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Treatment of Obsessive-Compulsive Disorder Complicated by Comorbid Eating Disorders

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Abstract

Purpose—Eating disorders and obsessive-compulsive disorder (OCD) commonly co-occur, but there is little data for how to treat these complex cases. To address this gap, we examined the naturalistic outcome of 56 patients with both disorders, who received a multimodal treatment program designed to address both problems simultaneously.

Methods—A residential treatment program developed a cognitive-behavioral approach for patients with both OCD and an eating disorder by integrating exposure and response prevention (ERP) treatment for OCD with ERP strategies targeting eating pathology. Patients also received a supervised eating plan, medication management, and social support. At admission and discharge, patients completed validated measures of OCD severity (the Yale-Brown Obsessive-Compulsive Scale—Self Report [Y-BOCS-SR]), eating disorder severity (the Eating Disorders Examination-Questionnaire), and depressive severity (the Beck Depression Inventory II [BDI-II]). Body mass index (BMI) was also measured. Paired-sample *t*-tests examined change on these measures.

Main Results—Between 2006 and 2011, 56 individuals completed all study measures at admission and discharge. Mean length of stay was 57 days (*SD* = 27). Most (89%) were on psychiatric medications. Significant decreases were observed in OCD severity, eating disorder severity, and depression. Those with bulimia nervosa showed more improvement than those with anorexia nervosa. BMI significantly increased, primarily among those underweight at admission.

Conclusion—Simultaneous treatment of OCD and eating disorders using a multimodal approach that emphasizes ERP techniques for both OCD and eating disorders can be an effective treatment strategy for these complex cases.

Keywords

anorexia nervosa; bulimia nervosa; cognitive-behavioral therapy; exposure therapy; OCD

Introduction

Cognitive-behavioral therapy (CBT) is effective for various psychiatric disorders (Barlow 2008). However, CBT's efficacy has largely been demonstrated in clinical trials that recruit individuals with one primary disorder, use disorder-specific CBT protocols, and exclude or standardize concomitant medication. Such trials do not mirror routine clinical practice where many patients suffer from multiple psychiatric disorders and receive a range of medications. Findings from clinical trials must be translated into effective treatment plans for these more complex cases.

Patients with comorbid obsessive-compulsive disorder (OCD) and an eating disorder are an example of this clinical dilemma, as these disorders often co-occur (Altman & Shankman, 2009; Angst *et al.*, 2004; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Swin-bourne & Touyz, 2007). Randomized controlled trials have identified evidence-based treatments for both disorders. For OCD, treatments include CBT emphasizing exposure and response prevention (ERP), serotonin reuptake inhibitors (SRIs) (clomipramine and the selective SRIs), or their combination (American Psychiatric Association, 2007). For eating disorders, treatments include: CBT emphasizing monitoring eating and cognitive restructuring (for anorexia nervosa [AN] and bulimia nervosa [BN]), fluoxetine (for BN), and weight restoration (for AN; American Psychiatric Association, 2006). However, OCD trials have generally excluded patients with eating disorders (e.g., Foa *et al.*, 2005; Goodman *et al.*, 1996; Hollander *et al.*, 2003; Tollefson *et al.*, 1994) and eating disorder trials usually do not report the frequency of comorbid OCD or its potential impact on treatment outcome (e.g., Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992; Goldstein, Wilson, Thompson, Potvin, & Rampey, 1995; McIntosh *et al.*, 2005).

Naturalistic studies suggest a complex relationship between OCD and eating disorders (Cumella, Kally, & Wall, 2007). One study of 508 females receiving treatment for an eating disorder (AN, BN, or eating disorder not otherwise specified [ED-NOS]), half of whom had comorbid OCD, found a reciprocal relationship, such that symptom improvement in one led to symptom improvement in the other (Olatunji, Tart, Shewmaker, Wall, & Smits, 2010). One implication of these findings is that simultaneous treatment of both conditions might lead to synergistic treatment effects.

ERP is highly effective for OCD as monotherapy or to augment SRIs (Foa *et al.*, 2005; Simpson *et al.*, 2008). ERP techniques have been used in BN with some success (Bulik *et al.*, 1998; Gray & Hoage, 1990; Kennedy *et al.*, 1995; Leitenberg, Rosen, Gross, Nudelman, & Vara, 1988; Wilson *et al.*, 1991). Pilot data suggest that exposure techniques may also help patients with AN (Steinglass *et al.*, 2011; Steinglass *et al.*, 2012). Thus, one approach to patients with both OCD and an eating disorder is to adapt ERP to address both OCD and pathological eating fears and behaviors, and to couple this as needed with other evidence-based interventions, such as weight restoration (for AN) and medication management (for OCD or BN).

In this paper, we describe the outcome of 56 patients with both OCD and an eating disorder (AN, BN, or ED-NOS), who entered a residential treatment program that offered a cognitive-behavioral program that integrated standard ERP for OCD with ERP strategies specifically adapted for eating disorder pathology. Patients also received a structured supervised eating plan, medication management, and other psychosocial supports. Using validated outcome measures, we investigated how effective this multimodal program was at treating such complex cases. On the basis of the literature for both disorders suggesting efficacy of cognitive-behavioral approaches and on the conceptual overlap in the

psychopathology underlying these conditions, we hypothesized that the treatment program would lead to clinically significant change in both OCD and eating disorder severity.

Methods

Overview

The data came from patients in the Comorbid Program, a residential treatment program at Rogers Memorial Hospital in Oconomowoc, WI. Patients provided written informed consent for their data to be used for research. The Rogers Center for Research and Training and the Institutional Review Board of the University of Wisconsin-Milwaukee approved the study.

The program

The Comorbid Program is an eight-bed, residential facility designed to treat adults (18 years of age) with OCD and coexisting eating disorders who have not adequately responded to outpatient treatment. Patients are not admitted with active psychosis, mania, homicidality, or suicidality; substance abuse or electroconvulsive therapy within the past 30 days; or mental retardation. There is no minimum body mass index (BMI) required at admission. Some patients are transferred from an inpatient setting where they may have achieved partial weight restoration. The program accepts patients who self-pay or have private insurance.

