventions. Recognizing that family involvement in OCD treatment varies substantially across studies in terms of type, intensity, and level of standardization, this distinction was based primarily on the degree of family involvement reported across trials. Child and

adolescent treatment necessarily involves some degree of parental involvement; thus, for the purpose of our review, interventions calling for regular check-in with parents at the end of each session or during specific parent-designated sessions were still considered individual treatments. This approach is in keeping with the convention used in the non-OCD anxiety literature. For example, the widely used Coping Cat intervention (Kendall, 1994) is considered an individual child treatment even though it specifies two specific parent sessions plus additional parental involvement as needed. By contrast, interventions for non-OCD anxiety including separate regular standardized parent sessions occurring in parallel to the child treatment are considered family-based (e.g., Barrett et al., 1996; Cobham, Dadds, & Spence, 1998). Notably, the term *family-focused* typically has been used to indicate systematized parent involvement in child treatment (e.g., Storch et al., 2007; Waters et al., 2001); few studies have included systematic intervention with parents and siblings (e.g., Barrett, Healy-Farrell, & March, 2004). Evaluative summaries are provided next for each of the studies retained for inclusion in this review. We begin by describing the most methodologically robust studies (Type 1) and then provide a review of the studies classified as Type 2 and 3 (based on Nathan and Gorman's criteria); we conclude with comments on the overall limitations of this body of work.

REVIEW OF TYPE 1 (RANDOMIZED CONTROLLED) STUDIES

As noted, two randomized controlled psychosocial outcome studies for children and/or adolescents with OCD (Barrett, Healy-Farrell, & March, 2004; POTS Team, 2004) met criteria for consideration as Nathan and Gorman (2002) criteria for Type 1 studies based on their design features, which included random assignment, blind assessment, clear description of eligibility criteria, sufficient statistical power, and state-of-the-art assessment and data analytic methods. One of these trials included a medication condition (POTS Team, 2004) and one (Barrett, Healy-Farrell, & March, 2004) compared ICBT and GCBT (both of which included a standardized family component) to a waitlist control condition (see Table 1).

Barrett and colleagues (2004) provided the first controlled comparison of family-focused individual versus family-focused group CBT for child and adolescent OCD. Both treatment conditions, which were contrasted against a waitlist control group, included a standardized family component, ''Freedom From Obsessions and Compulsions Using Cognitive-Behavioural Strategies'' (FOCUS; Barrett, 2007). Adapted from the March et al. individual treatment protocol (March & Mulle, 1998; March et al., 1994), the FOCUS program includes a structured parent and sibling protocol and allows both individual and group treatment delivery. The sample consisted of 77 youth ages 7 to 17 (M age = 11.7 years) with a primary diagnosis of OCD who either were medication free or agreed to maintain a stable medication regimen over the course of the study. Treatment integrity checks were also employed, along with assessment of participant and parent satisfaction with treatment. After active treatment, two booster sessions were conducted at 1 and 3 months posttreatment to provide further support to participants.

Treatment led to a 65% mean reduction on the Children's Yale–Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al., 1997), the gold standard clinician-rated severity measure for OCD, for participants in family-focused ICBT and a 61% mean reduction for family-focused GCBT. In addition, 88% of youth involved in the individual condition were diagnosis free at the completion of treatment, as were 76% of participants in the group condition. All youngsters in the waitlist condition continued to meet criteria for OCD at postassessment, although the relatively short duration of the waitlist condition (4–6 weeks) potentially limits the utility of comparisons between the active and no-treatment groups. There were no significant differences between treatment conditions, indicating that individual- and group-based treatments were equally effective at providing positive outcomes. These treatment outcome results were maintained at 3 and 6-month follow-up assessment, with no significant treatment condition differences evident at either follow-up point.

In a follow-up study, Barrett, Farrell, Dadds, and Boulter (2005) evaluated the long-term durability of family-focused ICBT and GCBT and investigated potential pretreatment predictors of long-term outcome. This study involved 48 participants ages 8 to 19 years who had received either family-focused ICBT or GCBT in the original study (90% of the original active treatment sample). Participants and parents were assessed at 12 and 18 months following treatment with standardized assessments, including diagnostic and symptom severity interviews, child self-report measures of anxiety and depression, and parental self-report of distress. Analyses indicated treatment gains were maintained for all participants, with 70% of participants in individual therapy and 84% in group therapy diagnosis free at follow-up. There were no significant differences between the individual or group conditions across measures. Results indicated that higher pretreatment CY-BOCS symptom severity scores and higher family dysfunction reported by mothers and fathers (measured by the Family Adjustment Device) predicted worse long-term outcome. These findings suggest that family-focused

TABLE 1
Description of Type 1

Study	*Sample*	*Treatment(s)*	*Design Elements*
ICBT			
POTS Team (2004)	$N = 112$ Age 7–17 50% female Ethnicity: NR Inc: OCD dx, CY-BOCS > 15, NIMH > 7, IQ > 80 Exc: MDD/bipolar, PDD, psychosis, primary TS, concurrent meds or therapy, 2 prior failed SRI or 1 failed CBT trials for OCD	ICBT–12 weeks ($N = 28$) Sertraline–12 weeks ($N = 28$) Combined tx–12 weeks ($N = 28$) Pill Placebo–12 weeks ($N = 28$) CBT based on March & Mulle (1998); 3 family sessions plus additional involvement as needed	+ outpatient setting (3 sites) + manualized protocol + sample eligibiltity characterized
ICBT + Family GCBT + Family			
Barrett, Healy-Farrell, & March (2004)	$N = 77$ Age 7–17 51% female Ethnicity: NR Inc: primary OCD Exc: TS, autism, MR, psychosis, organicity; stable meds ok	ICBT + Family–14 weeks ($N = 24$) GCBT + Family–14 weeks ($N = 29$) Waitlist – 4–6 weeks ($N = 24$) CBT based on March & Mulle (1998); Family tx: 14 group sessions for parents; 3 for siblings	+ outpatient setting + manualized protocol + sample eligibiltity characterized

Note: ICBT = individual cognitive behavioral therapy; POTS = Pediatric OCD Treatment Study; NR = not reported; OCD = obsessive-
of Mental Health Global Scale (Murphy, Pickar, & Alterman, 1982); MDD = Major Depressive Disorder; PDD = Pervasive
COM = Combination Treatment; PBO = Placebo; MR = Mental Retardation; ADIS–C = Anxiety Disorders Interview Schedule for *DSM–IV*
Children (March et al., 1997); FAD = McMaster Family Assessment Device (Epstein et al., 1983); DASS = Depression Anxiety Stress Scale
Inc = Inclusion Criteria; Exc = Exclusion Criteria.

Studies Included in Review

Measures (Informant)	*Results*	*Effect Size*	*Follow-Up*
OCD: CY-BOCS	CYBOCS: 46%↓for CBT, 30%↓SER, 53%↓COM, 15%↓PBO; COM > CBT = SER > PBO Remission (posttreatment CY-BOCS ≤ 10): 39% for CBT, 21% for SER, 54% for COM, 4% for PBO; COM = CBT, >SER, > PBO; CBT = SER, >PBO; SER = PBO	Within-group $CBT_{CYBOCS} = 2.61$ $SER_{CYBOCS} = 1.49$ $COM_{CYBOCS} = 4.20$ $PBO_{CYBOCS} = 1.12$ Between-group $CBT\text{-}PBO_{CYBOCS} = .99$ $SER\text{-}PBO_{CYBOCS} = .68$ $COM\text{-}PBO_{CYBOCS} = 1.46$ $COM\text{-}CBT_{CYBOCS} = .31$ $COM\text{-}SER_{CYBOCS} = .61$ $CBT\text{-}SER_{CYBOCS} = .27$	No follow-up data
OCD: ADIS-C (P) CY-BOCS (C) NIMH (C) OTHER: CDI (C) MASC (C) FAD (M,F) DASS (M,F) Sibling CDI, MASC, SAS	ADIS-C: 88% ICBT, 76% GCBT, 0% WL no Wk 14 OCD dx; ICBT = GCBT > WL CY-BOCS: 65%↓for ICBT, 61%↓GCBT, 5%↑WL; ICBT = GCBT > WL NIMH: 60%↓for ICBT, 63%↓GCBT, 4%↑WL; ICBT = GCBT > WL. No group differences on other measures. Age and med status not related to outcome	Within-group $ICBT_{CYBOCS} = 3.27$ $GCBT_{CYBOCS} = 4.03$ $WL_{CYBOCS} = -0.18$ Between-group $ICBT\text{-}WL_{CYBOCS} = 2.84$ $GCBT\text{-}WL_{CYBOCS} = 2.63$ $GCBT\text{-}ICBT_{CYBOCS} = .01$ $ICBT\text{-}WL_{NIMH} = 2.61$ $GCBT\text{-}WL_{NIMH} = 2.78$ $GCBT\text{-}ICBT_{NIMH} = .09$ $ICBT\text{-}WL_{MASC} = .06$ $GCBT\text{-}WL_{MASC} = .60$ $GCBT\text{-}ICBT_{MASC} = .66$ $ICBT\text{-}WL_{CDI} = .27$ $GCBT\text{-}WL_{CDI} = .82$ $GCBT\text{-}ICBT_{CDI} = .52$	Treatment gains maintained over 6 month follow-up. 18-month follow-up of 90% of active treatment groups found all participants to have maintained post-treatment gains with 70% of individual and 84% of group CBT participants diagnosis free (Barrett et al., 2005)

