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Short communication

Biomedical causal attributions for obsessive-compulsive disorder: Associations with patient perceptions of prognosis and treatment expectancy

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ABSTRACT

In recent years, with scientific advances and growing understanding of neurobiological processes, biomedical explanations of psychiatric disorders, including obsessive-compulsive disorder (OCD), have become more prominent in research and in clinical care. Patient perceptions of biomedical models of OCD have been understudied, particularly in how they relate to patients' beliefs about prognosis and treatment expectancy. The current study measured self-identified OCD patients' (N = 130) current beliefs about their own prognosis and treatment and how believable they found two explanatory models of OCD: 1) a biomedical model that attributes OCD to biological functioning in the brain and 2) an integrative biopsychosocial model. Correlational results indicated that patients who found the biomedical model to be highly believable expected that their OCD would be chronic and require long-term treatment. In contrast, ratings of believability in the model that integrated biological, psychological and social factors in explaining OCD were not associated with prognostic pessimism. Instead, we observed a trend in which stronger belief in the biopsychosocial model was associated with the belief that behavioral changes could improve symptoms of OCD. Notwithstanding limitations inherent in the correlational nature of this study, the current findings highlight the need to further investigate the clinical implications of OCD causal models

1. Introduction

Obsessive-compulsive disorder (OCD) affects approximately 2% of the population and is associated with significant distress and impairment in functioning (Ruscio, Stein, Chiu, & Kessler, 2010). Fortunately, effective treatments exist for OCD, including both psychotherapy and pharmacotherapy. The American Psychiatric Association's Practice Guidelines recommend either cognitive-behavioral therapy (CBT) consisting of exposure and response prevention (EX/RP) or pharmacotherapy with serotonin reuptake inhibitors (SRIs) as first-line OCD treatments (Koran & Simpson, 2013). In clinical practice, treatment usually begins with psychoeducation about OCD, including discussion of a model for how OCD symptoms are acquired and maintained. Such explanatory models are often presented by the clinician in order to provide the rationale for a given treatment. Several models have been proposed, including both biological and cognitive-behavioral models. However, very little research has focused on patient perceptions of these explanatory models and how they relate to patient beliefs regarding prognosis and treatment expectancy. This is an important issue, as substantial research suggests that patient beliefs and expectations

affect treatment response and symptom course (Kirsch, 1999). Thus the present study aimed to explore patient perceptions of two OCD explanatory models (specifically, biomedical and integrative biopsychosocial models) and how perceptions of these models relate to beliefs regarding OCD prognosis and treatment effectiveness.

In recent years, with scientific advances and growing understanding of neurobiological processes, biomedical explanations of psychiatric disorders, including OCD, have become more salient in research and in clinical care (Hyman, 2007). The biomedical approach attributes OCD to neurological, neurochemical, and genetic causal factors and emphasizes targeting these biological processes in treatment. Proponents of biomedical attributions suggest that this view may alleviate stigma associated with "mental illnesses" by emphasizing that these conditions are similar to medical illnesses (Corrigan & Watson, 2004). However, data on the effects of biomedical models on patient and public perception of psychological disorders are mixed (For review and critical analysis, see Deacon, 2013 and Schultz, 2015). Although biomedical explanations are associated with decreased self-blame and reduced shame (e.g., Deacon & Baird, 2009; Phelan, Cruz-Rojas, & Reiff, 2002; Lebowitz & Pyun, 2014), some studies indicate that biomedical

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attributions increase stigma, perceived dangerousness, and reduce empathy from others (e.g. Angermeyer, Holzinger, Carta, & Schomerus, 2011; Kvaale, Gottdiener, & Haslam, 2013; Lebowitz & Ahn, 2014; Read, Haslam, Sayce, & Davies, 2006). Haslam and Kvaale (2015) propose that biomedical and biogenetic explanations are associated with “mixed blessings,” that is, they have both positive and negative effects on stigma. Attributing the cause of the psychological disorder to biogenetic factors outside of the person’s control (attribution of uncontrollability) may help to reduce self-blame. On the other hand, biomedical explanations may cause an essentialist view of the problem (belief that it is inherent and unchangeable), thereby increasing desire for social distance from sufferers, as well as prognostic pessimism, low self-efficacy in addressing the issue oneself, and the perception that medication is more effective and necessary than psychosocial approaches. For this reason, biomedical models can have implications for how people view themselves as well as how they are viewed by clinicians and the public. Some argue that by solely adopting the biomedical conceptualization, we run the risk of biological reductionism or neurocentrism with adverse consequences, including increasing pessimism regarding prognosis and efficacy of treatment (e.g. Deacon, 2013; Lam & Salkovskis, 2007; Kemp, Lickel, & Deacon, 2014; Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Lebowitz, 2014; Lebowitz et al., 2014; Kvaale, Haslam, & Gottdiener, 2013).

