Published in final edited form as:

Int J Eat Disord. 2011 March ; 44(2): 134–141. doi:10.1002/eat.20784.

Rationale for the Application of Exposure Response Prevention to the Treatment of Anorexia Nervosa

Joanna E. Steinglass, MD, Robyn Sysko, PhD, Deborah Glasofer, Ph.D., Anne Marie Albano, PhD, H. Blair Simpson, MD, PhD, and B. Timothy Walsh, MD
Columbia University/New York State Psychiatric Institute 1051, Riverside Drive, New York, NY 10032

Abstract

Objective—Anorexia nervosa (AN) is a life threatening and difficult to treat illness with a high relapse rate. Current treatments are inadequate and new approaches to treatment are needed.

Method—We review the data on anxiety in AN, the relationship between anxiety disorders and AN, and the use of Exposure and Response Prevention in treatment.

Results—The overlap between AN and anxiety disorders suggest a model of AN in which baseline anxiety features yield eating related fears, avoidance behaviors, and ritualized safety behaviors that promote the underweight state and the perpetuation of the disorder. We propose an Exposure and Response Prevention treatment to prevent relapse in AN.

Discussion—Overlap between AN and anxiety disorders suggests that Exposure and Response Prevention may be a new and beneficial approach to preventing relapse in individuals with AN.

Keywords

Anorexia nervosa; exposure therapy; relapse prevention; anxiety; cognitive-behavior therapy; eating disorders; phobia; obsessive-compulsive disorder

Anorexia nervosa (AN) is a serious illness, affecting approximately 1% of women and one tenth as many men.(1) The disorder is characterized by severe restriction of food intake resulting in an inappropriately low body weight, intense fear of weight gain, and self-evaluation strongly related to body shape and weight. Mortality rates of individuals with AN can be as high as 10%, with the likelihood of death increasing with longer duration of illness.(2–4) Treatment for AN typically begins with acute weight restoration. Behavioral approaches that focus on weight restoration, developed in the 1960s and 1970s, led to inpatient and day treatment programs that, given adequate time, are successful in restoring body weight to near-normal levels. However, after successful weight restoration, many patients continue to exhibit significant eating disorder psychopathology, including fear of “fat,” excessive preoccupation with shape and weight, and abnormal eating behaviors. The rate of relapse following acute treatment is substantial, with as many as 30–50% of adult hospitalized patients requiring re-hospitalization within one year of discharge.(5–7) A recent treatment trial for patients with AN attempted to prevent relapse by providing cognitive behavioral therapy (CBT), emphasizing normalized eating and cognitive restructuring, with either fluoxetine or placebo and observed relapse rates of up to 57% after one year.(8) These high relapse rates highlight that acute weight restoration is only a first step in a more extensive recovery process for patients with AN,(9) and that current treatments available to

prevent relapse are not adequate. Given the substantial relapse rate in this population, new treatment approaches are needed.

Treatment approaches to AN, especially medication trials, have typically been based on phenomenological similarities between AN and other psychiatric illnesses. For example, the common occurrence of depressive symptoms among patients with AN, especially but not exclusively during the underweight state, led to a number of studies evaluating antidepressant treatments.⁽¹⁰⁾ However, these medication treatments have been disappointing. Nonetheless, the pursuit of parallels with other, better characterized disorders with interventions of established efficacy remains appealing.^(11–13) The purpose of this review is to describe: 1) the hypothesis that anxiety is central to the perpetuation of AN; 2) the rationale for utilizing CBT principles of Exposure and Response Prevention for treatment of AN; and 3) an outline of how Exposure and Response Prevention might be adapted for the treatment of individuals with AN.

Overlap Between Anxiety Disorders and AN

While AN is characterized by numerous psychological symptoms, behavioral disturbances are central to the illness's medical morbidity and functional impairment. Simply put, individuals with AN do not eat sufficient calories to maintain a normal weight. This core feature defines the illness, and there are numerous eating- and activity-related behaviors that serve to support the perpetuation of under-eating. Some of these behavioral phenomena (such as avoidance of feared foods, or stereotyped eating behaviors) overlap with disturbances seen in anxiety disorders, including phobic disorders and obsessive compulsive disorder. In addition, individuals with AN and those with anxiety disorders both display an overvaluation of an irrational belief system with dysfunctional behaviors organized around these beliefs. Notably, there symptoms of anxiety within the syndrome of AN, as well as co-occurrence of AN with anxiety disorders.

