

Exploring Mechanisms of Action in Exposure-Based Cognitive Behavioral Therapy for Eating Disorders: The Role of Eating-Related Fears and Body-Related Safety Behaviors

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Evidence-based cognitive behavioral therapy for eating disorders includes a component of exposure therapy, which involves patients confronting feared eating and body-related stimuli while preventing safety behaviors. With recent research demonstrating that eating-related fears and safety behaviors are central to eating disorder pathology, there is increased emphasis on improving the efficacy of exposure therapy in eating disorders. Doing so will require a better understanding of important mechanisms of action in this treatment. The present study explored how changes during treatment in eating-related fears and avoidance as well as body-related safety behaviors influence overall treatment outcomes. Individuals with eating disorders ($N = 71$) receiving exposure-based treatment completed measures of global eating disorder severity at admission and discharge. Hypothesized mechanisms of action were also assessed at admission and discharge as well as at a 2-week

time point after beginning treatment. Path modeling analyses showed that decreased eating-related cognitions (feared concerns about eating) and emotions (anxiety about eating) at the 2-week time point were prospectively predictive of lowered global eating disorder symptom severity at discharge. Additionally, reduced body checking and avoidance behaviors after 2 weeks of treatment were also associated with lower eating disorder severity at discharge. These findings highlight the importance of exposure-based therapy in eating disorders and the need to uniquely address eating-related fears and safety behaviors.

Keywords: exposure therapy; cognitive behavioral therapy; eating disorders; mechanisms of action

RESPONSIBLE FOR A VARIETY OF MEDICAL COMPLICATIONS, psychosocial impairment, and heightened risk of mortality (Westmoreland, Krantz, & Mehler, 2016), eating disorders (EDs) are severe mental health problems that necessitate swift and effective treatment. Fortunately, several treatment interventions are efficacious in the treatment of EDs. Among these interventions, cognitive behavioral therapy for eating disorders (CBT-ED) has shown the greatest

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amount of empirical support (Hay, 2013; Linardon, Wade, de la Piedad Garcia, & Brennan, 2017; Linardon, 2018) and is recommended as a frontline treatment intervention, according to clinical guidelines published by several national health organizations (e.g., National Institute for Health and Care Excellence [NICE], 2017). In accordance with these clinical guidelines, the term “CBT-ED” is used to encompass the range of different evidence-based forms of CBT for EDs (NICE, 2017).

A key component of CBT-ED is exposure therapy (Weissman et al., 2017), which involves guiding patients in confronting anxiety-evoking stimuli related to eating (e.g., “forbidden” foods) and body image (e.g., form-fitting clothing) while preventing engagement in ED safety behaviors (e.g., compulsive exercising, body checking). A variety of studies over the past several decades have produced somewhat mixed yet promising findings that exposure is often effective both as a component of the CBT-ED “package” and as a standalone intervention (Boswell, Anderson, & Anderson, 2015; Koskina, Campbell, & Schmidt, 2013). Despite this empirical evidence, many clinicians use exposure infrequently in their delivery of CBT-ED, choosing instead to emphasize other techniques (e.g., cognitive restructuring; Turner, Tatham, Lant, Mountford, & Waller, 2014; Waller, Stringer, & Meyer, 2012). However, in light of recent network analytic research demonstrating the central role of eating-related fear and safety behaviors in EDs (Forbush, Siew, & Vitevitch, 2016; Forrest, Jones, Ortiz, & Smith, 2018; Levinson et al., 2017; Smith et al., 2018), there is renewed interest in applying exposure to EDs and improving the effectiveness of the treatment (Boswell et al., 2015; Koskina et al., 2013; Reilly, Anderson, Gorrell, Schaumberg, & Anderson, 2017).

In the field of anxiety disorders and related problems, much work has focused on the mechanisms of change in exposure therapy. This work has led to identification of important change processes in exposure that are associated with superior treatment outcomes (e.g., Kumpula et al., 2017), thereby allowing for improvements to exposure-based treatment interventions. As pointed out by Jansen (2016), the field of EDs would benefit from conducting research of this nature, as it may contribute to improvements in extant CBT-ED interventions. Given the recent increased emphasis on refining exposure-based therapy for EDs in order to maximize treatment effectiveness, there is a need to explore potential mechanisms of action in exposure-based CBT for EDs.

The objective of the present research was to examine several theoretically derived potential mechanisms

of action in a sample of patients receiving exposure-based CBT in a partial hospitalization program for EDs. Global ED symptom severity was assessed at both pretreatment and posttreatment. Potential change mechanisms were also assessed at pretreatment and posttreatment as well as at a 2-week time point in the midst of treatment. Below, we describe the five potential mechanisms of action examined in this study organized into two broader categories: eating-related fear/avoidance and body-related safety behaviors.

