HHS Public Access

Author manuscript

Psychother Res. Author manuscript; available in PMC 2016 December 01.

Published in final edited form as:

Psychother Res. 2016 November ; 26(6): 727–736. doi:10.1080/10503307.2015.1065022.

Residential Treatment Outcomes for Adolescents with Obsessive-Compulsive Disorder

Rachel C. Leonard, Ph.D.[a,1], **Martin E. Franklin, Ph.D.**[b,2], **Chad T. Wetterneck, Ph.D.**[a,3], **Bradley C. Riemann, Ph.D.**[a,4], **H. Blair Simpson, M.D., Ph.D.**[c,d,5], **Kimberly Kinnear, M.A.**[a,6], **Shawn P. Cahill, Ph.D.**[e,7], and **Peter M. Lake, M.D.**[a,8]

[a]Rogers Memorial Hospital; Oconomowoc, WI, 34700 Valley Road, Oconomowoc, WI 53066

[b]Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, PA, USA

[c]Department of Psychiatry, Columbia University, New York, NY, USA

[d]Division of Clinical Therapeutics, New York State Psychiatric Institute, New York, NY, USA

[e]Department of Psychology, University of Wisconsin – Milwaukee, Milwaukee, WI, USA

Abstract

Objective—We examined outcomes from a residential treatment program emphasizing ERP to determine if the typically robust response to this treatment in outpatient settings extends to patients treated in this unique context.

Method—172 adolescents with primary OCD completed measures at admission and discharge. Almost all (92.4%) participants had at least two diagnoses and nearly half (44.2%) had three or more. Treatment consisted of intensive ERP (i.e., approximately 26.5 hours per week), additional cognitive behavioral therapy interventions, and medication management within a residential setting. In contrast to the samples reported on in the vast majority of other pediatric OCD trials, participants in the current study were living apart from their families and were immersed within the treatment setting, with staff members available at all times.

Results—Paired sample *t*-tests revealed significant decreases in OCD and depression severity.

Conclusions—Results suggest that residential treatment for adolescents with OCD using a multimodal approach emphasizing ERP can be effective for complex cases with significant comorbidity. Results were comparable with several randomized controlled trials.

Keywords

OCD; exposure therapy; cognitive behavior therapy; anxiety

[1]Corresponding Author: Rachel C. Leonard, Ph.D.; 34700 Valley Road, Oconomowoc, Wisconsin, 53066; Rogers Memorial Hospital; 262-646-1158; rleonard@rogershospital.org.
[2]marty@mail.med.upenn.edu; phone: 215-746-1230.
[3]cwetterneck@rogershospital.org; phone: 262-646-1759.
[4]briemann@rogershospital.org; phone: 262-646-1388.
[5]simpson@nyspi.columbia.edu; phone: 646-774-7000.
[6]kkinnear@rogershospital.org; phone: 1-800-767-4411.
[7]cahill@uwm.edu; phone: 414-229-5099.
[8]plake@rogershospital.org; phone: 262-646-5268.

Introduction

Obsessive-compulsive disorder (OCD) is characterized by recurrent intrusive thoughts, impulses, or images (i.e., obsessions) and/or repetitive behaviors that lessen anxiety (i.e., compulsions), with resulting impairment and/or distress (American Psychiatric Association, 2013). Left untreated, pediatric OCD is associated with significant impairment in academic, family and social functioning (Lack et al., 2009; Piacentini, Bergman, Keller, & McCracken, 2003), and it increases the risk of later psychiatric and psychosocial morbidity (Bolton, Luckie, & Steinberg, 1995; Hanna, 1995).

Recommended treatments for pediatric OCD include cognitive-behavioral therapy (CBT) with exposure and response prevention (ERP) for mild to moderate cases and CBT with ERP (referred to as CBT from this point onward unless otherwise noted) combined with serotonin reuptake inhibitors (SRIs) for moderate to severe cases (American Academy of Child and Adolescent Psychiatry, 2012; National Institute for Health and Clinical Excellence, 2005). Numerous randomized controlled trials (RCTs) have demonstrated the efficacy of CBT alone (e.g., Barrett, Healy-Farrell, & March, 2004; Pediatric OCD Treatment Study [POTS], 2004) or with SRIs (e.g., Franklin et al., 2011; POTS, 2004).

Storch and colleagues (2007) completed the only RCT to date examining intensive outpatient (i.e., 90-minute sessions each weekday for three weeks) CBT for pediatric OCD. Compared to weekly treatment, the intensive approach fared slightly better in remission and improvement rates at post-treatment; however, there were no significant differences across conditions by three-month follow-up.

Although these results are promising, there are a number of procedural details inherent in RCTs that may limit their external validity. Unlike participants treated in clinical practice settings, RCT participants are typically required to refrain from taking psychotropic medications or maintain a stable dosage prior to and throughout a study (e.g., Barrett et al., 2004; Storch et al., 2007). Some RCTs also exclude participants who have previously had a failed trial of CBT for OCD (e.g., POTS, 2004). In addition, RCTs often follow structured disorder-specific protocols. Therefore, examination of pediatric OCD treatment outcomes in naturalistic settings is warranted to test the limits of this treatment beyond the academic medical context and to examine its effects in patients who would likely be ineligible for RCTs. Specifically, further research is needed to examine outcomes when treatment is provided in a more flexible manner with complex patients, such as those with significant comorbidity and those who have previously failed to respond to treatment. Evidence from naturalistic studies supports the benefits of CBT for pediatric OCD in traditional outpatient settings (Nakatani, Mataix-Cols, Micali, Turner, & Heyman, 2009; Vande Voort, Svecova, Jacobsen, & Whiteside; Warren & Thomas, 2001). Naturalistic studies with adult outpatients have demonstrated the effectiveness of intensive CBT (e.g., Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000).

There is less research to date examining naturalistic outcomes following intensive CBT for pediatric OCD. Open trials examining intensive outpatient treatment have demonstrated significant symptom reduction (Franklin et al., 1998; Storch et al., 2010; Storch et al., 2006).

Other studies examining shorter-term intensive outpatient treatment reported significant improvement from pre- to post-treatment (Whiteside, Brown, & Abramowitz, 2008; Whiteside and Jacobsen, 2010) and from post-treatment to follow-up (Whiteside & Jacobsen, 2010). There is also evidence to support intensive outpatient group treatment for pediatric OCD (Olino et al., 2011).

