

Letter to the Editor: Recent Advances in Research on Cognition and Emotion in OCD: A Review

Gideon E. Anholt · Eyal Kalanthroff

© Springer Science+Business Media New York 2013

Abstract Cognitive theories of obsessive-compulsive disorder (OCD) ascertain that catastrophic (mis)interpretations of normally occurring intrusive thoughts are causal to the onset and maintenance of OCD. Recently, Calkins, Berman and Wilhelm have highlighted research validating the cognitive model. However, the current comment article stresses various findings that challenge basic premises of the cognitive theory. Moreover, a review of clinical studies investigating cognitive and behavioral therapies for OCD questions the added value of cognitive interventions over and above behavior therapy consisting of exposure and response prevention for this disorder. We suggest an alternative, potentially more useful route of investigation, stressing executive (dis)functions as the cause of OCD patients to (automatically) act on internal and external stimuli. We further suggest that dysfunctional beliefs proposed as paramount in the pathogenesis of OCD according to the cognitive model may be less important and specific than formerly believed.

Keywords Obsessive-compulsive disorder · OCD · Cognitive theory · Behavior therapy · Executive functions · Psychiatry

Obsessive-compulsive disorder (OCD) was considered untreatable until the 1960s, when Meyer [1] developed a new behavioral intervention, which later became known as "exposure and response prevention" (ERP). Since then, ERP has

turned into the treatment of choice for OCD [2]. However, several significant difficulties in the application of ERP were noted, motivating research of additional advancements in the treatment of OCD. Difficulties included high treatment refusal and dropout rates, as well as the finding that many patients maintained significant residual symptoms after treatment [3, 4]. Therefore, increased interest in the cognitive theory of OCD emerged from the 1980s onwards. It was believed that adding cognitive interventions to the treatment of OCD would (1) increase the efficacy of treatment for OCD, (2) help patients who did not succeed in behavior therapy and (3) reduce relapse rates [4]. These hopes have led to great interest and research investment in the cognitive theory and treatment of OCD, detailed in a recent paper authored by Calkins, Berman and Wilhelm [5]. The aforementioned paper highlights research validating the theoretical underpinnings of the cognitive model of OCD [5]. We propose, however, that analysis of current research findings indicates that cognitive theory and treatment of OCD have yet to achieve their set goals. In order to do this, we review research findings (1) concerning the assessment of beliefs hypothesized to be etiological in the onset and maintenance of OCD, (2) comparing the relative efficacy of ERP and cognitive therapy for OCD and (3) examining evidence for cognitive therapy being a viable option for ERP treatment failures. In addition we suggest it might be more useful to divert research attention to alternative theories, and offer a possible direction that relies on consistent findings showing inhibitory deficits in OCD patients [e.g., 6–8].

This article is part of the Topical Collection on *Anxiety Disorders*

G. E. Anholt (✉) · E. Kalanthroff
Department of Psychology, Ben-Gurion University of the Negev,
P.O.B. 653, Beer Sheva 84105, Israel
e-mail: ganholt@bgu.ac.il

E. Kalanthroff
Zlotowski Center for Neuroscience, Ben-Gurion University of the
Negev, P.O.B. 653, Beer Sheva 84105, Israel

OCD Beliefs

In 1997, a distinguished group of prominent OCD researchers comprising the Obsessive-Compulsive Cognitions Working Group (OCCWG) examined the state of the art knowledge of cognitive theory of OCD [9]. They reviewed existing

research and measurements of OCD cognitions and devised the Obsessive-Compulsive Questionnaire-87 (OBQ-87). It entails six belief domains believed to underlie the catastrophic (mis)interpretations of (normally occurring) intrusive thoughts, causal for the onset and maintenance of OCD symptoms: (1) inflated responsibility, (2) intolerance of uncertainty, (3) importance of thoughts, (4) control of thoughts, (5) overestimation of threat and (6) perfectionism. The OBQ has been revised using factor-analytic procedures to form the OBQ-44, containing three subscales, each combining two subscales from the original OBQ-87: (1) inflated responsibility/overestimation of threat, (2) perfectionism/intolerance of uncertainty and (3) importance/control of thoughts [10]. In this article we concentrate on research concerning the OBQ (and its various versions) since it is the most studied measure of OCD cognitions and contains most of the important cognitive constructs. Research using the OBQ reveals several important challenges to the cognitive theory:

