Title

Characterizing changes in obsessive—compulsive symptoms over the course of treatment for adolescent bulimia nervosa

Journal

International Journal of Eating Disorders, 55(10)

ISSN

0276-3478

Authors

Reilly, Erin E

Gorrell, Sasha

Brosof, Leigh

et al.

Publication Date

2022-10-01

DOI

10.1002/eat.23782

Peer reviewed

HHS Public Access

Author manuscript

Int J Eat Disord. Author manuscript; available in PMC 2023 October 01.

Published in final edited form as:

Int J Eat Disord. 2022 October ; 55(10): 1342–1351. doi:10.1002/eat.23782.

Characterizing Changes in Obsessive-Compulsive Symptoms Over the Course of Treatment for Adolescent Bulimia Nervosa

Erin E. Reilly, Ph.D.[1], **Sasha Gorrell, Ph.D.**[1], **Leigh Brosof, M.S.**[1,2], **James Lock, M.D., Ph.D.**[3], **Daniel Le Grange, Ph.D.**[1,4]

[1]Department of Psychiatry and Behavioral Sciences, University of California, San Francisco

[2]Department of Psychological and Brain Sciences, University of Louisville, Louisville, KY

[3]Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, California, USA

[4]Department of Psychiatry and Behavioral Neuroscience, the University of Chicago, Chicago, IL (Emeritus)

Abstract

Objective: Data suggest that obsessive-compulsive (OC) symptoms are commonly observed in adolescents with eating disorders and predict poorer treatment response. Further, emerging data among adults suggest that changes in OC symptoms relate to changes in eating disorder symptoms across treatment. Given evidence that early invention decreases risk for protracted illness, evaluating processes that may relate to treatment response will be useful in increasing the effectiveness of existing interventions. Therefore, the current investigation explored changes in general and eating disorder-specific OC symptoms throughout Family-Based Treatment (FBT) and Cognitive Behavioral Therapy (CBT) for bulimia nervosa, as well as associations among these changes and eating disorder outcomes at follow-up.

Method: Participants (N = 110) received 18 sessions of FBT or CBT and completed measurements of general and eating disorder-specific OC symptoms at baseline, end-of-treatment, and 6- and 12-month follow-up.

Results: Multi-level models indicated that across both treatments, there was no change in general OC symptoms, whereas all eating disorder-related OC symptoms decreased over treatment and follow-up. Exploratory analyses indicated that lower severity in discharge eating-disorder-specific OC symptoms contributed to lower eating pathology at follow-up.

Discussion: Together, findings support the efficacy of both FBT and CBT in helping to reduce eating disorder-specific OC symptoms and suggest that adjunctive intervention may be required for ameliorating general OC symptoms in this population.

Correspondence for this article should be addressed to: Dr. Erin Reilly, Department of Psychiatry and Behavioral Sciences, Weill Institute for Neurosciences, University of California, San Francisco, 401 Parnassus Avenue, San Francisco, CA 94143. erin.reilly3@ucsf.edu.

Keywords

bulimia nervosa; family-based treatment; cognitive-behavioral therapy; adolescent; obsessive-compulsive symptoms

Introduction

Obsessive-compulsive (OC) symptoms are common in adolescents with bulimia nervosa (BN); in clinical samples across the lifespan, up to 43% of individuals with BN report comorbid OCD diagnoses, with even higher numbers when considering subclinical, yet clinically significant, OC symptoms (Anderluh et al., 2003; Kaye et al., 2004; Mandelli et al., 2020; Swinbourne & Touyz, 2007). Data consistently suggest that OC symptoms, both general and eating disorder (ED)-specific (e.g., rituals related to meal consumption; compulsive exercise), may hold relevance to clinical outcomes. However, there are several limitations of existing work on OC symptoms in adolescent EDs, including a predominant focus on anorexia nervosa to the neglect of other ED diagnoses, lack of exploration of differential impact of ED-specific and general OC symptoms, and few studies exploring changes in different types of OC symptoms over the course of treatment. In this study, we seek to inform the development of improved interventions in BN by exploring changes in OC symptoms, a known predictor of treatment response in adolescent EDs (Le Grange et al., 2015; Le Grange et al., 2012), and associations between changes in OC symptoms and ED outcomes at follow-up in two commonly-used, evidence-based treatments for adolescent BN—Family-Based Treatment (FBT) and Cognitive-Behavioral Therapy (CBT).

OC symptoms can manifest in ED-specific domains, or present as co-occurring symptoms in a more general form (e.g., washing/counting rituals) (Altman & Shankman, 2009; Halmi, 2004; Halmi et al., 2005; Meyer et al., 2011). Of note, although work suggests that these two clusters of symptoms likely share psychological and behavioral mechanisms (e.g., intolerance of uncertainty; Williams & Levinson, 2021), existing research on OC symptoms in EDs does not typically distinguish between general and ED-specific OC symptoms; given some data suggesting that OC symptoms can emerge in concordance with malnutrition (Keys et al., 1950), it may be the case that these symptoms have differential features or respond in a different manner to intervention. Further, despite the fact that prevalence data suggest OC symptoms are commonly present in both anorexia nervosa *and* BN (Von Ranson et al., 1999), most work to date has focused on these symptoms in anorexia nervosa.

Across diagnostic categories, data have implicated OC symptoms as a predictor of poorer treatment response among adolescents with different EDs, and in some randomized controlled AN trials, as a moderator of response to different treatment formats (Le Grange et al., 2012; Lock et al., 2005; Madden et al., 2015). Within a prior analysis of the current dataset, greater ED-specific OC symptoms at baseline were associated with decreased likelihood of abstinence from BN behaviors at end-of-treatment (EOT) (Le Grange et al., 2015), tentatively supporting the assertion that ED-specific OC features are relevant to BN clinical outcomes and may be important to target in an effort to prevent relapse.

