Journal of Anxiety Disorders 24 (2010) 547–552

Contents lists available at ScienceDirect

Journal of Anxiety Disorders

Does cognitive-behavioral therapy response among adults with obsessive–compulsive disorder differ as a function of certain comorbidities?

Eric A. Storch[a,b,*], Adam B. Lewin[a], Lara Farrell[c], Mirela A. Aldea[a], Jeannette Reid[a], Gary R. Geffken[d], Tanya K. Murphy[a,b]

[a] Department of Pediatrics, University of South Florida, United States
[b] Department of Psychiatry, University of South Florida, United States
[c] Griffith University, Australia
[d] Departments of Psychiatry, Pediatrics, and Clinical & Health Psychology, University of Florida, United States

ARTICLE INFO

Article history:
Received 20 October 2009
Received in revised form 23 March 2010
Accepted 23 March 2010

Keywords:
Obsessive–compulsive disorder
Cognitive-behavioral therapy
Comorbidity
Treatment
Depression
Generalized anxiety disorder

ABSTRACT

This study examines the impact of several of the most common comorbid psychiatric disorders (i.e., generalized anxiety disorder (GAD); major depressive disorder (MDD); social phobia, and panic disorder) on cognitive-behavioral therapy (CBT) response in adults with obsessive–compulsive disorder (OCD). One hundred and forty-three adults with OCD (range = 18–79 years) received 14 sessions of weekly or intensive CBT. Assessments were conducted before and after treatment. Primary outcomes included scores on the Yale-Brown Obsessive–Compulsive Scale (Y-BOCS), response rates, and remission status. Sixty-nine percent of participants met criteria for at least one comorbid diagnosis. Although baseline OCD severity was slightly higher among individuals with OCD + MDD and OCD + GAD (in comparison to those with OCD-only), neither the presence nor the number of pre-treatment comorbid disorders predicated symptom severity, treatment response, remission, or clinically significant change rates at post-treatment. These data suggest that CBT for OCD is robust to the presence of certain common Axis-I comorbidities.
© 2010 Elsevier Ltd. All rights reserved.

Obsessive–compulsive disorder (OCD) is a debilitating psychiatric disorder with a lifetime prevalence rate of approximately 2% (Crino, Slade, & Andrews, 2005; Horwath & Weissman, 2000). Most affected individuals have a childhood onset (Samuels & Nestadt, 1997) and, left untreated, symptoms tend to pursue a chronic and deteriorating course (Eisen & Steketee, 1998). Moreover, a diagnosis of OCD places individuals at an increased risk for comorbid psychopathology by virtue of shared pathogenesis (Nestadt et al., 2001), commonalities in dysfunctional psychological processes (e.g., cognitive appraisals; Morillo, Belloch, & Garcia-Soriano, 2007), and distress related to illness-associated impairment. Conditions such as tic disorders, mood disorders, anxiety, 'obsessive–compulsive spectrum disorders' (e.g., body dysmorphic disorder, trichotillomania, hypochondriasis), and personality disorders commonly co-occur with OCD (Bienvenu et al., 2000; Chen & Dilsaver, 1995; Coffey et al., 1998; Nestadt et al., 2001; Samuels et al., 2000; Yaryura-Tobias et al., 2000). While there are numerous comorbid conditions associated with OCD, depression is the most frequent comorbid presentation in adults with OCD (Pinto, Mancebo, Eisen, Pagano, & Rasmussen, 2006).

Pharmacotherapy with serotonin reuptake inhibitors (SRIs) and psychotherapy using cognitive-behavioral therapy (CBT), including exposure and response prevention, are the treatments of choice for treating OCD. Although both treatment modalities have demonstrated efficacy among OCD patients (Abramowitz, 1997; Eddy, Dutra, Bradley, & Westen, 2004), results from randomized placebo-controlled trials (e.g., Foa et al., 2005) and meta-analytic studies have confirmed that CBT has some advantages over medication alone in terms of efficacy, durability after treatment withdrawal, and safety (Abramowitz, Whiteside, & Deacon, 2005; Kobak, Greist, Jefferson, Katzelnick, & Henk, 1998). There is a considerable body of literature attesting to the effectiveness of CBT for OCD. A recent, comprehensive meta-analysis of OCD treatment calculated effect sizes for cognitive and behavioral interventions (including exposure with response prevention) of 1.39–1.54 (Eddy et al., 2004). Given these findings, the Expert Consensus Guidelines (March, Frances, Carpenter, & Kahn, 1997) strongly recommend that patients with OCD receive CBT alone or in combination with SRI therapy as the first line treatment.

Despite efficacy of CBT, not all OCD patients respond to treatment, with as few as 25% experiencing full recovery (Fisher & Wells, 2005). Furthermore, approximately 25% of patients reportedly either refuse CBT or terminate treatment prematurely for

* Corresponding author at: Department of Pediatrics, University of South Florida School of Medicine, Rothman Center for Neuropsychiatry, 800 Sixth Street South, Fourth Floor North Box 7523, St. Petersburg, FL 33701, United States.
Tel.: +1 727 767 8230; fax: +1 727 767 7786.
E-mail address: estorch@health.usf.edu (E.A. Storch).

