Cognitive-Behavioral Therapy for Obsessive-Compulsive Disorder: A Meta-Analysis of Treatment Outcome and Moderators

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The present investigation employed meta-analysis to examine the efficacy of cognitive-behavioral therapy (CBT) for obsessive–compulsive disorder (OCD) as well as potential moderators that may be associated with outcome. A literature search revealed sixteen randomized-controlled trials (RCTs) with a total sample size of 756 participants that met inclusion criteria. Results indicated that CBT outperformed control conditions on primary outcome measures at post-treatment (Hedges's $g = 1.39$) and at follow-up (Hedges's $g = 0.43$). Subsequent analyses revealed few moderators of CBT efficacy. Neither higher pre-treatment OCD ($p = 0.46$) or depression symptom severity ($p = 0.68$) was significantly associated with a decrease in CBT effect size. Similarly, effect size did not vary as a function of 'type' of CBT, treatment format, treatment integrity assessment, blind assessment, age of onset, duration of symptoms, percentage of females, number of sessions, or percent comorbidity. However, active treatments showed smaller effect sizes when compared to placebo controls than when compared to waitlist controls. Effect sizes were also smaller for adult RCTs than child RCTs. Likewise, older age was associated with smaller effect sizes. However, an association between age and effect size was not observed when examining child and adult samples separately. This review indicates that while CBT is efficacious in the treatment of OCD, more research is needed to identify processes that may predict more favorable treatment responses.

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Obsessive-compulsive disorder (OCD) is characterized by recurrent obsessions or compulsions that cause marked distress and interfere with daily functioning (American Psychiatric Association [APA], 2000). Obsessions are defined as intrusive, repetitive thoughts, images, or impulses; and compulsions are purposeful, repetitive overt and covert behaviors or rituals performed in an effort to relieve obsessional distress. OCD affects between 2% and 3% of adults and 1% of children and adolescents (Flament et al., 1988; Karno et al., 1988). OCD can be a chronic illness when untreated and as many as 50% of adult OCD cases develop during childhood (Karno and Golding, 1991; Rasmussen and Eisen, 1990). OCD is also a very debilitating condition and sufferers often experience impairment in multiple areas of functioning resulting in a poorer quality of life (Olatunji et al., 2007). For example, research has shown that OCD is associated with significantly more functional impairment compared to healthy controls in areas of work, social life, and family life (Huppert et al., 2009). OCD has also been found to be associated with disability and increases in use of health-care services (Bobes et al., 2001), making it a significant public health concern.

Although once considered to be highly treatment resistant, researchers have made progress in identifying effective treatments for OCD over the last two decades. Controlled clinical trials consistently demonstrate that cognitive-behavioral therapy (CBT) substantially reduces symptoms of OCD (Franklin and Foa, 2002). CBT here refers to the class of interventions that are based on the basic premise that emotional disorders are maintained by cognitive and behavioral factors, and that psychological treatment leads to changes in these factors through cognitive and behavioral techniques (Beck and Emery, 2005). Currently, CBT that focuses on exposure and ritual prevention (ERP; Greist et al., 2003) is the psychological treatment of choice for OCD (NICE, 2006). ERP involves gradual prolonged exposure to fear-eliciting stimuli or situations combined with instructions to abstain from compulsive behavior. CBT that focuses on modifying dysfunctional beliefs about the presence or significance of the intrusive thoughts, commonly referred to as cognitive therapy (CT), has also shown promise in the treatment of OCD (van Oppen et al., 1995; Wilhelm et al., 2005).

Meta-analysis is the primary means by which researchers have synthesized the results from multiple treatment trials examining the efficacy of CBT for OCD. Although the use of meta-analytic data is not without limitations, this approach has proven useful in characterizing the general effectiveness of CBT in the treatment of OCD. In a meta-analysis of treatment outcome studies, Abramowitz (1997) found that ERP was highly effective in reducing OCD symptoms. CT approaches were also found to be at least as effective as ERP. A subsequent meta-analysis also found that effect sizes for

ERP was significantly greater than that of serotonin reuptake inhibitors for the treatment of OCD (Kobak et al., 1998). Eddy et al. (2004) later compared the effect sizes for ERP, CT, and their combination (CBT) for the treatment of OCD. Similar effect sizes were observed across these modalities, although slightly stronger for ERP and CBT conditions. Across all treatments, about two-thirds of the patients who completed treatment improved. A more recent meta-analysis also found that ERP, CT, and a combination of the two were very effective in reducing the symptoms of obsessions and compulsions and they seemed to show similar effectiveness (Rosa-Alcázar et al., 2008). One meta-analytic review of 16 controlled studies of ERP for adult OCD patients did find that the average OCD symptom reduction across studies was 48%, suggesting that the majority of patients remain symptomatic following treatment (Abramowitz et al., 2002). Thus, while meta-analyses have revealed high effect sizes for CBT in the treatment of OCD, it appears that certain characteristics may impinge on treatment efficacy.

