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Exposure-Based Family Therapy (FBT-E): An Open Case Series of a New Treatment for Anorexia Nervosa

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The overlap between anorexia nervosa (AN) and anxiety disorders has led to the development of anxiety-based etiological models of AN and anxiety-based interventions for AN, including exposure treatment. Family-based treatment (FBT) is an efficacious intervention for adolescents with AN; however, it has recently been proposed that FBT accomplishes parent-facilitated exposure and habituation to food and related triggers in the individual's natural environment. FBT was recently altered to include an explicit exposure component that targets the broad construct of anxiety, including fear, worry, and disgust. This case series examines the application of FBT with an exposure component (FBT-E) to a group of adolescents meeting diagnostic criteria for AN (n = 4) and eating disorder not otherwise specified–restricting type (SAN, n = 6). Ten outpatients (ages 12–17, mean age: 15.28) participated in a course of FBT-E. Session-by-session weight was examined, along with BMI at pre- and posttreatment and responses to self-report measures of eating disorder symptoms (Eating Disorder Examination Questionnaire; EDE-Q), depression and anxiety. Parent reports of their adolescents' anxiety were also collected. The results of this study provide preliminary evidence that FBT-E may effectively target disordered eating and anxiety symptoms and may be a viable alternative to traditional FBT. Implications and future directions are discussed.

Anxiety and Anorexia Nervosa

Anorexia nervosa (AN) is a chronic, severe condition that typically begins in adolescence (Hoek & Hoeken, 2003) and evidences poor treatment outcome, particularly among adults (Keel & Brown, 2010). A clear relationship exists between AN and anxiety disorders. AN is highly comorbid with anxiety disorders (Godart et al., 2003), and these disorders overlap in clinical phenomena such as perfectionism, rigidity, compulsivity, and harm avoidance (Collier & Treasure, 2004; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Strober, 2004), which may reflect a shared genetic vulnerability among individuals with these pathologies (Bulik, Slof-Op't Landt, van Furth & Sullivan, 2007; Halmi et al., 2005; Keel, Klump, Miller, McGue, & Lacono, 2005). Furthermore, anxiety (e.g., fear and worry about food) and avoidance behaviors such as severe dietary restriction are core features of AN. The similarities between AN and anxiety disorders have important implications for etiological theories and subsequent treatments of AN. For instance, etiological models of anxiety disorders may be used as a conceptual basis for etiological models of AN (Strober, 2004). AN treatment may be enhanced by targeting the anxiety experienced by these individuals (Hildebrandt, Bacow, Markella, & Loeb, 2012), and techniques that effectively target anxiety (e.g., exposure) may be a component of effective treatments for AN (Steinglass et al., 2011).

[1] Video patients/clients are portrayed by actors.

Keywords: anorexia nervosa; family-based treatment; exposure treatment; anxiety; adolescents

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Theoretical Models of Anxiety and AN

Several theoretical models of AN have been proposed based on the relationship between AN and anxiety, and each of these models has important treatment implications. Strober's (2004) fear conditioning model of AN posits a common etiology among anxiety disorders, anxious temperament, and eating disorders that centers on abnormal neurobiological functioning of structures that regulate emotional behaviors. According to this model, individuals with AN, similar to individuals with anxiety disorders, evidence neurobiological abnormalities that result in rapid fear conditioning to nonthreatening stimuli (e.g., food) and avoidance of these feared stimuli (e.g., food avoidance). In AN patients, this subsequently leads to weight loss. Behavioral avoidance coupled with an increased resistance to fear extinction maintains eating disorder pathology and accounts for the treatment-resistant nature of this illness. Exposure techniques are widely used to extinguish

conditioned fear responses among individuals with anxiety disorders (Antony & Barlow, 2002; Ougrin, 2011). Therefore, a fear conditioning model of AN logically suggests that exposure-based techniques may be used to treat this population (Steinglass et al., 2011). In accordance with this, exposure and response prevention (EXRP) treatment for AN has been developed and pilot-tested in this population; however, the results have been mixed. Specifically, adults with AN who received EXRP reported a reduction in food-related anxiety posttreatment; however, this did not correspond to a significant increase in caloric intake (Steinglass et al., 2012). This suggests that a fear conditioning model of AN may not adequately explain AN pathology, and therefore traditional exposure treatment may not fully target the core anxiety processes maintaining this disorder.

In contrast to the fear conditioning model, Pallister and Waller (2008) proposed a shared cognitive model of eating and anxiety disorders. This model asserts that pathological functioning results from an individual's schemas about the world ("the world is unsafe") and self ("I'm vulnerable"; "I'm unable to cope"), which, in the presence of environmental triggers (e.g., food), elicits cognitions about the individual's perceived vulnerability (e.g., "this food is dangerous"; "this food will make me fat") and the need for harm avoidance. These cognitions elicit anxiety, which then prompts the individual to engage in cognitive and behavioral strategies to prevent a feared consequence (e.g., rapid weight gain) or to avoid anxiety-evoking cognitions and the accompanying affect. These strategies are hypothesized to reinforce eating pathology; though these behaviors may reduce an individual's anxiety in the short term, they likely maintain the underlying schema. Anxiety is further maintained by attentional biases towards threatening stimuli, as this increases the detection rate of these stimuli and, consequently, overall levels of anxiety (Siep, Jansen, Havermans, & Roefs, 2011). Based on this cognitive model, AN treatment should focus on challenging underlying cognitions relating to perceived vulnerability and harm avoidance via techniques including behavioral experiments, reduction of safety behavior, and cognitive restructuring. Recent expansions of this cognitive model utilize methods such as cognitive remediation to increase cognitive flexibility and correct information processing biases (Abbate-Daga, Buzzichelli, Marzola, Amianto, & Fassino, 2012; Macleod, 2012). These cognitively focused treatment approaches are commonly utilized in recent models of cognitive behavioral therapy (CBT) for AN (Murphy, Straebler, Cooper, & Fairburn, 2010) and have some established efficacy (Shafran, Lee, Cooper, Palmer, & Fairburn, 2008); however, similar to EXRP, findings on CBT treatment for AN have been mixed (Wilson, Grilo, & Vitousek, 2007).