0887-6185/$ – see front matter © 2010 Elsevier Ltd. All rights reserved.
doi:10.1016/j.janxdis.2010.03.013

various reasons (e.g., lack of insight, difficulty engaging in exposures; Keeley, Storch, Merlo, & Geffken, 2008; McDonald, Marks, & Blizzard, 1988). Dar and Greist (1992) proposed several factors that may explain a poor response to CBT including, the presence of comorbid conditions, treatment noncompliance, and central nervous system depressing medications (e.g., benzodiazepines) that may impact on the success of exposure tasks.

To date, little research has attended to the relationship of patient variables and psychotherapy outcome. Of particular interest to the current investigation is the influence of psychiatric comorbidity on CBT outcome in an adult sample of OCD patients. With regard to the influence of comorbid depression on CBT response, the results of previous research have produced somewhat mixed findings that, for the most part, suggest that comorbid depression is associated with attenuated CBT response. Several studies have found that participants with comorbid depression responded less favorably than those who were not depressed (Abramowitz & Foa, 2000; Abramowitz, Franklin, Street, Kozak, & Foa, 2000; Cottreaux, Messy, Marks, Mollard, & Bouvard, 1993; Foa et al., 1983; Overbeek, Schruers, Vermetten, & Griez, 2002; Steketee, Chambless, & Tran, 2001). For example, Abramowitz and Foa (2000) found that OCD patients with comorbid major depressive disorder (MDD) who underwent a trial of CBT had significantly higher post-treatment and follow-up OCD severity scores than patients without MDD. Similarly, Steketee et al. (2001) found that patients with comorbid MDD fared worse on immediate and 6-month CBT outcome than those with OCD alone. Among youth with OCD, Storch et al. (2008) found that the presence of MDD negatively impacted response and remission rates. In contrast however, a number of studies failed to find an association between depression and treatment response (Foa, Kozak, Steketee, & McCarthy, 1992; O'Sullivan, Noshirvani, Marks, Montiero, & Lelliott, 1991; Orloff et al., 1994; Steketee, 1993; Zitterl et al., 2000). For example, Foa et al. (1992) found that depressed and non-depressed patients undergoing a trial of CBT responded equally as well, with both groups benefiting substantially from treatment.

Mixed findings have also emerged in pharmacological trials and CBT studies of OCD patients who present with other comorbid diagnoses. With regard to the influence of concurrent anxiety disorders, the results from one investigation suggested that the presence of comorbid posttraumatic stress disorder (PTSD) adversely impacts CBT response (Gershuny, Baer, Jenike, Minichiello, & Wilhelm, 2002). Similarly, Steketee et al. (2001) found that generalized anxiety disorder (GAD) predicted higher rates of dropout and poorer outcome 6 months after CBT completion. In contrast, others have not found anxiety comorbidities to negatively effect treatment response (Orloff et al., 1994; Steketee, Eisen, Dyck, Warshaw, & Rasmussen, 1999). For example, Steketee et al. (1999) found that neither the type nor the number of anxiety diagnoses predicted CBT outcome. Likewise, among children with OCD, Storch et al. (2008) found that the presence of an anxiety disorder did not impact CBT response. Investigations into the effect of personality disorders on treatment response have also been conflicting, with some studies finding that they predict poorer outcome (AuBuchon & Malatesta, 1994; Baer, Jenike, Black, & Treece, 1992; Minichiello, Baer, & Jenike, 1987; Ravizza, Barzega, Bellino, Bogetto, & Maina, 1995), and other studies failing to find an association (Dreessen, Hoekstra, & Arntz, 1997; Mavissakalian, Hamann, & Jones, 1990; Steketee et al., 1999, 2001).

Overall, research to date examining the role of comorbidity on treatment response in OCD has produced inconsistent findings. Furthermore, of the published studies examining outcome, there is limited data on the effect of comorbid psychiatric conditions in terms of rates of clinical remission and clinically meaningful symptomatic change (i.e., clinically significant change). Extant studies that have examined the impact of comorbidity on CBT outcome have been primarily confined to studies of comorbid depressive disorders and/or only examine either symptom improvement or response without attending to diagnostic remission. Additional research is needed to clarify the possible impact of comorbidity on CBT response, as this has significant clinical implications for assessment and treatment planning. For example, if the presence of comorbid depression is associated with attenuated response, sequential pharmacotherapy and/or a trial of behavioral activation (Lezuez, Hopko, & Hopko, 2001) to treat mood disturbance may be beneficial before initiating CBT. Similarly, if elevated levels of GAD are present, sequential pharmacotherapy and GAD-based cognitive therapy may be indicated.

Objectives of this investigation are therefore twofold. First, we sought to examine the influence of commonly occurring psychiatric comorbidity on post-treatment OCD-symptom severity. We expected that the pre-treatment comorbidity would be predictive of higher residual OCD symptoms following CBT. Second, we were interested in studying the impact of specific, common comorbid conditions on CBT response and remission rates as well as clinically significant change. Although previous findings are inconsistent, the extant data together with our clinical observations led us to expect that CBT response would be attenuated in participants with comorbid MDD relative to those without comorbid MDD.

