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Published in:
Journal of Affective Disorders

DOI:
[10.1016/j.jad.2017.08.072](https://doi.org/10.1016/j.jad.2017.08.072)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2018

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Tibi, L., van Oppen, P., van Balkom, A. J. L. M., Eikelenboom, M., Hendriks, G.-J., & Anholt, G. E. (2018). The relationship between cognitions and symptoms in obsessive-compulsive disorder. *Journal of Affective Disorders*, 225, 495-502. <https://doi.org/10.1016/j.jad.2017.08.072>

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Research paper

The relationship between cognitions and symptoms in obsessive-compulsive disorder

Lee Tibi^{a,*}, Patricia van Oppen^b, Anton J.L.M. van Balkom^b, Merijn Eikelenboom^b,
Gert-Jan Hendriks^{c,d,e}, Gideon E. Anholt^a

^a Department of Psychology, Ben-Gurion University of the Negev, Beer-Sheva, Israel

^b Department of Psychiatry and EMGO Institute for Health and Care Research, VU University Medical Center, GGZ InGeest, Amsterdam, The Netherlands

^c Institute of Integrated Mental Health Care “Pro Persona”, Centre for Anxiety Disorders “Overwaal”, Lent, The Netherlands

^d University Center for Psychiatry, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands

^e Department of Psychiatry, Radboud University Medical Centre, Radboud University Nijmegen, Nijmegen, The Netherlands

ARTICLE INFO

Keywords:

Obsessive Compulsive Disorder
Obsessive Compulsive Cognitions
Longitudinal
Cognitive theory
Cross-lagged design

ABSTRACT

Background: The cognitive theory of obsessive-compulsive disorder (OCD) ascertains that catastrophic (mis) interpretations of normally occurring intrusive thoughts are related to the maintenance of OCD. Nonetheless, findings supporting the relationship between cognitive biases and OCD symptoms are largely inconsistent. In the present study we examined the relationship between OCD cognitions and symptoms among 382 OCD patients participating in the longitudinal Netherlands Obsessive Compulsive Disorder Association (NOCDA) study.

Methods: OCD cognitions and OC, anxiety and depressive symptoms were assessed using self-report questionnaires at baseline and at two-year follow-up. Baseline multiple regression analyses assessed the specificity of OC cognitions to OCD symptoms. Cross-lagged analyses examined whether cognitions predict OCD symptoms at two-year follow up.

Results: Baseline analyses demonstrated significant relationships between comorbid anxiety, depressive severity and OC cognitions, adjusted for OCD symptoms ($\beta = .283$, $p < .001$ and $\beta = .246$, $p < .001$, respectively). OCD severity adjusted for comorbid symptoms was unrelated to cognitions at baseline ($\beta = .040$, $p = n.s$). Unique associations were found between cognitions and two OCD symptom subtypes (Impulses: $\beta = .215$, $p < .001$; Rumination/doubting: $\beta = .205$, $p < .001$). Longitudinal analyses yielded non-significant associations between OCD cognitions and symptom severity. Prospective analyses of cognitions and OCD symptom subtypes yielded significant effects for both bidirectional and unidirectional associations ($\beta = .11-.16$, $p < .05$).

Limitations: Given the naturalistic design of the study, we did not assess therapeutic interventions between baseline and follow-up.

Conclusions: Results only partially concord with the predictions of the cognitive theory of OCD. Future studies should focus on mechanisms alternative to cognitions when investigating the course of OCD.

1. Introduction

In the past decades, great interest emerged in the cognitive theory of obsessive-compulsive disorder (OCD), which suggests that misinterpretation of the significance of normal intrusions is the core problem in the etiology and maintenance of OCD (Salkovskis, 1985). Patients with OCD are assumed to interpret such intrusions as informative and important, as well as dangerous or predicting harm. These misinterpretations are hypothesized to be anxiety provoking and result in the performance of compulsions, which temporarily reduce the anxiety, and therefore preserve the symptoms through negative reinforcement.

An international group of OCD experts have identified several OC cognitions, which underlie the vulnerability to develop such negative misinterpretations in response to intrusions (Steketee et al., 2003). The obsessive beliefs questionnaire-44 (OBQ-44, Steketee et al., 2005) measures six belief domains hypothesized to characterize OCD patients: inflated responsibility and overestimation of threat, which pertain the belief that harmful things can happen if one is not careful; perfectionism and the need for certainty, which refer to the belief that one should be absolutely certain about everything; importance and control of thoughts, which depict the belief that thoughts are meaningful and should be controlled.

* Corresponding author.

E-mail address: tibi@post.bgu.ac.il (L. Tibi).