Patients with complex comorbidity or previous unsuccessful treatment attempts may require more intensive monitoring, multimodal interventions, and greater intensity of treatment delivery than even intensive outpatient CBT provides. Non-specific inpatient settings may help to ensure patient safety but do not provide an ideal venue for treatment of OCD due to the typically brief duration of treatment and lack of specialty providers. In contrast, residential treatment provides a greatly increased number of hours of CBT provided per week (approximately 26.5 in the current study), constant placement in the treatment setting and removal from direct contact with family members accommodating their symptoms, and around-the-clock staff monitoring and support to assist with ritual prevention, homework compliance, and other treatment targets. Residential treatment also provides more time to address comorbid conditions and provides opportunities for interaction with peers with similar symptoms, which often serves as an important source of motivation. Therefore, residential treatment may be beneficial for individuals presenting with significant comorbidity or previous unsuccessful treatment attempts.

There is little evidence to date examining the effectiveness of CBT for adolescents with OCD in a residential setting. Björgvinsson et al. (2008) provided naturalistic treatment outcome data for 23 adolescents who received residential treatment. Children's Yale-Brown Obsessive-Compulsive Scale (CY-BOCS; Scahill et al., 1997) scores significantly decreased from pre- to post-treatment. Further, 70% of their sample demonstrated reliable change (Jacobson & Truax, 1991). Participants experienced significant decreases in state and trait anxiety and cognitive beliefs associated with OCD but did not experience significant decreases in depressive symptoms. Therefore, this study provided initial support for the effectiveness of residential treatment in a small sample of adolescents with OCD. Additional research is needed to investigate residential treatment outcomes with a larger sample, and to examine whether gains are maintained following treatment.

In the current article, we present outcome data from a large sample of adolescents with extensive treatment histories and substantial comorbidity who received residential OCD treatment. Thus, the sample is more complex than those typically examined within RCTs; however, the treatment is more intensive and comprehensive. We will compare the results to several RCTs as benchmarks for evaluating the effectiveness of CBT for pediatric OCD. We also present follow-up data on a subset of the sample. We hypothesize that participants will demonstrate statistically significant and clinically meaningful reductions in OCD and depression symptom severity.

Method

Participants

Two hundred forty-six adolescents with primary OCD were admitted for the first time to the residential treatment program between February 2005 and April 2013. Of those 246, 215 had complete admission CY-BOCS-Self Report (CY-BOCS-SR; Piacentini, Langley, & Roblek, 2007) data, with 205 having both admission and discharge CY-BOCS-SR data. Those who had discharge data did not differ in admission CY-BOCS-SR scores from those who did not have discharge data, $t(213) = 1.304$, *ns*. Of these 205, 172 met the additional criterion of having an admission CY-BOCS-SR score of 16 or greater, indicating clinically significant OCD symptoms. Thus, only participants who completed the CY-BOCS – SR at pre- and post-treatment and had a pre-treatment score of 16 or greater were included. See Figure 1 for detailed information on participant flow. Participants were not excluded based on previous treatment history, medication usage, comorbid diagnoses, treatment duration, treatment response, or discharge circumstances. Exclusion criteria from the treatment program included the presence of a psychotic disorder, current substance dependence, acute suicidality or homicidality (i.e., significant threat or harm to oneself or others such that inpatient treatment would be necessary), or significant cognitive impairment. Average treatment duration was 78 days ($SD = 36$, range = 15 – 213). Data regarding reasons for discharge are not available, although typical reasons include the participant reaching maximum benefit, lack of funding, participant refusal to engage in treatment, or the need for inpatient treatment due to safety concerns. Treatment duration was flexible, with the goal prior to discharge being that participants complete 70% or more of their exposure hierarchy and demonstrate symptom improvement based on examination of self-report measures and/or observation of participant behavior. Participants were on average 15.45 years old ($SD = 1.22$, range = 13 – 17) and the majority of the sample was European American (89.0%, $n = 153$), followed by Hispanic (3.5%, $n = 6$), Asian (2.9%, $n = 5$), multiracial (2.9%, $n = 5$), and African American (0.6%, $n = 1$), with missing data on two participants. The sample included 89 males (51.7%) and 83 females (48.3%).

All participants completed a pre-admission telephone screening that included a CY-BOCS (Scahill et al., 1997). This was reviewed by a board-certified psychiatrist with extensive training in OCD evaluation and intervention. Upon admission, participants were evaluated by child and adolescent psychiatrists who specialize in the treatment of OCD and anxiety disorders. The psychiatrists made the official diagnoses following a diagnostic interview. This sample was characterized by extensive comorbidity (see Table 1).

Participants often presented with complicated treatment histories. All participants had psychiatric medication trials prior to admission. Nearly all (90.1%) had previously attempted outpatient therapy. Further, 39.5% had previously received inpatient treatment, indicating severe symptoms with significant impairment in functioning and/or safety concerns. In addition, 17.4% had previously attended partial hospital or day treatment programs, 11.0% had previously received intensive outpatient treatment, and 11.6% had previously completed residential treatment. Of the 20 participants with previous residential treatment, 11 had received residential treatment for OCD or anxiety.

Of the 172 participants, we attempted to contact participants who were approximately one year post-discharge during the time period in which the hospital was collecting follow-up data (follow-up data collection started in 2009). Eighty-nine participants met these criteria. Of these 89, we successfully contacted 49, and 44 agreed to participate. Unsuccessful attempts to contact individuals were due to inaccurate or changed contact information, placing numerous calls without reaching the individual or being able to leave a message, or leaving messages but not having the individual return the phone call. Participants provided follow-up data on average 1.49 years following discharge (*SD* = 0.57 years, range = .94 – 3.52 years).

Treatment

Treatment primarily consisted of ERP, which followed standard procedures (e.g., March & Mulle, 1998). Therapists and participants developed a hierarchy of situations that triggered their anxiety from least to most feared. Following this, therapists then assisted participants in facing these feared situations (i.e., exposures) in a prolonged, repetitive and graduated manner to promote habituation to the anxiety. Concurrently, engagement in any avoidance behaviors and/or rituals was proscribed (i.e., ritual prevention) in order to remove the connection between engagement in rituals and decreased anxiety. Therapists contacted participants' parents on a weekly basis to update them regarding treatment progress and any additional concerns. Participants met with a behavior therapist five days per week for staff-assisted exposure and completed additional self-directed exposure seven days per week, with the expectation that they complete approximately 26.5 hours of ERP per week. While the amount of staff-assisted and self-directed exposure work varied, it is estimated that approximately 25–50% of completed exposure work was staff-assisted. All participants also completed cognitive restructuring techniques to address their OCD-related fears and were regularly assigned thought challenging exercises in order to examine the evidence for and against their feared outcomes occurring. Additional CBT interventions (e.g., activity scheduling for depressive symptoms, interoceptive exposures for panic disorder symptoms) were often used to address comorbid symptoms. Participants also participated in process groups five days per week, met regularly with their therapist for non-CBT work (e.g., discharge planning, psycho-education with family members), participated in experiential therapy groups several times per week, and they attended school for 90 minutes each weekday.

