2020

Biology of Binge Eating Related Disorders and Proposal for Integration into Treatment

Karlyne Morawe
University of Denver

Biology of Binge Eating Related Disorders and Proposal for Integration into Treatment

Abstract

The concerningly low recovery and high cross over rates of eating disorders with binge eating behaviors suggests there might be a missing element in current treatment approaches commonly used to address eating disorders. Research supports the existence of significant biological correlation between disorders that are characterized by binge eating behaviors. This paper examines some of the biological processes in which these disorders show the most significant correlations. These include the organism's response to caloric restriction, the brain's response to feeding, the biological drive for reproduction, sensory association to feeding, and habituated responding to the feeding process. Additionally, we will explore three of the most common treatment modalities used to treat eating disorders and identify areas in which they may fail to address the biological underpinnings of these disorders. We then use these limitations to propose a novel treatment approach that combines the benefits of enhanced cognitive behavioral therapy with the targeted physiological treatment of appetite awareness training and prescriptive eating to create a more targeted, disorder specific modality based on the biological nuances that appear to maintain binge eating behaviors.

Document Type

Doctoral Research Paper

Degree Name

Psy.D.

Department

Graduate School of Professional Psychology

First Advisor

Jessica Bartley

Second Advisor

Lynett Henderson Metzger

Third Advisor

Lisa Brownstone

Keywords

Binge eating disorders, Treatment, Biology

Subject Categories

Biology | Clinical Psychology | Health Psychology | Psychology

Biology of Binge Eating Related Disorders and Proposal for Integration into Treatment

A DOCTORAL PAPER
PRESENTED TO THE FACULTY OF THE
GRADUATE SCHOOL OF PROFESSIONAL PSYCHOLOGY
OFFICE OF GRADUATE STUDIES
UNIVERSITY OF DENVER

IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE
DOCTOR OF PSYCHOLOGY

BY
KARLYNE MORAWE, M.A.
April 22nd, 2020

APPROVED: _____
Jessica Bartley, Psy.D., Chair

_____
Lynett Henderson Metzger, Psy.D., J.D.

_____
Lisa Brownstone, Ph.D.

Abstract

The concerningly low recovery and high cross over rates of eating disorders with binge eating behaviors suggests there might be a missing element in current treatment approaches commonly used to address eating disorders. Research supports the existence of significant biological correlation between disorders that are characterized by binge eating behaviors. This paper examines some of the biological processes in which these disorders show the most significant correlations. These include the organism's response to caloric restriction, the brain's response to feeding, the biological drive for reproduction, sensory association to feeding, and habituated responding to the feeding process. Additionally, we will explore three of the most common treatment modalities used to treat eating disorders and identify areas in which they may fail to address the biological underpinnings of these disorders. We then use these limitations to propose a novel treatment approach that combines the benefits of enhanced cognitive behavioral therapy with the targeted physiological treatment of appetite awareness training and prescriptive eating to create a more targeted, disorder specific modality based on the biological nuances that appear to maintain binge eating behaviors.

Biology of Binge Eating Related Disorders and Proposal for Integration into Treatment

Introduction

Eating disorders are a concerning presence in modern societies, with lifetime prevalence rates of 0.3%-0.9% for anorexia nervosa (AN), 1%-1.6% for bulimia nervosa (BN), 2.3%-3.5% for binge eating disorder (BED), and a point prevalence of 2.4% for eating disorders not otherwise specified (Smink, et al., 2012). There also exist concerning rates of crossover between eating disorders, most notably a 15%-25% crossover between BED to BN (Fichter & Quadflieg, 2007). The following proposal examines various eating disorders and outlines what can be considered a binge eating related disorder. It then explores the extant research on the biological underpinnings and environmental factors of these disorders to distinguish binge eating related disorders as etiologically unique. The paper then outlines the current existing empirical treatment of binge eating related disorders and identifies the benefits and shortcomings of these interventions in light of the biological processes and environmental factors that maintain these disorders. Finally, this proposal concludes with recommendations for the development of a treatment program that targets the unique nuances that maintain binge eating related disorders to increase long-term recovery rates and limit the crossover between eating disorders.

The significant percentage of relapse and crossover between all of the eating disorders lends to the relevance of this proposal. Stice, et al. (2010) sampled girls between 12 to 15 years of age to determine the presence of an eating disorder with the participants at initial contact and eight years later. This study established a lifetime prevalence of threshold AN, BN, and BED by age 20 as 0.6%, 6.1%, and 4.6%, respectively. Additionally, Stice et al. (2010) found significantly higher relapse rates for BN and BED (41% and 33%) than other eating disorders. Longitudinal studies found a crossover rate from BED to BN to be 15%-25% and from BN to

BED to be 7% (Fichter & Quadflieg, 2007). Moreover, virtually no crossover was noted to exist between AN and BED, while a 9%-11.2% crossover rate was found between AN and BN (Fichter & Quadflieg, 2007; Striegel-Moore et al., 2001). Fichter and Quadflieg (2007) attributed these findings to a shared biological and psychological underpinning between eating disorders with binge eating episodes that does not exist in other feeding and eating disorders.

Binge Eating Related Disorders

In the current paper, we use the term binge eating related disorder (BERD) to reference eating disorders that involve binge eating episodes. Of note, BERD is not a recognized classification for an eating disorder with binge eating episodes. However, given that the majority of available research assesses binge eating as a behavior, this paper will reference any eating disorder that includes binge eating episodes as a BERD with the intent to inform treatment approaches of disorders in which binge eating is central.

Three eating disorders with binge eating characteristics are addressed in the fifth edition of the Diagnostic and Statistical Manual (DSM-5) (American Psychiatric Association, 2013): BN, BED, and other specified feeding or eating disorders (OSFED) with binge eating specifiers. Each of these disorders share a similar criterion in that they all include binge eating episodes. A binge eating episode is characterized by uncontrolled eating of an excessive amount of food in a discrete period of time (American Psychiatric Association, 2013).

Bulimia Nervosa is characterized by recurrent episodes of binge eating followed by inappropriate compensatory behaviors with the intent to prevent weight gain (American Psychiatric Association, 2013). Individuals who meet criteria for BN are typically within a "normal weight or overweight range" and are highly influenced by body shape and weight (American Psychiatric Association, 2013, p. 347).

Binge Eating Disorder was recognized as a separate feeding and eating disorder in 2013 when it was included in the DSM-5. Though similar, what differentiates BED from BN is a lack of compensatory behaviors in response to binge eating episodes. Likewise, preoccupation with weight or body shape is not required to meet criteria for a diagnosis of BED. Furthermore, a distress criterion was included to distinguish between normal overeating and BED. This criterion stipulated that the individual must also experience "marked distress" associated with binge eating behavior. Finally, the frequency of binge eating episodes must occur at least weekly for three months. This frequency criteria may help to differentiate disordered overeating from episodic overeating (American Psychiatric Association, 2013; Decaluwe & Braet, 2003).

Other specified feeding and eating disorder is a diagnosis used to classify clinically significant symptoms of a feeding and eating disorder that do not meet full diagnostic criteria. There are two specifiers within this diagnosis including binge eating episodes; bulimia nervosa (of low frequency and /or limited duration) and binge eating disorder (of low frequency and/or limited duration). For both of these specifiers, binge eating episodes must be present; however, the frequency with which they occur is insufficient for a diagnosis of BN or BED. As such, our proposed BERD category includes BN, BED, and Other Specified Eating Disorders with binge eating episodes. Though AN may also have associated binge eating episodes, this type allows for binge eating or purging behaviors and some individuals with this subtype do not binge eat; therefore, there are likely biological variations from BERD.