1. Method

1.1. Participants

One hundred and forty-three patients (male = 68, female = 75) participated in this study across two sites. Enrollment sites included two university-based OCD specialty centers, one in the Southeastern United States and the other in South East Queensland, Australia. Demographic variables (i.e., age, gender, ethnicity) and clinical dimensions (i.e., symptom severity, comorbidity) did not differ across recruitment sites. Inclusion criteria required that subjects have a current primary diagnosis of OCD (as defined by the DSM-IV-TR; American Psychiatric Association, 2000) for a duration of at least one year. Exclusion criteria included diagnosis of schizophrenia or other psychotic disorders, active bipolar disorder, suicidality, abuse of alcohol or other illicit substance within 6 months, or a change in their psychotropic medication within 8 weeks of study enrollment. Participants ranged in age from 18 to 79 years old ($M = 31.16$, SD = 11.79). The vast majority of participants identified as Caucasian (97%) and the rest of the sample consisted of Black/African-decent (2%), Asian/Pacific Islander-decent (1%) and other race/ethnicity (1%).

1.2. Measures

Anxiety Disorders Interview Schedule-4th Edition. (Brown, Barlow, & Dinardo, 2004). The Anxiety Disorders Interview Schedule-4th Edition (ADIS-IV) is a semi-structured clinical interview based on DSM-IV diagnostic criteria. Although it was primarily designed for diagnosing anxiety disorders, the ADIS-IV also allows for the diagnosis of other Axis-I disorders (e.g., major depressive disorder), including the date of onset and degree of impairment. The disorder that causes the most distress and functional impairment is considered to be the primary diagnosis, while secondary diagnoses are assigned to other disorders that are present.

Yale-Brown Obsessive–Compulsive Scale. The Yale-Brown Obsessive–Compulsive Scale (Y-BOCS; Goodman et al., 1989a; Goodman et al., 1989b) is a 10-item semi-structured, clinician-administered measure of obsession and compulsion severity. Symptoms from the past week are rated on a 5-point Likert scale ranging from 0 to 4, with higher scores corresponding to greater

symptom severity. Items pertaining to obsessions and compulsions are summed to derive the Obsession and Compulsion Severity Scores. All items are summed to derive the Total Score. Internal consistency of the Total Severity Scale has been moderate to excellent (Goodman et al., 1989a; Woody, Steketee, & Chambless, 1995), as has inter-rater reliability (Goodman et al., 1989a, Woody et al., 1995). Total and subscale scores on the Y-BOCS have been significantly correlated with other measures of OCD symptoms, depression, and anxiety (Goodman et al., 1989b), and shows excellent treatment sensitivity (Foa et al., 2005).

Clinical Global Impression. The Clinical Global Impression scale (CGI-Severity and CGI-Improvement; Guy, 1976) consists of 7-point clinician ratings of symptom severity and improvement. Severity ratings (CGI-S) range from 0 (no illness) to 6 (extremely severe). Improvement (CGI-I) ratings include the following: −3 (very much worse), −2 (much worse), −1 (slightly worse), 0 (unchanged), +1 (slightly improved), +2 (much improved), and +3 (very much improved). This instrument has favorable psychometric properties and has been extensively used in treatment studies (e.g., Foa et al., 2005; Simpson et al., 2008).

Definition of response, remission, and clinically significant change. Consistent with previous research (e.g., Bergeron et al., 2002; Foa et al., 2005) the current study defined treatment response as a post-treatment CGI-Improvement score of "very much improved" or "much improved". Remission was defined as a severity rating on the Y-BOCS Total Score 1-week post-treatment of ≤12 (Frank et al., 1991; Simpson, Huppert, Petkova, Foa, & Liebowitz, 2006). Criteria for clinically significant change included a Y-BOCS reduction of 7 points and a post-treatment Y-BOCS ≤ 16 (Bjorgvinsson et al., 2008; Christensen & Mendoza, 1986; Goodman & Price, 1992; Jacobson, Follette, & Revenstorf, 1984; Jacobson & Truax, 1991). Although these outcomes are related constructs, they may be differentially affected as a function of comorbid conditions. Thus, separate sets of analyses were performed for response and remission rates and clinically significant change, respectively.

1.3. Data analyses

Statistical analyses were performed using SPSS 17.0. Preliminary analyses explored descriptive information for the OCD sample with and without comorbid diagnoses. To assess the main hypotheses of the study, the following analyses were conducted. First, one-way analyses of covariance (ANCOVA) were run to determine whether post-treatment Y-BOCS total and subscale scores depended upon comorbidity status while controlling for baseline symptom severity. Second, chi-square analyses were run to determine whether there were group differences on treatment response, remission, and clinically significant change depending upon comorbidity status. Given the small and unequal cell sizes, Fisher's Exact Tests were run to determine whether there were group differences on treatment response status across different comorbidities. No statistical correction was employed for Type I error.

2. Results

2.1. Preliminary analyses

The overall sample consisted of 143 patients, 98 (69%) of who met criteria for at least one comorbid psychiatric disorder. Comorbid diagnoses included: GAD ($n = 54$; 38%), MDD ($n = 44$; 31%), social phobia (SP; $n = 42$; 30%), and panic disorder (PD; $n = 24$; 17%). Other comorbid conditions were present in varying degrees (e.g., PTSD; specific phobia; tic disorders) but not included in analyses given their low frequency. In addition to OCD, participants met criteria for an average of 1.1 (SD = 1.0) additional DSM-IV Axis I conditions (range = 0–4). Thirty-four percent met criteria for one additional diagnosis, 25% for two additional diagnoses, 7% for three diagnoses, and 2% met criteria for four diagnoses exclusive of OCD.

