The effects of depression on the treatment of OCD in a residential sample

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Studies investigating the impact of depressive symptoms on obsessive-compulsive disorder (OCD) treatment have yielded mixed findings. The purpose of the study is to extend previous research, which primarily used outpatient samples, to determine whether depression affects OCD treatment outcome among patients receiving intensive residential treatment. OCD patients receiving residential treatment based primarily on exposure and response prevention (ERP) provided data regarding symptoms of depression and OCD at admission and discharge. Patients reported large and significant reductions in OCD symptoms over the course of treatment. Change in OCD symptoms was not significantly affected by depressive symptoms, including patients with severe depressive symptoms. Change in depressive symptoms over the course of treatment was, however, robustly related to change in OCD symptoms, especially among patients who began treatment with severe symptoms of depression. These findings suggest that cognitive-behavior therapy delivered in a residential treatment setting drastically reduces OCD symptoms regardless of depressive symptoms. (Bulletin of the Menninger Clinic 84[Special Issue], 12–33)

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Obsessive-compulsive disorder (OCD) is characterized by unwanted, recurrent thoughts, images, or impulses (i.e., obsessions) and/or repetitive behaviors or mental acts performed to reduce distress (i.e., compulsions) that lead to significant impairment and/or distress (American Psychiatric Association, 2013). Although treatments such as exposure and response prevention (ERP) have demonstrated success in reducing OCD symptoms (Franklin & Foa, 2011), many patients refuse ERP, terminate therapy prematurely, or fail to respond adequately (Franklin & Foa, 2008). One possible hindrance is the presence of comorbid disorders, such as major depressive disorder (MDD). Approximately one fourth of outpatient OCD samples have a comorbid MDD diagnosis (Antony, Downie, & Swinson, 1998; Yaryura-Tobias & Neziroglu, 1996), with lifetime prevalence rates of MDD reaching 50% or more (Crino & Andrews, 1996; Nestadt et al., 2001). Elevated levels of depression have been reported in both outpatient (Whittal, Thordarson, & McLean, 2005) and inpatient (Björgvinsson et al., 2013) OCD samples.

Depression may interfere with OCD treatment in several ways. Emotional arousal has been proposed as a key component of extinction learning (Craske et al., 2008) and thus exposure-based treatments (cf. Craske & Mystkowski, 2006). Depressive symptoms may blunt emotional arousal and therefore hinder extinction learning within the context of ERP (Craske, 2015; Zbozinek & Craske, 2017). In addition, Foa and colleagues (1983) speculated that depression may interfere with the habituation process by increasing emotional reactivity during exposure, thereby lengthening the amount of time needed for habituation and, potentially, effective treatment gains. This effect has been hypothesized to inhibit both within-session habituation and motivation to engage in exposures outside of session, which impedes the effectiveness of two of the main treatment components in ERP (Abramowitz, 2013).

A competing model of change underlying ERP is the inhibitory learning model. Unlike habituation-based models, the inhibitory learning model asserts that exposure allows for the development of safety-based associations that inhibit the previously learned

fear-based associations (Jacoby & Abramowitz, 2016). It should be noted that despite the promising evidence of the inhibitory learning model for ERP in animal samples and a general shift in the field toward acceptance of this model (Craske, 2015), the evidence base in human subjects is still preliminary (Abramowitz, Blakey, Reuman, & Buchholz, 2018). Importantly, parallel to the impact of depression on extinction learning, deficits in affect labeling and emotional arousal appear to hinder inhibitory learning (Craske, 2015). In addition, there is evidence that depression-related emotional reactivity can lead to experiential avoidance (by way of focus on distress elimination as opposed to distress tolerance), which can impede inhibitory learning (Abramowitz, 2013). As such, depression appears to be able to interfere with effective OCD treatment across theoretical models of change.