The biomedical model of OCD is often communicated to patients as part of the rationale for treating OCD with SRI medications: these medications alter the functioning of serotonin, a neurotransmitter relevant in the functioning of neural circuits implicated in OCD. However, this rationale may make some individuals believe that their symptoms cannot be changed without an intervention targeting these biological systems (e.g., medication, brain surgery) due to perceived genetic or biological determinism of their condition. As such, biological attributions may make some patients feel that non-biological interventions (e.g., changes in behavior and thought patterns promoted in psychotherapy) will be unlikely to help them. To date, most studies examining biomedical explanations of psychopathology have utilized non-clinical samples. Furthermore, these survey studies have examined causal explanations of general psychopathology (disorder-nonspecific), or focused on depression (e.g. Lebowitz et al., 2013) or schizophrenia (e.g. Read et al., 2006). In several studies with more clinically-relevant samples, focusing on individuals with depressive symptoms, generalized anxiety disorder, and eating disorder symptoms, biological attributions were associated with pessimism about prognosis (Farrell, Lee, & Deacon, 2015; Kemp et al., 2014; Lebowitz et al., 2014). As yet, however, no study has investigated patient perceptions of biological attributions in OCD specifically.

In parallel to the biomedical model, the cognitive-behavioral approach to OCD presents an alternative model of the factors involved in the development and maintenance of OCD. Most CBT models are biopsychosocial (non-reductionist) in that they are not overly deterministic as to the exact origin of the disorder, and instead emphasize the potential role of multiple factors and their combination, including biological or genetic factors as well as social and developmental learning. The CBT model primarily focuses on behavioral (and modifiable) factors that maintain symptoms and that serve as the targets for treatment. For example, the CBT model emphasizes the functional connection between obsessions and compulsions (i.e., compulsion relieve distress and are therefore negatively reinforced). This approach is frequently presented in the psychoeducation phase of CBT, as it supports the rationale for EX/RP: prolonged and repeated confrontations with distress-provoking stimuli without engaging in rituals may allow one to break out of the cycle of obsessions and compulsions.

In the current study, we investigated patient perceptions of these OCD explanatory models and their associations with patient beliefs about prognosis and treatment expectancy in a sample of individuals with self-identified OCD. Rather than experimentally manipulating which explanatory model participants saw (as in Deacon & Baird,

2009), we instead elected to present participants with both models and ask how believable each seemed to them as described below. The first model involved a biological attribution for OCD, while the second was an integrative model consisting of biological, cognitive, behavioral, and environmental elements (i.e., biopsychosocial model). Based on the findings reviewed above, we hypothesized that higher believability ratings in the biomedical model would be associated with greater prognostic pessimism and lower perceived efficacy of psychotherapeutic interventions. In contrast, we hypothesized that a higher belief in the integrative biopsychosocial model would be associated with lower prognostic pessimism and higher perceived efficacy of psychotherapeutic interventions.

2. Method

This study was administered via online survey, hosted by Qualtrics, an online survey development tool. Participants were adults (aged ≥ 18 years) who self-identified as having OCD who were recruited via study advertisements placed online at the following sites: the International OCD Foundation (IOCDF) website, social media groups (e.g. Facebook), and web forums for individuals who self-identify as having OCD. Full details of the recruitment materials and sources are available upon request from the authors. The survey was open to all individuals who chose to participate. Upon completion of the survey, participants were given the option to enter a raffle for a chance to win a \$50 gift card. Statistical analyses were run using IBM SPSS (Version 23). All study procedures were approved by the appropriate Institutional Review Board (IRB).

The sample consisted of 105 women and 24 men (total $N = 130$; one participant did not report a gender) with a mean age of 38.1 years ($SD = 12.8$, range 18–83). The sample was 84.6% non-Hispanic White, 8.5% Hispanic, 4.8% Asian/Pacific Islander, and 2.1% “other.” Most (60.8%) of the sample endorsed currently being in treatment for OCD, and 89.2% had a previous history of treatment.