The hallmark of this program is the use of ERP techniques to treat both OCD and pathological eating fears and behaviors. Standard ERP procedures are used for OCD symptoms (Kozak & Foa, 1997). Briefly, therapists help patients to generate a hierarchy of situations that trigger their anxiety or distress from least to most feared. Then, therapists help patients face these situations (“exposures”) to promote habituation to the anxiety that these situations trigger, starting with easier triggers and moving up the OCD hierarchy. In parallel, patients are asked to stop avoidance behaviors and rituals (“response prevention”) to break the connection between rituals and anxiety relief. Together, these procedures help disconfirm patients’ irrational beliefs. Therapists practice these steps with patients in session and assign specific exercises for between-session practice.

ERP procedures are also used to address eating disorder symptoms. As is standard in eating disorder programs, there is a minimum caloric goal per day for each patient to either restore weight if the patient is underweight (e.g., up to 90 calories/kg of body weight/day) or to maintain an appropriate weight (i.e., 50 calories/kg of body weight/day). However, what differs in this program is that a hierarchy of feared situations related to the eating disorder is also created, and the therapist helps patients face these situations in graduated steps of increasing difficulty. In addition, the menu plan is consistent with the ERP hierarchy: the initial food prescription is comprised entirely of foods low on the individual’s hierarchy and then changes as the patient habituates to the anxiety at one level and moves stepwise up the hierarchy. While this may resemble the introduction of challenge foods that are common in eating disorders treatment, it is unusual in the detail with which food fears are rated and in how the fear hierarchy shapes the menu plans. In this way, the approach more closely resembles ERP than classic CBT for eating disorders. Specific exposures to feared foods and eating situations (e.g., eating in public) are also done. To address fears about body shape, body image exposures may be conducted, including viewing disliked body parts in a mirror or wearing normally fitting rather than excessively loose clothing. Patients are asked to do exposures without engaging in compensatory behaviors related to their eating disorder (e.g., restricting during a future meal, vomiting, excessive exercise, or overeating in reaction to negative emotions). Two case histories are provided (see Table 6) that illustrate how such ERP procedures were applied to individual patients.

This ERP program is delivered each week-day in a three-hour block. During this block, patients participate in (1) individual therapy sessions (each lasting 30–45 minutes) with a behavioral specialist (typically a master's-level clinician); (2) supervised exposures with other staff; and (3) self-exposures. They are also assigned 90 minutes of homework each day to practice assigned exposures without ritualizing. The goal is for patients to reach at least 70% up their hierarchy of fear prior to discharge and to continue to the top as an outpatient. To complement these ERP techniques, cognitive restructuring techniques are used to challenge irrational thoughts related to OCD and the eating disorder, constituting about 10% of treatment time.

Adjunctive treatment is also provided. Dieticians see patients at least weekly to assess the caloric prescription and to monitor weight and adherence with the structured meal plan that follows the food hierarchy (as described above). Meals are initially supervised by staff. As patients improve, they are expected to eat more independently. Psychiatrists see patients at least weekly to monitor medications. Nurses see patients as needed regarding general medical issues. Social workers meet with patients individually for an hour once or twice a week (to provide supportive psychotherapy and discharge planning) and in a group format every weekday for an hour (to provide psychoeducation about OCD and eating disorders and to facilitate peer support). Social workers also conduct weekly family sessions (in person or by phone). These sessions are used to provide psychoeducation, to discuss how family members can support the patient's treatment goals, and to update the family on patient progress.

Assessments

Admission diagnoses were made by board-certified psychiatrists according to DSM-IV-TR criteria. To determine these diagnoses, these psychiatrists, all experts in OCD and anxiety disorders, completed in-depth reviews of patients' previous records as well as an interview with patients upon admission.

At admission and discharge, patients were asked to complete self-report outcome measures; BMI (kg/m^2) was also calculated. The self-report measures were (1) the Yale-Brown Obsessive-Compulsive Scale—Self Report (Y-BOCS-SR; Steketee *et al.*, 1996); (2) the Eating Disorder Examination Questionnaire (EDE-Q; Luce & Crowther, 1999); and (3) the Beck Depression Inventory-II (BDI-II; Beck *et al.*, 1996). The Y-BOCS-SR consists of a checklist of obsessions and compulsions and a scale (0–40) that assesses their severity in the last week. To facilitate comparison with prior OCD clinical trials (Simpson *et al.*, 2006), we also calculated the proportion of patients at discharge who met OCD response criteria (Y-BOCS-SR decrease $\geq 25\%$) and who had mild-to-minimal OCD symptoms (Y-BOCS-SR ≤ 12). The EDE-Q consists of 38 questions about behaviors and cognitions relating to eating, body shape, and weight in the prior 28 days; scores from four subscales (eating restraint, eating concerns, shape concerns, and weight concerns) are averaged to provide a global score. In a healthy young adult female population, the mean global EDE-Q (standard deviation [SD]) is approximately 1.6 (1.3) (Mond *et al.*, 2006; Welch, Birgegard, Parling, & Ghaderi, 2011). The BDI-II is a 21-item measure of depressive severity in the prior week. Finally, for the Y-BOCS-SR and the EDE-Q, we used Jacobson and Truax (1991) methods to calculate the proportion of patients who met reliable change (defined as Y-BOCS-SR change of at least 7 points and an EDE-Q change of at least .81) and the proportion who achieved end-state functioning within the nonpatient distribution (defined as within one SD of a normal population¹: Y-BOCS-SR ≤ 14 ; EDE-Q < 2.3). Normative data for the Y-BOCS-SR were taken from Steketee *et al.* (1996), in which the mean (SD) and test-retest reliability were 8.0 (6.6) and $r = .88$. Normative data for the EDE-Q global scale were taken from Ro *et al.* (2010), in which the corresponding values were 1.17 (1.11) and .93.