Although much remains unknown about the predictors of treatment outcome, Keijsers et al. (1994) did find that greater initial OCD severity and depression predicted poorer outcome for compulsive behavior after ERP. In a study of intensive residential treatment for those with severe OCD, Stewart et al. (2006) found that while depression severity did not predict treatment outcome, lower initial OCD severity predicted less severe OCD at discharge. Baseline severity of OCD symptoms was also found to be associated with poorer response to CBT in a pediatric sample (Ginsburg et al., 2008). Based on such findings, a recent qualitative review of clinical predictors of response to CBT for OCD concluded that greater symptom severity predicts poorer treatment response (Keeley et al., 2008). However, Rufer et al. (2006) found no relation between pre-treatment symptom severity and outcome among a large sample of CBT-treated in-patients with OCD. A recent study also found that more severe symptoms was only marginally associated with worse outcome for those who completed CT for OCD (Steketee et al., 2011). Furthermore, depressed mood did not predict treatment outcome. In fact more Axis I comorbid diagnoses (mainly major depression and anxiety disorders) predicted *more* improvement. Although differences in the extent to which depression relates to treatment outcome may be observed when assessing depression as either a categorical or dimensional construct, Anholt et al. (2011) recently found that depression, either as a continuous or categorical variable, was not predictive of treatment response among OCD patients that received behavior or cognitive therapy either alone or with fluvoxamine.

The extent to which initial symptom severity or initial depression severity predicts treatment outcome in CBT for OCD is at best unclear in the current literature. However, it is possible that individual studies lacked the power necessary to detect symptom severity or initial depression moderation. Meta-analysis of many trials may help clarify these discrepancies. Clarifying such associations may have important implications for treatment planning in CBT. For example, if initial symptom severity predicts OCD treatment outcome, this may be justification for the use of more prolonged and intensive versions of CBT for patients that do present with more severe OCD symptoms. Similarly, if more initial depression severity predicts OCD treatment outcome, this may be justification for the use of adjunctive treatments to CBT for OCD patients that are also depressed. In fact, the potential negative impact of comorbid major depression on CBT outcome has prompted formulations of a treatment program specifically for depressed OCD patients (Abramowitz, 2004). Examining the extent to which depression severity predicts OCD treatment outcome may also inform the time course of treatment. Among those with more severe depression, motivation and compliance with difficult exposure assignments in CBT may be problematic, thereby interfering with effective treatment of OCD symptoms. Treatment of depressive symptoms before beginning CBT for OCD in such cases may then maximize outcome.

Examination of the extent to which sample-specific characteristics, study-specific characteristics, and treatment-specific characteristics predict outcome in CBT for OCD may also prove to be informative. Such an approach would be consistent with the prescription of Gordon Paul (1967), who observed over 40 years ago that "…the question towards which all outcome research should ultimately be directed is the following: What treatment, by whom, is most effective for this individual with that specific problem, and under which set of circumstances?" (p. 111). Although no one study can address such a complicated question (Beutler, 1991), examination of moderators in the context of meta-analysis of treatment outcome studies examining the efficacy of CBT for OCD may be useful in beginning to address this question. Accordingly, the present investigation employs a meta-analytic approach to examine the efficacy of CBT for OCD. Although several meta-analyses of CBT for OCD have been published, this investigation extents prior work by examining moderators of treatment outcome. Four questions derived from the existing literature were examined.

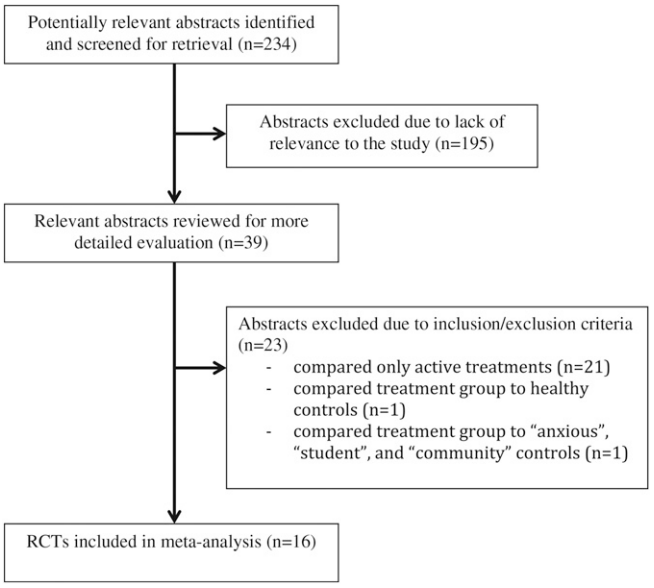
1. Do CBT treatments outperform control conditions on primary OCD outcome measures at post-treatment and at follow-up?
2. Do CBT treatments outperform control conditions on secondary depression outcome measures at post-treatment?
3. Does higher pre-treatment OCD severity and higher pretreatment depression scores predict lower effect sizes?
4. Does treatment type, control type, participant age, duration of symptoms, age of onset, percentage of female participants, percentage of comorbidity, number of sessions, inclusion of treatment integrity checks and/or blind assessors, population (adult or child), and treatment format (group or individual) moderate treatment effect sizes?

