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Behavioral Versus Cognitive Treatment of Obsessive-Compulsive Disorder: An Examination of Outcome and Mediators of Change

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Objective: To examine symptom change over time, the effect of attrition on treatment outcome, and the putative mediators of cognitive therapy (CT) versus behavior therapy (BT) for obsessive-compulsive disorder (OCD) using archival data. ***Method:*** Sixty-two adults with OCD were randomized to 20 sessions of CT ($N = 30$) or BT ($N = 32$) that consisted of 4 weeks of intensive treatment (16 hr total) and 12 weeks of maintenance sessions (4 hr). Independent evaluators assessed OCD severity using the Yale–Brown Obsessive Compulsive Scale (Y-BOCS) at baseline and at Weeks 4, 16 (posttreatment), 26, and 52 (follow-up). Behavioral avoidance, depressive symptoms, and dysfunctional beliefs regarding responsibility were also measured at each assessment. Study hypotheses were tested using multilevel modeling. ***Results:*** The slope of change in Y-BOCS scores was significantly greater in BT than in CT ($d = 0.69$), and those receiving BT had lower Y-BOCS scores at the final assessment than those receiving CT ($d = 1.17$). The greater slope of change in BT versus CT did not differ for dropouts versus completers. Reduction in depressed mood mediated changes in Y-BOCS across the 2 treatments, but a reduction in sense of responsibility and a decrease in avoidance did not. Instead, Y-BOCS improvements appeared to precede a decrease in avoidance. ***Conclusions:*** BT may have some therapeutic advantage over CT in the treatment of OCD, and this advantage does not appear to be due to a differential pattern of responding for treatment dropouts versus completers. Further, inconsistent with hypotheses, improvements in OCD symptoms were mediated by reductions in depressed mood instead of decreases in avoidance and responsibility. Theoretical, methodological, and clinical implications are discussed.

Keywords: obsessive-compulsive disorder, cognitive therapy, behavior therapy, exposure and response prevention, mediation

According to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000), obsessive-compulsive disorder (OCD) is an anxiety disorder characterized by recurrent obsessions or compulsions that cause marked distress and interfere with daily functioning. OCD has a lifetime prevalence rate of 2–3% in the general population (Kessler et al., 2005). Its onset is usually gradual, and, if left untreated, the course can be chronic, resulting in lower quality of life (Mataix-Cols et al., 2002; Olatunji, Cisler, & Tolin, 2007). The debilitating nature of OCD has led to increased efforts to identify empirically supported psychological treatments that can be broadly disseminated.

Currently, behavior therapy (BT) that focuses on exposure and response prevention (ERP; Greist et al., 2003) is the psychological treatment of choice for OCD (National Institute for Health and Clinical Excellence, 2006). BT is based on principles of learning; it derives from the theory that compulsive behavior is performed to avoid obsessional anxiety and is negatively reinforced by the reduction in distress that it engenders (Mowrer, 1951). The behavioral treatment derived from this theory involves gradual prolonged exposure to fear-eliciting stimuli or situations combined with instructions to abstain from compulsive behavior. The effectiveness of BT for OCD has been well established with approximately 75% of patients achieving responder status (Franklin & Foa, 2002). The efficacy of BT for OCD has also been demonstrated in several meta-analyses (Abramowitz, 1996, 1997; Eddy, Dutra, Bradley, & Westen, 2004; Rosa-Alcázar, Sánchez-Meca, Gómez-Conesa, & Marín-Martínez, 2008), and this line of research has shown that treatment gains often persist after treatment

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discontinuation (Abramowitz, 1998; Whittal, Robichaud, Thordarson, & McLean, 2008).

Although BT is widely regarded as a first-line psychological treatment for OCD (Olatunji, Cisler, & Deacon, 2010), it has been posited that cognitive approaches to treatment may offer promise because they target variables theorized to operate in the maintenance of the disorder, many of which are not specifically targeted in BT (Wilhelm et al., 2009). The cognitive model of OCD (J. S. Beck, 1995; Rachman, 1997; Salkovskis, 1989) suggests that the distinguishing characteristic of those with OCD is not the experience of intrusive thoughts per se but, rather, dysfunctional beliefs about the presence or significance of the intrusive thoughts. A prominent cognitive model of OCD is that inflated perceptions of responsibility, the belief that one has power to bring about or prevent crucial negative outcomes, accounts for the development and maintenance of OCD (Salkovskis, 1985; Salkovskis, Shafran, Rachman, & Freeston, 1999). Based on this formulation, cognitive therapy (CT) may have its desired effect by reducing perceptions of responsibility. Initial treatment outcome findings have provided supportive evidence for the efficacy of CT for OCD (Van Oppen et al., 1995; Wilhelm et al., 2005, 2009). Such findings have naturally raised questions regarding the relative efficacy of BT versus CT for OCD as well as regarding the key mediators underlying the OCD symptom improvement seen with these approaches.

Relative Efficacy of BT Versus CT

With respect to the question of whether BT has superior efficacy compared to CT, investigations have yielded mixed results. A number of studies comparing BT to CT have revealed no differences (Cottraux et al., 2001; Emmelkamp, Visser, & Hoekstra, 1988; Emmelkamp & Beens, 1991), although some have shown an advantage of BT over CT (e.g., McLean et al., 2001), and others have indicated an advantage of CT over BT (e.g., Van Oppen et al., 1995). Meta-analytic studies have generally reported comparable effect sizes for BT and CT for OCD (Eddy et al., 2004; Rosa-Alcázar, et al., 2008). Research employing more conservative criteria for determining efficacy, on the other hand, appears to favor BT over CT. For example, Fisher and Wells (2005) noted that BT and CT are roughly equivalent in terms of efficacy when the asymptomatic criterion (i.e., has the patient "recovered" from OCD?) is used as the index of outcome, but when using the conservative criterion for clinical significance put forth by Jacobson et al. (Jacobson, Follette, & Revenstorf, 1984; Jacobson & Revenstorf, 1988; Jacobson & Truax, 1991), BT outperforms CT.

Does this slight advantage of BT over CT suggest that these therapies have relatively similar outcomes, or is the lack of a clear difference between them due to the statistical and/or methodological limitations of the studies investigating their differential outcomes? Many of the studies had very small sample sizes (e.g., 18 in Emmelkamp et al., 1988; 21 in Emmelkamp & Beens, 1991) and therefore would only have been able to detect very large effect sizes. Many performed completer analyses (e.g., Emmelkamp & Beens, 1991; Van Oppen et al., 1995) dropping any participant with any missing data, which may have biased their results and reduced their power. Thus, even if these studies detected differences between treatments, it would be impossible to know if these results could be generalized to a greater population of OCD patients. Other studies used the last observation carried forward (LOCF; Cottraux et al., 2001; McLean et al., 2005) method, which assumes that the outcomes for dropouts would not have changed if they had continued in the study. This assumption is generally false (Hamer & Simpson, 2009) and therefore may bias the results (e.g., if treatment differences increase over time, LOCF underestimates treatment differences). We know of no studies in this area that used mixed effects regression models (referred to as multilevel models [MLM]), the preferred method to analyze psychiatric data (Hamer & Simpson, 2009).