Statistical methods

Treatment outcome was evaluated with paired-sample *t*-tests applied to admission and discharge scores for each continuous outcome measure (Y-BOCS-SR, EDE-Q, BMI, and BDI-II). Separate one-way analyses of variance (ANOVAs) explored whether the three eating disorder groups (AN, BN, and ED-NOS) differed in change scores on the four outcome measures. Significant group effects were followed by post-hoc *t*-tests for each pairwise comparison. Similar methods were used to explore whether treatment outcome differed between those at admission with low weight versus without low weight (BMI < 18.5 kg/m² vs. BMI ≥ 18.5 kg/m²). Unless otherwise specified, statistical tests were two-tailed with level of significance $\alpha = .05$.

Results

Sample

One hundred eight patients diagnosed with OCD and a comorbid eating disorder were admitted to the Comorbid Program between June 2006 and July 2011. Fifty-six (52%) completed all study measures (Y-BOCS-SR, EDE-Q, BDI-II, and BMI) at both admission and discharge and therefore are included in this report. Completeness of data was the only inclusion criteria, not duration of stay, circumstances of discharge, or response to treatment. The mean length of stay (SD) for these 56 people was 57 days (27, range 12–131).

Demographic and clinical features of these 56 people are provided in Tables 1 and 2. At admission, they had clinically significant OCD, with a mean Y-BOCS-SR (SD) of 24.6 (7.2). Somewhat more were diagnosed with AN than with BN or ED-NOS. The mean EDE-Q global score (SD) was 3.8 (1.5), reflecting significant pathology (Fairburn & Beglin, 1994). Mean BMI (SD) at admission was 21.7 (7.4), but ranged from 13.8 to 43.1, as would be expected in this heterogeneous group. The mean (SD) BDI-II score at admission was 32.6 (13.4).

Fifty-two people admitted to the program were not included in the analyses because they were missing data either at admission or discharge. Those with admission data did not significantly differ on these clinical features when compared to the study sample (i.e., mean Y-BOCS-SR scores [SD] = 25.5 [7.7], $t(96) = .60$, $p = .55$; mean EDE-Q global scale [SD] = 3.5 [1.7], $t(90) = -1.0$, $p = .31$; mean BMI [SD] = 21.0 [5.1], $t(106) = -.66$, $p = .46$; and mean BDI-II [SD] = 30.4 [14.5], $t(98) = -.78$, $p = .44$). The 52 not included in the study also did not differ in mean length of stay (53 days [34], range 3–161, $t(106) = -.71$, $p = .48$).

Treatment outcome

Patients experienced significant changes in all four outcome measures at discharge (Table 2). There were significant decreases in OCD severity, as measured by the Y-BOCS-SR. There were also significant decreases in eating disorder severity, as measured by the global EDE-Q score and reflected in all four EDE-Q subscales (eating restraint, eating concerns, shape concerns, and weight concerns). There were also significant increases in BMI and significant decreases in depressive severity, as measured by the BDI-II. All changes remained significant even with Bonferroni correction for eight comparisons (new $\alpha = .006$).

¹Note Jacobson and Truax (Jacobson & Truax, 1991, method b; see p. 13) operationally define return to normal functioning when, “[t]he level of functioning subsequent to therapy should fall within the range of the functional or normal population, where range is defined as within two standard deviations of the mean of that population.” Given that a Y-BOCS score of 16 is a commonly used criterion for admission into a study of treatment for OCD, we feel the criterion of being within two SDs of the mean for a normal sample after treatment is too liberal and have therefore opted for the more conservative criterion of having a posttreatment score that is within 1 SD of the normative mean.

At discharge, 45 of 56 (80%) met response criteria for OCD (25% Y-BOCS-SR reduction) and 28 of 56 (50%) had mild or minimal OCD symptoms (Y-BOCS-SR = 12). Following the methods of Jacobson and Truax (1991), 40 of 56 (71%) met the reliable change index (RCI) for the Y-BOCS-SR (7 points), and 32 of 56 (57%) had clinically significant change (Y-BOCS-SR = 14). At discharge, 36 of 56 (64%) met the RCI for the EDE-Q (.81 points), and 33 of 56 (59%) had clinically significant change (EDE-Q < 2.3).

The effect of eating disorder and BMI on outcome

As shown in Table 3, individuals with AN, BN, and ED-NOS did not significantly differ at admission in OCD, eating disorder, or depressive severity (all p -values > .25). Length of stay in days also did not differ (AN = 56.8 [29.8]; BN = 47.4 [20.6]; ED-NOS = 63.4 [25.5], $F(2, 53) = 1.5, p > .23$). Groups differed at admission on BMI, $F(2, 53) = 6.2, p < .01$. Post-hoc pairwise comparisons indicated individuals with AN had lower BMI scores than those with either BN or ED-NOS (both p -values < .01), those with BN did not differ from those with ED-NOS ($p > .69$). As shown in Table 3, individuals with AN, BN, and ED-NOS all showed reductions in OCD, eating disorder, and depressive severity. At the same time, BN patients had significantly more improvement than AN patients on the Y-BOCS-SR, EDE-Q, and BDI-II, and AN patients had significantly greater increases in BMI.

As shown in Table 4, individuals with and without low BMI did not significantly differ at admission in OCD, eating disorder, or depressive severity (all p -values > .40). Length of stay in days also did not differ (low BMI: 62.1 [24.9]; without low BMI: 52.9 [27.5], $F(1, 54) = 1.7, p > .20$). As shown in Table 4, patients with and without low BMI both showed large reductions in OCD, eating disorder, and depressive severity, with no significant group differences. Those with low BMI at admission had a significantly greater increase in BMI at discharge; this was expected given that they received a weight restoration (versus a weight maintenance) diet.