<http://dx.doi.org/10.1016/j.jad.2017.08.072>

Received 1 January 2017; Received in revised form 18 August 2017; Accepted 27 August 2017

Available online 30 August 2017

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Numerous studies have investigated the specific association between the OBQ score and OCD symptoms. For example, several studies found specific associations between OC cognitions and OCD symptoms, while controlling for anxiety or depressive symptoms (Janecek et al., 2003; Morillo et al., 2007; Sica et al., 2004; Wheaton et al., 2010; Wu and Carter, 2008). Support for the cognitive model of OCD was also provided by experimental studies using laboratory paradigms, which suggested that cognitive biases are related to the pathogenesis of obsessions (See Calkins et al., 2013 for a review). These studies manipulated OC beliefs such as inflated responsibility and demonstrated its effect on compulsive behaviors (e.g. Reeves et al., 2010).

However, the association between OC cognitions and OCD is not unambiguous, as researchers recently challenged the notion that dysfunctional cognitions are etiologically linked to OCD (Anholt and Kalanthroff, 2013; Gillan and Robbins, 2014), or specifically associated with OCD symptoms (Belloch et al., 2010; Tolin et al., 2006; Viar et al., 2011). First, several studies detected OC related beliefs in various other clinical populations. For example, Anholt and colleagues (Anholt et al., 2004) demonstrated that overestimation of threat was the only OC cognition in which OCD patients scored higher than pathological gamblers. In addition, a study that compared OC cognitions between medical clinic patients and OCD patients demonstrated a significantly increased OBQ score among the medical patient population (Baptista et al., 2011). Some researchers speculated that although accumulating findings suggest that OCD cognitions are transdiagnostic in nature, they hold a crucial role in the cognitive-behavioral model of OCD (Calkins et al., 2013). Nonetheless, two large clinical studies identified that about half of OCD patients did not endorse dysfunctional cognitions (i.e. the low belief group; Calamari et al., 2006; Taylor et al., 2006). Altogether, these findings cast doubt on the relevance of OC cognitions to OCD. An alternative theory would suggest that rather than a goal directed behavior that is led by the OCD beliefs system, compulsions in OCD are derived from excessive habit formation and inhibitory deficits (Anholt and Kalanthroff, 2013; Gillan and Robbins, 2014). In line with cognitive dissonance accounts, this theory proposes that OC cognitions may be post-hoc rationalizations to the compulsive behavior, rather than its instigator.

Several researchers speculated that the heterogeneous nature of OCD underlies the inconsistent findings with respect to the role of OC cognitions in OCD (Wheaton et al., 2010). This was based on findings that were suggestive of several replicable OCD subtypes: washing, doubting/obsessing, checking, ordering and hoarding (e.g. Leckman et al., 1997; McKay et al., 2004). According to the congruence hypothesis postulated by Tolin and colleagues (Tolin et al., 2006), certain symptom subtypes are expected to be associated with specific domains of obsessive cognitions. However, studies examining associations between cognitions and OC symptom subtypes have also yielded inconsistent results (Fergus and Carmin, 2014; Julien et al., 2008; Wheaton et al., 2010).

Studies up to date have largely used the OBQ to assess cognitions in OCD. While the OBQ is a broad and empirically validated measure of OC cognitions, it has a potential shortcoming for the assessment of specificity, since it is a nomothetic measure representing general beliefs common to several psychopathologies (e.g., Anholt et al., 2004). Perhaps using an idiographic instrument which relies on patients' idiosyncratic "hot" obsessional content may facilitate the understanding of the specific association between cognitive biases and symptoms in OCD. In addition, the OBQ demonstrated little sensitivity to treatment change among OCD patients, which is indicative of its limited clinical utility (Anholt et al., 2010). Furthermore studies largely investigated the association between OC cognitions and symptoms using cross-sectional designs (Novara et al., 2011). Consequently, it cannot address the hypothesis purporting that obsessive cognitions predict future OC symptoms, which is paramount to the cognitive model of OCD (Abramowitz et al., 2007).

The paucity of studies that investigated the time-lagged association

between OC cognitions and symptoms revealed mixed findings. In a five-year follow-up study conducted among students, Novara et al. (2011) reported that OC cognitions as measured by the OBQ predicted OCD symptoms only at baseline. On the other hand, Abramowitz and colleagues found that in a sample of future parents, prenatal OC cognitions prospectively predicted the development of OC symptoms at post-partum, after controlling for pre-existing OCD symptoms (Abramowitz et al., 2006). However, the prospective association between OC cognitions and symptoms was yet to be examined in a large clinical sample of OCD patients. Although analogue samples are relevant to the understanding of the OCD phenomena (e.g. Abramowitz et al., 2014), they have limited ecological validity, since participants would usually endorse symptoms in a minimal to non-existent severity (e.g. Abramowitz et al., 2007). In consideration of the findings corroborating the significance of OCD severity to the course and outcome of the disorder (Sharma et al., 2014), longitudinal studies investigating the relationship of cognitions and symptoms in OCD among clinical samples are needed.