A final potential explanation for the effect of depression on OCD treatment outcomes is the effect of depression on treatment engagement. Studies have shown significant correlations between depression and treatment attendance (Fals-Stewart & Schafer, 1993; Oldfield, Salkovskis, & Taylor, 2011) and treatment compliance (Abramowitz, Franklin, Street, Kozak, & Foa, 2000; Dowling et al., 2016). These findings are consistent with the results from studies that have shown that elevated levels of depression are correlated with lower motivation to engage in treatment (e.g., M. A. Cahill, Adinoff, Hosig, Muller, & Pulliam, 2003). Research has consistently shown that partial or poor engagement in ERP for OCD attenuates treatment outcomes (Brennan et al., 2014; Veale et al., 2016), which may be the mechanism through which depression exerts its effect on OCD treatment outcomes.

Studies examining depression's impact on OCD treatment have yielded mixed findings. A comorbid diagnosis of MDD has been associated with attenuated treatment outcomes following individual (Abramowitz & Foa, 2000; Motivala et al., 2018; Steketee, Chambless, & Tran, 2001), group (Raffin, Guimarães Fachel, Ferrão, Pasquoto de Souza, & Cordioli, 2009), and multimodal (Brennan et al., 2014) behavior therapy for OCD. Depression also interferes with response to medication and combined treatment approaches (Cottraux, Messy, Marks, Mollard, & Bouvard, 1993). Conversely, some studies have

shown nonsignificant relationships between depression and treatment outcome (Basoglu, Lax, Kasvikis, & Marks, 1988; Foa, Kozak, Steketee, & McCarthy, 1992; Hoogduin & Duivenvoorden, 1988; Mawson, Marks, & Ramm, 1982; O'Sullivan, Noshirvani, Marks, Monteiro, & Lelliot, 1991; Steketee, 1993; Storch et al., 2010). Finally, several studies have reported that exposure-based approaches are able to concurrently reduce depression and OCD symptom severity (Björgvinsson et al., 2008; Hofmann & Smits, 2008).

One explanation for these findings is that only *severe* depressive symptoms adversely affect OCD treatment. This was suggested by Foa (1979) and later examined by Abramowitz et al. (2000). A large outpatient OCD sample was separated into groups based on severity of depressive symptoms per scores on the Beck Depression Inventory (BDI; Beck, Ward, Mendelsohn, Mock, & Erlaugh, 1961). Results indicated that the severely depressed group demonstrated fewer treatment gains from ERP relative to the mildly and moderately depressed groups (Abramowitz et al., 2000). Although Abramowitz et al. demonstrated that outcome may be dependent upon depression severity, this has not been examined within samples with very severe OCD symptoms that require residential or inpatient treatment. Few studies have investigated the role of depression in these settings (cf. Jónsson, Kristensen, & Arendt, 2015; Veale et al., 2016). Stewart, Stack, Farrell, Pauls, and Jenike (2005) reported that admission depression scores were significantly positively correlated with length of stay, and that treatment responders have significantly lower levels of depression at pretreatment than nonresponders; moreover, a diagnosis of MDD has been shown to correlate with OCD severity upon treatment termination (Stewart, Yen, Stack, & Jenike, 2006). Importantly, however, regression analysis failed to demonstrate that pretreatment level of depression significantly predicted severity of OCD symptoms at treatment termination.

Given the mixed findings and the paucity of research in residential/inpatient settings, additional investigation is warranted. The current study aimed to explore the effects of depression on response to CBT for OCD. We sought to replicate and extend the findings of Abramowitz and colleagues (2000) in a severe sample in residential OCD treatment. Given the findings

reported in previous research, we anticipated that patients entering residential treatment with severe levels of depression would evince attenuated OCD symptom reductions relative to their less severely depressed counterparts. Residential/inpatient settings may provide more structure along with an intensive treatment protocol for OCD sufferers (Osgood-Hynes, Riemann, & Björgvinsson, 2003). This structured, intensive approach, where staff are trained to engage patients with treatment procedures throughout the day, may function similarly to behavioral activation and therefore mitigate the interfering effects of severe depression that have been previously reported. It is therefore possible that the effects reported by Abramowitz and colleagues (2000) will not be replicated among patients who received residential treatment.