1. METHOD

1.1. Study Selection

Well-controlled (see inclusion criteria below) randomized trials (RCTs) of CBT for OCD were selected using a comprehensive

FIGURE 1. Study Selection and Reasons for Exclusions.



search strategy. A search was conducted in the following databases: PsycINFO (1840 to December 2011), MEDLINE (1966 to December 2011), and Scopus (1869 to December 2011). The searches included the following terms: "cognitive behavioral" or "cognitive behavioral therapy", and "clinical trial," or "trial" alone and in combination with "obsessive-compulsive," "obsessive compulsive," "obsessive-compulsive disorder," "obsessive compulsive disorder," or "OCD". These words were searched as key words, title, abstract, and Medical Subject Headings. Also, we examined citation maps and used the "cited by" search tools. These findings were cross-referenced with references from reviews. In addition, we contacted authors of CBT trials for emerging publications. As depicted in Fig. 1, these initial search strategies produced 234 potential articles. Examination of the abstracts identified 39 relevant articles. Inclusion criteria for the meta-analysis were as follows: (a) participants who met full DSM-III-R, DSM-IV, or DSM-IV-TR criteria for obsessive-compulsive disorder; (b) adequate control condition (psychological placebo, wait-list control, or pill placebo); and (c) more than one session of CBT during the acute phase of treatment. Treatments were classified as CBT if they included cognitive techniques (e.g. cognitive restructuring, behavioral experiments, etc.), behavioral techniques (e.g. in-vivo exposure, imaginal exposure, etc.), acceptance/mindfulness techniques (e.g. engaging in valued behaviors despite anxiety), or a combination of these strategies. Exclusion criteria for the meta-analysis were: (a) single case studies; (b) treatment conditions based on augmentation of psychological treatment (i.e. D-cycloserine augmented ERP); and (c) studies with insufficient data, unless study authors were able to provide such data. Of the 39 identified studies, 23 were excluded based on these criteria. Twenty-one studies did not have an adequate control condition and only compared active treatments, one study compared the treatment group to healthy controls not diagnosed with OCD, and one study did not contain adequate control groups. Table 1 shows the sixteen studies with a total sample size of 756 participants that met the final inclusion criteria and were included in the meta-analyses.

1.2. Software

All analyses were completed with Comprehensive Meta-Analysis (Bornstein and Rothstein, 1999).

1.3. Procedure

Data on the following variables were collected when reported in each of the 16 included studies: treatment conditions (15 studies; ACT was not included in the analysis as only one study utilized this treatment type), control type (16 studies), treatment dose (number of sessions; 16 studies), number of participants (16 studies), mean age (of both adult and child groups separately and combined; 16 studies), mean age of onset (5 studies), mean duration of symptoms (9 studies), percentage of female participants (16 studies), percent comorbidity (a higher percentage refers to more comorbidity; 8 studies), population (adult or child; 16 studies), and treatment format (group or individual; 16 studies). Treatment integrity variables, including inclusion or exclusion of treatment integrity checks and blind assessors, were collected categorically for all 16 studies and assumed to be absent if not reported in the manuscript. Dependent variables were classified into categories including primary (OCD symptom severity) and secondary (depression symptoms and quality of life ratings).

Control conditions were classified into two categories: placebo or wait-list. Treatments that were categorized as placebo included: stress management training (SMT), relaxation (R), pill placebo, and anxiety management (AM). Wait-list (WL) was defined as a control condition in which participants did not receive any treatment for OCD symptoms for a specified amount of time. Six of the sixteen studies utilized placebos (5 psychological placebos and 1 pill placebo) as the control condition, 9 of the 16 studies had wait-list as the control condition, and 1 of the 16 studies included both a placebo condition and a wait-list condition.

1.4. Effect Size Calculation

Between-group effect sizes for each study were computed using Hedges's g (Rosenthal, 1991). Studies with multiple outcomes were categorized as above (see Table 1) and then combined within each domain. When the necessary data were available, Hedges's g was calculated directly using the following formula: $g = \bar{X}_T - \bar{X}_C / S_p$ where $\bar{X}_T$ is the mean of the treatment group, $\bar{X}_C$ is the mean of the comparison group, and S_p is the pooled standard deviation. If these data were not provided, Hedges's g was estimated using conversion equations for significance tests (e.g., t, F) (Rosenthal, 1991). All effect sizes were corrected for small sample sizes according to Hedges and Olkin (1985). Therefore, a smaller sample size reduces the estimated effect size helping control for different sample sizes across studies. These controlled effect sizes may then be interpreted conservatively with Cohen's convention of small (0.2), medium (0.5), and

TABLE 1. Studies Included in the Meta-Analysis.

