Obsessive-compulsive disorder with comorbid major depression: What is the role of cognitive factors?

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Abstract

Individuals with obsessive-compulsive disorder (OCD) commonly experience comorbid mood disturbances such as major depressive disorder (MDD). Previous studies that have compared OCD patients with and without MDD have revealed differences in demographic characteristics, clinical severity, and symptom presentation between these two patient groups. Previous studies have not, however, examined whether there are differences with respect to cognitive processes. The present study therefore aimed to address this gap in the literature. Eighty patients with OCD and no unipolar mood disorders were compared with 34 OCD patients with comorbid major depression on measures of OCD symptoms, cognitions, and insight, as well as on measures of depression and functional impairment. Whereas depressed OCD patients evidenced higher scores than non-depressed OCD patients on semi-idiographic measures of OCD symptoms and cognitions, this was not the case for nomothetic measures. Functional impairment and the tendency to misinterpret innocuous intrusive thoughts as significant emerged as unique predictors of depression within the entire sample of OCD patients. Results are discussed in terms of (a) the importance of semi-idiographic assessment of OCD, (b) possible explanations for the relationship between OCD symptoms, depression, and cognitive processes, and (c) the psychological treatment of comorbid OCD and MDD.

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Introduction

Individuals with obsessive-compulsive disorder (OCD) commonly experience comorbid Axis I and Axis II psychopathology over the course of their illness, with mood disturbances such as major depressive disorder (MDD) among the most common additional diagnoses. Although reported comorbidity rates differ among studies due to variability in sampling and methodology, research indicates that as many as one-quarter to one-half of OCD sufferers experience MDD (e.g., Crino & Andrews, 1996; Hong et al., 2004; Nestadt et al., 2001). Studies on the temporal nature of this comorbidity pattern indicate that in most (but not all) instances, OCD
symptoms predate the depressive symptoms (Bellodi, Scioto, Diaferia, Ronchi, & Smiraldi, 1992; Demal, Lenz, Mayrhofer, Zapotoczky, & Zitterl, 1993). This suggests that the mood disturbance often occurs as a response to the distress and functional impairment associated with obsessions and compulsions. Accordingly, there is evidence that depressed OCD patients have an earlier age of OCD onset and more severe obsessions and compulsions as compared to their non-depressed counterparts (e.g., Hong et al., 2004; Tukel, Meteris, Koyuncu, Tecer, & Yazici, 2006).

In addition to the aforementioned differences, depressed OCD patients show more severe general anxiety symptoms, higher rates of other comorbid conditions (e.g., anxiety disorders), higher rates of unemployment, and greater functional disability than do OCD patients without depression (Hong et al., 2004; Ricciardi & McNally, 1995; Tukel, Polat, Ozdemir, Sksut, & Turksoy, 2002; Tukel et al., 2006). Moreover, the presence of comorbid depression is associated with the occurrence of sexual and religious obsessions (Hasler, LaSalle-Ricci, & Ronquillo, 2005; Hong et al., 2004); and this is consistent with previous research indicating that these particular types of obsessions are experienced as more distressing than are other OCD symptoms (Abramowitz, Franklin, Schwartz, & Furr, 2003; McKay et al., 2004). When the prevalence and clinical severity findings reviewed above are considered in light of research demonstrating that comorbid MDD also attenuates response to the most effective treatment for OCD—exposure and response prevention—(e.g., Abramowitz & Foa, 2000), one recognizes the need to further study the differences between depressed and non-depressed OCD patients.

The existing literature provides important information regarding differences in demographic characteristics, clinical severity, and symptom presentation between OCD patients with and without MDD; yet, there is a surprising dearth of research investigating whether these groups of patients differ with respect to relevant cognitive processes. Cognitive models of psychopathology in general (e.g., Beck, 1976) posit that emotional distress arises from dysfunctional beliefs and appraisals about external or internal stimuli. For example, Beck (1976) proposed that depression arises from specific sorts of beliefs and interpretations related to loss and self-degradation (e.g., “I am a loser who is unlovable”). As applied to obsessional problems, Rachman (1993, 1997) proposed a cognitive model in which clinical obsessions arise when normally occurring intrusive thoughts are erroneously appraised as highly significant or threatening (e.g., “If I think of something terrible, I could cause it to happen”). Rachman (1993) further observed that in severe cases, such appraisals involve ideas of self-deprecation that are similar to the kinds of cognitions observed in people with MDD (e.g., “The fact that I think about terrible things means I am a terrible person”). Thus, in comorbid patients, the cognitive processes involved in obsessions might also play a role in depressive symptoms. In an attempt to elucidate this role, we addressed the following two questions in the present study: (a) do depressed OCD patients evidence a greater tendency to misinterpret the significance of intrusions relative to non-depressed OCD patients? and (b) are misinterpretations of intrusions uniquely related to comorbid depressive symptoms among individuals with OCD?