In light of the above-mentioned gap in the literature, we aimed to conduct a systematic investigation of the associations between OC cognitions and symptoms, in a large clinical sample of OCD patients. We report data from the Netherlands Obsessive Compulsive Disorder Association (NOCDA) study, a prospective observational study of a large clinical sample of OCD patients (Schuurmans et al., 2012). One of the main objectives of the NOCDA study is to examine the determinants that are associated with the outcome of OCD. This study was designed to test the contribution of the cognitive biases that characterize OCD to the course of OCD as measured by two time points: baseline and two-year follow-up. Specifically we sought to test the hypothesis postulated by OCD cognitive theorists according to which OC cognitions maintain OCD symptoms (Rachman, 1997; Salkovskis et al., 1999). Due to limited and inconsistent literature on the cross-sectional and longitudinal relationship between cognitions and symptoms in OCD, we addressed three subjects pertaining to the cognitive model of OCD: 1) the specificity of the relationship between OC cognitions and OCD symptoms; 2) the directionality of the relationship between cognitions and symptoms and 3) the contribution of cognitions to the maintenance of symptoms over time. Given that the OBQ describes generally held beliefs not necessarily specific to OCD, we assessed the appraisals of the patients' idiosyncratic obsessions, as they may better encompass the cognitive bias suggested to be unique to OCD (Steketee et al., 2005). We hypothesized that OC cognitions would be significantly associated with OCD, anxiety and depressive symptoms, but with stronger unique associations with OCD symptoms. In line with the cognitive theory of OCD, we expected higher ratings of OC cognitions would predict OCD symptoms at two year follow up.

2. Methods

2.1. Participants and procedures

Data were drawn from the NOCDA study, a large multicenter longitudinal study. The participants were 419 patients with a life-time diagnosis of OCD as determined by the Structured Clinical Interview for DSM-IV Disorders (First et al., 1999), aged 18 years and over and referred to one of the participating second-line mental health care centers. Comprehensive measurements and face to face interviews were performed at baseline and after two, four and six year follow-up by a trained research staff at one of the participating mental health care centers. Detailed sample characteristics and methodology of NOCDA are described elsewhere (Schuurmans et al., 2012).

For the purpose of the current study, only participants who fulfilled a current OCD diagnosis at baseline were included (N = 382). Since NOCDA is a naturalistic study, exclusion criteria were limited to an inadequate understanding of the Dutch language. The longitudinal association between OC cognitions and symptoms was assessed at two

time points: baseline and two-year follow-up (FU2). Of the 382 participants who provided baseline data, 104 (27.2%) participants were unavailable at FU2 (See Section 3.2).

2.2. Measures

2.2.1. OCD symptoms

OCD symptom severity was measured by the *Yale-Brown Obsessive-Compulsive severity Scale* (Y-BOCS; Goodman et al., 1989), a clinician-rated, 10-item severity scale, with each item rated from 0 (no symptoms) to 4 (extreme symptoms). It assesses current severity of obsessions and compulsions with total scores ranging from 0 to 40, and is the gold standard for assessing the severity of OCD symptoms. Its validity and reliability are well documented (Anholt et al., 2009). The *Padua Inventory-Revised* (PI-R; Sanavio, 1988) was used to assess OCD symptom subtypes. The PI-R consists of 41 self-rated items, which are rated on a five-point scale from 0 (not at all) to 5 (very much). This version entails 5 subscales: impulses (e.g., "in certain situations, I am afraid of losing my self-control and doing embarrassing things"), washing (e.g., "if I touch something which I think is 'contaminated' I immediately have to wash or clean myself"), checking (e.g., "I tend to keep on checking things more often than necessary"), rumination (e.g., "when doubts and worries come to my mind, I cannot rest until I have talked them over with a reassuring person") and precision (e.g., "before going to sleep I have to do certain things in certain order"). The PI-R was found to have excellent validity (van Oppen et al., 1995). For the purpose of the longitudinal analysis, we used the OCD symptoms data from all measurement points (i.e., baseline and FU2).

2.2.2. Depressive symptoms

We used the *Beck Depression Inventory* (BDI; Beck et al., 1961), a self-report inventory, to assess the severity of depressive symptoms. The BDI is one of the most widely used instruments for measuring the severity of depression in clinical research, with excellent reliability and validity (Beck and Steer, 1988).

2.2.3. Anxiety symptoms

Anxiety and panic symptoms were measured with the 21-item *Beck Anxiety Inventory* (Beck et al., 1988) a widely used and validated instrument. The BAI is a self-report instrument which assesses both somatic and cognitive components of anxiety. For the purpose of the current study, we used anxiety and depressive symptoms data from the baseline measurement.

2.2.4. OCD cognitions

We used the *Interpretation of Intrusions Inventory* (III; Obsessive Compulsive Cognitions Working Group, 2001) to assess OCD cognitions. The III is a 31-item semi-idiographic questionnaire that assesses erroneous appraisals of intrusive thoughts, images or impulses. The 31 items form three subscales: (a) importance of thoughts (e.g., "having this unwanted thought means I will act on it"), (b) responsibility (e.g., "if I ignore this thought, I could be responsible for serious harm"), and (c) control of thoughts (e.g., "I should be able to rid my mind of this thought"). With the help of a clinician, respondents identified one or two obsessional thoughts they have recently experienced that are either frequent or distressing. They then rated the extent to which they believe in each of the 31 statements as related to the identified intrusive thoughts, on a scale range from 0 ("did not believe this idea at all") to 100 ("convinced this idea was true"). Factor analysis of the III yielded a single factor, suggesting the total score be used in lieu of the 3 rationally derived subscales (Steketee et al., 2005). Therefore, we summed the three subscales of the III and calculated the III total score. Previous studies indicated that the III has good to excellent reliability and validity (Steketee et al., 2005, 2003). For the purpose of analysis, the III scores were divided by 100. In the NOCDA study, the III was administered in baseline and FU2. We used the III data from both time

points.