compulsive disorder; CY-BOCS = Children's Yale-Brown Obsessive-Compulsive Scale (Scahill et al., 1997); NIMH = National Institute Developmental Disorder; TS = Tourette's Syndrome; SRI = serotonin reuptake inhibitors; CBT = cognitive behavioral therapy; SER = Sertraline; Child Version (Silverman & Albano, 1996); CDI = Children's Depression Inventory (Kovacs, 1992); MASC = Multidimensional Anxiety Scale for (Lovibond & Lovibond, 1995); SAS = Sibling Accommodation Scale (Calvocoressi et al., 1995); WL = Wait List; Wk = Week; dx = diagnosis;

TABLE 2
Description of Type 2

Study	*Sample*	*Treatment(s)*	*Design Elements*
ICBT			
de Haan et al. (1998)	$N = 22$ Age 8–18 yrs 50% female Ethnicity: NR Inc: primary OCD Exc: TS, autism, MR, psychosis, organicity, primary MDD; BT or SRI meds past 6 mos	ICBT–12 sessions; family involvement not specified ($n = 12$) Clomipramine–12 weeks (m dose = 2.5 mg/kg) ($N = 10$) Outpatient Setting	+ outpatient setting + manualized protocol + sample eligibiltity characterized
Franklin et al. (1998)	$N = 14$ 10–17 yrs 71% female Ethnicity: NR Inc: primary OCD Exc: severe comorbid developmental disability 71% on concurrent SRI treatment	WT ($M = 16$ sessions over 4 months; $N = 7$) IT ($M = 18$ sessions over 1 month; $N = 7$), nonrandom assignment	+ outpatient setting (weekly and intensive treatment) + protocol driven + compared medication status and treatment intensity– sample eligibiltity not fully specified
ICBT + Family			
Storch et al. (2007)	$N = 40$ 7–17 yrs 55% female Ethnicity: 93% Caucasian Inc: primary diagnosis of OCD, CY-BOCS ≥ 16 Exc: psychosis, pervasive developmental disorder, bipolar disorder, current suicidality 60% on concurrent SRI treatment	14 sessions of weekly (WT; $N = 20$) or intensive (daily; IT; $N = 20$) treatment 1 parent present for every session Random assignment	+ outpatient setting + protocol driven − conditions differ in age and baseline symptom severity − limited interrater reliability on CY-BOCS − follow-up not conducted on all participants
GCBT			
Asbahr et al. (2005)	$N = 40$ Age 9–17 yrs 35% female Ethnicity: Latino Inc: primary OCD, treatment-naïve, NIMH > 7 Exc: primary MDD or ADHD, bipolar, PDD, PTSD, borderline PD, neurological disorder other than TS, autism, psychosis, organicity	GCBT–12 wks ($N = 20$) Sertraline–12 wks ($N = 20$) CBT based on March & Mulle (1998); 1 family session plus parent attended final 15 min of each session	+ outpatient setting + manualized protocol + sample eligibiltity characterized

Note: ICBT = individual cognitive behavioral therapy; yrs = years; Inc = Inclusion Criteria; Exc = Exclusion Criteria; NR = not reported; reuptake inhibitors; CY-BOCS = Children's Yale–Brown Obsessive-Compulsive Scale (Scahill et al., 1997); LOI–C = Leyton Obsessional CBCL = Child Behavior Checklist (Achenbach, 1991); CMI = Clomipramine; CBT = cognitive behavioral therapy; WT = weekly treatment; 1976); COIS–P = Children's Obsessive Compulsive Impact scale–Parent Report (Piacentini et al., 2003); CDI = Children's Depression for Children (March et al., 1997); NIMH = National Institute of Mental Health Global Scale (Murphy, Pickar, & Alterman, 1982); Personality Disorder; GCBT = group cognitive behavioral therapy; CGAS = Children's Global Assessment Scale (Shaffer et al., 1983);

Studies Included in Review

Outcome Measures	*Results*	*Effect Size*	*Follow-Up*
OCD: CY-BOCS LOI (C) OTHER: CDS (C) CBCL (P)	CY-BOCS: 60%↓, 66% response rate for ICBT; 33%↓, 50% response rate for CMI; ICBT > CMI No group differences on other measures	Within-group $CBT_{CYBOCS} = 1.58$ $CMI_{CYBOCS} = 1.45$ Between-group $CBT\text{-}CMI_{CYBOCS} = .86$ $CBT\text{-}CMI_{LOI} = .51$ $CBT\text{-}CMI_{CDS} = .32$ $CBT\text{-}CMI_{CBCL} = .27$	2 CBT nonresponders exhibited positive response following continued treatment
OCD: CY-BOCS Main Fear Main Ritual OTHER: HDRS	86% participants ≥ 50%↓ on CY-BOCS at posttreatment WT: 64%↓ CYBOCS IT: 70%↓ CYBOCS Significant reduction in severity of "main fear" and "main ritual" No significant impact on depressive symptoms (HDRS)	CY-BOCS $WEEKLY_{PRE\text{-}POST} = 2.48$ $INTENSIVE_{PRE\text{-}POST} = 3.57$ $CBT\text{-}ONLY_{PRE\text{-}POST} = 4.32$ $CBT + MEDS_{PRE\text{-}POST} = 2.29$ $HDRS_{PRE\text{-}POST} = .20$	10 of 12 available Ss were responders at 9-month follow-up
OCD: CY-BOCS CGI-S COIS-P FAS OTHER: CDI MASC	IT: 75% remission 90% response (CGI-I < 2) WT: 50% remission 5% response (CGI-I < 2)	CY-BOCS $WEEKLY_{PRE\text{-}POST} = 1.73$ $INTENSIVE_{PRE\text{-}POST} = 2.62$ CGI-S $WEEKLY_{PRE\text{-}POST} = 2.44$ $INTENSIVE_{PRE\text{-}POST} = 3.11$ COIS-P $WEEKLY_{PRE\text{-}POST} = 0.57$ $INTENSIVE_{PRE\text{-}POST} = 1.89$ FAS $WEEKLY_{PRE\text{-}POST} = 0.32$ $INTENSIVE_{PRE\text{-}POST} = 1.24$	No group differences at 3-month follow-up Follow-up data for 80% of total sample
OCD: CY-BOCS NIMH CGI-S CGAS OTHER: CDI (C) MASC (C)	Both treatment groups showed significant improvement on the CY-BOCS, NIMH, CGI, and CGAS Only the SER group had significant decrease on CDI and neither treatment group demonstrated significant improvement on the MASC	Insufficient data to calculate	9 month follow-up 5% (1/19) relapse in CBT vs. 53% (10/18) in SER following treatment discontinuation

OCD = obsessive–compulsive disorder; TS = Tourette's Syndrome; MR = Mental Retardation; MDD = Major Depressive Disorder; SRI = serotonin Inventory–Child Version (Berg, Rapoport, & Flament, 1986); CDS = Children's Depression Scale (Lang & Tisher, 1978; Luteijn, 1981); IT = intensive treatment; HDRS = Hamilton Depression Rating Scale (Hamilton, 1960); CGI–S = Clinical Global Impressions–Severity rating (Guy, Inventory (Kovacs, 1992); FAS = Family Accommodation Scale (Calvocoressi et al., 1995); MASC = Multidimensional Anxiety Scale ADHD = attention deficit hyperactivity disorder; PDD = Pervasive Developmental Disorder; PTSD = posttraumatic stress disorder; PD = SER = Sertraline.