Patients with AN commonly describe feeling nervous and unable to relax, and endorse physical symptoms of anxiety (e.g., muscle tension, shortness of breath, fidgeting).⁽¹⁴⁾ Anxiety symptoms are incorporated into diagnostic assessments across eating disorders^(27, 28) and the potential importance of this feature has been considered.⁽¹³⁾ For some patients, these symptoms improve with weight normalization.⁽¹⁵⁾ However, these studies report that individuals with AN endorse significantly higher levels of anxiety than healthy controls both while underweight *and* after weight restoration. Furthermore, patients' anxiety scores on the Spielberger State-Trait Anxiety Inventory (STAI⁽¹⁶⁾), remain elevated in comparison to healthy controls even after one or more years at normal weight.^(17, 18) One recent case-control study evaluated factors that may be associated with remission from AN, and reported that trait anxiety, as measured by the STAI, differentiated patients who remitted from AN from those who did not.⁽¹⁹⁾ Thus, while patients with AN report some amelioration of anxiety symptoms with weight restoration, they continue to endorse significantly more non-specific anxiety symptoms relative to controls even after maintaining a normal weight. This persistent anxiety has long been recognized clinically,⁽²⁰⁾ but has not generally been the focus of treatment.

In addition to the overlap between anxiety and eating disorder symptomatology, there is a high degree of comorbidity between anxiety disorders (e.g., social phobia, obsessive compulsive disorder (OCD), generalized anxiety disorder) and AN, with estimates ranging from 38%⁽²¹⁾ to 60%.⁽²²⁾ In a study of lifetime diagnoses,⁽²³⁾ 55–62% of patients with current or past AN had at least one DSM-IV⁽²⁰⁾ anxiety disorder diagnosis, with OCD and social phobia occurring most frequently and substantially higher than would be expected in non-eating disordered populations.⁽²⁴⁾ Studies have consistently found that in the majority

of participants queried, anxiety disorders precede the onset and diagnosis of AN.(21, 22) Furthermore, patients who were diagnosed with AN and another anxiety disorder (childhood generalized anxiety disorder) manifested more severe symptoms of AN.

Findings from genetic studies further support a relationship between anxiety disorders and AN. For example, one twin study found a shared genetic influence in the development of eating disorders and certain anxiety disorders (overanxious disorder and separation anxiety), though they did not look separately at AN.(25) Consistent with this finding, a subsequent family study also reported higher rates of anxiety disorders (OCD, generalized anxiety disorder, separation anxiety disorder, social phobia, and panic disorder) among first-degree relatives of patients with AN.(26)

In sum, the existing literature supports a link between anxiety and the perpetuation of AN, with anxiety symptoms and comorbid anxiety disorders occurring commonly among patients with AN, and some data indicating potentially shared biological features. Taken together, these findings suggest that anxiety may represent a useful target for clinical intervention in the treatment of AN.

A Model of Anxiety in AN

The core behavioral disturbance of AN – the avoidance of food believed likely to produce weight gain -- can be understood to result from several specific anxiety-related components (Figure 1). Two psychopathological models have proposed a link between anxiety and AN. (11, 12) Strober (11) has suggested that anxiety-related behaviors in AN may reflect an abnormality in “fear learning,” such that individuals with AN are more prone to learn fear associations (“fear conditioning”) than their healthy counterparts. This predisposition may account for the development of intense fear of weight gain after the initiation of dieting. In this model, the maladaptive avoidance of eating, especially of high-fat foods characteristic of AN can be understood as resulting from such fears. We have previously proposed a model (12) that emphasizes similarities between AN and OCD. This model emphasizes the obsessive-like intrusive thoughts around fear of weight gain, and the compulsive-like behaviors that attempt to provide reassurance. In this model, disturbances in neural systems known to be disturbed in OCD (frontostriatal circuits) are suggested to create a deficit in learning new behaviors, leaving patients with AN stuck repeating rigid, stereotyped dieting behavior. These models may be complementary, as AN may share features with *both* phobic disorders and obsessive-compulsive disorder, which will be referred to in this paper together as “anxiety disorders.” The model proposed here describes the role of anxiety in the clinical phenomena of AN as the basis of a new treatment approach focused on anxiety. As described in detail below, individuals with AN have anxious and obsessive characteristics that yield fears, avoidance, and ritualistic (or, “safety”) behaviors which perpetuate the core pathology.

Fear

“Fear of fat” is central to the diagnosis of AN; patients often conflate “fear of fat” as it relates to body shape or weight with a fear of ingesting fats. As with anxiety disorders, this fear can be conceptualized as an irrational belief driving avoidance behavior (i.e., insufficient caloric intake). Patients report anxiety in anticipation of a meal, concern about the content of the foods consumed, and fear of the effects of food on shape, weight, and mood,(27) akin to the responses to feared stimuli in anxiety disorders.(29) We propose that this fear is the organizing principle that leads to avoidance and ritual behaviors.