Method

Participants

Participants included 150 adults (men: n = 80, 53.3%; women: n = 70, 46.7%) admitted to an OCD residential treatment program over a 7-year period. Average length of stay was 63.70 days (SD = 40.06, range = 5–231). Average age was 33.63 years (SD = 12.64). The majority of participants were non-Hispanic White (n = 149, 99.3%); one participant was Asian. The majority (n = 86, 57.3%) had never been married, 45 (30.0%) were currently married, 13 (8.7%) were divorced, and 6 (4.0%) did not provide marital status. Most had received a college education or more: 3 (2.0%) had completed some high school, 25 (16.7%) had graduated from high school, 58 (38.7%) had completed some college, 43 (28.7%) had a bachelor's degree, 12 (8.0%) had a master's degree, and 2 (1.3%) had a doctoral degree.

Prior to admission, patients completed a 90-minute telephone assessment that included the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, & Mazure, 1989), which was reviewed by a licensed psychologist experienced in the diagnosis of OCD and anxiety disorders. Upon admission, patients met with a psychiatrist experienced in the diagnosis and treatment of OCD, who made the official diagnoses. Participants in the current study had a primary *DSM-IV-R* diagnosis of OCD (American Psychiatric Association, 2000), confirmed by both a psychiatrist

and a psychologist (i.e., the senior author, BR). In addition, 50 participants (33.3%) were diagnosed with MDD. Ninety participants (60.0%) had two diagnoses, 29 (19.3%) had three diagnoses, and 4 (2.7%) had four diagnoses. (See Appendix.)

Participants completed measures at admission and discharge. Only participants with an admission Y-BOCS-Self Report (Y-BOCS-SR; Baer, Brown-Beasley, Sorce, & Henriques, 1993) score of 16 or greater were included, because this score suggests clinically relevant severity and has been utilized in numerous treatment efficacy studies (e.g., Foa et al., 2005; Simpson et al., 2008). Only participants who completed the measures of interest (i.e., Y-BOCS-SR and BDI-II; Beck, Steer, & Brown, 1996) at admission and discharge were included in the sample. Participants were not excluded based on previous treatment history, medication usage, or comorbid diagnoses.

Most participants presented with complex treatment histories. Nearly all (n = 134, 89.3%; missing n = 13, 8.7%) had completed at least one psychiatric medication trial prior to admission, and most (n = 78, 52.0%; missing n = 53, 35.3%) had previously attempted outpatient psychotherapy. About half (n = 74; 49.3%; missing n = 17, 11.3%) had previously received inpatient treatment, 15 (10.0%; missing n = 46, 30.7%) had previously attended an intensive outpatient program, 12 participants (8.0%; missing n = 48, 32.0%) had previously attended a partial hospital program, and 28 participants (19.8%; missing n = 26, 17.3%) had previously received residential treatment. Of those 28 participants who had previously received residential treatment, 22 had received residential treatment for OCD.

Treatment

All participants received treatment in a residential OCD treatment program. Although pharmacotherapy was a common component of treatment, the primary emphasis was on ERP and cognitive restructuring. Weekday programming included a homework review group (30 minutes), therapist-aided and self-directed exposure (2.5 hours), therapist-aided cognitive restructuring (1 hour), recreational therapy (1 hour), and self-directed exposure and cognitive restructuring (90 minutes). Required weekend programming per day included a homework review group (30 minutes), self-directed exposures (2 hours), and

therapist-aided group cognitive restructuring (1 hour). Participants were instructed to resist ritualizing during exposure sessions or other times when they were experiencing low/moderate anxiety levels and encouraged to attempt to resist ritualizing when not completing exposure sessions and at higher levels of anxiety. In addition, participants engaged in therapist-aided cognitive restructuring that focused primarily on negative automatic thoughts as per Beck, Rush, Shaw, and Emery (1979). Staff were present 24 hours per day to support treatment goals. Furthermore, participants met regularly with a therapist for additional non-CBT work and weekly family sessions to ensure that family members understood the treatment process and plan for transition back home following discharge. For a detailed description of the treatment, see Osgood-Hynes and colleagues (2003).