CBT for OCD provides long-term symptom relief, and at present there is no evidence for significant differential effects for using CBT in either individual or group-based therapy formats. Notably, whereas the results for ICBT in the Barrett, Healy-Farrell, & March, (2004) trial are similar to those reported in the Type 2 and 3 trials (described next), the effect size for GCBT was considerably larger than those reported in the less methodologically rigorous studies described later in this review.

The POTS study (POTS Team, 2004) represents the first randomized controlled comparison of CBT, psychopharmacological treatment, their combination, and pill placebo for children and adolescents with OCD. The POTS study randomized 112 patients with OCD ages 7 through 17 years (M age = 11.7 years) recruited from three academic centers in the United States: Duke University, the University of Pennsylvania, and Brown University. Participants were randomly assigned to one of four conditions: ICBT alone, sertraline (a selective SRI, or SSRI) alone, combined ICBT and sertraline, or pill placebo for 12 weeks. Participants were seen weekly for medication adjustment based on an increasing dosage schedule, from 25 mg/d to a maximum of 200 mg/d over 6 weeks (POTS Team, 2004). To ensure a sample representative of treatment-seeking clients within the general community, inclusion and exclusion criteria were intentionally broad. Exclusion criteria involved the presence of primary major depression and/or Tourette's disorder, any pervasive developmental disorder, psychosis, concurrent treatment of OCD outside of the study, two previous failed attempts with an SRI medication or one failed CBT trial, any medical or neurological disorder, and pregnancy. Comorbid anxiety and/or externalizing disorders were allowed but could not be primary. The manualized CBT protocol was adapted from March and Mulle (1998) and consisted of 14 sessions conducted over 12 weeks, involving psychoeducation, cognitive training, symptom mapping, exposure and response prevention, and three parent sessions plus additional parent involvement as needed.

Posttreatment results indicated that all active treatments were significantly superior to the placebo condition in reducing OCD severity as measured by the CY-BOCS. On dimensional analyses of symptom severity, combined treatment (CBT + SSRI) proved to be superior to either CBT or SSRI alone, which did not differ from one another. However, when clinical remission (i.e., "excellent response" as indicated by posttreatment CY-BOCS ≤ 10) was used as the primary outcome measure, a significant advantage was found for the two CBT conditions, with the following response rates emerging: combination, 53.6%; CBT only, 39%; SSRI only, 21%; placebo, 3%. For these analyses, the combined condition did not differ from CBT alone; however, the combined treatment was superior to SSRI alone and to the placebo condition. The CBT alone condition did not significantly differ from the SSRI alone but did differ significantly from the placebo, whereas the SSRI treatment alone did not. Notably, study results were tempered by a significant Site $\times$ Treatment interaction, in which CBT alone was equivalent to combined treatment at one study site but not the other. The study authors interpreted this finding to indicate that under certain circumstances, optimal CBT may preclude the need for medication augmentation (POTS Team, 2004). Although the pattern of findings varied across different sets of analyses and is no doubt complicated by issues of sample size and power, findings from the POTS trial provide persuasive evidence for the efficacy of CBT, either alone or in conjunction with SSRI treatment.

REVIEW OF TYPE 2 TREATMENT STUDIES

The four Type 2 studies included in this review were classified as such because their designs were considered more rigorous than standard Type 3 trials but less stringent than those employed in Type 1 studies (see Table 2). Two studies (Franklin et al., 1998; Storch et al., 2007) examined ICBT delivered with different dose intensities and did not include a no-treatment or alternative treatment comparison condition, thus limiting their contribution to the evaluation of exposure-based CBT as, for example, either a well-established or probably efficacious treatment (Chambless et al., 1998; Chambless & Hollon, 1998; Chambless et al., 1996). Two additional studies were randomized controlled trials comparing CBT to pharmacotherapy (Asbahr et al., 2005; de Haan et al., 1998) that met the criteria for Type 2 (vs. Type 1) classification because they used a small sample size (de Haan et al., 1998) or lacked sufficient statistical detail to allow interpretation of study findings (Asbahr et al., 2005).

Franklin and colleagues (1998) conducted an open trial of CBT with 14 Caucasian youth (M age = 14.0 years, range = 10–17 years). A primary aim of the study was to determine whether a CBT intervention that omitted anxiety management techniques and instead relied on ERP as the central treatment strategy would be efficacious in treating youth with OCD. In addition, the study assessed whether treatment intensity (intensive vs. weekly) was related to treatment outcome. To examine this issue, half of the participants took part in intensive treatment, which involved an average of 18 sessions delivered over the course of 1 month. The other half received an average of 16 sessions over a span of 4 months. As noted earlier, assignment to treatment condition was not random but based on practical considerations specific to each participant. Of the 14 participants,

6 received CBT alone, and 8 received CBT while being continued on medication for OCD that was initiated prior to study entry. Following treatment, there was a mean reduction of 67% in CY-BOCS scores, with 12 of 14 participants showing at least 50% improvement in CY-BOCS symptom severity ratings. These improvements were maintained to 9-month follow-up assessment. Critically, outcomes did not vary by treatment intensity, and youth who received CBT alone fared equally as well as children concurrently taking medication.

More recently, Storch et al. (2007) compared the efficacy of intensive versus weekly individual family-based CBT for 40 children and adolescents with a primary diagnosis of OCD (M age = 13.3 years, range = 7–17 years). Treatment was delivered in a family-based format that required at least one parent to attend each session. In the intensive treatment arm, 14 sessions were conducted daily for 3 weeks; in the alternate treatment arm, sessions were held weekly for 14 weeks. Both treatments relied on the manual by Lewin, Storch, Merlo, Murphy, and Geffken (2005), with modifications designed to allow for greater parental psychoeducation and to facilitate the parental coaching during homework exercises. A standardized assessment battery was conducted pretreatment, posttreatment, and at 3-month follow-up, although posttreatment assessments were not done blind to treatment condition. Notably, despite a blinded randomization procedure, the intensive treatment group was significantly younger and had more severe OCD than the weekly treatment group.

Following treatment, illness remission (defined as a CY-BOCS total score of 10 or less) was achieved for 75% of participants in the intensive treatment and 50% in the weekly group. On the Clinical Global Improvement Scale, 90% of the intensive treatment completers were deemed treatment responders compared to 65% in the weekly treatment arm. Both groups evidenced a significant and similar improvement in psychosocial functioning and a significant decrease, although more so in the intensive versus weekly condition, in family accommodation of OCD symptoms. Although these findings suggest an initial advantage of intensive treatment over weekly treatment, the two treatment conditions demonstrated similar outcomes at 3-months posttreatment completion, a finding which suggests that youth who receive intensive treatment may continue to need additional clinical care.

In the first randomized controlled examination of CBT for child and adolescent OCD, de Haan et al. (1998) compared 12 weeks of ERP to 12 weeks of open treatment with clomipramine for 22 youth ages 8 to 18 years (M age = 13.7 years). Eligibility criteria included a primary diagnosis of OCD established on the basis of an unspecified semistructured interview and developmental history and the absence of a psychotic disorder, Tourette's disorder, pervasive developmental delay, mental retardation, or primary major depressive disorder. A standardized assessment protocol revealed significant improvement for both CBT and medication at posttreatment, although CBT led to a significantly greater decrease in CY-BOCS scores over time than pharmacotherapy (59.9% vs. 33.4%; $d_{\text{between group}} = 0.86$.[1] Nonresponders at 12 weeks were then treated openly with a combination of medication and CBT and evidenced a 55% response at 3-month follow-up. Although constrained by small sample size, these findings bolster support for the use of CBT as a frontline treatment for youth with OCD and point to its potential benefit over SRI treatment.

Asbahr et al. (2005) compared GCBT to sertraline in a randomized trial of 40 treatment naïve youth ages 9 to 17 years (M age = 13.05 years). Similar to Barrett, Healy-Farrell, & March, (2004), the GCBT format in this study was an adaptation of March and Mulle's (1998) treatment manual. A comprehensive standardized assessment battery was employed to assess participants at multiple time-points including pre- and posttreatment and 1, 2, 3, 6, and 9 months posttreatment. Consistent with de Haan et al. (1998), participants in both the psychosocial and medication conditions demonstrated significant improvement on the CY-BOCS at the end of treatment. Secondary measures of anxiety and depression also decreased over the course of treatment, with no group differences reported. However, at 9-month follow-up, youth in the GCBT condition reported significantly lower rates of symptoms compared to youth treated with sertraline. Although the Asbahr et al. (2005) article did not provide sufficient detail to allow determination of effect sizes for either treatment, the authors described their findings as convergent with those of Barrett, Healy-Farrell, & March, (2004) in demonstrating the efficacy of CBT delivered in group format and as providing further support for the potential long-term superiority of psychosocial versus psychopharmacological approaches.