Study	Conditions	*N*	Sample	Mean age	# Of sessions	Primary outcome measure	Secondary outcome measure
Anderson and Rees (2007)	CBT vs. WL	51	Adult	33.7	10	YBOCS	BDI
Barrett et al. (2004)	CBT vs. WL	53	Child	11.8	14	CYBOCS	CDI
Bolton and Perrin (2008)	CBT vs. WL	20	Child	13.2	12	CYBOCS	None
Cordioli et al. (2003)	CBT vs. WL	47	Adult	36.5	12	YBOCS	HAM-D
Fals-Stewart et al. (1993)	CBT vs. Psych PL	93	Adult	30.5	12	YBOCS	BDI
Fineberg et al. (2005)	CBT vs. Psych PL	47	Adult	39.3	12	YBOCS	None
Foa et al. (2005)	CBT vs. Pill PL	41	Adult	34.3	23	YBOCS	HAM-D
Freeston et al. (1997)	CBT vs. WL	29	Adult	35.8	12	YBOCS	BDI
Jones and Menzies (1998)	CBT vs. WL	21	Adult	38.5	10	MOCI	BDI
Lindsay et al. (1997)	CBT vs. Psych PL	18	Adult	32.8	15	YBOCS	BDI
O'Connor et al. (1999)	CBT vs. WL	26	Adult	37.3	5	YBOCS	None
Simpson et al. (2008)	CBT vs. Psych PL	108	Adult	39.2	17	YBOCS	HAM-D
Twohig et al. (2010)	CBT vs. Psych PL	79	Adult	37.0	8	YBOCS	BDI-II
Whittal et al. (2010)	CBT vs. Psych PL vs. WL	73	Adult	31.5	12	YBOCS	BDI
Wilhelm et al. (2009)	CBT vs. WL	29	Adult	33.4	22	YBOCS	BDI
Williams et al. (2010)	CBT vs. WL	21	Child	13.6	10	CYBOCS	CDI

Note. CBT = Cognitive Behavior Therapy, WL = Waitlist, PL = Placebo, CYBOCS = Children's Yale-Brown Obsessive Compulsive Scale, MOCI = Maudsley Obsessional-Compulsive Inventory, YBOCS = Yale-Brown Obsessive Compulsive Scale, BDI = Beck Depression Inventory, BDI-II = Beck Depression Inventory-II, CDI = Children's Depression Inventory, HAM-D = Hamilton Rating Scale for Depression.

large (0.8) effects (Cohen, 1988). Hedges's g also may be computed directly from Cohen's d with the following formula: $g = d(1 - 3/4(n_1 + n_2) - 9)$. When there were multiple outcomes per domain they were combined according to Borenstein et al. (2007). The overall mean effect size for all of the studies combined was computed using the following formula: $\bar{g} = \sum w_j g_j / \sum w_j$ where w_j is the weight for each study and g_j is the effect size for each study. Effect sizes were calculated with random effects models. The random effects analysis estimates the overall effect size assuming the studies included are only a sample of the entire population of studies and/or when the studies are heterogeneous.

2. RESULTS

2.1. Question 1: Does CBT Outperform the Control Conditions on Primary OCD Outcome Measures at Post-Treatment and Follow-up?

The post-treatment analysis included 16 studies with 756 participants. Consistent with prediction, Fig. 2 shows that CBT outperformed control conditions on primary outcome (i.e., OCD symptoms) measures at post-treatment showing a large effect size (Hedges's $g = 1.39$ [SE = 0.18, 95% CI: 1.04–1.74, $p = 0.000$]). The follow-up analysis included 3 studies with 111 participants. CBT outperformed control conditions on primary outcome measures at follow-up showing a medium effect size (Hedges's $g = 0.43$ [SE = 0.16, 95% CI: 0.12–0.74, $p = 0.01$]).

2.2. Question 2: Does CBT Outperform the Control Conditions on Secondary Depression Outcome Measures at Post-treatment and Follow-up

The post-treatment analysis included 9 studies with 559 participants. CBT outperformed control conditions on secondary outcome (i.e., depression symptoms) measures at post-treatment showing a medium effect size (Hedges's $g = 0.51$ [SE = 0.15, 95% CI: 0.21–0.82, $p = 0.00$]). Follow-up analysis was not possible as only 1 study reported sufficient data on secondary outcome measures.

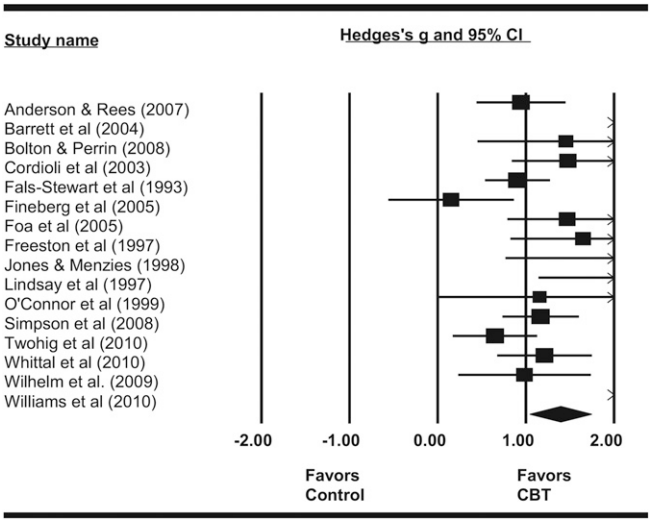
2.3. Heterogeneity

A heterogeneity analysis was conducted to test the assumption that the effect sizes were from a homogeneous sample (Hedges and Olkin, 1985). For this analysis all 16 studies were included with all time points (post-treatment and follow-up) on primary outcome measures. The test was significant, $Q(18) = 74.03$, $p = 0.000$, suggesting that the random effects analyses were most appropriate for this study. In addition, this significant heterogeneity suggests it may be appropriate to employ moderator analyses in an effort to identify potential sources of such between study variability.