Hildebrandt, Bacow, Markella, & Loeb (2012) proposed a broad anxiety-based model for AN, which focuses on a distinct typology of anxious emotions. Figure 1 summarizes the integrated model of anxiety. Fear operates under conditions of proximal threat and is associated with significant autonomic arousal and preparation for immediate action (Misslin, 2003). Worry develops under conditions of distal threat and/or high degree of uncertainty about the presence of the threat and physiological responses attenuate (Hoehn-Saric & McLeod, 2000; Starcevic & Berle, 2006). Disgust can be operationalized as the characteristic aversive response to distasteful, noxious, or unpleasant stimuli that pose threat in a range of domains from disease to toxicity and morality (Chapman & Anderson, 2012). Of these three emotions, disgust is the least well understood with regard to its phenomenology, its role in AN pathology, and its treatment. Research suggests that disgust is a distinct emotion with unique psychophysiological and neurobiological characteristics, including decreased heart rate (de Jong, van Overveld, & Peters, 2011), distinct facial expressions involving activation of the levator labii muscle (Cisler, Olatunji, & Lohr, 2009), and increased activation of the insula (Fusar-Poli et al., 2009). Though the role of disgust in AN is not fully understood, neuroimaging research has found increased activation of the anterior insula in AN patients in response to food stimuli (Kaye, 2008; Nunn, Frampton, Fuglset, Torzsok-Sonnevend, & Lask, 2011), suggesting that disgust may play a prominent role in this pathology and therefore may be an important treatment target.

The proposed model by Hildebrandt, Bacow, Markella, & Loeb (2012) also highlights the role of reward processing in maintaining avoidance behaviors. As indicated in Figure 1, processing of threats from any of five relevant domains (food; eating; interoceptive cues; shape and weight; and social evaluation) can lead to an interoceptively driven aversive response, an emotionally primed impulsive response, or both depending on the complexity of the trigger, environmental context, and specific learning history associated with the trigger. This avoidance may become highly reinforced either due to specific or general deficits/hypersensitivity in motivation-reward system (Keating, 2010). The emerging neuroscience of reward processing in AN suggests sensitivity to both pain and pleasure among patients (Keating, Tilbrook, Rossell, Enticott, & Fitzgerald, 2012) that may involve inability to inhibit sensory information (Bar, Berger, Schwier, Wutzler, & Beissner, 2013). Similarly, anticipatory processing may be overactive in contexts or triggers that signal a high degree of uncertainty (Frank, Roblek, et al., 2012). The sum effects of these abnormalities are a motivational state characterized by a high probability of favoring short-term avoidance over long-term gain.

According to the broad anxiety model, avoidance strategies emerge to manage the level of threat cued by

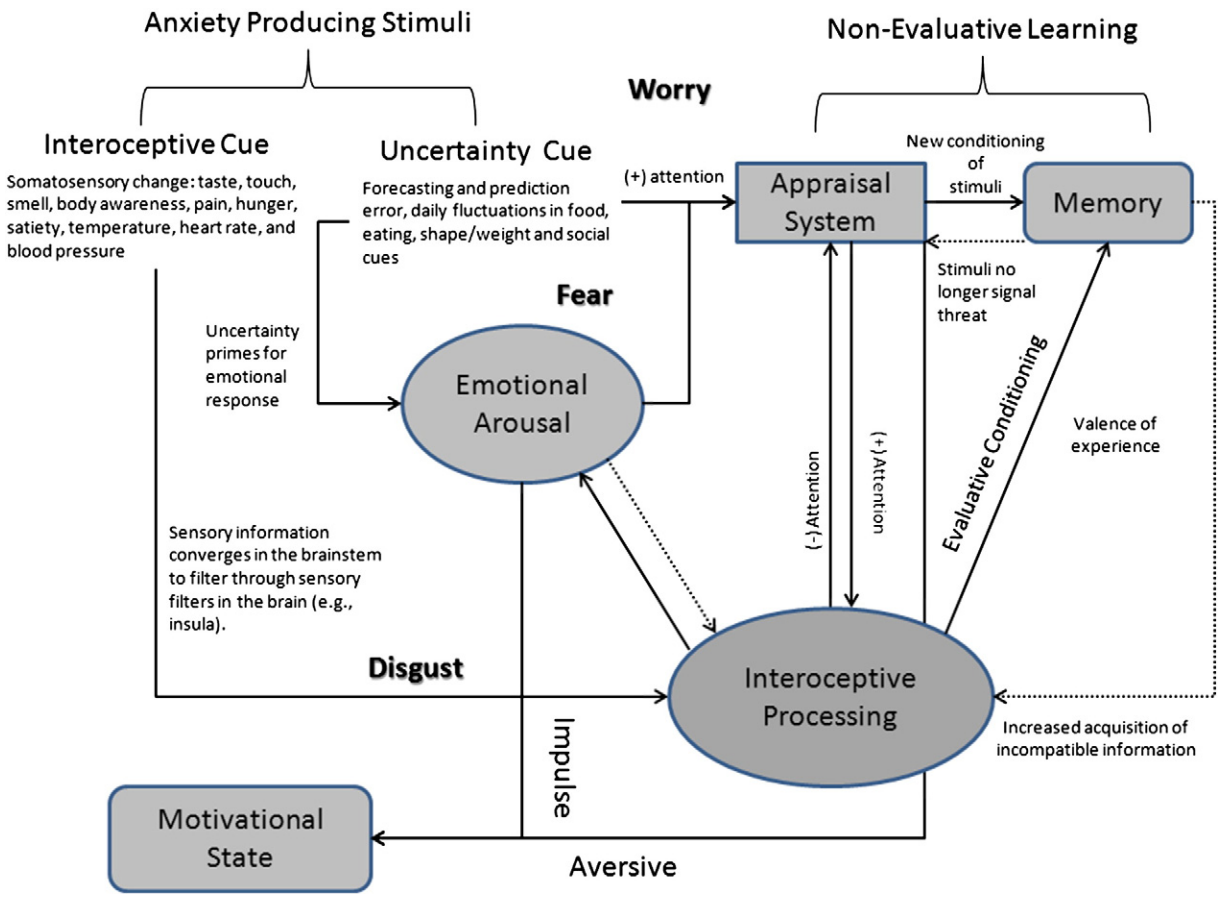


Figure 1. A model of fear, disgust, and worry in anorexia nervosa.

the specific trigger and its environment. These strategies encompass a wide range of behaviors including prevention strategies, safety behaviors, and compulsions. Such activities maintain anxiety because they reduce opportunities for alternate experiences that would challenge the individual's food and weight-related concerns (e.g., the belief that eating a brownie leads to weight gain), or lead to erroneous attributions. Specifically, nonoccurrence of the feared outcome (e.g., weight gain) is attributed to implementation of the safety behavior or ritual, rather than the improbability of the event occurring. In adolescents with AN, avoidance behaviors are often unintentionally reinforced by the individuals' parents, siblings, and peers who may initially support their child's avoidance of unhealthy foods and may continue to do so, after progression of the illness, due to fear of upsetting the individual. This motivation is understandable because initial encouragement to reduce avoidance would yield significant increases in anxiety. As the illness progresses, the individual's motivation to engage in normative activities (e.g., socialization; school activities) often decreases, while motivation to engage in food avoidance and other disordered eating behaviors increases, which further maintains eating pathology. It is hypothesized that anxiety generalizes to other triggers through associative learning (e.g., all calorically dense foods begin to elicit anxiety), consistent with the Strober (2004) hypothesis of rapid fear conditioning in this population.