Avoidance

Individuals with AN avoid caloric intake: they do not eat enough overall, and their consumption of high fat, calorie-dense food is significantly reduced in comparison to controls.(30, 31) Laboratory studies of eating behavior have shown that underweight patients take in a substantially lower fraction of calories derived from fat relative to normal controls (32) and that patients with AN, even immediately following weight restoration, avoid consumption of a calorie dense, unfamiliar food.(30, 33, 34) We propose that avoidance behaviors are responses to avoid experiencing eating-related anxiety.

Rituals

Individuals with AN demonstrate rigidly controlled, rule-bound eating patterns(35, 36), as well as abnormal, ritualized behaviors around eating that are used to decrease anxiety.(35, 37, 38) These behaviors can be likened to the safety behaviors used by individuals with phobic disorders, and the rituals or compulsions manifest in OCD. For example, individuals with AN develop irrational beliefs about “safe” and “unsafe” foods, which lead to narrow diets with a restricted range of acceptable choices. Additionally, individuals with AN may be unwilling to eat if they are served an item that deviates from what was expected (e.g., not eating an apple because an orange was anticipated). These behaviors can include regularly leaving a fraction of food on the plate, or eating at a regimented, slow pace. An individual with AN may clutch a napkin during a meal to diminish the intensity of the irrational belief that she is “dirty” because she is eating, or engage in a strict exercise routine after every meal to lessen the anxiety about the calories ingested.

When these idiosyncrasies become entrenched rituals, they contribute to reduced caloric consumption and/or increased caloric expenditure such that avoidance and rigid eating feed back on each other. Of note, rigidity and rituals surrounding food choices have been linked to perfectionist personality traits in patients with AN, which occur premorbidly.(39–41) Overall, these types of eating patterns, which stem from strict dietary rules, limit patients’ ability to achieve sufficient caloric or macronutrient intake. As a result, even after successful weight gain, patients frequently lose weight and become vulnerable to relapse.(42)

In summary, as depicted in Figure 1, AN can be conceptualized as traits of anxiety and obsessionality that result in a combination of fearful avoidance of calorie dense foods, irrational beliefs surrounding eating, and ritualized behaviors that manage the distress around eating. These psychological and behavioral features are present even after successful treatment aimed at restoring weight to within the normal range (14) and may be characterized as fear and avoidance behaviors, which could increase vulnerability to persistence of the disorder and relapse.(19) Effective psychological treatments for anxiety disorders include cognitive behavioral therapy; although CBT can consist of different procedures, data suggest that exposing people to the situations and thoughts that generate their fears is a key element. (43) Exposure is combined with response prevention to address not only the fears and avoidance behaviors but also the ritualistic behavior. Exposure and Response Prevention has been highly successful for both phobic disorders and OCD. Given the suggested clinical and biological overlap between anxiety disorders and AN, it is reasonable to consider whether Exposure and Response Prevention might also be adapted to aid patients with AN.

Cognitive Behavior Therapy in the Treatment of AN

The first stage of treatment for AN necessarily targets acute weight gain. Knowledge of the biology of starvation indicates that many symptoms experienced by underweight patients are a consequence of starvation itself, not specific manifestations of AN.(9) An empirically-supported approach to achieve acute weight gain is behaviorally-oriented therapy in

intensive settings (i.e., day program or inpatient). Behavioral approaches began to be described in the mid 1960s and 1970s, and the establishment of behavioral, incentive-based programs was reviewed by Bemis in 1987.(45) Data supported the utility and efficiency of reinforcement and incentives in “operant conditioning” paradigms to encourage weight gain. (45) The broad acceptance of this approach by the field suggests an appreciation for the role of behavioral learning in the treatment of patients with AN. Among the questions raised by Bemis’s review is whether the incentives used in the inpatient setting have durable post-hospitalization effects. The high rates of relapse after hospitalization suggest that inpatient behavioral treatment alone is often inadequate to foster lasting changes in eating patterns to sustain normal weight when the external structure of the hospital program is removed.(46) Yet given the success of behavioral therapy in helping patients change behaviors acutely, it seems relevant to consider how best to extend these approaches into outpatient relapse prevention treatment.