Maintaining Factors of Binge Eating-Related Disorders

Research demonstrates that numerous biological processes that do not exist in all eating disorders moderate the maintenance of binge eating behaviors in patients (Iacovina, et al., 2012). By understanding the biological factors that sustain the binge eating behaviors, clinicians may

develop more effective treatment programs that reduce crossover and increase remission rates among those with BERD. Some of the more powerful biological processes of binge eating include dietary restriction, hormonal responses to food, social influences on biological processes, and environmental conditioning through sensory association (Kelley, et al., 2002; Mathes, et al., 2009; Zunker et al., 2011). We will now explore each of these processes in more detail by outlining how each factor impacts biology and, in turn, contributes to the maintenance of BERD.

Dietary Restriction

A notable concern within the mental health community is the role dieting has in the development and maintenance of an eating disorder, particularly binge eating. In fact, studies using laboratory animals document a strong correlation between caloric restriction and binge eating (Hagan et al. 2002; Mathes et al. 2009). Dietary restraint encompasses the limitation or omission of food for the purpose of weight control (Drapeau & Gallant, 2013). Herman and Mack (1975) were the first to suggest the dietary restraint model, which suggests that chronic dietary restraint paradoxically increases the risk for overeating (Burton & Abbott, 2017). Polivy and Herman (1985) continued this research, concluding that dietary restriction was responsible for the onset and maintenance of the restriction-binge eating cycle. Moreover, an ecological momentary assessment (EMA) conducted by Zunker et al. (2011) that assessed participants over a two-week period also implicated self-reported dietary restriction as a predictor of binge eating.

Though early research attributes the restriction-binge cycle to disinhibited and uncontrolled eating, thus resulting in caloric restriction as a solution to the overeating, more recent research indicates that there are physiological factors that may also maintain these behaviors. The biological processes at work during the restriction-binge cycle likely begin in the brain and stimulate hormonal responses. Findings suggest that the hypothalamus, the region of

the brain responsible for maintaining the homeostatic balance of the body and regulating feeding behavior, is the focal point in which this process begins (Larder, et al., 2014; Saper & Lowell, 2014). The lateral hypothalamus is thought to receive signals that stimulate hunger, whereas the ventromedial hypothalamus is thought to create feelings of satiety (Choa, et al., 2019; Leibowitz, 1970; Meister, 2007). As such, in the event of dietary restriction, the hypothalamus is thought to respond as though nourishment is scarce and a threat to survival of the organism exists due to a scarcity of food (Nakamura, et al., 2017; Polivy, et al., 1994). This adaptive response likely triggers the lateral hypothalamus to increase the release of neuropeptide Y (NPY) and Ghrelin, hormones that reduce energy consumption and increase feeding cues to consume more nutrients, while the ventromedial hypothalamus suppresses satiety cues (Beck, 2006; Benton & Young, 2017; Kalra, et al., 1999). Additionally, the hypothalamus is suggested to instruct the body to store any nutrients it receives as fat to ensure future survival of the organism (Benton & Young, 2017). These biological processes activated by the hypothalamus may result in the need for increased quantities of food with the intent to store energy for future famine (Benton & Young, 2017). Evidence suggests that the consequence of caloric restriction and overeating is the return of the organism to its pre-restricting weight or higher, resulting in the maintenance of undesirable weight and future caloric restriction as the individual attempts to exert control over this biological process (Speakman, 2007; Speakman et. al., 2011). Specific to BERD, this process of restriction resulting in overeating behaviors to preserve the organism may be predictive of a binge eating episode.

Neural Activity and Hormonal Response to Food

During normal food consumption, the brain has been shown to respond in a specific way to different properties of the food. Specifically, the brain's neural activity and hormone

production seems to increase in response to highly palatable foods (Kelley, et al., 2005). The brain releases opioid peptides and dopamine within the nucleus accumbens (the reward center of the brain) in response to highly palatable and calorically dense foods (i.e., sweet and fatty). Despite some overlap in the processes of the brain in individuals with BN, AN, and BED in response to feeding, studies suggest there are notable biological variations that are likely responsible, in part, for the dissimilar presentation between BERD and other eating disorders.

For example, individuals with BED appear to have a higher sensitivity to the increase of dopamine in response to foods with a greater hedonic value compared with individuals with non-disordered eating behaviors (Davis et al., 2008). The resulting increase in pleasurable dopamine is suggested to produce a positive experience in relation to the consumption of these foods, while developing a correlation between the food and the positive affective experience (Boggiano, et al., 2005; Kelley et al., 2002; Kelley, et al., 2003; Kelley et al., 2005). Given similar findings were evident in animal studies of bulimia nervosa, it is possible that disorders in which binge eating is central share a heightened reward response to palatable foods that were not observed in animal models of anorexia nervosa (Avena & Bocarsly, 2012).

Research suggests there may also exist biological differences that are responsible for the varied maladaptive thought responses observed between the eating disorders. A within-group study by Brooks et al. (2011) examined the cognitive control differences between BN and AN and found that participants with BN demonstrated less activation than AN participants in regions of the brain responsible for modulating behavior. This finding may explain, in part, the reported lack of control over eating that is characteristic of a binge eating episode. Another maladaptive thought response associated with AN studied by Vocks, et al. (2011) is the fear of weight gain. In their study, Vocks et al. conducted a comparison study of the brain response to a subjectively

rated positive feeding stimuli in participants with AN and those with normal eating behaviors using magnetic resonance tomography. Their findings demonstrated an increase in brain activation of the amygdala that they suggest is responsible for modulating the fear response to feeding among those with AN, a response not similarly observed in BERD or normal eating.

There are notable variations in the brain's response to food which distinguish BN and AN as biologically different. Monteleone et al. (2017) used fMRI to examine the brain response to sweet and bitter tastes in participants with AN and BN found that while both groups of participants experienced similar brain activation in response to pleasant taste stimuli, BN participants demonstrated a reduced amygdalar response to aversive food stimuli when compared with that of AN and non-disordered participants. This noted difference in response to feeding may implicate the amygdala in the disinhibited eating patterns observed in patients with BN that is not present in patients with AN. Furthermore, a within-group study by Brooks et al. (2011) revealed increased insula activation in BN participants when presented pictures of foods they identified as pleasurable; however, the authors did not find this increased activation in the insula after presenting the images to AN participants. This heightened activation of the insula in BN participants might be at least in part responsible for the decline in cognitive control during food consumption experienced by individuals with BERD that is not experienced in other eating disorders. The notable higher sensitivity to increased dopamine in response to food, diminished brain activity in the regions of the brain responsible for modulating behavior, and reduced amygdalar response to aversive food stimuli observed in individuals who engage in binge eating behaviors that is not present in other eating disorders further differentiate BERD as biologically different from other eating disorders.

Social Effects on Disordered Eating Behaviors

There is a wealth of information available supporting the idea that the social behaviors of others influence eating behaviors in response to the organism's biological drive for reproduction. In a study to observe the group effects on eating behaviors, Howland, et al. (2012) found that participants under observation restricted food intake when in the presence of others who were instructed to restrict. The authors suggested that social networks and environmental factors influence feeding and eating behaviors, even without one's conscious awareness. Based on the restriction-binge model of eating disorders, social influences contribute to limiting food intake because of the intent to maintain socially preferred body types for the purpose of attracting a mate. This behavioral response to a biological process may predict caloric restriction patterns in response to environmental factors in eating disorders with binge eating features and is an important factor to consider when treating eating disorders in a group setting.