Descriptive clinical and demographic information for the OCD-only ($n = 45$) and OCD plus comorbid diagnosis groups ($n = 98$) is presented in Table 1. No differences were found between the OCD-only and OCD plus comorbidity groups in terms of age, ethnicity or gender ratio. However, the groups differed on baseline OCD severity, such that subjects with OCD and any comorbid diagnoses exhibited more severe OCD symptoms on the Y-BOCS Total Score, $F(1, 140) = 9.9$, $p = 0.002$; the Y-BOCS Obsession Severity Score, $F(1, 140) = 11.5$, $p < 0.001$; and the Y-BOCS Compulsion Severity Score, $F(1, 140) = 5.6$, $p = 0.019$ compared to subjects with OCD-only. This difference was also reflected in baseline CGI-S, $F(1, 141) = 5.0$, $p = 0.016$. With regard to specific comorbid conditions, those with OCD + MDD had significantly higher scores on the Y-BOCS Obsession Severity Score (at baseline) than did those with OCD-only ($F(1, 140) = 8.2$, $p = 0.005$); and those with OCD + GAD exhibited significantly more severe symptoms on the Y-BOCS Total Score ($F(1, 140) = 21.6$, $p < 0.001$); the Y-BOCS Obsession Severity Score ($F(1, 140) = 17.0$, $p < 0.001$); the Y-BOCS Compulsion Severity Score ($F(1, 140) = 18.0$, $p < 0.001$); and the CGI-S ($F(1, 141) = 8.1$, $p = 0.005$) than did those with OCD-only.

2.2. OCD-symptom reduction

Controlling for baseline OCD severity using ANCOVA, participants with comorbidity (panic disorder, social phobia, generalized anxiety disorder, and major depressive disorder) did not differ from subjects with OCD-only in terms of reduction in Y-BOCS scores (Total Score, Obsession Severity Score, or Compulsion Severity Score). Data are presented in Table 2. Further, Spearman Rank-Order Correlations indicate that post-treatment scores on the Y-BOCS (total, obsessions, and compulsions severity scores) as well as the CGI-I do not relate to the number of comorbid conditions. This difference was not significant ($\chi^2(1) = 1.44$, $p = 0.23$).

2.3. Treatment response, remission, and clinically significant change by comorbid condition

Seventy-six percent of participants (regardless of comorbidity status) were considered treatment responders based on CGI-I criteria (see above). The mean reduction on the Y-BOCS Total Score was 18.32 (SD = 6.64) points for responders, versus 4.64 (SD = 5.94) for treatment non-responders. Eighty-two percent of participants without a comorbid diagnosis (OCD-only) and 74% of participants with a comorbid diagnosis were treatment responders. This difference was not statistically significant ($\chi^2(1) = 1.35$, $p = 0.30$).

Forty-eight percent of all study participants ($n = 68$) were considered in-remission upon study completion (based on Y-BOCS scores; Simpson et al., 2006). For remitters, the mean reduction on the Y-BOCS Total Score was 21.06 (SD = 7.43); for the non-remitted patients, the mean Y-BOCS reduction was 11.69 (SD = 7.46). Of the 42 participants with no comorbid diagnoses, 53% ($n = 24$) were in remission. In the comorbid group, 45% ($n = 44$) were in remission. This difference was not statistically significant ($\chi^2(1) = 1.12$, $p = 0.29$).

Finally, 68% of participants overall met criteria for clinically significant change. The mean reduction on the Y-BOCS Total Score was 18.9 (SD = 7.0) points for subjects with clinically significant change responders versus a mean of 7.2 for those who did not achieve this status (SD = 6.6). Seventy-one percent of participants without a comorbid diagnosis (OCD-only) and 67% of participants with a comorbid diagnosis exhibited clinically significant change. This difference also was not statistically significant ($\chi^2(1) = 0.21$, $p = 0.64$).

Table 1
Baseline clinical characteristics of OCD patients with and without comorbid diagnoses. Data include mean (SD) unless otherwise specified.

Source	OCD, $n = 45$	OCD + any comorbidity, $n = 98$	OCD + PD, $n = 24$	OCD + SP, $n = 42$	OCD + GAD ,$n = 54$	OCD + MDD, $n = 44$
Sex (male), n	26	42	11	21	25	23
Age	30.2 (14.6)	31.1 (10.1)	30.0 (10.0)	30.9 (11.4)	30.1 (11.1)	32.4 (10.0)
Y-BOCS obsessions	12.4 (3.7)[a,b,c]	14.4 (3.1)[a]	14.5 (3.2)	14.5 (3.6)	15.3 (2.8)[b]	13.3 (2.5)[b]
Y-BOCS compulsions	12.94 (3.8)[a,b]	14.1 (3.2)[a]	14.8 (3.0)	14.5 (3.5)	15.5 (2.7)[b]	14.2 (3.3)
Y-BOCS Total Score	25.3 (6.8)[a,b]	28.8 (5.8)[a]	29.3 (5.9)	28.9 (6.5)	30.8 (5.2)[b]	29.2 (5.3)
CGI-S	4.6 (.97)[a,b]	4.9 (.89)[a]	4.9 (.95)	4.9 (.87)	5.1 (.90)[b]	5.0 (.90)

Note: Y-BOCS, Yale-Brown Obsessive–Compulsive Scale; CGI-S, Clinical Global Impression Severity Scale; OCD, obsessive–compulsive disorder; PD, panic disorder; SP, social phobia; GAD, generalized anxiety disorder; MDD, major depressive disorder. Matching superscripts indicate significant differences at the $p = .05$ level (minimum).

