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Cognitive Mediation of Symptom Change in Exposure and Response Prevention for Obsessive Compulsive Disorder

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Abstract

This study examined cognitive mediators of symptom change during exposure and response prevention (EX/RP) for obsessive compulsive disorder (OCD). Based on cognitive models of OCD, obsessive beliefs were hypothesized as a mediator of symptom change. Participants were 70 patients with primary OCD receiving EX/RP either as part of a randomized controlled trial ($n = 38$) or in open treatment following non-response to risperidone or placebo in the same trial ($n = 32$). Blinded evaluations of OCD severity and self-report assessments of three domains of obsessive beliefs (i.e., responsibility/threat of harm, importance/control of thoughts, and perfectionism/intolerance of uncertainty) were administered during acute (weeks 0, 4 and 8) and maintenance treatment (weeks 12 and 24). Study hypotheses were examined using cross-lagged multilevel modeling. Contrary to predictions, the obsessive beliefs domains investigated did not mediate subsequent OCD symptom reduction. In addition, OCD symptoms did not significantly mediate subsequent change in obsessive beliefs. The present study did not find evidence of cognitive mediation during EX/RP for OCD, highlighting the need to investigate other plausible mediators of symptom improvement.

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Keywords

obsessive-compulsive disorder; exposure and ritual prevention; cognitive theory; mediation; treatment

Introduction

Despite the large body of evidence supporting the efficacy of cognitive behavioral therapy (CBT) consisting of exposure and response prevention therapy (EX/RP) for OCD (Koran & Simpson, 2013; NICE, 2006; Ost, Havnen, Hansen & Kvale, 2015; Simpson & Foa, 2015), further refinement of the treatment is warranted to maximize patient outcomes. Randomized controlled trials (RCTs) indicate that, on average, patients receiving EX/RP show a 49% reduction in OCD symptoms at post-treatment (Öst et al., 2015). Across studies, approximately 75–80% of EX/RP treatment completers are deemed responders, defined as 25–30% reduction in OCD symptoms (e.g., Foa et al., 2005). Unfortunately, this leaves many patients with clinically significant symptoms, and only a minority of EX/RP patients achieve minimal symptoms (e.g., Simpson et al., 2008).

Better understanding the treatment processes that lead to symptom reduction (i.e., mechanisms of change) is a promising avenue for improving evidence-based therapies and for refining the scientific theories that underlie particular treatments (Kazdin, 2007). According to the cognitive mediation hypothesis, CBT leads to changes in dysfunctional beliefs related to pathological anxiety, which subsequently leads to symptom reduction (Clark & Beck, 2010). A large number of studies have shown that changes in anxiety-related cognitions pre- to post-treatment mediate symptom reduction when examined concurrently (see for review; Smits, Julian, Rosenfield, & Powers, 2012). However, such traditional pre-post mediation analyses cannot determine whether the mediator causes change in the outcome, the outcome causes changes in the mediator, or whether changes are caused by a third variable (Kazdin, 2007; Kazdin & Nock, 2003). The limitations of such analyses highlight the distinction made by Kazdin (2007) between a mediator, defined as an intervening variable that statistically accounts for the relationship between an independent and dependent variable, and a mechanism, which describes the processes that are actually responsible for the effect on the dependent variable. Properly designed mediational analyses can provide stronger evidence for causal inference without the limitations of traditional pre-post mediation analyses discussed previously. Such analyses in the case of the cognitive mediation hypothesis require demonstrating: 1) temporal precedence of the mediator (i.e., cognitive change), by examining the effect of cognitive change on *subsequent* changes in anxiety symptoms, and 2) specificity of the mediator, by testing for variables other than cognitive mediators that may be related to symptom improvement (Kazdin, 2007; Preacher, 2015; Smits et al., 2012). Thus, while preliminary evidence for the cognitive mediation hypothesis exists, further research is needed to provide more robust support for the idea that cognitive change is a mechanism of CBT's effects on anxiety symptom change.

Mechanisms of Change in OCD

A number of different types of cognitions have been theorized to play an important role in the reduction of OCD symptoms. Emotional processing theory (EPT; Foa & Kozak, 1986), which is the basis for EX/RP, emphasizes that exposure works by disconfirming beliefs about the likelihood of harm, the cost of that harm, and expectations that the feeling of anxiety will persist indefinitely (Foa, Huppert, & Cahill, 2006). Accordingly, erroneous beliefs related to a patient's obsessions about the probability of harm (e.g., I will definitely get sick if I touch that doorknob), the cost of harm (e.g., being sick will ruin everything) and the persistence of anxiety (e.g., I will feel anxious forever if I don't wash my hands) should be modified in order for symptom reduction to occur. Cognitive theories of OCD emphasize a related but distinct set of beliefs as playing a central role in symptom improvement. Specifically, the Obsessive Compulsive Cognition Working Group (OCCWG, 1997, 2001) proposed that three core domains were key to OCD: 1) inflated responsibility and overestimation of threat, 2) over-importance of thoughts and importance of controlling one's thoughts, and 3) intolerance of uncertainty and perfectionism. From the cognitive perspective, modifying these belief systems is central to the reduction of OCD symptoms (Wilhelm & Steketee, 2006).