2.4. Question 3: is Higher Pre-Treatment Severity and Depression Associated With Lower Effect Sizes?

The analysis for pre-treatment severity included 16 comparisons with 756 participants and revealed that higher pre-treatment severity was not significantly associated with lower effect size ($\beta = 0.05$, $p = 0.46$). The analysis for pretreatment

FIGURE 2. Effect Size Estimates (Hedges' g) for the Efficacy of CBT Compared to Control Condition on OCD Symptom Reduction.



depression included 14 comparisons with 751 participants and showed no significant relation between pre-treatment depression and effect size ($\beta = -0.02$, $p = 0.68$).

2.5. Question 4: Does Effect Size Vary as a Function of Treatment Type, Control Type, Mean Age, Percentage of Females, Number of Sessions, and Comorbidity?

The following analyses were completed using fully random effects categorical moderator analyses. First, we examined effect size as a function of two different treatment types: CT (3 studies) and ERP (12 studies). There were no significant differences in the magnitude of effect sizes in these two treatments: CT (Hedges's $g = 1.84$ [SE = 0.46, 95% CI: 0.94–2.74, $p = 0.00$]) and ERP (Hedges's $g = 1.35$ [SE = 0.20, 95% CI: 0.96–1.74, $p = 0.00$]).We also examined effect size as a function of two different control types: placebo (7 comparisons: 1 pill placebo and 6 psychological placebos) and waitlist (12 comparisons). Studies employing waitlist controls (Hedges's $g = 1.67$ [SE = 0.19, 95% CI: 1.31–2.04, $p = 0.00$)] showed larger effect sizes than studies using placebo controls (Hedges's $g = 0.92$ [SE = 0.20, 95% CI: 0.53–1.33, $p = 0.00$]). There were no significant differences after removing the study utilizing a pill placebo from the placebo control comparison ($n = 7$; Hedge's $g = 0.85$ [SE = 0.22, 95% CI: 0.43–1.28, $p = 0.00$)].

There were no significant differences in effect size between group ($n = 9$, Hedges's $g = 1.53$ [SE = 0.24, 95% CI: 1.06–2.00, $p = 0.00$]) and individual formats ($n = 10$, Hedges's $g = 1.24$ [SE = 0.22, 95% CI: 0.82–1.66, $p = 0.00$]). A significant difference in effect size was seen between adult ($n = 13$, Hedges's $g = 1.08$ [SE = 0.12, 95% CI: 0.85–1.32, $p = 0.00$]) and child populations ($n = 3$, Hedges's $g = 2.50$ [SE = 0.28, 95% CI: 1.94–3.05, $p = 0.00$]) with child populations being associated with significantly larger effect sizes. There were no significant differences in effect size between studies including treatment integrity checks ($n = 9$, Hedges's $g = 1.47$ [SE = 0.23, 95% CI: 1.01–1.92, $p = 0.00$]) and studies without treatment integrity checks ($n = 7$, Hedges's $g = 1.21$ [SE = 0.29, 95% CI: 0.65–1.77, $p = 0.00$]) or in studies with blind assessors ($n = 7$, Hedges's $g = 1.43$ [SE = 0.27, 95% CI: 0.90–1.95, $p = 0.00$]) and studies without blind assessors ($n = 9$, Hedges's $g = 1.31$ [SE = 0.25, 95% CI: 0.82–1.80, $p = 0.00$]).

Given that a significant difference in effect size was seen between adults and children, subsequent analyses were conducted to clarify this difference. The following analyses were completed using unrestricted maximum likelihood meta regressions. When including both adult and child studies, Fig. 3 shows that there was a significant relationship between effect size and mean age (16 studies), with older age associated with smaller effect sizes ($\beta = -0.06$, $p = 0.00$). However, when including only adult studies (13 studies), older age was not significantly associated with smaller effect sizes ($\beta = -0.04$, $p = 0.27$) and when including only child studies (3 studies), older age was also not significantly associated with smaller effect sizes ($\beta = 0.32$, $p = 0.65$). These findings suggest that the relation between age and effect size is best explained by the relationship between adult and child samples. However, the absence of an effect within children and adult samples may be due a restriction of age range. There was also not a significant relationship between effect size and each of the following: mean age of onset (5 studies; $\beta = -0.06$, $p = 0.53$), mean duration of symptoms (9 studies; $\beta = -0.02$, $p = 0.50$), percentage of females (16 studies; $\beta = -0.02$, $p = 0.08$), number of sessions (16 studies; $\beta = 0.02$, $p = 0.46$), or percent comorbidity (8 studies; $\beta = 0.01$, $p = 0.55$).

2.6. Publication Bias: "the File Drawer Problem"

Several authors suggest there may be a potential discrepancy between the number of published trials and the total number that are completed (Bakan, 1967; McNemar, 1960; Smart, 1964; Sterling, 1959). Therefore, any meta-analysis of published studies may be missing non-significant studies and therefore overestimate the overall effect size. Rosenthal and others have called this confound "The File Drawer Problem" (Rosenthal, 1991). A conservative method of addressing this problem is to assume that the effect sizes of all current or future *unpublished* studies in the omnibus effects size analysis are equal to 0 and compute the number of such studies it would require to reduce the overall effect size to a non-significant level (Rosenthal and Rubin, 1988). This value may be referred to as the "fail-safe *N*".