3. Measures

After providing consent, participants completed the online survey consisting of demographic questions, treatment history, and the following measures. Treatment history questions included two questions that asked (Yes/No) if participants had ever received medication for their OCD or had tried CBT consisting of EX/RP.

Dimensional Obsessive Compulsive Scale (DOCS) (Abramowitz, 2010) is a 20-item self-report measuring the severity of OCD symptoms across four dimension: contamination, responsibility for harm and mistakes, symmetry/ordering, and unacceptable thoughts. For each symptom dimension, five items (rated 0–4) assess severity in terms of: (1) time occupied by obsessions and compulsions, (2) avoidance, (3) distress, (4) functional impairment, and (5) difficulty disregarding the obsessions and refraining from compulsions. The four DOCS subscales are summed to create a total score reflecting overall OCD severity. The DOCS total score converges well with other measures of OCD symptoms and has excellent psychometric properties (Abramowitz et al., 2010).

Causal explanations (Deacon & Baird, 2009). Participants were presented with two causal explanations of OCD adapted from a similar study in depression by Deacon and Baird (2009). Participants read the following explanations:

3.1. Biological (brain) explanation

Research suggests that OCD is caused by problems in the brain. Specifically, data from brain imaging studies show that in individuals with OCD there are problems in communication between the front part of the brain (which involves planning and control over one’s actions) and deeper brain structures (which involve emotions such as fear and anxiety). These brain structures use neurotransmitters (basically,

chemical messengers) and data suggest that OCD involves an imbalance in these brain chemicals (particularly one called serotonin). Medications that affect serotonin (serotonin reuptake inhibitors, or SRIs) work to correct this chemical imbalance and restore normal brain function.

3.2. Biopsychosocial explanation

There are a number of reasons why a person might develop OCD, including having a family history of OCD, which may contribute both biological/genetic and environmental aspects. Stressful life experiences, like the death of a loved one, may also play a role. Individuals with OCD may begin having high levels of anxiety and over time develop problematic patterns of relying on rituals (compulsions) as a way to manage their anxiety. Some experts have suggested that specific “thinking mistakes” occur in individuals with OCD, which may cause them to believe that terrible things will happen if they do not do their rituals. Over time OCD can worsen as the person learns to rely on rituals more and more. It is probably a combination of biological, environmental, and psychological factors that causes OCD.

Each description (presented in a fixed order with the biological model first¹) was accompanied by a question asking how believable participants found it to be on a 5-point scale (from “not at all believable” to “extremely believable”). On a separate page, participants were then asked to reflect on their own OCD symptoms and rate questions assessing their beliefs about their own prognosis and how effective they expected different treatment strategies to be (Table 1). Responses were on a 5-point Likert scale, as shown in the table.

4. Analyses

Believability in the two models were each considered to be independent variables and correlated with the prognosis and treatment items (dependent variables). To guard against issues regarding false positive results with multiple correlations, we set a conservative alpha level of $p < .01$. Findings at $p < .05$ were considered trend-level. Where zero-order correlations indicated a significant relationship between model believability and an outcome variable, follow up partial correlations were conducted to control for OCD severity (DOCS scores).

5. Results

The sample mean on the DOCS ($M = 29.84, SD = 15.14$) was similar to that of clinical samples of OCD patients (Abramowitz et al., 2010). Descriptives of study items are presented in Table 2. The biopsychosocial model was rated as slightly more believable on average (compared to the biological model), but this difference did not reach statistical significance, $t(129) = 1.53, p = .127$. Notably, there was evidence for a ceiling effect for the believability of the biopsychosocial model, as the modal response was that this model was extremely believable (maximum). There was also a weak yet significant correlation in the model believability items ($r = .34, p < .001$).

We computed zero order correlation coefficients to examine relationships between the believability of a given causal explanation (biological vs. biopsychosocial) and perceptions of prognosis and treatment efficacy (Table 2). Strength of belief in the biological dysfunction model was significantly associated with worse prognosis (belief that OCD would be chronic problem and would require long-term treatment) and beliefs that medication or combined medication/

¹ The order was fixed with the biomedical model first out of concern that seeing the biopsychosocial model first might have made the exclusively biological model less believable in comparison (given that the elements of the biological model are nested in the biopsychosocial model). Thus to approximate impressions about the biological model (which the primary hypothesis concerned) without being biased by order effects it was presented first.