There are several advantages of MLM over traditional statistical methods (e.g., analysis of covariance, chi-square, multivariate analysis of covariance) in treatment outcome research (see Hamer & Simpson, 2009). First, MLM includes *all* participants, regardless of missing data. Thus, it does not require external imputation of missing data and hence avoids a major problem with LOCF, which imputes all missing data with the last observation, thereby introducing potential bias into the data. Indeed, consider the possibility that differences between BT and CT increase over time. In such case, LOCF may underestimate the difference between the two treatments in outcome, thereby increasing the likelihood of a null finding. Second, MLM assumes that data are missing at random (MAR), a much less stringent criterion than assuming that data are missing *completely* at random (MCAR), which is the assumption of traditional statistical methods and is rarely valid. Third, in contrast to traditional statistical methods, which base conclusions on a single data point at the end of the study, MLM uses all data points from all participants to plot individual change trajectories, thus providing more accurate and less variable estimates of the participant's overall improvement (Kraemer & Thiemann, 1989; Singer & Willett, 2003). In sum, by incorporating all data points from all participants, assuming less about the missing data, and avoiding data imputation methods that may bias the data, MLM generally provides more accurate estimates of treatment effects and more powerful tests of treatment differences (e.g., Quené & van den Bergh, 2004).

Differences between treatments can also be obscured when the model of symptom change does not correspond to the actual pattern of change (Liu, Rovine, & Molenaar, 2012). More specifically, symptoms tend to decrease rapidly during treatment and then level off over time during follow-up (i.e., approach an asymptote). Therefore it is possible that an exponential model (which models asymptotic curves) would yield between-group differences that would not be observed when data are analyzed using simple linear models (which do not correspond to the trajectory of symptom change). Accordingly, one aim of the present study is to investigate whether the lack of significant differences between BT and CT in a previously published study that employed LOCF and traditional statistical linear analyses will hold when these data are analyzed using MLM and nonlinear models.

Mediators of Therapeutic Change

With respect to identifying treatment mediators, the body of literature is small and findings are inconsistent. That is, although reducing behavioral avoidance in BT (Kirkby et al., 2000) and changing cognitions about inflated responsibility in CT (Ladouceur, Leger, Rheaume, & Dube, 1996) are both associated with reductions in OCD symptoms, the extent to which changes in behavioral approach and inflated responsibility account for symp-

tom improvement in BT and CT, respectively, is unclear. Interestingly, there is evidence that BT engenders cognitive changes that are associated with OCD symptom improvement (Emmelkamp, Van Oppen, & Van Balkom, 2002). Jónsson, Hougaard, and Bennedsen (2011) also recently found that change in inflated responsibility during a combination treatment of BT and CT remained significantly associated with OCD symptom change when controlling for change in depressive symptoms. In addition, Woody, Whittal, and McLean (2011) tested various cognitive mediators of changes in obsessions during CBT and found that only inflated responsibility emerged as a unique and significant predictor of change in obsessive symptoms, among their cognitive measures. However, further longitudinal analyses unexpectedly indicated that changes in symptoms led to changes in responsibility, but not vice versa. Taken together, these findings suggest that, although reductions in sense of responsibility may be related to change in OCD treatment, it may be the *result* of improvements in severity rather than a *cause* of such improvements.

The results from the Woody et al. (2011) study also highlight the importance of using more advanced statistical and methodological approaches to investigate the mechanisms of change during OCD treatment. Traditional mediation analyses (like the frequently used Baron & Kenny, 1986, approach) typically examine cross-sectional data and model a single mediator. Their model is reflected by the following regression equations:

$$Y = i_1 + c * T + \varepsilon_1$$

$$Y = i_2 + c' * T + b * M + \varepsilon_2$$

$$M = i_3 + a * T + \varepsilon_3,$$

where Y is the outcome, T is the independent variable (e.g., treatment), M is the mediator, i_j is the intercept, and ε_j is the error.

More recent extensions of Baron and Kenny (1986; e.g., the MacArthur approach; Kraemer, Kiernan, Essex, & Kupfer, 2008) have recognized the importance of establishing temporal precedence as an eligibility criterion for a mediator and of considering the possibility that the independent variable and the mediator may interact to affect the outcome. However, it is widely acknowledged that traditional, cross-sectional, single mediator mediation models do not demonstrate whether the mediator-to-outcome relation is due to the mediator causing changes in the outcome, or if the outcome causes changes in the mediator, or if their relation is due to other third variables (Kazdin, 2007; Kazdin & Nock, 2003; Smits, Julian, & Rosenfield, 2012; Smits, Rosenfield, McDonald, & Telch, 2006). Further, between-subjects mediation analyses (as is typical in the Baron & Kenny and MacArthur approaches), which tell us whether a mediator accounts for differences between treatment conditions, provide little insight into the mediators of *within-subject change over time* (Maxwell & Cole, 2007). Thus, more advanced mediation approaches are necessary to enhance causal inference and to derive information about what drives within-subject change over time.

Longitudinal mediation analysis (see MacKinnon, 2008) is a necessary, but not sufficient, condition to address these issues. With longitudinal data one can derive mediators of within-subject change over time, and one can use lagged analyses to establish temporal precedence. However, causal inference requires more stringent criteria. Building on the counterfactual framework (e.g., Rubin, 1974), Imai, Keele, and Tingley (2010) have shown that although randomization allows causal inference for the *a* (i.e., treatment to mediator) paths in mediation, randomization does not provide for causal inference on the *b* (i.e., mediator to outcome) paths. To establish causation for the *b* path, Imai et al. state that, "among those who share the same treatment status and the same pretreatment characteristics, the mediator can be regarded as if it were randomized (p. 313)." This criterion is rarely met, since individuals with different scores on the mediator generally have different scores on other potential confounds. Therefore, to enhance causal inference, it is critical to control for as many relevant confounders as is practical. Such relevant confounders should include other specific possible mediators as well as other nonspecific third variables that may be related to the proposed mediator (Smits et al., 2012). In addition, since we are conceptualizing a lagged mediation analysis, one should also control for the lagged effect of the previous level of the outcome, given that individuals who have different prior levels of the mediator may also have different prior levels of the outcome. Controlling for prior levels of the outcome has the added benefits of (a) partially controlling for other unmeasured confounds that may be related to both the mediator and the outcome and (b) controlling for the possibility that the outcome causes the mediator rather than vice versa.

In sum, to enhance causal inference about within-subject mediation of change over time, one should examine (a) longitudinal data and (b) lagged effects of the mediator on the outcome, (c) control for other putative mediators and third variables, and (d) control for lagged effects of the outcome (autoregressive effects).

Study Aims

The present study sought to build upon the previously reviewed work in a number of meaningful ways. First, we reanalyzed previously published data (Cottraux et al., 2001), utilizing more advanced and appropriate statistical methods (Hamer & Simpson, 2009; Kraemer & Thiemann, 1989) to add to the literature on the comparative efficacy of BT and CT for OCD. Specifically, instead of utilizing the last observation carried forward (LOCF) and endpoint analyses, which had indicated no significant differences between the BT and CT in the previous article (Cottraux et al., 2001), we employed MLM to analyze the data. In addition, rather than modeling linear change, which does not correspond to the pattern of change that is typically observed from the time that persons begin treatment until the time they present for a follow-up assessment, we examined various growth curve models (i.e., linear, quadratic, and exponential) to determine which model most accurately reflected the change in symptoms over time. Since we hypothesized that symptoms would rapidly decrease during treatment and approaching an asymptote during the follow-up period, we expected that an exponential model would most appropriately model the growth curve. Next, we investigated the nature of the missing data by incorporating pattern mixture modeling into our MLM analyses. Pattern mixture modeling does not assume that the data are MAR or MCAR but, by allowing for different growth curves for those with and without missing data, specifically investigates whether patients with missing data have different patterns of response than those without missing data (Enders, 2011; Hedeker & Gibbons, 2006).

