J. Behav. Ther. & Exp. Psychiat. 57 (2017) 113–117

Contents lists available at ScienceDirect

Journal of Behavior Therapy and Experimental Psychiatry

journal homepage: www.elsevier.com/locate/jbtep

Anxiety sensitivity as a predictor of outcome in the treatment of obsessive-compulsive disorder

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ARTICLE INFO

Article history:
Received 8 April 2016
Received in revised form
20 February 2017
Accepted 2 May 2017
Available online 6 May 2017

ABSTRACT

Background and objectives: To address the fact that not all individuals who receive cognitive-behavioral therapy (CBT) for obsessive-compulsive disorder (OCD) exhibit complete symptom reduction, research has examined factors that predict outcome; however, no studies have examined anxiety sensitivity (AS) as a predictor of outcome of CBT for OCD. AS refers to the fear of anxious arousal that results from mistaken beliefs about the dangerousness of anxiety-related body sensations. It is important to understand whether AS influences OCD treatment outcome, considering that (a) some obsessions directly relate to AS, and (b) OCD patients with high AS may be reluctant to engage in anxiety-provoking components of CBT for OCD.
Methods: Patients ($N = 187$) with a primary diagnosis of OCD who received residential CBT for OCD participated in this study, which involved completing a self-report battery at pre- and post-treatment.
Results: Results supported study hypotheses, in that (a) baseline AS positively correlated with baseline OCD severity, and (b) greater baseline AS prospectively predicted higher posttreatment OCD symptom severity even after controlling for pretreatment OCD and depression severity.
Limitations: The study was limited by its use of an older measure of AS, reliance on self-report measures, and nonstandardized treatment across participants.
Conclusions: Findings highlight the importance of AS in the nature and treatment of OCD. Clinical implications and future directions are discussed.

Obsessive-compulsive disorder (OCD) is characterized by distressing, unwanted intrusive thoughts, images, and doubts (i.e., obsessions) and/or urges to perform repetitive, deliberate rituals and other anxiety-reduction strategies to neutralize this distress (i.e., compulsions) (American Psychiatric Association [APA], 2013). Compulsive rituals are reinforced by the immediate decrease in anxiety they engender, yet the anxiety reduction is temporary and compulsions prevent the natural extinction of obsessional fear in the long term. Obsessive-compulsive symptoms cause considerable distress and functional impairment among the 2–3% of the population that experiences OCD at some point in their lifetime (Kessler et al., 2005).

Cognitive-behavioral therapy (CBT) using exposure and response prevention (ERP) procedures can be an effective treatment for OCD (e.g., Olatunji, Cisler, & Deacon, 2010). Exposure entails repeated systematic confrontation with situations and stimuli that provoke obsessional anxiety; response prevention involves resisting urges to perform escape and avoidance behaviors (e.g., compulsive rituals) during and after exposure trials. Yet despite its established efficacy, response to this intervention varies widely and some individuals are not able to adhere or respond. Accordingly, researchers have sought to identify factors that predict outcome. Severe depression and baseline OCD symptoms, for example, have been associated with attenuated response to CBT for OCD across a large body of research (Abramowitz & Foa, 2000; Abramowitz, Franklin, Kozak, Street, & Foa, 2000; Farrell et al., 2016; Foa et al., 1983; Knopp, Knowles, Bee, Lovell, & Bower, 2013; Steketee, Chambless, & Tran, 2001), although the relationship between baseline depression and OCD treatment outcome is not consistent and does not appear to influence outcomes in the

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http://dx.doi.org/10.1016/j.jbtep.2017.05.003
0005-7916/

long-term (e.g., Anholt et al., 2011).