Table 1
Questionnaire items assessing perceptions of prognosis and treatment.

Credibility	
1. How believable does this explanation seem to you?	
<input type="checkbox"/> Not at all believable	
<input type="checkbox"/> Somewhat believable	
<input type="checkbox"/> Moderately believable	
<input type="checkbox"/> Very believable	
<input type="checkbox"/> Extremely believable	
Prognosis	
2. To what extent would you believe you could eventually recover from your OCD? (Not at all, somewhat, moderately, very much, extremely)	
3. To what extent would you feel able to effectively control your OCD on your own? (Not at all, somewhat, moderately, very much, extremely)	
4. To what extent would you believe long-term treatment is necessary to overcome your OCD? (Not at all, somewhat, moderately, very much, extremely)	
5. To what extent would you expect your OCD to be a chronic problem that persists for years? (Not at all, somewhat, moderately, very much, extremely)	
Treatment	
6. How effective would you expect medication to be in treating your OCD? (Not at all, somewhat, moderately, very much, extremely)	
7. How effective would you expect psychotherapy to be in treating your OCD? (Not at all, somewhat, moderately, very much, extremely)	
8. How effective would you expect a combination of psychotherapy and medication to be in treating your OCD? (Not at all, somewhat, moderately, very much, extremely)	
9. To what extent would you believe that making changes in your attitudes and lifestyle would improve your OCD? (Not at all, somewhat, moderately, very much, extremely)	

Table 2
The relationship between strength of belief in biological and biopsychological causal model and beliefs regarding prognosis and treatment.

Domain and Item	Biological (brain) dysfunction model	Biopsychosocial model
Credibility	$M (SD) = 3.90 (.92)$	$M (SD) = 4.05 (.98)$
Prognosis		
Can eventually recover	$r = -.07$	$r = .03$
$M (SD) = 2.82 (1.1)$	$p = .442$	$p = .736$
Able to control myself	$r = -.01$	$r = .14$
$M (SD) = 2.54 (1.2)$	$p = .966$	$p = .127$
Need long-term treatment	$r = .34$	$r = -.02$
$M (SD) = 3.65 (1.2)$	$p < .001^*$	$p = .789$
Chronic problem	$r = .23$	$r = .06$
$M (SD) = 4.03 (1.0)$	$p = .009^*$	$p = .503$
Treatment		
Medication effectiveness	$r = .44$	$r = .13$
$M (SD) = 3.08 (1.1)$	$p < .001^*$	$p = .133$
Psychotherapy effectiveness	$r = .11$	$r = .16$
$M (SD) = 3.36 (1.0)$	$p = .218$	$p = .074$
Combined treatment effectiveness	$r = .29$	$r = .11$
$M (SD) = 3.72 (1.1)$	$p = .001^*$	$p = .209$
Effectiveness of changing attitude and lifestyle	$r = .01$	$r = .22$
$M (SD) = 3.41 (1.2)$	$p = .878$	$p = .013^{\ddagger}$

Trend-level[†] $p \leq .05$.

Significant ^{*} $p \leq .01$.

therapy would be effective. For each of these associations, partial correlations (controlling for DOCS severity) remained significant, indicating that differences in OCD severity did not drive these associations. Similarly, belief in the biomedical model remained significantly correlated with these three outcomes partialing out belief in the biopsychosocial model. Strength of belief in psychosocial model was correlated only with stronger expectancy that behavioral changes would help with OCD, though this correlation was significant only at a trend-level.

There was also a weak but significant association between past use of medication treatments and stronger belief in biological model ($r_s = .23, p < .01$) but no association between medication use and credibility of biopsychosocial model ($r_s = .02, p > .53$). There was no

significant association with past EX/RP and strength of belief in either model.

We used regression to examine the significant associations between belief in biological model and prognosis/treatment questions after controlling for history of treatment with medication. Believability of the biological model predicted each outcome variable (need for long term treatment, chronic problem, medication effectiveness, combined treatment effectiveness) above-and-beyond medication history (range in R^2 change .04 – .16, p 's < .01).

6. Discussion

The present study is the first investigation to assess the association of belief in a biological model and perception of prognosis and treatment in a sample of individuals with self-identified OCD. Like some other psychiatric disorders, OCD is often presented in the context of a causal model that attributes OCD to biological (brain) factors. For treatment of OCD, the first-line recommendations include either ERP or SRIs (Koran & Simpson, 2013). We were interested in exploring whether the strength of belief in a model that emphasizes biological factors would be associated with negative prognosis (assumption that biological factors are not as malleable to change) and decreased perceived efficacy of psychotherapeutic interventions (in contrast to medications).