Second, we examined the mediators of change in OCD symptoms, using a methodology that incorporated recent advances in mediation modeling, which we described above. We investigated the importance of avoidance and reductions in the sense of responsibility as putative agents of change in BT and CT. Consistent with theory, we hypothesized that reductions in avoidance would mediate OCD symptom improvements in BT, whereas reductions in sense of responsibility would mediate OCD symptom improvements in CT. From a behavioral perspective, avoidance behaviors are negatively reinforcing in that they maintain OCD symptoms in the long-term by preventing fear extinction (see Himle & Franklin, 2009). Foa and Kozak (1986) have also posited that prolonged exposure to threat cues to facilitate habituation is crucial to maximizing treatment outcome. This view also stresses preventing avoidance to reduce anxiety to previously feared cues, which makes it easier in the long run to resist compulsive urges. The effectiveness of BT for OCD then is based on the assumption that, by preventing avoidance, anxiety diminishes through the process of fear extinction. Indeed, ERP was largely derived from early learning models of OCD that identified avoidance as a crucial symptom maintaining mechanism (Dollard & Miller, 1950; Mowrer, 1951).

From a cognitive perspective however, changes in excessive responsibility may account for symptom reduction in CT, since beliefs that one has the power to bring about or prevent crucial negative outcomes can maintain OCD (Salkovskis, 1985; Salkovskis et al., 1999; Woody et al., 2012). We also included depression as a rival nonspecific mediator to avoidance and responsibility because it figures prominently in cognitive behavioral theories of the maintenance of OCD (e.g., Barlow, 2002), frequently accompanies OCD (e.g., Nestadt et al., 2001), and has been shown to be associated with treatment outcome (e.g., Overbeek, Schruers, Vermetten, & Griez, 2002). Including depression in our analyses allowed us to determine whether avoidance and responsibility were related to Y-BOCS over and above their relation with depression or only because they may have been related to depression.

In order to meet the four criteria enumerated previously to enhance causal inference in our mediation analysis, we modeled all three possible mediators (responsibility, avoidance, and depression) simultaneously using a longitudinal panel mediation analysis (see Seidel, Presnell, & Rosenfield, 2009; Tschacher & Ramseyer, 2009, for other studies using a similar approach). This approach uses prior levels of the mediators and outcomes (at the previous time point) to predict the mediators and outcomes at the next time point (see Figure 3). As noted above, this model incorporates temporal precedence and reduces bias in the estimates of the *b* paths by controlling for other potential mediators and controlling for reverse causality. It also provides additional data about the processes of change by simultaneously testing the effects of the outcome on the potential mediators and examining the effects of the mediators on the outcome.

Method

Participants

Patients ($N = 62$) from a study previously published by Cottraux et al. (2001) met diagnostic criteria for OCD based on a structured interview specifying the *DSM–IV* criteria. They were recruited from university hospitals and were included in the study if they scored 7 or more on the National Institute of Mental Health Obsessive-Compulsive Scale (Insel et al., 1983) and 16 or more on the Yale–Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman et al., 1989). Exclusion criteria included the presence of psychosis, Tourette syndrome, addiction, pregnancy, major depression and/or a Hamilton Depression Scale (Hamilton, 1960) score above 20, or suicidal ideation. Patients were 18–65 years of age and did not take psychotropic medication, apart from hypnotic drugs. The present study was approved by an ethics committee (CCPPRB, Lyon B), and all participants signed an informed consent form prior to participation.

Measures

Yale-Brown Obsessive-Compulsive Scale (Y-BOCS). The primary outcome was OCD severity as measured by the Y-BOCS (Goodman et al., 1989). The Y-BOCS is an assessor-rated measure of symptom severity for OCD and total scores on the Y-BOCS range from 0 to 40. The Y-BOCS has good internal consistency (Chronbach's alphas = .88–.91) and excellent interrater reliability (.98; Goodman et al., 1989). Internal consistency (Chronbach's alpha) in the current sample was .78.

Behavioral Avoidance Test (BAT). The BAT consisted of four homework assignments given by an independent assessor to examine behavioral avoidance (range 0–4) when confronting feared situations that are consistent with the patients OCD symptoms. The BATs were designed by the therapist in collaboration with the patient and were administered by an independent assessor that was blind to the treatment condition. This BAT format was adopted from prior research (Marks, Hallam, Connolly, & Philpott, 1977) and has been used in several treatment trials for anxiety disorders (for a review, see Marks, 1987), where it has been shown to demonstrate sensitivity to OCD treatment (Cottraux, Messy, Marks, Mollard, & Bouvard, 1993).

Salkovskis Responsibility Scale (SRS). The SRS is a 27-item scale that assesses patient's dysfunctional beliefs regarding their responsibility for negative events (Bouvard et al., 2001). That is, the scale measures the tendency to assume responsibility in a given situation, particularly situations involving intrusions and doubts. Total scores range from 0 to 115, and the scale has been shown to have adequate psychometric properties with reliability of .90 and .95 among controls and OCD patients, respectively (Bouvard et al., 2001).

Beck Depression Inventory (BDI). The BDI is a 21-item questionnaire of depression severity (A. T. Beck & Steer, 1987). Items are rated from 0 to 3, and scores range from 0 to 63. It is a well-established measure, and its psychometric properties are excellent with reliability as high as .95 in clinical samples (A. T. Beck, Steer, & Garbin, 1988).

Assessment Occasions

Participants were assessed at Weeks 0, 4, 16 (posttest), 26, and 52 (follow-up). An independent evaluator blind to the treatment condition administered the Y-BOCS, the primary outcome variable. The independent evaluator also administered a BAT at the week of each assessment. The participants completed the remaining measures.

Treatments

Figure 1 depicts the flow of participants through each stage of the treatment trial where patients were randomized to either CT or BT.[1] CT was delivered consistent with the Beckian model (e.g., A. T. Beck, Emery, & Greenberg, 1985; Salkovskis, 1985) and consisted of 20 hr of therapy over 16 weeks. CT had several components including explanation of the rationale, elicitation of intrusive and automatic thoughts, Socratic discussion, elicitation and discussion of dysfunctional danger, and responsibility schemas. One of the major goals was the modification of unrealistic interpretations, magical thinking and thought–action fusion. Behavioral experiments to test dysfunctional automatic thoughts and cognitive schemas were used after verbal challenging. The patient had to rate the intensity of the dysfunctional target thoughts after homework completion on a form. Patients were not given instructions to complete in vivo exposures with a habituation rationale but to confront the feared situations to modify their thoughts. Cognitive homework was discussed and assigned by the therapists. Sessions were given once or twice a week according to the patient's availability.

BT was delivered in the form of exposure and ritual prevention (ERP). The patients first received a rationale for the use of BT. The treatment consisted of therapist-aided imaginal and in vivo exposure with response prevention and family intervention was provided whenever the family was involved in the rituals (Foa & Wilson, 1991; Marks, 1987). The behavioral homework was discussed and assigned by the therapists. Patients recorded homework completion and rated anxiety on a form. BT consisted of 20 hr of therapy during 16 weeks that was presented in two phases. An initial intensive phase of 4 weeks, with two sessions lasting 2 hr each week (total 16 hr), was followed by a maintenance phase of 12 weeks, with one 40-min booster session every 2 weeks (total 4 hr). The rationale for treatments administration was to create a treatment context that would maximize the effects of the theorized mechanism for each OCD treatment (e.g., Foa et al., 2005). For CT, a progressive approach would allow for cognitive change to take place first, before gradual behavioral experiments that were used to modify automatic thoughts and schemas. However, an intensive approach for BT would better maximize the theorized extinction learning via a response prevention model that underlies the treatment's effectiveness. The therapists for both treatments were psychologists or psychiatrists that were certified in CBT. This certification required supervision of clinical cases, exams on cognitive behavioral case conceptualizations, and documented mastery of the theoretical foundations of CBT. The therapist also received an additional training of 20 hr. The therapists also had manuals for both treatments, with therapeutic guidelines, and received supervision as needed. A more detailed description of the design of the study has been reported elsewhere (Cottraux et al., 2001).