An additional consideration for treating eating disorders in a group setting is the sexual competition hypothesis (SCH). According to this model, intrasexual competition (ISC) is responsible, at least in part, for the development of eating disorders (Faer, et al., 2005). Intrasexual competition refers to the competitive biological drive of members within the same sex to attract members of the opposite sex. The SCH postulates that eating disorders arise in individuals from a biological drive to appear youthful in the attempt to attract a partner (Faer et al., 2005). A study by Li, et al. (2010) found this hypothesis to hold true problematically without regard to the participants sexual orientation. In westernized societies, thinness is commonly associated with youthfulness and fertility (Singh, 1993). Subsequently, the SCH implicates the desire for neoteny (in the hopes of attracting a mate) as a driving factor in the early development of eating disorders. Abed et al. (2012) collected data using female students, the General Competitiveness Scale (GCS), the Female Intrasexual Competition for Mates Scale (ISC-M), and

the Female Intrasexual Competition for Status Scale (ISC-S) to determine the intersexual competitiveness for mates and intrasexual competitiveness for status. When compared with results of the Eating Disorders Examination Questionnaire (EDE-Q), Abed et al. (2012) found that ISC drives disordered eating behaviors, and thus suggests that the highly competitive nature of eating disorders may be biologically driven. In a group setting, this biological drive may have an adverse effect on self-evaluation and inhibit one's ability to adhere to prescriptive eating protocols due to fear of weight gain or maintenance of excessive BMI. Furthermore, this biologically driven competition for thinness may also play a part in the 15-25% cross over rate from BED to BN found by Fichter and Quadflieg (2007). Overtime, the lack of eating restraint in the absence of compensatory behaviors associated with BED may lead to an increase in body image distress and thus act as a catalyst for the development of compensatory behaviors and, in turn, crossover to BN.

A person's desirability, and therefore the culture in which one lives, impacts the ability of the species to survive (Abed et al., 2012). In western culture, thinness is often associated with youth, and therefore plays a role in desirability (Abed et al., 2012). When in the presence of other members of the same sex, individuals often compare their level of desirability with others who are present (Faer et al., 2005). In individuals who have a negative self-image, the compulsion to increase their sexual desirability may lead to restrictive behaviors, leaving them to repeat the binge-restriction cycle commonly observed in BERD (Zunker et al., 2005). Furthermore, the behavioral influences of others in group settings also link with eating behaviors (Faer et al., 2005). When in a group setting, individuals with BERD are more likely to engage in eating behaviors based on the behaviors of others (Zunker et al., 2011). The behaviors driven by the biological need to attract a mate and social influences on eating may have an adverse impact

on the ability of an individual with BERD to maintain healthy eating behaviors and is an important consideration when treating this population in a group setting.

Environmental Conditioning Through Sensory Association

Conditioning appears to play a significant role in the establishment and maintenance of feeding and eating disorders. According to Mathes, et al. (2009), conditioned responding or environmental association can result in feeding without hunger. These associations present as cravings that may be triggered by environmental factors rather than a biologically driven need for food. Similarly, certain types of foods can also create a conditioned response in anticipation of consumption. Arana, et al. (2003) found after providing participants with normal eating habits with a list of highly motivational foods, that neural activity increased within the medial OFC and the amygdala. The authors attributed this increased activity to the release of NPY within the amygdala in anticipation of the desired foods. With the understanding that the OFC and amygdala are implicated in long-term memory, it is possible this response can be attributed to a sensory association of highly palatable foods with previous experiences, thus likely driving the need for consumption (Tyng, et al., 2017).

In conjunction with the release of NPY in anticipation of food, contextual cues associated with eating may play an important role in the conditioned response of binge eating. Research supporting this hypothesis comes from Petrovich, et al. (2006), who demonstrated that rats exposed to a conditioned stimulus associated with feeding overate, despite being in a state of satiety. Similar human studies found a significant correlation between food cues in a contextual setting and the elicitation of cravings for food (Sobik, et al., 2004). Additionally, the type of food consumed has also been implicated in overeating behaviors. For example, Jarosz, et al. (2006) established that rats consistently consumed sweet and fatty foods in response to environmental

cues in a non-deprived state. Further studies also show that repeated consumption of foods with hedonic properties result in neuroadaptations within the brain, further reinforcing a conditioned response to food (Kelley et al., 2003). In individuals with BERD, conditioned response to food and sensory association of highly palatable foods are expected to initiate feeding in the absence of hunger. Once this occurs, individuals with BERD may be more vulnerable to respond to the emotional experience of unnecessary food consumption with uncontrolled eating, thus resulting in a binge eating episode.

Neuroplasticity

Findings from neuroimaging studies connect automatic environmental responding to the neural plasticity of the brain. Neuroplasticity is a process in which the neurons of the brain alter in form and function and create or reinforce neural pathways, and this process is the foundation for learning and the acquisition of skills (Aarts, et al., 2001). Repeated exposure and response to the same environmental stimulus strengthens the neural pathways connecting the stimulus and the response, thereby creating a habitual response (Aarts, et. al, 2001). The more one repeats an action in response to a particular stimulus, the more ingrained the response to the stimuli becomes. The resulting effect is that over time, less effort and cognitive resources are required to initiate the same response. Gutierrez-Martos et al. (2018) conducted a study to examine the role of neuroplasticity in populations who engage in overeating behaviors. They aimed to determine the neurobiological effect of prolonged exposure to high-caloric foods in a rat population. Their findings demonstrated that repeated exposure and consumption of high-caloric foods resulted in neuroplastic and functional changes in the nucleus accumbens of rats. These modified neuropathways within the reward center of the brain in response to high-caloric foods are likely

responsible for the impulsive, uncontrolled behaviors often attributed to binge eating (Gutierrez-Martos et al., 2018).

In summary, when individuals engage in binge eating, they reinforce the neural pathways associated with overeating. Simultaneously, a cascade of dopamine and opioid peptides release in response to typical binge foods such as those with high sugar and fat content. This flood of neurotransmitters and hormones creates a temporary increase in positive affect (Boggiano, et al., 2005; Kelley et al., 2002; Kelley, et al., 2003; Kelley et al., 2005). As these reinforcers begin to dissipate, the individual's positive affect diminishes, typically resulting in feelings of guilt and shame for having lost control (Munsch et al., 2012). This emotional reaction to binge eating may lead to isolation and further binge eating, or caloric restriction. During food restriction, the body interprets the lack of available calories to a famine-like state and triggers the release of NPY and Ghrelin to stimulate hunger (Benton & Young, 2017). In response, the brain sends signals to the body to slow energy consumption and store available nutrients as fat (Benton & Young, 2017). The increase in NPY and Ghrelin can lead to uncontrolled eating, thus creating a recurring cycle. It is important to note that not all incidences of caloric restriction ensue with intention and that the binge-restriction cycle may also occur through unintended caloric restriction that may occur when regular eating is not encouraged.

Biological factors that include dietary restriction, hormonal responses to foods, biological response to social influences, and habituation to feeding are all components which differentiate BERD as distinctive from other feeding and eating disorders. When treating these disorders, these biological variations should be considered, and a model of treatment developed that addresses how each of these components present in BERD. By focusing treatment interventions on the neurobiological processes that maintain BERD, individuals can begin to rework

neuropathways through intentional behaviors. This treatment is designed with the intention of decreasing potential crossover rates and promoting long-term recovery increase within this population. We will now explore the current approaches used to treat eating disorder and examine ways in which they fail to address the biological components that maintain BERD.