However, while ED-specific OC symptoms seem to be relevant to treatment outcomes in adolescent EDs, less is known regarding whether existing interventions reduce or exacerbate general and ED-specific OC symptoms, and whether these changes relate to subsequent changes in ED outcomes. To date, one study of adults with transdiagnostic EDs suggested that changes in OC symptoms related to changes in ED symptoms, supporting the potential for reciprocal clinical relationships among these symptoms (Olatunji et al., 2010). Another early study in adult BN suggested no changes in comorbid OCD even after ED symptoms were remitted (Von Ranson et al., 1999). Understanding whether general *and* ED-specific OC symptoms decrease over the course of existing treatments for adolescents with BN would be useful in determining whether existing BN treatments are sufficient for targeting different types of OC symptoms and whether additional intervention is warranted for this cluster of symptoms. If it is the case that OC symptoms are targeted by FBT and CBT, this initial data may provide insights into potential mechanisms through which these treatments may exert their effects. Given the focus of existing treatments on regular eating and promoting flexibility in eating behaviors, it is possible that ED-specific OC symptoms will decrease over the course of treatment, with general OC symptoms remaining consistent. On the other hand, it could be the case that increases in flexibility in the eating domain and approaching feared stimuli (e.g., food) via refeeding will translate into more general gains in flexibility that result in changing general symptoms. Regardless, testing whether observed changes in different types of OC symptoms relate to ED outcomes will provide insights regarding whether targeting general and ED-specific OC symptoms is necessary for achieving remission from BN.

In this study, we evaluated changes in general and ED-specific OC symptoms throughout FBT and CBT and at 6- and 12-month follow-up with two objectives: first, we aimed to characterize quantitative changes in general and ED-specific OC symptoms over 18 weeks of treatment and at follow-up. Second, given past research suggesting associations between OC and ED symptom change (Olatunji et al., 2010), in an exploratory manner, we tested change in general and ED-specific OC symptoms throughout treatment as a predictor of global eating pathology at follow-up. Based on past work in adults (Olatunji et al., 2010), we expected greater reductions in OC symptoms to be related to lower eating pathology at 12-month follow-up. While we did have hypotheses for our project based on prior work in this area, due to small relative sample sizes and the secondary nature of analyses, we conceptualized this project as hypothesis generating, rather than confirming.

Method

Participants

We conducted secondary data analysis of a randomized controlled trial of FBT and CBT in adolescent BN (Clinical Trials Registration: NCT00879151; c.f., Le Grange et al., 2015), comprised of 110 individuals (aged 12-18), randomized to receive CBT ($n = 58$) and FBT ($n = 52$). We included the full sample in analyses probing ED-specific OC symptoms; for models exploring general OC symptoms, we included participants endorsing symptoms higher than established cutoffs for “mild” OC symptoms (Children’s Yale-Brown Obsessive Compulsive Scale [CYBOCS] total score > 5; $n = 59$; Lewin et al., 2014[1]); mean scores

on CYBOCS at baseline did not differ across treatment groups, $t(108) = .72$, $p = .473$. Of note, only 7.3% of the sample endorsed a full-threshold prior diagnosis of OCD, and the distribution of participants with formal OCD diagnoses did not differ across treatment condition, $\chi(1) = .03$, $p = .873$. Demographic information for the full sample and general-OC subsample are available in Table 1.

Measurements

Eating Disorder Examination (EDE; Fairburn, 2008).—Participants completed the EDE, a psychometrically-sound measurement of ED pathology over the past 28 days (Berg et al., 2012). We used the EDE global score to provide a continuous measurement of ED cognitive pathology. We also evaluated items gauging the frequency of objective binge-eating episodes and self-induced vomiting over the past 28 days as indicators of BN behaviors.

Obsessive-compulsive symptoms.—The **Children's Yale-Brown Obsessive Compulsive Scale,** (CYBOCS; Scahill et al., 1997) measures the nature of obsessions and compulsions through a checklist, with follow-up queries for distress, impairment, and frequency of each symptom endorsed. Cronbach's α for the CYBOCS across timepoints were excellent (range = .92-.95). The **Yale-Brown-Cornell Eating Disorders Scale (YBC-ED;** Bellace et al., 2012) interview gauges ED-specific preoccupations and rituals using a 65-item symptom checklist and a corresponding 19-item assessment probing associated impairment, intensity, and distress. Reliability was strong across timepoints (Cronbach's α = .89-.95).

Treatments

As described elsewhere (Le Grange et al., 2015), participants received 18 sessions of CBT adapted for adolescents (CBT-A; Lock, 2005), based on the protocol outlined by Fairburn (Fairburn, 2008) with developmentally appropriate adjustments as outlined by Lock (2005), or FBT, based on the published manual by Le Grange and Lock (Le Grange & Lock, 2007).

Procedures

Eligibility and recruitment procedures for the study are available in the main outcome report (Le Grange et al., 2015). Following randomization to a study condition, participants and their caregivers completed assessments at baseline, EOT, 6- and 12-month follow-up. There was attrition across timepoints, with assessments completed by 92 (83.6%) subjects at EOT, 70 (63.6%) at 6-month follow-up, and 71 (64.5%) at 12-month follow-up. All study procedures were approved by The University of Chicago and Stanford University Institutional Review Boards.

Statistical Analysis Plan

To explore changes in general and ED-specific OC symptoms over time, we ran multi-level models using the lme4 (Bates et al., 2015) and r2glmm packages in R (Jaeger,

[1]Of note, we conducted sensitivity analyses wherein we replicated study models using a higher cutoff for scores (i.e., CYBOCS total >14). The pattern of results remained unchanged.

2017). Within each model, we entered comorbid psychopathology (dichotomous variable indicating whether the individual had received any other psychiatric diagnosis other than an eating disorder), treatment assignment, and baseline EDE global scores as covariates. We also entered the interaction between time (weeks) and treatment condition to explore whether the trajectory of OC symptom change varied across groups. Within this aim, we addressed missing data through use of full-information maximum likelihood estimation, which represents accepted best practice for missing-data analysis (Baraldi & Enders, 2010; Enders & Bandalos, 2001).

