Available online at www.sciencedirect.com

ScienceDirect

Behavior Therapy 50 (2019) 165–176

Behavior Therapy

www.elsevier.com/locate/bt

Predictors and Moderators of Cognitive and Behavioral Therapy Outcomes for OCD: A Patient-Level Mega-Analysis of Eight Sites

Gail Steketee
Boston University

Jedidiah Siev
Swarthmore College

Iftah Yovel
The Hebrew University of Jerusalem

Keith Lit
Nova Southeastern University

Sabine Wilhelm
Massachusetts General Hospital and Harvard University Medical School

Cognitive (CT) and behavioral treatments (BT) for OCD are efficacious separately and in combination. Tailoring treatment to patient-level predictors and moderators of outcome has the potential to improve outcomes. The present study combined data from eight treatment clinics to examine the benefits of BT (n = 125), CT (n = 108), and CBT (n = 126), and study predictors across all treatments and moderators of outcome by treatment type. All three methods led to large benefits for OCD and depression symptoms. Residual gain scores for OCD symptoms were marginally smaller for BT compared to treatments containing CT. For depression, significantly more gains were evident for CBT than BT, and CT did not differ from either. Significantly fewer BT participants (36%) achieved clinically significant improvement compared to CT (56%), and this was marginally evident for CBT (48%). For all treatments combined, no predictors were identified in residual gain analyses, but clinically improved patients had lower baseline depression and stronger beliefs about responsibility/threat and importance/control of thoughts. Moderator analyses indicated that higher baseline scores on depression adversely affected outcomes for BT but not CT or CBT, and lower OCD severity and more education were associated with positive outcomes for CT only. A trend was evident for higher responsibility/threat beliefs to moderate clinical improvement outcomes for those receiving cognitive (CT and CBT), but not behavioral (BT) treatment. Medication status and comorbidity did not predict or moderate outcomes. Findings are discussed in light of models underlying behavioral and cognitive treatments for OCD.

This study was supported by a grant from the International OCD Foundation awarded to Dr. Wilhelm. The authors are grateful to the following colleagues who provided data for this study: Dr. Jonathan Abramowitz, Dr. Jean Cottraux, Dr. Michael Kyrios, Dr. Kieron O'Connor, Dr. Neil Rector, Dr. David Tolin, Dr. Maureen Whittal, and Dr. Sabine Wilhelm.

Data statement. All data files were shared with permission of the investigators of the 8 research sites, following IRB regulations regarding protection of human subjects. We do not have permission to share these data files with other investigators.

Address correspondence to Gail Steketee, Ph.D., Boston University School of Social Work, 264 Bay State Road, Boston, MA 02215; e-mail: steketee@bu.edu.

Keywords: obsessions; compulsions; cognitive therapy; exposure and response prevention

Obsessive-compulsive disorder (OCD) is chronic and prevalent, associated with distress, severe impairment, disability, and poor quality of life (Huppert, Simpson,

Nissenson, Liebowitz, & Foa, 2009; Kessler et al., 2005; Koran, Thienemann, & Davenport, 1996). Cognitive and behavioral treatments for OCD have proven efficacious in multiple studies (see McKay et al., 2015; Öst, Havnen, Hansen, & Kvale, 2015).

Exposure and response prevention (ERP), a form of behavior therapy (BT), is considered a first-line treatment for OCD (McKay et al., 2015) with response rates as high as 85% in patients who receive a complete course of treatment (Foa et al., 2005; Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000). ERP involves prolonged exposure in vivo and/or in imagery to obsessive stimuli without engaging in compulsions. Although efficacious, ERP can be emotionally challenging, and has been associated with moderate to high rates of treatment refusal and dropout (Abramowitz, Taylor, & McKay, 2009; Franklin & Foa, 2007; Öst et al., 2015), although a recent study suggests that ERP dropout rates may not differ from those for cognitive therapy (Ong, Clyde, Bluett, Levin, & Twohig, 2016).

Cognitive therapy (CT) is another form of therapy for OCD with a substantial evidence base and large effect sizes in individual treatment trials (e.g., Whittal, Robichaud, Thordarson, & McLean, 2008; Wilhelm et al., 2009; Wilhelm et al., 2005). CT for OCD typically follows Beck's model in which patients learn to identify and reconsider maladaptive ways of thinking (e.g., about responsibility for harm, overestimation of danger) that maintain their symptoms. CT commonly includes brief rather than lengthy exposures in the form of behavioral experiments that aim to test specific beliefs rather than produce habituation of discomfort, perhaps making them easier for some patients to tolerate (but see Ong et al., 2016).

Studies have also investigated the effects of combining cognitive and behavioral treatments (CBT), and a meta-analysis by Öst et al. (2015) indicated that CBT (defined somewhat loosely) performed well compared to waitlist, placebo, and medication treatments, although they noted that a number of studies had methodological problems. "CBT" is often used in the literature to refer to ERP and/or CT and/or their combination, based on the assumption that these treatments all contain components that alter both behavior and cognition, as well as emotional responses, with differences in emphasis. For the present paper, we define "CBT" as the deliberate combination of ERP plus CT (see below). To our knowledge, no direct comparisons of CBT (combined ERP + CT) to its individual components have been reported.

The ability to tailor individualized treatments is a primary motive to develop and test efficacious treatment alternatives, as not everyone benefits sufficiently from any given treatment approach. Moreover, some individuals may be unwilling to engage in a given treatment, and clinicians must consider patient values and preferences when offering treatment. By identifying predictors of outcome across treatments as well as moderators of response by treatment type, researchers may provide information about prognostic indicators that can guide treatment selection.

Several studies have examined predictors of outcome for BT for OCD (e.g., Abramowitz, 1997; Castle et al., 1994; de Araujo, Ito, & Marks, 1996; Foa et al., 1983; Kohls, Bents, & Pietrowsky, 2002; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002; Steketee, Chambless, & Tran, 2001). Unfortunately, results have been inconsistent, and small sample sizes for single studies have provided insufficient power to examine factors affecting treatment outcome adequately, especially when the variables studied have modest base rates among OCD patients (e.g., diagnosed comorbidity). These limitations are more problematic for CT because fewer studies have been conducted with OCD patients.

Three relatively recent meta-analyses of CBT methods for OCD have examined potential predictors and moderators of outcome, including therapy variables (e.g., duration) and several patient factors (e.g., age, gender, baseline symptom severity, symptom duration, comorbidity, medication status; Olatunji, Davis, Powers, & Smits, 2013; Öst et al., 2015; Rosa-Alcázar, Sánchez-Meca, Gómez-Conesa, & Marín-Martínez, 2008). Note that these studies differ somewhat in their inclusion/exclusion criteria (e.g., medication use), which may explain some differences in their findings. In addition, Keeley, Storch, Merlo, and Geffken (2008) provided a systematic review of studies of factors affecting outcome up to that time.

Among demographic variables, gender and age had no effect in two of the three meta-analyses, but Öst et al. (2015) found that studies with more women and older age showed lower treatment effects. None of these meta-analyses examined education level. Baseline OCD symptom severity had no impact in the Olatunji et al. (2013) study, but Öst et al. found that greater severity was associated with better outcome for CBT in placebo controlled trials. Duration of OCD symptoms showed no difference in the two studies that examined this variable.