1. *Discriminant validity*

The discriminant validity of the OBQ seems to be low since it does not separate OCD from other disorders very well. Although some studies found OCD patients to present with higher scores than anxious controls [e.g., 11], many studies detected significant increases in OBQ scores in various other patient populations. In the initial OCCWG validation study itself, a comparison of OCD patients with non-obsessional anxious patients revealed that in three of the six OBQ-87 scales, no significant differences emerged (intolerance of uncertainty, overestimation of threat and perfectionism; [10]). Anholt and colleagues [12] further demonstrated that overestimation of threat was the only subscale where OCD patients exhibited higher scores than pathological gamblers. In the same study, only two OBQ-87 subscales (inflated responsibility and overestimation of threat) were significantly increased in OCD patients relative to patients with panic disorder with or without agoraphobia [12]. In another research, participants with eating disorders exhibited OBQ-87 scores comparable to or higher than those with OCD [13]. Moreover, patients from a medical clinic for ambulatory and chronic diseases exhibited higher OBQ-87 scores than OCD patients in the domains of intolerance of uncertainty, overestimation of threat, inflated responsibility and perfectionism [14]. Thus, although some research findings support the importance of such beliefs in OCD, the specificity of such beliefs to OCD seems highly questionable.

2. *OCD patients with low OBQ scores and overlap between patients and healthy controls*

A study using cluster analysis with a sample of OCD patients reported that two clusters emerged—one with high levels of obsessive-compulsive (OC) beliefs and one with

low levels of OC beliefs [15]. Remarkably, about half of OCD patients did not show elevations in OBQ scores (i.e., the low-beliefs group). The low- and high-belief groups differed only in "harming obsessions" and checking, however, not in other OCD symptoms (contamination and grooming). Similarly, another study also reported that a large proportion of OCD patients do not show elevated OBQ-87 scores [16]. In this study, 66.35% of OCD patients scored below the clinical cutoff score (set at 2 SD above the mean score of healthy controls). Findings of a large proportion of OCD patients with low OBQ scores may be explained by specificity of these beliefs to certain symptoms and not to others [15]. However, this hypothesis is not compatible with inconsistencies in the literature, with some studies finding modest relations between OC beliefs and *all* symptom dimensions [e.g., 17], while other studies finding specific, (inconsistent between studies) belief-symptom subtype relations [16, 18–20]. Furthermore, a longitudinal 5-year study found (though using healthy controls and unrelated to specific stressful events) OBQ scores to be highly unstable, whereas OCD symptoms showed higher stability [21]. In that study, OBQ scores (of an Italian version consisting of 77 items) predicted OCD symptoms only at the baseline measurement, but not at the follow-up measurements of up to 5 years [21]. If certain beliefs function as a risk factor for OCD symptoms, one would expect opposite patterns; OC beliefs would be a stable underlying factor, whereas OCD symptoms would be elevated in times of stress.

3. *Sensitivity to treatment response*

Anholt and colleagues [16] found that the OBQ (both OBQ-87 and OBQ-44) exhibited relatively low sensitivity as a measure of treatment response. Interestingly though, a different study using a Brazilian sample found the OBQ-44 to be a highly sensitive measure of treatment change [22]. At this point, thus, results concerning the sensitivity of the OBQ to treatment response are mixed.

4. *Cultural differences*

In the initial validation of the OBQ-87 [10], data were collected at 17 sites, and the authors noted that variance-covariance matrices for the OBQ differed across languages. As a consequence, only data from English-speaking countries were pooled together and reported. In another study, OBQ-44 scores in a Dutch sample were found to be different from the scores reported by OCCWG [16]. Such cultural differences cast further doubt on the generalizability of these beliefs as an etiological factor for OCD.