Participants met with a psychiatrist approximately twice per week for medication management. Most participants (n = 131, 87.3%) were taking psychotropic medication upon admission, with 96 (64.0%) on at least two medications, 43 (28.7%) on three or more, 13 (8.7%) on four or more, and 3 (2.0%) taking five or more. Antidepressants were the most frequent medication (n = 122, 81.3%), followed by antipsychotics (n = 66, 44.0%), anti-anxiety (n = 59, 39.3%), mood stabilizers (n = 23, 15.3%), and stimulants or other medications for ADHD (n = 13, 8.7%), with one participant (0.7%) each taking an opioid or a sleep medication. Admission medication information was not available for eight participants (5.3%).

Instruments

Beck Depression Inventory-II (BDI-II). The BDI-II (Beck et al., 1996) is a 21-item self-report measure assessing depression severity. The BDI-II has shown excellent test–retest reliability, r = .93, and is strongly correlated with the original BDI (r = .93; Beck et al., 1996). A score of 0–13 is considered minimal depression; 14–19, mild depression; 20–28, moderate depression; and 29–63, severe depression.

Yale-Brown Obsessive-Compulsive Scale–Self-Report (YBOCS-SR). The self-report Y-BOCS (Baer et al., 1993) rates obsessions and compulsions, each on five items on a scale ranging from 0 to

4. Scores of 0–7 are considered sub-clinical; 8–15, mild; 16–23, moderate; 24–31, severe; and 32–40, extreme. Baer et al. (1993) found that the self-report version correlated very highly with the interview version (r = .97). Steketee, Frost, and Bogart (1996) reported that the Y-BOCS-SR displayed good internal consistency (α = .78) and good test–retest reliability over a 1-week interval (r = .79).

Analytic strategy

The effects of baseline depression on change in OCD symptom severity were examined with three mixed within-between analyses of variance (ANOVAs). The first ANOVA—with Y-BOCS-SR as the dependent variable (DV), time (admission and discharge) as the within-subjects factor, and admission BDI-II groups [minimally (n = 12), mildly (n = 19), moderately (n = 35), and severely depressed (n = 84)] as the between-subjects factor—was used to examine the differences in change in Y-BOCS-SR scores across the four depression groups. The second ANOVA—with Y-BOCS-SR as the DV, time as the within-subjects factor, and admission BDI-II scores (modeled as a continuous variable) as a covariate—was used to examine the relations between severity of depressive symptoms at admission and change in Y-BOCS-SR scores. The third ANOVA—with Y-BOCS-SR as the DV, time as the within-subjects factor, and comorbid MDD diagnosis (positive, negative) as a between-subjects factor—was used to examine differences in change in Y-BOCS-SR scores between participants with and without comorbid MDD.

We also examined the impact of change in depression (admission minus discharge BDI-II scores) on change in OCD severity with multiple regression analyses. Discharge Y-BOCS-SR was regressed onto admission Y-BOCS-SR and BDI-II change scores in the first regression. A second exploratory regression analysis was completed in the same manner among those with severe depression scores.

Lastly, we examined clinically significant change for both the Y-BOCS-SR and the BDI-II across each depressive group using the reliable change index (RCI; Jacobson & Truax, 1991) and end of treatment functioning (i.e., discharge scores). RCI was determined using published normative data from nonclinical samples employed in previous studies on residential treatment

for OCD (cf. Björgvinsson et al., 2013), and confidence intervals were calculated to delineate a threshold of reliable change with a cutoff score of two standard deviations (RCI = 1.96). Those who achieved reliable change and posttreatment scores in the nonclinical to mild range on the same measure (Y-BOCS-SR < 16; BDI < 10) demonstrated clinically significant change.