Traditional Treatment Approaches

The previously stated biological and environmental differences among eating disorders suggest the need for a treatment protocol that targets the specific variations unique to BERD. Currently, there are a few empirically supported treatment approaches used to address the behaviors of BERD. While each of these modalities address important aspects of these disorders, none of them holistically address the biological and environmental factors which maintain the disordered behaviors that are distinct to BERD as described above. Additionally, long-term recovery rates for each of the most commonly used approaches are concerningly low, further supporting that there is a missing element to each of these interventions. For instance, remission rates following one year after completion of CBT-E are about 30% for BN and 52% for BED. (Kass, et al., 2013; Sodersten, et al., 2017). We will now explore four of the methods used to treat BERD. These treatments include: Enhanced Cognitive Behavioral Therapy (CBT-E), Interpersonal Psychotherapy for Eating Disorders (IPT-ED), Dialectical Behavioral Therapy (DBT), and Appetite Awareness Training (AAT).

Enhanced Cognitive Behavioral Therapy for Eating Disorders

Enhanced Cognitive Behavioral Therapy for eating disorders is an empirically supported treatment approach that focuses on interrupting the “diet-binge cycle” through goal setting and self-monitoring (Iacovina, et al., 2012). Clinicians often deliver CBT-E the same way across diagnoses and in a group setting. Enhanced Cognitive Behavioral Therapy for eating disorders is

based on the cognitive behavioral theory of BN. The foundation of this approach centers on the notion that the distinctive cognitive and psychological core psychopathology within eating disorders present identically across diagnoses (Fairburn, 2008). Consequently, this treatment disregards the critical differences between BERD and other eating disorders. For example, one important difference that CBT-E fails to address is the reward response in the brain to hedonic properties of food observed in individuals with BERD versus other eating disorder. As previously discussed, the reward center of the brain in individuals with BERD are diminished in response to the hedonic properties of food whereas this same brain process is increased in the brain of an individual with AN (Frank et al. 2012).

Additionally, CBT-E incorporates practices to identify and challenge distorted thoughts associated with a patient's eating disorder (Fairburn, et al., 2003). These practices lead patients to the goal of CBT-E: To address the maintenance processes for eating disorders. These processes include dysfunctional cognitive frameworks, strict dieting and starvation, binge eating and compensatory behaviors, perfectionism, low self-esteem, emotion dysregulation, and interpersonal difficulties (Byrne, et al., 2011). Enhanced cognitive behavioral therapy integrates the mind and body to overcome the disordered eating by challenging the unhelpful cognitions associated with the eating disorder. The mechanism of change is posited to reside in the individual's ability to change their behavioral response to these thoughts (restriction or binging) and effectively interrupt the restriction-binge cycle (Fairburn, 2008).

Though initial remission rates of BN following completion of CBT-E are 50%, longitudinal studies show only a 30% continued remission rate after one year (Sodersten et. al., 2017). Lampard and Sharbanee (2015) suggest relapse following treatment occurs due to remaining mood intolerance, low self-esteem, and interpersonal problems. Enhanced Cognitive

Behavioral Therapy addresses some of the psychological factors associated with eating disorders; however, Sodersten et al. (2017) suggest that this treatment approach may fail to address the biological processes at work that maintain these disorders.

Interpersonal Psychotherapy for Eating Disorders

Interpersonal Psychotherapy for eating disorders (IPT-ED) is a treatment approach used to address the interpersonal difficulties typically present in eating disorders (Rieger, et al., 2010). IPT-ED is an evidence-based treatment approach with comparable recovery rates to CBT-E in both short term and long-term efficacy studies (Agras, el al., 2000; Cooper et al., 2016). Wilfley et al. (2002) found a 79% recovery rate for BED for CBT versus a 73% recovery rate for IPT at posttreatment measurements. One-year follow up recovery rates for BED treated with CBT and IPT were 59% versus 62%, respectively (Wilfley et al., 2002). Interpersonal functioning plays an important role in the development and maintenance of binge eating related disorders and may explain the long-term benefits of IPT-ED. Another consideration for treatment is that interpersonal difficulties often lead to increased isolation from peers and worsened self-esteem (Murphy, et al., 2012). The mechanism of change for IPT is thought to be that by increasing social support, decreasing interpersonal stress, facilitating emotional processing, and improving interpersonal skills, an individual can overcome the social isolation that accompanies eating disorders and change their relationship to the disorder, thus achieving remission and recovery (Lipsitz & Markowitz, 2013).

Additional research shows that IPT-ED is the indicated treatment approach for individuals with BED who present with more severe eating pathology and lower self-esteem (Wilson, et al., 2010). Despite the benefits of IPT-ED and higher long-term remission rates, CBT-E remains the recommended treatment for BN due to the delayed therapeutic effects of

IPT-ED when compared with CBT-E (Agras et al., 2000; Fairburn et al., 2015). Furthermore, there is limited information available pertaining to the mediators of change in IPT-ED, and modest research exists for this treatment approach when compared to CBT-E, leaving IPT-ED as the secondary treatment choice for binge eating related disorders (Murphy et al., 2012). Similar to CBT-E, IPT-ED fails to address biological factors which maintain BERD such as reinforced neuropathways related to habitual binge eating and the neurobiological differences that influence the excitatory response to foods with high hedonic value in the brains of these individuals.

Dialectical Behavior Therapy

Dialectical Behavior Therapy (DBT) works to address binge eating behaviors through the lens of the affect regulation model of binge eating (Iacovino, et al., 2012). Through the conceptualization of the affect regulation model, uncontrolled eating is a coping mechanism in the management of intolerable emotional experiences (Heatherton & Baumeister, 1991). Remission or recovery through this model occurs through integrating mindfulness, distress tolerance, emotion regulation, and interpersonal effectiveness (Linehan, 1993). Stress can trigger binge eating and food restriction in individuals with a history of binge eating (Russel, et al., 2016). Additionally, efficacy studies based on the affect regulation model that used DBT adopted for BED demonstrated an 82% abstinence rate from binge eating at the end of treatment (Telch, et al., 2001). Despite these findings and other studies to support the affective model of binge eating, a meta-analysis conducted by Haedt-Matt and Keel (2011) failed to support the affective model of binge eating, prompting their suggestion that DBT alone may not be the most effective treatment approach to address binge eating disorders.

Appetite Awareness Training

A treatment approach for binge eating with less of a relative literature base is the Appetite Awareness Training (AAT). The impetus to develop this model was to address the eating restraint challenges associated with eating disorders by reestablishing the client's ability to respond appropriately to internal hunger and satiety cues while simultaneously learning to control external factors (Craighead & Allen, 1995; Craighead, 2006). Research shows that individuals with BED tend to demonstrate less sensitivity to hunger and satiety cues both during binges and during normal feeding (Craighead & Allen, 1995). Appetite Awareness Training uses self-monitoring of hunger and fullness cues, thus focusing on the body's internal cues rather than focusing on calories or food types (Allen & Craighead, 1999).