2.2.5. Treatment information

Information about treatment between baseline and FU2 assessments was gathered through yes/no questions regarding contact patients had with mental health professionals at that time. Most of the participants (N = 248, 90.2%) contacted a mental health professional between baseline and FU2. In addition, information about selective serotonin reuptake inhibitors (SSRI) use over the past two weeks was gathered both at baseline and at FU2.

2.3. Analytic strategy

First we used independent *t*-tests and chi-square statistics to compare demographic and psychosocial characteristics of participants that were available at FU2 to those who dropped from the study. Next we analyzed the cross-sectional associations between OC cognitions and OC, depressive and anxiety symptoms at baseline using a series of regression analyses. The total III score was separately analyzed as the predictor of OCD symptom severity and subtypes as well as of comorbid anxiety and depressive symptoms. A total of 16 models were analyzed. First, unadjusted regression analyses were conducted to describe the associations between OC cognitions and symptom severity, subtypes and comorbid anxiety and depressive symptoms. In order to measure the unique associations between cognitions and symptoms, and due to possible overlap between various symptoms, we conducted additional adjusted analyses as following: 1) a model testing the association between OCD cognitions and symptom severity, adjusted for the effects of depressive and anxiety symptoms; 2) a model testing the association between OCD cognitions and symptom subtypes, adjusted for the effects of depressive and anxiety symptoms; 3) a model testing the association between OC cognitions and anxiety symptoms, adjusted for OCD severity and subtypes; 4) a model testing the association between OC cognitions and depressive symptoms, adjusted for OCD severity and subtypes. Due to multiple comparisons, we applied the Bonferroni correction with *p* level of significance at $p < .003 (.05/16)$. All analyses were conducted in SPSS (IBM, 21.0).

In order to examine the directionality of the relationship between OCD cognitions and symptoms and to test whether cognitions contribute to the maintenance of OCD symptoms at FU2, we conducted cross-lagged panel analyses, using AMOS program (IBM 21.0). A cross-lagged design is useful for examining the interrelationships between two (or more) constructs over time. This method requires at least two variables measured with synchronicity at two time points. If the causal structure between *x* and *y* does not change over time (i.e., if the assumption of stability is met), then the difference between cross-lagged correlations provides potentially useful information about the effect of *x* on *y* and vice versa (Hays et al., 1994). In addition, this analysis allows a simultaneous examination of the relationships between cognitions and symptoms across different time measurements. Fig. 1 presents the tested cross-lagged model. Briefly, this analysis yields three types of associations: a synchronous association, that corresponds to the

Fig. 1. Structural model of the cross-lagged analyses used to examine prospective associations between OCD cognitions and symptoms. Note: Pathways (S1) and (S2) denote synchronous associations at baseline and two year follow up, respectively. Pathways AR_{III} and AR_{OCD} denote autoregressive/stability associations at baseline and two year follow up. CL_{III→OCD} and CL_{OCD→III} denote the cross-lagged associations between baseline and two year follow up.

relationship between *x* and *y* at the same time point; an autoregressive association, that constitutes the stability effects of each variable in between baseline and follow up; a cross-lagged association, which is the effect of *x* at baseline on *y* at follow-up, or vice versa. Six cross-lagged models were analyzed, specifying the relationship between OCD cognitions with OCD severity and five symptoms subtypes measured by the PI-R. We followed guidelines for evaluating model fit using the selected fit indices: Non-normed-fit index (NNFI), with values $\geq .90$ indicating an acceptable fit, comparative-fit-index (CFI) with values over .90 represent good fit and the root mean square error of approximation (RMSEA), where values below .06 shows a good fit (Bentler and Bonett, 1980; Hu and Bentler, 1999). We handled missing data using full information maximum likelihood (FIML) estimates provided by the AMOS program (IBM 21.0), which allowed us to include all available data (any participant with data at baseline). In comparison with other methods such as means imputation, the FIML method was found to produce the least bias estimates of missing values (Shahar and Davidson, 2003).

3. Results

3.1. Characteristics of the study sample

Demographic data was collected from 382 OCD patients at baseline. The participants reported a mean age of 36.36 (10.92). Most of the participants were females ($n = 216$, 56.5%), and approximately one third of the sample were married ($n = 123$, 32.2%). At intake, a total of 163 participants (42.7%) reported using SSRI. Table 1 presents estimated means, standard deviations and bivariate correlations of the study variables.