CBT is widely recommended for treatment of eating disorders, based in part on its demonstrated utility for bulimia nervosa.(47) In a recently published CBT manual for eating disorders,(36) Fairburn describes the treatment of underweight patients as focusing on psychoeducation about starvation, increasing motivation to change behaviors, and addressing the overvaluation of shape and weight. To date, CBT for AN, as it has been manualized and studied, stems from a formulation of the disorder with “overconcern with shape and weight” as a central feature, and uses primarily cognitive techniques in session. Behavioral techniques such as self-monitoring and between session experiments (e.g. discontinuing weighing between sessions) are utilized in all forms of CBT. However, empirical support for CBT is mixed. Pike et al(48) found that CBT was helpful in preventing relapse among weight restored patients with AN, when compared with nutritional counseling. McIntosh et al(49), on the other hand, did not find that CBT was better than clinical management in improving weight gain among underweight patients with AN. In another study using CBT for relapse prevention in a medication study, the relapse rate remained substantial.(8) Channon et al(50) aimed to compare the more cognitively oriented CBT treatment, with a focus on challenging dysfunctional thoughts, to a more behavioral approach that included a graded hierarchy of feared foods and exposure for weight gain in the outpatient setting (described in more detail below). Neither treatment group demonstrated significant weight restoration, but this study was a weekly outpatient treatment which may not have provided sufficient structure for weight gain. CBT treatments for AN emphasize changing the cognitions that lead to dysfunctional behaviors and perpetuate the disorder.(36, 48) It is possible that CBT for AN could be enhanced through increased emphasis on behavioral techniques, specifically the use of in-session exposure to eating-related anxiety. Exposure and response prevention, focused on exposure to eating related fears, avoidance behaviors, and ritualized behaviors may therefore be a useful approach to treating the dysfunctional beliefs and behaviors that serve to perpetuate AN.

Using Exposure and Response Prevention Techniques in the Treatment of AN

Few studies have explicitly used exposure techniques to treat AN. The one randomized trial, mentioned above, compared cognitive therapy with an exposure-based approach in a small trial (n=7) and yielded unimpressive results in the weight gain phase of treatment; however, the authors did not describe the details of the exposure-based intervention;(50) it is not clear that *in-session* exposures were included, and it is not possible to draw conclusions about relapse prevention treatment from this trial. One study (51) used a behaviorally based biofeedback approach to train patients with AN and bulimia nervosa to eat at a normal rate. With this treatment, patients improved in weight and psychological measures, compared to a waitlist control group. However, this study involved several interventions, including the

medication cisapride, as well as structured eating. It is therefore difficult to determine the specific contribution of the feedback sessions. Several studies, including one pilot randomized trial(52) and two case reports, (53, 54) reported results using “systematic desensitization” (i.e., relaxation training in association with hierarchies of weight gain fears) at different stages of treatment for AN, with mixed results.

A single case study described a paradigm involving food exposure. Exposure to high fat foods in an underweight male patient led to a decrease in reported anxiety and increased diet variety.(55) A form of exposure therapy for body image called mirror exposure was examined in a pilot study of inpatients with AN. Key et al.(56) compared two forms of body image treatment, with and without mirror exposure exercises, and found that those who received the mirror exposure component showed significantly greater improvement in body image related anxiety than those who did not.

As previously described elsewhere, we (57) tested the acute effects of exposure in 11 underweight inpatients with AN. Specifically, patients participated in a standardized laboratory test meal before and after receiving 4 exposures to the same meal in the presence of a therapist. This intervention relied primarily on repeated exposure to a food stimulus and did not involve a fully developed Exposure and Response Prevention treatment. Nevertheless, patients demonstrated increased intake in the laboratory post-intervention. These findings, though preliminary, suggest that exposure therapy techniques may benefit patients with AN.

Adapting Exposure Therapy and Response Prevention for Treatment of AN

Exposure and Response Prevention is predicated upon patients’ learning through experience that feared consequences do not occur. To enable experiential learning, Exposure and Response Prevention utilizes session time to place the patient in direct contact with the feared stimuli so that the patient experiences “habituation” to anxiety (dissipation of anxiety), and learns to tolerate - not avoid - anxiety. Thus, for patients with AN, our proposed Exposure and Response Prevention treatment consists of in-session exposures to feared foods and feared eating situations. The psychoeducation component of treatment emphasizes emotion recognition, the concept of anxiety occurring on a scale (as opposed to being a binary, on/off experience), and understanding the natural time course of anxiety. The therapist aims to help the patient learn about the role of fear in perpetuating eating disorder symptoms, and about the importance of confronting rather than avoiding anxiety to reduce the strength of eating related anxiety or distress on behavior.

Identification of Anxiety & Feared Consequences

Therapy begins with helping the patient to recognize gradations of anxiety, in part through the development of a hierarchy of feared foods and situations and use of the Subjective Units of Distress Scale (SUDS). In this process, the individual identifies feared foods as well as feared eating situations, associated rituals and avoidance behaviors that will become the focus of treatment. The therapist helps the patient place these fears along a scale of increasing anxiety/fear. Most importantly, the therapist collaborates with the patient to identify the range of feared consequences. For example, a patient may be most able to articulate a “fear of fat,” but with probing from the therapist, the patient may also endorse fears that are more clearly and unarguably irrational: “fear of losing control/chaos,” “fear of being increasingly anxious and uncomfortable forever,” or “fear of instantaneous obesity.”