A number of studies have demonstrated that obsessive beliefs measured by the OBQ change over the course of cognitive and behavioral treatments (Anholt et al., 2010; Solem, Håland, Vogel, Hansen, & Wells, 2009; Storheim & O'Mahony, 2006), and that such changes are correlated with OCD symptom improvement (Adams, Riemann, Wetterneck, & Cisler, 2012; Emmelkamp, van Oppen, & van Balkom, 2002; Whittal, Thordarson, & McLean, 2005). Whether cognitive changes *account* for OCD symptom change during CBT, however, remains unclear. Wilhelm, Berman, Keshaviah, Schwartz, and Steketee (2015) found that changes in obsessive beliefs about perfectionism and certainty predicted OCD symptom change over the course of cognitive therapy. In the only large-scale study to examine obsessive beliefs as a mediator within an exposure-based treatment, Olatunji et al. (2013) found that beliefs significantly accounted for later improvement in OCD symptoms when examined as a single mediator of symptom change. However, when controlling for other putative mediators (i.e., depression and behavioral avoidance), only reductions in depression significantly predicted subsequent OCD improvement. Similarly, Woody, Whittal, and McLean (2011) found different results depending on the type of mediational analyses conducted. When using traditional (pre-post) mediation analyses to examine the role of obsessive beliefs during cognitive therapy, a number of different belief domains accounted for changes in obsessive symptoms. However, when using bivariate dual-change score models to examine whether change in weekly ratings of dysfunctional beliefs impacted subsequent ratings of obsessive symptoms (and vice versa), evidence for cognitive mediation was not found. Instead, change in obsessive symptoms was found to mediate subsequent change in cognitions (i.e., reverse mediation).

The findings of Woody et al. (2011) and Olatunji et al. (2013) demonstrate the importance of evaluating mediation using statistical methods that test for temporal precedence of the mediator to the outcome, reverse mediation, and potential third variables that may be driving the observed mediation relationship. The question as to whether change in cognitions

mediates change in OCD symptoms in CBT remains poorly understood for two reasons. First, the treatment delivered in Woody et al. (2011) and Wilhelm et al. (2015) emphasized cognitive techniques rather than systematic exposure, and mechanisms of change may be different when exposure is a central component of treatment. Second, while Olatunji et al. (2013) found no difference in mediation tests between behavioral and cognitive therapy, the only type of obsessive beliefs measured were responsibility for negative events, and other domains of beliefs may have a different relationship with symptom change. Additional research is warranted to clarify the role of cognitive change during exposure-based OCD treatment.

The present study applies a rigorous methodology recommended for mediation analyses (Kazdin, 2007; Preacher, 2015; Smits et al., 2012) to examine the cognitive mediation hypothesis during EX/RP for OCD. Based on cognitive theories of OCD (OCCWG, 2005), three categories of dysfunctional obsessive beliefs were examined as putative mediators in this study: beliefs regarding inflated responsibility/threat of harm, importance and control of thoughts, and perfectionism/intolerance of uncertainty. Consistent with Wilhelm et al.'s (2015) results and the cognitive mediation hypothesis, we hypothesized that all three categories of obsessive beliefs would mediate OCD symptom changes during EX/RP. Depression was included as an alternative mediator, given its positive association with OCD change in previous investigations (Olatunji et al., 2013; Zandberg et al., 2015).

Methods

Participants

Participants were 70 adults with OCD who were enrolled in an RCT evaluating the effectiveness of EX/RP vs. risperidone as a serotonin reuptake inhibitor (SRI) augmentation strategy (Simpson et al., 2013). Thus, all participants were on a stable dose of SRI throughout the trial. Participants included in the present study completed a pre-treatment assessment and attended two introductory sessions and at least one EX/RP exposure session. All participants met the following inclusion criteria: (a) principal diagnosis of OCD (≥ 1 year) as determined by the Structured Clinical Interview for DSM-IV Axis I Disorders, Research Version, Patient Edition (SCID; First, Spitzer, Gibbon, & Williams, 2002), and (b) received an SRI at a stable dose (≥ 12 weeks) yet remained at least moderately symptomatic (Yale-Brown Obsessive Compulsive Scale [Y-BOCS; Goodman et al., 1989; 1989b] score ≥ 16). The SCID was administered by trained clinicians (Master's Level or above) experienced in the treatment of OCD, and diagnostic information for all patients was reviewed with the principal investigators (the fourth and fifth authors) prior to entry into the study. Exclusion criteria for the study included: active bipolar or psychotic disorders, substance abuse or dependence in the previous three months, prominent suicidal ideation, severe depression as indicated by a score of ≥ 25 on the Hamilton Depression Scale (HAM-D; Hamilton, 1960), prior adjunctive EX/RP or antipsychotic treatment while on an SRI, or hoarding as the only OCD symptom.

Participant age ranged from 18 to 65 ($M = 32.6$, $SD = 11.0$). Forty-nine percent were female, and 94% were Non-Hispanic white. The average years of education completed was 15.5 years ($SD = 2.7$). Mean duration of OCD was 14.6 years ($SD = 10.5$). Overall, 41.4% of

participants ($n = 29$) had at least one additional concurrent Axis I diagnosis, with 24.3% meeting criteria for depressive disorder and 28.6% at least one other anxiety disorder.