Treatment Implications for Broad Anxiety Model

As discussed above, the predominant fear conditioning and shared cognitive anxiety models for AN rely on different techniques to reduce food avoidance and associated anxiety, but have limited efficacy. In contrast, family-based treatment (FBT) for adolescent AN has demonstrated efficacy (Eisler et al., 2000; Le Grange, Eisler, Dare, & Russell, 1992; Lock, Agras, Bryson, & Kraemer, 2005) as a purely outpatient treatment (Eisler et al., 2000; Le Grange et al., 1992; Lock et al., 2005; Loeb et al., 2007) as well as a post-hospitalization intervention to complete weight gain and prevent relapse (Russell, Szmukler, Dare, & Eisler, 1987). Follow-up studies also indicate that FBT produces durable results (Eisler et al., 1997; Eisler, Simic, Russell, & Dare, 2007; Lock, Couturier, & Agras, 2006). FBT's efficacy for individuals with the

highest degree of obsessive-compulsive symptoms, however, may be attenuated. FBT may work better than adolescent-focused therapy for these individuals (Le Grange et al., 2012); however, they require a 20-session, 1-year-long version of FBT (i.e., "long-term" treatment) rather than a 10-session, 6-month-long version of FBT (i.e., "short-term" treatment) to obtain this significant effect (Lock et al., 2005). This suggests that FBT may be enhanced via incorporation of additional treatment strategies targeting anxiety and avoidance behaviors.

Theoretical models of FBT consider family-level interventions primary and the proposed therapeutic mechanisms of action are not *explicitly* linked to anxiety (Loeb et al., 2012). In contrast, we proposed the broad anxiety model in efforts to explain the documented efficacy of FBTs for adolescents with AN (Hildebrandt, Bacow, Markella, & Loeb, 2012). We argued that effective FBT achieves intensive food exposure in the patient's natural environment with the aid of reduced uncertainty via increased parental structure around food/eating. Our broad-based anxiety model directly implicates the individual's anxiety in the pathogenesis and maintenance of AN, which is a point of divergence from FBT.

We have translated the Hildebrandt, Bacow, Markella, & Loeb (2012) broad anxiety model into a unique treatment, exposure-based Family Behavioral Therapy (FBT-E), that focuses on the use of exposure techniques and parental de/incentivization as well as direct delivery of adolescent coping skills to reduce symptoms of adolescent AN. Thus, this treatment incorporates some of the core components of FBT (enlisting parents to take charge of change process and reduction of patient/parent blame for eating disorder symptoms), but offers more explicit targets and instruction for addressing the anxiety-specific mechanisms that maintain the disorder. Most notably, FBT-E provides a conceptual model for the disorder; directs parents to intervene on the level of anxiety with a range of exposure techniques, including the use of an exposure model to guide refeeding; provides explicit homework assignments for the individual, patient, and family; and targets eating disorder and body image symptoms within the anxiety-exposure framework.

The expanded exposure framework for FBT-E matches exposure techniques to the proposed typology of anxious emotions. The existing EXRP interventions for AN involve the basic process of exposure to anxiety-provoking triggers (e.g., food) coupled with reduction in maladaptive avoidance responses (restriction, rituals, etc.; Steinglass et al., 2012). In EXRP exercises, exposure hierarchies are used and Subjective Units of Distress (SUDS) ratings are collected to document habituation. Patients and parents are asked to self-monitor these changes at meals between sessions and are expected to climb the hierarchy through the course of treatment. Our model further specifies that exposure should broadly target the individual's fear, worry, and disgust responses to the trigger, which may require adaptations of the traditional EXRP framework. Specifically, targeting worry may require specific worry exposure techniques (e.g., to address catastrophic thinking about future shape and weight changes; Hoyer et al., 2009). Disgust treatment may necessitate environmentally based exposures of greater frequency, duration, and intensity than traditional EXRP because research suggests that disgust may habituate more slowly than fear (Olatunji, Smits, Connolly, Willems, & Lohr, 2007; Viar-Paxton & Olatunji, 2012) and conditioning of the neutral stimulus occurs via interoceptive-driven learning. This type of learning has distinct characteristics; its acquisition doesn't require higher-order cognitive processing and its extinction may require an explicit change in the interoceptive experience.

The broad anxiety model of FBT-E also links anxiety to a motivational system that favors short-term avoidance over long-term functioning. In FBT-E parents are given psychoeducation about this link and asked to address the increased motivation to engage in avoidance by reducing familial and peer reinforcement of eating disorder behaviors and incentivizing alternative non-eating-disorder behaviors. Parents are directed to do this in a firm and empathetic manner. Ideally, this will allow the individual and family to establish a broader range of experiences that combat anxiety associated with AN. By positioning parents to decrease their child's access to avoidance and incentivize other (positive) life experiences (e.g., access to friends, meaningful leisure, etc.), parents can create positive associations between anxious triggers (eating, weight gain, etc.) and naturally rewarding experiences. This counter-conditioning may function not only to motivate adolescents to face their anxiety, but also facilitate extinction by changing the value of the trigger (rather than relying only on habituation to anxious emotion).

The expected time course of FBT-E is three phases and 20 sessions, similar to the intensive version of FBT. Both treatments focus on weight gain in Phase I, although through different mechanisms. In Phase II, both FBT and FBT-E focus on transfer of responsibility for feeding and weight management back to the adolescent. FBT-E, however, does this through continuation of exposure exercises and use of five different modules designed to address persistent eating disorder symptoms. Phase III of FBT-E is designed to be a test of the patient and family's mastery over anxiety and their ability to use what they have learned to prevent relapse or reduce any residual symptoms.

In this case series, we describe a pilot open trial of FBT-E utilized with a group of adolescents diagnosed with AN or subthreshold AN (i.e., SAN), which falls under the category of eating disorder not otherwise specified (i.e., EDNOS).