To test associations between change in OC symptoms over treatment and follow-up EDE global score outcomes, we used a multiple regression approach, entering EDE global scores at 12-month follow-up as the dependent variable, baseline EDE global scores and treatment assignment at Step 1, baseline OC symptoms at Step 2, and OC symptoms at EOT at Step 3. Entering both baseline and EOT OC symptoms simultaneously in the regression model results in EOT OC symptoms representing "change" in OC symptoms over treatment (as baseline OC symptoms are thus controlled for). This approach circumvents issues related to change scores (Norman, 1989), but allows us to explore associations between changes in OC symptoms throughout treatment with EDE global scores over time, as general and ED-specific OC scores at end-of-treatment, when controlling for baseline, serve as a proxy of variance attributable to treatment change. For models exploring associations between change in OC symptoms and BN behavioral symptoms (i.e., binge eating and purging episodes over the past 28 days), we conducted negative binomial regressions given that these were count variables (Schaumberg et al., 2018). Across regression analyses, we handled missing data using list-wise deletion; while list-wise deletion can be prone to bias, Little's MCAR test indicated data were missing completely at random. However, regression results should be interpreted with caution.

Results

Changes in General and ED-Related OC Symptoms

Descriptive statistics are available in Table 1. Within the general-OC multi-level model, significant effects were evidenced for baseline EDE global scores, indicating a positive association between EDE global scores and CYBOCS scores across time (Table 2). There was no effect of time, treatment, or their interaction, suggesting no mean changes in general OC symptoms over time and no treatment-related differences in change.

The ED-specific OC multi-level model indicated a significant effect of time, such that ED-related OC symptoms decreased over time. The effects of treatment and the interaction of treatment and time were not significant, suggesting no differences in mean levels of ED-related OC symptoms across groups nor differences in the slopes of symptoms over time.

Associations between Change in OC Symptoms and ED Symptoms at Follow-Up

EDE Global Scores.—Within the model exploring associations between general OC symptoms during treatment and EDE scores at follow-up (Table 3), only the effect of

baseline EDE global scores was significant. The effects of general OC symptoms at baseline and EOT were not significant, suggesting no association between changes in OC symptoms and follow-up ED symptoms.

The model exploring associations between ED-related OC symptoms and EDE global scores indicated a significant effect of ED-related OC symptoms at discharge, suggesting that after controlling for baseline ED-related OC symptoms, elevated ED-related OC symptoms at discharge (i.e., demonstrated less change) were associated with elevated ED symptoms at follow-up. No other effects were significant.

Binge Eating.—Within the negative binomial regression model exploring changes in general OC symptoms during treatment and binge-eating episodes at follow-up (Table 4), no effects were significant, indicating no links between change in general OC symptoms and binge eating. On the other hand, models testing changes in ED-related OC symptoms and binge eating at 12-month follow-up indicated significant effects for ED-specific OC symptoms at both baseline and EOT, indicating that greater YBC scores at baseline *and* end-of-treatment (elevations in ED-specific OC symptoms *and* those with less change) were related to more frequent binge-eating episodes at follow-up.

Self-Induced Vomiting.—Negative binomial models exploring changes in general OC symptoms predicting self-induced vomiting at 12-month follow-up suggested significant effects of baseline vomiting episodes and treatment group, such that individuals receiving CBT reported more frequent vomiting at 12-month follow-up. No effects of general OC symptoms were significant. The model exploring changes in ED-specific OC symptoms and vomiting episodes at follow-up indicated significant effects of baseline vomiting episodes, treatment group, and YBC at end-of-treatment, such that elevated frequency of vomiting at baseline, receiving CBT, and having greater ED-specific OC symptoms at end-of-treatment (i.e., proxy for less change in vomiting) were all associated with more frequent vomiting one year later.

Discussion

The current study extended prior research indicating that obsessionality relates to treatment response in EDs (Agras et al., 2014; Le Grange et al., 2015b; Lock et al., 2005) through examining the longitudinal course of general and ED-specific OC symptoms in two evidence-based treatments for adolescent BN. Altogether, our findings broadly support the importance of considering ED-specific OC symptoms in treatment for adolescent EDs and highlight several main avenues for potential work in this domain.

ED-specific OC Symptoms

Analyses focused on ED-related OC symptoms suggested mean decreases in YBC scores throughout treatment and follow-up. Findings indicated that these effects were similar across treatments. Thus, these results support the value of both FBT and CBT in reducing ED-related OC cognitions and behavior during BN treatment; this finding is notable considering that neither treatment is explicitly designed to incorporate treatment techniques commonly-used for obsessionality or rituals (e.g., exposure). However, it may be that behavioral

techniques used within CBT and FBT function as exposure exercises, consistent with recent theoretical accounts (Lock & Nicholls, 2020). Moving forward, the relative advantages of a family-based vs. individual approach in reducing ED-related OC behaviors, particularly considering individual difference factors (e.g., motivation to change rituals), warrant further investigation.

Findings also suggested that changes in ED-related OC symptoms accounted for significant variance in ED symptoms (EDE global scores; self-induced vomiting; binge episodes) at 12-month follow-up. Our results build upon literature supporting associations between baseline ED-related OC symptoms and outcome (Gorrell et al., 2022; Le Grange et al., 2012). Therefore, it may be important to support adolescents in reducing ED-related OC symptoms within treatment to promote optimal ED outcomes. Importantly, our efforts to explore changes in ED-related OC symptoms represent an initial step in this domain; future work should characterize heterogeneity in this effect and whether adjunctive interventions directly targeting ED-related OC symptoms (e.g., in vivo and/or imaginal exposure; Law & Boisseau, 2019) are warranted for subgroups less likely to show clinical change with standard treatments. Of note, given strong correlations between YBC and EDE global scores, it is also important to consider and test in future research the possibility that these measurements may be somewhat overlapping in content (i.e., ritualistic eating or obsessionality may be measured by EDE items).