3.2. Attrition analysis

In order to examine whether incomplete follow-up assessments were associated with the tested variables, we conducted an attrition analysis by comparing patients who completed both baseline and FU2 measurements ($N = 278$) to the patients who were absent from FU2 ($N = 104$). Independent *t*-tests and cross tabulation analyses were conducted on baseline measures of OCD subtypes and severity, anxiety and depressive symptoms, as well as OC cognitions, SSRI use and demographics. We found that compared to completers, non-completers had significantly increased depressive and anxiety symptom severity scores at baseline ($p < .05$). Therefore, these variables were entered as covariates in the prospective analyses.

Table 1
Descriptive statistics for the study variables.

Variable	M (SD)	YB	PI	BDI	BAI	III	YB FU2	PI FU2
<i>Baseline</i>								
Total Y-BOCS severity (YB)	21.18 (7.04)							
Padua inventory (PI)	62.16 (26.81)	.55**						
Beck Depression Inventory (BDI)	15.96 (9.81)	.44**	.57**					
Beck Anxiety Inventory (BAI)	18.19 (12.02)	.40**	.56**	.67**				
Interpretation of intrusion inventory (III)	14.70 (6.91)	.28**	.45**	.44**	.48**			
<i>FU2</i>								
Total Y-BOCS severity (YB FU2)	15.11 (8.98)	.49**	.45**	.30**	.28**	.18*		
Padua inventory (PI FU2)	46.30 (29.92)	.40**	.72**	.39**	.44**	.41**	.65**	
Interpretation of intrusion inventory	11.37 (7.20)	.20*	.40**	.37**	.38**	.55**	.41**	.57**

Note. Y-BOCS = Yale Brown Obsessive Compulsive Scale; FU2 = two year follow up.

* $p = .001$.

** $p < .0001$.

Table 2

Multiple regression analyses examining the effect of OC cognitions on OCD, anxiety and depressive symptoms.

Variable	R^2 change	β	<i>t</i>	<i>p</i>
Y-BOCS				
Unadjusted	.051	.226	4.509	.000
Adjusted for: BDI, BAI	.001	.040	.716	.474
PI-R: impulses				
Unadjusted	.175	.419	8.765	.000
Adjusted for: BDI, BAI	.035	.215	4.259	.000
PI-R: washing				
Unadjusted	.014	.118	2.261	.024
Adjusted for: BDI, BAI	.000	.003	.048	.962
PI-R: checking				
Unadjusted	.053	.236	4.604	.000
Adjusted for: BDI, BAI	.011	.122	2.136	.033
PI-R: rumination				
Unadjusted	.217	.466	10.008	.000
Adjusted for: BDI, BAI	.032	.205	4.500	.000
PI-R: precision				
Unadjusted	.084	.289	5.742	.000
Adjusted for: BDI, BAI	.018	.155	2.753	.006
BDI				
Unadjusted	.190	.436	9.422	.000
Adjusted for: Y-BOCS, PI-R	.050	.246	5.461	.000
BAI				
Unadjusted	.211	.459	9.796	.000
Adjusted for: Y-BOCS, PI-R	.065	.283	6.003	.000

Note: The III was the predictor in both unadjusted and adjusted models.

Y-BOCS = Yale Brown Obsessive compulsive severity scale; PI-R = Padua Inventory-Revised; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory.

3.3. Specificity: cross-sectional associations between OC cognitions and OCD, depressive and anxiety symptoms

Table 2 presents multiple regression analyses describing cross-sectional associations between the III and OCD, depressive and anxiety symptoms. Except for the washing subscale, all of the unadjusted associations between the III and both OCD and comorbid symptoms were statistically significant (β coefficients = .22–.46, $p < .003$). As evident from Table 2, the association between the III and symptom severity as measured by the Y-BOCS diminished after adjusting for comorbid anxiety and depressive symptoms ($\beta = .04$, $t = .71$, $p = \text{ns}$). We did find specific associations between the III and comorbid symptom severity adjusted for OCD symptomatology (BDI: $\beta = .24$, $t = 5.46$, $p < .003$; BAI: $\beta = .28$, $t = 6.00$, $p < .003$). In addition, we identified unique associations between the III and the impulses and ruminations subscales of the PI-R, adjusted for comorbid symptoms (impulses: $\beta = .21$, $t = 4.25$, $p < .003$; ruminations: $\beta = .20$, $t = 4.50$, $p < .003$).

Table 3
Standardized regression coefficients for the cross-lagged models.

OCD symptoms	Synchronous		Autoregressive		Cross-Lagged	
	S1	S2	AR _{III}	AR _{OCD}	CL _{III→OCD}	CL _{OCD→III}
Y-BOCS	.05	.41***	.58***	.40***	.08	.04
Padua Inventory subscales						
Impulses	.21***	.40***	.51***	.79***	– .04	.17**
Washing	.00	.31***	.58***	.72***	.07	.10
Checking	.11*	.41***	.59***	.68***	.11*	.00
Rumination	.24***	.53***	.53***	.56***	.12*	.12*
Precision	.14**	.43***	.55***	.72***	.12**	.11*

Note: The left column represents the OCD symptoms assessments used for the six cross-lagged models. Each row describes a separate model.