Procedure

Details regarding the RCT have been described elsewhere (see Simpson et al., 2013). In brief, the study was conducted at two outpatient research clinics in Philadelphia, Pennsylvania and New York, New York. One hundred OCD patients who were on a stable dose of SRI treatment prior to study entry were randomized to eight weeks of EX/RP ($n = 40$), risperidone ($n = 40$), or pill placebo ($n = 20$). Treatment responders at week 8 (defined as Y-BOCS decrease $\geq 25\%$) were eligible to enter the 6-month maintenance phase, and non-responders were given the choice to crossover to a different treatment. Among those initially randomized to EX/RP, two did not attend any exposure sessions, leaving 38 included in the present study (i.e., initial EX/RP group). Among non-responders to eight weeks of risperidone or pill placebo, 34 participants elected to crossover to EX/RP after non-response to either risperidone ($n = 22$) or pill placebo ($n = 12$); 32 of those participants attended two introductory sessions and at least one EX/RP exposure session and thus were included in the present sample (i.e., crossover EX/RP group). A recent study examining treatment outcome exclusively in the crossover EX/RP group demonstrated a similar, positive response pattern to EX/RP as the initial EX/RP group (McLean et al., 2015), supporting the decision to use an aggregated sample. Consequently, the final sample consisted of 70 OCD patients: 38 who were initially randomized to EX/RP, and 32 who crossed over to EX/RP.

EX/RP Treatment

Treatment consisted of an acute EX/RP phase and a maintenance phase. During acute EX/RP (week 0–8 for initial EX/RP; week 8–16 for crossover EX/RP), all participants received 17 twice-weekly 90-minute EX/RP sessions delivered over eight weeks by study therapists (PhD or PsyD). Therapist training consisted of an intensive 4-day workshop for therapists new to EX/RP, or reviewing training tapes for therapists with prior EX/RP experience, plus the treatment of at least two EX/RP cases under supervision of the last author and Dr. Shawn Cahill, the therapy supervisor for the trial. Active treatment cases were discussed in weekly supervision meetings with the last author and Dr. Cahill. EX/RP was provided using the Kozak and Foa (1997) manual, and consisted of 2 introductory sessions (gathering information & treatment planning), 15 exposure sessions, daily exposure and ritual prevention homework, and between-session telephone check-ins. Treatment did not involve any formal cognitive restructuring techniques, but dysfunctional cognitions were addressed in the context of exposures completed within and between sessions. Thirteen percent of EX/RP sessions were randomly sampled to evaluate treatment adherence. Therapists displayed excellent adherence, using 91% of prescribed therapy elements, and 97% of prescribed therapist behaviors.

During the 16-week maintenance EX/RP phase (week 8–24 for initial EX/RP; week 16–32 for crossover EX/RP), patients were offered additional 90-minute EX/RP sessions or 45-minute maintenance therapy sessions as follows: In the initial EX/RP group (who remained enrolled in the RCT), participants received up to 15 additional 90-minute EX/RP sessions as

needed to achieve excellent response, defined as Y-BOCS score ≤ 12 , and then transitioned to 45-minute maintenance sessions until the end of the maintenance phase. In the crossover group (who were in open treatment following non-response to their randomized condition), participants received additional sessions only as needed to maximize treatment gains and to prevent relapse in OCD.

Measures

Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989a; 1989b)—The Y-BOCS is a 10-item clinician administered, semi-structured interview of symptom severity for obsessions and compulsions in the past week. Items are rated on a 4-point scale (0 to 4; 0 = none, 4 = extreme), yielding two subscale scores (obsessions, compulsions) and a total score ranging from 0 to 40. Higher scores indicate greater OCD severity. The Y-BOCS has good internal consistency ($\alpha = .89$; Goodman et al., 1989a), excellent inter-rater reliability ($r = .72-.98$), and good test-retest reliability ($r = .90$; Kim, Dysken, & Kuskowski, 1990). The Y-BOCS has demonstrated adequate convergent validity with other indices of OCD in outpatient clinical samples ($r_s = .53-.74$), and adequate discriminant validity with measures of anxiety and depression ($r_s = .11-.58$; Goodman et al., 1989b). Internal consistency in the present study was .72, and intra-class correlations (ICCs) between independent evaluators were extremely high (ICC = .99; 95% CI, 0.97–1.00).

Obsessive Beliefs Questionnaire (OBQ; OCCWG, 2005)—The OBQ is a 44-item self-report measure of dysfunctional beliefs related to obsessions, which is derived from the original 87-item OBQ (OCCWG, 2001). The 44-item OBQ contains three empirically derived sub-scales: a) Responsibility/Threat Estimation (OBQ-RT; 16 items), which reflects beliefs about preventing harm from happening, the consequence of inaction, and responsibility for bad things happening (e.g., “*Harmful events will happen unless I am very careful*”); b) Perfectionism/Intolerance of uncertainty (OBQ-PIU; 16 items), which reflects rigid, absolute standards of completion and perfection, and excessive concern over mistakes and uncertainty (e.g., “*If I can’t do something perfectly, I shouldn’t do it at all*”); and c) Importance/Control of Thoughts (OBQ-ICT; 12 items), which reflects beliefs about the consequences of having intrusive and/or distressing thought or images and the need to rid oneself of intrusive thoughts (e.g., “*Having intrusive thoughts means I’m out of control*”). Items are rated on a 7-point Likert scale (1 to 7; 1 = Disagree very much, 4 = Neither agree nor disagree, 7 = Agree very much). The OBQ has demonstrated good psychometric properties, including internal consistency ($r = .89-.95$), and convergent and discriminant validity as established by modest but significant correlations with OCD symptom measures ($r = .27-.59$; OCCWG, 2005). The OBQ also has demonstrated criterion validity, in that OBQ scores have been found to be significantly greater in individuals with OCD compared to anxious controls, who themselves score more highly on the OBQ than healthy controls (OCCWG, 2005). In addition, the three-factor structure of the instrument has been found to be consistent across multiple samples (OCCWG, 2005). Internal consistencies in the present study were .96, .94, and .93 for the RT, PIU, and ICT subscales, respectively.