Comorbidity studied at a relatively gross level (percent with comorbid conditions for Olatunji et al., 2013, and greater or less than 50% comorbidity within samples for Rosa-Alcázar et al., 2008) showed no significant effect in either analysis. Olatunji et al. found no impact of baseline depression on effect size.

However, this contrasts with reviews of individual and meta-analytic study findings by McKay et al. (2015) and Keeley et al. (2008), which point to baseline depression, especially severe depression, as a commonly reported harbinger of adverse outcome. These meta-analyses did not examine the effect of OCD beliefs on outcomes. Reporting on a variety of variables affecting BT (with or without CT) outcomes for OCD, Keeley et al. concluded that the limited research on cognitive variables permitted no firm conclusions, but they suggested that more cognitive distortions may predict worse outcome given their association with OCD symptoms.

Among treatment variables, all three meta-analyses found no significant differences in effect size on standard OCD measures for ERP and CT. Treatment duration had no effect in the two meta-analyses that examined this variable. Two of the three meta-analyses examined medication status. Öst et al. found that studies with more patients on antidepressants reported lower effect sizes, indicating that the addition of medications did not improve outcomes. This contrasts with nonsignificant findings for medication use in Rosa-Alcázar et al.'s (2008) research.

Like many other researchers, Keeley et al. (2008) pointed to methodological inconsistencies that hampered the interpretation of findings about factors affecting OCD treatment outcome. Accordingly, despite repeated efforts, relatively little is known about who benefits and who does not. The purpose of the present study was to examine combined data from eight OCD treatment clinics to identify potential factors that affect the outcome of cognitive and behavioral treatments. Data for this "mega-analysis" were provided by investigators who had collaborated closely over several years to develop assessments of OCD-related beliefs and who had strong shared interests in understanding the mechanisms and impacts of behavioral and cognitive therapies for OCD. Combining data from multiple sites provides more power to detect effects, expands sample diversity and potential generalizability of results, and helps address the problem (especially for CT studies) of detecting predictors and moderators with relatively low base rates. The ability to identify potentially important factors is substantially increased with more than 350 participants from these eight sites which employed remarkably consistent inclusion and exclusion criteria, diagnostic interviews, and intervention methods.

The eight clinics included in this study provided BT, CT, or combined CBT treatment (see definitions below) to clinic patients, most of whom participated in randomized controlled trials. Based on the existing literature on treatment efficacy (e.g., Öst et al., 2015; Olatunji et al., 2013; Rosa-Alcázar et al., 2008), we expected that all three treatment approaches would lead to similar levels of clinical improvement, and that moderators of outcome might vary, given the different theoretical models on which the behavioral and cognitive therapies were based (see McKay et al., 2015). A number of demographic, clinical, and treatment variables were examined as potential predictors and moderators based on prior literature. Given the somewhat disparate findings from meta-analyses reporting on treatment predictors and moderators, we did not test specific hypotheses. However, two primary questions were addressed for each potential factor:

1. Does this variable predict improvement in OCD symptoms across all treatment types?
2. Does this variable moderate change in OCD symptoms differently across treatments?

Methods

DESIGN AND SAMPLE

Pre- and posttreatment data were collected from eight participating research clinic centers that studied cognitive and behavioral treatments alone and/or in combination for outpatients diagnosed with OCD. Sites were chosen because they used similar manualized treatment procedures and identical outcome measures. Investigators could include data from participants treated in research trials and/or in outpatient clinic settings that employed comparable assessment and treatment protocols. Investigators who contributed existing data sets are listed in Table 1 along with the sample sizes for each treatment method. Altogether, 125 patients were treated with BT, 108 with CT, and 126 with CBT. Five sites (Cottraux, O'Connor, Rector, Whittal, Wilhelm) representing 75% of the total sample reported that their data came mainly from participants in pilot studies or randomized research trials, and three sites (Abramowitz, Kyrios, Tolin) provided data mainly from nonrandomized clinic patients who received a standard protocol.

All participants were adults (age > 18) who received an interview-based diagnosis of OCD according to DSM-IV criteria (American Psychiatric Association, 1994). All received at least 12 sessions of manualized BT, CT, or CBT from trained and supervised therapists. Patients were required to be stable on or off psychotropic medications prior to and during treatment. Participants were excluded if they had current suicidal or psychotic symptoms, mania or other features necessitating psychiatric hospitalization, active substance abuse or dependence, and evidence of intellectual disability or other severe cognitive dysfunction. The Cottraux study (see Cottraux et al., 2001) excluded patients with major depressive disorder (MDD) or

Table 1
Demographics and Descriptive Statistics by Treatment Type and Site

	N	Treatment Type (*n*)	Age	% Women	Years Education	Number Sessions	Pre Y-BOCS	Post Y-BOCS	Pre BDI	Post BDI
Treatment Type										
BT	125	n/a	35.82 (11.89)	55%	14.43 (2.79)	16.00 (3.82)	24.08 (5.96)	13.86 (7.91)	17.91 (10.66)	11.09 (10.68)
CT	108	n/a	35.33 (10.03)	72%	14.77 (2.56)	17.12 (4.52)	25.20 (5.12)	12.63 (8.87)	17.71 (11.06)	9.41 (9.20)
CBT	126	n/a	36.57 (11.34)	54%	14.16 (2.79)	18.13 (2.00)	23.83 (5.80)	11.90 (6.67)	16.23 (10.00)	7.53 (7.57)
All	359	n/a	35.93 (11.14)	60%	14.44 (2.72)	17.08 (3.66)	24.33 (5.67)	12.80 (7.84)	17.27 (10.56)	9.33 (9.32)
Treatment Site										
Abramowitz	13	BT	33.46 (11.89)	31%	14.92 (3.48)	16.00 (0.00)	23.92 (3.97)	9.69 (5.39)	15.69 (8.64)	7.77 (7.36)
Cottraux	60	BT (30) CT (30)	35.83 (10.68)	73%	13.97 (2.54)	20.00 (0.00)	27.50 (4.66)	16.17 (8.86)	16.53 (9.55)	10.67 (9.41)
Kyrios	43	CBT	36.02 (11.81)	61%	12.78 (2.97)	16.00 (0.00)	24.58 (7.28)	10.12 (6.62)	17.35 (12.51)	6.02 (6.45)
O'Connor	95	BT (12) CT (16) CBT (67)	38.34 (10.77)	56%	14.80 (2.34)	20.00 (0.00)	23.66 (5.24)	12.59 (7.05)	17.17 (10.23)	8.82 (9.42)
Rector	26	BT (10) CBT (16)	29.92 (9.29)	54%	14.88 (2.41)	16.00 (0.00)	22.23 (3.82)	15.50 (6.49)	17.92 (8.92)	13.23 (10.45)
Tolin	31	BT	39.90 (12.42)	52%	14.50 (3.25)	15.51 (2.80)	24.67 (6.59)	15.71 (7.80)	20.75 (12.01)	10.24 (10.65)
Whittal	59	BT (29) CT (30)	34.92 (10.44)	63%	14.03 (2.04)	10.76 (1.63)	22.59 (5.21)	10.37 (7.40)	17.79 (10.17)	10.49 (9.65)
Wilhelm	32	CT	32.78 (10.97)	62%	16.75 (2.48)	18.91 (3.76)	24.75 (5.84)	11.44 (8.91)	14.34 (10.30)	6.50 (8.31)

Note. BT = behavior therapy; CT = cognitive therapy; CBT = cognitive-behavior therapy; Y-BOCS = Yale-Brown Obsessive-Compulsive Scale; BDI = Beck Depression Inventory. For BDI analyses, $n = 114$ for BT, $n = 103$ for CT, and $n = 115$ for CBT.