Cognitive Therapy of OCD

Several studies have directly compared cognitive and ERP therapies for OCD [23–27]. These comparisons did not detect

significant differences between these interventions in most post-treatment and follow-up measurements (of up to 5 years [28]). However, a recent study has found ERP to be superior to cognitive therapy of OCD [29]. Moreover, in a meta-analytic study, a slightly higher effect for ERP ($d(+) = 1.127$) than cognitive therapy ($d(+) = 1.090$) for OCD was found, with the addition of cognitive interventions to ERP faring least of all ($d(+) = 0.998$) [30]. Another meta-analytic study concluded that in the treatment of OCD, "the more behaviorally (as opposed to cognitively) oriented psychotherapies tended to be more efficacious, as consistent with other reviews" [31]. In an analysis of clinical significance, ERP was found to be superior to cognitive therapy of OCD, though both interventions were found to produce an equally low (25%) proportion of asymptomatic patients at post-treatment [32]. One might consider the benefit of providing alternatives that offer more treatment options for patients who do not respond to ERP. However, a recent study investigating effects of cognitive therapy (vs. fluvoxamine) as a second line treatment for OCD patients who did not respond to ERP found that cognitive therapy had very little effect on these patients, whereas fluvoxamine showed significantly larger effects [33]. This casts further doubt on whether ERP and cognitive treatment for OCD operate through differential mechanisms. Moreover, cognitive therapy and ERP were found to produce similar *cognitive* changes post-treatment [27]. In this vein, a study analyzing the process of change in ERP and cognitive treatment of OCD concluded that behavioral changes led to treatment gains in both treatment modalities [34].

An Alternative View

OCD patients were found to have deficits in executive functions and particularly in response inhibition [35]. These findings led Anholt and colleagues to suggest that due to response inhibition deficits, OCD patients may be more likely to (automatically) act on internal and external cues and that OC beliefs may develop over time [36, 37]. Findings on healthy participants support the notion that good response inhibition reduces effects of repeated checking (i.e., evokes less doubt as a result of repeated checking), whereas poor inhibition is related to increased effects of repeated checking (i.e., evokes more doubt as a result of repeated checking) [38]. In a similar line of research, OCD patients were found to rely more on habit rather than goal-directed learning, thus forming strong and inflexible stimulus-response associations [39]. Indeed, ERP may be conceptualized as a form of inhibition training—intentionally creating a strong urge to act, and practicing NOT acting upon it. Recently, some researchers have even challenged DSM's [40] assumption of temporal and causal relations between obsessions and compulsions [41]. These researchers have suggested that OCD may be better termed

compulsive-obsessive disorder (COD) and that obsessional or anxiety symptoms may be a consequence rather than a precursor of compulsions [41].

In conclusion, close to 30 years of research on the cognitive theory of OCD and about 25 years of research on the cognitive treatment of OCD have produced meager results in our ability to better treat OCD patients. It may be time to divert research efforts to other venues and consider the possibility that OC beliefs may be less important than previously believed.

Acknowledgment We would like to thank Prof. Avishai Henik for his useful comments.

Compliance with Ethics Guidelines

Conflict of Interest Gideon E. Anholt and Eyal Kalanthroff declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