Method

Participants

Participants were 10 female adolescents presenting to a hospital-based outpatient program specializing in eating disorder treatment. Four participants met criteria for AN outlined in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994). Of the 4 participants who met full DSM-IV criteria for AN, 2 presented with AN binge-eating/purging type, and 2 presented with the restricting type of the disorder. The remaining 6 participants presented with subthreshold anorexia nervosa (SAN). EDNOS (i.e., SAN and other subsyndromal variants of an eating disorder) was assigned if full DSM-IV criteria for AN was not met, but the adolescent clearly had a restrictive-type eating disorder in which weight loss was a prominent feature. Specifically, SAN was defined in one of two ways: weight loss to below 100% ideal body weight but above the 85% cutoff for AN, plus secondary amenhorrea, or weight loss to below the 85% cutoff plus oligomenhorrea (see Loeb, Lock, Le Grange, & Greif, 2012, for a discussion of the diagnostic features of SAN). Of note, SAN is quite common; one study found partial syndromes of eating disorders in a community sample in Australia to be present in 9.4% of female participants ages 15 to 17, and 1.4% of males (Patton, Coffey, Carlin, Sanci, & Sawyer, 2008). All participants included in this study were between 12 and 17 years of age (M = 15.28; SD = 1.52). Throughout the duration of this study, participants were not concurrently receiving any other treatment for eating disorders. Participants were excluded if they had suicidal ideation or were not medically stable enough for outpatient treatment (according to his/her pediatrician).

Clinicians

The FBT-E intervention was administered to participants by the first two authors of this case series, with half of the participants treated by the first author and the other half treated by the second author (see Table 1). Weekly supervision was held to ensure adherence to the manual. The mean length of treatment was 20.4 sessions (SD = 5.68). This is consistent with the length of FBT in outpatient trials, although previous research has considered 20 sessions of FBT to be long term and compared it with a shorter-term 10-session version of FBT (Lock et al., 2005). The majority of the participants (90%) completed the treatment in less than 22 sessions. One participant (who presented with full threshold AN and more severe symptoms) received the treatment for 34 sessions. In general, we observed that more symptomatic participants required a greater number of treatment sessions. The decision to end treatment was determined by the first and second authors in collaboration with the families, while considering objective criteria related to outcome (weight restoration and observable reductions in anxiety and avoidance). Families typically reported significant improvement and a desire to end treatment, or the therapist made the suggestion based on observed treatment gains and progression to the third and final phase of treatment (see Treatment section).

Measures

Adolescent Measures

Height and weight (BMI). To measure body mass index (BMI), participants were weighed in light indoor clothing (with belts or heavy objects, and shoes removed) at baseline and end of treatment as well as at every session. Height in inches was also measured using a standard yardstick. This information was used to calculate the adolescent's BMI percentile and percent ideal body weight (%IBW). The %IBW, a primary outcome index, was calculated using the following formula: (current weight/ideal weight) x 100, using the weight corresponding to the 50th percentile for age, height, and gender according to older National Center for Health Statistics (NCHS) norms (National Center for Health Statistics, 1973) as a metric for "ideal." According to DSM-IV, 85% of IBW is the suggested cutoff for which an individual may be considered to be below minimally normal weight for age and height. This measure allows for assessment of recovery from AN (and SAN) both clinically and diagnostically. Loeb et al. (2011) compared different reference points for diagnostic criteria using ROC curve analysis and found that the aforementioned method (%IBW using the weight corresponding to the 50th percentile BMI-for-age with newer NCHS norms as a proxy for ideal) had the greatest predictive validity with regard to treatment outcome for AN-spectrum adolescents (both AN and SAN).

Clinical Interview. Eating disorder diagnoses were assigned to patients by use of an open-ended, semistructured, psychiatric interview developed by the first author and administered to patients and their parents. This interview was written with item questions based directly on DSM-IV-TR (American Psychiatric Association, 2000) criteria and was not standardized. The interview includes questions regarding the following: demographic variables, chief complaint, history of present illness, treatment history, eating disorder symptoms, body image disturbance, presence of comorbid anxiety and depressive disorders, and family and social/developmental history. Eating disorder symptoms were assessed using wording from the DSM-IV criteria for AN and BN, simplified as necessary for younger participants and reworded when interviewing parents to allow them to provide their

Table 1
A Comparison of FBT-E and FBT Treatment Components

Session	FBT-E Approach	FBT Approach
Session 1	-Psychoeducation about anxiety & exposure -Assign roles to family members -Give parents Coach's Manual -Introducing contingency management -Plan for family meal (with challenge food)	-Gathering history of the illness -Separating the illness from the patient -Emphasizing blame reduction -Educating family about dangers of AN -Planning for family meal
Session 2	-Family Meal: -Collect SUDS before and after meal -Therapist and family feedback -Assign task of meal monitoring (forms) -Ask parents to also monitor Firm Empathy	-Family Meal: -Parents encouraged to get the adolescent to eat one more bite than s/he would typically eat -Monitoring not assigned/collected
Session 3	-Review and discuss monitoring forms -Create Fear and Avoidance Hierarchy [FAH; of feared foods and beverages] -Assign parent-facilitated exposure tasks -Implement contingency management -Review role of siblings (if present)	-Discussing and supporting parent(s) efforts at refeeding including use of functional incentives for eating -Continuing to separate the illness from the patient and reduce familial criticism -Review progress
Sessions 4–8	-Review monitoring forms weekly -Approach each item on FAH with parent's direct assistance; tackle safety behaviors -Teach adolescent skills for coping with anxiety -Evaluate need for optional modules	-Discussing and supporting parent(s) efforts at refeeding and reducing rituals -Continuing to separate the illness from the patient and reduce familial criticism -Evaluate readiness for Phase II
Sessions 9–16	-Criteria for Phase II are met -Gradual return to independent eating -Optional modules as needed for specific psychological symptoms using CBT: • Binge eating/purging • Body image concerns (mirror exposure) • AN specific worry (e.g. worry exposure)	-Criteria for Phase II are met -Gradual return to independent eating -Family encouraged to examine link between adolescent issues and the development of his/her AN - Continuing to separate the illness from the patient and reduce familial criticism
Sessions 17–20	-Relapse prevention -Planning for the future -Celebration of progress/termination	-Exploring adolescent issues with the family; planning for future issues -Checking in how parents are doing as a couple (when two parents present) -Terminating treatment

perspective on their adolescent's clinical presentation. Of note, this clinical interview captures denial of seriousness of low weight (part of Criterion C for AN), which is not captured in the Eating Disorder Examination Questionnaire (Fairburn & Beglin, 1994), described below. Diagnoses achieved by the interview were based on clinical judgment using all the information gathered. Interviews were conducted by the first two authors.

Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994). The EDE-Q is the questionnaire version of the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), an interview regarded as the gold-standard measure of the characteristic psychopathology of eating disorders. The EDE-Q is a 36-item self-report measure that focuses on the past 28 days and yields scores on four subscales: Restraint, Shape Concern, Weight Concern, and Eating Concern. Fairburn and Beglin (1994) have reported data on the concurrent validity of the EDE-Q in community and clinical populations. They note that the EDE-Q may be an acceptable alternative to clinical interviews when assessing those features of eating disorder symptomatology that are not subject to definitional problems, for example, binge eating. We elected to use the EDE-Q for this reason. Further, the clinical interviews utilized in our clinic (see above) are intended to capture DSM-IV eating disorder diagnoses, and the use of the EDE was thought to be redundant.

Children's Depression Inventory (CDI; Kovacs, 1981). Depression was included as an outcome measure in this study based on findings that comorbid depression and anxiety disorders are common amongst children and adolescents with AN. Research suggests that depression tends to onset subsequent to AN and abates with weight

restoration (Hughes, 2012). Therefore, depressive symptoms, when present, should ideally be reduced with successful eating disorder treatment. The CDI is a widely used self-report measure of depressive symptoms in children and adolescents 7 to 17 years of age. The scale consists of 27 items designed to assess a variety of depressive symptoms. The CDI has demonstrated good internal consistency as well as discriminant validity from measures of anxiety (Romano & Nelson, 1988; Saylor, Finch, Spirito & Bennett, 1984).

Screen for Child Anxiety and Related Disorders–Child Version (SCARED; Birmaher et al., 1999; Birmaher et al., 1997; Muris, Merckelbach, Schmidt & Mayer, 1999). The SCARED is a 41-item child/parent report instrument specifically developed to screen for the following childhood anxiety disorders: generalized anxiety, separation anxiety, somatic/panic, social phobia, and school phobia. Various studies have shown that the SCARED is a valid and reliable child anxiety instrument (Birmaher et al., 1999; Birmaher et al., 1997; Muris, Mayer, Bartelds, Tierney, & Bogie, 2001; Muris, Merckelbach, Ollendick, King, & Bogie, 2002).

Parent Measures

Screen for Child Anxiety and Related Disorders–Parent Version (SCARED; Birmaher et al., 1999; Birmaher et al., 1997). In addition to the aforementioned psychometric properties, the SCARED has been found to have reasonable parent-child agreement and thus was selected as the companion parent-based measure of child and adolescent anxiety disorders and symptomatology. This measure is identical to the child version, except it assesses parents' perceptions of their child's anxiety in all of the aforementioned domains.

FBT-E Approach

FBT-E is a manualized treatment in which exposure therapy is delivered in a family-based format. It is a novel approach based on theoretical understandings of anxiety and its treatment, in which EXRP (exposure with response prevention) is presented as an effective method to treat anorexia nervosa and the entire family is present for these sessions. FBT-E is inspired by the success and efficacy of traditional FBT and does retain many of the basic concepts and structure of FBT, including its implementation—a similar three-phase, 20-session format. However, we consider FBT-E to be a very new approach in terms of its definitive conceptualization of an eating disorder as maintained by anxiety, and its emphasis on similarities between AN and anxiety disorders, which drives the selection of interventions to be employed by the parents (and ultimately used independently by the adolescent). In particular, the treatment aims to target three aspects of eating disorder pathology using exposure-based interventions: fear, worry, and disgust. Further, from the first session onward, the therapist attempts to directly engage the adolescent in treatment and frame the parents and siblings as supporting members of the family "team." The adolescent is engaged in several ways: (a) direct psychoeducation about anxiety with information about how exposure can best help him/her overcome the fear, worries and disgust s/he may have about eating, weight gain and related stimuli; (b) direct provision of coping skills to the adolescent; and (c) incentivizing activities that help the adolescent work towards goals and approach feared and avoided stimuli.

The three phases of FBT-E are as follows: Phase I: Recovery From an Injury; Phase II: Getting Back Into the Game; and Phase III: The Playoffs. The FBT-E treatment approach is described below.

Phase I: Recovery From an Illness

The primary goals of Phase I of FBT-E are to present an anxiety-based model and anxiety-based treatment conceptualization of AN, explain the role of each family member in this treatment, and provide parents with clear instructions (in the form of a "Coach's Manual") regarding the refeeding process. This includes descriptions of the three types of anxiety commonly encountered in AN (fear, worry, and disgust) and how they may be recognized in the context of the adolescent's eating disorder (e.g., refusal of food, aversion to previously enjoyed foods accompanied by facial expressions signaling disgust, anticipatory worry about weight gain). These forms of anxiety are directly linked to the adolescent's avoidance of food and related triggers, while illustrating how anxiety in eating disorders can be overcome (i.e., by eating feared foods rather than avoiding them). This psychoeducation aims to help the adolescent and his/her family understand the patient's emotional and behavioral reactions to threatening stimuli and the process maintaining his/her eating disorder; thus, the adolescent may feel more empowered to overcome this disorder knowing that specific tools can be used to overcome anxieties. Contrary to the "grave scene," used in FBT to engage parents in refeeding, parents and family members are given the task in Session 1 of relating to the patient's anxiety through description of one of their own worst fears. After eliciting the worst fears from each family member, the therapist frames these fears in the context of the process of refeeding by asking individual family members to "imagine facing [feared situation] 3 to 5 times per day for the remainder of his/her life just to live." Family members are then assigned specific roles that are consistent with the emphasis on confronting different forms of anxiety; the parents are the "coaches," who help the patient face his/her fears of food and related stimuli;

siblings are "teammates" who play a supportive role; and the patient with AN (or SAN) is an essential member of the team ("star player") (see Video 1A-1C).

At the end of the first session, the parents receive a "Coach's Manual" which the therapist and parent(s) review without the patient present. The manual provides clear instructions for structuring the refeeding process (particularly when weight gain is indicated). Parents are, for example, instructed not to serve patients the most feared foods at the beginning of treatment and to gradually add more feared foods over time. Further, parents are provided with guidelines for supervised mealtimes derived from behavioral treatments (e.g., ignore negative behavior, attend to positive behavior, give labeled praise and model a calm, supportive stance when confronted with the adolescent's anxiety reaction).

The parent-coaches are instructed to bring a picnic meal to the next treatment session that is representative of a meal they may eat at home, including a "challenge" food (e.g., a food that the adolescent has been avoiding) that is not unreasonably difficult but that is currently avoided to some degree. This session directly targets fear and is the family's first concrete exposure practice, aided by the therapist (see Video 2A-2D).