In-session Exposure to Feared Eating-Related Situations

Exposures are designed to elicit a patient’s emotional experience *in the moment* and to, in the process, identify and address feared consequences. Some exposures for an individual

with AN might focus on a particular feared food, such as pizza. Other exposures might focus on relaxing rigid control during eating, for example by eating with closed eyes. The therapist helps the patient to maintain focus on every aspect of sensation during the exposure and asks the patient to attend to the physical feelings – both the sympathetic nervous system activation and sensations of fullness. The therapist then helps the patient to recognize these sensations as manifestations of anxiety - as feelings, not realities. Exposure and Response Prevention for AN generates anxiety *in session* and uses techniques to 1) break the association between the feared stimuli and anxiety; 2) break the association between rituals and the relief from anxiety; and 3) disconfirm the irrational beliefs that the stimuli are dangerous. Similar to extinction learning in animals,(61) the individual must learn the absence of the feared outcome (or the absence of total catastrophe) in the presence of the feelings of anxiety and in the absence of ritualizing behaviors.

Response Prevention

During all in-session exposures, the patient is expected to eat without the use of rituals that might consist of blotting with a napkin, or avoidance techniques such as looking away. The therapist helps the patient to avoid “checking out,” or avoiding awareness of eating-related stimuli through placement of pizza boxes or other pizza cues around the room such that anywhere the patient looks she sees pizza reminders. In common with current CBT recommendations, between-session practice emphasizes elimination of all fear-driven responses to eating, such as self-weighing, compulsive exercise, or purging behavior. Response prevention additionally emphasizes elimination of ritualized eating (e.g. maintaining rigid eating schedules, or eating foods in a particular order) and of anxiety avoidance strategies (e.g. watching television while eating) during between-session practice. Response prevention has been found to be particularly important in the treatment of OCD because rituals function as a method of avoidance, and if rituals persist, patients avoid the exposures.(62)

We hypothesize that Exposure and Response Prevention sessions allow for learning of new, safer associations and the subsequent diminution of anxiety disordered symptoms in individuals with AN. Exposure and Response Prevention for AN presents the patient with a new model for understanding eating disordered behaviors, a new perspective that emphasizes the role of fear of food and fat in maintaining a vulnerability to relapse of AN.

Conclusion

As described above, current treatments for AN discuss the importance of altering the distorted thinking that may contribute to relapse.(9, 36) Irrational beliefs are clearly an important part of the syndrome of AN, yet psychotherapy and previous medication interventions have been disappointing in altering outcome. Exposure and Response Prevention emphasizes the relationship between thoughts, feelings and behaviors, but shifts the emphasis of treatment to behavioral techniques. Notably, in phobic disorders and OCD, Exposure and Response Prevention has been found to be a powerful way to change *both* anxiety driven behaviors *and* irrational beliefs.(58, 59) The overlap between AN and anxiety disorders suggests that a treatment with demonstrated success in improving outcome of anxiety disorders merits consideration for individuals with AN.

Reduced food intake among patients with AN, especially of calorie dense foods, is associated with high levels of anxiety, avoidance behaviors around meals, and rituals to manage eating related anxiety. These characteristics strongly resemble the core disturbances of anxiety disorders, including phobic disorders and OCD. These similarities suggest that psychotherapeutic approaches that are effective for these anxiety disorders (i.e., exposure and response prevention) may be useful for AN. Limited previous research has suggested

that exposure therapy is a potentially useful component of treatment for AN, yet it has not been systematically studied. Applying Exposure and Response Prevention to the treatment of AN could alter patients' experiences around eating to increase flexibility of food choice and caloric consumption, thereby improving their ability to maintain weight and prevent relapse. We propose an application of Exposure and Response Prevention to target relapse prevention in AN: this consists of a graded sequence of exposures to the individual's hierarchy of feared eating situations, as well as cessation of rituals and avoidance behaviors. As patients with AN have phobic reactions to food, rituals around food, and distorted cognitive beliefs about the feared consequences of eating food, adaptation of Exposure and Response Prevention for the treatment of AN represents a new and promising approach to this devastating illness.