Results

Baseline Characteristics and Attrition

Of the 85 participants screened, 62 met inclusion criteria, completed the baseline assessment and were randomly assigned to CT ($N = 30$) or BT ($N = 32$). As can be seen in Table 1, there were no significant between-group differences on any of the demographic or study variables at baseline. Between Week 4 and Week 16, two (3.2%) participants dropped out; total cumulative attrition during the follow-up period was nine (14.5%) by Week 26 and 14 (22.6%) by Week 52. Consequently, we obtained data from 285 of the possible 310 (62×5) assessments (91.9%). There were no between-group differences in attrition rate (9/32 = 28% in BT vs. 5/30 = 17% for CT; Fisher exact test $p = .37$).

Outcome and Mediation Analyses

Overview. We used MLM to analyze the data. Given that our MLM analysis involved repeated measures over time, we modeled the error covariance matrix as auto correlated, with heterogeneous variances over time. And because we tested differences between nested models in some of our analyses, we used maximum-likelihood estimation instead of restricted maximum-likelihood estimation. We used robust standard errors to calculate the t statistics for the regression coefficients (these are robust to violations of multivariate normality) and used the Satterthwaite approximation to calculate the degrees of freedom for the t tests (resulting in differing dfs for each test). Post hoc power analyses using the program PinT (Power in Two-level Models; Snijders & Bosker, 1993) indicated that we had greater than .80 power to detect an effect size as small as $d = 0.34$ (between a small [$d = 0.20$] and a medium [$d = 0.50$] effect size).

Treatment effects. First, we investigated the slopes of change over time for our outcome measure (Y-BOCS). As expected, Y-BOCS scores followed a nonlinear growth curve (see Table 2 for mean scores at each time point), improving rapidly during treatment and leveling off during follow-up (see Figure 2). We expected the exponential growth curve to most appropriately approximate the pattern of change of Y-BOCS over time. However, we evaluated various growth curve models (linear, quadratic, and exponential) on their deviance statistics (–2 log likelihood, Akaike information criterion [AIC], and Bayesian information criterion [BIC]) to determine if another model more accurately reflected the change in Y-BOCS over time (Singer & Willett, 2003). The linear model included Time, Condition, and Time × Condition as predictors in the model. The quadratic model added Time^2 and Time^2 × Condition as additional predictors. The exponential growth curve, in which the outcome levels off at an asymptote, was modeled directly with the log of the outcome as the dependent variable, with Time, Condition, and Time × Condition as predictors (as specified by Singer & Willett, 2003). Additionally, we standardized the dependent variable in each analysis (both the raw Y-BOCS scores and the log-transformed Y-BOCS scores) to enhance the comparison among models. Results for each model are shown in Table 3. All three deviance statistics were lower in the quadratic versus the linear model, and chi-square tests comparing the two models showed that the quadratic model fit the data better than the linear model, $\chi^2(2) = 27.49$, $p < .001$, for difference in the –2 log likelihood. Further, all three deviance statistics were

[1] Because this study was completed prior to the Journal Article Reporting Standards (JARS; APA Publications and Communications Board Working Group on Journal Article Reporting Standards, 2008) and the Consolidated Standards of Reporting Trials (CONSORT; Altman et al., 2001; Moher, Schulz, & Altman, 2001), detailed data regarding participant flow from screening to study exit were not collected.

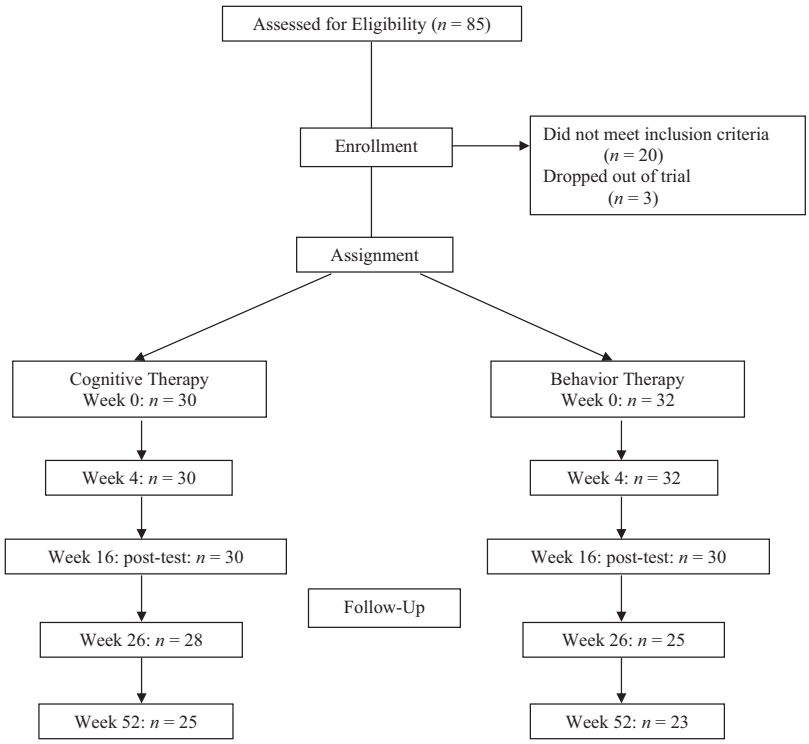

Figure 1. Flow of participants through each stage of the treatment trial.

much lower for the exponential vs. the quadratic models (e.g., 66.29 points lower for the BIC). No significance tests between the quadratic and exponential models were possible because these were not nested and because we would be are comparing a model with *z*-scored Y-BOCS as the dependent variable versus a model with *z*-scored *log* of Y-BOCS as the dependent variable. However, a difference >10 for BIC is considered large (Raftery, 1995; Singer & Willett, 2003), and the difference between these models is considerable larger than 10 (66.29). Further, in the quadratic model, the plot of the growth curve turned substantially higher during follow-up, whereas the actual Y-BOCS scores during follow-up either leveled off (CT) or continued to decrease (BT). Hence, the quadratic model did not reflect the pattern of the data accurately. Thus, the exponential model appeared to be the best fit for the data, so we modeled the change of Y-BOCS over time as an exponential curve (see Figure 2). Accordingly, all analyses used the log of Y-BOCS as the dependent variable, and therefore references to Y-BOCS in this results section refer to the log of Y-BOCS.

Results indicated that the slope of change in Y-BOCS over time was significantly different for the two treatment conditions, $b = .14$, $t(115) = 2.08$, $p = .039$, $d = 0.69$.[2] Over the entire course of the study (52 weeks), the slope of change in the log of Y-BOCS was greater in BT than in CT, $b = -.48$, $t(117) = -10.08$, $p < .001$, $d = 1.80$ for BT, and $b = -.34$, $t(115) = -7.30$, $p < .001$, $d = 1.11$ for CT (see Figure 2). Although these slopes cannot be directly translated into a specific slope of change for the raw Y-BOCS scores because the growth curve was curvilinear, the average rate of change was 5.11 and 3.60 units on the Y-BOCS scale per assessment for BT and CT, respectively. Further, because Time was centered at the final assessment in this analysis (Time was coded −4,-3,-2,-1,0),[3] the treatment condition main effect tested treatment condition differences in Y-BOCS at the final assessment. This test showed that those in BT had lower Y-BOCS scores at the final assessment than those in CT, $b = .57$, $t(60) = 2.11$, $p = .037$, $d = 1.17$ (estimated means at the final assessment: BT = 8.73, CT = 13.10; see Figure 2).