Results

Data were examined for violations of the assumption of normality with the Shapiro-Wilk test. This was not significant ($p > .05$) for any of the BDI-II groups at either admission or discharge Y-BOCS-SR, indicating no violations of normality. Means, range, and standard deviations for each depression group are presented in Table 1. There were no significant differences in length of stay, age, gender, marital status, or level of education between the four levels of depression, $p > .05$ for all variables.

Examination of the effect of admission depression on admission and discharge OCD severity

The first mixed ANOVA, which examined differences in Y-BOCS-SR change over time across the four depressive groups, revealed a significant main effect of time, $F(1, 146) = 273.09$, $p < .001$, $\eta_p^2 = .65$, indicating very large and significant reduction in Y-BOCS-SR from admission to discharge. There was a significant main effect of depressive group, $F(3, 146) = 5.30$, $p < .01$, $\eta_p^2 = .10$, indicating that the average level of Y-BOCS-SR severity across admission and discharge varied as a function of depressive group. The Time × Depressive Group interaction was

Table 1. **Age and days in treatment by levels of depression**

		Age		Treatment Duration	
Level of Depression	***n***	***M***	***SD***	***M***	***SD***
Minimally Depressed	12	27.00	8.19	60.08	33.59
Mildly Depressed	19	36.74	14.33	55.74	22.70
Moderately Depressed	35	33.74	12.51	64.60	46.13
Severely Depressed	84	33.83	12.68	65.64	41.57

not significant, $F(3, 146) = 2.02$, $p = .11$, $\eta_p^2 = .04$. This indicates that the differences in Y-BOCS-SR change over the course of treatment did not significantly vary across the four depressive groups. Post hoc *t* tests were consistent with this finding. Patients in all depressive groups showed large, significant, and comparable reductions in Y-BOCS-SR from admission to discharge (Table 2).

The second mixed ANOVA, which tested the effects of depressive severity (modeled as a continuous covariate) at admission on change in Y-BOCS-SR severity scores over time, revealed a nonsignificant Time × Depressive Severity interaction, $F(1, 148) = 0.41$, $p = .53$, $\eta_p^2 = .00$. Like the nonsignificant Time × Depressive Group interaction from the first ANOVA, this finding indicates that reductions in OCD symptoms over the course of treatment did not vary as a function of pretreatment depressive severity.

The third mixed ANOVA, which examined if change in Y-BOCS-SR scores over the course of treatment varied as a function of comorbid MDD diagnosis, revealed a Time × MDD Diagnosis interaction that was trending toward statistical significance, but was not, $F(1, 142) = 2.79$, $p = .10$, $\eta_p^2 = .02$. This finding suggests that differences in Y-BOCS-SR change were similar between patients with and without a comorbid MDD diagnosis. Inspection of Y-BOCS-SR change scores revealed that patients

Table 2. **Admission and discharge Y-BOCS-SR by level of depression**

		Y-BOCS-SR					
		Admission		Discharge		Significance Test of Change	
Level of Depression	*n*	*M*	*SD*	*M*	*SD*	*t, p*	Cohen's *d*
Minimally Depressed	12	27.83	5.84	12.67	4.94	6.868, < .001	2.80
Mildly Depressed	19	24.58	4.72	15.32	5.06	8.505, < .001	1.89
Moderately Depressed	35	27.26	4.74	16.11	5.45	11.104, < .001	2.18
Severely Depressed	84	29.98	5.52	17.79	7.28	14.903, < .001	1.89

Note. Y-BOCS-SR = Yale-Brown Obsessive-Compulsive Scale–Self Report.

with a comorbid MDD diagnosis showed slightly smaller reductions in Y-BOCS-SR over the course of treatment (M = 10.38, SD = 7.28) than their counterparts without a comorbid MDD diagnosis (M = 12.41, SD = 6.79), although this difference was not significant. Importantly, a one-way ANOVA showed that the two groups did not have significantly different discharge Y-BOCS-SR scores, $F(1, 142) = 2.29$, $p = .13$.