Tourette's disorder; other studies permitted inclusion of these comorbidities.

MEASURES

Diagnostic Interviews

All sites used structured interviews to diagnose OCD and comorbid conditions. Most (65%) utilized the Structured Clinical Interview for DSM-IV-Patient Version (SCID-P; First, Spitzer, Gibbon, & Williams, 1995) and 21% completed the Anxiety Disorders Interview Schedule (ADIS; Brown, Di Nardo, & Barlow, 1994). The remaining 13% received another structured interview such as the Mini-International Neuropsychiatric Interview (MINI) or a detailed clinical interview and checklist based on DSM-IV criteria.

Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989). The Y-BOCS served as the primary outcome measure. This 10-item scale rates the severity of obsessions and compulsions with respect to time spent, interference, distress, resistance and control. This measure showed good interrater reliability for the total score ($r = .95$; Goodman et al., 1989) and good sensitivity to treatment effects (DeVeaugh-Geiss, Landau, & Katz, 1989). The self-report and interview forms of the Y-BOCS have been found to be comparable (e.g., Steketee, Frost, & Bogart, 1996). Seven sites used the interview format and one (Kyrios) used self-report.

Beck Depression Inventory (BDI). The BDI (Beck, Steer, & Garbin, 1988) is a 21-item self-report scale that focuses primarily on cognitive symptoms of depression which are rated from 0 to 3 with higher scores indicating greater severity. The BDI has shown good internal consistency in psychiatric and nonpsychiatric samples (mean α of 0.86 and 0.81, respectively) and good congruent validity with other measures of depression (Beck et al., 1988).

Obsessive Beliefs Questionnaire-44 (OBQ-44; OCCWG, 2005). This self-report measure assesses three types of OCD-related beliefs: responsibility/threat estimation (RT), importance/control of thoughts (ICT), and perfectionism/certainty (PC). The OBQ-44 has demonstrated good internal consistency across these subscales, and good criterion-

related validity in clinical and nonclinical samples (OCCWG, 2005). In the current combined sample, some participants completed the OBQ-44 whereas others completed the original 87-item OBQ; the latter was rescored to match the 44-item version.

TREATMENTS

Site investigators were invited to submit data for patients who received BT, CT, or CBT as defined below.

BT

Deliberate and prolonged exposure, often with the use of a hierarchy of feared situations, to obtain habituation of anxiety/negative emotions plus graduated or intensive efforts to prevent rituals and avoidance behaviors. Exposures and response prevention are conducted in sessions and assigned as homework. Informal cognitive restructuring may be included during exposures (e.g., regarding the likelihood of harm) but formal CT methods (see below) are not employed or are a very minor part of the therapy.

CT

Focus on stepping back from maladaptive thoughts and beliefs and developing a new perspective (i.e., cognitive restructuring) with some but not necessarily all of the following techniques: Socratic questioning, examining the evidence, monitoring thoughts and altering maladaptive thoughts to more accurate ones, taking another perspective, responsibility pie, calculating the probability of harm, using analogies and metaphors. Brief behavioral experiments may be included but prolonged exposure and planned response prevention is not.

CBT

Formal cognitive restructuring as defined for CT above, including using several of the techniques in a deliberate fashion, plus deliberate and prolonged exposure and response prevention as defined for BT above.

Treatment type was verified by an investigator report detailing the therapy protocol(s) for each site. Per report, all three treatment protocols also contained common elements including education and treatment rationale; identifying obsessions/rituals to target during treatment; self-monitoring of obsessions, rituals, and discomfort; between-session homework assignments; and relapse prevention training during final sessions. Study participants received individual outpatient treatment for an average of 17.1 sessions (range 7–22) over an average of 15 weeks (range 8–40). Therapists at all sites received extensive training before delivering treatment, as well as weekly supervision. Adherence to standard therapy procedures was monitored via video or audiotapes for 100% of CT patients, 72% of BT patients, and 93% of CBT patients. Differences in methods preclude analyses of fidelity across sites, but published reports from two sites indicated good fidelity (e.g., mean = 8.7 of 10 for Whittal and 4.1 of 5 for Wilhelm).

DATA ANALYSES

Baseline comparisons across sites and across treatment types for demographic, treatment, and symptom severity variables utilized chi-square analyses for categorical data and one-way ANOVAs with post-hoc comparisons via the least significant difference (LSD) method for continuous data. Paired-samples *t*-tests were used to examine pre-post treatment efficacy on OCD symptoms and mood. Two methods, residual gain scores and clinically significant change, were used to compare outcomes for BT, CT, and CBT and to examine predictors and moderators of change. These methods are described below.

Residual Gain (RG) Scores

RG scores control for initial pretreatment differences as well as measurement error over time and are useful for studying correlates of change in large samples because they rescale an individual's score relative to gains experienced by others at the same initial level (Manning & Dubois, 1962; Steketee & Chambless, 1992). RG scores were computed for each participant across the entire sample including all treatment conditions by converting pre- and posttreatment scores to Z scores and calculating relative change by subtracting the posttreatment score (Z_2) from the pretreatment score (Z_1) multiplied by the correlation (r_{12}) between scores at pre- and posttreatment (i.e., $RG = Z_1^* r_{12} - Z_2$). Higher scores reflect greater change.

RG scores for Y-BOCS and BDI scores were compared via one-way ANOVAs for BT, CT, and CBT. To detect potential predictors and moderators of change, first linear regressions were used to analyze predictor-group interactions with RG scores for Y-BOCS as the dependent variable. For continuous independent variables, we used hierarchical multiple regression with the centered predictor and treatment group entered in the first block, and the interaction terms entered in the second block to determine if predictor-by-group interaction terms added significantly to the variance accounted for over and above the main effects of the predictor and group. Simple effects were explored by calculating regression slopes within each treatment group when interactions were significant. For categorical independent variables, 2-way ANOVAs were used to detect significant interactions between variables and treatment groups.

Clinically Significant Change

Determination of clinically significant change for each participant was based on the 2-part method recommended by Jacobson, Roberts, Berns, and McGlinchey (1999) to determine (a) whether participants showed reliable change (Jacobson & Truax, 1991) indicating that their pre-post change scores exceeded random fluctuations due to measurement error, and (b) whether their posttest scores fell below clinical levels. Calculations for the reliable change index (RCI) used the standard error of difference (SE_{diff}) between these two scores, based on the standard error of measurement (SE_m) of the Y-BOCS. Because the Y-BOCS SE_m is not known, the standard deviation of the pretreatment measurement and the test-retest coefficient (r_{xx}) reported by Woody, Steketee & Chambless (1995) were employed as follows:

$$SE_m = SD \times \sqrt{1-r_{xx}} = 5.68 \times \sqrt{1-0.61} = 3.55$$

$$SE_{diff} = \sqrt{2 \times (SE_m)^2} = \sqrt{2 \times (3.55)^2} = 5.02$$

SE_{diff} was then multiplied by 1.96; change scores that exceeded this value (9.83) indicated reliable change. The second criterion, clinical change, was computed by subtracting 2 SD's (5.68 × 2) from the mean (24.33) of the pretreatment Y-BOCS scores for clinical OCD samples; posttreatment Y-BOCS scores that fell below this cutoff of 12.97 indicated symptoms below clinical levels. Per Jacobson et al. (1999), participants who met both criteria were classified as having made a clinically significant change: n = 165, 46.0% across the entire sample.