Participants met with a psychiatrist several times per week for medication management and supportive psychotherapy. The majority of participants (90.7%) were taking at least one psychotropic medication upon admission, with 57 participants (33.1%) taking two, 36 participants (20.9%) taking three, 19 participants (11.0%) taking four, one participant (0.6%) taking five, and one participant (0.6%) taking six psychotropic medications upon admission. This information was not available for one participant. Antidepressants were the most frequent type of medication taken at admission (*n* = 146, 84.9%), followed by antipsychotics (*n* = 79, 45.9%), benzodiazepines and other anxiolytics (*n* = 36, 20.9%), stimulants and other medications for attention deficit hyperactivity disorder (*n* = 27, 15.7%), and mood stabilizers (*n* = 16, 9.3%).

Measures

Beck Depression Inventory-II (BDI-II; Beck et al., 1996)

The BDI-II is a 21-item measure of depression symptom severity for individuals ages 13 and older. Items are rated on a 4-point scale for a total score ranging from 0–63. The BDI-II has excellent test-retest reliability (Beck et al., 1996) and internal consistency (Beck et al., 1996; Steer, Kumar, Ranieri, & Beck; 1998). Pre-treatment and post-treatment BDI-II data were available for a subsample of 126 participants. This is in part due to the hospital changing from using the BDI-II to the Quick Inventory of Depressive Symptomatology – Self Report (QIDS-SR; Rush et al., 2003) near the end of 2012. Of note, data from this subsample were reported in Leonard, Jacobi, Riemann, Lake, and Luhn (2014); however, that paper did not include any follow-up data and the primary focus was on the impact of level of depressive symptoms on outcome.

CY-BOCS-SR (Piacentini et al., 2007)

The CY-BOCS-SR is a 10-item self-report measure of OCD symptom severity. The severity scale contains five items each to assess the severity of obsessions and compulsions on a scale from 0 to 4, for a total score ranging from 0 to 40. Conelea, Schmidt, Leonard, Riemann, & Cahill (2012) found that the CY-BOCS-SR significantly correlated with scores on the interview-based CY-BOCS ($r = .77$), with self-report scores potentially under-reporting symptoms compared to an interview-based assessment.

QIDS-SR (Rush et al., 2003)

The QIDS-SR is a 16-item self-report measure of depression symptom severity. The QIDS-SR was developed to assess all aspects of depression included in the DSM-IV-TR (American Psychiatric Association, 2000) and to capture a wide range of symptom severity. Items are rated from 0 to 3, with scoring based on symptom clusters such that the highest rated item pertaining to a symptom cluster is scored. Scores range from 0 to 27, with higher scores indicating greater depression severity. The hospital switched from the BDI-II to the QIDS-SR near the end of 2012; therefore, a small sample ($n = 12$) completed the QIDS-SR at pre-treatment and post-treatment.

Statistical Methods

Paired-sample *t*-tests were applied to (1) pre-treatment and post-treatment scores for the CY-BOCS-SR and BDI-II to evaluate treatment outcome and (2) to post-treatment and follow-up scores for participants with available follow-up data. We calculated the proportion of patients at discharge who met response criteria defined as a CY-BOCS-SR decrease of 25% (Simpson, Huppert, Petkova, Foa, & Liebowitz, 2006; Storch, Lewin, De Nadai, & Murphy, 2010). We also examined the proportion of participants who demonstrated excellent response with mild-to-minimal symptoms defined by a discharge CY-BOCS-SR of 10 or less. This criterion was chosen to allow comparison with POTS (2004).

Jacobson and Truax's (1991) methods were used to assess reliable change (the change necessary to be statistically reliable) and clinically significant change (the extent to which treatment leads to improvement such that an individual falls outside the range of the

dysfunctional population or within the range of the functional population; Jacobson & Truax, 1991). Data from non-clinical samples are required to calculate clinically significant change; however, non-clinical data using the CY-BOCS were unavailable. Therefore, in accordance with Abramowitz, Whiteside, and Deacon (2005), non-clinical Y-BOCS data from adults reported in Steketee, Frost, and Bogart (1996) were used to estimate the normative sample ($M = 7.2$, $SD = 4.5$, test-retest reliability = 0.88). To calculate the reliable change index (RCI), each participant's CY-BOCS-SR change scores were divided by the standard error of the difference. This score is considered reliable if it is greater than 1.96 (Jacobson & Truax, 1991). A CY-BOCS-SR score of 15.50 or less at discharge was used to indicate clinically significant change, which was calculated using method *c* from Jacobson and Truax (1991). Only participants who met this criterion and demonstrated reliable change were considered to demonstrate clinically significant change.

Results were compared to several RCTs as benchmarks. RCTs were chosen for benchmarking if they included 20 or more children or adolescents with primary OCD who received individual treatment consisting of CBT alone or with medications, and reported CY-BOCS scores at pre- and post-treatment. To allow for comparison, effect sizes were calculated as the difference from pre- to post-treatment divided by the pre-treatment standard deviation (Morris & DeShon, 2002).

Results

Primary Outcomes

Primary outcomes are presented in Table 2, as are the percentage of participants who met criteria for response (≥25% CY-BOCS-SR reduction), excellent response (discharge CY-BOCS-SR ≤ 10), reliable change (see above) and those who met criteria for both reliable and clinically significant change (see above). Overall, participants significantly improved in CY-BOCS-SR and BDI-II or QIDS-SR scores. To determine if the significant change was due to differences in duration of treatment, number of treatment days was correlated with change scores and was not significant for CY-BOCS-SR change, $r = -0.081$, *ns*; BDI-II change, $r = 0.014$, *ns*; or QIDS-SR change, $r = -0.106$, *ns*.

Benchmarking Comparisons

The descriptive characteristics of the current sample and the samples in RCTs used for comparison are provided in Table 3. Effect sizes are displayed in Figure 2. According to Cohen's (1977) guidelines, all samples demonstrated large effects, with effect sizes ranging from 1.30 (brief CBT condition in Bolton et al., 2011) to 4.20 (CBT plus sertraline condition in POTS, 2004).