General OC Symptoms

Results probing changes in general OC symptoms suggested no significant decreases over the course of treatment and follow-up. Although this finding contrasts with prior research suggesting that comorbid symptoms (e.g., anxiety; depression) may decrease in FBT and CBT (Le Grange et al., 2020; Simic et al., 2022; Trainor et al., 2020), the focus in FBT and CBT is intently on ED symptoms; therefore, additional intervention is likely required to bring about change in obsessions or compulsions more generally; it is also possible that these findings are related to the relatively low levels of general OC symptoms in our sample and would be different in a larger sample with greater symptom elevations or clinically-diagnosed OCD. It is finally possible that despite some work suggesting that OCD is present in BN samples, they may be less clinically important or present differently in BN samples than they do in AN samples.

In contrast with some past work suggesting reciprocal changes between general OC and ED symptoms in treatment (Olatunji et al., 2010), our data do not suggest that changes in general OC symptoms are necessary for improving ED outcomes among individuals with BN who do not endorse clinical diagnoses of OCD. To reconcile these mixed findings, future research should elucidate the circumstances under which intervention on general OC symptoms may be warranted due to interference in ED outcomes or associations with other outcomes of interest (e.g., quality of life; negative affect), or whether OC symptoms are more or less important to treat depending on other factors (e.g., the degree to which the ED presentation is restrictive). Altogether, ongoing work characterizing and clarifying the role of comorbid, general OC symptoms in ED treatment is indicated.

Limitations

There are study limitations that are important to note. First, few male-identifying subjects enrolled in the trial, precluding our ability to examine the effect of gender. Second, in analyses probing changes in general OC symptoms, our sample sizes were modest and likely underpowered to detect small-to-medium effects. In a parallel manner, attrition in our sample, while comparable to other RCTs in adolescent EDs (Le Grange et al., 2016; Stefini et al., 2017), may bias our findings. Additionally, although we did have data on psychotropic medication prescribed within the sample (i.e., 9.1% prescribed psychotropic medications), we did not collect data on the symptoms for which this medication was prescribed, limiting our ability to explore the influence of OCD-related medication on outcomes. Particularly given ample data suggesting medications for treatment of OC features (Abramowitz et al., 2005), future work should use more explicit measurements of medication and explore the potential ways through which medication use may be useful alongside psychotherapy for adolescent BN. Notably, our decision to complete analyses regarding general OC symptoms in a subsample of our population endorsing mild OC symptoms (rather than exploring these analyses only among individuals with diagnosed OCD or more severe symptoms) was made to balance considerations regarding power as well as data suggesting that general OC symptoms are continuous in nature; however, this decision ultimately results in limitations regarding the generalizability of our findings to samples with differing levels of OC symptoms. Altogether, our analyses represent a hypothesis-generating endeavor that should be used as a first step toward future work in this domain.

Conclusions

Although OC symptoms relate to ED treatment response (e.g., Le Grange et al., 2012), less is known about how these symptoms respond to existing interventions and relate to changes in ED symptoms over time. Both CBT and FBT appear to be helpful in reducing ED-specific OC symptoms; however, results suggest that general OC symptoms do not significantly change during a standard course of treatment. Future work must focus on further delineating how to help adolescents to achieve overall improvement in OC symptoms during evidence-based treatment for BN.

Data Sharing Statement

The data supporting the current study are available upon reasonable request from the 4th and 5th authors.

References

Abramowitz JS, Whiteside SP, & Deacon BJ (2005). The effectiveness of treatment for pediatric obsessive-compulsive disorder: A meta-analysis. Behavior Therapy, 36(1), 55–63. 10.1016/S0005-7894(05)80054-1

Agras WS, Lock J, Brandt H, Bryson SW, Dodge E, Halmi KA, Jo B, Johnson C, Kaye W, Wilfley D, & Woodside B (2014). Comparison of 2 Family Therapies for Adolescent Anorexia Nervosa: A Randomized Parallel Trial. JAMA Psychiatry, 71(11), 1279. 10.1001/jamapsychiatry.2014.1025 [PubMed: 25250660]

Altman SE, & Shankman SA (2009). What is the association between obsessive–compulsive disorder and eating disorders? Clinical Psychology Review, 29(7), 638–646. 10.1016/j.cpr.2009.08.001 [PubMed: 19744759]

Anderluh MB, Tchanturia K, Rabe-Hesketh S, & Treasure J (2003). Childhood Obsessive-Compulsive Personality Traits in Adult Women With Eating Disorders: Defining a Broader Eating Disorder Phenotype. American Journal of Psychiatry, 160(2), 242–247. 10.1176/appi.ajp.160.2.242 [PubMed: 12562569]

Baraldi AN, & Enders CK (2010). An introduction to modern missing data analyses. Journal of School Psychology, 48(1), 5–37. 10.1016/j.jsp.2009.10.001 [PubMed: 20006986]

Bates D, Mächler M, Bolker B, & Walker S (2015). Fitting Linear Mixed-Effects Models Using lme4. Journal of Statistical Software, 67(1), 1–48. 10.18637/jss.v067.i01

Bellace DL, Tesser R, Berthod S, Wisotzke K, Crosby RD, Crow SJ, Engel SG, Le Grange D, Mitchell JE, Peterson CB, Simonich HK, Wonderlich SA, & Halmi KA (2012). The Yale-Brown-Cornell eating disorders scale self-report questionnaire: A new, efficient tool for clinicians and researchers. International Journal of Eating Disorders, 45(7), 856–860. 10.1002/eat.22023 [PubMed: 22532411]