All associations are adjusted for baseline depressive and anxiety severity.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

3.4. Prospective associations between OC cognitions and OCD symptoms

3.4.1. Covariates

Since the majority of participants contacted a mental health care professional between baseline and FU2, we first examined whether a change in OCD symptoms and the III scores between baseline and FU2 was associated with the patients' contact with a health care professional. We calculated the difference between the two measurements (i.e. baseline and FU2) for the Y-BOCS severity scale, the PI-R and the III score. Using independent *t*-tests, we examined whether there was a significant difference between the change in symptoms and cognitions score from baseline to FU2 as a function of the contact with a health care professional. We found no significant between group differences for any of the above mentioned measures. Similarly, SSRI use at FU2 was unrelated to change both in OCD cognitions and symptoms. Due to significant differences in baseline scores of BAI and BDI between completers and non-completers, depression and anxiety symptoms at baseline were entered to the model as covariates.

3.4.2. Cross-lagged models

Tables 3, 4 present the coefficients of the synchronous, autoregressive and cross-lagged associations and their corresponding fit indices, respectively. All the analyzed models demonstrated an excellent fit to the data (i.e. NNFI: .96–1.00; CFI: .98–1.00; RMSEA: .00–.04). As evident from Table 3, autoregressive paths demonstrated a relative stability of cognitions and symptoms between baseline and FU2. Stability effects of OC cognitions across models were moderate, and differed depending on the instrument analyzed; the Y-BOCS severity scale demonstrated a moderate stability effect (β coefficient = .40) and the PI-R showed a moderate to high stability over two year, as a function of symptom subtype (β coefficients: .56–.79). Baseline synchronous effects were generally small, as the associations between OCD cognitions and symptoms were adjusted for depressive and anxiety

Table 4
Fit Indices of the cross lagged models.

Model	χ^2	Df	NNFI	CFI	RMSEA
Y-BOCS severity scale	13.20	12	.99	.99	.01
Padua Inventory subscales					
Impulses	21.68*	12	.96	.98	.04
Washing	19.20	12	.97	.99	.04
Checking	14.82	12	.98	.99	.02
Rumination	13.94	12	.99	.99	.02
Precision	10.38	12	1.00	1.00	.00

Note: NNFI: non-normed fit index; CFI: comparative fit index; RMSEA: root-mean-square error of approximation.

* $p = .04$.

symptoms. The synchronous associations in the subsequent wave were moderate; however, the correlations between cognitions and OCD symptoms at FU2 were unadjusted for mood symptomatology.

3.4.3. Reciprocal relations between OC cognitions and OCD symptoms

In order to examine the directionality of the relationship between cognitions and symptoms, as well as to test the contribution of cognitions to symptoms at FU2, we examined the cross-lagged coefficients (β values). As shown in Table 3, the cross-lagged path from baseline OC cognitions to symptom severity as measured by the Y-BOCS at FU2 was not significant ($\beta = .05$, $p = .30$). Similarly, the path from symptom severity at baseline to cognitions at FU2 was non-significant ($\beta = .08$, $p = .15$). Next, we observed the six cross-lagged models in which OCD symptoms were assessed via the PI-R. In the impulses subscale model, cognitions did not predict symptoms at FU2 ($\beta = -.04$, $p = .37$). However, a significant cross-lagged association was evident as the impulses subscale predicted III score at FU2 ($\beta = .16$, $p < .01$). The washing subscale demonstrated no significant cross-lagged paths between cognitions and symptoms, although the path predicting cognitions from symptoms showed a trend toward significance ($\beta = .10$, $p = .06$). In the checking subscale, baseline cognitions significantly predicted symptoms at FU2 ($\beta = .11$, $p < .05$), while the cross lagged path from symptoms to cognitions was non-significant. For both rumination and precision subscales, the cross-lagged paths from cognitions to symptoms and symptoms to cognitions demonstrated small but significant loadings (β : .11–.12, $p < .05$).

4. Discussion

In the past decade there has been considerable debate in the literature regarding the role of cognitions in OCD symptoms (e.g., [Anholt and Kalanthroff, 2013](#)). This debate is of paramount importance to OCD theory and research since it addresses one of the basic assumptions of cognitive behavioral therapy (CBT) for OCD: that interpretation of intrusive thoughts is related to the development and maintenance of OCD symptoms. This study replicated and extended previous findings regarding the role of cognitions in OCD, in a large clinical sample. The first hypothesis was partly supported, as the cross-sectional analyses demonstrated a nonspecific association between cognitions and OCD symptoms, particularly with respect to symptom severity. The second hypothesis concerning the longitudinal analysis was not supported. Prospectively, baseline OC cognitions did not predict OCD symptom severity at FU2, and there was a largely bidirectional association between cognitions and symptom subtypes.