Table 2
Effect of baseline comorbidity status on post-treatment Y-BOCS scores.

	Post-treatment Y-BOCS Total Mean (SD)	*F*-value	*p*-value	Post-treatment Y-BOCS Obsessions Mean (SD)	*F*-value	*p*-value	Post-treatment Y-BOCS Compulsions Mean (SD)	*F*-value	*p*-value
OCD-only	11.7 (6.3)	–	–	5.8 (3.3)	–	–	5.9 (3.6)	–	–
OCD + any comorbidity	13.3 (8.3)	0.31	0.58	6.8 (4.2)	0.58	.45	6.5 (4.6)	0.08	0.77
OCD + PD	12.3 (8.52)	0.001	0.98	6.0 (4.0)	0.02	.90	6.2 (4.7)	0.004	0.95
OCD + SP	14.2 (8.8)	1.1	0.28	7.2 (4.2)	1.5	.27	6.9 (4.8)	0.68	0.41
OCD + GAD	12.4 (8.2)	0.002	0.88	6.3 (4.2)	0.01	.97	6.2 (4.6)	0.05	0.82
OCD + MDD	13.7 (7.9)	0.42	0.52	6.5 (3.9)	0.98	.33	6.5 (4.2)	0.04	0.84

Note: OCD, obsessive–compulsive disorder; PD, panic disorder; SP, social phobia; GAD, generalized anxiety disorder; MDD, major depressive disorder; Y-BOCS, Yale-Brown Obsessive Compulsive Inventory.

Table 3
Percentage of individuals meeting responder or remission criteria based on the presence of comorbidity pre-treatment.

	Responder		Remitter		Clinically Significant Change	
	Yes	No	Yes	No	Yes	No
OCD-only	82.2%	17.8%	53.3%	46.7%	71.0%	29.0%
OCD + any comorbidity	73.5%	26.5%	44.9%	55.1%	67.0%	33.0%
OCD + PD	87.5%	12.5%	50.0%	50.0%	66.7%	33.3%
OCD + SP	71.4%	28.6%	38.1%	61.9%	71.4%	28.6%
OCD + GAD	81.5%	18.5%	44.9%	55.1%	70.4%	29.6%
OCD + MDD	75.0%	25.0%	45.5%	54.5%	84.1%	15.9%

Note: OCD, obsessive–compulsive disorder; PD, panic disorder; SP, social phobia; GAD, generalized anxiety disorder; MDD, major depressive disorder.

Fisher's Exact Tests revealed no differences in rates of response, remission, or clinically significant change between subjects with OCD-only compared to participants with specific comorbid diagnoses: panic disorder, social phobia, generalized anxiety disorder, or major depressive disorder (see Table 3). Similarly, there were no differences in response ($\chi^2(4) = 6.3$, $p = 0.17$), remission ($\chi^2(4) = 6.1$, $p = 0.18$) or clinically significant change ($\chi^2(4) = 5.9$, $p = 0.22$) on the basis of the number of baseline comorbidities.

3. Discussion

While CBT is indicated as the first line treatment of choice for OCD, more information is needed about how comorbid diagnoses might impact outcome. Previous studies have examined the impact of depressive disorders on response rates (e.g., Abramowitz et al., 2000); however, little data exists on symptom remission, which is considered a practical indicator of change that corresponds with improved quality of life (Huppert, Simpson, Nissenson, Liebowitz, & Foa, 2009). The present study extends the literature by investigating the impact of the presence and type of a number of comorbid diagnoses on CBT response, remission, and clinically significant change rates. Overall, 69% of the current sample met criteria for at least one comorbid diagnosis and, not surprisingly, participants with a comorbid diagnosis experienced more severe pre-treatment symptoms relative to those without a comorbid diagnosis.

Contrary to predictions and past results (e.g., Abramowitz & Foa, 2000; Cottreaux et al., 1993), the presence of a comorbid diagnosis was not associated with a statistically significant attenuated treatment response. Seventy-four percent of participants with a comorbidity were classified as treatment responders, while 82% of participants with OCD alone responded to treatment. This is similar to extant studies which found that neither the type nor number of comorbid diagnoses impacted treatment outcome (Foa et al., 1992; Steketee et al., 1999). Contrary to our hypothesis, the presence of comorbid MDD (or any of three common co-occurring anxiety disorders) did not influence treatment outcomes. Our clinical observation suggests that many individuals experience reduced depressive symptoms following successful OCD treatment, a finding which has been replicated empirically (Barrett, Healy-Farrell, & March, 2004). Taken together, these data suggest that the comorbid disorders examined in this study may not play a major role in impacting response rates among CBT-seeking adults. Such findings suggest that people with the comorbid disorders examined within this study need not be excluded from CBT research studies. On balance, inclusion and exclusion criteria for our sample limited the inclusion of certain comorbidities that could hinder response such as bipolar disorder, schizophrenia-spectrum disorders or substance abuse. It is quite likely – and consistent with our clinical experiences – that these diagnoses would negatively impact treatment response/remission. As well, it is possible that those participants who were severely depressed or otherwise impacted by their comorbid condition were less likely to present for treatment thereby providing somewhat of a misrepresentation of the role of such comorbid conditions in CBT outcome.