EATING-RELATED FEAR/AVOIDANCE

Multiple network analytic studies have converged on the finding that eating-related fears, such as the fear of exorbitant weight gain or inability to tolerate anxiety while eating, are central to eating disorders across diagnoses (Levinson et al., 2017; Smith et al., 2018). In tandem with this key finding, there have been several recent calls for exposure-based interventions to more directly address eating-related fear in order to maximize treatment outcomes (e.g., Murray, Loeb, & Le Grange, 2016). As an example of this, a prior study has shown that greater reductions in eating-related fear over the course of exposure therapy predicted increased caloric intake at a test meal (Steinglass et al., 2012).

Research in the anxiety disorders field has raised questions about mechanisms of change in exposure therapy; namely, whether change on an emotional level (i.e., habituation of feared responding) versus a cognitive level (i.e., violation of feared expectancies) is most influential in treatment outcome (e.g., Vervliet, Craske, & Hermans, 2013). Based on this research, Levinson and Byrne (2015) have highlighted the importance of examining eating-related fear on both an emotional and cognitive, as well as behavioral level. Therefore, in the present study, we assessed the following components of eating-related fear as potential mechanisms of action in exposure-based CBT: (a) anxiety about eating (emotional), (b) food avoidance behaviors (behavioral), and (c) feared concerns about eating (cognitive).

BODY-RELATED SAFETY BEHAVIORS

In contrast to the aforementioned research indicating that feared concerns are central to EDs, the prevailing cognitive behavioral model of EDs posits that overvaluation of body shape and weight are the core pathological features (e.g., Fairburn, Cooper, & Shafran, 2003). Recent empirical work provides robust support for this model (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Forrest et al., 2018). Behavioral manifestations of overvaluation of weight and shape include several body-related “safety behaviors” (i.e., behaviors that reduce

anxiety in the short term and strengthen anxiety in the long-term). In particular, compulsive body checking (e.g., scrutinizing one's physique repeatedly in the mirror) as well as body image avoidance (e.g., dressing in baggy clothing to conceal one's physique) have emerged as the predominant expression of shape and weight overvaluation (Trottier, MacDonald, McFarlane, Carter, & Olmsted, 2015). Indeed, a recent network analysis showed that body checking was the strongest central feature in a large, transdiagnostic ED sample (Forbush et al., 2016), thereby highlighting the importance of body image safety behaviors as targets for treatment. In exposure therapy for EDs, individuals are encouraged to refrain from these body-related safety behaviors while confronting scenarios that evoke body image-related anxiety (e.g., wearing a bathing suit). The overall benefits of exposure therapy may be primarily driven by facilitation of reduced body checking and avoidance behaviors, which could theoretically diminish the intensity of ED patients' overvaluation of body shape and weight. In the present study, we elected to examine body checking and body image avoidance as two additional mechanisms of action in exposure-based CBT.

STUDY OVERVIEW AND HYPOTHESES

A sample of patients ($N = 71$) diagnosed with EDs received treatment in a hospital-based day treatment program that predominantly emphasized exposure-based CBT. Global ED severity was assessed upon admission to the program as well as upon discharge. Variables explored as potential mechanisms of action were assessed upon admission and discharge, as well as at a 2-week time point after admission. Path modeling analyses were used to examine the effects of changes in mechanisms of action variables on overall treatment response.

We predicted that patients would experience significant reductions in ED symptom severity from admission to discharge. Additionally, we hypothesized that the magnitude of reductions in eating-related anxiety, food avoidance behaviors, and feared concerns about eating would be associated with the extent of overall ED symptom relief. We further hypothesized that reductions in body checking and body image avoidance would also be associated with reduced ED symptom severity.

Methods

PARTICIPANTS AND PROCEDURE

Data for the present study were obtained from a group of 122 adults with a principal ED diagnosis who sought treatment in an ED-specialty partial hospitalization (i.e., day treatment) program within a large behavioral hospital between May 2016 and

July 2018. Our data analyses included individuals whose length of treatment was at least 4 weeks, thereby allowing us to explore the effects of our potential change mechanism variables, which were assessed at a 2-week time point within the treatment program. Using this approach allowed for adequate treatment change over time for all three time points. That is, at least 2 weeks of treatment separated each time point. Baseline assessments were used as Time 1. Two-week follow-up was used as Time 2. Assessments completed at discharge (4 weeks or beyond) were used as Time 3. Average length of stay in the program was 5.73 weeks ($SD = 1.84$), range = 4–9 weeks.