Missing data effects. We used pattern mixture modeling (Hedeker & Gibbons, 2006) to examine whether treatment effects varied as a function of attrition. These models do not assume MAR and can be used to determine whether results vary as a function of the missing data pattern (Hedeker & Gibbons, 2006). Since assumptions regarding missing data cannot be definitively answered using observed data, it is suggested that more than one missing data pattern be evaluated. In our data, there were only three different missing data types since once a patient missed an assessment, that patient missed all subsequent assessments. The three types of missing data were dropout between Weeks 4 and 16 ($n = 2$), dropout between Weeks 16 and 26 ($n = 7$), and dropout between Weeks 26 and 52 ($n = 5$). Since the first group only contained two patients, we combined it with the second group, as per the recommendations of Hedeker and Gibbons (2006, p. 304). We then performed two pattern mixture models. The first included two missing data dummy variables: dropout before Week 26 and

[2] The effect size measure for the slopes of change is estimated as $d = b/\sqrt{\tau_{11}}$ (Raudenbush & Liu, 2001).

[3] Alternately coding Time to reflect number of weeks from baseline to the current assessment yielded identical results as coding it as assessment number. We also investigated whether sex or gender moderated slope of change in Y-BOCS, but neither did.

Table 1
Baseline Characteristics

Variable	CT (n = 30)				BT (n = 30)				Statistics	p
	M	*SD*	*n*	%	*M*	*SD*	*n*	%		
Age (years)	36.83	9.80			34.84	11.38			$t = 0.74$	.47
Sex										
Female			25	83.33			21	65.63	$\chi^2 = 2.54$	.11
Male			5	16.67			11	34.38		
OCD duration	15.48	11.86			11.32	8.72			$t = 1.56$	.13
Axis I comorbidity			13	43.33			10	31.25	$\chi^2 = 0.97$	.33
OC personality disorder			15	50.00			11	34.38	$\chi^2 = 1.55$	.21
Y-BOCS	28.60	5.14			28.50	4.89			$t = 0.08$	.94
Responsibility	78.43	23.70			77.44	22.99			$t = 0.17$	.88
Avoidance	3.40	1.13			3.41	1.19			$t = 0.02$	.98
BDI	18.37	10.47			16.00	9.32			$t = 0.94$	.35

Note. CT = cognitive therapy; BT = behavior therapy; OCD = obsessive-compulsive disorder; OC = obsessive-compulsive; Y-BOCS = Yale–Brown Obsessive-Compulsive Scale (Goodman et al., 1989); BDI = Beck Depression Inventory (A. T. Beck & Steer, 1987).

dropout after Week 26. The second included just one dummy variable coding dropout at any time in the study. Each missing data code was added to the exponential MLM model as a main effect and as a moderator of Time, Condition, and Time × Condition. Following Hedeker and Gibbons, we compared the –2LL of the two missing data models to that of the basic growth curve model described in the previous section. Neither missing data models fit the data significantly better than the basic model, $\chi^2(8) = 9.07$, $p > .34$, for the comparison of the model with the two missing data codes to the basic model, and $\chi^2(4) = 5.56$, $p > .23$, for the comparison of the model with one missing data code to the basic model. Further, none of the Time × Condition × missing data codes was significant ($ps > .53$), indicating that the Condition × Time interaction was not significantly different for patients with missing data than for patients with complete data. Finally, the Condition × Time interaction was significant in all these models, and the value of its coefficients never varied more than 9% from the basic model. Although the small number of dropouts limits conclusions about nonsignificant differences, there is no evidence in these analyses that missing data/dropouts significantly impacted the basic MLM models.

Mediation analyses. In our mediation analyses, we modeled multiple mediators (i.e., responsibility, avoidance, and depression) simultaneously using a panel mediation analysis. The use of simultaneous multiple mediators helps minimize relations that may be due to third variables, and the panel approach helps to separate mediator-to-outcome effects from outcome-to-mediator effects (i.e., reverse causality). Specifically, as can be seen in Figure 3, our panel approach to mediation analysis tests the lagged relations between the mediator and outcome while controlling for the lagged effect of the outcome on the mediator. In addition, our panel approach tests the lagged effect of the outcome on the mediators while controlling for earlier levels of the mediator, which provides estimates of the effect of the outcome on the mediators.

The *a* paths, which represent the change in the mediators over time, were calculated as per MacKinnon (2008).[4] Lagged relations were not necessary in calculating the *a* paths (the effect of Time on the mediators) because reverse causality is nonsensical in this situation. We first derived the most appropriate growth curve model for each mediator, comparing linear, quadratic, and exponential models for each mediator on three deviance statistics (–2 log likelihood, AIC, BIC). Results are displayed in Table 3. For each mediator, the quadratic model was superior to the linear model, $\chi^2 = 13.18$, $p = .001$ for responsibility; $\chi^2 = 59.27$, $p < .001$ for avoidance; and $\chi^2 = 8.47$, $p = .01$ for BDI. Further, the exponential model was superior to the quadratic model for BDI (BIC = 654 for the quadratic model vs. 609 for the exponential model). Thus, we modeled responsibility and avoidance using quadratic growth curves and modeled BDI using an exponential growth curve. Analyses indicated that none of the *a* paths were moderated by treatment condition ($ps > .20$), so the interactions between time and treatment were dropped from these models. As can be seen in Figure 3, this final model yielded significant *a* paths for each of the mediators.

The calculation of the *b* paths (the effect of the mediators on the outcome) and the *r* paths (reverse mediation paths: the effect of the outcome on the mediators) involved (a) the lagged paths between prior levels of the mediator to current levels of the outcome, controlling for prior levels of the outcome (for the *b* paths), and (b) the lagged effect of prior levels of Y-BOCS on each mediator, controlling for earlier levels of the mediator (for the *r* paths, for reverse causation). This required a variation of the typical MLM mediation model (see MacKinnon, 2008, for a typical longitudinal MLM mediation model), which calculates *concurrent* relations between the mediator and outcome. Initial analyses included treatment condition as a moderator of all of the *b* paths. However, results indicated that treatment condition did not moderate any of these paths. Thus, the interaction terms were dropped from the

[4] Sample MLM equations for the *a* path linking Time to BDI were

$$\text{Level 1}: \ BDI_{it} = \beta_{0i} + \beta_{1i} * Time_{it} + \varepsilon_{it};$$

$$\text{Level 2 (for the slope over Time)}: \ \beta_{1i} = \gamma_{10} + \gamma_{11} * Condition_i + \mu_{1i},$$

where BDI_{it} is the BDI for individual i at assessment t, Time is coded –4 to 0 for Assessments 1–5, and Condition is coded 0 = BT, 1 = CT. Condition was nonsignificant for all of the Level 2 equations for each mediator, so it was dropped from the Level 2 equations, and the analyses were recomputed. MLM equations for the other mediators were similar. Level 2 equations for the intercepts were similar to the equations for the slope.