Rosenthal suggested the following equation to compute a fail-safe *N*: $X = K(K\bar{Z}^2 - 2.706)/2.706$ where K is the number of studies in the meta-analysis and $\bar{Z}$ is the mean Z obtained from the K studies (Rosenthal, 1991). Rosenthal also suggested that findings may be considered robust if the required number of studies (X) to reduce the overall effect size to a non-significant level exceeded $5K + 10$ which in this study would be 90 (Rosenthal, 1991). Analyses revealed that it would require more than 991 current or future unpublished studies with an effect size of 0 to bring the overall

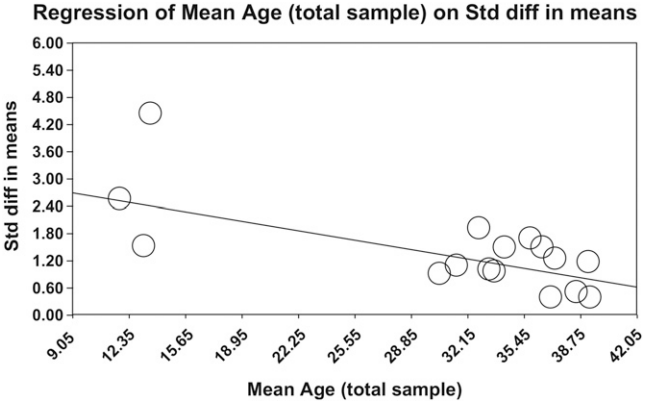
effect size of the primary analyses within the non-significant range, suggesting that the findings in this meta-analysis are robust as the fail-safe-*N* (991) is much larger than the convention for robust cutoff or $5K + 10$ (90).

3. DISCUSSION

The present investigation employed meta-analysis to examine the efficacy of CBT for OCD. Consistent with predictions, CBT out performed control conditions on primary OCD symptom outcome measures at post-treatment showing a large effect size. This finding is consistent with prior meta-analyses demonstrating that CBT is highly effective in reducing OCD symptoms (Abramowitz, 1997; Rosa-Alcázar et al., 2008). Importantly, the present study included a number of studies that have been published since these previous meta-analyses, and thus adds to the evidence base of CBT for OCD. The present investigation also found that CBT outperformed control conditions on primary OCD symptom outcome measures at follow-up showing a medium effect size. Although the sustained efficacy (the ability to produce lasting symptomatic changes) of CBT-based approaches for OCD has been questioned (e.g., Eddy et al., 2004), these meta-analytic findings suggest that OCD treatment gains attributed to CBT (relative to controls) are observed after treatment has been terminated. While these findings are encouraging, additional research is needed to adequately determine the extent to which CBT produces longer lasting symptom changes for patients with OCD (Olatunji et al., 2010). This will require future studies to include substantially longer follow-up intervals so more definite inferences can be made regarding the durability of CBT for OCD. Interestingly, the overall controlled effect size for CBT was significantly larger at post-treatment ($g = 1.39$) compared to follow-up ($g = 0.51$). This suggests that the effect of CBT for OCD may indeed diminish after the acute treatment phase. Future research aimed at identifying strategies that may be employed during and after CBT in order to better sustain treatment gains for patients with OCD may prove valuable.

The present study also found that CBT outperformed control conditions on secondary outcome measures of depression at post-treatment showing a medium effect size. Follow-up analysis of secondary outcome measures was not possible as only one trial included sufficient data for this analysis. One cross-national study found that the lifetime prevalence of major depressive disorder among those with OCD ranged from 12.4% to 60.3% across seven countries. In the United States, researchers have found a concurrent comorbidity rate of 36% and a lifetime comorbidity rate of 54% (Nestadt et al., 2001; Steketee, 1999). Furthermore, having comorbid major depressive disorder at intake has been found to be associated with a decreased likelihood of recovery from OCD over a 15-year period (Marcks et al., 2011). The prevalence and negative impact of depression in OCD may limit the general utility of CBT. Indeed, the specific and problem focused nature of CBT has been the basis of concerns

FIGURE 3. The Relationship Between CBT for OCD Effect Size and Mean Age.



that such a treatment may not generalize to "real-life" patients who frequently present with co-occurring conditions (Westen et al., 2004). However, the present meta-analysis revealed that CBT for patients with OCD also produced robust changes in secondary symptoms of depression. The post-treatment effects of CBT for OCD on depression observed in the present investigation are also consistent with those of Hofmann and Smits (2008) who found that CBT significantly outperformed placebo in reducing depression in OCD. It is generally accepted that OCD predates depression, suggesting that depressive symptoms typically occur in response to the distress and functional impairment associated with OCD (Abramowitz, 2004). Accordingly, targeting OCD may be expected to also lead to reductions in symptoms of depression.