Limited studies are available to support the efficacy of AAT; however, studies that integrate appetite monitoring into other treatment approaches show promise. Appetite-focused CBT (CBT-AF) amalgamates appetite awareness training and CBT to re-sensitize clients to internal appetite cues and thereby "normalize" their eating while simultaneously addressing the cognitive distortions associated with BN (Dicker & Wilcoxon, 2004). While researching the effectiveness of CBT-AF, Dicker and Wilcoxon (2004) found that participants reported a higher willingness to participate in appetite monitoring to regulate calorie intake versus other methods that focus on the foods to limit intake. Similarly, Hill, et al. (2011) conducted a study using appetite-focused dialectical behavior therapy (DBT-AF) to treat binge eating with compensatory behaviors. The authors of this study found that the combination of appetite-focused techniques and DBT resulted in an increased positive response rate to treatment and a lower attrition rate versus DBT alone. Moreover, 61.5% of the participants no longer met criteria for BN following treatment, making DBT-AF another promising approach in the treatment of BERD.

Appetite Awareness Training focuses on normalizing eating patterns by identifying seven points of intervention (Craighead, 2006). These points are: 1) getting too hungry, 2) breaking food rules, 3) ignoring fullness, 4) eating when food is available, 5) emotional eating, 6) giving up, and 7) planning to binge or overeat. Appetite Awareness Training incorporates flexibility in the type of interventions utilized at each point to prevent binge eating by controlling environmental and cognitive factors while also drawing focus to feelings of internal satiety to stop eating. The goal of the treatment is to practice the intervention repetitively to strengthen the neuropathways that make controlled eating easier (Craighead, 2006). Diligent practice appears to augment the neural connections and seemingly improves the efficiency of appropriate responding to food cues, thus making target practices more automatic in the face of cues that once activated binge eating.

Transdiagnostic Treatment

The majority of therapy groups that treat BERD use a transdiagnostic treatment approach. Modalities such as CBT-E justify this approach through the premise that all eating disorders share the same core psychopathology that present in similar behaviors and thought patterns (Fairburn, 2008). Unfortunately, transdiagnostic approaches to treatment fail to address the specific psychological and physiological nuances of each disorder. For example, an individual who presents for treatment of BN may experience temporary relief from guilt and shame following a purge, while someone with AN may achieve this feeling by restricting food intake. Even further, someone who presents with BED may only experience these feelings during a binge eating episode or not at all as was found by Munsch, et al. (2012) in their ecological momentary assessment study on the breakdown of emotion regulation in eating disorders.

Another area of concern pertaining to transdiagnostic treatment of eating disorders is the competitive environment that may result from the presence of individuals who experience different cognitive distortions commonly observed among different eating disorders. Intrasexual competition (ISC), suggests that a crucial factor in the relapse rates of individuals with an eating disorder may lie in the desire for women to achieve thinness with the intent to attract a mate (Abed et al., 2012). Given a diagnostic criteria for AN and BN is preoccupation with weight gain, body image concerns seemingly play an important role in the maintenance of these disorders. Conversely, a diagnosis of BED does not necessitate that the individual has concerns in regard to their body image, further complicating the transdiagnostic approach to the treatment of eating disorders.

An additional consideration is that clinicians may encourage participants to avoid or discourage many topics during transdiagnostic treatment. For instance, weight is often a topic that is "taboo," with the intent to prevent triggering those in treatment who experience preoccupation with their appearance. These off-limits topics prevent individuals from having a space in which they can talk about their body experiences and process their beliefs associated with body shape and size. Additionally, they are unable to witness the experience of others, thereby normalizing their own. Transdiagnostic approaches further neglect the unique neurobiological and etiological considerations evident between eating disorders, leaving a notable gap in the psychoeducation provided to participants in the treatment of their eating disorders.

The Way Forward

Considering the available research, current transdiagnostic treatment approaches are likely not the most effective approach to addressing the biological underpinnings specific to

BERD. Therefore, the development of a more targeted treatment protocol that addresses the areas of 1) dietary restriction, 2) hormonal responses to food, 3) social influences, and 4) sensory association specific to BERD may improve treatment outcomes, reduce cross-over rates, and improve long-term recovery rates.

Proposed Treatment Model

A treatment protocol that targets the biological underpinnings specific to BERD may contribute to an increase in remission and long-term recovery rates while addressing the issue that current treatment approaches for BERD progress as though all eating disorders are based on a common etiology. As previously described, BED, BN, and OSFED with binge eating episodes remain neurobiologically different from AN, and therefore clinicians should approach these distinct disorders differently in treatment. Clinicians can implement interventions to directly address and interrupt the binge eating cycle in at least three different areas. These areas are: a) following the dissipation of dopamine and opioid peptides, b) during eating to modify and regrow helpful neural pathways, and c) during feeding to control the release of NPY and Ghrelin. (see Figure 1).

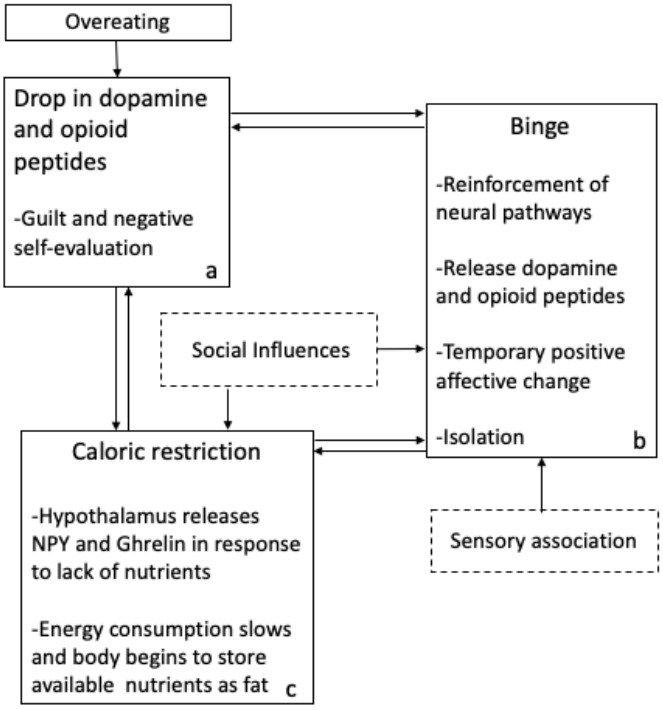

Figure 1: The binge eating cycle and points of intervention

Current treatment protocols address important psychological processes that exist in all eating disorders, such as negative affective experiences and distorted thoughts associated with weight and eating behaviors. The most effective of these treatment approaches, CBT-E, addresses the psychological aspects of BERD by challenging the dysfunctional cognitive frameworks and distorted thinking that leads to maladaptive eating behaviors. By challenging and modifying behavioral responses to the decrease in dopamine and opioid peptides following a binge, the frequency of subsequent binge eating or caloric restriction may also be reduced (Fairburn, 2008). Unfortunately, challenging unhelpful cognitions is rarely enough to maintain remission in BERD populations, and further interventions are likely necessary to address the remaining biological processes (Mathes et al., 2009). Research suggests that, to effectively treat BERD, clinicians must remain mindful of common components of eating disorders while simultaneously addressing the unique biological processes at work.