A majority of our final sample of patients ($N = 71$) identified as female ($n = 65$; 91.5%) and European American ($n = 65$; 91.5%). Mean age was 28.11 years ($SD = 9.49$), range = 18–57 years. Principal ED diagnoses were determined by program psychiatrists via semistructured interview combined with reviewing patient responses on the EDE-Q at admission (see below) and included the following: bulimia nervosa ($n = 30$, 42.3%), anorexia nervosa, restricting type ($n = 16$, 22.5%), anorexia nervosa, binge-eating/purging type ($n = 10$, 14.1%), binge eating disorder ($n = 8$, 11.3%), avoidant/restrictive food intake disorder ($n = 2$, 2.8%), and other specified or unspecified eating disorder ($n = 5$, 7.0%). Additionally, a majority of the sample was diagnosed with a comorbid anxiety disorder ($n = 44$; 62.0%) or mood disorder ($n = 36$; 50.7%). Most patients ($n = 59$, 83.1%) were prescribed psychotropic medication that was managed by a program psychiatrist.

Within 2 days after their admission to the partial hospitalization program, patients provided informed consent for research and completed the Time 1 study measures that were included within a larger battery of assessment instruments. The treatment program predominantly emphasized exposure-based CBT and encouraged patients to systematically confront feared eating and body-image-related stimuli while preventing engagement in ED safety behaviors. The treatment was primarily facilitated by a program therapist with assistance from a dietitian who aided specifically in the facilitation of exposure to feared foods. Program therapists provided psychoeducation on the role of eating-related fears and avoidance as well as other safety behaviors in the maintenance of ED pathology and worked in collaboration with the patient to develop an exposure hierarchy of various ED-related feared stimuli (e.g., eating in feared scenarios, wearing anxiety-evoking clothing, etc.).

Importantly, study therapists were trained and receiving ongoing supervision to ensure that exposure was implemented in a manner consistent with

recent recommendations from Reilly and colleagues (2017) to incorporate key principles of inhibitory learning theory into the treatment. Accordingly, therapists were encouraged to: (a) facilitate expectancy violation with patients during exposure, (b) vary the level of fear intensity over the course of exposure, (c) vary the contexts in which exposure practices occurred, and (d) make efforts to deepen extinction learning over multiple exposure activities, such as pairing exposure to feared foods with exposure to feared eating scenarios (e.g., eating calorific foods at a large, crowded restaurant).

The treatment program was coordinated on business days (i.e., weekdays) for 6.5 hours each day. Patients met with their therapist daily to: (a) review exposure homework activities completed the previous day, (b) complete therapist-aided exposure in session, and (c) plan further exposure homework activities. In total, the program facilitated patients' engagement in 3 hours of exposure homework activities daily that were supervised by program therapists and other staff. Two meals and two snack times also provided daily opportunities for exposure to feared foods and various eating scenarios (e.g., buffet-style restaurants). Ancillary components of the program comprised a small minority of the overall activity schedule and included: nondirective, supportive psychotherapy in a group format (30 minutes daily), nutritional education groups (twice weekly for 60 minutes), and weekly family therapy sessions to provide education and periodic updates to patients' family members. On their 14th day of the treatment program, patients completed the questionnaires comprising our hypothesized mechanisms of action. Finally, patients completed Time 3 study measures within 2 days of their discharge from the program.

MEASURES

Eating Disorder Examination Questionnaire Version 6.0 (EDE-Q)

The EDE-Q (Fairburn & Beglin, 1994) is a 28-item self-report questionnaire adapted from Eating Disorder Examination interview (Fairburn & Cooper, 1993). The EDE-Q is designed to assess cognitive and behavioral features of eating disorders. Participants use a scale ranging from 0 (*no days*) to 6 (*every day*) to indicate the frequency of experiencing each item (e.g., "Have you had a definite fear that you might gain weight?") during the past 4 weeks. The EDE-Q has previously demonstrated excellent psychometric properties in both clinical and nonclinical samples (Mond et al., 2004). Based on work by Peterson and colleagues (2007), we used the EDE-Q global scale score to characterize overall ED severity at both admission (Time 1) and discharge (Time 3). The

EDE-Q has previously demonstrated excellent test-retest reliability and internal consistency (Luce & Crowther, 1999) and good criterion validity and concurrent validity (Mond et al., 2004). In the current study, internal consistencies for the EDE-Q global scale were good at baseline ($\alpha = .83$) and excellent at discharge ($\alpha = .92$).

Body Checking Questionnaire (BCQ)

The BCQ (Reas, Whisenhunt, Netemeyer, & Williamson, 2002) is a 23-item measure that assesses the frequency of engaging in various body checking behaviors. Participants use a scale ranging from 1 (*never*) to 5 (*very often*) to indicate the frequency of engaging in each type of body checking described (e.g., "I check the diameter of my legs to make sure they're the same size as before."). The BCQ has been shown to have good test-retest reliability, concurrent validity, and internal consistency (Reas et al., 2002). In the current study, the BCQ showed excellent internal consistency at all three time points (α s = .93–.95).