Berg KC, Peterson CB, Frazier P, & Crow SJ (2012). Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature: Psychometrics of the EDE and EDE-Q. International Journal of Eating Disorders, 45(3), 428–438. 10.1002/eat.20931 [PubMed: 21744375]

Claudat K, Brown TA, Anderson L, Bongiorno G, Berner LA, Reilly E, Luo T, Orloff N, & Kaye WH (2020). Correlates of co-occurring eating disorders and substance use disorders: A case for dialectical behavior therapy. Eating Disorders, 28(2), 142–156. 10.1080/10640266.2020.1740913 [PubMed: 32301683]

Enders CK, & Bandalos DL (2001). The Relative Performance of Full Information Maximum Likelihood Estimation for Missing Data in Structural Equation Models. Structural Equation Modeling: A Multidisciplinary Journal, 8(3), 430–457. 10.1207/S15328007SEM0803_5

Fairburn CG (2008). Cognitive Behavior Therapy and Eating Disorders. Guilford Press.

Gorrell S, Byrne CE, Trojanowski PJ, Fischer S, & Le Grange D (2022). A scoping review of non-specific predictors, moderators, and mediators of family-based treatment for adolescent anorexia and bulimia nervosa: A summary of the current research findings. Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity. 10.1007/s40519-022-01367-w

Halmi KA (2004). Obsessive-Compulsive Personality Disorder and Eating Disorders. Eating Disorders, 13(1), 85–92. 10.1080/10640260590893683

Halmi KA, Tozzi F, Thornton LM, Crow S, Fichter MM, Kaplan AS, Keel P, Klump KL, Lilenfeld LR, Mitchell JE, Plotnicov KH, Pollice C, Rotondo A, Strober M, Woodside DB, Berrettini WH, Kaye WH, & Bulik CM (2005). The relation among perfectionism, obsessive-compulsive personality disorder and obsessive-compulsive disorder in individuals with eating disorders. International Journal of Eating Disorders, 38(4), 371–374. 10.1002/eat.20190 [PubMed: 16231356]

Jaeger B (2017). Package 'r2glmm.' R Found Stat Comput Vienna Available CRAN R-Project Org/ Package= R2glmm.

Kaye WH, Bulik CM, Thornton L, Barbarich N, & Masters K (2004). Comorbidity of Anxiety Disorders With Anorexia and Bulimia Nervosa. American Journal of Psychiatry, 161(12), 2215–2221. 10.1176/appi.ajp.161.12.2215 [PubMed: 15569892]

Keys A, Brozek J, Henschel A, Mickelsen O, & Taylor HL (1950). The Biology of Human Starvation Volume II. JSTOR.

Law C, & Boisseau CL (2019). Exposure and Response Prevention in the Treatment of Obsessive-Compulsive Disorder: Current Perspectives. Psychology Research and Behavior Management, Volume 12, 1167–1174. 10.2147/PRBM.S211117 [PubMed: 31920413]

Le Grange D, Eckhardt S, Dalle Grave R, Crosby RD, Peterson CB, Keery H, Lesser J, & Martell C (2020). Enhanced cognitive-behavior therapy and family-based treatment for adolescents with an eating disorder: A non-randomized effectiveness trial. Psychological Medicine, 1–11. 10.1017/S0033291720004407

Le Grange D, Hughes EK, Court A, Yeo M, Crosby RD, & Sawyer SM (2016). Randomized Clinical Trial of Parent-Focused Treatment and Family-Based Treatment for Adolescent Anorexia Nervosa.

Journal of the American Academy of Child & Adolescent Psychiatry, 55(8), 683–692. 10.1016/j.jaac.2016.05.007 [PubMed: 27453082]

Le Grange D, & Lock J (2009). Treating bulimia in adolescents: A family-based approach. Guilford.

Le Grange D, Lock J, Agras WS, Bryson SW, & Jo B (2015a). Randomized Clinical Trial of Family-Based Treatment and Cognitive-Behavioral Therapy for Adolescent Bulimia Nervosa. Journal of the American Academy of Child & Adolescent Psychiatry, 54(11), 886–894.e2. 10.1016/j.jaac.2015.08.008 [PubMed: 26506579]

Le Grange D, Lock J, Agras WS, Bryson SW, & Jo B (2015b). Randomized Clinical Trial of Family-Based Treatment and Cognitive-Behavioral Therapy for Adolescent Bulimia Nervosa. Journal of the American Academy of Child & Adolescent Psychiatry, 54(11), 886–894.e2. 10.1016/j.jaac.2015.08.008 [PubMed: 26506579]

Le Grange D, Lock J, Agras WS, Moye A, Bryson SW, Jo B, & Kraemer HC (2012). Moderators and mediators of remission in family-based treatment and adolescent focused therapy for anorexia nervosa. Behaviour Research and Therapy, 50(2), 85–92. 10.1016/j.brat.2011.11.003 [PubMed: 22172564]

Lewin AB, Piacentini J, De Nadai AS, Jones AM, Peris TS, Geffken GR, Geller DA, Nadeau JM, Murphy TK, & Storch EA (2014). Defining clinical severity in pediatric obsessive-compulsive disorder. Psychological Assessment, 26(2), 679–684. 10.1037/a0035174 [PubMed: 24320764]

Lock J (2005). Adjusting Cognitive Behavior Therapy For Adolescents With Bulimia Nervosa: Results Of Case Series. American Journal of Psychotherapy, 59(3), 267–281. 10.1176/appi.psychotherapy.2005.59.3.267 [PubMed: 16370133]

Lock J, Agras WS, Bryson S, & Kraemer HC (2005). A Comparison of Short- and Long-Term Family Therapy for Adolescent Anorexia Nervosa. Journal of the American Academy of Child & Adolescent Psychiatry, 44(7), 632–639. 10.1097/01.chi.0000161647.82775.0a [PubMed: 15968231]