To date, no studies have examined *anxiety sensitivity* (AS) as a predictor of CBT outcome in OCD. AS refers to mistaken beliefs about the dangerousness of anxiety-related body sensations that generate the fear of anxious arousal (e.g., Reiss & McNally, 1985). Specifically, individuals with elevated AS are hypervigilant to and (mis)appraise ambiguous body sensations as particularly dangerous. For example, someone with high anxiety sensitivity might misinterpret chest tightness as a sign of a heart attack, dizziness as a sign of "losing control," or racing thoughts as an indicator that one is "going crazy" and about do something embarrassing or harmful. Although AS is often associated with panic disorder, it is considered a transdiagnostic process (Taylor, 1999); moreover, AS has demonstrated moderate to strong associations with OCD symptom severity in individuals diagnosed with OCD (Pearson *r*s range 0.28–0.30; partial *r*s range 0.28–0.64; Calamari, Rector, Woodard, Cohen, & Chik, 2008; Deacon & Abramowitz, 2006; Laposa, Collimore, Hawley, & Rector, 2015; Norton, Sexton, Walker, & Norton, 2005; Zinbarg, Barlow, & Brown, 1997; for a review see Robinson & Freeston, 2014) as well among nonclinical individuals (Pearson *r*s range 0.30–0.56; David et al., 2009; Keough, Riccardi, Timpano, Mitchell, & Schmidt, 2010; Sexton, Norton, Walker, & Norton, 2003; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012).

Although research in this area is limited, a few studies have highlighted relationships between specific *dimensions* of AS (i.e., the fear of anxiety and/or anxious arousal because of feared physical, mental, or social consequences) and OCD symptoms (e.g., Wheaton, Mahaffey, Timpano, Berman, & Abramowitz, 2012). Previous investigators have posited that the cognitive dimension of AS in particular is strongly related to OCD given that misappraisals of one's own thinking (e.g., overestimating the importance of thoughts) are a cardinal feature of OCD (Rachman, 1997, 1998). Although some research supports this hypothesis (e.g., Cox, Borger, & Enns, 1999; Sexton et al., 2003; Wheaton et al., 2012), aggregate findings are inconsistent (Deacon & Abramowitz, 2006). Moreover, the cross-sectional nature of previous studies precludes investigators from determining whether AS predicts changes in OCD symptom severity *over time* (e.g., following treatment).

There are a number of reasons to examine AS as a predictor of poorer outcome with CBT for OCD. First, anxious arousal is often provoked during exposure therapy. Thus, individuals with high AS are apt to become afraid not only of exposure stimuli per se, but also the arousal sensations induced when conducting exposures. Given that AS is a strong predictor of panic attacks (e.g., Schmidt, Zvolensky, & Maner, 2006), OCD patients with high AS might be prone to experiencing panic episodes during exposure, which could lead to avoidance behavior and hamper both adherence and confidence in the treatment techniques, thereby leading to suboptimal outcome. Furthermore, some patients' obsessions might directly relate to ambiguous somatic sensations; for example, a patient who presents with contamination-related OCD may be especially hypervigilant for (and anxious in response to) feelings of nausea, which often accompany anxious arousal.

One methodological obstacle to examining predictors of outcome in OCD is that patients with high levels of concurrent anxiety and depression (i.e., comorbidity) are often excluded from randomized controlled trials (RCTs) in efforts to maximize the internal validity of such studies. Yet this restricts the range of comorbid psychopathology variables (e.g., depression, AS), potentially obscuring relationships in secondary analyses of outcome predictors. We addressed this issue in the present investigation by using a large sample of individuals with OCD seeking treatment outside the context of an RCT. Our data were collected within a residential program specializing in CBT for OCD in which individuals often met criteria for comorbid conditions (e.g., major depression) and were concurrently using psychotropic medication. Although these sample characteristics might attenuate internal validity (e.g., treatment was less standardized than in controlled studies), it afforded the best opportunity to observe the effects of AS on CBT response in a diverse OCD clinical sample receiving CBT in a service setting.

The present study was designed to examine the extent to which baseline levels of AS predict treatment outcome in a sample of individuals with a clinical diagnosis of OCD undergoing CBT above and beyond established predictors of attenuated response (i.e., baseline OCD and depressive symptom severity). On the basis of previous research and the conceptual considerations noted above, we expected that baseline AS would be positively correlated with baseline OCD severity. We also hypothesized that greater baseline levels of AS would prospectively predict higher posttreatment OCD symptom severity above and beyond pretreatment OCD and depression severity.