A second cognitive factor that could be involved in the overlap between OCD and depression is insight—the degree to which the individual recognizes the senselessness of his or her obsessions and compulsions (e.g., Kozak & Foa, 1994). Whereas most OCD patients realize to some extent that their symptoms are unrealistic and excessive, insight exists on a continuum with some individuals believing strongly that their obsessive fears are rational and their compulsive behaviors are necessary to prevent disasters (Foa & Kozak, 1995). Studies of OCD patients indicate that poor insight is associated with more severe OCD symptoms (e.g., Deacon & Abramowitz, 2005) and higher levels of self-reported symptoms of depression (Turksoy, Tukel, Ozdemir, & Karah, 2002), leading to the hypothesis that OCD patients with MDD would evidence poorer insight than non-depressed OCD patients. Although Tukel et al. (2006) reported data consistent with this prediction, poor insight was assessed as either present or absent on the basis of DSM-IV criteria, rather than as measured by a psychometrically validated continuous measure.

In the present study, we therefore aimed to replicate and extend the literature on comorbid OCD and depression by examining the role of cognitive processes. Consistent with previous work, we expected to find that depressed OCD patients (i.e., with comorbid MDD) would show more severe OCD symptoms and functional disability relative to non-depressed OCD patients. We hypothesized that depressed OCD patients would also show a stronger tendency to misinterpret the significance of intrusive thoughts and evidence poorer insight into the senselessness of their OCD symptoms relative to non-depressed OCD patients. Furthermore,
we examined whether these cognitive factors uniquely contributed to the prediction of depressive symptoms in our OCD sample.

**Method**

**Participants**

Participants in the present study included 114 adult (≥18 years) outpatients with a primary DSM-IV-TR diagnosis of OCD seeking evaluation and treatment for this condition. Thirty-four of these patients met DSM criteria for both OCD and current MDD (“OCD-MDD” group), and 80 met criteria for OCD, but for no unipolar mood disorders (“OCD-NUMD” [meaning, No Unipolar Mood Disorder] group). Data were combined from consecutive referrals at two outpatient treatment facilities: 82 patients (72%) were evaluated at the Mayo Clinic in Rochester, Minnesota and 32 patients (28%) at the University of Florida in Gainesville, Florida.

**Procedure**

Diagnostic evaluations took place in OCD specialty clinics located within their respective academic medical centers. Each patient received a 1.5-h diagnostic assessment performed by a trained assessor who administered a structured diagnostic interview: the Mini International Neuropsychiatric Interview (Sheehan et al., 1998) was used at the Mayo Clinic site, and the Anxiety Disorders Interview Schedule for DSM-IV (Brown, DiNardo, & Barlow, 1994) was used at the Florida site. The interview also included the Yale–Brown Obsessive Compulsive Scale (Y-BOCS; described below) and the Brown Assessment of Beliefs Scale (BABS; described below). Axis II psychopathology was assessed by means of a semi-structured interview based on DSM-IV criteria. Additional self-report measures (see below) were completed in advance of the clinic appointment.

After completing the initial interview and formulating a diagnosis, the first interviewer presented the assessment data to a more expert clinician (the first or second author depending on location), who subsequently met and reviewed the assessment data with the patient. Although formal inter-rater reliability checks were not conducted, only patients for whom both interviewers agreed on diagnostic status (OCD-MDD or OCD-NUMD) were included in the study (100% inter-rater agreement). Because the design of the present study had not been conceptualized when these clinic data were collected, interviewers conducting the assessments were blind to the present study hypotheses.