1. Meyer V. Modification of expectations in cases with obsessional rituals. *Behav Res Ther.* 1966;4:273–80.
2. Olatunji BO, Davis ML, Powers MB, et al. Cognitive-behavioral therapy for obsessive-compulsive disorder: a meta-analysis of treatment outcome and moderators. *J Psychiatr Res.* 2013;47:33–41.
3. Park L, Jefferson J, Greist J. Obsessive-compulsive disorder. *CNS Drugs.* 1997;7:188–202.
4. Clark DA. Cognitive behavioral treatment of obsessive-compulsive disorders: a commentary. *Cogn Behav Pract.* 1999;6:408–15.
5. Calkins AW, Berman NC, Wilhelm S. Recent advances in research on cognition and emotion in OCD: A review. *Curr Psychiatry Rep.* 2013;15:357–64.
6. Morein-Zamir S, Fineberg NA, Robbins TW, et al. Inhibition of thoughts and actions in obsessive-compulsive disorder: extending the endophenotype? *Psychol Med.* 2010;40:263–72.
7. de Wit SJ, de Vries FE, van der Werf YD, et al. Presupplementary motor area hyperactivity during response inhibition: a candidate endophenotype of obsessive-compulsive disorder. *Am J Psychiatry.* 2012;169:1100–8.
8. Lennertz L, Rampacher F, Vogeley A, et al. Antisaccade performance in patients with obsessive-compulsive disorder and unaffected relatives: further evidence for impaired response inhibition as a candidate endophenotype. *Eur Arch Psychiatry Clin Neurosci.* 2012;262:625–34.
9. Obsessive Compulsive Cognitions Working Group. Cognitive assessment of obsessive-compulsive disorder. *Behav Res Ther.* 1997;35:667–81.
10. Obsessive Compulsive Cognitions Working Group. Psychometric validation of the Obsessive Beliefs Questionnaire and the Interpretation of Intrusions Inventory: Part I. *Behav Res Ther.* 2003;41:863–78.
11. Julien D, Careau Y, O'Connor KP, et al. Specificity of belief domains in OCD: validation of the French version of the Obsessive Beliefs Questionnaire and a comparison across samples. *J Anxiety Disord.* 2008;22:1029–41.