To accomplish this, an amalgamation of treatment approaches may prove useful. One such promising but underutilized treatment approach that focuses on satiety rather than food is Appetite Awareness Training (AAT). Certain aspects of AAT can integrate with CBT-E to target many of the biological processes that maintain binge eating behaviors, such as unregulated levels of dopamine and opioid peptides, reinforcement of neural pathways associated with binge eating, and elevations of NPY and Ghrelin in response to food and caloric restriction. AAT manages the fluctuations of hormones due to feeding patterns by first managing the quantity of food consumed without the restriction of food groups or types. This approach is likely to reduce subsequent binge eating episodes for individuals with BERD by reducing feelings of deprivation. Studies support this hypothesis and show that individuals who focus on feelings of satiety rather than the type and quantity of food they consume regularly report less frequency in binge eating (Craighead & Allen, 1995). To lessen the biological response to hedonic properties of food, clinicians should encourage those with a BERD to identify feelings of satisfaction such as sensations of stomach distention to indicate the occurrence of adequate feeding. This may be accomplished by teaching a modified version of mindful eating and appetite monitoring. Given the heightened amygdalar response to thoughts of certain foods seen in imaging studies of BED participants, as well as the subsequent increase in NPY and Ghrelin, treatment that focuses on types of food may in fact play a part in the initiation of the binge cycle (Mathes et al., 2009). Appetite monitoring has the participant identify the frequency of feeding, feelings of fullness, and emotional responses to the process of feeding, whereas other approaches to the treatment of BERD, such as CBT-E, focus instead on monitoring the type of food, the location of feeding, and the thoughts associated with feeding. Additionally, moderate consumption of food is the focus of AAT, with the intent to return the participant to intuitive eating patterns. This treatment

discourages labeling foods as good or bad and educates the participants on the importance and function of all types of foods to de-stigmatize high fat and sugary foods, and in turn integrate these into normal feeding (Craighead, 2006).

Another recommended approach to treating BERD is prescriptive eating, a common treatment for eating disorders in inpatient settings that is rarely implemented in outpatient treatment. Prescriptive eating encourages a patient to adhere to a meal plan that designates the consumption of meals at specific times each day. The meal plan includes a balance of the necessary food groups and entails encouraging the participant to measure their food intake to ensure caloric restriction is not a factor. An issue with most prescriptive eating approaches is that food is the focus of this intervention. Research has shown treatment focused on food is less desirable by BN populations versus treatment which focuses on the physical sensations of satiety rather than food intake and thus should be avoided (Dicker & Wilcoxon, 2004). Prescriptive eating focuses on eating episodes based on feelings of satiety. These types of treatments may prove more useful in the treatment of BERD due to a heightened neurological response to the hedonic properties of food in this population when compared with other eating disorders (Arana et al., 2003; Zhang, et al., 2011). For example, a participant is encouraged to eat from all food groups, limiting groups which cause a spike in the reward centers of the brain such as sugary and fatty-foods and balance these with foods with higher protein and fiber. The participant is then encouraged to focus on foods that leave them feeling satisfied longer and with more energy. By learning to pay attention to their body's response to foods and learning to re-label food, not as good or bad, but rather helpful and not helpful, they can begin to focus on the act of eating as nourishing rather than distressing. Furthermore, prescriptive eating encourages an individual to consume five to six meals throughout the day rather than three large meals. The regimented and

mindful approach to eating of AAT combined with the regularity of prescriptive eating may help to weaken the neural pathways associated with overeating and strengthens those related to the frequent consumption of smaller meals, thus breaking the habit of mindless and impulsive eating that often accompanies BERD (Craighead, 2006). In addition to frequent consumption of meals, this hybrid approach to eating also asks participants to slow the pace of their meal consumption. Obesity studies have shown that slow and paced eating may manage the release of NPY and Ghrelin to prevent overeating, while allowing the individual to attend to feelings of satiety (Galhardo et al., 2012).

Despite the utility of appetite awareness and prescriptive eating for all forms of eating disorders, there are additional considerations which may adversely impact long-term recovery from BERD, some of which may be exacerbated in a transdiagnostic setting. For example, SCH of eating disorders suggests the competitiveness of eating disorders triggers symptoms of the disorders due to shame and negative self-concept (Faer et al., 2005). In AN, this presents as caloric restriction whereas in BED, an increase in depression and self-criticism results in an increase in frequency of binge eating (Nettersheim, et al., 2018). Additionally, when in an environment in which caloric restriction is present, an individual with BERD may also engage in restrictive behaviors. Studies suggest the social influences of eating patterns return the patient with a BERD to the binge-restriction cycle, thus maintaining the disorder. Some such influences may include body shaming, social effects on eating patterns, and sensory association of habitual eating; three factors that vary greatly between BERD and other eating disorders. For instance, while both AN and BERD patients experience habitual responses to eating, an individual with BERD experiences an increase in the reward center of the brain in anticipation of food whereas individuals with AN experience an anxiogenic response relieved by food restriction (Kaye &

Bailer, 2011). Additionally, restrictive behaviors are encouraged by weight stigma ideals and enforced by medical constructs that emphasize weight-normative health, whereas overeating is discouraged and shamed by medical providers and thin-body idealists alike (Tylka et al., 2014).

The overt nature of binge eating results in social isolation, secretive eating, and increased guilt and shame in the BERD populations (Fairburn, 2008). This suggests another area for intervention lies in challenging weight stigma and body idealism and encouraging healthy lifestyle changes that do not focus on weight. An approach that already encourages these changes is called Health at Every Size (HAES). The premise of the HAES concept is that body size and shape does not correlate with health and a shift in the approach to weight management should be made to discourage the stigma that thin means healthy (Robinson, 2005). If this altered approach to health is presented in a disorder specific forum, weight-inclusive practices can be tailored to meet the specific needs of the participants and psychoeducation can be provided in a more meaningful way.

Limitations

Some limitations for this proposal should be noted. Though the existing research pertaining to BERD demonstrates many strong biological similarities between disorders with a binge eating component, we were unable to find any research that focused solely on binge eating behaviors as inclusion criteria. Current research commonly examines the biology of BN or BED compared with that of AN or a control. For the purpose of this proposal, similarities among these disorders were inferred; however, further research to ensure that all eating disorders with a binge eating component are more biologically similar than not may be beneficial. Furthermore, to date, research to support the efficacy of a program that integrates elements of AAT, CBT-E, and prescriptive eating in a BERD specific group setting has not been conducted. Though various

studies support the use of these approaches in treating BERD independently, the efficacy of an amalgamated approach should be tested to determine if greater long-term recovery from BERD and/or decreased crossover rates between eating disorders can be achieved.

Conclusion

In conclusion, AAT and CBT-E, though effective, are not without shortcomings in relation to the treatment of BERD. Prescriptive eating approaches and CBT-E, as stand-alone treatments, fail to address the specific psychological and biological processes that maintain binge eating behaviors. An amalgamation of these treatment approaches delivered in a disorder specific setting may better target the biological processes which work to maintain BERD. By addressing these disorders as unique feeding and eating disorders with distinctive biological processes, clinicians can provide education and effective interventions to increase the successful management of these disorders and minimize the effects of group influences associated with transdiagnostic treatment of eating disorders (Ashmore, et al., 2008).

References

Aarts, E., Eikelder, H., & Korst, J. (2001). Neural plasticity. *International Encyclopedia of Social and Behavioral Sciences*, 10542-10546

Abed, R., Mehta, S., Figueredo, A. J., Aldridge, S., Balson, H., Meyer, C., & Palmer, R. (2012). Eating disorders and intrasexual competition: Testing an evolutionary hypothesis among young women. *The Scientific World Journal, 2012*. https://doi.org/10.1100/2012/290813

Agras, W. S., Walsh, B. T., Fairburn, C. G., Wilson, G. T., & Kraemer, H. C. (2000). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry, 57*(5), 459-466. doi:10.1001/archpsyc.57.5.459

Allen, H. N. & Craighead, L. W. (1999). Appetite monitoring in the treatment of binge eating disorder. *Behavior Therapy, 30*(2), 253-272. doi: 10.1016/S0005-7894(99)80007-0

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Ashmore, J. A., Friedman, K. E., Reichmann, S. K, & Musante, G. J. (2008). Weight-based stigmatization, psychological distress, and binge eating behavior among obese treatment-seeking adults. *Eating Behaviors, 9*(2), 203-209. doi: 10.1016/j.eatbeh.2007.09.006

Arana, F. S., Parkinson, J. A., Hinton, E., Holland, A. J., Owen, A. M., & Roberts, A, C. (2003). Dissociable contributions of the human amygdala and orbitofrontal cortex to incentive motivation and goal selection. *Journal of Neuroscience, 23*(29), 9632-9638.