References

1. Steinhausen HC. The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*. 2002; 159(8):1284–1293. [PubMed: 12153817]
2. Crisp AH, Callender JS, Halek C, Hsu LK. Long-term mortality in anorexia nervosa. A 20-year follow-up of the St George's and Aberdeen cohorts. *Br J Psychiatry*. 1992; 161:104–7. [PubMed: 1638303]
3. Moller-Madsen S, Nystrup J, Nielsen S. Mortality in anorexia nervosa in Denmark during the period 1970–1987. *Acta Psychiatr Scand*. 1996; 94(6):454–9. [PubMed: 9020999]
4. Patton GC. Mortality in eating disorders. *Psychol Med*. 1988; 18(4):947–51. [PubMed: 3270837]
5. Channon S, DeSilva WP, Hemsley D, Perkins R. A controlled trial of cognitive-behavioral and behavioral treatment of anorexia nervosa. *Behavioral Research and Therapy*. 1989; 27:529–535.
6. Eckert E, Halmi KA, Marchi P, Grove W, Crosby R. Ten-year follow-up of anorexia nervosa: clinical course and outcome. *Psychological Medicine*. 1995; 25:143–156. [PubMed: 7792349]
7. Pike KM. Long-term course of anorexia nervosa: response, relapse, remission, and recovery. *Clin Psychol Rev*. 1998; 18(4):447–75. [PubMed: 9638357]
8. Walsh BT, Kaplan AS, Attia E, Olmsted M, Parides M, Carter JC, et al. Fluoxetine after weight restoration in anorexia nervosa: a randomized controlled trial. *JAMA*. 2006; 295(22):2605–12. [PubMed: 16772623]
9. Association AP. Treatment of patients with eating disorders, 3rd edition. *American Journal of Psychiatry*. 2006; 163(7 Supplement):4–54. [PubMed: 16925191]
10. Steinglass, JE.; Walsh, BT. Medication treatment in anorexia nervosa, bulimia nervosa and binge eating disorder. In: Brewerton, TD., editor. *Eating Disorders*. New York: Marcel Dekker; 2004.
11. Strober M. Pathologic fear conditioning and anorexia nervosa: on the search for novel paradigms. *Int J Eat Disord*. 2004; 35(4):504–8. [PubMed: 15101066]
12. Steinglass J, Walsh BT. Habit learning and anorexia nervosa: a cognitive neuroscience hypothesis. *Int J Eat Disord*. 2006; 39(4):267–75. [PubMed: 16523472]
13. Waller G. A 'trans-transdiagnostic' model of the eating disorders: a new way to open the egg? *Eur Eat Disord Rev*. 2008; 16(3):165–72. [PubMed: 18383203]
14. Attia E, Haiman C, Walsh BT, Flater SR. Does fluoxetine augment the inpatient treatment of anorexia nervosa? *Am J Psychiatry*. 1998; 155(4):548–51. [PubMed: 9546003]
15. Pollice C, Kaye WH, Greeno CG, Weltzin TE. Relationship of depression, anxiety, and obsessiveness to state of illness in anorexia nervosa. *Int J Eat Disord*. 1997; 21(4):367–76. [PubMed: 9138049]
16. Spielberger, CD.; Gorsuch, RL.; Lushene, RE. *Manual for the state-trait inventory*. Palo Alto, CA: Consulting Psychologists Press; 1970.
17. Wagner A, Aizenstein H, Venkatraman VK, Fudge J, May JC, Mazurkewicz L, et al. Altered reward processing in women recovered from anorexia nervosa. *Am J Psychiatry*. 2007; 164(12):1842–9. [PubMed: 18056239]