Medication status

Most patients ($n = 50$ [89%]) were receiving psychiatric medications at admission. The proportion of patients receiving different classes of medication is shown in Table 5(a), with the most common class being antidepressants. At discharge, there was a small increase in the use of each class of medication (Table 5(b)). Only a few patients changed their medication, with the addition of a medication being more common than its removal (Table 5(c)). Antipsychotic medication was the class most commonly added (25% of patients); benzodiazepine was the class most commonly removed (9% of patients). There were no significant differences at admission in the percentage of patients with the three eating disorder diagnoses who were prescribed antidepressants, antipsychotics, mood stabilizers/anticonvulsants, or benzodiazepines (all $\chi^2 < 1.9$, all p -values > .40). At discharge, there were no differences among the three eating disorders in the percentage of patients prescribed antidepressants, antipsychotics, or mood stabilizers/anticonvulsants (all $\chi^2 < 2.5$, all p -values > .42), but there was a trend toward a differences in benzodiazepines (AN = 38%, BN = 12%, and ED-NOS = 63%; $\chi^2(2) = 5.9, p < .06$). There were no significant difference at admission or at discharge in the percentage of patients in the two BMI groups (BMI < 18.5 kg/m² vs. BMI ≥ 18.5 kg/m²) who were prescribed any of the four classes of psychiatric medications (antidepressants, antipsychotics, mood stabilizers/anticonvulsants, or benzodiazepines; all $\chi^2 < 2.3$, all p -values > .13).

Discussion

This study investigated the outcome of patients diagnosed with OCD and an eating disorder, who entered a residential treatment program designed to address both disorders simultaneously. The uniqueness of the program was the use of ERP that focused not only on OCD fears and rituals but also on fears and rituals around food and body image. At discharge, the patients had clinically meaningful reductions in OCD severity, eating disorder symptoms, and depressive severity, and increases in BMI. These data suggest that simultaneous treatment of OCD and eating disorders with a multimodal approach can be highly effective.

Despite the presence of comorbid eating disorders, patients in this program experienced robust decreases in OCD severity at discharge. At discharge, the rate of OCD response (80%) and proportion of patients with mild to minimal OCD symptoms (50%) in this sample were similar to those reported in ERP trials that recruited OCD patients without comorbid eating disorders and had similar OCD severity at baseline (Foa *et al.*, 2005; Simpson *et al.*, 2006; Simpson *et al.*, 2008). Significant reductions in OCD severity were observed across all eating disorder diagnoses regardless of BMI. At the same time, BN patients showed more improvement in OCD symptoms than AN patients.

Despite the presence of comorbid OCD, patients in this program also experienced significant reductions in eating disorder severity as measured by the EDE-Q. Improvements occurred across all the eating disorder diagnoses (although BN patients showed more improvement than AN patients) as well as in those with and without low BMI at admission. Shape concern, although improved, remained more elevated at discharge than other EDE-Q subscales, consistent with other eating disorder studies where shape concerns show less improvement or take longer to improve (Fairburn *et al.*, 2009; Rosen, 1996). There was also a significant increase in weight in those with AN and in those with low BMI at admission.

These improvements in OCD symptoms and eating disorder severity occurred despite the high level of depression experienced by many on admission. Moreover, this sample also reported significant reduction in depressive symptoms at discharge, even though the CBT program did not systematically target depression. Since few (7%) started antidepressant treatment during the hospital stay, the reduction in depression was likely due to the psychosocial elements of the program or secondary to improvement in OCD or eating disorder psychopathology. Prior studies found that treatment of OCD or of eating disorders can improve depression (Attia *et al.*, 1998; Foa *et al.*, 1992; Meehan *et al.*, 2006; Storch *et al.*, 2010).

Because of the naturalistic design and multimodal treatment plan, we cannot determine whether the observed improvements in OCD or eating disorder severity were due to the integrated ERP component, other specific aspects of the program (e.g., structured eating), nonspecific effects (e.g., residential setting, social support, and time), or medication changes that occurred during the program. However, some tentative hypotheses can be made. First, the significant decreases in OCD and eating disorder severity were unlikely only due to medication, since many more patients responded to this treatment program than were newly prescribed antidepressants or antipsychotics, two classes of medications with known effects in these conditions as well as known side effects of weight gain. Importantly, patients with AN or low BMI at admission showed the greatest increases in BMI, but were not more likely to be on antipsychotics or antidepressants at admission or discharge than the other groups. Second, the robust decreases in OCD severity are most likely due to the ERP component, given the very low response rates in OCD to nonspecific interventions (Huppert *et al.*, 2004; Simpson *et al.*, 2008). Moreover, although eating disorder treatment on its own

may reduce OCD symptoms (Cumella *et al.*, 2007; Olatunji *et al.*, 2010), no data indicate that it can do so when OCD is as severe as it was in this sample of patients. Finally, the decrease in OCD severity could be responsible in large part for the decrease in depressive severity, as this degree of improvement in comorbid depression after successful ERP treatment has been observed (Foa *et al.*, 1992). Of note, the greatest improvement in depression occurred in those with BN, who also had the largest decreases in OCD severity.

It seems reasonable to assume that improvements in eating disorder symptoms were due in part to the non-ERP components of the program (e.g., sustained nutrition, observation after meals, and supportive psychotherapy). Reducing OCD symptoms with ERP may also have facilitated improvements in eating disorder symptoms (Olatunji *et al.*, 2010). At the same time, the exposure techniques that targeted fear of food and eating could have directly reduced pathological eating behaviors and cognitions, thereby improving eating and facilitating treatment response. Consistent with the latter, ERP techniques have been found useful in BN (Bulik *et al.*, 1998; Kennedy *et al.*, 1995), and exposures focused on fear of food were found to lead to healthier eating behaviors in AN (Steinglass *et al.*, 2012). Any of these possibilities support a multimodal program like this one for people with both OCD and an eating disorder.

The study had several limitations stemming in large part from its naturalistic design and from the fact that the data come from patients treated in a busy clinical setting. First, our sample was restricted to those who entered the residential program and completed all study assessments. This potentially could have led to a biased sample. On the other hand, study participants were not selected based on treatment response and did not differ from nonstudy patients either on clinical features at admission or on length of stay. Second, diagnoses were made by a psychiatrist during an intake evaluation; validated diagnostic instruments were not used to confirm these diagnoses and AN subtypes were not coded. Third, although medications at admission and discharge are included in the research database, exact doses were not; moreover, prior treatment history was not systematically recorded. Fourth, the outcome measures (other than BMI, which was objectively measured) were self-reports that were judged feasible to deliver in this clinical context; the focus was symptom severity. As a result, we cannot describe the frequency of different types of OCD symptoms in this sample. This would be an important area of future study. Finally, the length of stay was variable and there is no follow-up data. Future research should focus on whether the acute reductions in symptoms on this residential unit lead to improved psychosocial functioning and quality of life after discharge.