The present investigation also employed meta-analysis to examine the extent to which initial OCD and depression symptom severity was associated with outcome. Neither higher pre-treatment OCD symptom severity nor higher pre-treatment depression was significantly associated with a lower CBT effect size. In contrast to previous work (e.g., Foa et al., 1983; Keeley et al., 2008), the present findings combining 16 RCTs suggest that higher pre-treatment depression severity is not a robust predictor of treatment outcome. Rather than definitively concluding however that pre-treatment OCD symptoms and pre-treatment depression symptoms have no effect on treatment outcome, it may be that only severe OCD and depression hinders outcome. For example, Abramowitz et al. (2000) examined the relationship between pretreatment levels of depression and outcome of ERP for patients with OCD who had a wide range of depression severity. After grouping patients on the basis of their baseline depression levels, those with the most severe depression evidenced significantly lower rates of improvement with ERP compared to those who had moderate, mild, or no depression. Similarly, mild to moderate levels of depression may be predictive of treatment outcome among those with more severe OCD (Stewart et al., 2006).

Examination of the effect size as a function of placebo and waitlist controls revealed that CBT compared to waitlist

controls showed larger effect sizes than CBT compared to placebo controls, even after removing one study utilizing a pill placebo from the placebo control group. This finding is inconsistent with a recent meta-analysis of psychological treatments of OCD that found the effect size for the comparisons that used an active control group (psychological and or pharmacological placebo) did not significantly differ to that in studies which used a non-active, waiting-list control group (Rosa-Alcázar et al., 2008). Based on these findings, Rosa-Alcazar et al. also concluded that non-specific effects can be considered to be of a practically negligible magnitude and of no clinical relevance in the psychological treatment of OCD. Prior research has shown that patients with OCD are less likely to respond to placebo than patients with generalized social phobia or panic disorder (Huppert et al., 2004). One interpretation of the conflicting findings is that non-specific effects may influence treatment outcome in OCD to a lesser degree than other anxiety disorders, but that is not to say that non-specific effects have no influence on OCD treatment outcome. In fact, examination of the effect size for CBT compared to waitlist controls and that of CBT compared to placebo controls in the present investigation would suggest that the differences are far from negligible in magnitude.

Comparison of effect sizes as a function of two different treatment types: CT and ERP revealed no significant differences. This suggests that variants of CBT for OCD do not appear to differ from each other. This finding is largely consistent with prior meta-analytic studies that have found relatively equivalent effects for different CBT interventions for OCD, including CT and ERP (Abramowitz, 1997; Abramowitz et al., 2002; Eddy et al., 2004; Rosa-Alcázar et al., 2008). These equivalent findings have been difficult to interpret given that the different CBT techniques have been theorized to have their effects through distinct mechanisms. In ERP for example, a central mechanism is extinction (or habituation) resulting from systematic exposure (or decrease in avoidance) to fear-related stimuli and prevention of escape or neutralizing behaviors (Himle and Franklin, 2009). CT on the other hand is thought to have its desired effects by identifying, challenging, and modifying, underlying distorted beliefs that drive patients to perform compulsive rituals. Accordingly to some, the equivalent effects may be expected given non-specific factors are shared between the different CBT treatments (e.g., Messer and Wampold, 2002). However, this interpretation seems unlikely given that CBT outperforms placebo control conditions in the present investigation, a finding that has also been found in prior meta-analysis (Hofmann and Smits, 2008). An alternative view is that the different CBT interventions have equivalent outcomes because they incorporate similar specific treatment strategies. Like ERP, CT often involves the use of exposure ('behavioral experiments') designed to disprove irrational thoughts. Behavioral procedures in the form of exposure appear to constitute the critical ingredient in different forms of CBT for OCD (Deacon and Abramowitz, 2004), and this common critical ingredient may account for their equivalent effects.

Examination of the extent to which other sample-specific characteristics, study-specific characteristics, and treatment specific characteristics predicted outcome revealed very little robust effects. Specifically, the CBT effect size for OCD did not vary as a function of treatment format, treatment integrity assessment, blind assessment, age of onset, duration of symptoms, percentage of females, number of sessions, or percent comorbidity. The absence of the influence of methodological characteristics of the studies is especially notable as it casts doubt on the presence of a systematic bias in the effect size estimates. The absence of an association between percent comorbidity is also notable. Comorbidity in OCD may result in more dysfunction and lower quality of life. Accordingly, an intuitive prediction is that a higher percentage of diagnostic comorbidity should be associated with a reduction in CBT effect size. However, our findings are consistent with prior research showing that comorbidity is generally unrelated to CBT effect sizes for the anxiety disorders more broadly (Olatunji et al., 2010) and OCD more specifically (Rosa-Alcázar et al., 2008). Nevertheless, the extent to which specific comorbid diagnosis influences CBT treatment outcome for OCD requires further research. For example, Storch et al. (2008) found that the presence of comorbid disruptive behavior and attention-deficit/hyperactivity disorders had a particularly negative impact on treatment response among youth with OCD that received CBT. One interpretation of this finding is that while comorbidity in general may not influence treatment outcome, specific comorbid disorders that influence one's ability to attend and process information efficiently may.