18. Bailer UF, Frank GK, Henry SE, Price JC, Meltzer CC, Becker C, et al. Serotonin transporter binding after recovery from eating disorders. *Psychopharmacology (Berl)*. 2007; 195(3):315–24. [PubMed: 17690869]
19. Yackobovitch-Gavan M, Golan M, Valevski A, Kreitler S, Bachar E, Lieblich A, et al. An integrative quantitative model of factors influencing the course of anorexia nervosa over time. *Int J Eat Disord*. 2009; 42(4):306–17. [PubMed: 19040269]
20. Association AP. *Diagnostic and Statistical Manual of Mental Disorders*. 4. Washington, D.C: American Psychiatric Association Press; 2000. Text Revision
21. Raney TJ, Thornton LM, Berrettini W, Brandt H, Crawford S, Fichter MM, et al. Influence of overanxious disorder of childhood on the expression of anorexia nervosa. *Int J Eat Disord*. 2008; 41(4):326–332. [PubMed: 18213688]
22. Bulik CM, Sullivan PF, Fear JL, Joyce PR. Eating disorders and antecedent anxiety disorders: a controlled study. *Acta Psychiatr Scand*. 1997; 96(2):101–7. [PubMed: 9272193]
23. Kaye WH, Bulik CM, Thornton L, Barbarich N, Masters K. the Price Foundation Collaborative G. Comorbidity of Anxiety Disorders With Anorexia and Bulimia Nervosa. *Am J Psychiatry*. 2004; 161(12):2215–2221. [PubMed: 15569892]
24. Halmi KA, Eckert E, Marchi P, Sampugnaro V, Apple R, Cohen J. Comorbidity of psychiatric diagnoses in anorexia nervosa. *Archives of General Psychiatry*. 1991; 48:712–718. [PubMed: 1883254]
25. Silberg JL, Bulik CM. The developmental association between eating disorders symptoms and symptoms of depression and anxiety in juvenile twin girls. *J Child Psychol Psychiatry*. 2005; 46(12):1317–26. [PubMed: 16313432]
26. Strober M, Freeman R, Lampert C, Diamond J. The association of anxiety disorders and obsessive compulsive personality disorder with anorexia nervosa: evidence from a family study with discussion of nosological and neurodevelopmental implications. *Int J Eat Disord*. 2007; 40 (Suppl):S46–51. [PubMed: 17610248]
27. Sunday SR, Halmi KA, Einhorn A. The Yale-Brown-Cornell Eating Disorder Scale: a new scale to assess eating disorder symptomatology. *Int J Eat Disord*. 1995; 18(3):237–45. [PubMed: 8556019]
28. Cooper Z, Cooper PJ, Fairburn CG. The validity of the eating disorder examination and its subscales. *Br J Psychiatry*. 1989; 154:807–12. [PubMed: 2597887]
29. Barlow, DH.; Durand, VM. *Abnormal Psychology: An Integrative Approach*. 4. Belmont, CA: Thompson-Wadsworth; 2005.
30. Nova E, Varela P, Lopez-Vidriero I, Toro O, Cenal MJ, Casas J, et al. A one-year follow-up study in anorexia nervosa. Dietary pattern and anthropometrical evolution. *Eur J Clin Nutr*. 2001; 55(7): 547–54. [PubMed: 11464228]
31. Misra M, Tsai P, Anderson EJ, Hubbard JL, Gallagher K, Soyka LA, et al. Nutrient intake in community-dwelling adolescent girls with anorexia nervosa and in healthy adolescents. *Am J Clin Nutr*. 2006; 84(4):698–706. [PubMed: 17023694]
32. Hadigan CM, Anderson EJ, Miller KK, Hubbard JL, Herzog DB, Klibanski A, et al. Assessment of macronutrient and micronutrient intake in women with anorexia nervosa. *Int J Eat Disord*. 2000; 28(3):284–92. [PubMed: 10942914]
33. Sysko R, Walsh BT, Schebendach J, Wilson GT. Eating behavior among women with anorexia nervosa. *Am J Clin Nutr*. 2005; 82(2):296–301. [PubMed: 16087971]
34. Windauer U, Lennerts W, Talbot P, Touyz SW, Beumont PJ. How well are ‘cured’ anorexia nervosa patients? An investigation of 16 weight-recovered anorexic patients. *Br J Psychiatry*. 1993; 163:195–200. [PubMed: 8075911]
35. Sunday SR, Einhorn A, Halmi KA. Relationship of perceived macronutrient and caloric content to affective cognitions about food in eating-disordered, restrained, and unrestrained subjects. *Am J Clin Nutr*. 1992; 55(2):362–71. [PubMed: 1734673]
36. Fairburn, CG. *Cognitive Behavior Therapy and Eating Disorders*. New York: Guilford Press; 2008.
37. Slade PD. A short anorexic behaviour scale. *British Journal of Psychiatry*. 1973; 122:83–85. [PubMed: 4683028]