Secondary Outcomes: Follow-Up Data

CY-BOCS-SR—CY-BOCS scores did not significantly change from post-treatment ($M = 11.00$, $SD = 5.96$) to follow-up ($M = 10.30$, $SD = 7.88$), $t(43) = 0.602$, $p = .55$. Of the 136 participants who met response criteria at post-treatment, 39 had follow-up data available, with 35 (89.7%) maintaining responder status at follow-up. Of the 72 participants who met

excellent response criteria at post-treatment, 20 had follow-up data available, with 15 (75.0%) maintaining excellent response.

BDI-II—BDI-II scores significantly increased from post-treatment ($M = 6.21$, $SD = 6.45$) to follow-up ($M = 8.98$, $SD = 8.31$), $t(42) = -2.392$, $p = .021$, $d = -0.43$ although scores remained in the minimal range.

QIDS-SR—No participants had QIDS-SR data available at follow-up.

Discussion

This study examined the outcome of residential treatment for a large sample of severe, treatment refractory adolescents with OCD and considerable comorbidity. At post-treatment, participants had clinically significant and meaningful reductions in OCD severity. Most participants (79.1%) experienced symptom response and 41.9% experienced excellent response with mild-to-minimal symptoms following treatment. Further, 80.8% demonstrated reliable change and 64.0% demonstrated clinically significant change. These findings confirm and extend previous research on the beneficial effects of a multimodal residential treatment program for adolescents with OCD and substantial comorbidity.

Benchmarking data revealed that reductions in OCD symptoms in the current study are in line with those from several RCTs examining CBT alone or with psychotropic medication (e.g., Bolton et al., 2011; Franklin et al., 2011; Piacentini et al., 2011; POTS, 2004; Storch et al., 2007). This is in spite of substantial comorbidity (92.6% had at least one comorbid diagnosis compared to 50% – 80% in RCTs compared) and extensive treatment histories in the current sample, with all participants having previously received psychotropic medication and approximately 90% having previous experience with outpatient psychotherapy. Further, approximately 40% of the current sample had previously received inpatient treatment, and several participants had previously received intensive outpatient, day treatment, or residential treatment. Examination of a subsample of individuals who provided follow-up data indicated that gains in OCD symptoms were maintained on average 1.5 years post-treatment. Compared to the benchmarking samples, the current study employed a flexible treatment duration. While some participants likely discharged prematurely, others remained in treatment until it was determined that they had achieved maximum benefit and were ready to transition to a less intensive treatment setting (e.g., intensive outpatient or traditional outpatient settings). This flexible treatment duration may, in combination with the intensive residential treatment setting, help mitigate the impact of the complexity of the sample on outcomes. Future comparisons may benefit from examining how residential treatment compares to traditional outpatient CBT provided over a more flexible treatment duration.

Participants also experienced substantial improvement in depressive symptoms, with, on average, moderate depressive symptoms at pre-treatment and minimal depressive symptoms at post-treatment (Leonard et al., 2014). This is in line with other studies demonstrating reductions in depressive symptoms following CBT for OCD (e.g., Brown, Lester, Jassi, Heyman, & Krebs, 2014; Meyer et al., 2014). The subsample of participants who provided follow-up data reported, on average, depressive symptoms in the minimal range

approximately 1.5 years post-treatment despite an increase from post-treatment to follow-up. This contrasts with Björgvinsson et al. (2008), who did not report significant improvement in depressive symptoms in their sample of adolescents who received residential treatment. It is possible that the multimodal treatment approach presented in the current study led to improvements in depressive symptoms through the inclusion of recreational therapy or other program elements in addition to ERP, or through specific treatment strategies targeting depressive symptoms (i.e., behavioral activation) that were used with some participants.

Because of the naturalistic design and multimodal treatment plan, we cannot determine whether improvements in symptoms of OCD and depression were due to CBT, other specific aspects of treatment (e.g., art therapy, process group), nonspecific aspects of treatment (e.g., residential setting, structured schedule, social support), or medication changes during treatment. Despite this, some hypotheses are offered below.

First, it is unlikely that decreases in symptoms were only due to medication, as all participants had received psychotropic medications prior to admitting. The majority (90.7%) were taking at least one psychotropic medication upon admission, with 84.9% taking at least one antidepressant and 45.9% taking an antipsychotic upon admission. Despite this, it is possible that medication changes impacted our results. At discharge, 95.3% of participants were taking at least one medication, with 90.1% taking at least one antidepressant and 52.3% taking at least one antipsychotic medication. Thus, while we believe it unlikely that symptom improvement was solely due to medication effects, medication changes were made throughout treatment and therefore this possibility cannot be excluded. Second, substantial decreases in OCD symptoms are more likely due to CBT than nonspecific aspects of treatment, given the much lower response rates to nonspecific treatments for OCD (Piacentini et al., 2011).

Improvements in depressive symptoms may have resulted from improvements in OCD symptom severity. In fact, Anholt and colleagues (2011) indicated that depressive symptoms are likely to improve with successful treatment of OCD. For some participants, additional interventions were used to target depressive symptoms and in these cases likely contributed to improvements in mood as well. Further, nonspecific effects (e.g., structured schedule requiring engagement in numerous types of activities, regulated sleep schedule, social contact) likely played a role in reducing depressive symptoms, as decreased avoidance and increased activation are key components of empirically supported treatments for depression (Kanter et al., 2010).

Study Limitations and Future Directions

The present study has several limitations. First, structured diagnostic interviews were not completed as they were not part of the admissions protocol. Although structured clinical interviews were not completed, all diagnoses were determined based on information from a referring professional, a pre-admission screening including the CY-BOCS Symptom Checklist which was then reviewed by a board certified child and adolescent psychiatrist with extensive expertise in the assessment and treatment of OCD, and through an in-person interview with an experienced board-certified child and adolescent psychiatrist. Second, this study was heavily reliant upon self-report measures; therefore it is possible that participants

were under- or over-reporting. Data from our study of self-report versus interviewer CY-BOCS scores (Conelea et al., 2012) would suggest otherwise, but this remains a possibility. Third, most participants were prescribed psychotropic medications as part of their treatment. Data regarding the impact of medication changes on symptoms of OCD and depression were not available and therefore it is possible that medication effects significantly impacted outcomes. As described above, however, we believe it is unlikely that medication alone led to symptom improvement. In addition, the majority of the sample was of European American background; therefore, the results of this study may not generalize to individuals from non-European American racial or ethnic backgrounds.

All participants were prescribed approximately 26.5 hours of ERP per week. Due to varying levels of adherence, however, the amount of ERP each participant actually received was likely variable, and we do not have an objective measure of patient adherence. Finally, while the follow-up results are promising, these should be interpreted with caution due to the small sample size ($n = 44$) compared to the overall sample. In addition, information on treatment received between post-treatment and follow-up was not available. Future studies would benefit from a larger follow-up sample, with information regarding treatment received during the intervening time period.