Individuals with additional Axis I and II disorders were included in the study as long as OCD was considered a primary (principal) diagnosis. In the OCD-MDD group, 12 patients (25.3%) evidenced additional comorbid conditions (i.e., generalized anxiety disorder, social phobia, specific phobia, eating disorders, bipolar disorder, panic disorder); in the OCD-NUMD group, 28 patients (26.2%) evidenced additional comorbid conditions (i.e., generalized anxiety disorder, panic disorder, eating disorders, phobias). $\chi^2$-Analysis indicated that the proportion of patients with additional comorbid conditions did not differ significantly between the two groups, $\chi^2 = (N = 141, df = 1) = 1.06, p = \text{n.s.}$

**Assessment instruments**

We used a multitrait–multimethod approach to assessment that included both interview and self-report measures. In addition, given the substantial heterogeneity and highly patient-specific nature of OCD symptom presentation (e.g., contamination, harm, sex, religion, washers, checkers; McKay et al., 2004), we used nomothetic as well as idiographic measures to assess obsessions, compulsions, and OCD-relevant cognitive variables. The two treatment facilities used slightly different assessment batteries. Thus, we only included data derived from measures used at both sites and with enough observations to afford adequate power for meaningful analyses. The following instruments were therefore included in the present study:

**Yale–Brown Obsessive Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, Mazure, Delgado et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann et al., 1989):** Global severity of OCD was measured using the Y-BOCS, a semi-structured clinical interview that includes a symptom checklist and 10-item
semi-idiographic severity scale. The symptom checklist is first used to identify the patient’s particular obsessions and compulsions. The severity scale then assesses the patient’s main obsessions (items 1–5) and compulsions (items 6–10) on the following five parameters: (a) time, (b) interference, (c) distress, (d) resistance, and (e) degree of control. The clinician rates each item from 0 (no symptoms) to 4 (extreme) based on the past week. Two subscales (obsessions and compulsions; range 0–20) can be added to produce a total severity score that ranges from 0 to 40. The Y-BOCS interview has satisfactory psychometric properties (Goodman, Price, Rasmussen, Mazure, Delgado et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann et al., 1989; Storch et al., 2005) and is considered the gold standard measure of obsessive-compulsive symptoms.

Brown Assessment of Beliefs Scale (BABS; Eisen et al., 1998): Insight into the senselessness of obsessions and compulsions was assessed with the BABS; a 6-item continuous measure of conviction in obsessional fears. The BABS is a semi-idiographic measure administered as a semi-structured interview. The patient and clinician first identify one or two of the patient’s specific obsessional fears (e.g., “I will get AIDS from flushing a public toilet,” “I will hit someone with my car without realizing”). Next, these beliefs are rated on the following parameters: (a) conviction in the belief, (b) perception of others’ views, (c) explanation of differing views, (d) fixity of the belief, (e) attempts to disprove the belief, and (f) insight (recognition of a psychiatric etiology). Item scores range from 0 (normal) to 4 (pathological) and are summed to produce a total score ranging from 0 to 24. The scale has good psychometric properties as reported by Eisen et al. (1998).

Obsessive-Compulsive Inventory-Revised (OCI-R; Foa et al., 2002): Severity of OCD symptoms was assessed nomothetically using the OCI-R, an 18-item self-report questionnaire based on the earlier 84-item OCI (Foa, Kozak, Salkovskis, Coles, & Amir, 1998). Participants rate the degree to which they are bothered or distressed by OCD symptoms in the past month on a 5-point scale from 0 (not at all) to 4 (extremely). The OCI-R assesses six dimensions of OCD symptoms: (a) washing, (b) checking/doubting, (c) obsessing, (d) neutralizing, (e) ordering, and (f) hoarding. Research suggests the OCI-R possesses good internal consistency (total score alphas = .81 to .93 across samples) and construct validity (Abramowitz & Deacon, 2006; Foa et al., 2002).

OCD is a heterogeneous condition, yet each patient presents with a specific set of obsessions and compulsions (e.g., contamination/washing, ordering/arranging). Therefore, items on the OCI-R that do not pertain to a patient’s idiosyncratic presentation might be irrelevant and could suppress the representativeness of the total score. To address this issue in our analyses, we also derived scores from the OCI-R subscale that was most elevated (most severe) for each patient. Hereafter, we refer to this dependent variable as “OCI-R main symptom” (range = 0–12).