The findings from this study suggest that simultaneous treatment of OCD and eating disorders can be highly effective when a multimodal approach is used that emphasizes ERP techniques for both OCD and eating disorders and includes weight restoration and medication management. Naturalistic studies like these are an important way to begin to evaluate the effectiveness of evidence-based treatment approaches when they are applied in novel ways to real-world patients.

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References

- Altman SE, Shankman SA. What is the association between obsessive-compulsive disorder and eating disorders? *Clinical Psychology Review*. 2009; 29(7):638–646. [PubMed: 19744759]
- American Psychiatric Association. Treatment of patients with eating disorders. third edition. Vol. 163. *American Journal of Psychiatry*; 2006. p. 4-54.
- American Psychiatric Association. Practice guideline for the treatment of patients with obsessive-compulsive disorder. *American Journal of Psychiatry*. 2007; 164:1–56. [PubMed: 17202533]
- Angst J, Gamma A, Endrass J, Goodwin R, Ajdacic V, Eich D, Rossler W. Obsessive-compulsive severity spectrum in the community: Prevalence, comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience*. 2004; 254(3):156–164. [PubMed: 15205969]
- Attia E, Haiman C, Walsh BT, Flater SR. Does fluoxetine augment the inpatient treatment of anorexia nervosa? *American Journal of Psychiatry*. 1998; 155(4):548–551. [PubMed: 9546003]
- Barlow, DH., editor. *Clinical handbook of psychological disorders*. 4th ed. Guilford Press; New York: 2008.
- Beck AT, Steer RA, Ball R, Ranieri W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *Journal of Personality Assessment*. 1996; 67(3):588–597. [PubMed: 8991972]
- Bulik CM, Sullivan PF, Carter FA, McIntosh VV, Joyce PR. The role of exposure with response prevention in the cognitive-behavioural therapy for bulimia nervosa. *Psychological Medicine*. 1998; 28(3):611–623. [PubMed: 9626717]
- Cumella EJ, Kally Z, Wall AD. Treatment responses of inpatient eating disorder women with and without co-occurring obsessive-compulsive disorder. *Eating Disorders*. 2007; 15(2):111–124. doi: 10.1080/10640260701190634. [PubMed: 17454070]
- Fairburn CG, Beglin SJ. Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders*. 1994; 16(4):363–370. [PubMed: 7866415]
- Fairburn CG, Cooper Z, Doll HA, O'Connor ME, Bohn K, Hawker DM, Palmer RL. Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: A two-site trial with 60-week follow-up. *American Journal of Psychiatry*. 2009; 166(3):311–319. [PubMed: 19074978]
- Fluoxetine Bulimia Nervosa Collaborative Study Group. Fluoxetine in the treatment of bulimia nervosa. A multicenter, placebo-controlled, double-blind trial. *Archives of General Psychiatry*. 1992; 49(2):139–47. [PubMed: 1550466]
- Foa EB, Kozak MJ, Steketee GS, McCarthy PR. Treatment of depressive and obsessive-compulsive symptoms in OCD by imipramine and behaviour therapy. *British Journal of Clinical Psychology*. 1992; 31(Pt, 3):279–292. [PubMed: 1393157]
- Foa EB, Liebowitz MR, Kozak MJ, Davies S, Campeas R, Franklin ME, Tu X. Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*. 2005; 162(1):151–161. [PubMed: 15625214]
- Goldstein DJ, Wilson MG, Thompson VL, Potvin JH, Rampey AH Jr. Long-term fluoxetine treatment of bulimia nervosa, Fluoxetine Bulimia Nervosa Research Group. *British Journal of Psychiatry*. 1995; 166(5):660–666. [PubMed: 7620754]
- Goodman WK, Kozak MJ, Liebowitz M, White KL. Treatment of obsessive-compulsive disorder with fluvoxamine: A multi-centre, double-blind, placebo-controlled trial. *International Clinical Psychopharmacology*. 1996; 11(1):21–29. [PubMed: 8732310]
- Gray JJ, Hoage CM. Bulimia nervosa: Group behavior therapy with exposure plus response prevention. *Psychological Reports*. 1990; 66(2):667–674. [PubMed: 1971954]
- Hollander E, Koran L, Goodman WK, Greist J, Ninan P, Yang HM, Barbato LM. A double-blind, placebo-controlled study of the efficacy and safety of controlled-release fluvoxamine in patients