Avena, N. M., & Bocarsly, M. E. (2012). Dysregulation of brain reward systems in eating disorders: Neurochemical information from animal models of binge eating, bulimia

nervosa, and anorexia nervosa. *Neuropharmacology, 63*(1), 87-96. doi: 10.1016/j.neuropharm.2011.11.010

Beck, B. (2006). Neuropeptide Y in normal eating and in genetic and dietary-induced obesity. *Philosophical Transactions of the Royal Society B: Biological Sciences, 361*(1471), 1159-1185. doi: 10.1098/rstb.2006.1855

Benton, D. & Young, H. A. (2017). Reducing calorie intake may not help you lose body weight. *Perspectives on Psychological Science, 12*(5), 703-714. doi:10.1177/1745691617690878

Boggiano, M. M., Chandler, P. C., Viana, J. B., Oswald, K. D., Maldonado, C. R., Wauford, P. K., & Disterhoft, J. F. (2005). Combined dieting and stress evoke exaggerated responses to opioids in binge eating rats. *Behavioral Neuroscience, 119*(5), 1207-1214. doi: 10.1037/0735-7044.119.5.1207

Brooks, S. J., O'Daly, O.G., Uher, R., Friederich, H., Giampietro, V., Brammer, M., Williams, S. C., Schioth, H.B. Treasure, J., & Campbell, I. C. (2011). Differential neural responses to food images in women with bulimia versus anorexia nervosa. *PLoS One, 6*(7). doi: 10.1371/journal.pone.0022259

Byrne, S. M., Fursland, A., Allen, K. L., & Watson, H. (2011). The effectiveness of enhanced cognitive behavioral therapy for eating disorders: An open trial. *Behavior Research and Therapy, 49*(4), 219-226. doi: 10.1016/j.brat.2011.01.006

Burton, A. L. & Abbott, M. J. (2017). Conceptualizing binge eating: A review of the theoretical and empirical literature. *Behavior Change, 34*(3), 168-198. doi:10.1017/bec.2017.12

Choa, H., Digruccio, M., Chen, P., & Li, C. (2019). Type 2 corticotropin-releasing factor receptor in the ventromedial nucleus of hypothalamus is critical in regulating feeding and

lipid metabolism in white adipose tissue. *Endocrinology, 153*(1), 166-176. doi: 10.1210/en.2011-1312

Cooper, Z., Allen, E., Bailey-Straebler, S., Basden, S., Murphy, R., O'connor, M. E., & Fairburn, C. G. (2016). Predictors and moderators of response to enhanced cognitive behaviour therapy and interpersonal psychotherapy for the treatment of eating disorders. *Behaviour Research and Therapy, 84,* 9-13. doi: 10.1016/j.brat.2016.07.002

Craighead, L. W. (2006). *The Appetite Awareness Workbook: How to Listen to Your Body and Overcome Bingeing, Overeating, and Obsession with Food*. Oakland, CA; New Harbinger Publications.

Craighead, L. W. & Allen, H. N. (1995). Appetite awareness training: A cognitive behavioral intervention for binge eating. *Cognitive and Behavioral Practice, 2*(2), 249-270. doi: 10.1016/s1077-7229(95)80013-1

Davis, C., Levitan, R. D., Kaplan, A. S., Carter, J., Reid, C., Curtis, C., Patte, K., Hwang, R., & Kennedy, J. L. (2008). Reward sensitivity and the D2 dopamine receptor gene: A case-control study of binge eating disorder. *Progress in Neuropsychopharmacology & Biological Psychiatry, 32*(3), 620-628. doi: 10.1016/j.pnpbp.2007.09.024

Decaluwé, V. & Braet, C. (2003). Prevalence of binge eating disorder in obese children and adolescents seeking weight-loss treatment. *International Journal of Obesity, 27*(3), 404-409. doi: 10.1038/sj.ijo.0802233

Dicker, S. L., & Wilcoxon, L. (2004). Appetite-focused cognitive-behavioral therapy in the treatment of binge eating with purging. *Cognitive Behavioral Practice, 11*(2), 213-221. doi: 10.1016/s1077-7229(04)80032-4

Drapeau, V., & Gallant, A. (2013). The low satiety phenotype-15. *Satiation, Satiety and the Control of Food Intake,* 273-297. doi: 10.1533/9780857098719.5.273

Duarte, C., & Pinto-Gouveia, J. (2017). Self-defining memories of body image shame and binge eating in men and women: Body image shame and self-criticism in adulthood as mediating mechanisms. *Sex Roles, 77*(5-6), 338-351. doi: 10.1007/s11199-016-0728-5

Duarte, C., & Pinto-Gouveia, J. (2016). Body image flexibility mediates the effect of body image-related victimization experiences and shame on binge eating and weight. *Eating Behaviors, 23*, 13-18. doi: 10.1016/j.eatbeh.2016.07.005

Faer, L. M., Hendriks, A., Abed, R. T., & Figueredo, A. J. (2005). The evolutionary psychology of eating disorders: Female competition for mates or for status? *Psychology and Psychotherapy, 78*(3), 397-417. doi: 10.1348/147608305x42929

Fairburn, C. G. (2008). *Cognitive Behavior Therapy and Eating Disorders*. New York, NY: Guilford Press.

Fairburn, C. G., Bailey-Straebler, S., Basden, S., Doll, H. A., Jones, R., Murphy, R., O'Connor, M. E., & Cooper, Z. (2015). A transdiagnostic comparison of enhanced cognitive behaviour therapy (CBT-E) and interpersonal psychotherapy in the treatment of eating disorders. *Behaviour Research and Therapy, 70*, 64-71. doi: 10.1016/j.brat.2015.04.010

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A "transdiagnostic" theory and treatment. *Behaviour Research and Therapy, 41*(5), 509-528.

