DO WE NEED A COGNITIVE THEORY FOR OBSESSIVE-COMPULSIVE DISORDER?

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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. However, we suggest that various research findings challenge basic premises of the cognitive theory. Furthermore, results of clinical trials investigating cognitive and behavioral therapies for OCD challenge the added value of cognitive interventions over and above behavior therapy consisting of exposure and response prevention (ERP) for OCD. It is maintained that there is a need to search for alternative theories to improve OCD understanding and treatment. Executive dysfunctions and particularly response inhibition deficits are suggested as a potential alternative research route.

Key words: OCD, executive functions, cognitive therapy

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Do we need a cognitive theory for OCD? What has been achieved in 30 years of cognitive therapy OCD research?

The cognitive (appraisal) theory of obsessive-compulsive disorder (OCD) suggests that catastrophic interpretations of normally occurring intrusive thoughts constitute a major factor in the onset and maintenance of OCD (Salkovskis 1985, 1999; Rachman 1997). The rationale for the development of this theory has been the realization that behavior therapy of OCD (consisting of exposure and response prevention, ERP), though highly effective, suffers from serious shortcomings. These include high drop-out and treatment refusal rates as well as residual symptoms at the end of treatment (Clark 1999, Park et al. 1997). Several questionnaires have been constructed to measure OCD beliefs underlying this appraisal process, and most importantly the obsessive-compulsive questionnaire (OBQ; OCCWG 1997, 2003). Changing these beliefs and consequent catastrophic interpretations of intrusive thoughts has been the target of cognitive therapy for OCD (Salkovskis 1996, van Oppen and Arntz 1994).

Recently, we have reviewed evidence for the validity of the cognitive theory and efficacy of cognitive therapy for OCD (Anholt and Kalanthroff 2013). Concerning the cognitive theory we proposed that (1) the cognitive theory lacks specificity between OCD and other psychopathologies – as OCD beliefs are increased in various psychopathologies (Anholt et al. 2004, Julien et al. 2008), (2) the cognitive theory lacks specificity between OCD and healthy participants (Anholt et al. 2010). This is manifested in high OCD beliefs in many non-OCD participants (Anholt et al. 2010). Furthermore, about 50% of OCD patients do not exhibit elevated OCD beliefs, and no consistent symptom-belief patterns were detected (Anholt et al. 2010, Taylor et al. 2006), (3) ERP produces similar changes in OCD beliefs as cognitive therapy (Whittal et al. 2005), and (4) OCD beliefs seem to vary significantly between samples derived from various cultures, a finding that is not commensurable with the view of these beliefs as a general etiological factor for OCD (Anholt et al. 2010, OCCWG 2003). Concerning research investigating efficacy of cognitive therapy for OCD we suggested that (1) cognitive therapy seems to be slightly inferior to both ERP and the combination of interventions (Rosa-Alcázar et al. 2008), (2) cognitive therapy does not seem to help participants who are non-responders to ERP (van Balkom et al. 2012). The overall picture from these findings suggests that many cognitive theory assumptions remain unsupported and that there is little evidence that it has advanced our ability to improve treatment of OCD.

How do we proceed?

One option is to consider that measurement of cognitive constructs and cognitive interventions derived to alter them are too rational and nonexperiential. They may include excessive verbalization and logical

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challenging of beliefs. Hypothetically, cognitive beliefs may be causal to OCD symptoms whereas behavioral methods facilitate better change in beliefs and symptoms, possibly due to the more experiential nature of these interventions. One line of support for this notion can be found in the additive effects of imaginary exposure to in vivo exposure for OCD (Rosa-Alcázar et al. 2008). Such exposure is typically administered for catastrophic scenarios that are internal (e.g., burning in hell, being subjected to social condemnation as consequence of behaving in a sexually unacceptable manner). This finding suggests that taking into consideration more than environmental triggering stimuli is essential for OCD treatment. However, even in the case of specific phobia where a highly behavioral (often one session) treatment is clearly the most effective intervention, important attention is given to catastrophic beliefs in directing exposure to feared scenarios (Davis et al. 2012). Yet, construction of exposures to falsify catastrophic beliefs may be achieved in a largely behavioral attitude, without the excessive verbal dialogue characterizing cognitive therapy of OCD.

From beliefs to executive (dys)functions?