Lock J, Fitzpatrick KK, Agras WS, Weinbach N, & Jo B (2018). Feasibility Study Combining Art Therapy or Cognitive Remediation Therapy with Family-based Treatment for Adolescent Anorexia Nervosa. European Eating Disorders Review, 26(1), 62–68. 10.1002/erv.2571 [PubMed: 29152825]

Lock J, & Nicholls D (2020). Toward a greater understanding of the ways family-based treatment addresses the full range of psychopathology of adolescent anorexia nervosa. Frontiers in Psychiatry, 10. psyh. 10.3389/fpsyt.2019.00968

Madden S, Miskovic-Wheatley J, Wallis A, Kohn M, Hay P, & Touyz S (2015). Early weight gain in family-based treatment predicts greater weight gain and remission at the end of treatment and remission at 12-month follow-up in adolescent anorexia nervosa. International Journal of Eating Disorders, 48(7), 919–922. 10.1002/eat.22414 [PubMed: 26488111]

Mandelli L, Draghetti S, Albert U, De Ronchi D, & Atti A-R (2020). Rates of comorbid obsessive-compulsive disorder in eating disorders: A meta-analysis of the literature. Journal of Affective Disorders, 277, 927–939. 10.1016/j.jad.2020.09.003 [PubMed: 33065835]

Meyer C, Taranis L, Goodwin H, & Haycraft E (2011). Compulsive exercise and eating disorders. European Eating Disorders Review, 19(3), 174–189. 10.1002/erv.1122 [PubMed: 21584911]

Norman GR (1989). Issues in the use of change scores in randomized trials. Journal of Clinical Epidemiology, 42(11), 1097–1105. 10.1016/0895-4356(89)90051-6 [PubMed: 2809664]

Olatunji BO, Tart CD, Shewmaker S, Wall D, & Smits JAJ (2010). Mediation of symptom changes during inpatient treatment for eating disorders: The role of obsessive–compulsive features. Journal of Psychiatric Research, 44(14), 910–916. 10.1016/j.jpsychires.2010.02.011 [PubMed: 20359715]

Scahill L, Riddle MA, McSWIGGIN-HARDIN M, Ort SI, King RA, Goodman WK, Cicchetti D, & Leckman JF (1997). Children's Yale-Brown Obsessive Compulsive Scale: Reliability and Validity. Journal of the American Academy of Child & Adolescent Psychiatry, 36(6), 844–852. 10.1097/00004583-199706000-00023 [PubMed: 9183141]

Schaumberg K, Reilly EE, Anderson LM, Gorrell S, Wang SB, & Sala M (2018). Improving prediction of eating-related behavioral outcomes with zero-sensitive regression models. Appetite, 129, 252–261. 10.1016/j.appet.2018.06.030 [PubMed: 29958864]

Simic M, Stewart CS, Konstantellou A, Hodsoll J, Eisler I, & Baudinet J (2022). From efficacy to effectiveness: Child and adolescent eating disorder treatments in the real world (part 1)—treatment course and outcomes. Journal of Eating Disorders, 10(1), 27. 10.1186/s40337-022-00553-6 [PubMed: 35189967]

Stefini A, Salzer S, Reich G, Horn H, Winkelmann K, Bents H, Rutz U, Frost U, von Boetticher A, Ruhl U, Specht N, & Kronmüller K-T (2017). Cognitive-Behavioral and Psychodynamic Therapy in Female Adolescents With Bulimia Nervosa: A Randomized Controlled Trial. Journal of the American Academy of Child & Adolescent Psychiatry, 56(4), 329–335. 10.1016/j.jaac.2017.01.019 [PubMed: 28335877]

Swinbourne JM, & Touyz SW (2007). The co-morbidity of eating disorders and anxiety disorders: A review. European Eating Disorders Review, 15(4), 253–274. 10.1002/erv.784 [PubMed: 17676696]

Trainor C, Gorrell S, Hughes EK, Sawyer SM, Burton C, & Le Grange D (2020). Family-based treatment for adolescent anorexia nervosa: What happens to rates of comorbid diagnoses? European Eating Disorders Review, 28(3), 351–357. 10.1002/erv.2725 [PubMed: 31995262]

Trottier K, Monson CM, Wonderlich SA, & Olmsted MP (2017). Initial Findings From Project Recover: Overcoming Co-Occurring Eating Disorders and Posttraumatic Stress Disorder Through Integrated Treatment: Integrated CBT for Eating Disorders and PTSD. Journal of Traumatic Stress, 30(2), 173–177. 10.1002/jts.22176 [PubMed: 28398626]

Vall E, & Wade TD (2015). Predictors of treatment outcome in individuals with eating disorders: A systematic review and meta-analysis. International Journal of Eating Disorders, 48(7), 946–971. 10.1002/eat.22411 [PubMed: 26171853]

Von Ranson KM, Kaye WH, Weltzin TE, Rao R, & Matsunaga H (1999). Obsessive-compulsive disorder symptoms before and after recovery from bulimia nervosa. American Journal of Psychiatry, 156(11), 1703–1708. [PubMed: 10553732]

Williams BM, & Levinson CA (2021). Intolerance of uncertainty and maladaptive perfectionism as maintenance factors for eating disorders and obsessive-compulsive disorder symptoms. European Eating Disorders Review, 29(1), 101–111. [PubMed: 33220014]

Public Health Significance Statement

Bulimia nervosa is associated with significant increases in mortality and societal cost, and there is a pressing need for innovations within available treatments for young people with this disorder. In the current study, we explore the extent to which existing evidence-based treatments for adolescent bulimia nervosa are effective in targeting obsessive-compulsive symptoms, a known predictor of treatment response and common co-morbidity in this population.

Table 1.