In this study we used the III to assess OC cognitions, which, as opposed to the OBQ, explicitly addresses the *appraisals* of one's idiosyncratic intrusions. One would expect that more negative interpretations of intrusions will be associated with more severe symptoms. It is possible that biased interpretations are related to other aspects of OCD. For example, several studies suggested that effortful strategies to deal with intrusions are particularly relevant to the emotional disturbance often manifested among OCD patients ([Fergus and Carmin, 2014](#); [Freeston et al., 1992](#)). Conversely, [Taylor et al. \(2010\)](#) suggested that controlling for depression and anxiety is tantamount to partialling out some of the effects cognitions have on OCD symptoms. Nonetheless, in this study OCD cognitions were uniquely associated to concurrent depressive and anxiety severity rather than to OCD severity, which is consistent with the notion that OC cognitions are not specific to OCD and might be related to comorbid symptom severity, even among OCD patients.

Consistent with the specific relationship demonstrated between cognitions and impulses subtype, [Taylor et al. \(2010\)](#) reported that the high belief subgroup of OCD patients demonstrated elevated levels on the impulses subscale. However, [Steketee et al. \(2005\)](#) found that the impulses subscale was not associated with the III or with the OBQ, controlling for depression and anxiety symptoms. Interestingly, we found the direction of the relationship between cognitions and impulses

subtype to be reversed than the direction that would be predicted by the cognitive model (i.e. symptoms predicted cognitions). Perhaps individuals view their obsessions related to impulses and harm to self and/or others and, over time, develop post-hoc beliefs such as: “if I have such horrible thoughts I must be careful or else I’ll be responsible for doing harm/I should be able to control such thoughts”. Consistent with our finding, the rumination subtype (which is akin to the doubting and obsessing subscales in other OCD self-report questionnaires; McKay et al., 2004) was associated with the III in several studies (Abramowitz et al., 2009; Julien et al., 2008; Tolin et al., 2008). Nonetheless, since the PI-R was constructed prior to the OBQ and the III, some of the items, particularly of the rumination subscale, may overlap to items of the III describing importance/control of thought. For example: “unpleasant thoughts come into my mind against my will and I cannot get rid of them” from the rumination subscale, and “having intrusive thoughts means I am out of control” from the III. This might also explain the unique association found between cognitions and this subscale.

With respect to the relationship between cognitions and OCD symptom subtypes, some reservation should be made, as several studies observed that OCD symptoms may be represented by a single, general dimension (e.g. Olatunji et al., 2015). Thus, inconsistency in the literature with respect to the relationship between specific symptoms and cognitions may be explained, to some extent, by the possibility that categorizing symptoms to subtypes is not reflective of the clinical reality. For example, according to the dimensional approach in OCD, checking and reassurance seeking compulsions are associated with cognitions about causing harm (Abramowitz et al., 2010). However, a patient suffering from unacceptable thoughts or contamination fears might be similarly preoccupied with doubting, reassurance seeking and checking behaviors, and not present with increased cognitions about causing harm. In addition, perhaps the heterogeneous and idiosyncratic nature of OCD symptoms challenges the development of content valid instruments (Abramowitz et al., 2010). Thus, assessment of OCD symptoms based on symptom function rather than content may be more useful and valid in describing the OCD phenomena. Efforts in this direction were made by Abramowitz et al. (2010), who constructed an instrument assessing OCD symptoms based on function rather than form. However, due to the idiosyncratic nature of OCD, it is likely that there are numerous motivations behind the symptoms which are too heterogeneous to categorize. Future studies should further elucidate this challenge of OCD assessment.

Results of the cross-lagged analyses were mostly inconsistent with previous studies, which relied on non-clinical populations. For example, among undergraduate students, OCD related beliefs at baseline significantly predicted changes in distress associated with OCD symptoms (Coles et al., 2008). Abramowitz et al. (2006) found that among new mothers and fathers, OC cognitions at childbirth significantly predicted OCD symptoms postpartum. Finally, Novara et al. (2011) examined longitudinal aspects of OCD cognitions in a five-year prospective study of university students, demonstrating that OCD cognitions predicted symptoms only at baseline. All of the above-mentioned studies focused on the hypothesized relationship according to which cognitions predict symptoms, excluding analyses of the opposite association. Thus, while two of the three above-mentioned studies supported the cognitive model, they did not attempt to repudiate the alternative (i.e. that symptoms derive cognitions). In line with our findings regarding the bidirectional relationship between OC cognition and symptom subtypes, Gillan and Robins (2014) postulated that the cognitive biases characteristic of OCD may be a consequence of compulsive behavior. Similarly, Anholt and colleagues suggested that OCD patients observe their own behavioral tendencies and reactions associated with intrusions, and interpret these as indicating that the intrusions are important. Over time generalizations of this process are established in the form of metacognitive beliefs (Anholt et al., 2012).

The cognitive theory of OCD is rooted in the notion that cognitive change predicts behavioral change in symptoms (Beck et al., 1979).