Hamilton Depression Scale (HAM-D; Hamilton, 1960)—The HAM-D is a widely used interview assessment of depression consisting of 17 items addressing somatic and

psychological symptoms of depression. Items are rated from 0 to 4 or 0 to 2, where 0 is none/absent. The total score ranges from 0 to 52, with higher scores indicating more depressive symptoms. The severity ranges for the HAM-D are as follows: no depression (0–7); mild depression (8–16); moderate depression (17–23); and severe depression (24) (Zimmerman, Martinez, Young, Chelminski, & Dalrymple, 2013). The scale has strong psychometric properties, including internal consistency ($\alpha = .79$), inter-rater reliability ($r = .94$), and test-retest reliability ($r_s = .64-.98$; Trajkovic et al, 2011). Convergent validity with other measures of depression in most studies is adequate ($r_s = >.60$), and the HAM-D has shown to have good classification accuracy between depressed and non-depressed individuals (Bagby, Ryder, Schuller, & Marshall, 2004). Internal consistency in the present study was .77, and ICCs between independent evaluators were excellent (ICC = 0.99; 95% CI, 0.98–1.00).

Assessment Time Points

Patients were assessed by independent evaluators blind to treatment and completed self-report measures at EX/RP baseline (i.e., prior to receiving EX/RP), mid- and post-acute EX/RP treatment, and one and four months into the maintenance phase of treatment. Assessments were conducted at weeks 0, 4, 8, 12, and 24 for the initial EXRP group, and at weeks 8, 12, 16, 20, and 32 for the crossover EXRP group since the latter started EX/RP at week 8.

Data Analysis

To meet the requirements for establishing that maladaptive cognitions are a mediator of therapeutic change (i.e., temporal precedence, observed covariation, and specificity; Kazdin, 2007; Preacher, 2015), we used lagged panel mediation analyses to examine the bidirectional associations between proposed mediators (i.e., obsessional beliefs as assessed by OBQ subscales) and outcome variable (i.e., OCD symptoms as assessed by YBOCS). Prior levels of the mediator and outcomes at Time_(t) were used to predict the mediator and outcomes at Time_(t+1). Depressive symptoms (HAM-D) were included as a nonspecific mediator to test the specificity of the proposed mediator (Kazdin, 2007). This procedure helps to establish the temporal precedence of obsessional beliefs as the putative mediator, controlling for autoregressive effects (i.e., the lagged effect of prior levels of the outcome), and separates mediator-to-outcome effects from outcome-to-mediator effects (i.e., “reverse causation”; Smits et al., 2012).

Multilevel modeling (MLM) in which Level-1 variables (repeated measurement of the OBQ, Y-BOCS, and HAM-D) were nested within Level-2 units (participants) was conducted. All Level-1 variables included five time points: pre- (0-week), mid- (4-week) and post-acute EX/RP treatment (8-week); and 1-month (12-week) and 4-months (24-week) into the maintenance phase of EX/RP treatment. The observed pattern of change trajectory was nonlinear (i.e., curvilinear, exponential), with the mediators and outcomes decreasing rapidly during acute treatment and then leveling off during the maintenance treatment. For this reason, we compared an individual growth model with a nonlinear, natural log time variable to a model with a linear time variable by evaluating multiple fit indices: deviance statistics (–2 times the log-likelihood [–2LL], Akaike’s Information Criterion [AIC], and Bayesian

Information Criterion [BIC]). Results indicated a better fit for the non-linear model across all study variables by using maximum likelihood estimation (ML), thus a log time variable was used for the study analysis.

MLM models were built sequentially using a series of models (Raudenbush, Bryk, & Congdon, 2004) via HLM 6.08 using Restricted Maximum Likelihood Estimation (REML), as this is a preferred estimation strategy for models with few Level-2 units (McCoach, 2010). The first step in the analyses was to estimate unconditional means models with no predictor variable in it, which serves as a baseline model to assess the variance accounted for that exists in Level 1 and 2 (i.e., intraclass correlation; ICC). Separate models were estimated for each study measure (Y-BOCS, OBQ subscales, and HAM-D). In the next step, time was added as a Level-1 covariate to estimate unconditional growth models to examine the rates of change over time for each study measure and if there was interindividual variability in change trajectories. In the third step, to examine whether obsessional beliefs act as a hypothesized mediator of change, we estimated a time-lagged autoregressive model for each OBQ subscale (RT, PIU, and ICT), in which effects of lagged OBQ subscales at Time_(t) on Y-BOCS at time_(t+1), controlling for autoregressive effect (i.e., lagged Y-BOCS at Time_(t)). Lagged HAM-D at Time_(t) was also included as a Level-1 covariate to control for its confounding influence. In the last step, to determine whether “reverse mediation” was present, we tested: 1) the effect of lagged YBOCS at Time_(t) on OBQ subscales at time_(t+1), controlling for lagged OBQ subscales at Time_(t); and 2) the effect of lagged YBOCS at Time_(t) on HAM-D at time_(t+1), controlling for lagged HAM-D at Time_(t). At Level 2, the intercept and the slopes for OBQ (Y-BOCS for reverse mediation model) and HAM-D were estimated as randomly varying across Level-2 units (i.e., random effect). The slopes for time and autoregressive variable were estimated as fixed across all Level-2 units because of too few degrees of freedom to estimate their effects at random. The sample size at both levels was sufficient for accurate estimation for MLM analysis (cf. Maas & Hox, 2005).