The Body Image Avoidance Questionnaire (BIAQ)

The BIAQ (Rosen, Srebnik, Saltzberg, & Wendt, 1991) is a 19-item measure that assesses the frequency of engaging in avoidant behaviors related to body image anxiety. Participants use a scale ranging from 0 (*never*) to 5 (*always*) to indicate the frequency of engaging in each type of body image avoidance described (e.g., "I wear clothes that will divert attention from my weight."). The BIAQ has previously demonstrated good test-retest reliability, concurrent validity, and internal consistency (Rosen et al., 1991). In the current study, the BIAQ had adequate to good internal consistencies across the three time points (α s = .74–.82).

The Fear of Food Measure (FOFM)

The FOFM (Levinson & Byrne, 2015) is a 23-item measure that assesses eating-related fear and avoidance. Participants are asked to rate items on a 7-point Likert scale (from 1 = *not at all characteristic* to 7 = *very much characteristic*). The FOFM has three subscales: Anxiety about Eating (AE; e.g., "I feel anxious when I eat"), Feared Concerns about eating (FC; e.g., "I worry that eating will make me dissatisfied with my body"), and Food Avoidance Behaviors (FAB; e.g., "I try to avoid eating when I can"). This three-factor version has good factor, convergent, divergent, and incremental validity (Levinson & Byrne, 2015). In the current study, internal consistencies for the AE (α s = .93–.97), FC (α s = .90–.94), and FAB (α s = .86–.89) subscales were good to excellent across the three time points.

DATA ANALYTIC PLAN

Path modeling and indirect effect analyses were conducted using Mplus program Version 8.0 (Muthen, Muthen, Asparouhov, & Nguyen, 2017). The maximum likelihood with robust standard errors (MLR) was used for standardized path estimates, which accounts for missing data and controls for any potential violations of multivariate normality. Model fit was evaluated using the following fit indices: (a) comparative fit index (CFI; Bentler, 1990), (b) Tucker-Lewis incremental fit index (TLI; Tucker & Lewis, 1973), (c) root mean square error of approximation (RMSEA; Steiger & Lind, 1980), and (d) Standardized Root Mean Square Residual (SRMR; Bentler, 1995). Recommendations from Hu and Bentler (1999) were used for evaluating index magnitudes. For the CFI and TLI, values of .95 and above were considered very good and values between .90 and .95 were considered adequate. For the RMSEA and SRMR, values of .05 and lower were considered very good and values between .08 and .05 were considered adequate. A Swain correction factor was conducted for small sample size using RGui for the CFI and RMSEA (Boomsma & Herzog, 2013). First, model fit of the autoregressive path model was tested. After establishing adequate model fit, indirect effects were estimated using bootstrapping with 5000 draws. Indirect effects were significant if confidence intervals (CI) did not include zero. Five models were planned to test the indirect effect of fear of food (three subscales), body checking, and body avoidance on change in eating disorder symptoms over the course of treatment. We controlled for the effect of body mass index (BMI) in all models.

Planned Model 1

First, we tested a path model including baseline (T1) eating disorder symptoms, anxiety about eating at each time point (T1, two-week follow-up [T2], and discharge [T3]), and discharge eating disorder symptoms, controlling for BMI. If significant paths were observed, we tested whether there was an indirect effect of anxiety about eating on change in eating disorder symptoms from baseline to discharge.

Planned Model 2

We tested a path model including T1 eating disorder symptoms, food avoidance behaviors at each time point (T1, T2, T3), and discharge eating disorder symptoms, controlling for BMI. If significant paths were observed, then, we tested whether there was an indirect effect of food avoidance behaviors on change in eating disorder symptoms from baseline to discharge.

Table 1

Descriptive Statistics for Treatment Outcome Variables

	Admission <i>M (SD)</i>	2 weeks <i>M (SD)</i>	Discharge <i>M (SD)</i>	<i>d</i>
BMI	28.47 (11.50)	-	29.11 (11.25)	.06
FOF AE	36.54 (14.18)	29.41 (9.59)	21.61 (10.94)	1.18
FOF FC	39.37 (14.28)	32.61 (9.78)	23.44 (10.75)	1.26
FOF FAB	20.87 (9.35)	15.58 (6.00)	12.20 (6.86)	1.06
BIAQ	45.70 (14.34)	38.87 (15.07)	34.76 (16.23)	.71
BCQ	60.28 (20.58)	57.32 (17.92)	44.44 (14.55)	.89
EDE-Q	3.65 (1.41)	-	1.78 (0.97)	1.54

Notes. All variables (aside from BMI) significantly decreased from admission to discharge ($ps < .001$). BMI = Body mass index; FOF AE = Fear of food, anxiety about eating subscale; FOF FC = Fear of food, feared concerns subscale; FOF FAB = Fear of food, food avoidance behaviors subscale; BIAQ = Body image avoidance questionnaire; BCQ = Body checking questionnaire; EDE-Q = Eating disorder examination-questionnaire.