There are some limitations inherent to this study which should be acknowledged. First, raters were aware that participants were receiving treatment, and inter-rater reliability of the ADIS was not measured. However, due to the post-hoc nature of the anal-

yses, raters were not aware of the hypotheses of the present study and raters were trained to strict reliability standards as part of their preparation as independent evaluators for other studies. Second, our sample was homogeneous in terms of ethnicity; thus, results may not be immediately generalizable. On balance, participants were recruited from two active treatment facilities located on separate continents and thus are believed to reflect treatment seeking adults. Third, assessments were made immediately following treatment; it will be important to collect follow-up data to determine the duration of treatment effects and remission rates. As a whole, the mental health field lacks data on 'recovery' or extended duration without relapse (Frank et al., 1991). Fourth, it will be important to examine remission rates of the comorbid diagnoses when conducting future studies. Fifth, this study is unable to examine the relationship of pre-treatment co-morbid symptom severity (e.g., anxiety/depressive symptoms) and treatment outcome. Understanding these relations may be helpful in providing additional insight regarding prognostic indicators of CBT for OCD. Finally, this research focused on generally moderate levels of commonly occurring comorbidities, and it does not address how other diagnoses (e.g., psychosis, bipolar disorder, substance abuse, or Axis II psychopathology) – or more severe levels of the presently studied diagnoses – would affect CBT outcomes. Within these limitations, these data provide additional evidence that CBT is a robust and effective treatment for OCD and that the presence of a comorbid diagnosis may did not negatively impact treatment response or remission rates.

References

Abramowitz, J. (1997). Effectiveness of psychological and pharmacological treatments for obsessive–compulsive disorder: A quantitative review. *Journal of Consulting and Clinical Psychology*, *65*, 44–52.

Abramowitz, J., & Foa, E. (2000). Does comorbid major depressive disorder influence outcome of exposure and response prevention for OCD? *Behavior Therapy*, *31*, 795–800.

Abramowitz, J., Franklin, M. E., Street, G. P., Kozak, M. J., & Foa, E. B. (2000). Effects of comorbid depression on response to treatment for obsessive–compulsive disorder. *Behavior Therapy*, *31*, 517–528.

Abramowitz, J., Whiteside, S. P., & Deacon, B. J. (2005). The effectiveness of treatment for pediatric obsessive–compulsive disorder: A meta-analysis. *Behavior Therapy*, *36*, 55–63.

AuBuchon, P. G., & Malatesta, V. J. (1994). Obsessive–compulsive patients with comorbid personality disorder: Associated problems and response to a comprehensive behavior therapy. *Journal of Clinical Psychiatry*, *55*, 448–453.

Baer, L., Jenike, M. A., Black, D. W., & Treece, C. (1992). Effect of axis II diagnosis on treatment outcome with clomipramine in 55 patients with obsessive–compulsive disorder. *Archives of General Psychiatry*, *49*, 862–866.

Barrett, P., Healy-Farrell, L., & March, J. S. (2004). Cognitive-behavioral family treatment of childhood obsessive–compulsive disorder: A controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*, 46–63.

Bergeron, R., Ravindran, A. V., Chaput, Y., Goldner, E., Swinson, R., van Ameringen, M. A., et al. (2002). Sertraline and fluoxetine treatment of obsessive–compulsive disorder: Results of a double-blind, 6-month treatment study. *Journal of Clinical Psychopharmacology*, *22*, 148–154.

Bienvenu, O. J., Samuels, J. F., Riddle, M. A., Hoehn-Saric, R., Liang, K. Y., Cullen, B. M., et al. (2000). The relationship of obsessive–compulsive disorder to possible spectrum disorders: Results from a family study. *Biological Psychiatry*, *48*, 287–293.

Bjorgvinsson, T. P., Wetterneck, C. T. P., Powell, D. M. P., Chasson, G. S. M., Webb, S. A. P., Hart, J. M., et al. (2008). Treatment outcome for adolescent obsessive–compulsive disorder in a specialized hospital setting. *Journal of Psychiatric Practice*, *14*, 137–145.

Brown, T. A., Barlow, D. H., & Dinardo. (2004). *Anxiety Disorders Interview Schedule Adult Version (ADIS-IV): Client interview schedule*. New York: Oxford University Press.

Coffey, B. J., Miguel, E. C., Biederman, J., Baer, L., Rauch, S. L., O'Sullivan, R. L., et al. (1998). Tourette's disorder with and without obsessive–compulsive disorder in adults: Are they different? *Journal of Nervous and Mental Disease*, *186*, 201–206.

Cottreaux, J., Messy, P., Marks, I. M., Mollard, E., & Bouvard, M. (1993). Predictive factors in the treatment of obsessive–compulsive disorder with fluvoxamine and or behavior therapy. *Behavioral Psychotherapy*, *21*, 45–50.

Chen, Y. W., & Dilsaver, S. C. (1995). Comorbidity for obsessive–compulsive disorder in bipolar and unipolar disorders. *Psychiatry Research*, *59*, 57–64.