In recent years there seems to be an ever-growing interest in the role of executive dysfunctions in OCD (Pauls et al. 2014). One of the functions most investigated is response inhibition, or the ability to stop an action that is no longer necessary. Inhibition deficits have been further suggested as an endophenotype of OCD (Bannon et al. 2002, de Wit et al. 2012). We have suggested that such deficits may make OCD patients more prone to respond to intrusive thoughts by subtle movements, which may in turn make these thoughts seem more likely to happen and underlie catastrophic appraisals of these thoughts (Anholt et al. 2012). A subsequent elaboration of this theory entails viewing compulsive behaviors as reflecting a failure in resolution of task conflict. This notion is based on Gibson's affordances theory, according to which, objects in the environment are not only perceived in terms of their external features but also its affordances – how they can be used (i.e., actions; Gibson 1979). OCD patients may have deficient ability to inhibit automatically triggered actions – resulting in the performance of task irrelevant reactions (e.g., repeatedly pressing a light switch when this action is not necessary; Kalanthroff et al. 2013, 2014). Such ideas are strengthened by the research of Gillan and colleagues, where OCD patients were found to have the tendency for overreliance on habits, showing the learned responses even when unnecessary and not more reinforced (Gillan et al. 2011). These findings have led to the suggestion that obsessions may be a posthoc rationalization of compulsive actions rather than causal to them (thus naming OCD compulsive-obsessive disorder, or COD; Robbins et al. 2012).

The idea that OCD symptoms are (at least partially) caused by executive dysfunctions is contested. Some researchers view these dysfunctions as a consequence of obsessive symptoms (or maybe) of obsessive thoughts that overload and wear out the cognitive system, rather than causal to OCD (Abramovitch et al. 2012). Furthermore, executive dysfunctions seem to be subtle and shared by many psychopathologies (Abramovitch et al. 2014). One possible explanation concerns a dual process. Subtle executive dysfunctions may constitute vulnerability factors. However, high emotional valence may exacerbate these subtler dysfunctions to produce clinically meaningful symptoms (enhanced through paradoxical effects of compulsive behaviors; van den Hout and Kindt 2003). It is likely that such triggering stimuli will be related to self-sensitive domains (Doron and Kyrios 2005). Consider, for example, the postnatal period – one of the sensitive times for OCD onset, where concerns about parenthood may interact with inhibition deficits to produce excessive checking.

In conclusion, we suggest that cognitive theory has not yet lived up to expectations in demonstrating validity and improving treatment efficacy for OCD. The study of executive dysfunctions and particularly inhibition deficits may form the basis for developing additional interventions and improving our understanding of OCD.

References

Abramovitch A, Abramowitz JS, Mittelman A (2013). The neuropsychology of adult obsessive-compulsive disorder: a meta-analysis. *Clinical Psychology Review* 33, 1163-71.

Abramovitch A, Dar R, Hermesh H, Schweiger A (2012). Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: implications for a novel executive overload model of OCD. *Journal of Neuropsychology* 6, 161-91.

Anholt GA, van Oppen P, Cath DC, Emmelkamp PM, Smit JH, van Balkom AJ (2010). Sensitivity to change of the Obsessive Beliefs Questionnaire. *Clinical Psychology and Psychotherapy* 17, 154-9.

Anholt GE, Emmelkamp PM, Cath DC, van Oppen P, Nelissen H, Smit, JH (2004). Do patients with OCD and pathological gambling have similar dysfunctional cognitions? *Behaviour Research and Therapy* 42, 529–37.

Anholt GE, Kalanthroff E (2013). Letter to the Editor: Recent advances in research on cognition and emotion in OCD: a review. *Current Psychiatry Reports* 15, 416.

Anholt GE, Linkovski O, Kalanthroff E, Henik A (2012). If I do it, it must be important: Integrating basic cognitive research findings with cognitive behavior theory of obsessive-compulsive disorder. *Psicoterapia Cognitiva e Comportamentale* 18, 69-77.

Bannon S, Gonsalvez CJ, Croft RJ, Boyce PM (2002). Response inhibition deficits in obsessive-compulsive disorder. *Psychiatry Research* 110, 165-174.

Clark DA (1999) Cognitive behavioral treatment of obsessive-compulsive disorders: a commentary. *Cognitive and Behavioral Practice* 6, 408–15.