The program PRODCLIN (MacKinnon, Fritz, Williams, & Lockwood, 2007) was used to test the significance of the indirect/mediating effects by calculating the asymmetric confidence limits. If the 95% confidence interval (CI) for the strength of the indirect effects does not include zero, the mediated pathway is significant. Percent mediation (P_M) was calculated as an indicator of effect size (MacKinnon, Fairchild, & Fritz, 2007), representing the proportion of the change in outcome variable that is accounted for by the proposed mediator.

Results

Participation and Attrition Analyses

Of the 70 participants in our sample, 3 dropped out during the acute phase (i.e., completed fewer than five 90-minute EX/RP sessions) and another 9 dropped during the maintenance phase. The dropouts did not differ from the remaining participants on any demographics or pre-treatment measures (all $ps > .05$) and all assessment data collected prior to dropout was included in the current analyses. The mean number of 90-minute EX/RP sessions during the acute phase was 15.5 ± 3.1 for the total sample (initial EX/RP: 16.5 ± 1.3 ; crossover EX/RP:

14.2±4.1). The mean number of additional sessions during the maintenance phase was 8.1± 9.6 for the total sample (initial EX/RP: 14.0±8.9; crossover EX/RP: 1.0±3.6).

Pattern mixture modeling (Hedeker & Gibbons, 1997) was used to examine the influence of missing observations due to dropout. Participants who dropped from the acute phase were combined with those who dropped during the maintenance phase as the former contained only three patients, per the recommendations of Hedeker and Gibbons (1997, p.69). A dummy-coded variable “dropout” was constructed as the Level-2 covariate and added to the nonlinear MLM model. The main effect and its interaction with time were nonsignificant for the Y-BOCS and OBQ subscales, indicating that the slopes of change over time for the outcome and proposed mediators were not significantly different for those who dropped out. Accordingly, the missing-data due to dropouts did not significantly impact the basic MLM models.

MLM Results

The analyses began by estimating unconditional means models (i.e., one-way random ANOVA model) to determine the variance explained that exists at each level (i.e., ICC). The ICC value was 0.78 for the Y-BOCS, indicating that the 78% of variance in OCD symptoms was between-person. The ICC values were 0.53, 0.60, 0.52, and 0.52 for OBQ-RT, OBQ-PIU, OBQ-ICT, and HAM-D, respectively, indicating that approximately half of the variances were between-person for the proposed mediators. The -2LL values of the models were 2178.7, 2759.9, 2732.6, 2499.9, and 1791.3 for YBOCS, OBQ-RT, OBQ-PIU, OBQ-ICT, and HAM-D, respectively.

Next, we estimated unconditional growth models by including time as a Level-1 covariate and allowing both the intercept and slope to vary across participants. Table 1 displays the raw observed means and standard deviations for all study measures at each wave of data collection. Overall, participants experienced significant declines in symptoms of OCD (mean slope = -8.90, $p < .001$), depression (mean slope = -2.10, $p < .001$), and obsessional beliefs (mean slopes = -12.22 to -16.87, $ps < .001$) during EX/RP treatment. Meanwhile, it is noted that these variables decreased rapidly during the acute phase (week 0–8) and leveled off during the maintenance phase (week 8–24). By examining the Level-1 variance components, there was significant inter-individual variability in trajectories of change in obsessional beliefs, OCD, and depressive symptoms (all $ps < .001$). The -2LL value of the models were 1872.7, 2570.1, 2564.7, 2272.1, and 1737.5 for Y-BOCS, OBQ-RT, OBQ-PIU, OBQ-ICT, and HAM-D, respectively.

Subsequently, panel mediation analyses were used to examine the temporal and reciprocal associations between OCD symptoms and hypothesized mediators (i.e., OBQ-RT, OBQ-PIU, and OBQ-ICT). For each mediator, we tested three time-lagged autoregressive models: a) lagged OBQ predicts Y-BOCS, controlling for both lagged Y-BOCS and lagged HAM-D; b) lagged Y-BOCS predicts OBQ, controlling for lagged OBQ; and 3) lagged Y-BOCS predicts HAM-D, controlling for lagged HAM-D. The bivariate correlations of all study variables at pretreatment were shown in Table 1.