Between-group comparisons of the proportion of participants who achieved clinically significant change between each pair of treatment types used likelihood ratios estimated based on chi-square comparisons. Predictors and moderators of clinically significant improvement versus nonimprovement were examined using independent-samples t-tests and chi-square tests to compare groups.

Significance levels were set at $p < .05$ with two exceptions: (1) predictor analyses when one of the two outcome variables (RG, clinically improvement status) was significant and the other was marginal ($p < .10$) and (2) treatment comparisons and moderator analyses where sample sizes were smaller.

Results

SAMPLE CHARACTERISTICS

Analyses included 359 participants of whom 125 received BT, 108 received CT, and 126 received CBT. Table 1 provides demographic information and descriptive statistics for the three treatment types and the eight different sites. Of 242 (67%) participants for whom race/ethnicity data were available, a large majority were Caucasian (91%, n = 221). Of the 324 (90%) participants whose marital status was known, 147 (45%) were married or cohabitating, 144 (44%) were single and never married, and 33 (10%) were divorced, separated, or widowed. Employment status was reported for 265 (74%) participants, of whom 162 (61%) were employed, 93 (35%) unemployed, and 10 (4%) on disability. Medication status was reported for 268 participants (75%), of whom 158 (59%) received medications and 110 (41%) did not. The breakdown by treatment type for these 268 participants was as follows: for BT, 71% (n = 60) were on medications, 25 were not (40 missing data); for CT, 58% (n = 33) were taking medications, 24 were not (57 missing data); for CBT, 52% (n = 65) were on medications, 61 were not (no missing data).

PRETREATMENT ANALYSES

Pretreatment Differences by Site

For demographic variables no differences were detected for gender (p = .13), but significant differences emerged for age, $F(7, 349) = 2.91$, p = .01, η_p^2 = .06, education, $F(7, 327) = 7.05$, $p < .001$, η_p^2 = .13, and duration of OCD symptoms, $F(4, 175) = 5.11$, p = .001, η_p^2 = .11. Examination of means (see Table 1) indicated that the Rector sample was somewhat younger (30 versus 33–40 across other sites), and the Wilhelm sample had more education (16.8 years), compared to 13–15 years for other sites. Tolin's sample had a shorter duration of OCD compared to all other groups. Differences were also evident for the percent of patients taking medications (type not specified), $F(6, 261) = 9.76$, $p < .001$, η_p^2 = .18, with the majority on medications in 5 sites (68–100% for Abramowitz, Rector, Tolin, Whittal, Wilhelm) and a minority in 2 sites (39–44% for O'Connor and Kyrios). Data were not available for the Cottraux site.

ANOVAs comparing sites on symptom measures showed that pretreatment total Y-BOCS scores differed significantly across sites, $F(7, 351) = 4.51$, $p < .001$, η_p^2 = .08. Post-hoc LSD analyses indicated that this effect was due primarily to greater severity in the Cottraux sample (M = 27.50, SD = 4.66) compared to the other seven sites (means ranged from 22.23 for Rector to 24.75 for Wilhelm), ps < .04; there were no significant differences between any other pair of sites. Average pretreatment BDI scores did not differ by site, $F(7, 341) = 0.95$, p = .47, η_p^2 = .02, although the Cottraux site excluded patients with major depression. No site differences were detected for OBQ subscales (ps > .44).

Pretreatment Differences by Treatment Type

Baseline differences across treatment types were examined for demographic variables, symptom severity, and number of sessions. No differences emerged on age or years of education, but the sex ratio was more evenly balanced for BT (55% women) and CBT (54% women) relative to CT (72% women), $\chi^2(2) = 9.02$, $p = .01$. Pretreatment Y-BOCS and BDI scores did not differ by treatment type: Y-BOCS, $F(2, 356) = 1.91$, $p = .15$, $\eta_p^2 = .01$; BDI, $F(2, 346) = 1.10$, $p = .34$, $\eta_p^2 = .006$. No significant differences emerged for OBQ subscales, all ps > .25. The three treatment types differed significantly in the average number of sessions given, $F(2, 356) = 11.26$, $p < .001$, $\eta_p^2 = .06$. Individuals received more sessions of combined CBT ($M = 18.13$, $SD = 2.00$) than either BT ($M = 16.00$, $SD = 3.82$), $p < .001$, or CT ($M = 17.12$, $SD = 4.52$), $p = .03$, and CT had significantly more sessions than BT, $p = .02$.

EFFICACY OF TREATMENT TYPES

All three treatment types were highly efficacious and led to large reductions in OCD symptoms and depression as evident from Table 1. Pre-post t-test comparisons indicated that for BT, the average pre-post Y-BOCS reductions were 10.22, $t(124) = 15.15$, $p < .001$, $d = 1.39$; for CT average reductions were 12.57, $t(107) = 17.14$, $p < .001$, $d = 1.83$; and for CBT average reductions were 11.93, $t(125) = 19.50$, $p < .001$, $d = 1.75$. All three treatments significantly reduced depression according to t-tests on BDI scores[1]: for BT, average reductions in BDI were 6.82, $t(113) = 8.65$, $p < .001$, $d = 0.81$; for CT average improvement was 8.30, $t(102) = 11.62$, $p < .001$, $d = 1.18$; and for CBT mean improvement was 8.70, $t(114) = 9.37$, $p < .001$, $d = 0.89$.

TREATMENT COMPARISONS

Residual Gain Scores

Using the average total Y-BOCS residual gain scores as the dependent measure, a one-way ANOVA examining differences among the three treatment types was not significant, $F(2, 356) = 2.86$, $p = .06$, $\eta_p^2 = .02$. Given the p value of .06, we proceeded with post-hoc LSD analyses which revealed that total Y-BOCS residual gain scores were smaller for BT ($M = -.15$, $SD = .91$) compared to both CT ($M = .09$, $SD = .99$), $p = .04$, $d = 0.26$, and CBT ($M = .08$, $SD = .79$), $p = .04$, $d = 0.27$, with small effect sizes. The difference between CBT and CT was negligible, $p = .92$, $d = 0.01$. The pattern of differences for obsessions and compulsions separately was similar to that for the Y-BOCS total score.

[1] For BDI analyses, $n = 114$ for BT, $n = 103$ for CT, and $n = 115$ for CBT.

A similar comparison using BDI residual gain scores indicated a significant difference among treatments, $F(2, 329) = 3.77$, $p = .02$, $\eta_p^2 = .02$. Post-hoc LSD analyses revealed that CBT ($M = .14$, $SD = .80$) outperformed BT ($M = -.14$, $SD = .83$), $p = .007$, $d = 0.34$, with a small to medium effect. However, CT ($M = .03$, $SD = .64$) did not differ significantly from either CBT, $p = .29$, $d = 0.15$, or BT, $p = .11$, $d = 0.22$.