Demographic Information and Descriptive Statistics

Full Sample (n = 110)				
Variable	***n* (%)**		**Variable**	***n* (%)**
Child Race			**Father Race**	
White	81 (73.6%)		White	75 (68.2%)
Black	6 (5.5%)		Black	7 (6.4%)
Asian	8 (7.3%)		Asian	7 (6.4%)
Native Hawaiian/Pacific Islander	2 (1.8%)		Native Hawaiian/Pacific Islander	1 (0.9%)
More than 1 Race	13 (11.8%)		More than 1 Race	2 (1.8%)
Child Ethnicity			Missing	18 (16.4%)
Hispanic/Latino	27 (24.5%)		**Father Ethnicity**	
Not Hispanic or Latino	83 (75.5%)		Hispanic/Latino	14 (12.7%)
Child Gender			Not Hispanic or Latino	78 (70.9%)
Female	103 (93.6%)		Missing	18 (16.5%)
Male	7 (6.4%)		**Mother Race**	
Family Income			White	88 (80.0%)
<50k/year	35 (30.9%)		Black	3 (2.7%)
50-80k/year	13 (11.8%)		Asian	12 (10.9%)
81-100k/year	12 (10.9%)		Native Hawaiian/Pacific Islander	2 (1.8%)
101-150k/year	20 (18.2%)		More than 1 Race	4 (3.6%)
>150k/year	35 (30.9%)		Missing	1 (0.9%)
Declined	2 (1.8%)		**Mother Ethnicity**	
Missing	6 (5.5%)		Hispanic/Latino	25 (22.7%)
Psychotropic Medication			Not Hispanic or Latino	84 (76.4%)
SSRI	9 (8.2%)		Missing	1 (0.9%)
SNRI	1 (0.9%)		**Comorbid Disorders**	
Tricyclic Antidepressant	1 (0.9%)		Depression	37 (33.6%)
NDRI	1 (0.9%)		Depression NOS	20 (18.2%)
Antianxiety Medication	1 (0.9%)		GAD	17 (15.5%)
Any Medication	10 (9.1%)		OCD	8 (7.3%)

Variable	***M(SD)***	**Range**	ADHD	5 (4.5%)
Child Age (years) (n = 110)	15.80 (1.51)	12-18	Dysthymia	6 (5.5%)
Mother Age (years) (n = 104)	47.01 (6.15)	29-60	ODD	1 (0.9%)
Father Age (years) (n = 84)	50.70 (7.27)	31-75	PTSD	3 (2.7%)
Length of Illness (months) (n = 110)	18.89 (17.16)	3-100	Social Phobia	2 (1.8%)
EDE Global at BL (n = 110)	3.58 (1.30)	0.43-5.73	Tic Disorder	1 (0.9%)
EDE Global at EOT (n = 93)	1.82 (1.46)	0.00-5.65	Anxiety NOS	2 (1.8%)
EDE Global at 6m (n = 70)	1.55 (1.39)	0.00-5.20		
EDE Global at 12m (n = 71)	1.30 (1.17)	0.00-5.37		
YBC Total BL (n = 110)	18.78 (7.16)	0.00-31.00		
YBC Total at EOT (n = 89)	9.16 (8.29)	0.00-26.00		

YBC Total at 6m (n = 70)	8.44 (8.70)	0.00-28.00
YBC Total at 12m (n = 72)	6.26 (7.52)	0.00-29.00

OC Subgroup (n = 59)

Variable	*n* (%)	Variable	*n* (%)
Child Race		**Father Race**	
White	41 (69.5%)	White	40 (67.8%)
Black	4 (6.8%)	Black	4 (6.8%)
Asian	4 (6.8%)	Asian	3 (5.1%)
Native Hawaiian/Pacific Islander	2 (3.4%)	Native Hawaiian/Pacific Islander	1 (1.7%)
More than 1 Race	8 (13.6%)	More than 1 Race	2 (3.4%)
Child Ethnicity		Missing	9 (15.3%)
Hispanic/Latino	12 (20.3%)	**Father Ethnicity**	
Not Hispanic or Latino	47 (79.7%)	Hispanic/Latino	5 (8.5%)
Child Gender		Not Hispanic or Latino	45 (76.3%)
Female	57 (96.6%)	Missing	9 (15.3%)
Male	2 (3.4%)	**Mother Race**	
Family Income		White	47 (79.7%)
<50k/year	18 (30.5%)	Black	1 (1.7%)
50-80k/year	8 (13.6%)	Asian	6 (10.2%)
81-100k/year	6 (10.2%)	Native Hawaiian/Pacific Islander	2 (3.4%)
101-150k/year	11 (18.6%)	More than 1 Race	2 (3.4%)
>150k/year	12 (20.3%)	Missing	1 (1.7%)
Declined	0 (0.0%)	**Mother Ethnicity**	
Missing	4 (6.8%)	Hispanic/Latino	11 (18.6%)
Psychotropic Medication		Not Hispanic or Latino	47 (79.7%)
SSRI	5 (8.5%)	Missing	1 (1.7%)
SNRI	1 (1.7%)	**Comorbid Disorders**	
Tricyclic Antidepressant	0 (0.0%)	Depression	23 (39.0%)
NDRI	0 (0.0%)	Depression NOS	11 (18.6%)
Antianxiety Medication	0 (0.0%)	GAD	11 (18.6%)
Any Medication	5 (8.5%)	OCD	8 (13.6%)

Variable	*M(SD)*	Range	ADHD	3 (5.1%)
Child Age (years) (n = 59)	15.86 (1.46)	12-18	Dysthymia	3 (5.1%)
Mother Age (years) (n = 54)	47.50 (6.59)	29-60	ODD	1 (1.7%)
Father Age (years) (n = 46)	50.54 (7.35)	31-68	PTSD	1 (1.7%)
EDE Global at BL (n = 59)	3.67 (1.36)	0.43-5.73	Social Phobia	0 (0.0%)
EDE Global at EOT (n = 52)	2.17 (1.57)	0.00-5.65	Tic Disorder	1 (1.7%)
EDE Global at 6m (n = 40)	1.81 (1.47)	0.00-5.20	Anxiety NOS	2 (3.4%)
EDE Global at 12m (n = 39)	1.66 (1.27)	0.00-5.37		
CY-BOCS Total BL (n = 59)	15.03 (7.86)	5.00-38.00		
CY-BOCS Total at EOT (n = 52)	6.96 (9.85)	0.00-32.00		
CY-BOCS Total at 6m (n = 41)	7.78 (8.50)	0.00-26.00		
CY-BOCS Total at 12m (n = 39)	7.77 (10.22)	0.00-28.00		