REVIEW OF TYPE 3 (UNCONTROLLED) STUDIES

Collectively, the retained Type 3 treatment studies (Benazon et al., 2002; Himle et al., 2003; March et al.,

[1]Following Cohen (1988), between group effect sizes were calculated as follows:

$$d = (\bar{x}_t - \bar{x}_c)/\sqrt{\frac{(n_t - 1)s_t^2 + (n_c - 1)s_c^2}{n_t + n_c}}$$

TABLE 3
Description of Type 3

Study	*Sample*	*Treatment(s)*	*Design Elements*
ICBT			
March, Mulle & Herbel (1994)	$N = 15$ 8–18 yrs 67% female Ethnicity: NR Inc: primary OCD, Exc: not specified 93% on concurrent SSRI treatment that varied for some	$M = 10$ sessions (range 3–21) Treatment included Anxiety Management Component (AMT) including relaxation, self-talk, and other coping strategies	+ outpatient setting + protocol driven − sample eligibiltity not fully specified
Scahill et al. (1996)	$N = 7$ 9–16 yrs 71% female Ethnicity: NR Inc: moderately severe OCD Exc: not specified 71% on concurrent SRI treatment	M = 15 sessions Separate therapist for child and parent components of treatment	+ outpatient setting + protocol driven − sample eligibiltity not fully specified
Benazon, Ager, & Rosenberg (2002)	$N = 16$ 8–17 years 50% female Ethnicity: 100% Caucasian Inc: OCD diagnosis, CY-BOCS > 16, Exc: bipolar or unipolar depression, tic disorder, conduct disorder, learning disorder, psychosis, MR, autism, seizure disorder history, contraindicated medical condition, any concomitant treatment, substance abuse past 6 mos, significant suicide/homicide risk	$M = 12$ sessions Treatment based on March & Mulle (1998) and Schwartz's (1996) Four Steps Program Four parent sessions	+ outpatient setting + protocol driven + sample eligibiltity characterized
Piacentini et al. (2002)	$N = 42$ 5–17 yrs 60% female Ethnicity: NR Inc: primary OCD diagnosis Exc: not specified 52% on concurrent medication	$M = 12.5$ sessions All families attended first two sessions with subsequent involvement depending on specifics of case. Most families attended multiple additional sessions	+ outpatient setting + protocol driven + large sample size + examined predictors of response − sample eligibility not fully specified − CY-BOCS not used at outcome
ICBT + Family			
Waters, Barrett & March (2001)	$N = 7$ 10–14 yrs 57% female Ethnicity: NR Inc: primary OCD, CGAS < 70 Exc: primary MDD or other anxiety disorder, TS, schizophrenia, MR, organicity	$M = 14$ sessions Parallel manualized parent skills-training component. 4 joint parent-child sessions.	+ outpatient setting + protocol driven + sample eligibiltity characterized + assessment of child/family functioning
Storch et al. (2006)	$N = 7$ 9–13 years 43% female Ethnicity: 86% Caucasian Inc: primary OCD-PANDAS subtype diagnosis Exc: psychosis, pervasive developmental disorder, bipolar disorder, current suicidality 86% concurrent medication	$M = 14$ sessions All sessions included at least one parent	+ test of exportability + protocol driven + comprehensive assessment − small sample size − concurrent medication for portion of sample

Studies Included in Review

Outcome Measures	*Results*	*Effect Size*	*Follow-Up*
OCD: CY-BOCS NIMH CGI	50%↓ CY-BOCS, 52%↓ on NIMH, 69% on CGI. 67% participants $\geq$ 50%↓ on CY-BOCS at posttreatment 40% asymptomatic (NIMH $\leq$ 2 at posttreatment)	CY-BOCS$_{\text{PRE-POST}}$ = 1.57	Gains maintained at 18 month follow-up; Successful medication discontinuation in 6 patients with CBT booster sessions
OCD: CY-BOCS	61%↓ CY-BOCS 3 Ss refusing CBT showed no symptom change over study interval	CY-BOCS$_{\text{PRE-POST}}$ = 2.05	All Ss evidenced symptom exacerbation by 6 months post tx, with 5 of 7 responding to booster CBT
OCD: CY-BOCS NIMH OTHER: HAM-A HDRS	63% participants $\geq$ 50%↓ on CY-BOCS at posttreatment 44% asymptomatic (NIMH $\leq$ 2 at posttreatment) Significant reduction in HAM-A and HDRS scores	CY-BOCS$_{\text{PRE-POST}}$ = 1.65 HAM-A$_{\text{PRE-POST}}$ = 1.09 HDRS$_{\text{PRE-POST}}$ = 0.99	No follow-up data
OCD: NIMH CGI-I	79% response rate (CGI-I < 2) 45%↓ NIMH No difference between CBT alone or CBT + medication Poorer response predicted by higher CY-BOCS Obsession score and greater OCD-related academic impairment both at baseline	NIMH OVERALL$_{\text{PRE-POST}}$ = 2.08 CBT-ONLY$_{\text{PRE-POST}}$ = 2.14 CBT + MEDS$_{\text{PRE-POST}}$ = 2.12	No follow-up data
OCD: ADIS-C/P CY-BOCS NIMH CGI OTHER: CGAS CDI FAS FAD	86% OCD diagnosis free at posttreatment 60% mean reduction in CY-BOCS and NIMH Significant reduction in family accommodation No change in family functioning as measured by FAD	CY-BOCS$_{\text{PRE-POST}}$ = 2.15 NIMH$_{\text{PRE-POST}}$ = 2.20 CGAS$_{\text{PRE-POST}}$ = 1.41 CDI$_{\text{PRE-POST}}$ = .83 FAS$_{\text{PRE-POST}}$ = 2.28	Improvements maintained at 3 month follow-up
OCD: CY-BOCS ADIS-P CGI-S CGI-I OTHER: TODS-PR CDI MASC	86% response (CGI < 2) 71% diagnosis free at posttreatment	CY-BOCS$_{\text{PRE-POST}}$ = 3.38 ADIS-P$_{\text{PRE-POST}}$ = 2.30 CGI-S$_{\text{PRE-POST}}$ = 2.90	50% responders at 3-month follow up

(*Continued*)

TABLE 3

Study	*Sample*	*Treatment(s)*	*Design Elements*
Valderhaug et al. (2007)	$N = 28$ 8–17 years 50% female Ethnicity: Norwegian Inc: primary OCD diagnosis Exc: MR, anorexia nervosa, Tourette's syndrome, psychotic disorder, PDD 20% on concurrent medication	$M = 12$ sessions All families attended first two sessions with subsequent involvement depending on specifics of case. Most families attended multiple additional sessions	+ outpatient setting + protocol driven + sample eligibility characterized − small sample
GCBT			
Thienemann et al. (2001)	$N = 18$ 13–17 yrs 12 male 6 female Ethnicity: NR Inc: OCD diagnosis Exc: not specified Exc: not specified	14 weekly sessions based on March & Mulle (1998). Adapted to 2-hr format	+ outpatient setting + protocol driven − sample eligibiltity not characterized + assessed parenting stress
Himle et al. (2003)	$N = 19$ 12–17 yrs 11 male 8 female Ethnicity: NR Inc: OCD diagnosis Exc: not specified 68% on concurrent medication	7 90-min adolescent sessions 1 optional parent session covering psychoeducation about OCD plus discussion of OCD-related family problems	+ outpatient setting + protocol driven + thorough assessment − #parents attending parent session not reported
Martin &Thienemann (2005)	$N = 14$ 8–14 yrs 4 male 9 female Ethnicity: NR Inc: primary OCD diagnosis Exc: inability to attend group due to scheduling conflicts 64% on concurrent medication	14 weekly sessions based on March & Mulle (1998). Adapted to 90-min format	+ outpatient setting + protocol driven + thorough assessment