Table 2
Means and Standard Deviations of Study Variables at Each Time Point

	Baseline		Week 4		Week 16		Week 26		Week 52	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Y-BOCS										
CT	28.60	5.14	20.60	7.58	16.13	8.98	14.32	7.49	14.64	8.31
BT	28.50	4.89	20.03	8.55	16.13	8.84	12.48	10.12	10.57	8.95
BAT										
CT	3.40	1.13	1.60	1.50	0.50	1.04	0.50	1.07	0.48	0.82
BT	3.41	1.19	1.41	1.46	0.97	1.27	0.76	1.42	0.61	1.31
SRS										
CT	78.43	23.70	72.30	24.34	62.13	23.69	56.36	23.62	60.16	20.02
BT	77.44	22.99	68.94	22.30	62.97	20.66	57.16	23.50	58.43	21.48
BDI										
CT	18.37	10.47	12.73	9.44	9.97	8.97	10.07	11.38	10.68	10.05
BT	16.00	9.33	13.91	10.84	11.37	9.94	9.24	10.51	8.61	9.17

Note. Y-BOCS = Yale–Brown Obsessive-Compulsive Scale (Goodman et al., 1989); BAT = Behavioral Avoidance Test (Marks, Hallam, Connolly, & Philpott, 1977); SRS = Salkovskis Responsibility Scale (Bouvard et al., 2001); BDI = Beck Depression Inventory (A. T. Beck & Steer, 1987); CT = cognitive therapy; BT = behavior therapy.

model, and the analyses were recomputed.[5] Results from these final analyses are shown in Figure 3. BDI (and not responsibility or avoidance) was related to later changes in Y-BOCS. Further, tests of reverse causality indicated that Y-BOCS was related to later changes in avoidance. There was no evidence that responsibility was either a cause or a consequence of Y-BOCS.

We used the program PRODCLIN (MacKinnon, Fritz, Williams, & Lockwood, 2007) to test the significance of the mediated pathways ($a_i^*b_i$ for the paths from Time through the mediators to Y-BOCS, and $a_i^*r_i$ for the paths from Time to Y-BOCS$_{t-1}$ to the mediators$_t$). PRODCLIN performs an asymmetric distribution of products test for the product of the two segments of the mediated pathway (a^*b). This test is more powerful and has more appropriate Type I error rates than the Baron and Kenny (1986) criteria for mediation (MacKinnon, Lockwood, Hoffmann, West, & Sheets, 2002). Mediated pathways ($a_i^*b_i$ or $a_i^*r_i$) whose confidence intervals do not include 0 are significant. The only significant mediated pathways were the Time-to-BDI-to-YBOCS indirect path, a^*b = –.03, 95% CI: [–.01, –.07], and the Time-to-YBOCS-to-Avoidance indirect path, a^*b = –.05, 95% CI: [–.005, –.09].

Traditional Mediation Analyses

It is instructive to compare the results of the above lagged multimediator analysis to the results that would have been obtained if we had performed more traditional, less complex analyses. Again, differences between the approach in the current analysis and the traditional, cross-sectional single mediator approach are (a) multimediator versus single-mediator analyses and (b) lagged panel mediation analyses versus concurrent mediator analyses. Are the results different if we perform three separate single mediator analyses (one for each potential mediator) rather than a single multimediator analysis? Yes, but the results partially depend on whether we employ a traditional *concurrent* mediator analysis or a lagged mediation analysis. Specifically, all three mediators (instead of BDI alone) emerge as significant when conducting three single *concurrent* mediator analyses (as in the typical Baron and Kenny model), for BDI: a^*b = –.06, 95% CI: [–.03, –.09], for responsibility: a^*b = –.02, 95% CI: [–.01, –.03], and for avoidance: a^*b = –.05, 95% CI: [–.02, –.08]. When conducting three separate single lagged mediation analyses (reflecting the MacArthur approach), BDI (a^*b = –.07, 95% CI: [–.04, –.12]) and responsibility (a^*b = –.02, 95% CI: [–.001, –.03]), but not avoidance (a^*b = .02, 95% CI: [–.01, .06] emerge as mediators of Y-BOCS. Are the results different if we perform a traditional, *concurrent* multimediator analysis instead of a *lagged* multimediator panel mediation analysis? Yes, both BDI (a^*b = –.06, 95% CI: [–.03, –.09]) and avoidance (a^*b = –.07, 95% CI: [–.04, –.10]) are mediators of Y-BOCS in a traditional concurrent multimediator analysis.

Discussion

Although previous research has found BT and CT to be effective for the treatment of OCD (Eddy et al., 2004; Rosa-Alcázar, et al., 2008), the literature addressing which psychological treatment is more effective has been inconsistent. This inconsistency may be partially due to statistical and/or methodological limitations of prior studies examining the differential efficacy of BT and CT for OCD. To address these limitations, the present study reanalyzed previously published data (Cottraux et al., 2001) utilizing more advanced and appropriate statistical methods. With the application

[5] Sample MLM equations for the *b* paths linking mediators to Y-BOCS in the mediation analysis were

$$\text{Level 1: } Y-\text{BOCS}_{it} = \beta_{0i} + \beta_{1i} * \text{Time}_{it} + \beta_{2i} * Y-\text{BOCS}_{it-1} + \beta_{3i} * \text{Resp}_{it-1} + \beta_{4i} * \text{Avoid}_{it-1} + \beta_{5i} * \text{BDI}_{it-1} + \varepsilon_{it};$$

$$\text{Level 2 (for the lagged relation between avoidance and Y-BOCS): } \beta_{4i} = \gamma_{40} + \gamma_{41} * \text{Condition}_i + \mu_{4i},$$

where Y-BOCS$_{it}$ is Y-BOCS for individual i and assessment t (t = 2, 5), Y-BOCS$_{it-1}$ is Y-BOCS for individual i at assessment t − 1, and Avoid$_{it-1}$ is avoidance for individual i at assessment t − 1. Condition was nonsignificant for all of the Level 2 equations for each predictor, so it was dropped from the Level 2 equations, and the analyses were recomputed. Level 2 equations for the other predictors were similar to the one for avoidance.

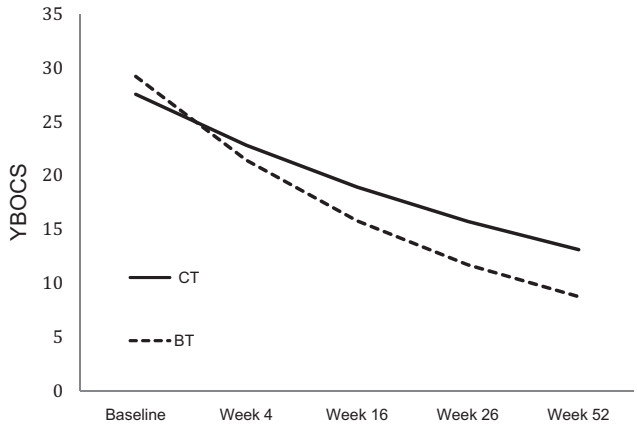

Figure 2. Growth-curve model of obsessive-compulsive disorder symptoms over time. YBOCS = Yale–Brown Obsessive-Compulsive Scale (Goodman et al., 1989); CT = cognitive therapy; BT = behavior therapy.

of MLM to analyze the data, the results revealed that the slope of change in OCD symptom severity over time was significantly greater in BT than in CT. Furthermore, OCD patients in BT had significantly lower symptom severity scores after a 52-week follow-up period than those in CT. Although examination of the means for Y-BOCS shows that the effects of BT and CT are almost identical during treatment, this was not the case after treatment. This finding highlights the importance of conducting extended follow-up assessments in treatment outcome research, allowing for clearer delineation of which psychological treatments produce lasting symptom relief for patients with OCD. One potential implication of these findings for future research examining the relative efficacy of BT and CT for OCD is that repeated symptom assessment over the long term may be required for differences between the treatments to emerge.