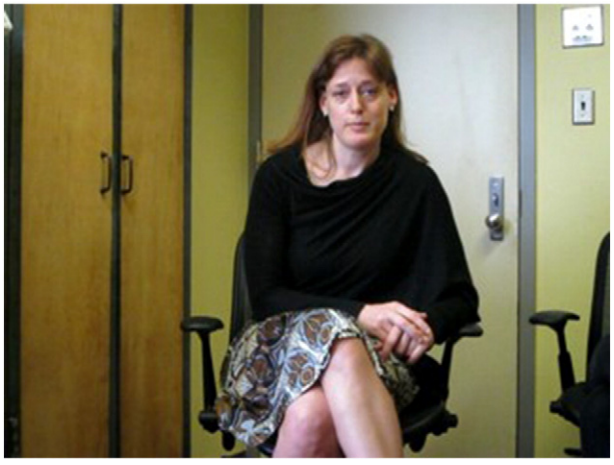
During this initial phase of treatment, FBT-E focuses on reviewing the rationale behind conducting food exposures and reducing safety behaviors, creating a fear-and-avoidance hierarchy (FAH), and explaining how to properly use contingency management to reinforce successful exposures. The "coaches" (i.e., parents) are assigned the task of serving the first item on the hierarchy to the "star player" (i.e., patient) at home and it is explained that they will be gradually tackling each item on the FAH throughout the course of Phase I. Exposure, therefore, takes place primarily at

Video 2. Family Meal.

home, allowing the adolescent (with the aid of his/her parent-coaches) to naturalistically confront feared and disgusted foods in a systematic way. The family is informed by the therapist that while both fear and disgust may be targeted effectively with exposure, it may take repeated trials of the exposure intervention to produce extinction of the disgust response that the patient finds particularly aversive (whether in terms of taste, texture, smell, fat, or carbohydrate content). The patient is encouraged to place such items further along the hierarchy and to allow sufficient time for repeated exposure practice at home; in-office exposures may also occur in which the family brings a food that is primarily disgust driven into session and coaching is provided by the therapist (see Video 3A-3C).

The therapist also explains to parent-coaches the importance of monitoring and curtailing any avoidance behaviors (e.g., cutting food into little pieces, weighing

Video 1. Engaging Adolescent.



Video 3. Disgust Exposure.

food, or chewing slowly) that the adolescent exhibits. Parents are encouraged to use contingency management to help enhance the adolescent's motivation for success. For example, privileges (such as exercise, spending time with friends) may be contingent upon the player's completion of meals with his/her family. During this phase, active coping on the part of the adolescent is also encouraged; skills for managing anxiety while confronting threatening stimuli (e.g., "riding the wave" of the anxiety during a meal, taking deep breaths, giving oneself a pep talk) are taught to the adolescent directly, with coaches and teammates present. The adolescent is encouraged to practice these skills during exposures outside of session.

Phase II: Getting Back Into the Game

In FBT-E, Phase II typically takes place from Sessions 9 through 16 (although more or less time is allotted to complete this phase if needed) and these sessions occur bimonthly (as opposed to weekly). There are specific criteria for progressing from Phase I to Phase II. The adolescent (a) should be weight-restored or at a stable weight health-wise; and (b) all foods on the hierarchy should be completed. The adolescent may need additional practice consuming feared foods in different contexts (e.g., with friends) and is encouraged to continue incorporating these into all meals; however; the adolescent will have successfully exhibited a reduction in anxiety of all of the items on the hierarchy to proceed with Phase II. The second phase of FBT-E involves a gradual increase in independence around eating. A first step may involve allowing the adolescent to serve him/herself portions of food and a larger step may involve choosing his/her own meals and going out to eat with friends, unsupervised. The FBT-E manual allows for a return to Phase I, if needed, if weight is lost or the adolescent exhibits a significant reemergence of anxiety and avoidance (e.g., not being able to eat in social situations). There is no specific prescription for weight maintenance and the ultimate goal of this phase is to fully normalize eating. Siblings also continue to be involved as teammates.

Phase II of FBT-E also addresses any residual eating disorder signs and symptoms, including (a) symptoms of rumination/worry that are interfering with exposure completion (i.e., worry that eating certain foods will lead to weight gain), (b) binge eating and purging, (c) compulsive exercise, (d) body image concerns, and (e) co-occurring internalizing symptoms. FBT-E includes five specific modules with suggested cognitive behavioral techniques for addressing each of these domains (see Table 2). These CBT interventions, which are employed on an as-needed basis, can be used with the adolescent alone and may also include his/her parent(s). For example, in the case of binge eating and/or purging, increased parental supervision may be indicated. At the same time, the adolescent may be independently educated about the benefit of regular meals in curtailing hunger and ways in which to manage binge triggers. With worry and rumination, the adolescent is coached in how to defuse his/her worries and engage in worry exposure; the parent may also be given psychoeducation about these symptoms. With body image concerns, a variety of exposure techniques may be used along with empirically supported CBT interventions for body image (Fairburn, 2008). The use of mirror-exposure for individuals with severe body image may also be indicated (Hildebrandt, Loeb, Troupe, & Delinsky, 2012). Both the adolescent and parent may be enlisted in monitoring and curtailing compulsive exercise. These modules have been extremely helpful in addressing residual symptomatology and fully addressing all aspects of the eating disorder. The

Table 2
FBT-E Phase II Optional Treatment Modules

Modules	Description
Optional Module 1	*Psychoeducation
-Binge Eating and NO Purging	*Regular Eating
	*Appetite awareness/triggers
Optional Module 2	*Psychoeducation about ineffectiveness and/or impact of compensatory behavior
-Purging and/or Exercising	*Increased parental supervision
	*Limit setting around behavior
Optional Module 3	*Monitor body checking and avoidance
-Body Image Concerns	*Exposure to triggers (i.e. mirror exposure)
	*Parent coaching
	*CBT interventions (i.e. Fairburn, 2008)
Optional Module 4	*Worry Exposure
-Worry About Eating/Weight Gain	

Video 4. Worry Exposure.

modules are described more specifically in Table 2 (see Video 4A-4C).

In this study, we utilized one or more of the aforementioned modules with each of our participants. This speaks to the utility of addressing the psychological symptoms of a restrictive eating disorder. In particular, almost all participants appeared to benefit from at least a brief discussion of body image. Further, at least half of our participants mentioned rumination/worry specific to their eating disorder symptoms or recovery, often pertaining to fear of weight gain (e.g., that weight would continue to increase even after treatment ended) or that their weight gain would result in fatness (which necessitated both a discussion of body image as well as cognitive and exposure-based tools). Only one of our participants reported binge-purge symptoms (in this case, this module was employed) and several presented with comorbid internalizing symptoms (requiring a discussion of how to improve these symptoms in the process of recovery).