Note: Effect size (Cohen's $d = M_1–M_2/_{s\text{pooled}}$. ICBT = individual cognitive behavioral therapy; yrs = years; NR = not reported; Inc = Children's Yale–Brown Obsessive-Compulsive Scale (Scahill et al., 1997); NIMH = National Institute of Mental Health Severity rating (Guy, 1976); tx = treatment; MR = Mental Retardation; HAM-A = Hamilton Anxiety Rating Scale (Hamilton, 1960); Depressive Disorder; TS = Tourette's Syndrome; ADIS-C/P = Anxiety Disorders Interview Schedule for *DSM-IV* Child/Parent Version 1995); FAD = McMaster Family Assessment Device (Epstein, Baldwin, & Bishop, 1983); PANDAS = pediatric autoimmune neuropsychiatric Anxiety Scale for Children (March et al., 1997); PDD = Pervasive Developmental Disorder; K-SADS-PL = Schedule for Affective Disorders and vioral therapy; CBCL = Child Behavior Checklist (Achenbach, 1991); PSI = Parenting Stress Index (Abidin, 1995); COIS-P/C = Children's

Continued

Outcome Measures	*Results*	*Effect Size*	*Follow-Up*
OCD: K-SADS-PL CGAS CY-BOCS NIMH CGI-S CGI-I	75% response rate (> 50% reduction in symptoms) 60.6% CY-BOCS reduction posttreatment	$CY\text{-}BOCS_{PRE\text{-}POST} = 3.52$ $CGI\text{-}S_{PRE\text{-}POST} = 2.76$	68.8% CY-BOCS reduction at 6-month follow-up
OCD: CY-BOCS NIMH CGI OTHER: MASC CDI CBCL PSI	25% mean reduction in CY-BOCS 50% participants $\geq$ 25%$\downarrow$ on CY-BOCS at posttreatment Significant reductions in MASC, CDI, CBCL total scores No change in either maternal or paternal Parenting Stress (PSI)	$CY\text{-}BOCS_{PRE\text{-}POST} = 1.15$ $NIMH_{PRE\text{-}POST} = 0.78$ $MASC_{PRE\text{-}POST} = 0.44$ $CDI_{PRE\text{-}POST} = 0.29$ $CBCL_{PRE\text{-}POST} = 0.64$ $PSI\text{-}Mother_{PRE\text{-}POST} = 0.00$ $PSI\text{-}Father_{PRE\text{-}POST} = 0.00$	No follow-up data
OCD: CY-BOCS	30% mean reduction in CY-BOCS No difference between CBT alone or CBT + medication No difference between CBT alone or CBT + tic disorder	$CY\text{-}BOCS_{PRE\text{-}POST} = 0.82$	No follow-up data
OCD: CY-BOCS NIMH CGI-I Target OCD Symptoms COIS-P COIS-C OTHER: MASC-P MASC-C CDI CBCL-T CBCL-I	24.8% mean reduction in CY-BOCS 43% participants $\geq$ 25%$\downarrow$ on CY-BOCS at post-treatment. Average posttreatment CGI-I rating was "much improved" Significant reductions in COIS-P, CDI, CBCL total and internalizing scores No significant change in COIS-C or MASC-P or MASC-C	$CY\text{-}BOCS_{PRE\text{-}POST} = 1.05$ $NIMH_{PRE\text{-}POST} = 1.00$ $COIS\text{-}P_{PRE\text{-}POST} = 0.91$ $COIS\text{-}C_{PRE\text{-}POST} = 0.22$ $TARGET\ Sx\ 1_{PRE\text{-}POST} = 0.40$ $TARGET\ Sx\ 2_{PRE\text{-}POST} = 0.26$ $MASC\text{-}P_{PRE\text{-}POST} = 0.22$ $MASC\text{-}C_{PRE\text{-}POST} = 0.20$ $CDI_{PRE\text{-}POST} = 0.42$ $CBCL\text{-}T_{PRE\text{-}POST} = 0.52$ $CBCL\text{-}I_{PRE\text{-}POST} = 0.49$ $PSI\text{-}Mother_{PRE\text{-}POST} = 0.00$ $PSI\text{-}Father_{PRE\text{-}POST} = 0.00$	No follow-up data

Inclusion Criteria; Enc = Exclusion Criteria; OCD = obsessive–compulsive disorder; SSRI = selective serotonin reuptake inhibitors; CY-BOCS = Global Scale (Murphy, Pickar, & Alterman, 1982); CGI-I = Clinical Global Impressions-Improvement; CGI–S = Clinical Global Impressions– CGAS = Children's Global Assessment Scale (Shaffer et al., 1983); HDRS = Hamilton Depression Rating Scale (Hamilton, 1960); MDD = Major (Silverman & Albano, 1996); CDI = Children's Depression Inventory (Kovacs, 1992); FAS = Family Accommodation Scale (Calvocoressi et al., disorders associated with streptococcus; TODS–PR = Tourette's Disorder Scale–Parent Rated (Shytle et al., 2003); MASC = Multidimensional Schizophrenia for School-Age Children-Present and Lifetime Version (Kaufman, Birmaher, Brent, & Rao, 1997); GCBT = group cognitive beha- Obsessive Compulsive Impact Scale-Parent/Child Report (Piacentini et al., 2003).

TABLE 4
Classification of Psychosocial Treatments for Obsessive–Compulsive Disorder in Children and Adolescents

Psychosocial Treatment	*Citation for Evidence*
Well-Established Treatments	
None	—
Probably Efficacious Treatments	
Individual Cognitive Behavioral Therapy	POTS (2004)
Individual Cognitive Behavioral Therapy + Sertraline	POTS (2004)
Possibly Efficacious Treatments	
Family-Focused Individual Cognitive Behavioral Therapy	Barrett et al. (2004)
Family-Focused Group Cognitive Behavioral Therapy	Barrett et al. (2004)
Experimental Treatments	
Group Cognitive Behavioral Therapy	Asbahar et al. (2005) Thienemamm et al. (2001) Himle et al. (2003) Martin et al. (2005)

Note: POTS = Pediatric OCD Treatment Study Team.

1994; Martin & Thienemann, 2005; Piacentini et al., 2002; Scahill et al., 1996; Storch et al., 2006; Thienemann et al., 2001; Valderhaug et al., 2007; Waters et al., 2001) exhibit a number methodologically rigorous design features, including the use of standardized treatment protocols that incorporate cognitive and/or family treatment components, reliable and valid assessment measures, and follow-up designs to evaluate treatment durability. These features constitute a substantial improvement over the single-case design studies that characterized the earliest published Type 1, 2, and 3 trials (March, 1995). The seven Type 3 studies of ICBT or individual plus family CBT included in this review each led to significant improvements in OC symptoms ($d_{\text{within group}} = 1.57$–$4.32$),[2] with mean reductions on the CY-BOCS ranging from 51 to 70% (see Table 3). Furthermore, these trials suggest that treatment gains generally are maintained at follow-up assessments, which ranged in mean time of follow-up from 3 months (Scahill et al., 1996; Waters et al., 2001) to 21 months posttreatment (March et al., 1994). The three retained studies of GCBT (Himle et al., 2003; Martin & Thienemann, 2005; Thienemann et al., 2001) were similarly rigorous in design and execution and also found significant, although less dramatic, improvements in OC symptomatology for group ($d_{\text{within group}} = 0.82$–$1.15$) as compared to individual treatment.

Individual Treatment Studies

In one of the first open trials of manualized CBT for children and adolescents with OCD, March et al. (1994) evaluated an exposure-based CBT protocol with 15 consecutive participants in a university-based clinical research program (M age = 14.3 years, range = 8–18 years, 87% Caucasian), assessing youth at posttreatment and follow-up (range = 3–21 months posttreatment). Results indicated that 6 participants were asymptomatic on the National Institute of Mental Health-Global Obsessive Compulsive Scale (NIMH-GOCS; Insel, Hoover, & Murphy, 1983), a clinician-rated single item index of overall OCD severity, immediately posttreatment and 9 demonstrated at least a 50% reduction in CY-BOCS symptoms. There was no indication of any patient relapse at follow-up assessment and booster treatment enabled 6 participants to discontinue medication with minimal to no return of symptoms.

Scahill and colleagues (1996) utilized a standardized CBT protocol with a parental component to treat seven youngsters with OCD (M age = 13.0 years, range = 10.8–15.8 years). The treatment protocol was conducted across 14 individual sessions and parental education sessions were conducted every other week from Sessions 3 to 11 with the goal of training parents to serve as coaches during homework exercises. All participants demonstrated a clinically significant reduction of symptoms, with a mean reduction of 61% on the CY-BOCS. These gains were maintained at 3-month follow-up, and booster sessions again were effective in preventing relapse in the majority of participants. Notably, three children who met criteria for OCD but elected not to receive the treatment showed no change in CY-BOCS scores from baseline to follow-up.