Christensen, L., & Mendoza, J. L. (1986). A method of assessing change in a single subject: An alteration of the RC index. *Behaviour Therapy*, *17*, 305–308.

Crino, R., Slade, T., & Andrews, G. (2005). The changing prevalence and severity of obsessive-compulsive disorder criteria from DSM-III to DSM-IV. *American Journal of Psychiatry*, *162*, 876–882.

Dar, R., & Greist, J. H. (1992). Behavior therapy for obsessive compulsive disorder. *Psychiatric Clinics of North America*, *15*, 885–894.

Dreessen, L., Hoekstra, R., & Arntz, A. (1997). Personality disorders do not influence the results of cognitive and behavior therapy for obsessive–compulsive disorder. *Journal of Anxiety Disorders*, *11*, 503–521.

Eddy, K. T., Dutra, L., Bradley, R., & Westen, D. (2004). A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive–compulsive disorder. *Clinical Psychology Review*, *24*, 1011–1030.

Eisen, J., & Steketee, G. (1998). Course of illness in obsessive–compulsive disorder. In: L. J. Dickstein, M. B. Riba, & J. M. Oldham (Eds.), *Review of psychiatry*. Washington, DC: American Psychiatric Press.

Fisher, P. L., & Wells, A. (2005). How effective are cognitive and behavioral treatments for obsessive-compulsive disorder? A clinical significance analysis. *Behaviour Research and Therapy*, *43*, 1543–1558.

Foa, E. B., Grayson, J. B., Steketee, G. S., Doppelt, H. G., Turner, R. M., & Latimer, P. R. (1983). Success and failure in the behavioral treatment of obsessive–compulsives. *Journal of Consulting and Clinical Psychology*, *51*, 287–297.

Foa, E. B., Kozak, M. J., Steketee, G. S., & McCarthy, P. R. (1992). Treatment of depressive and obsessive–compulsive symptoms in OCD by imipramine and behavior therapy. *British Journal of Clinical Psychology*, *31*, 279–292.

Foa, E. B., Liebowitz, M. R., Kozak, M. J., Davies, S., Campeas, R., Franklin, M. E., et al. (2005). Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive–compulsive disorder. *American Journal of Psychiatry*, *162*, 151–161.

Frank, E., Prien, R. F., Jarrett, R. B., Keller, M. B., Kupfer, D. J., Lavori, P. W., et al. (1991). Conceptualization and rationale for consensus definitions of terms in major depressive disorder. Remission, recovery, relapse, and recurrence. *Archives of General Psychiatry*, *48*, 851–855.

Gershuny, B. S., Baer, L., Jenike, M. A., Minichiello, W. E., & Wilhelm, S. (2002). Comorbid posttraumatic stress disorder: Impact on treatment outcome for obsessive–compulsive disorder. *American Journal of Psychiatry*, *159*, 852–854.

Goodman, W. K., & Price, L. H. (1992). Assessment of severity and change in obsessive compulsive disorder. *Psychiatric Clinics of North America*, *15*, 861–869.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Delgado, P., Heninger, G. R., et al. (1989). The Yale-Brown Obsessive Compulsive Scale. II. Validity. *Archives of General Psychiatry*, *46*, 1012–1016.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., et al. (1989). The Yale-Brown Obsessive Compulsive Scale. I. Development, use, and reliability. *Archives of General Psychiatry*, *46*, 1006–1011.

Guy, W. (1976). *Clinical global impressions ECDEU assessment manual for psychopharmacology* Rockville, MD: National Institute for Mental Health. (Vol. Revised DHEW Pub. (ADM) pp. 218–222).

Horwath, E., & Weissman, M. (2000). The epidemiology and cross-national presentation of obsessive–compulsive disorder. *Psychiatric Clinics of North America*, *23*, 493–506.

Huppert, J. D., Simpson, H. B., Nissenson, K. J., Liebowitz, M. R., & Foa, E. B. (2009). Quality of life and functional impairment in obsessive–compulsive disorder: A comparison of patients with and without comorbidity, patients in remission, and healthy controls. *Depression and Anxiety*, *26*, 39–45.

Jacobson, N. S., Follette, W. C., & Revenstorf, D. (1984). Psychotherapy outcome research: Methods for reporting variability and evaluating clinical significance. *Behaviour Therapy*, *15*, 336–352.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology*, *59*, 12–19.

Keeley, M. L., Storch, E. A., Merlo, L., & Geffken, G. R. (2008). Clinical predictors of response to cognitive-behavioral therapy for obsessive–compulsive disorder. *Clinical Psychology Review*, *28*, 118–130.

Kobak, K. A., Greist, J. H., Jefferson, J. W., Katzelnick, D. J., & Henk, H. J. (1998). Behavioral versus pharmacological treatments of obsessive–compulsive disorder: A meta-analysis. *Pscyhopharmacology*, *136*, 205–216.

Lezuez, C. W., Hopko, D. R., & Hopko, S. D. (2001). A brief behavioral activation treatment for depression. *Behavior Modification*, *25*, 255–286.

March, J. S., Frances, A., Carpenter, D., & Kahn, D. A. (1997). The expert consensus guideline series: Treatment of obsessive–compulsive disorder. *Journal of Clinical Psychiatry*, *58*(Suppl. 4), 65–72.