Examination of the effect of change in depression scores on change in OCD severity

A multivariate regression analysis tested if change in BDI-II scores (admission minus discharge) predicted discharge Y-BOCS-SR scores among all patients. The overall regression model explained 31.3% of the variance in discharge Y-BOCS-SR, $F(2, 147) = 33.55$, $p < .001$. Admission Y-BOCS-SR and BDI-II change accounted for 18.9% and 18.7% of the variance in discharge Y-BOCS-SR, respectively. Lower BDI-II change was significantly associated with higher discharge Y-BOCS-SR, $\beta = -.44$, $t = -6.33$, $p < .001$.

Previous research has indicated that only severe levels of depression may hinder OCD treatment (Abramowitz et al., 2000). As such, we utilized an additional regression to test if change in BDI-II from admission to discharge predicted discharge Y-BOCS-SR for participants in the severely depressed group. The overall regression model explained 50% of the variance in discharge Y-BOCS-SR, $F(2, 83) = 39.90$, $p < .001$. Admission Y-BOCS-SR and BDI-II change accounted for 12.9% and 38.2% of the variance in discharge Y-BOCS-SR, respectively. Lower BDI-II change was significantly associated with higher discharge Y-BOCS-SR, $\beta = -.62$, $t = -7.83$, $p < .001$.[1]

Reliable change and clinically significant change

Percentages for reliable change and clinically significant change are reported in Table 3. Comparisons across the largest three groups (minimal, moderate, and severe depression) display a

1. All analyses using change scores were also examined by computing residual gain scores to correct for problems in using raw scores (see Steketee & Chambless, 1992). In all instances, there were no differences in the pattern of results; therefore, change scores were used in the body of the article for ease of interpretation.

Table 3. **Percentage of reliable change and clinically significant change on the Y-BOCS-SR and BDI by level of depression**

		Reliable Change		Clinically Significant Change	
Level of Depression	***n***	**Y-BOCS-SR**	**BDI-II**	**Y-BOCS-SR**	**BDI-II**
Minimally Depressed	12	91.7%	50.0%	75.0%	[a]
Mildly Depressed	19	78.9%	68.4%	47.4%	84.2%
Moderately Depressed	35	82.9%	77.1%	45.7%	60.0%
Severely Depressed	84	79.8%	86.9%	39.3%	48.8%
Total Sample	150	79.7%	77.7%	43.8%	56.5%[b]

Note. Y-BOCS-SR = Yale-Brown Obsessive-Compulsive Scale–Self Report. BDI-II = Beck Depression Inventory. [a]All participants started in the minimally depression range of BDI < 13 and therefore could not meet criteria for clinically significant change. [b]This percentage represents those eligible for clinically significant change (n = 141). Those in the minimally depressed group could not achieve clinically significant change.

pattern of similar levels of reliable change and clinically significant change by groups for OCD severity. However, higher rates of clinically significant change were achieved by those in the depression groups with lower initial levels.

Discussion

This study investigated the effects of depression on OCD treatment outcome in a residential OCD treatment center. We probed these effects at multiple levels by measuring relations between (a) depressive grouping at admission and change in OCD symptoms (as in Abramowitz et al., 2000); (b) continuous depressive severity at admission and change in OCD symptoms; (c) comorbid MDD diagnosis and change in OCD symptoms; and (d) change in depressive symptoms and change in OCD symptoms. Our results indicated that patients demonstrated significant and comparable lessening of OCD symptoms regardless of depression severity at admission. Although we found that patients with a comorbid diagnosis of MDD responded marginally less to CBT for OCD than their counterparts without comorbid MDD, this effect was small and nonsignificant, and the two groups did not differ in OCD symptom severity at discharge. Inspection of effect sizes showed that those with minimal depression reported the largest OCD symptom reductions,