Piacentini et al. (2002) conducted an open trial of manual-guided CBT with 42 children and adolescents (M age = 11.7 years, range = 5–17 years). Approximately half the sample (52%) was on psychotropic medication for their OCD at the time of referral, and these youth remained on a stable dose over the course of treatment. A manualized family treatment

[2]Within group effect sizes were calculated using the following formula: $d = (X_{\text{post}} - X_{\text{pre}})/_{\text{spooled}}$ (Cohen, 1988).

component provided psychoeducation about OCD and guidelines for helping parents to disengage from involvement with their child's OCD symptoms and to foster generalization and maintenance of treatment gains. However, the nature and extent of parental involvement varied according to the clinical picture of individual patients and was negotiated between the therapist and the family. Results indicated a 45% mean reduction in NIMH-GOCS ratings for both CBT alone and CBT plus medication, with 79% of all participants rated as significantly improved as measured using the CGI. The response rate did not differ between the CBT-only condition and the concurrent medication condition. Results also demonstrated that a poorer response to treatment, based on NIMH-GOCS score reductions, was related to more severe obsessions, greater anxiety, and poorer academic and social functioning, as measured by the Child OC Impact Scale (COIS; Piacentini et al., 2003), a rating scale measure of OCD-specific functional impairment, at baseline. Unfortunately, follow-up data were not available for this sample.

Benazon et al. (2002) evaluated an ICBT protocol via an open trial of 16 treatment naive Caucasian youth, ages 8 to 17 years (mean age not provided). Treatment was based on the integration of two existing therapy manuals (March & Mulle, 1998; Schwartz, 1996) and participants were assessed with a standardized assessment battery. At posttreatment, 10 participants had experienced a 50% reduction in CY-BOCS scores, and 44% were considered asymptomatic as judged by a NIMH-GOCS score of 2 or less. Participants also demonstrated a decrease in anxiety severity and a decrease in the severity of depressive symptoms. Follow-up data were not provided.

Individual Treatment With Family Involvement Studies

Waters et al. (2001) treated seven children and young adolescents ages 10 to 14 years (mean age not reported) with a 14-week individual treatment protocol that included a structured weekly parent skills training component. Results indicated that at posttreatment, 86% of participants were diagnosis free, with a mean reduction of 60% on both CY-BOCS and NIMH-GOCS severity ratings. These improvements were maintained to 3-month follow-up. Significant reductions were also found in family accommodation from pre- to posttreatment.

More recently, efforts have been made to assess the effectiveness of CBT for treating the pediatric autoimmune neuropsychiatric disorders associated with streptococcus (PANDAS) subtype of OCD. Storch et al. (2006) conducted a small open trial of seven children (M age = 11.1 years, range = 9–13 years) with PANDAS. Participants received 3 weeks of intensive CBT that involved 14 sessions of daily CBT with parental involvement designed to enhance compliance (Lewin et al., 2005). Treatment was adapted from POTS study (POTS Team, 2004) previously described. At the end of treatment, six of the seven participants were rated treatment responders by blind raters using standardized assessments. Of these youth, 50% maintained responder status at 3-month follow-up.

Individual Treatment Effectiveness Studies

As a growing number of efficacy studies have demonstrated that exposure-based CBT may be helpful in treating patients with OCD in controlled laboratory or hospital settings, attention has turned to whether these treatments are exportable to community settings. In the first published effectiveness study of CBT for childhood OCD, Valderhaug et al. (2007) examined the use of manual-guided individual + family CBT in three community outpatient clinics in Norway. Twenty-eight youth (M age = 13.3 years, range = 8–17 years) with a primary diagnosis of OCD were assessed pre- and posttreatment using an extensive standardized assessment battery. All participants except one (who was symptom free after seven sessions) completed 12 weeks of treatment following the manualized approach of Piacentini et al. (2002). Significant improvements were demonstrated on all outcome measures, with a mean symptom reduction of 60.6% on the CY-BOCS immediately posttreatment and 68.8% at 6-month follow-up.

Group Treatment Studies

Thienemann and colleagues (2001) treated 18 adolescents (M age = 15.2 years, range = 13–17 years) using a protocol that involved 14 weekly sessions of GCBT with 15 min of parental participation incorporated into each session. Group size ranged from 5 to 9 youngsters. OCD symptoms decreased significantly from pre- to posttreatment with a mean reduction of 25% in CY-BOCS scores and small reductions in Multidimensional Anxiety Scale for Children (March et al., 1997), Children's Depression Inventory (Kovacs, 1992) and Child Behavior Checklist (Achenbach, 1991) scores. The authors noted that their study did not allow for an examination of how group composition affected outcomes and aptly pointed out that although some youth are likely to benefit more than others from GCBT, the study design did not allow them to untangle this issue. In addition, participants were not required to be stable on medication over the course of treatment.

Himle et al. (2003) used GCBT to examine whether response to treatment was influenced by the presence of comorbid tics. Nineteen adolescents (M age = 14.63 years, range = 12–17 years) took part in a 7-week open

trial during which they received weekly 90-min sessions of CBT. Of the 19 participants, 8 had tic-related OCD and the remainder were classified as non-tic-related cases. Unfortunately, neither baseline diagnoses nor tic severity were assessed using standardized measures. All youth demonstrated significant improvements in CY-BOCS scores, and there were no differences across tic-related and non-tic-related adolescents, suggesting that comorbidity may not undermine response to CBT.

Martin and Thienemann (2005) evaluated a GCBT protocol with middle school children ages 8 to 14 years. Youth and their parents participated in 14 weeks of treatment based on the March and Mulle (1998) treatment manual but adapted to a weekly 90-min group format. Groups were composed of four to six families, and in addition to standardized assessments used pre- and posttreatment, weekly adherence ratings were obtained. The results indicated a substantial reduction in OCD symptoms as evidenced by a mean reduction of 25% on the CY-BOCS. In addition, youth were rated "much improved" on the NIMH-GOCS, and parents reported a significant decrease in OCD-related functional impairment posttreatment as measured by the COIS (Piacentini et al., 2003).

EVALUATIVE CONCLUSIONS

Limitations of Type 1 Treatment Studies

Although clinical trials by Barrett, Healy-Farrell, & March, (2004) and the POTS Team (2004) provide promising evidence to support the efficacy of CBT delivered either in individual format or in family-focused individual or group formats, a number of limitations merit consideration. Findings from the trial conducted by Barrett, Healy-Farrell, & March, (2004) are constrained by the lack of a primary outcome measure integrating both child- and parent-report information and by the use of a waitlist condition that was only 4 to 6 weeks in duration. In addition, it was not possible to assess the effects of treatment versus no treatment at follow-up, and low rates of response at 3- and 6-month follow-up limit interpretation of follow-up data. Likewise, findings from POTS are tempered by small sample size, limited power, and a Site $\times$ Condition interaction that obscures a clear picture of study findings. Furthermore, understanding of the durability of treatment gains is limited, at present, by the absence of follow-up data.

Limitations of Type 2 Treatment Studies

As with their Type 1 counterparts, all four of the Type 2 studies reviewed herein also provide support for the efficacy of exposure-based CBT for children and adolescents with OCD. Of interest, however, neither Franklin et al. (1998) nor Storch et al. (2007) demonstrated the durable superiority of intensive versus weekly CBT for childhood OCD. However, methodological concerns suggest caution in interpreting these findings. Most important, in both studies, the intensive and weekly treatment groups evidenced notable baseline differences in terms of both illness severity and demographic status. Other limitations include small sample size, lack of standardized diagnostic assessment (Franklin et al., 1998), and notable sample attrition at follow-up (Storch et al., 2007). In addition, neither study utilized blind outcome assessors. Likewise, although the large effect sizes reported by de Haan and colleagues (1998) are noteworthy, the small sample size in their randomized trial raises questions about the generalizability of these findings to the larger population. Finally, findings from Asbahr et al. (2005), although also promising, are undermined by the absence of adequate statistical information.

Limitations of Type 3 Treatment Studies

Taken together, the open trials conducted to date have provided a strong preliminary foundation to support the use of CBT in treating child and adolescent OCD. Type 3 studies of individual treatment suggest that it may be useful for both children and adolescents, that it is associated with favorable outcomes regardless of baseline medication status (Piacentini et al., 2002), and that it may be effective in the community (Valderhaug et al., 2007) and with PANDAS subtypes (Storch et al., 2006). In addition, work to date points to a number of factors that may influence treatment response, including baseline symptom severity, academic, and social functioning (Piacentini et al., 2002).