Interpretation of Intrusions Inventory (III; Obsessive Compulsive Cognitions Working Group [OCCWG], 2005): The III is a 31-item semi-idiographic questionnaire that assesses erroneous appraisals and interpretations of unwanted, distressing intrusive (obsessional) thoughts, images or impulses. After reading a definition of unwanted mental intrusions (which includes several examples), respondents were helped (by a clinician) to identify one or two obsessional thoughts they have recently experienced that are either frequent or distressing. They then rate the extent to which they believe in each of the 31 statements as related to the identified intrusive thoughts (e.g., “Thinking this thought could make it happen”). Strength of belief is rated by 10 s (e.g., 0, 10, 20, etc.) from 0 (“I did not believe this idea at all”) to 100 (“I was completely convinced this idea was true”). The 31 items form three subscales: (a) importance of thoughts, (b) responsibility, and (c) control of thoughts.

Thought–Action Fusion Scale (TAFS; Shafran, Thordarson, & Rachman, 1996): The TAFS is a 19-item self-report measure of the tendency to believe that thoughts are equivalent to actions. Twelve items assess moral TAF, which is the belief that thoughts are the moral equivalent of actions (e.g., “Having a blasphemous thought is almost as sinful to me as a blasphemous action”). Three assess likelihood-self TAF, which is the belief that merely thinking about harm coming to oneself increases the likelihood of being harmed (e.g., “If I think of myself being in a car accident this increases the risk that I will have a car accident”). The remaining four items assess likelihood-other TAF, which is the belief that thinking about harm coming to someone else increases the likelihood of that person being harmed (e.g., “If I think of a relative/friend losing their job, this increases the risk that they will lose their job”). Agreement with each item is rated on a scale from 0 (disagree strongly) to 4 (agree strongly). The instrument’s psychometric properties are good and have been described by Shafran et al. (1996).
Sheehan Disability Scale (SDS; Sheehan, 1986): The SDS is a commonly used 3-item measure of the degree to which clinical symptoms interfere with work, social/leisure activities, and family/home responsibilities. It is regarded as a measure of quality of life among anxiety patients (Mendlowicz & Stein, 2000). Internal consistency of the SDS in clinical samples ranges from .56 to .86 (Mendlowicz & Stein, 2000).

Beck Depression Inventory (BDI; Beck, Steer, & Brown, 1996): The BDI 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 has excellent reliability and validity and is widely used in clinical research (Beck et al., 1996).

Results

Site differences

In a preliminary set of analyses, we examined whether patients evaluated at the two study sites differed on demographic and psychopathology variables. These analyses indicated no such differences \( p > .05 \) on any demographic characteristics, including group membership (OCD-MDD vs. OCD-NUMD). With two exceptions, there were also no significant site differences on the psychopathology variables. The exceptions were that participants enrolled at the University of Florida had higher scores on the Y-BOCS Total Score and on the Y-BOCS Obsessions subscale. These differences are likely due to the Florida site’s specialization in the treatment of refractory patients. Because there were no site differences in the proportion of patients with comorbid MDD, we combined data from both sites in the analyses presented below.

Demographic characteristics

Table 1 presents demographic characteristics for the entire sample and for the OCD-MDD and OCD-NUMD groups. Between-group comparisons were made using \( \chi^2 \)-tests for categorical variables and \( t \)-tests for continuous variables, and these also appear in Table 1. As can be seen, no significant differences emerged on any of these variables. As expected, preliminary analyses revealed a significant between group differences on the BDI. The OCD-MDD group reported being substantially more depressed than the OCD-NUMD group.