References

Abramowitz JS, Foa EB, Franklin ME. Exposure and ritual prevention for obsessive-compulsive disorder: Effects of intensive versus twice-weekly sessions. Journal of Consulting and Clinical Psychology. 2003; 71:394–398. [PubMed: 12699033]

Abramowitz JS, Whiteside SP, Deacon BJ. The effectiveness of treatment for pediatric obsessive-compulsive disorder: A meta-analysis. Behavior Therapy. 2005; 36:55–63.

Anholt GE, Aderka IM, van Balkom AJLM, Smit JH, Hermesh H, de Haan E, van Oppen P. The impact of depression on the treatment of obsessive-compulsive disorder: Results from a 5-year follow up. Journal of Affective Disorders. 2011; 135:201–207. [PubMed: 21889802]

American Academy of Child and Adolescent Psychiatry. Practice parameter for the assessment and treatment of children and adolescents with obsessive-compulsive disorder. Journal of the American Academy of Child & Adolescent Psychiatry. 2012; 51:98–113. [PubMed: 22176943]

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed.. Washington, DC: Author; 2000.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed.. Arlington, VA: American Psychiatric Publishing; 2013.

Barrett P, Healy-Farrell L, March JS. Cognitive-behavioral family treatment of childhood obsessive compulsive disorder: A controlled trial. Journal of the American Academy of Child & Adolescent Psychiatry. 2004; 43:46–62. [PubMed: 14691360]

Beck, AT.; Steer, RA.; Brown, GK. Manual for the Beck Depression Inventory. 2nd ed.. San Antonio, TX: Psychological Corporation, Harcourt Brace; 1996.

Björgvinsson T, Wetterneck CT, Powell DM, Chasson GS, Webb SA, Hart J, et al. Treatment outcome for adolescent obsessive-compulsive disorder in a specialized hospital setting. Journal of Psychiatric Practice. 2008; 14:137–145. [PubMed: 18520782]

Bolton D, Luckie M, Steinberg D. Long-term follow-up of obsessive-compulsive disorder treated in adolescence. Journal of the American Academy of Child &Adolescent Psychiatry. 1995; 34:1441–1450. [PubMed: 8543511]

Bolton D, Perrin S. Evaluation of exposure and response-prevention for obsessive compulsive disorder in children and adolescents. Behavior Therapy and Experimental Psychiatry. 2008; 39:11–22.

Bolton D, Williams T, Perrin S, Atkinson L, Gallop C, Waite P, Salkovskis P. Randomized controlled trial of full and brief cognitive-behaviour therapy and wait-list for paediatric obsessive-compulsive disorder. The Journal of Child Psychology and Psychiatry. 2011; 52:1269–1278. [PubMed: 21644984]

Brown HM, Lester KJ, Jassi A, Heyman I, Krebs G. Paediatric obsessive-compulsive disorder and depressive symptoms: Clinical correlates and CBT treatment outcomes. Journal of Abnormal Child Psychology. 2014

Conelea CA, Schmidt ER, Leonard RC, Riemann BC, Cahill S. The Children's Yale-Brown Obsessive Compulsive Scale: Clinician versus self-report format in adolescents in a residential treatment facility. Journal of Obsessive-Compulsive and Related Disorders. 2010; 1:69–72.

Franklin ME, Abramowitz JS, Kozak MJ, Levitt JT, Foa EB. Effectiveness of exposure and ritual prevention for obsessive-compulsive disorder: Randomized compared with non-randomized samples. Journal of Consulting and Clinical Psychology. 2000; 68:594–602. [PubMed: 10965635]

Franklin ME, Kozak MJ, Cashman LA, Coles ME, Rheingold AA, Foa EB. Cognitive-behavioral treatment of pediatric obsessive-compulsive disorder: an open trial. Journal of the American Academy of Child & Adolescent Psychiatry. 1998; 37:412–419. [PubMed: 9549962]

Franklin ME, Saptya J, Freeman JB, Khanna M, Compton S, Almirall D, et al. Cognitive behavior therapy augmentation of pharmacotherapy in pediatric obsessive-compulsive disorder: The pediatric OCD treatment study II (POTS II) randomized controlled trial. Journal of the American Medical Association. 2011; 306:1224–1232. [PubMed: 21934055]

Franklin ME, Tolin DF, March JS, Foa EB. Treatment of pediatric obsessive-compulsive disorder: a case example of intensive cognitive-behavioral therapy involving exposure and ritual prevention. Cognitive and Behavioral Practice. 2001; 8:297–304.

de Haan E, Hoogduin KAL, Buitelaar JK, Keijesers GPJ. Behavior therapy versus clomipramine for the treatment of obsessive-compulsive disorder in children and adolescents. Journal of the American Academy of \Child & Adolescent Psychiatry. 1998; 37:1022–1029. [PubMed: 9785713]

Hanna GL. Demographic and clinical features of obsessive-compulsive disorder in children and adolescents. Journal of the Academy of Child & Adolescent Psychiatry. 1995; 34:19–27.

Jacobson NS, Truax P. Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. Journal of Consulting and Clinical Psychology. 1991; 59:12–19. [PubMed: 2002127]

Kanter JW, Manos RC, Bowe WM, Baruch DE, Busch AM, Rusch LC. What is behavioral activation? A review of the empirical literature. Clinical Psychology Review. 2010; 30:608–620. [PubMed: 20677369]

Lack CW, Storch EA, Keeley ML, Geffken GR, Ricketts ED, Murphy Tk, Goodman WK. Quality of life in children and adolescents with obsessive-compulsive disorder: base rates, parent-child agreement, and clinical correlates. Social Psychiatry and Psychiatric Epidemiology. 2009; 44:935–942. [PubMed: 19255701]

Leonard RC, Jacobi DM, Riemann BC, Lake PM, Luhn R. The effect of depression symptom severity on OCD treatment outcome in an adolescent residential sample. Journal of Obsessive Compulsive and Related Disorders. 2014; 3:95–101.

March, J.; Mulle, K. OCD in Children and Adolescents: A Cognitive-Behavioral Treatment Manual. New York: Guilford Press; 1998.

Marien WE, Storch EA, Geffken GR, Murphy TK. Intensive Family-Based Cognitive-Behavioral Therapy: Applications for Treatment of Medication-Resistant Pediatric Obsessive-Compulsive Disorder. Cognitive and Behavioral Practice. 2009; 16:304–316.