Note: BL = baseline; EOT = end of treatment; EDE = Eating Disorder Examination; CYBOCS = Children's Yale-Brown Obsessive Compulsive Scale; YBC = Yale-Brown-Cornell Eating Disorders Scale; NOS = not otherwise specified; GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder; ADHD = attention deficit-hyperactivity disorder; ODD = oppositional defiant disorder; PTSD = post-traumatic stress disorder

Table 2.

Results from Multi-Level Models (Aim 1) Exploring Changes in General and ED-Specific OC Symptoms Over Treatment and Follow-Up

Multi-level Models													
CY-BOCS Total (General OC Symptoms)							**YBC Total (ED-Specific OC Symptoms)**						
Predictor	***Est.***	***SE***	***df***	***t***	***p***	R^2_p	**Predictor**	***Est.***	***SE***	***df***	***t***	***p***	R^2_p
Intercept	15.64	3.35	110.95	4.67	<.001		Intercept	17.84	2.16	185.62	8.27	<.001	
Time (weeks)	−0.10	0.07	145.78	−1.43	.155	.01	**Time (weeks)**	**−0.15**	**0.04**	**259.07**	**−3.37**	**<.001**	**.03**
Comorbid diagnosis	1.29	1.99	58.81	0.65	.520	.00	Comorbid diagnosis	0.57	1.24	110.69	0.46	.645	.00
Tx group	−2.73	2.05	121.64	−1.34	.184	.01	Tx group	−1.21	1.28	204.87	−0.95	.343	.00
EDE Global BL	**1.87**	**0.66**	**61.56**	**2.83**	**.006**	**.07**	**EDE Global BL**	**2.64**	**0.45**	**113.25**	**5.83**	**<.001**	**.17**
Time x Tx Group	−0.00	0.05	146.17	−0.08	.934	.00	Time x Tx Group	−0.02	0.03	261.89	−0.85	.397	.00
Full Model						.18	Full Model						.48

Note. EDE = Eating Disorder Examination; Tx= Treatment; BL = baseline; EOT = end of treatment; bolded effects are significant at $p < .05$.

Table 3.

Results from Regression Analyses Testing Associations between Changes in OC symptoms in Treatment and Change in EDE Global Scores at 12-month Follow-Up

Regression Analysis									
CYBOCS Change- EDE Global 12-Month FU					**YBC Change- EDE Global 12-Month FU**				
Predictor	***b*-value**	***SE***	***t***	***p***	**Predictor**	***b*-value**	***SE***	***t***	***p***
EDE Global BL	**.45**	**.17**	**2.74**	**.010**	EDE Global BL	.06	.13	0.47	.640
Tx Group	−.34	.39	−0.86	.398	Tx Group	−.18	.24	−0.75	.459
CYBOCS BL	−.01	.03	−0.46	.649	YBC BL	.02	.02	0.70	.489
CYBOCS EOT	.04	.02	1.52	.138	**YBC EOT**	**.08**	**.02**	**4.71**	**<.001**
Overall Model	$F(4, 35) = 3.73$, $p = .014$, Adjusted $R^2 = .24$				Overall Model	$F(4, 63) = 9.46$, $p = <.001$, Adjusted $R^2 = .38$			

Note. EDE = Eating Disorder Examination; Tx= Treatment; BL = baseline; EOT = end of treatment; bolded effects are significant at $p < .05$.

Table 4.

Results from Negative Binomial Regression Analyses Testing Associations between Changes in OC symptoms in Treatment and Change in Binge Eating and Self-Induced Vomiting at 12-Month Follow-Up

DV: Binge Eating Episodes, 12-Month Follow-Up									
CYBOCS Change					**YBC Change**				
Predictor	***Est.***	***SE***	***z***	***p***	**Predictor**	***Est.***	***SE***	***z***	***p***
EDE Binge Ep., BL	0.01	0.05	0.25	.803	EDE Binge Ep., BL	−0.06	0.04	−1.55	.121
Tx Group	0.98	1.02	0.97	.333	Tx Group	−0.66	0.83	0.82	.423
CYBOCS BL	0.03	0.07	0.46	.643	**YBC BL**	**0.22**	**0.09**	**2.50**	**.012**
CYBOCS EOT	0.02	0.06	0.26	.792	**YBC EOT**	**0.14**	**0.05**	**2.70**	**.007**
DV: Vomiting Episodes, 12-Month Follow-Up									
CYBOCS Change					**YBC Change**				
Predictor	***Est.***	***SE***	***z***	***p***	**Predictor**	***Est.***	***SE***	***z***	***p***
EDE Vomiting, BL	**0.07**	**0.01**	**5.56**	**<.001**	**EDE Vomiting, BL**	**0.06**	**0.01**	**5.22**	**<.001**
Tx Group	**1.51**	**0.69**	**2.19**	**.028**	**Tx Group**	**2.01**	**0.66**	**3.04**	**.002**
CYBOCS BL	−0.01	0.04	−0.16	.877	YBC BL	−0.04	0.05	−0.72	.471
CYBOCS EOT	0.09	0.05	1.85	.064	**YBC EOT**	**0.14**	**0.04**	**3.14**	**.002**

Note. EDE = Eating Disorder Examination; Tx= Treatment; BL = baseline; EOT = end of treatment; bolded effects are significant at $p < .05$.