Lagged Mediation Model 1 (OBQ-RT)—As depicted in Figure 1, lagged OBQ-RT significantly decreased over time (Path *a*), $B = -16.87$, $t(69) = -8.05$, $p < .001$, but was not associated with Y-BOCS scores after controlling for earlier level of Y-BOCS and depression (Path *b*), $B = -0.03$, $t(69) = -1.21$, $p = .186$. The indirect effect of time on Y-BOCS through lagged OBQ-RT was nonsignificant ($ab = 0.47$; 95% CI $[-0.188, 1.172]$), indicating that reductions in beliefs regarding inflated responsibility and overestimation of threat did not mediate subsequent OCD improvement. Lagged Y-BOCS was not associated with OBQ-RT ($B = -0.07$, $p = .710$) and the indirect effect was nonsignificant ($ab = 0.59$; 95% CI $[-2.51, 3.70]$), indicating the absence of reverse mediation. Additionally, lagged HAM-D was unrelated to Y-BOCS score ($B = 0.09$, $p = .314$).

Lagged Mediation Model 2 (OBQ-PIU)—As seen in Figure 2, lagged OBQ-PIU significantly decreased over time (Path *a*), $B = -14.23$, $t(69) = -6.75$, $p < .001$, but was not associated with Y-BOCS scores after controlling for prior level of Y-BOCS (Path *b*), $B = -0.02$, $t(69) = -0.83$, $p = .413$. The indirect effect of time on Y-BOCS through lagged OBQ-PIU was nonsignificant ($ab = 0.24$; 95% CI $[-0.334, 0.846]$), suggesting that reductions in beliefs regarding perfectionism/intolerance of uncertainty did not mediate subsequent OCD improvement. Lagged Y-BOCS was not associated with OBQ-PIU ($B = -0.07$, $p = .710$) and the indirect effect was nonsignificant ($ab = 2.72$; 95% CI $[-0.45, 5.97]$), indicating the absence of reverse mediation. Lagged HAM-D was unrelated to Y-BOCS score ($B = 0.09$, $p = .315$).

Lagged Mediation Model 3 (OBQ-ICT)—As depicted in Figure 3, lagged OBQ-ICT significantly decreased over time (Path *a*), $B = -12.22$, $t(69) = -9.59$, $p < .001$, but was significantly *negatively* correlated with Y-BOCS after controlling for prior level of Y-BOCS (Path *b*), $B = -0.07$, $t(69) = -2.20$, $p = .031$. The indirect effect of time on Y-BOCS through lagged OBQ-ICT was significant ($ab = 0.83$; 95% CI $[0.054, 1.656]$), suggesting that reductions in beliefs about the overimportance of thoughts and importance of controlling one's thoughts mediated later *increases* in OCD symptoms. Lagged Y-BOCS was not associated with OBQ-ICT ($B = -0.03$, $p = .744$) and the indirect effect was nonsignificant ($ab = 0.29$; 95% CI $[-1.324, 1.901]$), indicating the absence of reverse mediation. Lagged HAM-D was unrelated to Y-BOCS score ($B = 0.10$, $p = .220$).

Sensitivity Analyses

Traditional Mediation Analyses—To examine whether the null results of our mediation analyses were due to the testing multiple mediators simultaneously or to time-lagged relationships, we conducted single concurrent and single lagged mediation analyses, with time as the independent variable, Y-BOCS as the outcome variable, and OBQ subscales and HAM-D as single mediators in four separate analyses. Single concurrent mediation analyses indicated that the indirect effects for all four mediators were significant (OBQ-RT: $ab = -0.93$, 95% CI $[-1.57, -0.35]$; OBQ-PIU $ab = -0.95$, 95% CI $[-1.60, -0.40]$; OBQ-ICT: $ab = -0.87$, 95% CI $[-1.52, -0.26]$; HAM-D: $ab = -0.68$, 95% CI $[-1.12, -0.33]$). In contrast, separate single lagged mediation analyses showed that the indirect effects of all four mediators were nonsignificant (OBQ-RT: $ab = 0.34$, 95% CI $[-0.39, 1.09]$; OBQ-PIU $ab =$

0.09, 95% CI [-0.57, 0.75]; OBQ-ICT $ab = 0.62$, 95% CI [-0.14, 1.43]; HAM-D: $ab = -0.09$, 95% CI [-0.50, 0.29]).

Treatment Group as a Moderator—To address whether the unexpected results of our mediation analysis were impacted by sample composition (i.e., initial EX/RP plus crossover EX/RP group), we conducted moderated mediation analyses including “treatment group” (i.e., initial EX/RP vs. crossover EX/RP) as a Level-2 covariate on each of the b paths (i.e., the association between lagged OBQ subscales and Y-BOCS). With Y-BOCS as the outcome variable, MLM analyses showed that all cross-level interaction effects between the OBQ subscales and treatment group were nonsignificant (OBQ-RT: $B = 0.03$, $p = .389$; OBQ-PIU: $B = 0.06$, $p = .125$; OBQ-ICT: $B = 0.03$, $p = .642$), indicating that the observed mediation findings presented above were not impacted by the inclusion of crossover participants.