However, there are findings questioning the precedence of cognitive over behavioral change. For example, Su et al. (2016) examined OC cognitions as mediators of symptom change during exposure and response prevention among OCD patients. The researchers found that none of the cognitions mediated subsequent symptom reduction. In addition, Woody et al. (2011) found that among OCD patients entering treatment, more severe obsessional symptoms led to a strengthening of appraisals of personal significance over time. Their analysis showed that prior obsession severity is a convincing leading indicator of subsequent change in appraisals of personal significance. Similarly, Craske et al. (2014) suggested that exposure practice can elicit belief disconfirmation in CBT for OCD. This is consistent with the finding demonstrating that some of the symptom subtypes predicted cognitions at FU2.

There are some inherent limitations in the present study. First, the longitudinal analyses are based on two-year span data. This time frame may be too extensive to test whether cognitions predict OCD symptoms, as suggested by the cognitive model (Salkovskis, 1985). Nonetheless, OCD symptoms tend to be chronic and relatively stable (Tibi et al., 2017; Visser et al., 2014). Furthermore, in the current study we found a sizable autoregressive effect of most of the OCD symptoms, suggesting that the symptoms were stable over time (Selig and Little, 2012). However, it emerged that baseline cognitions did not contribute to the maintenance of symptoms. Having said that, the mean Y-BOCS severity scale score decreased at FU2 to the near subclinical range. Nonetheless, it is expected that cognitions will contribute to the variance of symptoms beyond symptoms at baseline. Another limitation concerns inferences from a longitudinal design with two time points. In order to establish the directionality of OCD cognitions and symptoms, future studies should include multiple assessment points. In addition, the self-report instruments we used in this study might measure nonspecific aspects such as negative affect, which limits the ability to draw inferences on the specific relationship between OC cognitions and various symptoms. A related limitation concerns the possible overlap between the III and the PI-R items, which makes it difficult to differentiate cognitions from symptoms. Future studies should investigate the prospective relationship between cognitions and symptoms using up-to-date dimensional OCD symptom measures. Moreover, the fact that the NOCDA study included patients diagnosed with OCD, limits the ability to study the relationship between OC cognitions and OCD symptoms, since it is impossible to make inferences about causality or etiology in an already diagnosed population. However, we can surmise about the relevance of OC cognitions to the course of OCD. Finally, given the naturalistic design of the study, we did not control treatments received by the participants between baseline and FU2. Nonetheless, this limitation was partly mitigated as there were no significant associations between the contact the patients had with a health care professional and OC cognitions or symptoms. Future longitudinal studies should carefully monitor treatments received by the participants throughout the follow-up period (i.e., assess the presence of exposure sessions and homework assignments).

Strengths of this study include a prospective investigation of the relationship between cognitions and symptoms in a large clinical sample of OCD patients, and the use of a multidimensional approach to measure OCD symptomatology. The clinical implications of our study concern the implementation of cognitive interventions in OCD, which are based on the cognitive model postulated by Salkovskis (1985). In their discussion on maximizing the effect of exposure therapy, Craske et al. (2014) suggested that cognitive restructuring should not be employed before or during exposure sessions, as it might reduce the expectancy of an aversive outcome (i.e., “how likely is it that you’ll cause harm in case you’ll leave the light on?”) and thus mitigate extinction learning. In line with the finding that behavioral symptoms are predictive of OC cognitions, the researchers speculated that the exposure should precede cognitive interventions, which serve to consolidate what was learned during exposure practice.

Our findings emphasize the need to focus clinical research efforts on alternative mechanisms to those based on the traditional cognitive theory of OCD. While some cognitive models emphasize the role of changing cognitions as an antecedent for symptoms change, others incorporate issues of identity, values and attachment style as potential facilitators of treatment outcome for OCD (Doron and Moulding, 2009; Haaland et al., 2011; Sookman et al., 1994). These approaches suggest intriguing and promising lines for the enhancement of behavioral therapy for OCD. Additional research directions may include adding components of evidence based interventions such as acceptance and commitment therapy addressing psychological flexibility and cognitive diffusion (Bluett et al., 2014).

Acknowledgements

The research infrastructure needed to complete the baseline measurements was financed almost exclusively by the participating organizations: Academic Department VU Medical Centre/GGZinGeest, Amsterdam, The Netherlands; Innova Research Centre, Mental Health Care Institute GGZ Centraal, Marinade Wolf Anxiety Research Centre, Ermelo, The Netherlands; Center for Anxiety Disorders “Overwaal”, Lent, The Netherlands; Dimence, GGZ Overijssel; Department of Psychiatry, Leiden University Medical Centre, Leiden, The Netherlands; ‘Vincent van Gogh Institute’ Mental Health Care Centre Noorden Midden-Limburg, Venray, The Netherlands; Academic Anxiety Center, PsyQ Maastricht University, Division Mental Health and Neuroscience, Maastricht, The Netherlands, except for the field work coordinator, which was financed by a research grant number: 5.6.2.180505 from ‘Stichting tot Steun VCVGZ’, awarded to Patricia van Oppen and Anton J. L. M. van Balkom.

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