Clinically Significant Improvement

For the entire sample, 165 of 359 participants (46.0%) met criteria for clinically significant improvement (reliable change plus posttreatment scores in the nonclinical range); 105 met neither criteria and 89 met partial criteria. The breakdown for treatments was 45 of 125 (36.0%) met these criteria for BT, 60 of 108 (55.6%) for CT, and 60 of 126 (47.6%) for CBT. Comparisons of these proportions revealed that significantly more CT than BT participants showed clinical improvement, $\chi^2(1) = 8.95$, $p = .003$, and that rates for CBT were marginally greater than BT, $\chi^2(1) = 3.48$, $p = .06$. CT did not differ from CBT, $p = .23$.

PREDICTORS AND MODERATORS OF RESIDUAL GAIN SCORES

The following variables were examined: Baseline Y-BOCS, BDI and OBQ-44 scores, duration of OCD, medication status, presence of comorbid clinical disorders, number of treatment sessions, chronological age, education level, and gender. When all treatment groups were combined, none of these predictors were significant at $p < .05$ in RG analyses. Higher depression (BDI) and responsibility/threat beliefs (OBQ-RT) tended to predict worse outcome, ps < .09, but with small effects.

Among potential moderators of treatment type, significant interactions for residual gain scores were detected for pretreatment BDI (full model $F[5, 343] = 3.69$, $R^2 = .05$, $p = .003$, $F\text{-}change[2, 343] = 4.37$, $R^2\ change = .02$, $p = .01$), pretreatment Y-BOCS total (full model $F[5, 353] = 2.49$, $R^2 = .03$, $p = .03$, $F\text{-}change[2, 353] = 3.32$, $R^2\ change = .02$, $p = .04$), and years of education (full model $F[5, 329] = 2.50$, $R^2 = .04$, $p = .03$, $F[2, 329] = 3.54$, $R^2\ change = .02$, $p = .04$). Follow-up exploration indicated that higher levels of baseline depression (BDI) were associated with worse treatment outcomes in the BT group, $\beta = -0.26$, $p = .004$, $R^2 = .07$, but not in the CT or CBT groups. In contrast, higher initial OCD symptoms (Y-BOCS) were linked to worse outcomes in the CT group, $\beta = -0.19$, $p = .05$, $R^2 = .03$, but not in the BT or CBT groups. Finally, higher levels of education were associated with better treatment outcome only for those who received CT, $\beta = 0.23$, $p = .02$, $R^2 = .05$. Figure 1 illustrates these findings.

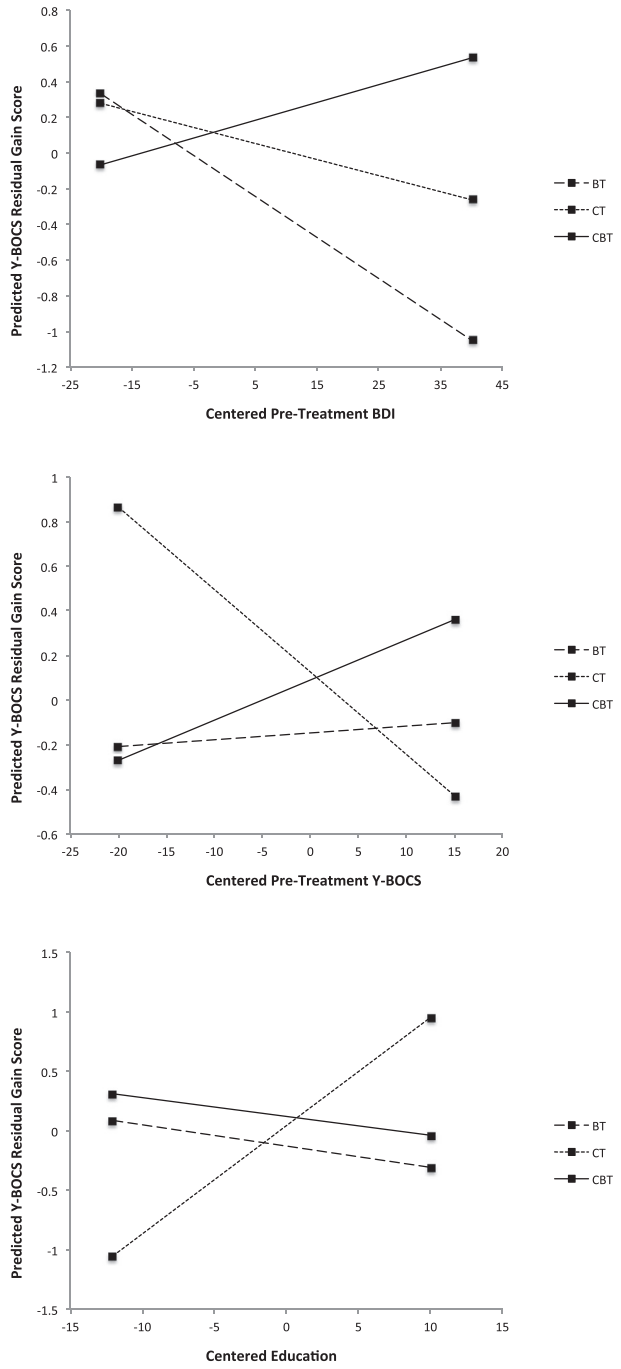

FIGURE 1 Graphs of significant interactions of baseline variables with outcomes of behavior therapy, cognitive therapy and combined cognitive behavioral treatment.

PREDICTORS AND MODERATORS OF CLINICALLY SIGNIFICANT IMPROVEMENT

In examining the same variables in relation to clinically significant improvement for all treatment groups combined, only pretreatment BDI and OBQ responsibility/threat and importance/control of thoughts were significant. Clinically improved patients averaged lower pretreatment BDI scores, t (261) = 2.62, p = .009, d = 0.33, and higher scores (stronger beliefs) for both OBQ-RT, t (132) = 2.05, p = .04, d = 0.36, and OBQ-ICT, t (132) = 2.00, $p < .05$, d = 0.36. Although other variables were not significant, nonsignificant trends with small effects were detected, suggesting that clinically improved patients had lower pretreatment Y-BOCS scores, t (268) = 1.78, p = .08, d = 0.22, and were more educated, t (255) = 1.69, p = .09, d = 0.22. For all other variables (age, sex, number of sessions, OCD duration, OBQ-PC), ps > .20.

Within-treatment-group analyses indicated that baseline BDI affected outcome differently across groups: Clinically improved patients who received BT had lower initial BDI scores than did those who were not, t (84) = 3.53, p = .001, d = 0.76, whereas baseline BDI was not associated with clinical improvement status among those who received CT or CBT, ps > .12. In addition, pretreatment Y-BOCS and education level operated differently across groups: Clinically improved patients in CT had lower pretreatment Y-BOCS scores, t (87) = 2.98, p = .004, d = 0.67, and were more educated, t (83) = 2.37, p = .02, d = 0.54. In contrast, neither variable affected clinical improvement among those treated with BT or CBT, ps > .50.

Additionally, nonsignificant trends with medium to large effects were found for OBQ responsibility/threat beliefs (RT): clinically improved patients in CT and CBT reported stronger baseline responsibility/threat beliefs, for CT, t (25) = 1.97, p = .06, d = 0.78, for CBT, t (78) = 1.85, p = .07, d = 0.45. OBQ-RT beliefs did not appear to be a factor for those who received BT, p = .49. Although a significant effect was found across treatments in the combined sample for beliefs about the importance and control of thoughts (OBQ-ICT), those beliefs did not distinguish clinically improved patients from any treatment type, ps > .11. No other pretreatment variables affected improvement status among participants who received CBT, all ps > .21.