1. Method

1.1. Participants

Participants were considered eligible to participate in the current study if they (a) had a primary diagnosis of OCD, (b) were admitted for residential treatment at the Obsessive-Compulsive Disorders Center at Rogers Memorial Hospital (RMH) in Oconomowoc, Wisconsin, (c) provided informed consent to participate, and (d) had completed pre- and post-treatment assessments. Patients with comorbid psychotic symptoms or current substance abuse were considered ineligible. On average, approximately 28.5 h of ERP were completed per week, including both staff-assisted and self-directed exposure practice. Most participants ($n = 163$; 87.2%) were also taking psychiatric medications; primarily, selective serotonin reuptake inhibitors (SSRIs; $n = 125$; 66.8%). Psychiatric medications were adjusted upon the on-site psychiatrist's assessments of the patient's needs (information on the precise number of participants whose medication changed during the course of CBT was unfortunately not available). The majority ($n = 153$; 81.8%) had secondary diagnoses, which are shown in Table 1.

The final sample included 187 adults (51.9% women; $n = 97$) who had a mean age of 30.49 years ($SD = 12.24$) and had received

Table 1
Secondary diagnosis of study participants.

Secondary diagnosis	*n*	(%)
Mood disorders	62	(33.2)
Major depressive disorder	10	(5.3)
Bipolar I disorder	1	(0.5)
Other mood disorder	51	(27.3)
Anxiety-related disorders	36	(19.3)
Panic disorder	4	(2.1)
Posttraumatic stress disorder	1	(0.5)
Social anxiety disorder	10	(5.3)
General anxiety disorder	21	(11.2)
Obsessive-compulsive and related disorders	5	(2.7)
Trichotillomania	1	(0.5)
Tic disorder/Tourette's syndrome	1	(0.5)
Body dysmorphic disorder	3	(1.6)
Developmental disorders	2	(1.1)
Attention deficit/hyperactivity disorder	1	(0.5)
Learning disability	1	(0.5)
Eating disorders	3	(1.6)
Anorexia nervosa	1	(0.5)
Bulimia nervosa	1	(0.5)
Other eating disorder	1	(0.5)
Other psychological disorder	45	(24.1)

an average of 14.93 years ($SD = 2.29$) of education. The majority of participants ($n = 167$; 89.3%) identified as White/European American, with 3.7% ($n = 7$) identifying as Asian and 1.1% ($n = 2$) identifying as African American. Eight (4.3%) participants self-identified as being of Hispanic origin. Three participants (1.6%) did not self-disclose their race/ethnicity. Most participants ($n = 128$; 68.4%) were single; 24.6% ($n = 46$) were married and 2.1% ($n = 4$) were divorced (marital status was undocumented for two participants).

1.2. Procedure

1.2.1. Assessment

Prior to admission to the residential treatment program, all prospective patients completed an initial assessment with a trained intake staff member. This included the clinician-rated version of the semi-structured Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, & Mazure, 1989a, 1989b) symptom checklist and severity rating to determine the presence of DSM-IV OCD (APA, 2000). The clinical director of the Obsessive-Compulsive Disorder Center ([AUTHOR INITIALS]) reviewed the results of this assessment with the intake interviewer and determined if the patient was appropriate for admission. Individuals were only admitted to the clinic for treatment, and thus included in the current study, if there was 100% diagnostic agreement between the intake interviewer and clinical director. Each participant also completed a self-report assessment packet that included the study measures described further below, which was re-administered at posttreatment. As part of the admissions process, patients provided consent to allow their responses to the study measures to be used for both clinical and research purposes. The consent procedures and study measures were approved by both the RMH Human Subjects Committee and the Rogers Center for Research and Training.

