

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

Tom Hildebrandt, Terri Bacow, Rebecca Greif, and Adrienne Flores, *Mount Sinai School of Medicine*

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 etiolog-

ical 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).

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

¹ Video patients/clients are portrayed by actors.

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, Straebl, 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

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