Planned Model 3

We tested a path model including T1 eating disorder symptoms, feared concerns about eating at each time point (T1, T2, T3), and discharge eating disorder symptoms, controlling for BMI. If significant paths were observed, we tested whether there was an indirect effect of feared concerns on change in eating disorder symptoms from baseline to discharge.

Planned Model 4

We tested a path model including T1 eating disorder symptoms, body checking at each time point (T1, T2, T3), and discharge eating disorder symptoms, controlling for BMI. If significant paths were observed, we tested whether there was an indirect effect of body checking on change in eating disorder symptoms from baseline to discharge.

Planned Model 5

We tested a path model including T1 eating disorder symptoms, body image avoidance at each time point (T1, T2, T3), and discharge eating disorder symptoms, controlling for BMI. If significant paths were observed, we tested whether there was an indirect effect of body image avoidance on change in eating disorder symptoms from baseline to discharge.

Results

DESCRIPTIVE STATISTICS AND PAIRED T-TESTS

See Table 1 for means and standard deviations of all self-report measures at admission, after 2 weeks in the program, and at discharge. Results from paired samples *t*-tests indicated that, aside from BMI¹, all

¹ Based on the helpful suggestion of a reviewer, we analyzed BMI change from admission to discharge via a separate paired-samples *t*-test including only those 26 patients diagnosed with one of the two anorexia nervosa subtypes. Results indicated that BMI increase from admission ($M = 18.06$, $SD = 8.83$) to discharge ($M = 19.22$, $SD = 9.43$) was marginally significant amongst this smaller subset of patients, $t(25) = 1.91$, $p = .09$.

variables significantly reduced from admission to discharge. Effect sizes for these reductions are also listed in Table 1.

EFFECT OF LENGTH OF STAY ON DISCHARGE EATING DISORDER SYMPTOMS

As length of stay in the program varied between patients, we assessed whether it would be necessary to control for length of stay in subsequent path modeling analyses. We found that there was no significant association between length of stay and global ED severity at discharge ($p = .26$) while controlling for global ED severity at admission. Accordingly, length of stay was not included in the five path models we conducted.

MODEL 1: ANXIETY ABOUT EATING

Please see Figure 1 for full model results for fear of food. Model fit was adequate to excellent (CFI = 0.99, TLI = 0.93, RMSEA = 0.13, SRMR = 0.02). Model fit remained adequate to excellent, though slightly improved when using Swain R correction for small sample size (CFI = 0.99, RMSEA = 0.12). T1 eating disorder symptoms ($b^* = .11, p = .049$)

significantly predicted T2 anxiety about eating, while controlling for BMI ($p = .744$) and baseline anxiety about eating ($p < .001$). In addition, T2 anxiety about eating ($b^* = .48, p < .001$) significantly predicted T3 eating disorder symptoms, while controlling for BMI ($p = .094$) and T1 eating disorder symptoms ($p = .475$). There was no significant indirect effect of anxiety about eating ($p = .152, 95\% \text{ CI } [-0.01, 0.08]$) from admission to discharge eating disorder symptoms. Thus, although change in anxiety about eating did not mediate the observed change in global ED symptom severity over the course of treatment, lower anxiety about eating at the two-week time point was predictive of decreased ED severity at discharge.

MODEL 2: FOOD AVOIDANCE BEHAVIORS

Model fit was excellent (CFI = 1.00, TLI = 1.07, RMSEA = 0.00, SRMR = 0.01). Model fit remained excellent when using Swain R correction for small sample size (CFI = 1.00, RMSEA = 0.00). T1 eating disorder symptoms ($b^* = .17, p = .035$) significantly predicted T2 food avoidance behaviors, while controlling for BMI ($p = .724$) and baseline food