Phase III: The Playoffs

Phase III is the final phase of the treatment, and sessions now occur once per month (and may involve anywhere from one to four or more sessions). The playoffs occur when all treatment objectives are met, the adolescent is eating independently, and all disordered eating behaviors and additional eating-disorder-related issues are successfully addressed. Similar to many cognitive behavioral treatments, the final phase of treatment entails reviewing progress, discussing any remaining symptoms that need to be addressed, discussing relapse prevention, and planning for the future. The therapist explores what is needed for the adolescent to maintain gains and how to handle bumps in the road and potential lapses. Upon termination, the family is encouraged to do something to celebrate their progress, such as going out for pizza after a championship.

Results

Statistical Analysis

Descriptive changes in primary outcomes pre-post are reported and Standard Error of Measurement (SEM; Anastasi, & Urbina, 1997) is used to provide a standardized metric of individual within-subject change in eating disorder, anxiety, and depressive symptoms over treatment. SEM is calculated using the following formula: standard deviation of the instrument multiplied by the square root of one minus its reliability coefficient. Repeated measures analysis of variance (RM-ANOVAs) were also used to test for significant group changes in eating disorder, anxiety, and depressive symptoms over treatment.

Effects of Treatment

Figure 2 presents trajectories for each individual's percentage of ideal body weight (%IBW) over the course of treatment. All participants gained weight over the course of treatment and 9/10 reached >85% IBW. Two thirds of these individuals (6/9) achieved this threshold by Session 8 with the remaining three participants achieving >85% ideal body weight by Session 16.

Table 3 summarizes the changes in %IBW, eating, depressive, and anxiety symptoms over the course of treatment. Consistent with previous research, we used a criterion of 1 SEM to define meaningful individual clinical change (Wyrwich, Nienaber, Tierney, & Wolinsky (1999). With regard to eating symptoms, all EDE-Q subscales improved over treatment and 9/10 individuals demonstrated a >1 SEM reduction in EDE-Q total score. Furthermore, 8/10 individuals evidenced >1 SEM reduction in the Restraint and Weight Concern Subscales, and 7/10 demonstrated >1 SEM reduction in the Eating and

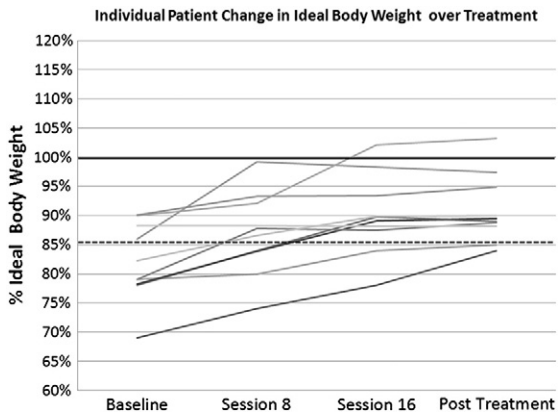


Figure 2. Ideal Body Weight (IBW) pretreatment through posttreatment.

Table 3
Adolescent's Depression, Eating Disorder, and Anxiety Symptoms

	Pre Mean (SD)	Post Mean (SD)	F-value	P level	SEM Change
%IBW	0.81(0.07)	0.91(0.06)	7.71(3,6)	$p < .05$	-
CDI	16.9(6.66)	9.7(9.43)	6.63(1, 9)	$p < .05$	0.35
EDE Global	3.71(1.60)	2.46(1.59)	6.72 (1, 9)	$p < .05$	0.14
Res	3.47(2.14)	2.15(1.64)	2.97(1, 9)	$p = .119$	0.51
Eating Con	3.16(1.78)	1.93(1.63)	5.34(1, 9)	$p < .05$	0.64
Shape Con	4.43(1.43)	4.26(1.88)	4.72(1, 9)	$p = .058$	0.49
Weight Con	3.79(2.04)	2.5(1.68)	4.26(1, 9)	$p = .069$	0.63
C-Total Score	25.80(8.94)	16.10(10.48)	16.67(1, 9)	$p < .01$	0.16
P-Total Score	23.88(8.48)	15.50(3.59)	6.46(1, 7)	$p < .05$	0.17

Note. SEM = standard error of measurement. SEM for total sample $\sigma_x\sqrt{1\text{-}r_{xx}}$.

Shape Concern Subscales. Results of the RM-ANOVAs indicated that all eating disorder symptom scales improved over treatment and the improvements reflected moderate to large effects. However, these effects were only significant for global eating symptoms and eating concerns. Changes in shape in weight and dietary restraint subscales were all in the predicted direction with moderate effect sizes, but they did not reach statistical significance. A total of 8/10 participants had an EDE-Q score of less than 4 at posttreatment, which is the cutoff for clinical significance. Moreover, 6/10 participants had an EDE-Q score less than 2.77, which is within 1 standard deviation of the mean EDE-Q Total Score for young adult women (Mond, Hay, Rodgers, & Owen, 2006).

Similar findings emerged for self and parent-rated adolescent anxiety and depressive symptoms (see Table 3). Patients reported significant improvement in depressive symptoms and 8/10 individuals evidenced >1 SEM reduction in CDI scores over the course of treatment. As previously mentioned, depressive symptoms often improve upon weight restoration and therefore reduction in depression scores may be secondary to weight gain. Anxiety symptoms also improved over the course of treatment. Specifically, 90% individuals evidenced > 1 SEM reduction in anxiety scores as rated by the SCARED-child version and 75% evidenced >1 SEM reduction in anxiety scores according to the SCARED-parent version. Results from RM-ANOVA support significant improvements in depressive symptoms and anxiety symptoms, based on child and parent report.

Discussion

This pilot study sought to examine symptom change over the course of an exposure-based behavioral version of FBT (FBT-E) for adolescents with AN or SAN. There is a strong rationale for altering traditional FBT to explicitly target anxiety, as several etiological and clinical models of AN are based on the relationship between AN and anxiety (Hildebrandt, Bacow, Markella, & Loeb, 2012; Pallister & Waller, 2008; Strober, 2004). Furthermore, a longer version of the leading treatment for adolescents with AN is necessary for patients reporting higher levels of obsessive-compulsive symptoms (Lock et al., 2005), suggesting that a greater focus on anxiety and avoidance behaviors may further improve upon treatment outcomes. The results of this study provide preliminary evidence that FBT-E may be effective at targeting both eating disorder and anxiety symptoms as rated by both patients and their parents. At the end of treatment 9/10 participants reached a threshold of >85% IBW. This is comparable to findings reported in a prior open trial of FBT for SAN and AN patients in which 80% of individuals assigned to FBT had reached 85% IBW at posttreatment (Loeb et al., 2007). The treatment appeared to be well tolerated by families as evidenced by the absence of dropout and spontaneous feedback provided to therapists suggesting this treatment could potentially be used as an alternative to traditional FBT.