Data about current medication status was examined for the subset of participants (n = 268) for whom data were available, among whom 59% were taking medications and the remainder were not. The proportion meeting both criteria for clinically significant improvement, 47% (n = 127), was very similar to the 46% who met these criteria for the entire sample; 27% (n = 73) met neither criterion. Both across and within the treatment conditions, medication status did not influence who achieved clinical improvement versus who achieved neither criterion, $\chi^2(1) < 0.34$, $p > .55$ for all analyses.

Axis I comorbidity data were available for 116 participants, of whom 45% (n = 52) had at least one comorbid disorder and 55% (n = 64) did not.

Within this subset, 39% ($n = 45$) met both criteria for clinically significant improvement, and 35% ($n = 41$) met neither. Across treatment conditions, the difference in clinical improvement rates between those with comorbidity (60%) and those without comorbidity (47%) was not significant, $\chi^2(1) = 1.33$, $p = .25$. Likewise, comorbidity status did not influence clinical improvement for any group, $\chi^2(1) < 0.94$, $p > .33$, for all analyses.

Discussion

The primary purpose of this study was to identify predictors of treatment response across interventions for OCD as well as moderators for BT, CT, and CBT using data from eight treatment clinics. Combining sites increased the sample size and appeared justified by the generally similar demographic features, with few exceptions. Baseline severity of OCD, depression and beliefs was also comparable across sites, with only one site (Cottraux) reporting higher Y-BOCS scores. Treatment outcomes were assessed via residual gain scores and achievement of clinically significant improvement, and several demographic, clinical, and treatment variables were analyzed as potential predictors and/or moderators of outcome.

In comparing the treatment efficacy of BT, CT, and CBT, we found large and significant decreases in OCD and depressive symptoms, consistent with numerous outcome studies (Abramowitz, 1997; Franklin et al., 2000; Franklin & Foa, 2007; McKay et al., 2015; Wilhelm et al., 2009; Wilhelm et al., 2005). Both CT and CBT led to slightly larger treatment gains compared to BT as indexed by changes on the Y-BOCS obsessions, compulsions, and total scores. Similarly, the percentage of patients who achieved clinically significant change with BT was significantly lower than with CBT and tended to be lower than CT. These results were surprising, given that previous studies have mainly found no differences between BT and CT (Olatunji et al., 2013; Öst et al., 2015; Rosa-Alcázar et al., 2008). Although the differences between treatments in the present study were small (ds = 0.26 - 0.27 for Y-BOCS total scores), they appear substantially different from the results of Öst et al.'s (2015) comprehensive meta-analysis, which found very small and nonsignificant differences between BT and CT (Hedges' $g = 0.07$ in favor of BT) across 37 published trials of CBT for OCD. As additional patients who followed standard protocols could be included in the data sets we collected, patients in the current study may have been drawn from a somewhat broader pool than those from published trials, so these findings may reflect a somewhat more community-based sample.

Pretreatment depression severity significantly predicted clinical improvement for combined treatments, and a trend was found in residual gain analyses. This differs from Olatunji et al.'s (2013) meta-analytic finding that baseline depression severity did not influence effect size. However, Keeley et al. (2008) noted that across the literature many studies show both that depression predicts behavioral treatment outcome and that it does not. They suggest that the conflicting findings may partly result from studies that excluded patients with major depression (only Cottraux's site did so in the current sample), noting that studies that examined the diagnosis of MDD consistently found a relationship to poor outcome.

Our findings also showed that depression had a significant impact only for those receiving BT, suggesting that CT or CBT may be more helpful for severely depressed OCD patients. Perhaps depression reduces motivation to engage in ERP more than for CT (see also Keeley et al., 2008), given the significant anxiety most patients experience during exposure treatment. Abramowitz and colleagues have suggested that depression may hinder habituation during BT and that patients with comorbid MDD may be more inclined to misinterpret intrusive thoughts (Abramowitz, Storch, Keeley, & Cordell, 2007). If so, interventions that include cognitive therapy may provide more benefit. In addition, the skills learned in CT for OCD (challenging distorted thinking about contamination and beliefs about the self and others) were developed originally for use with depressed patients (Beck et al., 1979) and may improve both depressive and OCD symptoms. ERP methods may not translate as directly to depressive symptoms. Overall, depression appears to be a complex factor that may affect outcomes differently for individual patients and treatment methods.

Stronger beliefs about responsibility and threat estimation also predicted clinically significant improvement in the total sample, and showed marginal findings in residual gain analyses. Although findings for within-treatment-group analyses were marginal, the medium to large effects suggested that among patients who received CT or CBT, those with higher responsibility/threat beliefs were more likely to improve clinically, whereas BT outcomes were not affected by these beliefs. From a clinical standpoint, BT appears to be a reasonable treatment regardless of the severity of such beliefs. It is also tempting to conclude that patients who overestimate risk may benefit particularly from cognitive treatments that directly target such beliefs, but such a conclusion would require a direct test of the relative efficacy of CT versus BT at different levels of OBQ-RT. Further study of these issues is needed to guide clinical decision-making.

Because data about these OCD-related beliefs were only available for a subset of our entire sample,

analyses of individual treatment groups were less than optimally powered, and therefore our interpretations are tentative and require replication. It is not clear why effects for beliefs were significant for responsibility/threat estimation but not for beliefs about the importance and control of thoughts and perfectionism/certainty. Perhaps it is related to the prominence of contamination and checking symptoms in our sample, which have been more strongly associated with beliefs about risk and threat estimation than with beliefs about the importance and control of thoughts (OCCWG, 2005; Taylor, McKay, & Abramowitz, 2005). Nonetheless, the absence of moderating effects for other belief domains in the current study remains a question for future research.

For combined treatments, baseline OCD severity assessed via Y-BOCS did not predict outcomes across treatments, but lower pretreatment OCD severity predicted clinically significant improvement, affecting CT patients, but not those receiving BT or CBT. This finding suggests that inclusion of exposure techniques may be particularly critical in the treatment of severe OCD. Additionally, more education predicted better overall outcomes in residual gain analyses and moderated outcomes, affecting those receiving CT but not BT or CBT. Perhaps a positive response to CT is more likely when patients have more verbal and critical thinking skills when starting treatment. Alternatively, more educated patients may simply be more comfortable with the Socratic learning process central to CT, and therefore may make gains more quickly in the time-limited treatments delivered in these clinical trials.

Variables that did not predict treatment response in the total sample or moderate outcomes for specific approaches included gender, number of sessions, duration of OCD, medication status, and the presence of a comorbid clinical disorder. Gender has rarely predicted or moderated outcome in other studies, and the number of sessions showed little variation, making it difficult to detect differences overall and by therapy method. While OCD duration and medication status are generally supported as predictors in the literature (Olatunji et al., 2013; Rosa-Alcázar et al., 2008), comorbidity apart from depression has been little studied. Rosa-Alcázar et al. (2008) found that more comorbidity was associated with larger treatment effects, but a single study accounted for their marginal finding, and Olatunji et al. (2013) did not find an association in their more recent meta-analysis. Unfortunately, despite the larger overall sample size, the data provided did not allow us to determine whether some comorbid conditions might be more problematic than others.