Discussion

The present study used cross-lagged multilevel modeling to evaluate whether dysfunctional obsessive beliefs mediated OCD symptom improvement among 70 participants receiving EX/RP. Based on cognitive models of OCD, we specifically examined three central domains of obsessive beliefs (OCCWG, 2005) as possible cognitive mediators: inflated responsibility/threat of harm, perfectionism/intolerance of uncertainty, and importance and control of thoughts. Although obsessive beliefs decreased significantly across EX/RP, contrary to our hypotheses, obsessive beliefs did not significantly mediate symptom change. Our analytic approach meets the recommended criteria for demonstrating mediators of treatment change proposed by Kazdin (2007) and Preacher (2015), including temporal precedence of the mediator with respect to the outcome, dose-response relationship, and establishing the specificity of the hypothesized mediator.

The null mediation results found here mirror the results of previous investigations that employed similar methodology to examine cognitive mediation (e.g., Olantunji et al., 2013; Woody et al., 2011) during OCD treatment. Notably, these two previous studies and ours each examined varying types of obsessive beliefs (e.g., beliefs about responsibility, appraisals of personal significance of obsessions, and the three domains of the OBQ-44, respectively) within different forms of CBT (i.e., cognitive therapy and EX/RP). Thus, null findings with regard to cognitive change and reduction in OCD symptoms do not appear to be exclusive to certain belief types or therapies. Wilhelm et al. (2015) did find that changes in perfectionism/certainty beliefs and dependency/incompetence schemas accounted for OCD symptom improvement during cognitive therapy. However, such changes only accounted for a small portion of change in OCD symptoms (16%), and – more importantly – changes in beliefs were analyzed as a predictor of subsequent reduction in OCD symptoms rather than a mediator of the effect of time on symptom change.

Surprisingly, we found that beliefs regarding the importance and control of thoughts (OBQ-ICT) mediated *increases* in OCD symptoms, which is inconsistent with previous research (Woody et al., 2011). However, several features point to this finding being a statistical artifact: First, when using a traditional pre-post (concurrent) mediation analyses, OBQ-ICT significantly mediated decreases in OCD symptoms, as expected. Second, when conducting

single lagged mediation analysis, the association between lagged OBQ-ICT and Y-BOCS, although negative, was nonsignificant ($B = -0.05$, $t(69) = -1.55$, $p = .125$). It is only when Y-BOCS was simultaneously regressed on multiple mediators (i.e., lagged OBQ-ICT, autocorrelation of Y-BOCS, and lagged HAM-D), that the regression weight of lagged OBQ-ICT on Y-BOCS increased and emerged as significant, suggesting the presence of a suppressor variable (Conger, 1974). Thus, replication in other samples is needed before interpretations about this finding can be made.

The current study highlights the importance of using rigorous statistical approaches that account for temporal precedence when conducting mediation research. Namely, when the same data were analyzed using traditional, single mediational analysis, cognitive mediation findings were significant in the present sample. Thus, consistent with previous research (e.g., Smits et al, 2012), a concurrent relationship between cognitive changes and symptom improvement was evident. However, when evaluating temporal precedence of OCD versus cognitive change (using lagged analyses), the “mediation” effect whereby lower obsessive beliefs preceded lower OCD symptoms appears to be eliminated. This suggests that although cognitions and OCD symptoms both improve during exposure-based treatment, cognitive change does not appear to drive subsequent reduction in OCD symptoms.

Taken together, the current body of research shows limited support for the theory that changes in dysfunctional beliefs related to responsibility and threat estimation, perfectionism and intolerance of uncertainty, or the importance and control of thoughts are responsible for OCD symptom improvement. Within treatments emphasizing exposure in particular, the current evidence fails to support such a causal relationship, suggesting that other change mechanisms are likely at play. These null findings are consistent with broader theoretical critiques of the cognitive model of OCD (Cougale & Lee, 2014), which have noted that beliefs such as importance of control of thoughts show poor specificity to OCD, and may in fact only apply to a distinctive subset of OCD patients.

Importantly, other change mechanisms have been proposed that warrant further investigation. For example, inhibitory learning models (Craske et al., 2008) highlight the importance of violation of expectancy in promoting positive outcomes in exposure therapy, emphasizing discrepancy between anticipated and actual outcomes following exposure exercises. Measures such as the Violation of Expectancy Measure for Imaginal Exposure (VEMIE; Tuerk & Mangelin, N.d.) allow for ideographic assessment of a patient’s beliefs before and after exposure exercises to evaluate how changes in these beliefs may relate to symptom reduction. Alternatively, some research has highlighted the importance of meta-cognitions in the maintenance and treatment of OCD (Wells 1997; Rees & Anderson, 2013). Changes in meta-cognitions may be a mediator of symptom change, capturing a domain of cognitive beliefs related to OCD that the OBQ-44 does not measure (Solem et al., 2009). Finally, some research eschews beliefs and cognitions altogether. For example, recent studies have demonstrated that individuals with OCD have deficits in response inhibition (Kalantharoff, Anholt, Keren, & Henik, 2013) and a reliance on habit rather than goal-directed learning (Gillan et al., 2011). This has led Anholt and Kalantharoff (2013) to propose that EX/RP exerts its effects not due to change in beliefs about negative outcomes, but as a form of inhibition training. Additional research using robust mediation analyses is necessary

in order to better understand potential mechanisms of symptom change during CBT for OCD so that interventions more directly targeting these mechanisms can be developed and tested.