1.2.2. Treatment

Treatment consisted of ERP, which followed the procedures described by Kozak and Foa (1997). Therapists and participants developed a list of situations for exposure that triggered their anxiety from least to most feared and then assisted participants in facing these feared situations in a prolonged, repetitive, and graduated manner while at the same time helping them to resist engaging in avoidance behaviors and/or rituals. Participants also engaged in cognitive restructuring to help them examine and disconfirm their irrational beliefs. Additional interventions (e.g., behavioral activation for symptoms of depression) were often used to address comorbid symptoms. Further, participants met regularly with their therapist for non-CBT work (e.g., psychoeducation with family members), participated in a process group once per week, and participated in experiential therapy groups several times per week.

1.3. Measures

1.3.1. Dimensional Obsessive-Compulsive Scale (DOCS; Abramowitz et al., 2010)

The DOCS is a 20-item self-report measure with four empirically derived subscales that assesses the severity of the four most consistently replicated OCD symptom dimensions: (a) contamination (DOCS-C), (b) responsibility for harm and mistakes (DOCS-H), (c) unacceptable thoughts (DOCS-UT), and (d) symmetry/ordering (DOCS-S). Each subscale begins with a description of the symptom dimension as well as prototypical examples of fears, rituals, and avoidance behaviors. Next, within each symptom dimension, five items (rated 0 to 4) assess the following parameters of severity: (a) time occupied by obsessions and rituals, (b) avoidance behavior, (c) associated distress, (d) functional interference, and (e) difficulty disregarding the obsessions and refraining from the compulsions. The DOCS has demonstrated excellent reliability in clinical samples ($\alpha = 0.94$-0.96), and the measure converges well with other measures of OCD symptoms (Abramowitz et al., 2010).

1.3.2. Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996)

The BDI-II is a 21-item self-report scale that assesses the severity of affective, cognitive, motivational, vegetative, and psychomotor components of depression. Scores of 10 or less are considered normal; scores of 20 or greater suggest the presence of clinical depression. The BDI-II has excellent reliability and validity and is widely used in clinical research (Beck et al., 1996).

1.3.3. Anxiety Sensitivity Index (ASI; Reiss, Epstein, & Gursky, 1986)

The ASI is a unidimensional 16-item self-report measure of beliefs regarding the dangerousness of anxious arousal (e.g., "It scares me when my heart beats rapidly"). Participants rate their agreement with each statement on a 0 (*very little*) to 4 (*very much*) scale; higher scores indicate greater AS. The ASI has demonstrated adequate reliability and validity in previous research (Peterson & Reiss, 1992; Reiss, Peterson, Gursky, & McNally, 1986). Three ASI subscales were computed according to the principal components solution recommended by Taylor, Koch, Woody, and McLean (1996): ASI-Physical Concerns (items 6, 7, 8, 9, 10, and 11; Cronbach $\alpha = 0.81$), ASI-Mental Concerns (items 2, 12, and 15; Cronbach $\alpha = 0.80$), and ASI-Social Concerns (items, 1, 3, 4, 5, 13, 14, and 16; Cronbach $\alpha = 0.75$).

2. Results

Data were first screened to assess concordance with statistical assumptions. Distributions of scores on study measures were free of significant skew and kurtosis (all values < 2). DOCS, ASI, and BDI-II scores fell within the range of expected values. The sample exhibited clinically significant baseline AS ($M = 23.94$; $SD = 12.28$) and depressive symptoms ($M = 26.28$; $SD = 13.47$) prior to treatment. The sample's mean DOCS total score indicated clinically significant OCD symptom severity prior to treatment ($M = 32.73$; $SD = 15.07$). At posttreatment, the mean DOCS total score was 16.59 ($SD = 11.74$). A paired-samples t-test revealed that the change in DOCS total scores from pre-to posttreatment was significant, $t(123) = 13.22$, $p < 0.001$, $d = 1.18$.