Our study has a number of limitations that are inherent in a mega-analysis in which data are combined across sites that did not deliberately coordinate assessment and treatment delivery. Such procedures undoubtedly varied somewhat across sites, although the comparability of studies with regard to pretreatment scores on standard measures and overall outcomes across trials is generally reassuring. All investigators were experienced clinical researchers from the U.S., Canada, Europe, and Australia who met frequently at international conferences to share clinical and research information about OCD. All were familiar with the Y-BOCS and with the administration of ERP and CT methods to OCD patients. Accordingly, while the absence of formal coordination introduces noise into the data, it also provides breadth that enhances the generalizability of findings.

Although the overall sample size was reasonably large for a clinical trial, it was notably smaller for individual treatment breakdowns (BT, CT, CBT) and for some measures for which there were missing data. We did not replace missing data for individual studies as our focus was on examining predictors for treatment completers rather than for intention-to-treat samples. Accordingly, power is not as high for analyses of some variables (e.g., OCD-related beliefs) as for others.

Unfortunately, reliability data were not available to verify OCD and comorbid diagnoses, nor was interrater reliability of Y-BOCS interviewers reported, even for the several published studies reporting findings for these participants. The lack of standardized interviews (SCID, ADIS) for a small portion of participants is an additional limitation. With regard to treatment fidelity, all site investigators reported that clinicians were well-trained and supervised weekly in the therapy protocol. Five of the eight sites reported assessing treatment adherence using various methods (e.g., therapist-rated postsession checklist, peer ratings of taped sessions, independent clinician ratings on a 10-point scale, blind independent clinician ratings of 30% of randomly selected sessions), but only two sites reported findings, both showing good fidelity (Whittal et al., 2008; Wilhelm et al., 2009). In addition, clinic procedures may have varied on other factors (e.g., emphasis on CT and ERP techniques, medication use) that affected outcomes.

Regardless of limitations, this mega-analysis combining data from multiple sites that employed similar assessments and treatments provided an avenue for investigating predictors and moderators of outcomes across and within treatments on moderately large samples. Findings pointed to the importance of higher baseline depression as potentially problematic across treatments but especially for

delivery of exposure and response prevention. Beliefs about responsibility and threat also appear to be important across treatments, but especially for cognitive treatments where more educated patients may benefit particularly. Understanding the mechanisms for these associations will require more investigation to identify mediators of these effects, but it seems likely that clinical judgment will be needed to determine how best to apply cognitive behavioral treatments to maximum effect.

Conflict of Interest Statement

The authors of the paper submit the following information about potential conflicts of interest:

Gail Steketee: Dr. Steketee was Co-PI on a grant funded by the International OCD Foundation (IOCDF) to Dr. Sabine Wilhelm to support this project. The funding enabled us to employ research assistants to gather and analyze the data. I am not aware of any specific bias with regard to outcomes due to the source of funding. She has received royalties from Elsevier Publications, Guilford Publications, New Harbinger Publications, Oxford University Press, and Houghton-Mifflin-Harcourt, as well as speaking honoraria from various academic institutions and foundations, including the funder IOCDF.

Jed Siev: No conflicts of interest.

Iftah Yovel: No conflicts of interest.

Keith Lit: No conflicts of interest.

Sabine Wilhelm: Dr. Wilhelm has received research support in the form of free medication and matching placebo from Forest Laboratories for clinical trials funded by the NIH. Dr. Wilhelm is a presenter for the Massachusetts General Hospital Psychiatry Academy in educational programs supported through independent medical education grants from pharmaceutical companies; she has received royalties from Elsevier Publications, Guilford Publications, New Harbinger Publications, and Oxford University Press. Dr. Wilhelm has also received speaking honorarium from various academic institutions and foundations, including the International Obsessive Compulsive Disorder Foundation and the Tourette Association of America. In addition, she received payment from the Association for Behavioral and Cognitive Therapies for her role as Associate Editor for the *Behavior Therapy* journal, as well as from John Wiley & Sons, Inc. for her role as Associate Editor on the journal *Depression & Anxiety*. Dr. Wilhelm has also received salary support from Novartis.

References

Abramowitz, J. S. (1997). Effectiveness of psychological and pharmacological treatments for obsessive-compulsive disorder: A quantitative review. *Journal of Consulting and Clinical Psychology*, *65*(1), 44–52. https://doi.org/10.1037/0022-006X.65.1.44

Abramowitz, J. S., Storch, E. A., Keeley, M. L., & Cordell, E. (2007). Obsessive–compulsive disorder with comorbid major depression: What is the role of cognitive factors? *Behaviour Research and Therapy*, *45*, 2257–2267. https://doi.org/10.1016/j.brat.2007.04.003

Abramowitz, J. S., Taylor, S., & McKay, D. (2009). Obsessive-compulsive disorder. *The Lancet*, *374*(9688), 491–499. https://doi.org/10.1016/S0140-6736(09)60240-3

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review*, *8*, 77–100. https://doi.org/10.1016/0272-7358(88)90050-5

Brown, T. A., Di Nardo, P. A., & Barlow, D. H. (1994). *Anxiety disorders interview Schedule for DSM-IV: Adult version*. New York: Oxford University Press.

Castle, D. J., Deale, A., Marks, I. M., Cutts, F., Chadhoury, Y., & Stewart, A. (1994). Obsessive-compulsive disorder: Prediction of outcome from behavioural psychotherapy. *Acta Psychiatrica Scandinavica*, *89*(6), 393–398. https://doi.org/10.1111/j.1600-0447.1994.tb01535.x

Cottraux, J., Note, I., Yao, S. N., Lafont, S., Note, B., Mollard, E., ... Dartigues, J. (2001). A randomized controlled trial of cognitive therapy versus intensive behavior therapy in obsessive compulsive disorder. *Psychotherapy and Psychosomatics*, *70*(6), 288–297. https://doi.org/10.1159/000056269

de Araujo, L. A., Ito, L. M., & Marks, I. M. (1996). Early compliance and other factors predicting outcome of exposure for obsessive-compulsive disorder. *British Journal of Psychiatry*, *169*(6), 747–752. https://doi.org/10.1192/bjp.169.6.747

DeVeaugh-Geiss, J., Landau, P., & Katz, R. (1989). Treatment of obsessive compulsive disorder with clomipramine. *Psychiatric Annals*, *19*(2), 97–99. https://doi.org/10.3928/0048-5713-19890201-11

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (1995). *Structured clinical interview for DSM-IV axis I disorders*. New York: New York State Psychiatric Institute.