The current study has several limitations. First, all participants in the current sample were on a stable dose of SRI throughout the trial. The use of SRIs may have particularly restricted the range of depressive symptoms in the sample, potentially limiting our ability to replicate the findings of Olatunji et al. (2013) regarding depression as a mediator of change in OCD symptoms. Thus, the present results require replication in non-medicated samples of OCD patients. Second, the study sample consisted of predominantly non-Hispanic white adults. The results might not generalize to those outside this demographic profile. Third, the sample size for this study was relatively small ($n = 70$). Although the current study did meet criteria for adequate sample size for MLM (Maas & Hox, 2006), a larger sample size would allow for increased confidence in the present results. Finally, OCD symptoms and obsessive beliefs were assessed every four weeks during treatment instead of every session or weekly, which might contribute to the lack of expected mediational effects in our study. We included assessments from the maintenance phase to capture further changes in our variables of interest, but as can be seen in Table 1, obsessive beliefs and OCD symptoms declined dramatically during acute treatment (week 0 to week 8), while reductions during maintenance were smaller. Therefore, it is possible that cognitive mediation might have been observed in the first eight weeks if more assessment time points were available for analysis (e.g., weekly assessments). Such an approach provides a more fine-grained analysis of change in mediators and symptoms and may detect individual variation in when the change is made during treatment (Kazdin, 2007). Of note, however, numerous other studies have detected lagged mediation effects with assessments conducted at intervals of four weeks or longer, thus such a limitation is not necessarily prohibitive (McLean, Su, & Foa, 2015; McLean, Yeh, Rosenfield, & Foa, 2015; Olatunji et al., 2013).

Conclusions

The present study contributes to our knowledge of mechanisms of change in OCD during EX/RP by testing the cognitive mediation hypothesis within a manualized course of EX/RP. Using cross-lagged multi-level analyses, we replicated previous findings (Olatunji et al., 2013; Woody et al., 2011) showing that reductions in obsessive beliefs do not mediate OCD symptom improvement during CBT, suggesting that other mediators not examined in the present study may have an important role in OCD symptom reduction during exposure-based treatments. Additional studies employing rigorous tests of mediation are needed to elucidate the mechanisms of change in evidence-based OCD treatment.

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Figure 1. Lagged Mediation Model 1

Changes in OBQ-RH mediating reduction in Y-BOCS Y-BOCS = Yale-Brown Obsession Compulsive Scale; OBQ-RT = Responsibility/Threat Estimation subscale of the 44-item Obsessional Beliefs Questionnaire. HAM-D = Hamilton Depression Rating Scale.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 2. Lagged Mediation Model 2

Changes in OBQ-PIU mediating reduction in Y-BOCS Y-BOCS = Yale-Brown Obsession Compulsive Scale; OBQ-PIU = Perfectionism/Intolerance of Uncertainty subscale of the 44-item Obsessional Beliefs Questionnaire. HAM-D = Hamilton Depression Rating Scale.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 3. Lagged Mediation Model 3

Changes in OBQ-IC mediating reduction in Y-BOCS Y-BOCS = Yale-Brown Obsession Compulsive Scale; OBQ-ICT = Importance/Control of Thoughts subscale of the 44-item Obsessional Beliefs Questionnaire. HAM-D = Hamilton Depression Rating Scale.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 1

Means, Standard Deviations, and Correlations for Study Measures

Measures	Mean intercept	Mean slope	Pretreatment <i>M(SD)</i>	4-week <i>M(SD)</i>	8-week <i>M(SD)</i>	12-week <i>M(SD)</i>	24-week <i>M(SD)</i>	1	2	3	4
1. Y-BOCS	26.49 ^{***}	-8.90 ^{***}	26.53 (4.01)	20.97 (5.97)	15.49 (6.52)	14.32 (7.31)	13.09 (8.32)	—			
2. OBQ-RT	65.32 ^{***}	-16.87 ^{***}	63.87 (27.45)	56.85 (23.06)	46.00 (21.82)	41.11 (22.13)	37.31 (20.72)	.11	—		
3. OBQ-PIU	72.32 ^{***}	-14.23 ^{***}	70.70 (24.31)	66.09 (21.86)	56.12 (24.30)	52.09 (26.11)	47.94 (25.80)	.13	.63 ^{***}	—	
4. OBQ-ICT	41.87 ^{***}	-12.22 ^{***}	41.20 (18.47)	34.78 (14.88)	28.05 (14.17)	24.11 (13.33)	22.92 (13.11)	.11	.59 ^{***}	.44 ^{***}	—
5. HAM-D	7.94 ^{***}	-2.10 ^{***}	8.21 (5.87)	6.26 (4.90)	4.77 (3.93)	5.38 (5.01)	4.24 (3.99)	.29 [*]	.29 [*]	.17	.32 ^{**}

Note. Correlations between study variables were calculated with pretreatment values. Y-BOCS = Yale-Brown Obsession Compulsive Scale; OBQ-RT = Responsibility/Threat Estimation subscale of the 44-item Obsessional Beliefs Questionnaire (OBQ); OBQ-PIU = Perfectionism/Intolerance of Uncertainty subscale of the 44-item OBQ; OBQ-ICT = Importance/Control of Thoughts subscale of the 44-item OBQ; HAM-D = Hamilton Depression Scale.

* $p < .05$.

** $p < .01$.

*** $p < .001$.