Clinical characteristics

Table 2 presents the means and standard deviations for both patient groups (and for the entire sample) on the clinical variables, as well as the results of \( t \)-tests examining differences between the OCD-MDD and OCD-NUMD groups. Between group effect sizes (Cohen’s \( d \)) are also presented in Table 2. Due to the strong intercorrelations among the three SDS items (\( r \) ranged from .67 to .72) and among the three III subscales (\( r \) ranged from .72 to .82), multivariate analyses of variance were performed on these two measures. For both analyses, each dependent variable was roughly univariate normal (skewness and kurtosis estimates < 1). Additionally, analysis of homogeneity of variance-covariance matrices across design cells for both analyses as indicated by the Box-M test revealed non-significant results \( p > .05 \), suggesting that the assumption of

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographic characteristics for the entire sample and by patient group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total sample ((N = 114))</td>
</tr>
<tr>
<td>Mean (SD) age (years)</td>
<td>33.30 (11.9)</td>
</tr>
<tr>
<td>Number of females (%)</td>
<td>50 (44.3)</td>
</tr>
<tr>
<td>Number of Caucasian (%)</td>
<td>82 (96.5)</td>
</tr>
<tr>
<td>Mean (SD) BDI</td>
<td>17.02 (10.9)</td>
</tr>
</tbody>
</table>

Note: *\( p < .01 \).
OCD-MDD = Obsessive-compulsive disorder with major depressive disorder; OCD-NUMD = Obsessive-compulsive disorder with no unipolar mood disorder; BDI = Beck Depression Inventory.
variance-covariance was not violated. Even so, we used the relatively conservative Pillai’s trace for the estimation of \( F \)-statistics in both analyses.

Both groups evidenced moderate-to-severe OCD symptoms, yet whereas the OCD-MDD group demonstrated significantly more severe OCD symptoms on the Y-BOCS, no such difference was found on the OCI-R, any of its subscales, or for the OCI-R main symptom variable. The magnitudes of between group effect sizes on the Y-BOCS were moderate. Effect sizes derived from the OCI-R were small.

The omnibus MANOVA for the SDS measure revealed significant between group differences in impairment ratings (\( F[3, 88] = 7.29, p < .01, \eta^2 = .20, \) Pillai’s trace = .20). Consistent with the results reported above, the OCD-MDD group indicated greater impairment in work, social, and family domains relative to the OCD-NUMD group. Effect sizes for the SDS subscales were uniformly large.

**Cognitive variables**

Our main interest in the present study was whether depressed and non-depressed OCD patients differed on cognitive variables associated with OCD. To examine our hypothesis that depressed OCD patients would
show a greater tendency to misinterpret the significance of intrusive thoughts, we compared the two patient groups on the TAF and III; group mean scores, F- and t-test results, and effect sizes are also presented in Table 2. As can be seen, no differences were found on any of the TAF subscales, and effect sizes were small. In contrast, the omnibus MANOVA for the III measure revealed significant between-group differences (F[3, 85] = 3.71, p < .05, η² = .12, Pillai’s trace = .12). On all three of the subscales, the OCD-MDD group evidenced significantly higher scores than did the OCD-NUMD group. The magnitudes of the between group effect sizes for the III were medium.

With respect to insight into the senselessness of OCD symptoms, BABS scores indicated that both groups had reasonably good recognition that their obsessions and compulsions were excessive or unreasonable. Although the OCD-MDD group evidenced a higher mean BABS score (i.e., poorer insight) than did the OCD-NUMD group (which was in the hypothesized direction), only a non-significant trend (p = .06) was found. The effect size for this comparison was small to medium.

### Predicting MDD and depressive symptoms

To further explore the relationship between cognitive factors and depressive symptomatology within OCD, we conducted hierarchical linear and logistic multiple regression analyses to examine the relative contribution of clinical and cognitive variables to the prediction of BDI scores (linear regression) and a comorbid diagnosis of MDD (logistic regression) in the entire sample (N = 114). In both analyses, the Y-BOCS total score was entered in the first step to control for group differences in symptom severity. The following variables (which demonstrated significant differences between the OCD-MDD and OCD-NUMD groups) were simultaneously entered into the second step of each regression equation as predictors: SDS total score, III, and BABS.1

Tolerance statistics for these analyses were acceptable (range .69–.99) and correlations among predictor variables weak to modest (r = .07–.37). Results of these analyses are presented in Tables 3 and 4. In the hierarchical linear multiple regression analysis, after controlling for total Y-BOCS scores, results indicated that the three predictor variables (SDS, III, and BABS) explained a significant proportion of the additional variance in BDI scores (ΔR² = .36, p < .001), and the SDS and III contributed significant, unique variance to the model. Results from the hierarchical logistic multiple regression analysis predicting comorbid MDD also indicated that the predictors explained a significant portion of additional variance after controlling for the Y-BOCS total score (AR² = .31, p < .001; see Table 4). As in the first analysis, the SDS and III again emerged as the only significant, unique predictors of MDD comorbidity.