Foa, E. B., Grayson, J. B., Steketee, G. S., Doppelt, H. G., Turner, R. M., & Latimer, P. R. (1983). Success and failure in the behavioral treatment of obsessive-compulsives. *Journal of Consulting and Clinical Psychology*, *51*(2), 287. https://doi.org/10.1037/0022-006X.51.2.287

Foa, E. B., Liebowitz, M. R., Kozak, M. J., Davies, S., Campeas, R., Franklin, M. E., . . . Tu, X. (2005). randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *162*, 151–161. https://doi.org/10.1176/appi.ajp.162.1.151

Franklin, M. E., Abramowitz, J. S., Kozak, M. J., Levitt, J. T., & Foa, E. B. (2000). Effectiveness of exposure and ritual prevention for obsessive-compulsive disorder: Randomized compared with nonrandomized samples. *Journal of Consulting and Clinical Psychology*, *68*(4), 594. https://doi.org/10.1037/0022-006X.68.4.594

Franklin, M. E., & Foa, E. B. (2007). Cognitive behavioral treatment of obsessive-compulsive disorder. In P. E. Nathan & J.M. Gorman (Eds.), *A guide to treatments that work* (3rd ed., pp. 431–446). New York: Oxford University Press.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., ... Charney, D. S. (1989). The Yale-Brown Obsessive Compulsive Scale: I. Development, use, and reliability. *Archives of General Psychiatry*, *46*(11), 1006–1011. https://doi.org/10.1001/archpsyc.1989.01810110048007

Huppert, J. D., Simpson, H. B., Nissenson, K. J., Liebowitz, M. R., & Foa, E. B. (2009). Quality of life and functional impairment in obsessive–compulsive disorder: A comparison of patients with and without comorbidity, patients in remission, and healthy controls. *Depression and Anxiety*, *26*(1), 39–45. https://doi.org/10.1002/da.20506

Jacobson, N. S., Roberts, L. J., Berns, S. B., & McGlinchey, J. B. (1999). Methods for defining and determining the clinical significance of treatment effects: Description, application, and alternatives. *Journal of Consulting and Clinical Psychology*, *67*, 300–307. https://doi.org/10.1037/0022-006X.67.3.300

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology*, *59*, 12–19. https://doi.org/10.1037/0022-006X.59.1.12

Keeley, M. L., Storch, E. A., Merlo, L. J., & Geffken, G. R. (2008). Clinical predictors of response to cognitive-behavioral therapy for obsessive–compulsive disorder. *Clinical Psychology Review*, *28*(1), 118–130. https://doi.org/10.1016/j.cpr.2007.04.003

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602. https://doi.org/10.1001/archpsyc.62.6.593

Kohls, S., Bents, H., & Pietrowsky, R. (2002). Predictors of success and failure in the behavior therapy of obsessive-compulsive disorders. *Verhaltenstherapie*, *12*(2), 98–107. https://doi.org/10.1159/000064373

Koran, L. M., Thienemann, M. L., & Davenport, R. (1996). Quality of life for patients with obsessive-compulsive disorder. *The American Journal of Psychiatry*, *153*(6), 783. https://doi.org/10.1176/ajp.153.6.783

Manning, W. H., & Dubois, P. H. (1962). Correlational methods in research on human learning. *Perceptual and Motor Skills*, *15*, 287–321. https://doi.org/10.2466/pms.1962.15.2.287

Mataix-Cols, D., Marks, I. M., Greist, J. H., Kobak, K. A., & Baer, L. (2002). Obsessive-compulsive symptom dimensions as predictors of compliance with and response to behaviour therapy: Results from a controlled trial. *Psychotherapy and Psychosomatics*, *71*(5), 255–262. https://doi.org/10.1159/000064812

McKay, D., Sookman, D., Neziroglu, F., Wilhelm, S., Stein, D. J., Kyrios, M., Matthews, K., & Veale, D. (2015). Efficacy of cognitive-behavioral therapy for obsessive–compulsive disorder. *Psychiatry Research*, *225*(3), 236–246. https://doi.org/10.1016/j.psychres.2014.11.058

Obsessive Compulsive Cognitions Working Group. (2005). Psychometric validation of the Obsessive Belief Questionnaire and Interpretation of Intrusions Inventory—Part 2: Factor analyses and testing of a brief version. *Behaviour Research and Therapy*, *43*(11), 1527–1542. https://doi.org/10.1016/j.brat.2004.07.010

Olatunji, B. O., Davis, M. L., Powers, M. B., & Smits, J. A. (2013). Cognitive-behavioral therapy for obsessive-compulsive disorder: a meta-analysis of treatment outcome and moderators. *Journal of Psychiatric Research*, *47*(1), 33–41. https://doi.org/10.1016/j.jpsychires.2012.08.020

Ong, C. W., Clyde, J. W., Bluett, E. J., Levin, M. W., & Twohig, M. P. (2016). Dropout rates in exposure with response prevention for obsessive-compulsive disorder: What do the data really say? *Journal of Anxiety Disorders*, *40*, 8–17. https://doi.org/10.1016/j.janxdis.2016.03.006

Öst, L.-G., Havnen, A., Hansen, B., & Kvale, G. (2015). Cognitive behavioral treatments of obsessive–compulsive disorder. A systematic review and meta-analysis of studies published 1993–2014. *Clinical Psychology Review*, *40*, 156–169. https://doi.org/10.1016/j.cpr.2015.06.003

Rosa-Alcázar, A. I., Sánchez-Meca, J., Gómez-Conesa, A., & Marín-Martínez, F. (2008). Psychological treatment of obsessive–compulsive disorder: A meta-analysis. *Clinical Psychology Review*, *28*(8), 1310–1325. https://doi.org/10.1016/j.cpr.2008.07.001

Steketee, G., & Chambless, D. L. (1992). Methodological issues in predicting treatment outcome. *Clinical Psychology Review*, *12*, 387–400. https://doi.org/10.1016/0272-7358(92)90123-P

Steketee, G., Chambless, D. L., & Tran, G. Q. (2001). Effects of axis I and II comorbidity on behavior therapy outcome for obsessive-compulsive disorder and agoraphobia. *Comprehensive Psychiatry*, *42*(1), 76–86. https://doi.org/10.1053/comp.2001.19746

Steketee, G., Frost, R., & Bogart, K. (1996). The Yale-Brown Obsessive Compulsive Scale: Interview versus self-report. *Behaviour Research and Therapy*, *34*(8), 675–684. https://doi.org/10.1016/0005-7967(96)00036-8

Taylor, S., McKay, D., & Abramowitz, J. S. (2005). Hierarchical structure of dysfunctional beliefs in obsessive-compulsive disorder. *Cognitive Behaviour Therapy*, *34*(4), 216–228. https://doi.org/10.1080/16506070510041167

Whittal, M. L., Robichaud, M., Thordarson, D. S., & McLean, P. D. (2008). Group and individual treatment of obsessive-compulsive disorder using cognitive therapy and exposure plus response prevention: A 2-year follow-up of two randomized trials. *Journal of Consulting and Clinical Psychology*, *76*, 1003–1014. https://doi.org/10.1037/a0013076

Wilhelm, S., Steketee, G., Fama, J. M., Buhlmann, U., Teachman, B. A., & Golan, E. (2009). Modular cognitive therapy for obsessive-compulsive disorder: A wait-list controlled trial. *Journal of Cognitive Psychotherapy*, *23*(4), 294–305. https://doi.org/10.1891/0889-8391.23.4.294

Wilhelm, S., Steketee, G., Reilly-Harrington, N. A., Deckersbach, T., Buhlmann, U., & Baer, L. (2005). Effectiveness of cognitive therapy for obsessive-compulsive disorder: An open trial. *Journal of Cognitive Psychotherapy*, *19*(2), 173–179. https://doi.org/10.1891/jcop.19.2.173.66792

Woody, S. R., Steketee, G., & Chambless, D. L. (1995). Reliability and validity of the Yale-Brown Obsessive–Compulsive Scale. *Behaviour Research and Therapy*, *33*, 597–605. https://doi.org/10.1016/0005-7967(94)00076-V

Received: October 24, 2017
Accepted: April 12, 2018
Available online: 18 April 2018