

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

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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 nonresponse 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.

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

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; Öst, 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 posttreatment (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 posttreatment 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: (a) temporal precedence of the mediator (i.e., cognitive change), by examining the effect of cognitive change on *subsequent* changes in anxiety symptoms, and (b) 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 disconfirm-

ing 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: (a) inflated responsibility and overestimation of threat, (b) overimportance of thoughts and importance of controlling one's thoughts, and (c) 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 Obsessive Beliefs Questionnaire (OBQ; OCCWG, 2005) 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

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