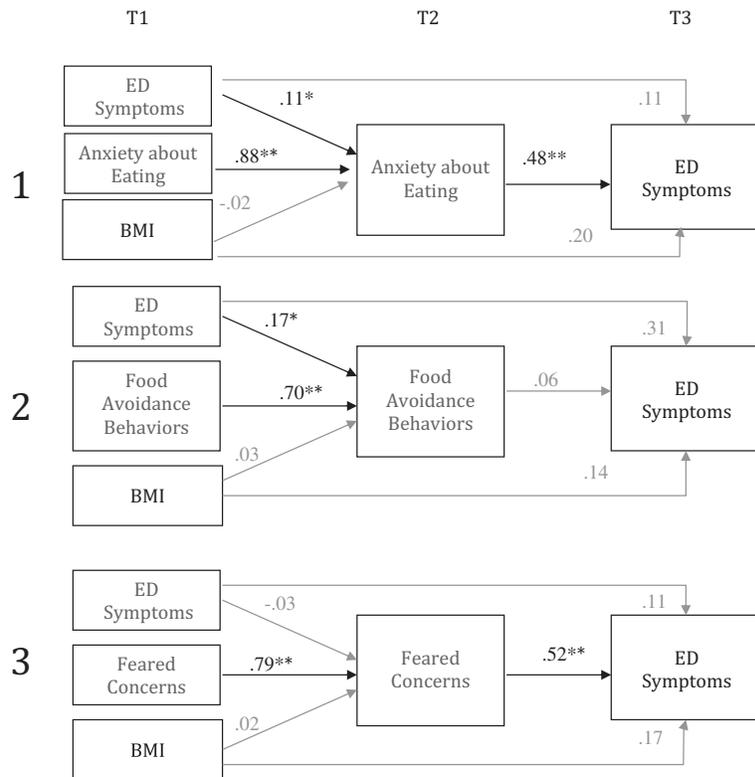


FIGURE 1 Path model of the relationships between eating disorder (ED) symptoms, and each of the three subscales of the Fear of Food Measure: Model 1) Anxiety about Eating, Model 2) Food Avoidance Behaviors, and Model 3) Feared Concerns), while controlling for Body Mass Index (BMI). T1 = Time 1; T2 = Time 2; T3 = Time 3; ** = $p < .001$, * = $p < .05$.

avoidance behaviors ($p < .001$). However, T2 food avoidance behaviors ($p = .683$) did not significantly predict T3 eating disorder symptoms. There was no significant indirect effect of food avoidance behaviors ($p = .861$, 95% CI [-0.04, 0.05]) from admission to discharge eating disorder symptoms. Thus, frequency of food avoidance behaviors appeared to be unassociated with global ED severity at discharge.

MODEL 3: FEARED CONCERNS ABOUT EATING

Model fit was poor to excellent (CFI = 0.97, TLI = 0.75, RMSEA = 0.18, SRMR = 0.02). Model fit remained poor to excellent, though slightly improved when using Swain R correction for small sample size (CFI = 0.97, RMSEA = 0.18). T1 eating disorder symptoms ($p = .748$) did not significantly predict T2 feared concerns about eating. T2 feared concerns ($b^* = .52$, $p < .001$) significantly predicted T3 eating disorder symptoms, while controlling for BMI ($p = .184$) and T1 eating disorder symptoms ($p = .398$). There was no significant indirect effect of feared concerns ($p = .775$, 95% CI [-0.08, 0.06]) from admission to discharge eating disorder symptoms. Thus, although change in feared concerns about eating did not mediate the observed change in global ED symptom severity over the course of treatment, lower feared concerns about eating at the two-week time point was predictive of decreased ED severity at discharge.

MODEL 4: BODY CHECKING

Please see Figure 2 for full model results. Model fit was adequate to excellent (CFI = 0.97, TLI = 0.90, RMSEA = 0.11, SRMR = 0.04). Model fit remained adequate to excellent when using Swain R correc-

tion for small sample size (CFI = 0.97, RMSEA = 0.11). T1 eating disorder symptoms ($p = .689$) did not significantly predict T2 body checking, while controlling for T1 BMI ($p = .005$). T2 body checking ($b^* = .48$, $p < .001$) significantly predicted T3 eating disorder symptoms, while controlling for T1 eating disorder symptoms ($p = .613$). There was no significant indirect effect of body checking ($p = .324$, 95% CI [-0.04, 0.13]) from admission to discharge eating disorder symptoms. Thus, although change in body checking did not mediate the observed change in global ED symptom severity over the course of treatment, lower body checking at the two-week time point was predictive of decreased ED severity at discharge.

MODEL 5: BODY IMAGE AVOIDANCE

Please see Figure 3 for full model results. Model fit was excellent (CFI = 1.00, TLI = 1.04, RMSEA = 0.00, SRMR = 0.01). Model fit remained excellent when using Swain R correction for small sample size (CFI = 1.00, RMSEA = 0.00). T1 eating disorder symptoms ($p = .671$) did not significantly predict T2 body image avoidance, while controlling for T1 BMI ($p = .011$) and T1 body image avoidance ($p < .001$). T2 body image avoidance ($b^* = .61$, $p = .001$) significantly predicted T3 eating disorder symptoms, while controlling for T1 eating disorder symptoms ($p = .309$). There was no significant indirect effect of body image avoidance ($p = .695$, 95% CI [-0.08, 0.05]) from admission to discharge eating disorder symptoms. Thus, although change in body image avoidance did not mediate the observed change in global ED symptom severity over the course of treatment, lower body image avoidance at the two-week time point was predictive of decreased ED severity at discharge.