The present findings are inconsistent with those of Cottraux et al. (2001), who reported no significant differences between BT and CT in their original analysis. There are multiple factors that may account for the different pattern of findings. For example, Cottraux et al. applied the LOCF and end-point (completer) analyses to this data. As previously noted, LOCF assumes that the outcome for OCD patients would not have changed if they had continued in the study. However, this assumption is often false (Hamer & Simpson, 2009) and can therefore bias the results. Another factor that may account for the different pattern of findings is that Cottraux et al. modeled change in OCD symptoms in a linear fashion. However, a linear approach does not correspond to the pattern of change that is typically observed before, during, and after treatment. Given that improper modeling of change over time can lead to inaccurate estimates of the growth curve parameters (Liu et al., 2012), which may mask differences between treatments, the present approach evaluated various growth curve models to determine which model most accurately reflected the change in OCD symptoms over time.

The present findings highlight the value of utilizing more powerful statistical tools when examining differences between treatments. Failure to employ more appropriate models of the growth curve that properly model missing data and that provide the most powerful statistical tests could potentially yield very different findings. In addition to the statistical and/or methodological implications for treatment outcome research, the present findings favoring BT over CT in the treatment of OCD also have important clinical implications. For example, the findings are inconsistent with Wampold and colleagues' notion that different psychological treatments are equally effective (Ahn & Wampold, 2001; Messer & Wampold, 2002; Wampold et al., 1997). There may be specific characteristic features of BT that are not observed in CT that allow for more favorable outcomes in the treatment of OCD. A central mechanism hypothesized to operate in BT is extinction resulting from systematic exposure (or decrease in avoidance) to fear-related stimuli and prevention of escape or neutralizing behaviors (Himle & Franklin, 2009). In contrast, the purported mechanism of CT is the modification of irrational beliefs. Directly encouraging behavioral change, a characteristic feature of BT, may be a more effective long term strategy for reducing symptoms of OCD than cognitive restructuring, a central feature of CT.

Encouragement of behavioral change is an incidental feature of CT, implemented through behavioral experiments in which patients engage in avoided activities or situations intended to test their beliefs. Thus, while BT and CT differ in theory, there is some overlap in their practice. Given that a behavioral strategy is not entirely unique to BT, it remains unclear as to why the slope of change in Y-BOCS was significantly greater overall in BT than in CT and why those in BT had lower Y-BOCS scores at the final assessment than those in CT. One explanation may be differences in the focus and duration of session activity. That is, whereas the different psychotherapies may share a focus on behavioral change, they vary with respect to how much of the session content is dedicated to maximizing this change. It has been observed that changes in behavior are critical to achieving remission of OCD symptoms (Tolin, 2009). Accordingly, treatments that dedicate more of the session activity on this behavior change may produce superior continued improvement and maintenance of gains during

Table 3
Deviance Statistics for the Linear, Quadratic, and Exponential Growth Models of the Study Variables

Variable	Deviance statistic	Linear model	Quadratic model	Exponential model
Y-BOCS	−2 log likelihood	603.80	576.31	521.33
	AIC	623.80	600.31	541.33
	BIC	660.32	644.14	577.85
Responsibility	−2 log likelihood	544.94	531.76	551.40
	AIC	560.94	549.76	567.40
	BIC	590.16	582.64	596.62
Avoidance	−2 log likelihood	637.56	578.29	636.67
	AIC	653.56	596.29	652.67
	BIC	682.78	629.16	681.88
BDI	−2 log likelihood	612.05	603.58	563.63
	AIC	628.05	621.58	579.15
	BIC	657.27	654.45	608.85

Note. Y-BOCS = Yale–Brown Obsessive-Compulsive Scale (Goodman et al., 1989); BDI = Beck Depression Inventory (A. T. Beck & Steer, 1987); AIC = Akaike information criterion; BIC = Bayesian information criterion.

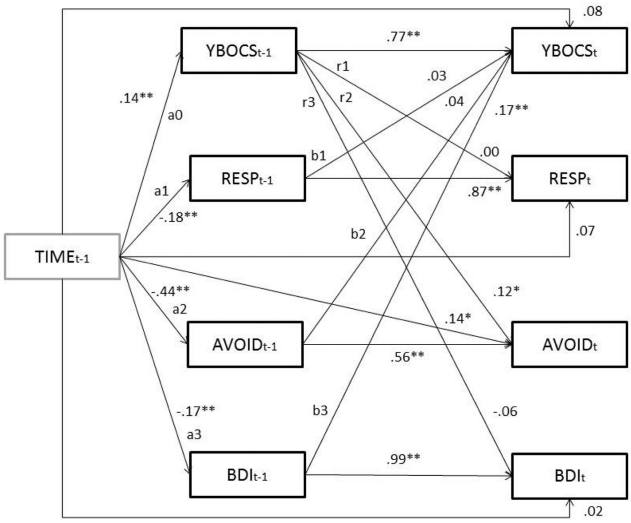

Figure 3. Panel mediation model. YBOCS = Yale–Brown Obsessive-Compulsive Scale (Goodman et al., 1989); RESP = responsibility; AVOID = behavioral avoidance; BDI = Beck Depression Inventory (A. T. Beck & Steer, 1987). $^{*}p < .05$. $^{**}p < .001$.

follow-up. However, we would like to note that patients in the CT group did exhibit only mild OCD symptoms at posttreatment, suggesting that a purely cognitive oriented treatment does provide significant symptom relief for patients with OCD.

Although the present study provides some support for the superior efficacy of BT, refusal and dropout rates in BT have been shown to be high, and some patients retain OCD symptoms after BT (Emmelkamp & Foa, 1983; Kozak, Liebowitz, & Foa, 2000). It is for this reason that many have advocated for the use of cognitive interventions that are not as "aversive" as BT for the treatment of OCD (Wilhelm et al., 2009). Despite such claims, it remains unclear if BT and CT treatment effects vary as a function of attrition. To investigate this, we analyzed differences between the two treatments incorporating pattern-mixture modeling into MLM. This approach allowed for delineating whether patients with missing data have different patterns of response than those without missing data. The findings suggested that dropouts did not respond significantly differently to the treatments than did completers. Further, even when we modeled patients with missing data separately from those with complete data, thereby not requiring data to be MAR, we still found that patients did better in BT than in CT. Although the failure to find different trajectories between completers and dropouts may have been partially due to the small number of dropouts ($N = 14$) or to the fact that almost all dropouts occurred *after* full treatment (i.e., 12 out of 14), these results do bolster confidence in our overall results and suggest that they are valid even if data are not MAR.