Another intuitive prediction is that more therapy should be associated with an increase in CBT effect size. However, the president findings did not support this prediction as number of sessions was unrelated CBT effect sizes. This finding is largely consistent with prior research showing that the duration of treatment (in weeks), intensity of treatment (number of hours a week), and magnitude of treatment (total number of hours) was generally unrelated to treatment outcome (Rosa-Alcázar et al., 2008). This finding may have significant implications for treatment planning for patients with OCD. That is, there may be no added benefit to extensive sessions given that standard CBT for OCD can stay cost effective without losing efficacy. What may be necessary (and perhaps sufficient) to maximize gains is the application of the specific treatment procedures that is indicated for patients with OCD in a time sensitive manner while also ensuring adherence to those procedures (Simpson et al., 2011). Interestingly, Abramowitz (1996) did find that while the number of weeks in treatment, the number of treatment sessions per week, and the total number of sessions was unrelated to OCD treatment outcome, longer treatment sessions was associated with better outcomes. This finding raises the possibility that there may be some advantages to a more intensive approach to the treatment of OCD (Abramowitz et al., 2003).

The absence of an effect of sample-specific characteristics, study-specific characteristics, and treatment-specific characteristics on outcome may reflect a limitation of the moderation approach which was based on only 16 studies that combined different treatments. The moderation analysis was also derived from RCT samples that may not always be representative of the OCD population at large. This may be especially relevant for examining the extent to which depression predicts outcome. Indeed, RCTs often exclude very depressed patients (in order to maximize internal validity), which restricts the range of depression severity in these studies, potentially obscuring relationships with outcome. Despite the limitations of the moderation approach, a significant difference in effect size was seen between adult and child populations with child populations being associated with significantly larger effect sizes. Similarly, there was a significant relationship between effect size and mean age when including both adult and child studies, with older age associated with smaller effect sizes. This finding suggests that CBT may be more effective for child populations with OCD relative to adult populations with OCD. This is consistent with the view that children and adolescents with OCD respond well to CBT, and may be more likely to recover than adults (Huppert and Franklin, 2005). The stronger effect of CBT for children with OCD may be observed for several reasons. For example, Huppert and Franklin observe that a supportive family system may provide a greater incentive for children to follow treatment procedures. It may also be the case that children and adolescents are more malleable than adults in terms of changing their cognitions and behavior. Children and adolescents may spend less time than adults enriched in obsessive beliefs that contribute to the maintenance of OCD symptoms, making them more likely to benefit from treatment.

The present meta-analytic investigation suggests that CBT is effective in the treatment of OCD and that disorder-specific symptom reduction continue to be observed at follow-up. Although findings were robust against the 'file drawer problem', few data points were available for follow-up assessments. This is an important limitation of the literature on the efficacy of CBT for OCD and more treatment outcome research aimed at delineating the lasting effects of psychological treatments for OCD are needed. Few moderators of outcome were also observed in the present investigation. Regarding the important question posed by Paul (1967) of what treatment, by whom, is most effective for this individual with that specific problem, and under which set of circumstances, it may be the case that CBT is generally effective for most individuals under most circumstances. The finding that higher pre-treatment OCD symptom severity, pretreatment depression symptom severity, and percent comorbidity were not significantly associated with a decrease in effect size highlights the effectiveness of CBT for patients with wide range of symptom complexity. Accordingly CBT should translate well to community clinics where OCD patients with more symptom severity, more depression, and more comorbid conditions are likely to present. The present findings did reveal that CBT is perhaps more effective for children with OCD relative to adults with OCD. However, the prescriptive implication of this finding is less clear. Although CBT should clearly be a first-line treatment for children and adults with OCD, it remains unclear if a distinction between children and adults would predict a different pattern of outcomes between two, or more, treatment modalities. Unfortunately, the majority of RCTs that have been evaluated for the treatment of OCD have focused mainly on the efficacy of CBT. The availability of more RCTs examining the efficacy of non-CBT based OCD treatments will be valuable in identifying prescriptive variables that will facilitate individually tailored OCD treatment.

The exclusive focus on CBT treatments specifically also limits inferences that can be made about psychological treatments for OCD more broadly. The use of only well-controlled RCTs of CBT for OCD is perhaps another limitation of the study as it resulted in the availability of a relatively small number of studies for various comparisons. Although the use of well-controlled RCTs does allow for greater confidence in the present findings, there is a need for more well-controlled RCTs of CBT for OCD that will allow for more substantive examination of moderators of treatment outcome. However, the very nature of RCTs may render them ill-suited for detecting important predictors, especially if stricter eligibility criteria and more rigid delivery of treatment protocols eliminate the very factors that influence how OCD patients respond to treatment. Accordingly, effectiveness trials in community settings may have value in efforts to identify predictors of treatment outcome. The assessment of OCD as a unitary outcome is also a limitation of the present investigation. Prior research suggests that OCD consists of distinct symptom dimensions (Mataix Cols et al., 2005). Mataix-Cols et al. (2002) found that OCD patients with higher scores on the hoarding dimension were more likely to drop out prematurely from a randomized trial of CBT and to improve less than nonhoarding OCD patients. In a meta-analysis, patients with primary obsessive thoughts without rituals tended to improve less with CBT than those who had overt motor rituals (Christensen et al., 1987). Future research is needed to examine the extent to which prognostic and prescriptive moderators of CBT vary across the putative symptom dimensions of OCD.

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* Indicates studies used in the meta-analysis.