As predicted, in our sample, belief in the biological model was significantly correlated with medication expectancy beliefs, such that the more believable the biological model seemed the more medications were anticipated to be helpful (either as monotherapy or in combination with psychotherapy). Research does not however support the idea that medication is necessary for all individuals with OCD to achieve wellness. EX/RP has a higher response rate than medications, and many people find EX/RP alone to be sufficient (NICE, 2013). Relatedly, the more believable the biological model seemed, the more individuals expected that their OCD would run a chronic course and require long term treatment. These associations remained significant after controlling for patients' reported past history of medication treatment and current level of OCD symptom severity.

In contrast to the biological model, believability of the integrative biopsychosocial model was not associated with prognostic pessimism items. Instead, belief in the biopsychosocial model was only correlated with the belief that behavioral changes could improve symptoms of OCD, though the significance of this relationship was trend-level. It should be noted however that this model was rated as being highly believable on average, which may have created a restriction of range issue affecting the ability to detect significant correlations with this item.

Patients with OCD have several options regarding treatment. Future research should investigate the clinical impact of presenting a comprehensive (vs. biologically-exclusive) model, which may allow patients more flexibility in their preference and treatment choice. Although OCD may involve biological factors, which should be included as part of psychoeducation, it may be most important to emphasize the treatable nature of the disorder. By contextualizing OCD and its treatment within a comprehensive model, individuals' perceived efficacy of non-pharmacological treatment may be improved and relatedly their willingness to engage in and respond to this treatment. For example, Maher et al. (2012) reported that individuals who had greater levels of EX/RP treatment expectancy had better EX/RP outcomes, and this relationship was mediated through improved patient adherence to EX/RP procedures (e.g. completion of exposure homework, and efforts to implement ritual prevention). Similarly, in a trial of CBT for pediatric OCD, Lewin, Peris, Bergman, McCracken, and Piacentini (2011) reported that higher treatment expectations were linked to better treatment response, lower attrition, better homework compliance, and reduced impairment at post-treatment. It should be noted though that some trials have not found expectancy beliefs to relate to OCD treatment outcomes (Lax,

Baçoğlu, & Marks, 1992; Vogel, Hansen, Stiles, & Götestam, 2006). There is also research to suggest that providing an intervention consisting of psychoeducation, with an emphasis on change within biological explanations, can reduce prognostic pessimism and increase individuals' perceived agency for up to 6 weeks following the intervention (Lebowitz & Ahn, 2015; Farrell, Lee, & Deacon, 2015).

There are several methodological limitations of the study that are notable. First, the study was correlational in nature, which prevents conclusions regarding causation. Although previous experimental studies suggest that the way illness are described can shape the beliefs patients hold about prognosis and treatment (e.g., Haslam & Kvaale, 2015; Lebowitz, 2014), in our study it could be that pre-existing beliefs about prognosis and treatment influence how believable these models seemed. Thus longitudinal and experimental research is needed to explore the processes by which patients come to form beliefs about prognosis and treatment expectancy and how this related to OCD models. The sample also consisted of individuals with self-identified OCD who self-selected into the study and may not be necessarily representative of the population of all individuals with OCD. There was also no clinician evaluation to ensure that participants met formal diagnostic criteria for OCD, though the sample mean on the OCD severity measure (DOCS) was similar to that reported in clinical samples. Additionally, previous treatment information was not systematically acquired. Participants were asked a simple "yes/no" question to indicate if they had ever in the past received medication or CBT consisting of EX/RP to treat their OCD, but specific details of dose, duration and type were not collected. In addition, the causal models were presented in a fixed order (with the biomedical explanation presented first) which may have caused order effects. We also did not assess for what explanations of OCD participants had been given prior to the present study. Despite these limitations, this study contributes to a growing body of research on the influence of biomedical explanations of psychiatric illnesses and offers preliminary data that we hope will encourage future research on causal models in this population. Our results are consistent with previous research in other conditions, suggesting that belief in biological attributions may be associated with negative beliefs about prognosis and decreased perceived efficacy of psychotherapeutic interventions. More research is needed to further investigate how best to explain OCD symptoms to patients to maximize their ability to engage in first-line treatment.

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