Merlo LJ, Lehmkuhl HD, Geffken GR, Storch EA. Decreased family accommodation associated with improved therapy outcome in pediatric obsessive-compulsive disorder. Journal of Consulting and Clinical Psychology. 2009; 77:355–360. [PubMed: 19309195]

Meyer JM, McNamara JP, Reid AM, Storch EA, Geffken GR, Mason DM, Bussing R. Prospective relationship between obsessive-compulsive and depressive symptoms during multimodal treatment in pediatric obsessive-compulsive disorder. Child Psychiatry & Human Development. 2014; 45:163–172. [PubMed: 23756717]

Morris SB, DeShon RP. Combining effect size estimates in meta-analysis with repeated measures and independent-groups designs. Psychological Methods. 2002; 7:105–125. [PubMed: 11928886]

Nakatani E, Mataix-Cols D, Micali N, Turner C, Heyman I. Outcomes of cognitive behaviour therapy for obsessive-compulsive disorder in a clinical setting: A 10-year experience from a specialist OCD service for children and adolescents. Child and Adolescent Mental Health. 2009; 14:133–139.

National Institute for Health and Clinical Excellence (NICE). Obsessive-compulsive disorder: core interventions in the treatment of obsessive-compulsive disorder and body dysmorphic disorder. London: National Institute for Health and Clinical Excellence; 2005a. p. CG31www.nice.org.uk

Olatunji BO, Davis ML, Powers MB, Smits JAJ. Cognitive-behavioral therapy for obsessive-compulsive disorder: A meta-analysis of treatment outcome and moderators. Journal of Psychiatric Research. 2013; 47:33–41. [PubMed: 22999486]

Olino TM, Gillo S, Rowe D, Palermo MPH, Nuhfer EC, Birmaher B, Gilbert AR. Evidence for successful implementation of exposure and response prevention in a naturalistic group format for pediatric OCD. Depression and Anxiety. 2011; 28:342–348. [PubMed: 21456041]

Pediatric OCD Treatment Study (POTS) Group. Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder. Journal of the American Medical Association. 2004; 292:1969–1975. [PubMed: 15507582]

Pence SL, Storch EA, Geffken GR. Barriers to Evidenced-Based Care: A Case Study in Pediatric Obsessive-Compulsive Disorder. Annals of the American Psychotherapy Association, Spring. 2010:58–61.

Piacentini J, Bergman L, Chang S, Langley A, Peris T, Wood JJ, McCracken J. Controlled comparison of family cognitive behavioral therapy and psychoeducation/relaxation training for child obsessive-compulsive disorder. Journal of the American Academy of Child & Adolescent Psychiatry. 2011; 50:1149–1161. [PubMed: 22024003]

Piacentini J, Bergman RL, Keller M, McCracken J. Functional impairment in children and adolescents with obsessive-compulsive disorder. Journal of Child and Adolescent Psychopharmacology. 2003; 13(Suppl 1):S61–S69. [PubMed: 12880501]

Piacentini, J.; Langley, A.; Roblek, T. Cognitive-behavioral treatment of childhood ocd: It's only a false alarm, therapist guide. New York, NY: Oxford University Press; 2007.

Rush AJ, Trivedi MH, Ibrahim HM, Carmody TJ, Arnow B, Klein DN, et al. The 16-item Quick Inventory of Depressive Symptomatology (QIDS) Clinician Rating (QIDS-C) and Self-Report (QIDS-SR): A psychometric evaluation in patients with chronic major depression. Biological Psychiatry. 2003; 54:573–583. [PubMed: 12946886]

Scahill L, Riddle MA, McSwiggin-Hardin M, Ort SI, King RA, Goodman WK, Leckman JF. Children's Yale-Brown obsessive-compulsive scale: Reliability and validity. Journal of the American Academy of Child & Adolescent Psychiatry. 1997; 36:844–852. [PubMed: 9183141]

Simpson HB, Huppert JD, Petkova E, Foa EB, Liebowitz MR. Response versus remission in obsessive-compulsive disorder. Journal of Clinical Psychiatry. 2006; 67:269–276. [PubMed: 16566623]

Steer RA, Kumar G, Ranieri WF, Beck AT. Use of the Beck Depression Inventory-II with adolescent psychiatric outpatients. Journal of Psychopathology and Behavioral Assessment. 1998; 20:127–137.

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, Geffken GR, Merlo LJ, Mann G, Duke D, Munson M, Adkins J, et al. Family-based cognitive-behavioral therapy for pediatric obsessive-compulsive disorder: Comparison of intensive and weekly approaches. Journal of the American Academy of Child & Adolescent Psychiatry. 2007; 46:469–478. [PubMed: 17420681]

Storch EA, Gerdes AC, Adkins JW, Gefken GR, Star J, Murphy T. Behavioral treatment of a child with PANDAS. Journal of the American Academy of Child & Adolescent Psychiatry. 2004; 43:510–511. [PubMed: 15100555]

Storch EA, Lehmkuhl HD, Ricketts E, Geffken GR, Marien W, Murphy TK. An open trial of intensive family based cognitive-behavioral therapy in youth with obsessive-compulsive disorder who are

medication partial responders or nonresponders. Journal of Clinical Child & Adolescent Psychology. 2010; 39:260–268. [PubMed: 20390817]

Storch EA, Murphy TK, Geffken GR, Mann G, Adkins J, Merlo LJ, et al. Cognitive-behavioral therapy for PANDAS related obsessive-compulsive disorder: findings from a preliminary wait-list controlled open trial. Journal of the American Academy of Child & Adolescent Psychiatry. 2006; 45:1171–1178. [PubMed: 17003662]

Vande Voort JL, Svecova J, Jacobsen AB, Whiteside SP. A retrospective examination of the similarity between clinical practice and manualized treatment for childhood anxiety disorders. Cognitive and Behavioral Practice. 2010; 17:322–328.

Warren R, Thomas JC. Cognitive-behavior therapy of obsessive-compulsive disorder in private practice: An effectiveness study. Journal of Anxiety Disorder. 2001; 15:277–285.