Despite the promise of these results, however, studies in this category suffer from a number of flaws endemic to open trial designs. With limited exceptions, the Type 3 studies previously reviewed are limited by relatively small sample sizes, the absence of a control condition, variability in the quality of assessment measures and the amount of treatment provided to participants and family members, and absent or inconsistent timing of follow-up assessments.

The available group treatment studies are equally difficult to evaluate. Although previous research in the field of child and adolescent anxiety has demonstrated that group-based treatment for the management of anxiety is helpful (e.g., Barrett, 1998; Mendlowitz et al., 1999; Shortt, Barrett, & Fox, 2001; Silverman et al., 1999), and apparently similarly efficacious, as individual approaches (Flannery-Schroeder & Kendall, 2000) the limited open trial data previously described make it difficult to draw conclusions about the comparative

efficacy of group versus ICBT for OCD. In contrast to findings from Barrett, Healy-Farrell, and March (2004) controlled trial, data from the Type 3 open trials suggest that treatment outcomes may be less impressive for group as compared to ICBT, with posttreatment CY-BOCS reductions of 25% to 32% for group treatment, compared to 50% to 85% in individual treatment studies. As some youth are likely to be more responsive to GCBT than others, it is also unclear at this juncture who is most likely to benefit or how group configuration affects treatment response.

Critically, given that the group-based studies conducted to date are open trials, they include the limitations of the other Type 3 studies discussed earlier, including issues related to sample size, assessment procedures, and lack of randomization or control groups. It is also important to note that youth in two of the group treatment studies (Martin & Thienemann, 2005; Thienemann et al., 2001) were not on stable medication over the course of treatment, a factor that makes it difficult to determine whether improvements were related to the effects of psychosocial treatment, medication change, or placebo effects. Thus, although these studies provide some preliminary support for group-based CBT of youth with OCD, controlled evaluations comparing individual versus group delivery of CBT are necessary to advance our understanding of the relative efficacy of these treatment modalities.

EVIDENCE-BASED STATUS OF TREATMENTS FOR OCD

Based on this review, there are no treatments that currently meet the criteria for a well-established treatment as specified by Chambless et al. (1998), Chambless et al. (1996), and Chambless and Hollon (1998). At present, the most thoroughly examined intervention, exposure-based ICBT, meets the requirements for designation as a probably efficacious psychological intervention (see Table 4). As noted earlier, the distinction of probably efficacious requires either (a) two independent randomized controlled trials demonstrating the superiority of the treatment in question to waitlist control or (b) at least one study demonstrating the treatment to be superior to pill or psychological placebo or an alternative treatment. Exposure-based ICBT meets these requirements based on findings from the POTS Team (2004) suggesting that ICBT is equivalent to (and possibly superior to) the established SSRI, sertraline, and superior to pill placebo. In addition, although thorough examination of combined treatment strategies for child and adolescent OCD is beyond the scope of this review, combination treatment (ICBT and sertraline) appears to meet the criteria for probably efficacious based on its superiority to SSRI medication alone and possible superiority to CBT alone (POTS Team, 2004).

The results of this review also indicate that both family-focused ICBT and family-focused GCBT treatment can be considered possibly efficacious treatments based on Barrett, Healy-Farrell, & March, (2004), who demonstrated the superiority of both interventions to a waitlist control condition (Table 4). Finally, our review suggests that GCBT without an intensive structured family component remains an experimental treatment because of the absence of controlled data on this intervention modality. Certainly, these classifications are likely to change as additional trials are completed; however, they mark significant advances in the treatment of child and adolescent OCD over the past few years.

IMPLICATIONS AND CLINICAL GENERALIZABILITY

Despite the promising efficacy demonstrated to date for CBT, certain methodological limitations in the existing literature must be noted. First, the vast majority of studies to date have utilized primarily Caucasian samples that are relatively free of many of the serious comorbidities commonly associated with OCD (e.g., depressive disorder, tic disorders, externalizing disorders). Indeed, with the exception of the Latino sample employed by Asbahr et al. (2005), the vast majority of studies reporting ethnicity demographics have incorporated samples that are more than 85% Caucasian (e.g., Storch et al., 2007; Storch et al., 2006) or, in some cases, fully Caucasian (e.g., Benazon et al., 2002). When reported, minority participants typically have been of Latino descent and have constituted too small a number (e.g., $n = 1$) from which to draw evaluative conclusions about ethnic group differences related to treatment response (e.g., Storch et al., 2007; Storch et al., 2006). Thus, it remains unclear whether treatment efficacy varies as a function of child ethnicity or race.

With regard to comorbidity, studies that have been flexible with regard to exclusion criteria or that have included highly comorbid cases tend to fall into the Type 3 category and thus suffer from a number of methodological issues that undermine generalization. At the same time, the stringent exclusion criteria applied in some Type 1 studies makes it difficult to determine how *typical* the evaluated samples are of community-based treatment referrals. Indeed, a review of the mean severity ratings across the studies included in this review (based on CY-BOCS total scores) indicates that all samples were within *moderate* range of severity at

pretreatment. As increased baseline symptom severity and functional impairment have been associated with a poorer treatment response (Barrett et al., 2005; Piacentini et al., 2002) for youth with *moderate* OCD severity, it is unclear how effective CBT, at least as administered in the studies previously described, will be in more demographically diverse populations and for youth with more severe and or complicated clinical presentations.

Second, even within the relatively homogenous samples studied thus far, there is still considerable room for improvement in treatment outcomes. Indeed, in spite of the relatively robust effect sizes noted for CBT and combined treatment, a substantial proportion of children and adolescents in both the CBT and medication trials noted above demonstrated a less-than-optimal response to treatment. For example, in the POTS trial, only 39% of youngsters receiving CBT only and only 53% of those receiving CBT + SSRI were considered to be even reasonably symptom free at the end of 12 weeks of treatment. In addition, the degree to which CBT positively influences psychosocial functioning, although examined in a handful of Type 2 and 3 studies, has not been well addressed by existing Type 1 trials. This is an important omission given the demonstrated negative impact of OCD on the social, academic, and family functioning of affected children and adolescents. It is interesting to note that, in contrast to the Type 1 psychosocial treatment literature, at least two randomized controlled multisite psychopharmacological trials for childhood OCD have demonstrated the efficacy of SSRI medication in improving psychosocial functioning (Geller et al., 2001; Liebowitz et al., 2002).

In addition, questions remain as to the optimal mode of CBT delivery and findings regarding the relative benefits of GCBT versus ICBT merit further examination. By and large, effect sizes from open trials of GCBT tend to be smaller than for ICBT. However, Barrett, Healy-Farrell, & March, (2004) did not find this disparity in their comparison of individual versus group treatment, a finding that may reflect the more rigorous design features of their Type 1 trial. As Asbahr et al. (2005) did not provide details to facilitate calculation of effect sizes, it is difficult to draw conclusions in this area and more research is needed to elucidate this issue.

Likewise, the role of families in treating child and adolescent OCD requires closer scrutiny. Despite the ample documentation of the impact of child and adolescent OCD on family functioning (e.g., Piacentini et al., 2003; Renshaw, Steketee, & Chambless, 2005; Waters & Barrett, 2000), the incremental efficacy of family (typically parent) participation in child treatment has yet to be examined systematically. Moreover, systematic family involvement in treatment has not yet been shown to significantly enhance either child outcomes or family functioning (e.g., Barrett, Healy-Farrell, Piacentini, & March, 2004). At the same time, the largest effect sizes of CBT reported thus far are derived from a treatment protocol with arguably the highest "dose" of family intervention (Barrett, Healy-Farrell, & March, 2004). Given the notable impact of OCD on the family (Renshaw et al., 2005) as well as the developmental considerations intrinsic to working with children and adolescents, it is no surprise that the general consensus in the field dictates that some degree of family involvement in the treatment of child and adolescent OCD is merited (e.g., Barrett, Healy-Farrell, & March, 2004; Piacentini et al., 2006; Renshaw et al., 2005). However, the nature of family involvement in therapy is unlikely to be a one size fits all proposition, and flexibility will be needed to address the widely differing needs (e.g., accommodation of OC symptoms vs. anger/frustration) of individual families presenting for treatment.