### Discussion

The present study examined differences between OCD patients with and without comorbid MDD with respect to clinical and cognitive variables, and to explore OCD-relevant cognitive processes as unique predictors of MDD comorbidity.

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1The SDS and III total scores were used due to the strong intercorrelations between the three SDS items (r ranged from .67 to .72) and three III subscales (r ranged from .72 to .82).
predictors of depressive symptoms in individuals with OCD. With regard to our between-group comparisons, and consistent with previous research (e.g., Tukel et al., 2006), depressed OCD patients experienced more severe levels of overall distress and functional impairment relative to non-depressed OCD patients. However, when all of our data are considered together, our primary hypotheses appear to receive only mixed support. Concerning OCD symptom severity, between-group differences in the predicted direction were found on the Y-BOCS, but not on the OCI-R. Similarly, with respect to the propensity toward misinterpreting the significance of intrusive thoughts, the OCD-MDD group had higher scores on the III than did the OCD-NUMD group, but no such difference was found on the TAF. Finally, only a non-significant trend in the hypothesized direction emerged regarding insight into the senselessness of OCD symptoms. Although at first glance support for our hypotheses appears mixed, a closer inspection reveals that all data from the semi-idiographic measures of OCD-related variables (i.e., the Y-BOCS, III, and BABS) are consistent (or tending to be consistent) with our predictions and with findings from earlier studies (e.g., Tukel et al., 2006); whereas this was not the case for the nomothetic OCD-relevant measures (i.e., OCI-R and TAF). Specifically, ratings based on patients’ specific presentation of OCD revealed that relative to non-depressed OCD patients, those with comorbid MDD had more severe obsessions and compulsions, a greater tendency to misinterpret the significance of intrusive thoughts, and poorer insight into the senselessness of their symptoms. In contrast, ratings of symptoms and cognitions assumed to be representative of people with OCD in general (i.e., on nomothetic measures) did not differ between patient groups. We believe these differential findings for semi-idiographic and nomothetic measures are consequential in light of the highly heterogeneous and idiosyncratic (patient-specific) nature of OCD (e.g., McKay et al., 2004). Because semi-idiographic measures take into account the patient’s specific obsessions, compulsions, and beliefs, these sorts of measures are likely to provide greater accuracy regarding the assessment of obsessive-compulsive complaints than are nomothetic measures.

Our regression analyses indicated that two variables—functional impairment and erroneous interpretations of intrusive thoughts—evidenced unique associations with depressive symptoms and with the presence of comorbid MDD. In contrast, the severity of obsessions, compulsions, and insight into the senselessness of these symptoms, did not show such a relationship. The finding that misinterpretations of intrusive thoughts and impairment were uniquely related to depression suggests that functionality and cognitive distortions are somehow involved in depressive symptoms when they occur in the context of OCD. We cannot, however, infer the direction of this relationship since the present study was cross-sectional (correlational). Therefore, prospective and experimental studies, which are better able to resolve such causal arrow ambiguity, would be a valuable next step.

Table 4
Predictors of comorbidity with MDD among patients with obsessive-compulsive disorder in the final step of the Logistic Regression Model

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE</th>
<th>Wald</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Y-BOCS</td>
<td>.01</td>
<td>.08</td>
<td>.03</td>
<td>1.01 (.87–1.17)</td>
</tr>
<tr>
<td>SDS</td>
<td>.21</td>
<td>.08</td>
<td>6.21*</td>
<td>1.23 (1.05–1.45)</td>
</tr>
<tr>
<td>III</td>
<td>.00</td>
<td>.00</td>
<td>4.48*</td>
<td>1.00 (1.00–1.00)</td>
</tr>
<tr>
<td>BABS</td>
<td>.08</td>
<td>.07</td>
<td>1.10</td>
<td>1.08 (.94–1.25)</td>
</tr>
</tbody>
</table>

Note: ΔNagelkerke $R^2 = .31$, Δχ² = 19.46, df = 3, $p < .001$.
*p < .05.
*aModel fit statistics: Nagelkerke $R^2 = .46$, −2 log likelihood = 58.01, $\chi^2 = 27.52$, df = 4, $p < .001$.