- with obsessive-compulsive disorder. *Journal of Clinical Psychiatry*. 2003; 64(6):640–647. [PubMed: 12823077]
- Huppert JD, Schultz LT, Foa EB, Barlow DH, Davidson JR, Gorman JM, Woods SW. Differential response to placebo among patients with social phobia, panic disorder, and obsessive-compulsive disorder. *American Journal of Psychiatry*. 2004; 161(8):1485–1487. [PubMed: 15285978]
- Jacobson NS, Truax P. Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology*. 1991; 59(1):12–19. [PubMed: 2002127]
- Kaye WH, Bulik CM, Thornton L, Barbarich N, Masters K. Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *American Journal of Psychiatry*. 2004; 161(12):2215–2221. [PubMed: 15569892]
- Kennedy SH, Katz R, Neitzert CS, Ralevski E, Mendlowitz S. Exposure with response prevention treatment of anorexia nervosa-bulimic subtype and bulimia nervosa. *Behaviour Research and Therapy*. 1995; 33(6):685–689. [PubMed: 7654160]
- Kozak, MJ.; Foa, EB. *Mastery of obsessive-compulsive disorder*. Graywind Publications Incorporated, Oxford University Press, Inc.; New York, New York: 1997.
- Leitenberg H, Rosen JC, Gross J, Nudelman S, Vara LS. Exposure plus response-prevention treatment of bulimia nervosa. *Journal of Consulting and Clinical Psychology*. 1988; 56(4):535–541. [PubMed: 3198810]
- Luce KH, Crowther JH. The reliability of the Eating Disorder Examination-Self-Report Questionnaire Version (EDE-Q). *International Journal of Eating Disorders*. 1999; 25(3):349–351. [PubMed: 10192002]
- McIntosh VV, Jordan J, Carter FA, Luty SE, McKenzie JM, Bulik CM, Joyce PR. Three psychotherapies for anorexia nervosa: A randomized, controlled trial. *American Journal of Psychiatry*. 2005; 162(4):741–747. [PubMed: 15800147]
- Meehan KG, Loeb KL, Roberto CA, Attia E. Mood change during weight restoration in patients with anorexia nervosa. *International Journal of Eating Disorders*. 2006; 39(7):587–589. [PubMed: 16941630]
- Mond JM, Hay PJ, Rodgers B, Owen C. Eating Disorder Examination Questionnaire (EDE-Q): Norms for young adult women. *Behaviour Research and Therapy*. 2006; 44(1):53–62. [PubMed: 16301014]
- Olatunji BO, Tart CD, Shewmaker S, Wall D, Smits JA. Mediation of symptom changes during inpatient treatment for eating disorders: The role of obsessive-compulsive features. *Journal of Psychiatric Research*. 2010; 44(14):910–916. [PubMed: 20359715]
- Ro O, Reas DL, Lask B. Norms for the Eating Disorder Examination Questionnaire among female university students in Norway. *Nordic Journal of Psychiatry*. 2010; 64(6):428–432. [PubMed: 20429744]
- Rosen JC. Body image assessment and treatment in controlled studies of eating disorders. *International Journal of Eating Disorders*. 1996; 20(4):331–343. [PubMed: 8953321]
- Simpson HB, Foa EB, Liebowitz MR, Ledley DR, Huppert JD, Cahill S, Petkova E. A randomized, controlled trial of cognitive-behavioral therapy for augmenting pharmacotherapy in obsessive-compulsive disorder. *American Journal of Psychiatry*. 2008; 165(5):621–630. [PubMed: 18316422]
- Simpson HB, Huppert JD, Petkova E, Foa EB, Liebowitz MR. Response versus remission in obsessive-compulsive disorder. *Journal of Clinical Psychiatry*. 2006; 67(2):269–276. [PubMed: 16566623]
- Steinglass J, Albano AM, Simpson HB, Carpenter K, Schebendach J, Attia E. Fear of food as a treatment target: Exposure and response prevention for anorexia nervosa in an open series. *International Journal of Eating Disorders*. 2012; 45(4):615–621. [PubMed: 21541979]
- Steinglass JE, Sysko R, Glasofer D, Albano AM, Simpson HB, Walsh BT. Rationale for the application of exposure and response prevention to the treatment of anorexia nervosa. *International Journal of Eating Disorders*. 2011; 44(2):134–141. [PubMed: 20127936]
- Steketee G, Frost R, Bogart K. The Yale-Brown Obsessive Compulsive Scale: Interview versus self-report. *Behaviour Research and Therapy*. 1996; 34(8):675–684. [PubMed: 8870295]

- Storch EA, Lewin AB, Farrell L, Aldea MA, Reid J, Geffken GR, Murphy TK. Does cognitive-behavioral therapy response among adults with obsessive-compulsive disorder differ as a function of certain comorbidities? *Journal of Anxiety Disorders*. 2010; 24(6):547–552. [PubMed: 20399603]
- Swinbourne JM, Touyz SW. The comorbidity of eating disorders and anxiety disorders: A review. *European Eating Disorders Review*. 2007; 15:253–274. [PubMed: 17676696]
- Tollefson GD, Rampey AH Jr, Potvin JH, Jenike MA, Rush AJ, Kominguez RA, Genduso LA. A multicenter investigation of fixed-dose fluoxetine in the treatment of obsessive-compulsive disorder. *Archives of General Psychiatry*. 1994; 51(7):559–567. [PubMed: 8031229]
- Welch E, Birgegard A, Parling T, Ghaderi A. Eating disorder examination questionnaire and clinical impairment assessment questionnaire: General population and clinical norms for young adult women in Sweden. *Behaviour Research and Therapy*. 2011; 49(2):85–91. [PubMed: 21185552]
- Wilson GT, Eldredge KL, Smith D, Niles B. Cognitive-behavioral treatment with and without response prevention for bulimia. *Behaviour Research and Therapy*. 1991; 29(6):575–583. [PubMed: 1759956]

Table 1

Demographic and clinical characteristics at admission of 56 patients

Variable	Mean (SD) or number (%)
<i>Demographic characteristics</i>	
Age in years	24.8 (8.5)
Female	45 (80%)
Caucasian	54 (96%)
Not married ^a	53 (98%)
Education level ^b	
< High school diploma	3 (6%)
High school diploma or GED	9 (17%)
Some college	22 (42%)
College degree	18 (35%)
<i>Eating disorder diagnosis by admitting physician</i>	
Anorexia nervosa	23 (41%)
Bulimia nervosa	14 (25%)
Eating disorder not otherwise specified	19 (34%)
<i>Mood disorder diagnosis by admitting physician</i>	
None	34 (61%)
Major depressive disorder	15 (26%)
Bipolar disorder	4 (7%)
Depressive disorder not otherwise specified	3 (5%)

^a n = 54.

^b n = 52.

Table 2

Treatment outcome of 56 patients

Outcome measures	Admission mean (SD)	Discharge mean (SD)	Paired <i>t</i> -test results
Y-BOCS-SR	24.6 (7.2)	13.3 (7.2)	$t(55) = 10.9, p < 0.001$
EDE-Q global score	3.8 (1.5)	2.2 (1.4)	$t(55) = 8.7, p < 0.001$
<i>Subscales</i>			
Eating restraint	3.1 (1.9)	1.3 (1.4)	$t(55) = 7.6, p < 0.001$
Eating concerns ^a	3.3 (1.5)	1.6 (1.3)	$t(54) = 8.7$
Shape concerns ^a	4.6 (1.6)	3.2 (1.8)	$t(54) = 6.2$
Weight concerns	4.1 (1.8)	2.6 (1.8)	$t(55) = 6.5$
BMI	21.7 (7.4)	22.7 (6.1)	$t(55) = 3.9, p < 0.001$
BDI-II	32.6 (13.4)	15.4 (15.0)	$t(55) = 9.6, p < 0.001$

Notes. BDI-II, Beck Depression Inventory – 2; BMI, Body Mass Index; EDE-Q, Eating Disorders Examination Questionnaire; Y-BOCS-SR, Yale-Brown Obsessive-Compulsive Scale—Self-Report.