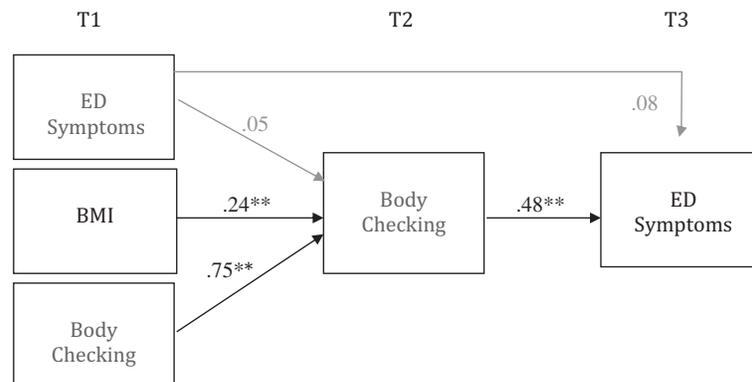


FIGURE 2 Path model of the relationship between eating disorder (ED) symptoms and Body Checking, while controlling for Body Mass Index (BMI). T1 = Time 1; T2 = Time 2; T3 = Time 3; ** = $p < .001$.

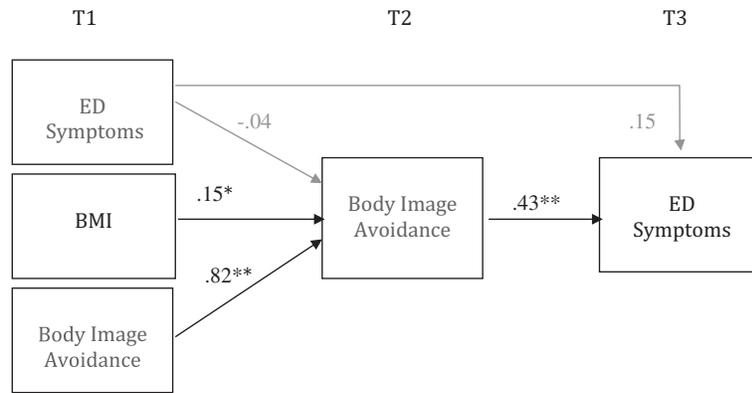


FIGURE 3 Path model of the relationship between eating disorder (ED) symptoms and Body Image Avoidance, while controlling for Body Mass Index (BMI); T1 = Time 1; T2 = Time 2; T3 = Time 3; ** = $p < .001$, * = $p < .05$.

Discussion

Despite recent research underscoring the applicability of exposure therapy to EDs, as well as the efficacy of CBT approaches that emphasize exposure, there is a paucity of work examining mechanisms of action in this treatment. Identifying such mechanisms of action in exposure-based CBT will allow for more targeted applications of this treatment that may improve the overall reduction in ED symptoms that patients experience (Levinson, Vanzhula, & Brosof, 2018). The present study was, to our knowledge, the first exploration of several variables as potential change mechanisms that have previously been theorized to be influential in the treatment of EDs.

In a sample of patients completing at least 4 weeks of exposure-based CBT for EDs in a hospital-based day treatment program, we found that patients experienced substantial reductions in global ED severity as well as other associated ED features (e.g., eating-related fears/avoidance, body image safety behaviors) from admission to discharge. We believe this finding itself has very important clinical implications. Although CBT-ED containing a component of exposure therapy is the established treatment of choice for EDs, many clinicians “shy away” from exposure (Turner et al., 2014; Waller et al., 2012). Deemphasizing exposure in CBT-ED may at least partly explain why a sizeable proportion of patients derive minimal benefit from CBT-ED despite a number of modifications intended to improve treatment outcome over the past several decades (Agras, Fitzsimmons-Craft, & Wilfley, 2017).

With regard to exploration of potential mechanisms of action in exposure-based CBT for EDs, our data analyses revealed some important direct effects of hypothesized change variables on overall

treatment outcome. Specifically, two of the three FOFM subscales that measure both eating-related anxiety and concern about feared eating-related outcomes prospectively predicted global ED severity; lower scores on these two subscales at the 2-week time point (Time 2) was predictive of decreased global ED severity (i.e., lower EDE-Q global scale scores) at discharge (Time 3). Additionally, there were similar findings with regard to body-related safety behaviors. Lower Time 2 frequency of body checking and body image avoidance behaviors prospectively predicted lowered global ED severity at discharge. This finding mirrors work in the field of anxiety disorders, suggesting that safety behaviors play an influential role in treatment outcome for clinical anxiety (Blakey & Abramowitz, 2016). Given the temporal precedence that was established between these variables in our path models, it can be reasonably inferred that reducing eating-related anxiety and feared concerns as well as body checking and avoidance has a helpful impact on lowering global ED severity over the course of exposure-based CBT.