Mavissakalian, M., Hamann, M. S., & Jones, B. (1990). DSM-III personality disorders in obsessive–compulsive disorders: Changes with treatment. *Comprehensive Psychiatry*, *31*, 432–437.

McDonald, R., Marks, I. M., & Blizzard, R. (1988). Quality assurance of outcome in mental health care: A model of routine care in clinical settings. *Health Trends*, *20*, 111–114.

Minichiello, W. E., Baer, L., & Jenike, M. A. (1987). Schizotypal personality disorder: A poor prognostic indicator for behavior therapy in the treatment of obsessive–compulsive disorder. *Journal of Anxiety Disorders*, *1*, 273–276.

Morillo, C., Belloch, A., & Garcia-Soriano, G. (2007). Clinical obsessions in obsessive–compulsive patients and obsession-relevant intrustive thoughts in non-clinical, depressed and anxious subjects: Where are the differences? *Behaviour Research and Therapy*, *45*, 1319–1333.

Nestadt, G., Samuels, J., Riddle, M. A., Liang, K. Y., Bienvenu, O. J., Hoehn-Saric, R., et al. (2001). The relationship between obsessive–compulsive disorder and anxi-

ety and affective disorders: Results from the Johns Hopkins OCD Family Study. *Psychological Medicine*, *31*, 481–487.

Orloff, L. M., Battle, M. A., Baer, L., Ivanjack, L., Pettit, A. R., Buttolph, M. L., et al. (1994). Long-term follow-up of 85 patients with obsessive–compulsive disorder. *American Journal of Psychiatry*, *151*, 441–442.

O'Sullivan, G., Noshirvani, H., Marks, I. M., Montiero, W., & Lelliott, P. (1991). Six-year follow-up after clomipramine and exposure therapy for obsessive–compulsive disorder. *Journal of Clinical Psychiatry*, *52*, 150–155.

Overbeek, T., Schruers, K., Vermetten, E., & Griez, E. (2002). Comorbidity of obsessive–compulsive disorder and depression: Prevalence, symptom severity, and treatment effect. *Journal of Clinical Psychiatry*, *63*, 1106–1112.

Pinto, A., Mancebo, M. C., Eisen, J. L., Pagano, M. E., & Rasmussen, S. A. (2006). The Brown Longitudinal Obsessive–Compulsive Study: Clinical features and symptoms of the sample at intake. *Journal of Clinical Psychistary*, *67*, 703–711.

Ravizza, L., Barzega, G., Bellino, S., Bogetto, F., & Maina, G. (1995). Predictors of drug treatment response in obsessive–compulsive disorder. *Journal of Clinical Psychiatry*, *56*, 368–378.

Samuels, J., & Nestadt, G. (1997). Epidemiology and genetics of obsessive–compulsive disorder. *International Review of Psychiatry*, *9*, 61–71.

Samuels, J., Nestadt, G., Bienvenu, O. J., Costa, P. T., Jr., Riddle, M. A., Liang, K. Y., et al. (2000). Personality disorders and normal personality dimensions in obsessive–compulsive disorder. *British Journal of Psychiatry*, *177*, 457–462.

Simpson, H. B., Foa, E. B., Liebowitz, M. R., Ledley, D. R., Huppert, J. D., Cahill, S., et al. (2008). A randomized, controlled trial of cognitive-behavioral therapy for augmenting pharmacotherapy in obsessive–compulsive disorder. *American Journal of Psychiatry*, *165*, 621–630.

Simpson, H. B., Huppert, J. D., Petkova, E., Foa, E. B., & Liebowitz, M. R. (2006). Response versus remission in obsessive–compulsive disorder. *Journal of Clinical Psychiatry*, *67*, 269–276.

Steketee, G. (1993). Social support and treatment outcome of obsessive–compulsive disorder at 9-month follow-up. *Behavioural Psychotherapy*, *21*, 81–95.

Steketee, G., Chambless, D. L., & Tran, G. Q. (2001). Effects of axis I and II comorbidity on behavior therapy outcome for obsessive–compulsive disorder and agoraphobia. *Comprehensive Psychiatry*, *42*, 76–86.

Steketee, G., Eisen, J., Dyck, I., Warshaw, M., & Rasmussen, S. (1999). Predictors of course in obsessive–compulsive disorder. *Psychiatric Research*, *89*, 229–238.

Storch, E. A., Merlo, L. J., Larson, M., Geffken, G. R., Lehmkuhl, H. D., Jacob, M. L., et al. (2008). The impact of comorbidity on cognitive-behavioral therapy response in pediatric obsessive compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*, 583–592.

Woody, S. R., Steketee, G., & Chambless, D. L. (1995). Reliability and validity of the Yale-Brown Obsessive-Compulsive Scale. *Behaviour Research and Therapy*, *33*, 597–605.

Yaryura-Tobias, J. A., Grunes, M. S., Todaro, J., McKay, D., Neziroglu, F. A., & Stockman, R. (2000). Nosological insertion of axis I disorders in the etiology of obsessive–compulsive disorder. *Journal of Anxiety Disorders*, *14*, 19–30.

Zitterl, W., Demal, U., Aigner, M., Lenz, G., Urban, C., Zapotoczky, H. G., et al. (2000). Naturalistic course of obsessive–compulsive disorder and comorbid depression: Longitudinal results of a prospective follow-up study of 74 actively treated patients. *Psychopathology*, *33*, 75–80.