^a n = 55 due to one patient with anorexia nervosa who did not complete these subscales.

Table 3

The impact of clinical diagnosis at admission on treatment outcome

Outcome measure	Anorexia nervosa <i>n</i> = 23		Bulimia nervosa <i>n</i> = 14		Eating disorder-NOS <i>n</i> = 19		ANOVA results on change scores
	Admission mean (SD)	Discharge mean (SD)	Admission mean (SD)	Discharge mean (SD)	Admission mean (SD)	Discharge mean (SD)	
Y-BOCS-SR ^a	24.0 (6.3)	15.3 (6.6)	22.9 (8.2)	7.6 (5.6)	26.6 (7.4)	14.9 (7.1)	<i>F</i> (2,53) = 3.4, <i>p</i> < 0.05 ^b
EDE-Q	3.8 (1.6)	2.6 (1.3)	4.0 (1.3)	1.6 (1.3)	3.6 (1.4)	2.1 (1.5)	<i>F</i> (2,53) = 3.4, <i>p</i> < 0.05 ^c
BMI	18.0 (2.3)	19.5 (2.0)	24.0 (6.1)	23.8 (5.5)	24.8 (10.0)	25.6 (8.1)	<i>F</i> (2,53) = 3.9, <i>p</i> < 0.05 ^d
BDI-II	31.7 (13.8)	20.8 (16.2)	30.1 (10.5)	5.8 (5.1)	35.6 (15.0)	16.0 (15.4)	<i>F</i> (2,53) = 5.6, <i>p</i> < 0.01 ^e

Notes: BDI-II, Beck Depression Inventory—2; BMI, body mass index; EDE-Q, Eating Disorders Examination Questionnaire; Y-BOCS-SR = Yale-Brown Obsessive-Compulsive Scale—Self-Report.

^a Proportion of patients with a Y-BOCS-SR change of at least 25% at discharge: anorexia nervosa, 16 of 23 (70%); bulimia nervosa, 14 of 14 (100%); eating disorder-NOS, 15 of 19 (79%).

^b Post-hoc pairwise comparisons for change in Y-BOCS-SR: anorexia nervosa < bulimia nervosa (*p* = 0.012). Other pairwise comparisons did not reach significance.

^c Post-hoc pairwise comparisons for change in EDE-Q: anorexia nervosa < bulimia nervosa (*p* = 0.013). Other pairwise comparisons did not reach significance.

^d Post-hoc pairwise comparisons for change in BMI: anorexia nervosa > bulimia nervosa (*p* = 0.008). Other pairwise comparisons did not reach significance.

^e Post-hoc pairwise comparisons for change in BDI-II: anorexia nervosa < bulimia nervosa (*p* = 0.002) and anorexia nervosa < eating disorder-NOS (*p* = 0.029). Other pairwise comparisons did not reach significance.

Table 4

The impact of BMI at admission on treatment outcome

Outcome measure	BMI < 18.5 kg/m ² <i>n</i> = 23		BMI > 18.5 kg/m ² <i>n</i> = 33		ANOVA results on change scores
	Admission mean (SD)	Discharge mean (SD)	Admission mean (SD)	Discharge mean (SD)	
Y-BOCS-SR	23.6 (6.5)	14.1 (6.5)	25.3 (7.7)	12.7 (7.7)	$F(1,54) = 2.2$, ns
EDE-Q	3.9 (1.4)	2.6 (1.4)	3.8 (1.5)	1.9 (1.4)	$F(1,54) = 2.1$, ns
BMI	16.8 (1.3)	19.0 (1.2)	25.2 (7.9)	25.2 (6.9)	$F(1,54) = 35.6$, $p < 0.001$
BDI-II	32.0 (12.0)	17.4 (15.1)	33.1 (14.5)	14.0 (14.9)	$F(1,54) = 1.5$, ns

Notes. BDI-II, Beck Depression Inventory—2; BMI, body mass index; EDE-Q, Eating Disorders Examination Questionnaire; Y-BOCS-SR, Yale-Brown Obsessive-Compulsive Scale—Self-Report.

Table 5

Psychiatric medications at admission and discharge, and change in psychiatric medications in 56 patients

Measure	Number of subjects (%)		
<i>a. Psychiatric medications on admission^a</i>			
None	6 (11%)		
Antidepressants (TCAs, SSRIs, SNRIs, bupropion)	40 (71%)		
Benzodiazepines	22 (39%)		
Antipsychotics	23 (41%)		
Mood stabilizers and anticonvulsants (e.g., lithium, valproic acid, gabapentin)	20 (36%)		
Other (e.g., adderall, campal, methadone, trazodone)	17 (30%)		
<i>b. Psychiatric medications on discharge^a</i>			
None	3 (5%)		
Antidepressants	43 (77%)		
Benzodiazepines	26 (46%)		
Antipsychotics	35 (62%)		
Mood stabilizers and anticonvulsants	23 (41%)		
Other	26 (46%)		
<i>c. Change in psychiatric medications</i>			
	No change ^b	Added	Removed
Antidepressants	51 (91%)	4 (7%)	1 (2%)
Benzodiazepines	42 (75%)	9 (16%)	5 (9%)
Antipsychotics	40 (71%)	14 (25%)	2 (4%)
Mood stabilizers and anticonvulsants	49 (88%)	5 (9%)	2 (4%)

Notes. SNRIs, serotonin and norepinephrine reuptake inhibitors (e.g., venlafaxine, duloxetine); SSRIs, selective serotonin reuptake inhibitors (e.g., fluoxetine, fluvoxamine, paroxetine, sertraline, citalopram); TCAs, tricyclic antidepressants (e.g., clomipramine).