12. Anholt GE, Emmelkamp PM, Cath DC, et al. Do patients with OCD and pathological gambling have similar dysfunctional cognitions? *Behav Res Ther.* 2004;42:529–37.
13. Lavender A, Shubert I, de Silva P, et al. Obsessive-compulsive beliefs and magical ideation in eating disorders. *Br J Clin Psychol.* 2006;45:331–42.
14. Baptista MN, Magna LA, McKay D, et al. Assessment of obsessive beliefs: Comparing individuals with obsessive-compulsive disorder to a medical sample. *J Behav Ther Exp Psychiatry.* 2011;42:1–5.
15. Taylor S, Abramowitz JS, McKay D, et al. Do dysfunctional beliefs play a role in all types of obsessive-compulsive disorder? *J Anxiety Disord.* 2006;20:85–97.
16. Anholt GA, van Oppen P, Cath DC, et al. Sensitivity to change of the Obsessive Beliefs Questionnaire. *Clin Psychol Psychother.* 2010;17:154–9.
17. Wu KD, Carter SA. Further investigation of the Obsessive Beliefs Questionnaire: factor structure and specificity of relations with OCD symptoms. *J Anxiety Disord.* 2008;22:824–36.
18. Myers SG, Fisher PL, Wells A. Belief domains of the Obsessive Beliefs Questionnaire-44 (OBQ-44) and their specific relationship with obsessive-compulsive symptoms. *J Anxiety Disord.* 2008;22:475–84.
19. Tolin DF, Woods CM, Abramowitz JS. Relationship between obsessive beliefs and obsessive-compulsive symptoms. *Cogn Ther Res.* 2003;27:657–69.
20. Coles M, Hornig B. A prospective test of cognitive vulnerability to obsessive-compulsive disorder. *Cognit Ther Res.* 2006;12:723–34.
21. Novara C, Pastore M, Ghisi M, et al. Longitudinal aspects of obsessive compulsive cognitions in a non-clinical sample: a five-year follow-up study. *J Behav Ther Exp Psychiatry.* 2011;42:317–24.
22. Bortolotto CF, Braga DT, Gomes JB, et al. Psychometric properties of the Brazilian version of the Obsessive Beliefs Questionnaire (OBQ-44). *J Anxiety Disord.* 2012;26:430–4.
23. Emmelkamp PM, Visser S, Hoekstra RJ. Cognitive therapy vs. exposure in vivo in the treatment of obsessive-compulsives. *Cognit Ther Res.* 1988;12:103–14.
24. Emmelkamp PM, Beens H. Cognitive therapy with obsessive-compulsive disorder: a comparative evaluation. *Behav Res Ther.* 1991;29:293–300.
25. Van Oppen P, de Haan E, van Balkom AJ, et al. Cognitive therapy and exposure in vivo in the treatment for obsessive-compulsive disorder. *Behav Res Ther.* 1995;33:379–90.
26. Cottraux J, Note I, Yao SN, et al. A randomized controlled trial of cognitive therapy versus intensive behavior therapy in obsessive compulsive disorder. *Psychother Psychosom.* 2001;70:288–97.
27. Whittal ML, Thordarson DS, McLean PD. Treatment of obsessive-compulsive disorder: cognitive behavior therapy vs. exposure and response prevention. *Behav Res Ther.* 2005;43:1559–76.
28. van Oppen P, van Balkom AJ, de Haan E, et al. Cognitive therapy and exposure in vivo alone and in combination with fluvoxamine in obsessive-compulsive disorder: a 5-year follow-up. *J Clin Psychiatry.* 2005;66:1415–22.
29. Olatunji BO, Rosenfield D, Tart CD, et al. Behavioral versus cognitive treatment of obsessive-compulsive disorder: An examination of outcome and mediators of change. *J Consult Clin Psychol.* 2013;81:415–28.
30. Rosa-Alcázar AI, Sánchez-Meca J, Gómez-Conesa A, et al. Psychological treatment of obsessive-compulsive disorder: a meta-analysis. *Clin Psychol Rev.* 2008;28:1310–25.
31. Eddy KT, Dutra L, Bradley R, et al. A multidimensional meta-analysis of pharmacotherapy for obsessive-compulsive disorder. *Clin Psychol Rev.* 2004;24:1011–30.
32. Fisher PL, Wells A. How effective are cognitive and behavioral treatments for obsessive-compulsive disorder? A clinical significance analysis. *Behav Res Ther.* 2005;43:1543–58.
33. van Balkom AJ, Emmelkamp PM, Eikelenboom M, et al. Cognitive therapy versus fluvoxamine as a second-step treatment in obsessive-compulsive disorder nonresponsive to first-step behavior therapy. *Psychother Psychosom.* 2012;81:366–74.
34. Anholt GE, Kempe P, de Haan E, et al. Cognitive versus behavior therapy: processes of change in the treatment of obsessive-compulsive disorder. *Psychother Psychosom.* 2008;77:38–42.
35. Chamberlain SR, Blackwell AD, Fineberg NA, et al. The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neurosci Biobehav Rev.* 2005;29:399–419.
36. Anholt GE, Linkovski O, Kalanthroff E, et al. If I do it, it must be important: Integrating basic cognitive research findings with cognitive behavior theory of obsessive-compulsive disorder. *Psicoterapia Comportamental e Cognitiva.* 2012;18:69–79.
37. Kalanthroff E, Anholt EG, Keren R, et al. What should I (not) do? Control over irrelevant tasks in obsessive-compulsive disorder patients. *Clin Neuropsychiatry.* 2013;10:37–40.
38. Linkovski O, Kalanthroff E, Anholt GE, et al. Did I turn off the stove? Good inhibitory control can protect from Influences of repeated checking. *J Behav Ther Exp Psychiatry.* 2013;44:30–6.
39. Gillan CM, Pappmeyer M, Morein-Zamir S, et al. Disruption in the balance between goal-directed behavior and habit learning in obsessive-compulsive disorder. *Am J Psychiatry.* 2011;168:718–26.
40. American Psychiatric Association: Diagnostic and statistical manual of mental disorders (4th ed., text rev.), Washington, DC: 2000.
41. Robbins TW, Gillan CM, Smith DG, et al. Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. *Trends Cogn Sci.* 2012;16:81–91.