To test our first hypothesis that AS would be associated with baseline OCD symptoms, we computed zero-order correlations between ASI total and subscale scores and pretreatment DOCS total scores. Correlations among study variables are presented in Table 2. These analyses revealed significant positive correlations among pretreatment DOCS and ASI total and subscale scores (all $ps < 0.001$).

Table 2
Zero-Order bivariate correlations among study variables.

Variable	1	2	3	4	5	6
1. Posttreatment DOCS	–					
2. Baseline DOCS	0.52	–				
3. Baseline BDI-II	0.26	0.36	–			
4. Baseline ASI-Total	0.45	0.39	0.42	–		
5. Baseline ASI-Physical	0.41	0.31	0.26	0.86	–	
6. Baseline ASI-Mental	0.35	0.27	0.48	0.76	0.45	–
7. Baseline ASI-Social	0.35	0.40	0.41	0.92	0.67	0.66

DOCS = Dimensional Obsessive-Compulsive Scale; BDI-II = Beck Depression Inventory-II; ASI = Anxiety Sensitivity Index; all $ps < 0.01$.

To test our second hypothesis that baseline scores on ASI subscales would predict posttreatment OCD symptom severity above and beyond baseline OCD and depressive symptom severity, we computed a hierarchical linear regression model. Results are shown in Table 3.

In Step 1, baseline DOCS (to control for pretreatment OCD severity) and BDI-II (to control for depressive symptom severity) scores jointly explained a significant amount of variance (28.62%) in posttreatment DOCS scores, $F(2, 106) = 21.276$, $p < 0.001$. Adding ASI subscale scores in Step 2 of this model account for statistically significant additional variance (R^2 change = 0.05, $p = 0.049$), such that higher pretreatment AS predicted poorer OCD treatment response even after controlling for pretreatment OCD and depression severity. However, after accounting for pretreatment DOCS and BDI-II scores, individual ASI subscales did not uniquely predict significant variance in posttreatment DOCS scores (i.e., ASI subscales only jointly explained significant outcome variance).

Based on our findings that depressive symptoms were only moderately correlated with baseline and posttreatment DOCS scores, we conducted an exploratory hierarchical regression excluding BDI-II scores, such that baseline DOCS were entered in Step 1, and the ASI subscales were entered in Step 2 of the model predicting posttreatment DOCS scores. The pattern of results was generally consistent with the primary analysis. Specifically, pretreatment OCD symptoms explained for a significant amount (27.2%) of posttreatment OCD symptoms in Step 1, $F(1, 108) = 41.63$, $p < 0.001$, yet the addition of the ASI subscales in Step 2 accounted for an additional 6.0% of outcome variance, $F^2_{\Delta R}$ (3, 105) = 3.15, $p = 0.028$. In this simplified model, the ASI physical subscale accounted for a marginally significant amount of unique outcome variance, $\beta = 0.198$, $t(104) = 1.83$, $p = 0.070$, $spr^2 = 0.021$.

3. Discussion

It is important to understand why certain OCD patients do not adhere or respond to CBT, the gold-standard intervention for OCD. In this vein, trait levels of AS represent a relevant and promising predictor of treatment outcome. Accordingly, the current study examined the extent to which baseline AS predicted treatment outcome in a sample of individuals with a clinical diagnosis of OCD undergoing CBT. Consistent with our first hypothesis, AS was positively correlated with baseline OCD severity. This is in line with previous work (see Robinson & Freeston, 2014) and indicates that increased fear of arousal-related body sensations is associated with increased OCD symptom severity. Our second hypothesis, that greater baseline levels of AS would prospectively predict higher posttreatment OCD symptom severity after controlling for pretreatment OCD and depression severity (i.e., factors consistently documented to influence OCD treatment response), was also supported. Even after controlling for pretreatment OCD and depression severity (which did not significantly predict CBT treatment outcome in our sample), the construct of AS accounted for significant, incremental variance in posttreatment OCD severity; although, no individual AS dimension emerged as a unique predictor. This suggests that the fear of anxious arousal in general predicts the outcome of CBT for OCD, and therefore represents a promising target for enhancing treatment response.