In weighing the relative strength of these direct effects, reduced feared concerns about eating exerted the most impact on global ED severity at discharge ($\beta = .52$), closely followed by anxiety about eating and body checking (both β s = $.48$). Given that these variables span cognitive, behavioral, and affective domains, it is important that clinicians simultaneously pursue reductions in these domains and assess for the success of this periodically throughout the course of CBT-ED. A recent meta-analysis showed that several leading treatment interventions for anorexia nervosa (including CBT) demonstrated no reliable advantage over treatment-as-usual interventions in reducing cognitive and affective features of anorexia (Murray,

Quintana, Loeb, Griffiths, & Le Grange, 2018). It is thus imperative that we tailor CBT-ED to more directly address these influential variables, specifically targeting cognitive and affective aspects. Again, increasing emphasis on exposure therapy appears to be a viable way forward in this regard.

Contrary to prediction, our path models yielded no significant indirect effects of the hypothesized mechanisms of action on change in global ED symptom severity from admission to discharge. Although the path model assessing whether global ED symptom reduction from admission to discharge was mediated by reduced anxiety about eating approached statistical significance, our relatively small sample size likely undermined our power to detect this indirect effect. It can be reasonably argued that a larger sample size likely would have yielded a statistically significant indirect effect, whether for anxiety about eating or other mechanisms of action we assessed. We hope that future, larger powered studies, will assess if this potential mechanism of action mediates change. At a minimum, we believe that the present findings will generate more targeted hypotheses as to which mechanisms of action may play a critical role in influencing global ED symptom reduction. Future research studies with larger sample sizes may provide a more rigorous test of these hypotheses.

The treatment effect sizes in the present study were quite large across our key outcome variables (*ds* range .71–1.54). Although these effects could be accounted for by a number of factors, we believe it is defensible to posit that the “infusion” of key inhibitory learning theory principles into exposure per the recommendations of Reilly and colleagues (2017) was at least partly responsible for the large treatment effects. Also, whereas these large effects were derived from a group of patients who received a relatively high “dosage” of exposure, previous work has revealed smaller treatment less exposure (Farrell et al., in press; Levinson & Byrne, 2015). Taken together, this work suggests the presence of “dose-response” relationship with regard to exposure therapy and overall ED symptom severity; a higher volume of the treatment appears to be associated with more robust symptom relief. Accordingly, given the tendency for there to be variations in CBT-ED “packages” (Waller et al., 2012), these findings contribute to the growing argument to strengthen CBT-ED with a higher concentration of exposure therapy (e.g., Becker & Waller, 2017). We hope future research will test if such a dose-response relationship exists.

There are several limitations to the present study. First, the relatively small size of our patient sample likely contributed to our path modeling analyses

being insufficiently powered to detect significant indirect effects (while still able to detect direct prospective effects) of hypothesized mechanisms of action on treatment outcome. Based on the pattern of results observed in our study, it seems plausible that a similar study with a larger sample size would provide better clarity as to which change mechanisms were most influential in driving overall therapeutic benefit. Additionally, given that our data were collected in the design of an effectiveness study to examine treatment outcome in a “real-world setting,” the lack of internal validity of the study raises doubt over whether the exposure-based component of the program was primarily responsible for patients’ therapeutic benefit. Although the program emphasized exposure-based CBT as the chief therapeutic modality, it cannot be ruled out that other ancillary program components, such as weekly family therapy sessions or nutrition education groups, accounting for a significant portion of the overall therapeutic benefit observed. Future research may consider assessing mechanisms of change in exposure-based CBT for EDs as a purely standalone treatment intervention. Although our study explored the influence of several hypothesized mechanisms of action that were derived from empirical research, these mechanisms are largely unique to EDs. As such, the present study did not take into account the potential influence that broader, transdiagnostic variables (e.g., experiential avoidance, rumination; Rawal, Park, & Williams, 2010) may have on treatment outcome. Future research should examine the influence of both ED-specific and transdiagnostic variables that are theorized to play an important role in influencing ED treatment outcomes.

Strengths of the present study include our use of a diagnostically heterogeneous sample of individuals with EDs, the high degree of external validity inherent in our effectiveness study design, and the examination of a number of variables as theoretically derived mechanisms of action. This study represents the first of its kind to explore potential mechanisms of action in the context of exposure-based CBT for EDs. We identified variables known from previous research to be central to the maintenance of EDs and demonstrated that several of these variables directly impacted treatment outcome in a naturalistic treatment setting. Specifically, after 2 weeks of treatment, lowered eating-related anxiety, feared concerns about eating, body checking, and body image avoidance all contributed to decreased global ED severity at discharge. Our use of path modeling analyses allowed us to account for multicollinearity between variables we hypothesized to influence treatment outcome. It is

our hope that this study will serve as a catalyst for further similar research aimed at identifying important mechanisms of action in exposure-based CBT for EDs. Doing so will allow for further refinement of the treatment to uniquely target critical variables that influence treatment outcome.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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