The broad anxiety model and intervention have important implications with regard to our understanding of AN pathology and effective treatment approaches for this population. Perhaps the most unique aspect of this model and the FBT-E approach is the inclusion of disgust as a primary anxious emotion maintaining food avoidance. Disgust-based interventions were used explicitly with all but one of our participants to at least some degree (ranging from brief psychoeducation about fear and disgust to a discussion of where to place more disgust-laden items on the hierarchy, to an open, structured disgust exposure). Experimental evidence indicates that extinction of disgust conditioning requires a greater intensity and duration than fear (Mason & Richardson, 2010). Perhaps most relevant to our understanding of the resistance of food avoidance to extinction is the potential for disgust conditioning to occur through evaluative (interoceptively driven) and nonevaluative (cognitively driven) mechanisms. The former type of conditioning is particularly

resistant to extinction (Olatunji, Forsyth, & Cherian, 2007), in part because the conditioned interoceptive responses appear to persist well beyond changes in cognition. The implications for AN are clear. If disgust conditioning is at least in part responsible for food avoidance, the interoceptive discomfort associated with food/eating will persist beyond changes in perceived threats to these triggers Successful extinction of these types of relationships may involve an alternative approach. Counterconditioning (i.e., pairing neutral or positive stimuli with aversive trigger) has been shown to outperform classic extinction (i.e., habituation to trigger in the absence of feared consequence; Raes & De Raedt, 2012) Understanding this option clinically, parents would be asked to pair positive experiences with food/eating triggers. For example, playing the patient's favorite music during the meal or having the siblings engage the patient in a fun game during dinner.

The influence of disgust on food avoidance may also explain relapse among many adolescents. By utilizing the family, FBT-E is able to achieve prolonged exposures to triggering stimuli (e.g., food), multiple times per day, within the individual's home environment where avoidance behaviors are entrenched. Other forms of AN treatment, such as day treatment and inpatient treatment, similarly involve repeated daily exposures; however, these interventions are executed outside the individual's natural environment and therefore may target disgust and other evaluative associations less effectively. Moreover, in contrast to FBT-E, these forms of treatment may inhibit generalization of learning and lead to problems associated with transition to the real-world environment. As a result, individuals receiving these treatments are at high risk for relapse following termination.

FBT-E is also distinct from other treatments with regard to how it targets motivational deficits in AN patients. Resistance to change has been documented in the AN psychotherapy literature (Vitousek, Watson, & Wilson, 1998) and more recent theoretical work (Jappe et al., 2011). A number of neuroimaging studies have begun to identify impairments in motivation-reward system (Frank, Reynolds, et al., 2012; Wagner et al., 2007). Specifically, research suggests that dysregulations in the reward system of AN patients may facilitate food avoidance as well as avoidance of activities that typically elicit pleasure (e.g., socialization with others; (Frank, Roblek, et al., 2012), both of which likely maintain eating pathology. Techniques aimed at enhancing motivation in AN outpatient treatment (i.e., pros and cons; motivational interviewing, etc.) have been previously proposed (Vitousek et al., 1998); however, these strategies may not be adequately effective because they rely on the afflicted individual to overcome these impairments in reward processing through self-initiation. FBT-E uniquely addresses this motivational paradox by helping parents create an environment for the afflicted adolescent that incentivizes non-eating-disordered behavior (e.g., food exposure) as well as rewarding developmentally appropriate activities (e.g., socializing with friends). The parents are directed to deliver these contingencies in a firm but empathetic way in order to reduce blame and increase support for their child's return to a healthy developmental trajectory. Further, adolescents themselves are made a part of the treatment process and provided with specific skills for coping with anxiety. Anecdotally, adolescents are the most resistant to eating disorder treatment, and our clinical experience suggests that they particularly struggle with having their locus of agency completely removed during the first phase of FBT. FBT-E may improve upon this by giving the adolescents a framework within which to understand their symptoms of anxiety and specific tools to manage them.

Resistant patients with lower motivation and lesser interest in reinforcers are harder to treat and tend to be more severe. In Phase I of FBT-E, the resistant patient and his/her family is tasked with removing the intermediate obstacle of food-related anxiety. Illness severity and other obstacles that complicate treatment may require longer duration of treatment to fully reach the goal of dealing directly with food-related anxiety. For example, one patient in this study, who was dealing with a number of stress-related setbacks that limited full engagement in the treatment model, received 34 sessions of therapy. In general, with more severe and less motivated patients, FBT-E would explicitly engage the parents and adolescent in identifying the right set of contingencies to ensure appropriate engagement in treatment and to also allow this system to be flexible to adapt to setbacks and other stressors that may complicate treatment.

Limitations

There are a number of limitations to this study, most importantly the absence of a control group, which makes it impossible to determine if this treatment is efficacious. However, this pilot study does provide some evidence of patient tolerability and evidence that both mood and eating disorder symptoms improve over treatment. In addition, the small sample size prevented a thorough evaluation of the effectiveness of the five additional modules available to therapists in Phases II and III. The flexibility in module use, similar to CBT-E, is clinically appealing but it poses many methodological challenges for establishing efficacy. In particular, the choice of modules by the therapist may introduce a larger effect of therapist competency in the absence of testable or empirically derived algorithms that dictate these clinical choices. We used an open-ended clinical interview combined with the questionnaire version of the EDE (i.e., the EDE-Q) to determine eating disorder diagnosis.

This is an empirically sound assessment method; however, we did not use the interview version of the EDE in our study and acknowledge this as a limitation. The majority of our sample had a diagnosis of SAN, rather than AN, and therefore it is possible that FBT-E is more effective for individuals with less severe symptoms. We used greater than 85% of IBW as a marker of weight restoration. This outcome criterion is consistent with prior research (Eisler et al., 1997; Loeb et al., 2007); however, using individual growth curves may have been a more refined measure of weight outcome. Nine out of 10 participants ended treatment at greater than 85% IBW. Though all patients had lost a significant amount of weight prior to the commencement of treatment, it is important to note that 4/10 participants entered the study at greater than 85% IBW. Moreover, the symptom presentation in individuals with SAN may not be homogeneous. We did not measure the level of obsessive-compulsive symptoms among participants and therefore treatment outcome for these individuals remains unknown.

Future Directions

A larger randomized controlled trial is needed to establish efficacy of this treatment. In addition, we want to identify the mechanisms that distinguish this treatment from existing interventions. In particular, it will be important to establish the impact of FBT-E on food avoidance, particularly food avoidance influenced by disgust-conditioning. This would have important implications for understanding how to improve treatment outcome for patients who evidence an elevated disgust response to food and maintain this disgust response upon weight restoration.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.cbpra.2013.10.006.

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