MEDIATORS, MODERATORS, AND PREDICTORS OF TREATMENT RESPONSE

As the understanding of child and adolescent OCD and its treatment moves forward, a natural question pertains to which treatments work best and for whom. By and large, there is only limited work addressing predictors of treatment response for youth with OCD and, unfortunately, this question remains largely unanswered. The lack of research on this important topic undoubtedly is related to the relatively small sample sizes and, hence, limited available statistical power, characterizing the psychosocial treatment studies for childhood OCD published to date. In the only study examining demographic factors published to date, Piacentini et al. (2002) did not find a relationship between treatment response and age, gender, or baseline medication status.

Although the adult literature suggests that comorbidity, motivation, fixity of beliefs, and family-level variables such as expressed emotion or patient perceptions of criticism are predictive of worse response to exposure-based CBT (Abramowitz & Foa, 2000; Chambless & Steketee, 1999; de Haan et al., 1997; Foa, Abramowitz, Franklin, & Kozak, 1999), these variables have yet to be examined in child and adolescent populations. However, the limited data that are available suggest that pretreatment individual and family functioning may be important predictors of psychosocial treatment response (Barrett et al., 2005; Piacentini et al., 2002) with greater symptom severity and worse anxiety, academic, and social functioning predictive of poorer outcomes.

Work formally testing these variables as mediators or moderators of treatment outcome has yet to be

conducted. Indeed, the only examination of putative moderators to date has involved secondary analysis of the POTS data (March et al., 2007), which found comorbid tics to moderate treatment outcome for youth receiving sertraline but not CBT. This finding argues for the use of CBT as the frontline intervention for youngsters with comorbid tic conditions (March et al., 2007).

RECOMMENDATIONS FOR BEST PRACTICE

Based on the literature published to date, exposure-based CBT appears to be a consistently beneficial intervention for child and adolescent OCD producing remission rates of disorder ranging from 40% to 85% across studies (Barrett, Healy-Farrell, & March, 2004; Benazon et al., 2002; POTS Team, 2004; Waters et al., 2001). CBT also has generated between-group effect sizes on the CY-BOCS ranging from 0.99 to 2.84 for the Type 1 studies and within-group effect sizes on the CY-BOCS from 1.57 to 4.32 (ICBT) and 0.82 to 1.15 (GCBT) for the Type 2 and 3 studies. The large to very large effect sizes demonstrated for the Type 1 CBT studies contrast notably with the psychopharmacological treatment literature. A recent meta-analysis of 12 published randomized placebo-controlled medication trials for childhood OCD comprising 1,044 participants found only a modest effect size for psychopharmacological intervention (pooled standardized mean difference = 0.46; 95% confidence interval = 0.37–0.55; Geller et al., 2003). Support for the potential advantage of CBT over psychopharmacological intervention also is provided by de Haan et al.'s (1997) finding that CBT was more efficacious than medication and by findings from the POTS (2004) trial. However, given the relatively small sample sizes for each of these studies, additional larger scale comparative trials clearly are needed to clarify this important issue.

In addition to the controlled CBT literature, uncontrolled trials suggest that CBT appears to be equally efficacious when delivered as monotherapy or as an adjunct to preexisting pharmacotherapy (Franklin et al., 1998; Piacentini et al., 2002; Storch et al., 2007). CBT also appears to deliver significant improvement for youth when delivered individually (i.e., Barrett, Healy-Farrell, & March, 2004; Franklin et al., 1998; Piacentini et al., 2002; POTS Team, 2004; Scahill et al., 1996) or in group format (i.e., Barrett, Healy-Farrell, & March, 2004; Fischer et al., 1998; Thienemann et al., 2001). Finally, although not blindly assessed, CBT efficacy appears durable to at least 18 months posttreatment (Barrett et al., 2005; March et al., 1994). The results of our review, especially when taken in the context of the child psychopharmacology literature (e.g., Geller et al., 2003), provide strong support for the use of individual CBT as a first-line intervention for children and adolescents with OCD seen in outpatient or day treatment settings and suggest that individual CBT accompanied by a structured family intervention and/or delivered in group format are reasonably viable therapeutic alternatives (see Table 4).

FUTURE DIRECTIONS FOR YOUTH OCD TREATMENT RESEARCH

Historically, OCD during childhood and adolescence has been considered a difficult condition to treat relative to other anxiety disorders. Given that the protocols described in this article differ substantially from traditional CBT approaches to child anxiety disorders in their emphasis on exposure plus response prevention, and in light of the current recognition of an underlying neurological basis to the pathogenesis of OCD, there is some leverage to this widely held belief. At the same time, the outlook for youth with OCD is promising. An examination of the current state of the literature reveals strong evidence for good, durable outcomes following 10 to 14 sessions of CBT. However, as noted earlier, there are a number of issues that need to be addressed in future research trials.

First, replication studies that can further understanding of best-treatment guidelines and help to establish the parameters of current treatments are in order. These studies must focus on further evaluating the relative efficacy of individual and group treatments, as well as examining the longer term durability of therapy outcome beyond 18 months posttreatment. Future trials with sophisticated designs and assessment protocols are also warranted to develop more specific treatment guidelines that can prescribe particular treatment modalities (i.e., individual vs. group; CBT alone vs. CBT + medication) for specific clinical presentations. In addition, controlled evaluations of CBT with more severe samples, including inpatient samples and youngsters with significant comorbidities, most notably tic, externalizing, and depressive disorders, are necessary to inform us of the efficacy of this intervention across a broader range of patients and clinical presentations.

Second, it is important to develop strategies for treating current treatment nonresponders. Intensive treatments, multimodal intervention, and more intensive family involvement are all intuitively promising strategies in this arena and future work must address the relative contribution of each as well as sequencing algorithms that may be used to guide individualized treatment approaches.

Third, closer examination of family involvement in treatment is in order. A growing body of literature

points to family factors characteristic of children and adolescents with OCD and suggests that these variables may influence treatment outcomes (see Renshaw et al., 2005, for a review). Although the majority of the child and adolescent treatment protocol involve some degree of family participation, on the whole, the open-ended and flexible nature of this involvement has prevented rigorous examination of what this component contributes to treatment. Indeed, even though both the expert consensus guidelines (March et al., 1997) and the American Academy of Child and Adolescent Psychiatry (1998) practice parameters recommend family involvement in treatment, there is as yet no direct empirical evidence to support this recommendation. Carefully controlled studies assessing the incremental efficacy of family involvement above and beyond the benefits associated with individual treatment have yet to be undertaken. Thus, an important step for future research will entail closer examination of family involvement in treatment as well as which particular family factors (e.g., accommodation, blame, conflict, etc.) are most relevant for youth outcomes.

Fourth, controlled research examining the central components of CBT for child and adolescent OCD as well as mediators and moderators of treatment response and mechanisms of action has yet to be conducted. For example, although the efficacy of primarily cognitive interventions has garnered some support in the adult literature (e.g., Abramowitz, 1997), this issue remains to be addressed in younger populations. Certainly, the non-OCD anxiety literature emphasizes the value of behavioral components of CBT interventions (e.g., exposure; Kendall et al., 2005), and it will be important to examine how developmental considerations bring to bear on the active ingredients of CBT treatment for child and adolescent OCD. Translational research efforts investigating the neurocognitive, neurobiological, and even genetic underpinnings of CBT efficacy (e.g., Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996) will also be necessary both to better understand how these treatments work and to guide the refinement of more effective interventions as well as methods for predicting optimal intervention candidates.

Finally, efforts will be needed to transport CBT treatments for child and adolescent OCD into community settings where they can be delivered effectively by *real* clinicians and applied to *real* patient populations. There is certainly an argument to be made that evidence-based outcomes are based on "unrealistic" conditions. For example, typical treatment trials are conducted by *expert* clinicians (usually doctoral level) with regular professional team supervision occurring throughout the trial to ensure first-class treatment integrity. Moreover, the often strict inclusion and exclusion criteria frequently means the most severe or *difficult* patients (i.e., those with comorbidity, or who have failed previous treatment attempts) are not actually included in these trials. Thus it will be important for the research community to develop systematic approaches to disseminating evidence-based protocols into the community through adequate professional training programs, to evaluate the effectiveness of these interventions across a broad range of settings (hospitals, community centers, schools), and to find ways to sustain evidence-based practice in the real world, with outcomes that can compare favorably to those reported in our clinical trials. Valderhaug et al. (2007) have taken important first steps in demonstrating the effectiveness of CBT delivered in community settings for youth with OCD; however, there is much remaining work to be done in this arena. The promising future of CBT for child and adolescent OCD, which can be considered a probably efficacious treatment based on more than 10 years of systematic evaluations, hinges on this pivotal next step.

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