2One might argue that the OCI-R main symptom variable represents a semi-idiosyncratic measure of OCD since it considers the patient’s most severe symptom. Each OCI-R subscale, however, contains only three items. Therefore, although this variable arguably provides a measure of the severity of the patient’s “main” or “primary” OCD symptom (as assessed by the OCI-R), it is still bound by the particular examples included in the three items of any particular subscale. Given the heterogeneity of obsessions and compulsions, we consider this variable a nomothetic measure of OCD.
As mentioned above, the cross-sectional nature of our data precludes causal inferences regarding the nature of the relationship among cognitive processes, OCD, and depression; this relationship is likely to be complex. Our findings, however, could be considered within the context of Rachman’s (1993) suggestion that in some individuals with OCD, misinterpretations of the presence and significance of intrusive obsessional thoughts become highly personalized, resulting in self-blame for having (and not being able to control) such unacceptable thoughts. Self-blame and self-deprecation lead to feelings of depression and other negative mood states (e.g., Beck, 1976). Additionally, negative mood states have been shown to increase vulnerability to intrusive obsessional thoughts (e.g., England & Dickerson, 1988; Sutherland, Newman, & Rachman, 1982). Accordingly, depression might foster obsessional thinking (Niler & Beck, 1989; Reynolds & Salkovskis, 1991), thereby completing a vicious cycle of obsessional thoughts → misinterpretations → depressed mood → more obsessional thoughts. Importantly, such a model remains speculative since we did not include measures of self-blame or self-deprecation in the present study, nor could we examine causal relationships.

Although research suggests that comorbid MDD attenuates response to exposure and response prevention treatment for OCD (e.g., Abramowitz & Foa, 2000), no studies have addressed whether treatment outcome for depressed OCD patients can be improved by augmenting this therapy with additional psychological treatment techniques. One hypothesis raised by the present findings is that cognitive therapy, which is an effective treatment for uncomplicated OCD (e.g., Wilhelm & Steketee, 2006) as well as for depression (e.g., DeRubeis et al., 2005), would be a useful intervention for depressed OCD patients. Adding cognitive therapy to exposure-based therapy might help patients to explicitly identify, challenge, and modify dysfunctional beliefs and interpretations that maintain both depressive and obsessional symptoms. The use of cognitive therapy along with traditional exposure-based methods in the treatment of comorbid OCD and depression has been described elsewhere (Abramowitz, 2004). Alternatively, behavioral activation—which is an effective treatment for depression that does not explicitly modify cognitions—might also be considered as an augmentation strategy.

Our present findings should be interpreted within the context of several limitations. As mentioned previously, our data are correlational and cross-sectional, and as such do not provide a basis for causal inferences. The relationship between depression and OCD symptoms is likely a complex one, and might differ from patient to patient. Prospective and experimental studies should be undertaken to further elucidate the nature of this relationship. Other limitations include the demographically homogeneous nature of our sample (e.g., largely Caucasian) and that we did not assess family members perceptions or experiences. Regarding the latter point, it is conceivable that family members of a patient with OCD and comorbid depression may engage in heightened levels of symptom accommodation and expressed emotion by virtue of the additional burden created by the co-occurring illnesses. Finally, the variables in our regression model accounted for only a portion of the variability in depressive symptoms. This suggests that future research is necessary to further pinpoint the processes that account for the development of clinical depression in patients with OCD.

Finally, we suggest a number of avenues for future research. Firstly, additional cognitive OCD-relevant variables such as inflated responsibility, intolerance of uncertainty, perfectionism, and the need to feel that things are “just right” should be examined in the context of comorbid OCD and depression. Secondly, clinical observations suggest that whereas some depressed OCD patients experience separate co-principal diagnoses of these disorders, others are characterized by depression that is secondary to having OCD (e.g., “I am depressed because of how my life is ruined by OCD”). Unfortunately, we did not include an assessment of this issue in the present study, yet it would be useful to examine whether these two groups of patients differ with respect to symptom severity, functional impairment, and cognitive variables. Answers to these questions might have implications for the treatment of depressed OCD patients.

References