Davis TE 3rd, Ollendick TH and Ost LG (eds). (2012). *Intensive one-session treatment of specific phobias.* Springer, New-York.

de Wit SJ, de Vries FE, van der Werf YD, Cath DC, Heslenfeld DJ, Veltman EM, et al. (2012). Presupplementary motor area hyperactivity during response inhibition: A candidate endophenotype of obsessive-compulsive disorder. *American Journal of Psychiatry* 169, 1100-1108.

Doron, G, Kyrios M (2005). Obsessive compulsive disorder: a review of possible specific internal representations within a broader cognitive theory. *Clinical Psychology Review* 25, 415-32.

Gibson JJ (1979). *The ecological approach to visual perception*. Houghton Mifflin, Michigan.

Gillan CM, Papmeyer M, Morein-Zamir S, Sahakian BJ, Fineberg NA, Robbins TW, de Wit S (2011). Disruption in the balance between goal-directed behavior and habit learning in obsessive-compulsive disorder. *American Journal of Psychiatry* 168, 718-726.

Julien D, Careau Y, O'Connor KP, Bouvard M, Rheaume J, Langlois F, et al. (2008). Specificity of belief domains in OCD: validation of the French version of the Obsessive Beliefs Questionnaire and a comparison across samples.

Journal of Anxiety Disorders 22, 1029-41.
Kalanthroff E, Anholt GE, Henik A (2014). Always on guard: Test of highvs. Low control conditions in obsessive-compulsive disorder patients. *Psychiatry Research* 219, 322-8.
Kalanthroff E, Anholt GE, Keren R, Henik A (2013). What Should I (not) Do? Control over Irrelevant Tasks in Obsessive-Compulsive Disorder Patients. *Clinical Neuropsychiatry* 10, 37-40.
Obsessive Compulsive Cognitions Working Group (1997). Cognitive assessment of obsessive-compulsive disorder. *Behaviour Research and Therapy* 35, 667-81.
Obsessive Compulsive Cognitions Working Group (2003). Psychometric validation of the Obsessive Beliefs Questionnaire and the Interpretation of Intrusions Inventory: Part I. *Behaviour Research and Therapy* 41, 863-78.
Park L, Jefferson J, Greist J (1997). Obsessive-compulsive disorder. *CNS Drugs* 7, 188-202.
Rachman SJ (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy 16,* 793-802.
Pauls Dl, Abramovitch A, Rauch SL, Geller DA (2014). Obsessive-compulsive disorder: an integrative genetic and neurobiological perspective. *Nature Reviews Neuroscience* 15, 410-24.
Robbins TW, Gillan CM, Smith DG, de Wit S, Ersche KD (2012) Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. *Trends in Cognitive Sciences* 16, 81-91.
Rosa-Alcázar AI, Sánchez-Meca J, Gómez-Conesa A, Marín-Martínez F (2008). Psychological treatment of obsessive–compulsive disorder: A meta-analysis Clinical Psychology Review 28, 1310-1325.
Salkovskis, PM (1996). Cognitive-behavioral approaches to the understanding of obsessional problems. In RM Rapee (ed) *Current controversies in the anxiety disorders.* Guilford Press, New York.
Salkovskis PM (1985). Obsessional-compulsive problems: a cognitive-behavioral analysis. *Behaviour Research and Therapy 23,* 571-583.
Salkovskis PM (1999). Understanding and treating obsessive-compulsive disorder. *Behaviour Research and Therapy 37,* s29-s52.
Taylor S, Abramowitz JS, McKay D, Calamari JE, Sookman D, Kyrios M, et al. (2006). Do dysfunctional beliefs play a role in all types of obsessive-compulsive disorder? *Journal of Anxiety Disorders* 20, 85-97.
van den Hout M, Kindt M (2003). Repeated checking causes memory distrust. *Behaviour Research and Therapy* 41, 301-316.
van Balkom AJ, Emmelkamp PM, Eikelenboom M, Hoogendoorn AW, Smit JH, van Oppen P (2012). Cognitive therapy versus fluvoxamine as a second-step treatment in obsessivecompulsive disorder nonresponsive to first-step behavior therapy. *Psychotherapy and Psychosomatics* 81, 366-74.
Van Oppen P, Zrntz A (1994). Cognitive therapy for obsessive-compulsive disorder. *Behaviour Research and Therapy* 32, 79-87.
Whittal ML, Thordarson DS, McLean PD (2005). Treatment of obsessive compulsive disorder: cognitive behavior therapy vs. exposure and response prevention. *Behaviour Research and Ther*apy 43, 1559-76.