Table 3
Hierarchical linear regression predicting posttreatment DOCS total scores.

	ΔR^2	B	SE_B	β	t	p	sr^2
Step 1	0.29					<0.001	
Baseline DOCS		0.41	0.07	0.50	5.63	<0.001	0.213
Baseline BDI-II		0.06	0.08	0.08	0.85	0.397	0.005
Step 2	0.05					0.049	
Baseline DOCS		0.35	0.08	0.43	4.63	<0.001	0.138
Baseline BDI-II		−0.02	0.09	−0.02	−0.19	0.853	<0.001
ASI-Physical		0.42	0.23	0.20	1.79	0.077	0.021
ASI-Mental		0.64	0.46	0.17	1.40	0.166	0.013
ASI-Social		−0.15	0.28	−0.07	−0.54	0.590	0.002

DOCS = Dimensional Obsessive-Compulsive Scale; BDI-II = Beck Depression Inventory-II; ASI = Anxiety Sensitivity Index; sr^2 = squared semipartial correlation.

An important next step is to consider the mechanism through which AS hinders the effects of CBT for OCD. Although it was not measured directly in the present study, we hypothesize that elevated AS served to amplify exposure difficulty. Specifically, exposures that generated sensations of anxious arousal may have been experienced as more challenging and/or distressing not only because of the fear of the stimuli themselves (e.g., "Touching the toilet will make me sick"), but also because of the threat associated with physiological arousal (e.g., "If my heart races, I will have a heart attack"). In effect, OCD patients with high AS are simultaneously confronting two conditioned fear stimuli during exposures, which might understandably lead to nonadherence with prescribed exposure tasks. To test this hypothesis, future work examining AS as an outcome predictor might include measures of the quality of engagement in ERP as well as measures of physiological arousal during exposures (e.g., heart rate and skin conductance).

Regarding clinical implications of our findings, it may be beneficial to first conduct interoceptive exposure (IE; deliberately inducing feared-yet-safe body sensations without engaging in arousal-reduction strategies) with high AS patients prior to exposures to OCD-related stimuli with the aim of extinguishing the fear of arousal-related sensations. This might increase willingness to comply with ERP procedures for OCD. Consistent with inhibitory learning approaches to fear extinction, CBT might also involve the simultaneous use of IE and in vivo exposure to optimize consolidation of long-term extinction learning (e.g., Arch & Abramowitz, 2015; Craske et al., 2008, 2014). For example, a patient who interprets her trembling hands as indicating that she is highly likely to act on her unwanted obsessional thoughts of stabbing a loved one might conduct tailored IE exercises (i.e., holding a pushup position to induce trembling) immediately prior to holding a knife while near a loved one (in vivo exposure). This procedure is consistent with the principle of deepened extinction as described by Rescorla (2006), in which multiple fear cues are combined during exposure.

Strengths of the present study included the large clinical sample (which maximizes power) as well as the variability of patient symptomatology (which offers greater external validity and sample generalizability than many RCTs). On the other hand, one limitation of this research was the use of the original ASI (Reiss et al., 1986), rather than the newer ASI-3 (Taylor et al., 2007). Second, assessments relied on self-report measures administered at pre- and posttreatment only; future research should administer assessments at additional time points, as well as include clinician-rated assessments of symptom severity. A third limitation regards the differences in treatment duration and multimodal intervention at Rogers Memorial Hospital, as well as the fact that some participants may have had medication adjustments during treatment (if deemed appropriate by the on-site psychiatrist). Along these lines, it is also possible that patients in our sample did conduct OCD exposures that in some way addressed their AS concerns. Finally, because our sample was receiving residential treatment, our findings may not apply to routine clinical settings. Thus, future research examining the role of particular dimensions of AS in various approaches of standardized CBT for OCD should be conducted.

Acknowledgements, declaration of interest, and role of funding organizations

The authors have no conflicts of interest to disclose. The authors did not receive support from funding organizations to conduct this research. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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