Watson HJ, Rees CS. Meta-analysis of randomized, controlled treatment trials for pediatric obsessive-compulsive disorder. Journal of Child Psychology and Psychiatry. 2008; 49:489–498. [PubMed: 18400058]

Whiteside SP, Jacobsen AB. An uncontrolled examination of a 5-day intensive treatment for pediatric OCD. Behavior Therapy. 2010; 41:414–422. [PubMed: 20569789]

Whiteside SP, Brown AM, Abramowitz JS. Five-day intensive treatment for adolescent OCD: A case series. Journal of Anxiety Disorders. 2008; 22:495–504. [PubMed: 17543497]

Williams TI, Salkovskis PM, Forrester L, Turner S, White H, Allsopp MA. A randomized controlled trial of cognitive behavioural treatment for obsessive compulsive disorder in children and adolescents. European Child & Adolescent Psychiatry. 2010; 19:449–456. [PubMed: 19921305]

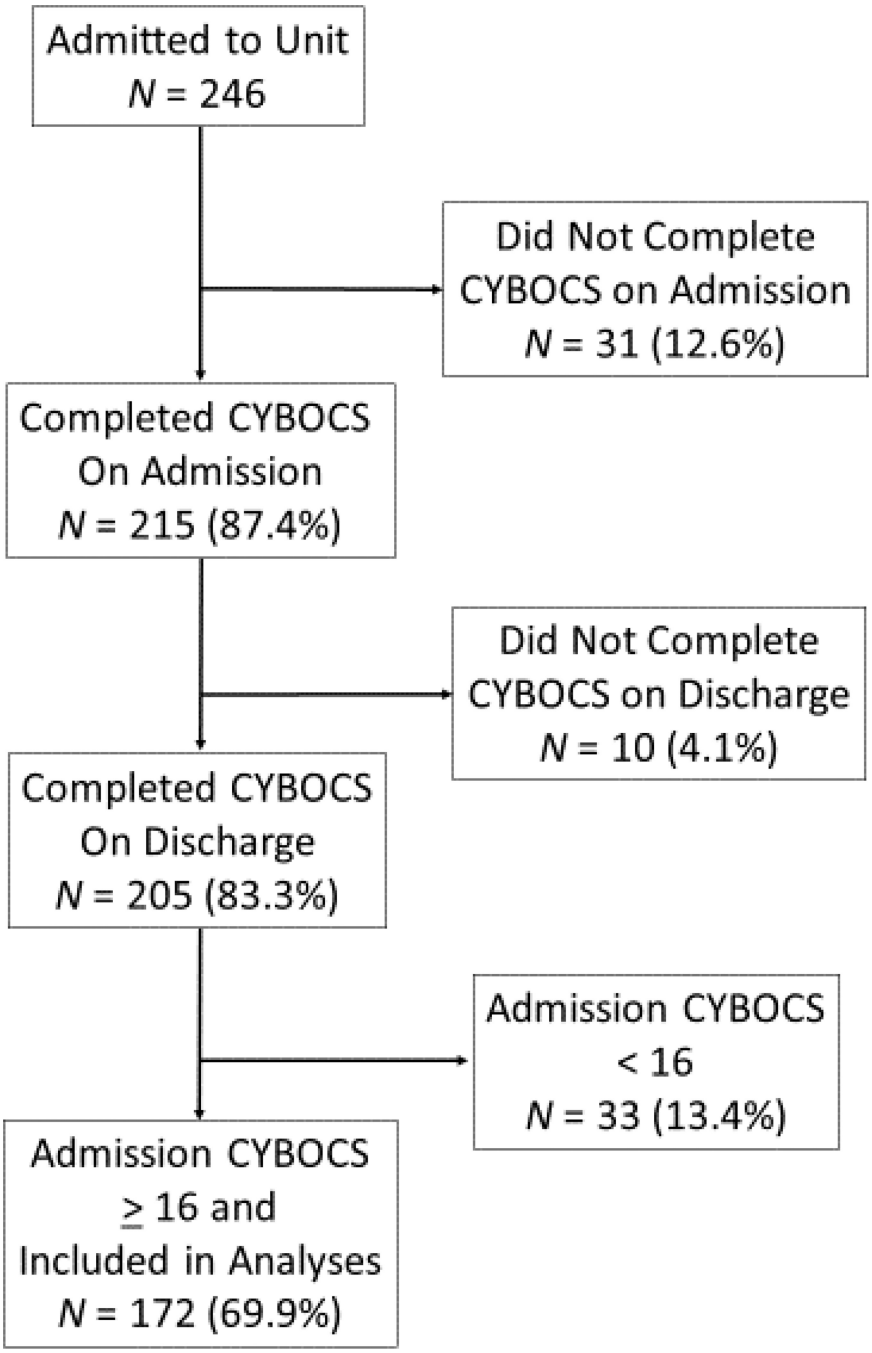

Figure 1. Participant flow

Note. percentages are out of the total, *N* = 246

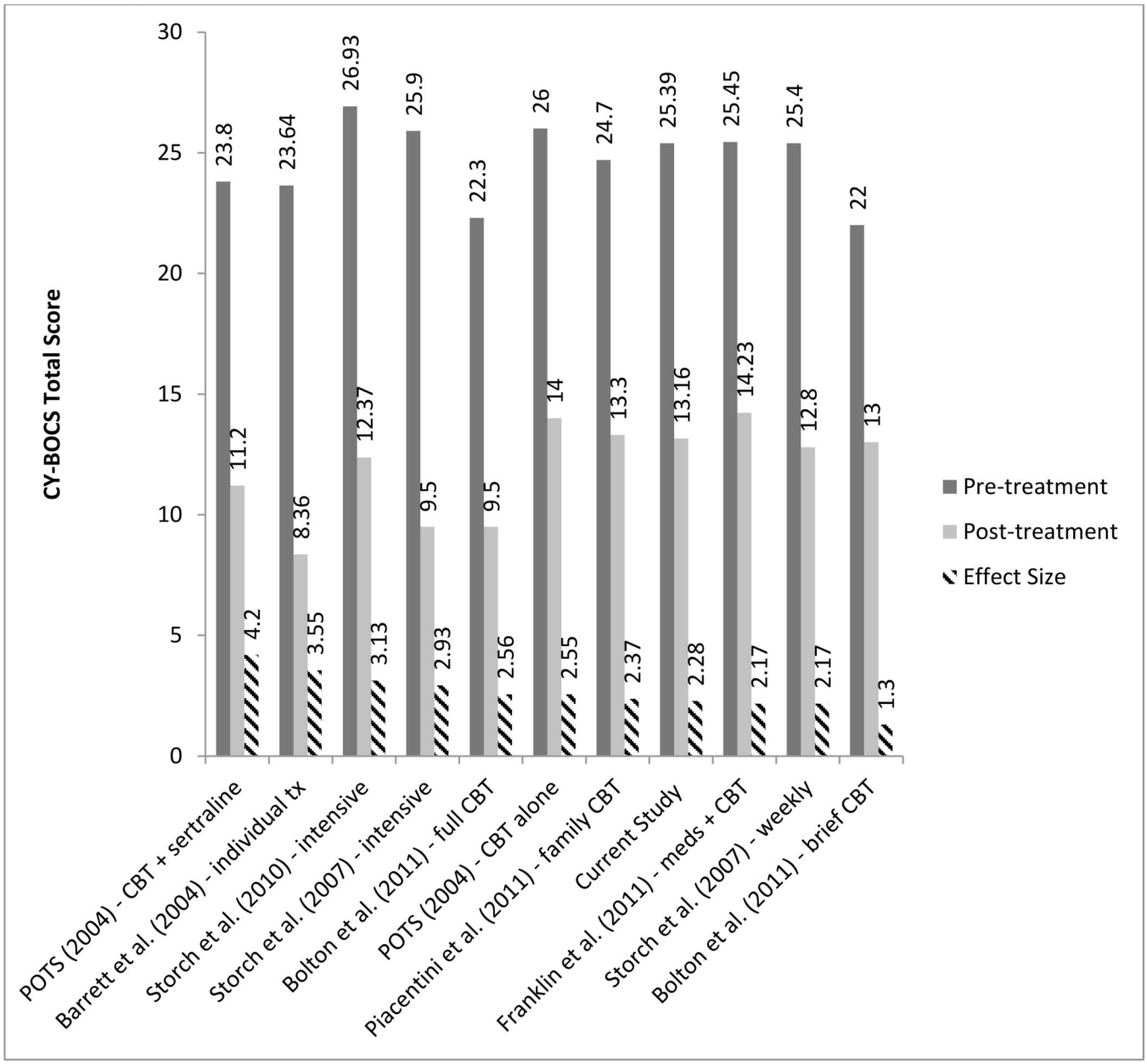

Figure 2. Pre-and post-treatment CY-BOCS scores and effect sizes

Note. Sample size for the current study was 172, with sample size for the additional studies/ study conditions presented above ranging from 20 to 49. Effect size calculated following Morris and DeShon (2002; mean difference divided by pre-treatment SD).

Table 1

Number and Type of Comorbid Diagnoses

Number of Diagnoses	***N* (%)**
1 (OCD)	13 (7.6%)
2	82 (47.7%)
3	51 (29.7%)
4	18 (10.5%)
5	8 (4.7%)
Comorbid Diagnoses	*N* (%)
Mood disorder not otherwise specified (NOS)	82 (47.7%)
Attention deficit hyperactivity disorder	36 20.9%)
Major depressive disorder	36 (20.9%)
Autism spectrum disorder (any)	20 (11.6%)
Generalized anxiety disorder	19 (11.0%)
Social anxiety disorder	17 (9.9%)
Tic disorder (any)	15 (8.7%)
Eating disorder NOS	7 (4.1%)
Panic disorder	6 (3.5%)
Learning disorder (any)	5 (2.9%)
Anorexia nervosa	4 (2.3%)
Substance use disorder (any)	4 (2.3%)
Other	18 (10.5%)

Table 2

Outcome Data

Measure	Admission *M* (*SD*), range	Discharge *M* (*SD*), range	*t*(df), *p*	*d*	Follow-up *M* (*SD*), range	*t*(df), *p*	% met response criteria	% met excellent response criteria	% met RCI criteria	% met clinically significant change criteria
CY-BOCS-SR (*n* = 172, 44) *	25.39 (5.36), 16 – 40	13.16 (7.57), 0 – 39	19.044 (171), <001	2.28	10.30 (7.88), 0 – 34	0.602(43), .55	79.1%	41.9%	80.8%	64.0%
BDI-II (*n* = 126, 43) *	20.22 (12.90), 0 – 57	7.66 (8.12), 0 – 43	11.737 (125), <.001	0.97	8.98 (8.31), 0 – 27	−2.392(42), < .05	N/A	N/A	N/A	N/A
QIDS-SR (*n* = 12, 0) *	11.17 (5.91), 4 – 21	6.50 (4.10), 2 – 13	2.548 (11), < .05	0.79	N/A	N/A	N/A	N/A	N/A	N/A