^aThe sum of the percentages exceeds 100% because polypharmacy was common.

^bThe No Change category includes both those who were not taking the indicated class of medication at admission nor taking it at discharge, and those who were taking the indicated class medication at admission and continued to take it at discharge.

Table 6

Patient perspectives

Ms A

“Ms A.” was a 21-year-old college student seeking treatment for AN and OCD. Ms A. arrived for treatment weighing 111 pounds with a height of 5 feet 6 inches (BMI = 17.9). Upon admission, Ms A. had an EDE-Q score of 4.2, a Y-BOCS-SR score of 28, and a BDI-II score of 29. She reported restricted intake in both overall calories and types of foods eaten. Ms A. endorsed overexercising, continuously adding and being preoccupied with the number of calories consumed throughout every day, weighing herself multiple times per day, spending 90 minutes per day examining her appearance in mirrors to make sure her stomach appears flat, and comparing her body to others in response to anxiety regarding eating and her appearance. Ms A. also endorsed OCD symptoms including concern with order with excessive time spent perfectly making her bed, folding clothes, and arranging items and obsessions regarding perfection with school work including rereading material, and rewriting notes or papers. She spent significant amounts of time checking her work for mistakes and frequently sought reassurance from others.

Upon admission, Ms A. met with her cognitive-behavioral therapist to create an exposure hierarchy for both OCD symptoms and her eating disorder. Ms A. was expected to complete numerous hours of exposure work per day and to track any OCD or eating-related rituals she performed. Her exposure work was expected to include a combination of OCD-specific exposures and eating disorder-specific exposures. At the beginning of treatment, she started with mid-level exposures including eating wheat bread or string cheese, writing a sentence with a spelling error, leaving her pillow crooked on her bed, and reading one sentence without rereading it. Later in treatment she moved up her hierarchy to do exposures such as being served and eating a dessert after dinner that she did not choose herself, going to a fast food restaurant for lunch and ordering and eating a high calorie cheeseburger, reading a chapter from a text book and writing a summary paper without rereading or rewriting, completely disorganizing her closet (i.e., shoes in a pile on the floor, hangers facing multiple directions, clothing not organized by type, or color) and leaving it this way for weeks, and leaving dirty clothes on the bedroom floor for days. Her therapist helped her to challenge fear-related thoughts (e.g., “If I don’t reread this information, I may fail at school”), and thoughts regarding her body image (e.g., “unless I am thin, no one will like me”).

After 10 weeks of treatment, Ms A. gained 19 lbs to achieve weight restoration, and also experienced a significant reduction in time spent engaging in rituals. Ms A. was able to eat a wider variety of foods. By discharge, she had completed 70% of her exposure hierarchy and was able to successfully resist engaging in her ordering rituals and checking behaviors related to her AN and to her OCD symptoms the majority of the time. She continued to experience some anxiety around eating dessert items. She also reported significantly improved mood. At the time of discharge, she had BMI of 21.0, an EDE-Q score of 2.7, a Y-BOCS-SR score of 15, and a BDI-II score of 12.

Ms B.

“Ms B.” was a 20-year-old college student seeking treatment for BN and OCD. She reported a history of restrictive eating followed by binge eating and purging that began during her senior year of high school. On days where she was restricting, Ms B. reported that she closely checked food labels for caloric and fat content and added up her caloric intake as she ate (i.e., “counted calories”). Several days of restricting were typically followed by several days of binge eating and purging, during which she typically had several instances per day of eating large quantities (approximately 1000 calories) in one sitting, accompanied by a sense of loss of control. After each binge episode, she induced vomiting. Her symptoms had been worsening: her restricting days included fewer calories and her binge eating days included greater quantities of food and more episodes of purging. Upon admission, she weighed 140 pounds and stood at 5 feet 7 inches (BMI = 21.9 kg/m²). Ms B. denied overexercising or use of laxatives, diuretics, enemas, or appetite suppressants. She weighed herself several times per day and checked her appearance in the mirror multiple times per day to evaluate what she viewed as trouble areas, wore excessively loose clothing, and often covered her stomach with her arms or other objects such as a blanket or pillow. In addition to her eating disorder symptoms, Ms B. also endorsed intrusive thoughts about harming others and frequent checking and reassurance seeking to make sure she had not caused someone harm. She reported religious obsessions and prayed hundreds of times per day to attempt to “cancel out” sinful or blasphemous thoughts or actions. She sought reassurance multiple times per day from her pastor and others regarding her religious obsessions and confessed on a regular basis for what she considered to be sinful thoughts. Her reassurance seeking and confessing often involved her mother, creating considerable stress in their relationship. Ms B. also endorsed depressive symptoms (e.g., depressed mood, loss of interest in previously enjoyed activities, feelings of guilt, feelings of hopelessness, and fatigue) that started approximately six months ago. Upon admission, Ms B. had an EDE-Q score of 3.9, a Y-BOCS-SR score of 27, and a BDI-II score of 23.

Ms B. and her cognitive-behavioral therapist developed an exposure hierarchy. She was assigned to track the number of times per day she submitted to or resisted engaging in rituals or maladaptive behaviors, including calorie counting, weighing herself, checking food labels, checking that she has not caused harm, praying, reassurance seeking, and confessing. Exposures assigned early in her treatment included sleeping with scissors next to her bed, driving over a speed bump without checking to make sure she did not run anyone over, eating chicken breasts, sitting without covering her stomach, and purposefully dropping a Bible on the floor without praying, seeking reassurance, or confessing. Later in treatment, Ms B. worked on exposures such as holding a butcher knife to her therapist’s back, writing a swear word in a Bible in pen, eating a single serving of potato chips without purging, wearing appropriately sized clothing, trying on a swimsuit, eating red meat, and purposefully thinking a “bad” thought in church. Ms B. was also taught to challenge her fear-related thoughts, depressive thoughts, and maladaptive thoughts about her body image and eating. At discharge, Ms B. had an EDE-Q score of 1.5, a Y-BOCS-SR score of 16, and a BDI-II score of 10.