38. Tappe KA, Gerberg SE, Shide DJ, Andersen AE, Rolls BJ. Videotape assessment of changes in aberrant meal-time behaviors in anorexia nervosa after treatment. *Appetite*. 1998; 30(2):171–84. [PubMed: 9573451]
39. Strober M. Personality and symptomatological features in young, nonchronic anorexia nervosa patients. *Journal of Psychosomatic Research*. 1980; 24:353–359. [PubMed: 7205724]
40. Halmi KA, Sunday SR, Strober M, Kaplan A, Woodside DB, Fichter M, et al. Perfectionism in anorexia nervosa: variation by clinical subtype, obsessionality, and pathological eating behavior. *American Journal of Psychiatry*. 2000; 157(11):1799–1805. [PubMed: 11058477]
41. Srinivasagam NM, Kaye WH, Plotnicov KH, Greeno C, Weltzin TE, Rao R. Persistent perfectionism, symmetry, and exactness after long-term recovery from anorexia nervosa. *American Journal of Psychiatry*. 1995; 152(11):1630–1634. [PubMed: 7485626]
42. Schebendach JE, Mayer LE, Devlin MJ, Attia E, Contento IR, Wolf RL, et al. Dietary energy density and diet variety as predictors of outcome in anorexia nervosa. *Am J Clin Nutr*. 2008; 87(4):810–6. [PubMed: 18400701]
43. Barlow, DH. *Anxiety and its disorders: The nature and treatment of anxiety and panic*. New York: Guilford Press; 2002.
44. Kozak, MJ.; Foa, EB. *Mastery of Obsessive-Compulsive Disorder*. New York: Oxford University Press; 1997.
45. Bemis KM. The present status of operant conditioning for the treatment of anorexia nervosa. *Behav Modif*. 1987; 11(4):432–63. [PubMed: 3334127]
46. Wilson GT, Grilo CM, Vitousek KM. Psychological treatment of eating disorders. *American Psychologist*. 2007; 62(3):199–216. [PubMed: 17469898]
47. Chavez M, Insel TR. Eating Disorders: National Institute of Mental Health's Perspective. *American Psychologist*. 2007; 62(3):159–166. [PubMed: 17469895]
48. Pike KM, Walsh BT, Vitousek K, Wilson GT, Bauer J. Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *Am J Psychiatry*. 2003; 160(11):2046–9. [PubMed: 14594754]
49. McIntosh VV, Jordan J, Carter FA, Luty SE, McKenzie JM, Bulik CM, et al. Three psychotherapies for anorexia nervosa: a randomized, controlled trial. *Am J Psychiatry*. 2005; 162(4):741–7. [PubMed: 15800147]
50. Channon S, de Silva P, Hemsley D, Perkins R. A controlled trial of cognitive-behavioural and behavioural treatment of anorexia nervosa. *Behav Res Ther*. 1989; 27(5):529–35. [PubMed: 2684134]
51. Bergh C, Brodin U, Lindberg G, Sodersten P. Randomized controlled trial of a treatment for anorexia and bulimia nervosa. *Proc Natl Acad Sci U S A*. 2002; 99(14):9486–91. [PubMed: 12082182]
52. Goldfarb LA, Fuhr R, Tsujimoto RN, Fischman SE. Systematic desensitization and relaxation as adjuncts in the treatment of anorexia nervosa: a preliminary study. *Psychol Rep*. 1987; 60(2):511–8. [PubMed: 2884684]
53. Hallsten EA Jr. Adolescent anorexia nervosa treated by desensitization. *Behav Res Ther*. 1965; 3(2):87–91. [PubMed: 5828571]
54. Hauserman N, Lavin P. Post-hospitalization continuation treatment of anorexia nervosa. *Journal of Behavior Therapy and Experimental Psychiatry*. 1977; 8:309–313.
55. Boutelle KN. The use of exposure with response prevention in a male anorexic. *J Behav Ther Exp Psychiatry*. 1998; 29(1):79–84. [PubMed: 9627827]
56. Key A, George CL, Beattie D, Stammers K, Lacey H, Waller G. Body image treatment within an inpatient program for anorexia nervosa: the role of mirror exposure in the desensitization process. *Int J Eat Disord*. 2002; 31(2):185–90. [PubMed: 11920979]
57. Steinglass J, Sysko R, Schebendach J, Broft A, Strober M, Walsh BT. The application of exposure therapy and D-cycloserine to the treatment of anorexia nervosa: a preliminary trial. *J Psychiatr Pract*. 2007; 13(4):238–45. [PubMed: 17667736]
58. Foa, EB.; Kozak, MJ. Psychological treatments for obsessive compulsive disorder. In: Mavissakalian, MR.; Prien, RP., editors. *Long-term treatments of anxiety disorders*. Washington, D.C: American Psychiatric Press; 1996.

59. Whittal ML, Thordarson DS, McLean PD. Treatment of obsessive-compulsive disorder: cognitive behavior therapy vs. exposure and response prevention. *Behav Res Ther.* 2005; 43(12):1559–76. [PubMed: 15913543]
60. Dimidjian S, Hollon SD, Dobson KS, Schmaling KB, Kohlenberg RJ, Addis ME, et al. Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *J Consult Clin Psychol.* 2006; 74(4):658–70. [PubMed: 16881773]
61. Davis M, Myers KM. The role of glutamate and gamma-aminobutyric acid in fear extinction: clinical implications for exposure therapy. *Biol Psychiatry.* 2002; 52(10):998–1007. [PubMed: 12437940]
62. Foa EB, Steketee G, Milby JB. Differential effects of exposure and response prevention in obsessive-compulsive washers. *J Consult Clin Psychol.* 1980; 48(1):71–9. [PubMed: 7365045]

Figure 1. Model of Anorexia Nervosa

Traits of high baseline anxiety and obsessionality interact with environmental factors such that patients develop maladaptive behaviors, including food avoidance, and rigid eating patterns (or dieting practices), and they experience high levels of anxiety around eating. These behaviors are interrelated in that rigid dieting leads to increased anxiety about food and vice versa. These behaviors result in a diet that is low fat (low energy density) and limited in variety. This, in turn, promotes weight loss. The low weight state feeds back on the baseline traits and leads to increased levels of anxiety and obsessionality.