although patients at all levels of depression demonstrated large reductions in OCD symptoms. Our observed effects sizes are comparable to or greater than effect sizes reported in a meta-analysis of outpatient OCD treatment (d = 1.20–1.75; Eddy, Dutra, Bradley, & Westen, 2004) and previously published inpatient/residential treatment studies (d = 1.10 in Björgvinsson et al., 2013, and d = 1.35 in Boschen, Drummond, & Pillay, 2008). Lastly, regression analyses showed that change in depressive symptoms over the course of treatment was a robust predictor of change in OCD symptoms; as depressive symptoms reduced, so did OCD symptoms, or, alternatively, depressive symptoms were more likely to improve as OCD symptoms decreased. Regardless of causal inference, the relation between improvement in depressive symptoms and OCD symptoms was quite pronounced for patients entering treatment with severe depressive symptoms; change in depressive symptoms predicted 38% variance in discharge OCD symptoms after accounting for admission OCD severity. This suggests that patients who begin OCD treatment with severe depression but do not achieve significant reductions in depressive symptoms are likely to have an attenuated response to CBT for OCD.

Findings from the current study suggest that patients with severe OCD and depression treated in a residential setting make progress on OCD symptom severity that is nearly equivalent to their less depressed counterparts. This differs from previous studies with outpatient samples, which reported that patients with severe depression demonstrated less improvement in OCD symptoms than patients with less severe depression (Abramowitz et al., 2000). There were some notable differences between the current sample and the sample included in Abramowitz and colleagues' (2000) study. Distribution across levels of depression differed between the two studies. The present residential sample reported more severe symptoms of OCD and depression than the sample studied by Abramowitz and colleagues. Most of Abramowitz and colleagues' sample fell in the mild (34.5%) or moderate (34.5%) levels, and most of the residential sample (56%) was severely depressed. Perhaps most importantly, the present sample received intensive residential CBT whereas Abramowitz and colleagues' sample received outpatient treatment. The residential group

had continuous support from a variety of staff and a structured schedule of other possibly therapeutic activities (e.g., group cognitive restructuring, art therapy, experiential therapy, process groups, group outings). Collectively, these differences may have enabled residential patients to devote significantly more hours to treatment each week. This may be why the severely depressed patients fared so well, and thus clinical recommendations for the treatment of OCD with comorbid severe depression may include a higher ERP dose and greater support and structure to maximize treatment engagement and between-session compliance.

Increasing the "dose" and inherent structure addresses two concerns raised by Abramowitz et al. (2000) on the interference of depression in OCD treatment. First, they suggested that depressed individuals may experience less habituation and therefore require more treatment to achieve gains like those who are less depressed. Second, they posited that depression may lead to decreased treatment compliance. ERP is challenging work even for those who are not depressed; thus, having a structured environment and support from a variety of highly trained staff could help with homework completion.

The residential treatment setting and schedule require patients to be active and interact socially, whereas outpatients with OCD and depression may spend much of their time isolating themselves. This is a significant distinction, because inactivity, isolation, and avoidance are theorized to contribute to the development and maintenance of depression (e.g., Martell, Addis, & Jacobson, 2001), and depression treatments focused on increasing activation toward pleasant and meaningful behavior (i.e., behavioral activation; Lejuez, Hopko, & Hopko, 2001; Martell et al., 2001) have considerable empirical support (e.g., Cuijpers, van Straten, & Warmerdam, 2007; Ekers, Richards, & Gilbody, 2008; Mazzucchelli, Kane, & Rees, 2009). Indeed, recent work from our group suggests that ERP within a residential treatment setting increases behavioral activation, which mediated the effects of ERP on depression (Blakey, Abramowitz, Leonard, & Riemann, 2019). Moreover, experts recommend adding activation strategies to traditional OCD treatment for those with comorbid depression (Abramowitz, 2004; Ledley, Pai, & Franklin, 2007).