Note: Effect size calculated following Morris and DeShon (2002; mean difference divided by pre-treatment SD), response = a CY-BOCS-SR decrease of 25% or more from admission to discharge, excellent response defined = discharge CY-BOCS-SR of 10 or less, reliable change index (RCI) and criteria for clinically significant change calculated according to Jacobson & Truax (1991), with clinically significant change indicated by a participant meeting criteria for the RCI and having a discharge CY-BOCS-SR of less than 15.5. BDI-II data were also reported in Leonard et al. (2014).

* Sample size provided first for admission and discharge data, then for follow-up data.

Table 3

Descriptive Characteristics of the Current Study and RCT Comparison Samples

	N	*M* Age (Years)	Gender (% Female)	% with No Comorbidity	Treatment
Current Study	172	15.45	48.3	7.6	Intensive daily ERP plus medication management and adjunctive treatment.
Barrett et al. (2004) – individual	24	10.75	50.0	21.0*	14 weekly 90 min. sessions and 2 booster sessions. Adjunctive pharmacotherapy allowed if stable 3 mos. prior to treatment.
Bolton et al. (2011) – Full CBT	36	15.00	58.0	Not reported	12 sessions of CBT over 3 mos. Pharmacotherapy allowed if stable 6 weeks prior to study.
Bolton et al. (2011) – Brief CBT	36	14.33	64.0	Not reported	5 sessions of CBT plus use of workbook over 3 mos. Pharmacotherapy for OCD allowed if stable 6 weeks prior to study.
Franklin et al., 2011 – medications plus CBT	42	12.71	54.8	50.0	14 60 min. sessions over 12 weeks. 7 35 min. medication management visits over 12 weeks. Sample on SRIs. Sample included partial responders to SRIs.
Piacentini et al. (2011) – Family CBT	49	12.40	59.2	30.6	12 90 min. sessions over 14 weeks of CBT with structured family intervention. Psychotropic medication proscribed.
POTS (2004) – CBT alone	28	11.40	50.0	20.0*	14 60 min. sessions over 12 weeks. Psychotropic medication proscribed.
POTS (2004) – CBT plus sertraline	28	11.70	60.7	20.0*	14 60 min. sessions over 12 weeks. Weekly medication visits.
Storch et al. (2007) – intensive	20	12.00	50.0	25.0	14 90 min. sessions provided daily (weekdays). Adjunctive pharmacotherapy allowed.
Storch et al. (2007) - weekly	20	14.50	40.0	30.0	14 90 min. sessions provided weekly. Adjunctive pharmacotherapy allowed.
Storch et al. (2010) - intensive	30	13.40	50.0	16.7%	14 90 min. sessions provided over 3 weeks. Sample included partial responders and nonresponders to SRIs. Adjunctive pharmacotherapy allowed.

*% with no comorbidity provided for full sample and not specific to the treatment condition.