The present investigation also examined specific and nonspecific mediators of OCD treatment effects with methodology that incorporates recent advances in mediation modeling. Specifically, multiple mediators were modeled simultaneously using a panel mediation analysis in order to demonstrate specificity of mediator effects and to enhance causal inference of the effect of the mediators on the OCD symptom reduction. Based on theory, we predicted that reductions in avoidance would mediate OCD symptom improvements in BT, whereas reductions in sense of responsibility would mediate OCD symptom improvements in CT. We also predicted that these mediated effects would be independent of the influence of a reduction in depressed mood, a rival nonspecific mediator. Contrary to predictions, changes in sense of responsibility and avoidance were not significant mediators of changes in Y-BOCS over time, and this finding did not vary by treatment condition. In fact, rather than the reduction in avoidance leading to a decrease in OCD symptoms, the opposite was true: decreases in OCD symptoms were related to later decreases in behavioral avoidance. This finding suggests that the degree to which OCD patients are able to reduce avoidance of feared stimuli is largely dependent upon the extent to which they experience symptom relief. A similar pattern of associations was observed for cognitive mediators in Woody et al. (2011). They found that changes in OCD symptoms led to changes in the mediator (responsibility), but not vice versa. The Woody et al. study results, coupled with our results for avoidance, underscore the importance of examining reverse causality in mediation analyses. Otherwise, one might conclude that the mediator caused the outcome when the opposite is actually true (Smits et al., 2012).

Why did we not replicate the Woody et al. (2011) finding that OCD symptoms led to changes in responsibility? There are at least two possible explanations. First, the time lags in our study (up to 26 weeks) were much longer than those in the Woody et al. study (1 week). It is possible that the lags in the present study were too long to detect a causal effect. Second, the Woody et al. study examined *between-subjects* lagged relations (i.e., did participants with greater change in Y-BOCS have greater subsequent change in responsibility?), whereas the present study examined *within-subject relations* (within each participant, were higher levels of Y-BOCS related to higher levels of responsibility at the next assessment, over time). Maxwell and Cole (2007) have shown that between-subjects mediation is rarely the same as within-subject mediation, and the latter is more appropriate and in line with theoretical predictions.

The absence of a mediated effect for responsibility and avoidance raises important questions about specific mechanisms of change in psychological treatment of OCD and may highlight the importance of utilizing advanced statistical analyses when examining treatment mechanisms. Specifically, all three mediators (instead of BDI alone) emerged as significant when conducting three single *concurrent* mediator analyses, and both BDI and responsibility (instead of BDI alone) emerged as mediators of Y-BOCS when conducting three single lagged mediation analyses. These results replicate previous findings suggesting that responsibility mediates changes in OCD symptoms during BT. However, responsibility was not found to be a significant mediator of Y-BOCS in either of the *multimediator* analyses (neither the concurrent nor the lagged panel mediation analyses). These findings as a whole suggest that the previously obtained relation between responsibility and Y-BOCS may have been due to third variables (either BDI or avoidance). Accordingly, multimediator models may help rule out false positives for mediators by partially eliminating third-variable explanations.

The results for the three single lagged mediation analyses can also give us other important information. The fact that two of the three mediators (BDI and responsibility) were found to be significant in the single mediator analyses suggests that the relatively

long lags in our study were not too long to prevent us from finding significant lagged mediating relationships. Thus, our failure to find significant relations between some of the mediators and outcome in our major cross lagged multimediator analysis may not have been due to the long lags between assessments, although the long lags may have made those relations weaker.

Certainly, the finding that the reduction in depressed mood emerged as the single significant mediator is noteworthy. This observation is consistent with research, which has shown that more severe depression predicts less improvement during BT (e.g., Foa et al., 1983), suggesting that reducing depression may be important for achieving clinically significant gains in the treatment of OCD. However, this has not been a consistent finding in the literature. For example, Foa et al. (1987) found that reduction of depression with imipramine had no effect on the subsequent treatment of OCD with ERP. Given prior research suggesting that targeting depression among patients with OCD may prevent the failure to habituate to anxiety-evoking stimuli during exposure and may increase motivation for therapy (Abramowitz, Franklin, & Street, 2000), future research is clearly needed to more precisely delineate when and why depression mediates OCD treatment outcome.

Our findings that reduced avoidance *results* from OCD symptom improvement and that a reduction in the sense of responsibility does not mediate OCD symptom change after controlling for the influence of depression symptom reduction suggest that the effects of treatments for OCD (regardless of whether it is BT or CT) may be accounted for by a rather complex pattern of relations among hypothesized mediators. For example, it is possible that reductions in the sense of responsibility cause depression symptom reduction, which in turn causes OCD symptom severity change, which results in decreased avoidance. Testing more complex mediation models should be the topic of future research (Smits et al., 2012). This work will ultimately help guide the development of more potent intervention strategies for OCD and related problems.

Although the present study has several strengths, including examination of differences between two psychological treatments in patterns of OCD symptom change over time, examination of multiple mediators of OCD symptom change over time, and examination of mediation through a lagged panel approach, which allowed for the consideration of bidirectional relationships, one reanalysis of one trial cannot determine more generally whether BT is more efficacious than CT in the treatment of OCD. In addition, the original design is not without limitations that are difficult to overcome even with more advanced and appropriate statistical methods. For example, although differences in treatment administration allowed for possible maximization of the distinct mechanisms that have been proposed to account for the effectiveness of CT and BT, this difference in the timing of treatment dosages may also have limited direct comparisons between the two treatments. Indeed, both treatments involve a total of 20 hr of treatment over 16 weeks, but the first 4 weeks were more intensive in BT than in CT. The initial intensive treatment, rather than BT per se, may account for the greater slope of change in OCD symptoms compared to the slope of change observed for those in the CT condition. This potential interpretation of the present findings highlights the need for future research that creates a comparable treatment administration context that also allows for the maximization of the mechanisms of action of distinct psychological treatments for OCD. Here, we should also note that the absence of a treatment effect on the mediator variables precludes making inferences with respect to mediators of BT versus CT. Thus, it is possible that the observed mediation effects in this study simply speak to how the proposed mediators relate to OCD symptom severity over time instead of symptom severity change with specific treatments (i.e., BT or CT).

The present study is also limited by the one-dimensional assessment of OCD symptoms and mediators of change. More notable rates of improvements are commonly observed in OCD patients with cleaning and checking compulsions, whereas less improvement may be observed among those with exactness, counting, hoarding, or slowness rituals (Abramowitz, Foa, & Franklin, 2003; Ball, Baer, & Otto, 1996). Assessment of the purportedly distinct dimensions of OCD may reveal a more complex pattern where outcome varies as a function of treatment and/or OCD symptom dimension. Another possible limitation is the potential overlap between the instruments that assess the mediators and outcomes. For example, thematic overlap may partially account for the association between avoidance (proposed mediator) and Y-BOCS severity score (outcome). Although none of the items of the Y-BOCS directly measures avoidance, two Y-BOCS items do assess interference, which may encompass avoidance. This potential thematic overlap between the mediator and the outcome makes it difficult to assert causal claims. It also highlights the importance of developing and incorporating measures of proposed mediators and outcomes that are distinct. Additionally, many aspects of cognitive misappraisals that have been implicated in maintenance of OCD, including overimportance of thoughts, excessive concern about the importance of controlling one's thoughts, overestimation of threat, intolerance of uncertainty, and perfectionism (Obsessive Compulsive Cognitions Working Group, 1997) were not assessed in the current study. Although appraisals involving inflated responsibility have been most strongly related to OCD symptom severity and reduction (see Woody et al., 2011), the extent to which changes in other cognitive appraisals may mediate changes in OCD during treatment should be a focus of future research. Here, it would be interesting to test whether responsibility appraisals, though characteristic of those with aggressive/harm obsessions, are less relevant for washers who fear "feeling" contaminated, or patients preoccupied with symmetry or order, as has been suggested by some (McKay et al., 2004). Together, these observations suggest that the assessment of a wider array of OCD symptom dimensions and obsessive beliefs in future studies that are adequately powered may allow for a more specific examination of theoretically consistent mediators of treatment outcome.

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