Of necessity, the residential setting requires a certain level of social interaction between residents, often including a shared bedroom between two residents. Depressed individuals often have social skills deficits (Weissman & Paykel, 1974); accordingly, social skills training has been incorporated into behavior therapy for depression for many years (e.g., Bellack & Hersen, 1979) and included in some behavioral activation treatments (see Kanter et al., 2010, for a review). The social activity inherent in the residential treatment setting provides opportunities to practice social skills, which may decrease depressive symptoms. These opportunities are not directly available in an individual outpatient treatment setting.

Other unique aspects of a residential setting may enhance treatment outcomes. Although most work is conducted individually or with a behavior therapist, patients meet daily as a group with staff members to discuss progress, difficulties, and plans for success. Exposures are often conducted in common areas of living; patients view firsthand the struggles and, more importantly, the eventual successes of other patients, and thus may learn from the experience of those nearing completion of treatment. This can add credibility to the treatment process and foster support for each other in reaching treatment goals. It is common for patients in this setting to point out to another patient when he or she is engaging in ritualistic behaviors, to encourage response prevention, or to support each other to engage in exposure work. Therefore, many principles found to be important in group psychotherapy, such as universality, guidance, and instillation of hope (Yalom & Leszcz, 2005), may assist in the residential treatment settings.

This study has a few notable limitations. It is a naturalistic design of participants who were aware of and considered residential treatment for OCD. Therefore, generalization of the results may be limited by the sample demographics (e.g., income, race, ethnicity). Another limitation is the inability to account for only ERP/CBT to explain gains in treatment. The effects of medication on symptoms were not assessed or controlled; however, most participants had been taking psychotropic medication for long periods of time upon admission, suggesting minimal benefit prior to and while in residential

treatment. Furthermore, other therapeutic interventions may have contributed to treatment outcome (e.g., experiential therapy, behavioral activation, and challenging depressive cognitions). Although all participants received certain core aspects of treatment (i.e., ERP, experiential therapy, psychiatrist meetings), not all received behavioral activation or thought challenging for depressive cognitions. Also, each patient's level of compliance with individual treatment techniques may have varied; future studies should attempt to measure adherence to treatment. Two final limitations are use of self-report measures and the lack of structured diagnostic assessments. Although this research was conducted in a naturalistic study, with an emphasis on dimensional severity of depression rather than solely exploring the effects of depression based on diagnosis, future research could use more stringent assessment ,including clinician-administered measures such as the Y-BOCS, Montgomery Asberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979), and a structured diagnostic assessment.

In conclusion, the findings from this study suggest that a residential treatment approach is effective at reducing both symptoms of OCD and depression, even when depressive symptoms are severe. It is possible that residential treatment and the benefits it conveys may combat exposure-interfering effects of depression. This is an idea that requires further inquiry; future researchers should explore if CBT dose or setting moderates relations between depressive severity and response to CBT for OCD. Although the naturalistic design could be a weakness to internal validity, these results show efficacy in a real-world option for patients with OCD and depression.

Appendix

Table A1. **Number of comorbid diagnoses within the entire sample and across levels of depressive severity**

Comorbid diagnosis	Minimal Depression (*n* = 12)	Mild Depression (*n* = 19)	Moderate Depression (*n* = 35)	Severe Depression (*n* = 84)	Entire Sample (*N* = 150)
None	8	11	12	29	60
Mood Disorder	4	6	15	42	67
Trichotillomania	0	0	0	1	1
BDD	0	0	0	3	3
Panic Disorder	0	0	0	3	3
PTSD	0	0	0	1	1
Social Phobia	0	1	2	4	7
GAD	0	0	4	2	9
BDD	0	0	1	5	3
Eating Disorder	0	1	0	10	11
Substance Abuse/ Dependence	1	2	4	2	9
Other	0	2	3	7	12

Note. Percentages are not provided given that many patients carried more than one comorbid diagnosis. BDD = body dysmorphic disorder; PTSD = posttraumatic stress disorder; GAD = general anxiety disorder.

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