Fichter, M. M. & Quadflieg, N. (2007). Long-term stability of eating disorder diagnosis. *International Journal of Eating Disorders, 40*(S3), S61-S66. doi: 10.1002/eat.20443

Frank, G. K. (2013). Altered brain reward circuits in eating disorders: Chicken or egg? *Current Psychiatry Reports, 15*(10), 1-7. doi: 10.1007/s11920-013-0396-x

Frank, G. K., Reynolds, J. R., Shott, M. E., Jappe, L., Yang, T. T., Tregellas, J. R., & O'Reilly, R. C. (2012). Anorexia nervosa and obesity are associated with opposite brain reward response. *Neuropsychopharmacology, 37*(9), 2031-2046. doi: 10.1038/npp.2012.51

Galhardo, J., Hunt, L. P., Lightman, S. L., Sabin, M. A., Bergh, C., Sodersten, P., & Shield, J. P. (2012). Normalizing eating behavior reduces body weight and improves gastrointestinal hormonal secretion in obese adolescents. *The Journal of Clinical Endocrinology & Metabolism, 97*(2), E193-E201. doi: 10.1210/jc.2011-1999

Gutiérrez-Martos, M., Girard, B., Mendonça-Netto, S., Perroy, J., Valjent, E., Maldonado, R., & Martin, M. (2018). Cafeteria diet induces neuroplastic modifications in the nucleus accumbens mediated by microglia activation. *Addiction Biology, 23*(2), 735-749. doi: 10.1111/adb.12541

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin, 137*(4), 660-681. http://dx.doi.org/10.1037/a0023660

Hagan, M. M., Shuman, E. S., Oswald, K. D., Corcoran, K. J., Profitt, J. H., Blackburn, K., Schwiebert, M. W., Chandler, P. C., & Birbaum, C. (2002). Incidence of chaotic eating behaviors in binge eating disorder: Contributing factors. *Behavioral Medicine, 28*(3), 99-105. doi: 10.1080/08964280209596048

Heatherton, T. F. & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin, 110*(1), 86-108. doi: 10.1037/0033-2909.110.1.86

Herman, C., & Mack, D. (1975). Restrained and unrestrained eating 1. *Journal of Personality, 43*(4), 647-660. doi: 10.1111/j.1467-6494.1975.tb00727.x

Hill, D. M., Craighead, L. W., & Safer, D. L. (2011). Appetite-focused dialectical behavior therapy for the treatment of binge eating with purging: A preliminary trial. *International Journal of Eating Disorders, 44*(3), 249-261. doi: 10.1002/eat.20812

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the national comorbidity survey replication. *Biological Psychiatry, 61*(3), 348-358. doi: 10.1016/j.biopsych.2006.03.040

Howland, M., Hunger, J. M., & Mann, T. (2012). Friends don't let friends eat cookies: Effects of restrictive eating norms on consumption among friends. *Appetite, 59*(2), 505-509. doi: 10.1016/j.appet.2012.06.020

Iacovino, J. M., Gredysa, D. M., Altman, M., & Wilfley, D. E. (2012). Psychological treatments for binge eating disorder. *Current Psychiatry Report, 14*(4), 432-446. doi: 10.1007/s11920-012-0277-8

Jarosz, P. A., Sekhon, P., & Coscina, D. V. (2006). Effect of opioid antagonism on conditioned place preferences to snack foods. *Pharmacology, Biochemistry and Behavior, 83*(2), 257-264. doi: 10.1016/j.pbb.2006.02.004

Kalra, S. P., Dube, M. G., Pu, S., Xu, B., Horvath, T. L., & Kalra, P. S. (1999). Interacting appetite-regulating pathways in the hypothalamic regulation of body weight. *Endocrine Reviews, 20*(1), 68-100. doi: 10.1210/er.20.1.68

Kass, A., Kolko, R., & Wilfley, D. (2013). Psychological treatment for eating disorders. *Current Opinion Psychiatry, 26*(6), 549-555. doi: 10.1097/YCO.0b013e328365a30e

Kaye, W. H. & Bailer, U. F. (2011). Understanding the neural circuitry of appetitive regulation in eating disorders. *Biological Psychiatry, 70*(8), 704-705. doi: 10.1016/j.biopsych.2011.08.018

Kelley, A. E, Bakshi, V. P., Haber, S. N., Steininger, T. I, Will, M. J., & Zhang, M. (2002). Opioid modulation of taste hedonics within the ventral striatum. *Physiology and Behavior, 76*(3), 365-377. doi: 10.1016/s0031-9384(02)00751-5

Kelley, A. E., Will, M. I., Steininger, T. L., Zhang, M., & Haber, S. N. (2003). Restricted daily consumption of a highly palatable food (chocolate Ensure®) alters striatal enkephalin gene expression. *European Journal of Neuroscience, 18*(9), 2592-2598. doi: 10.1046/j.1460-9568.2003.02991.x

Kelley, A. E., Baldo, B. A., Pratt, W. E., & Will. M. J. (2005). Corticostriatal-hypothalamic circuitry and food motivation: Integration of energy, action and reward. *Physiology & Behavior, 86*(5), 773-795. doi: 10.1016/j.physbeh.2005.08.066

Lampard, A. M., & Sharbanee, J. M. (2015). The cognitive-behavioural theory and treatment of bulimia nervosa: An examination of treatment mechanisms and future directions. *Australian Psychologist, 50*(1), 6-13. doi: 10.1111/ap.12078

Larder, R., Lim, C. T., & Coll, A. P. (2014). Genetic aspects of human obesity. *Handbook of Clinical Neurology Handbook, 124*, 93-106. doi: 10.1016/b978-0-444-59602-4.00006-x

Leibowitz, S. F. (1970). Reciprocal hunger-regulating circuits involving alpha-and beta-adrenergic receptors located, respectively, in the ventromedial and lateral hypothalamus. *Proceedings of the National Academy of Sciences of the United States of America, 67*(2), 1063-1070. doi: 10.1073/pnas.67.2.1063

Li, N. P., Smith, A. R., Griskevicius, V., Cason, M. J., & Bryan, A. (2010). Intrasexual competition and eating restriction in heterosexual and homosexual individuals. *Evolution and Human Behavior, 31*(5), 365-372. doi: 10.1016/j.evolhumbehav.2010.05.004

Linehan, M. M. (1993). *Diagnosis and treatment of mental disorders. Skills training manual for treating borderline personality disorder.* New York, NY; Guilford Press.

Lipsitz, J. D. & Markowitz, J. C. (2013). Mechanisms of change in interpersonal therapy (IPT). *Clinical Psychology Review, 33*(8), 1134-1147. doi: 10.1016/j.cpr.2013.09.002

Mathes, W. F., Brownley, K. A., Mo X. & Bulik, C. M. (2009). The biology of binge eating. *Appetite, 52*(3), 545-553. doi: 10.1016/j.appet.2009.03.005

Meister, B. (2007). Neurotransmitters in key neurons of the hypothalamus that regulate feeding behavior and body weight. *Physiology & Behavior, 92*(1-2), 263-271. doi: 10.1016/j.physbeh.2007.05.021

Monteleone, A. M., Monteleone, P., Esposito, F., Prinster, A., Volpe, U., Cantone, E., Pellegrino, F., Canna, A., Milano, W., Aiello, M., Di Salle, F., & Maj, M. (2017). Altered processing of rewarding and aversive basic taste stimuli in symptomatic women with anorexia nervosa and bulimia nervosa: An fMRI study. *Journal of Psychiatric Research*, 90, 94-101. doi: 10.1016/j.jpsychires.2017.02.013

Munsch, S., Meyer, A. H., Quartier, V., & Wilhelm, F. H. (2012). Binge eating in binge eating disorder: A breakdown of emotion regulatory process. *Psychiatry Research, 195*(3), 118-124. doi: 10.1016/j.psychres.2011.07.016

Murphy, R., Straebler, S., Basden, S., Cooper, Z., & Fairburn, C. G. (2012). Interpersonal Psychotherapy for Eating Disorders. *Clinical Psychology & Psychotherapy, 19*(2), 150-158. doi: 10.1002/cpp.1780

Nakamura, Y., Yanagawa, Y., Morrison, S. F., & Nakamura, K. (2017). Medullary reticular neurons mediate neuropeptide Y-induced metabolic inhibition and mastication. *Cell Metabolism, 25*(2), 322-334. doi: 10.1016/j.cmet.2016.12.002

Nettersheim, J., Gerlach, G., Herpertz, S., Abed, R., Figueredo, A. J., & Brüne, M. (2018). Evolutionary psychology of eating disorders: An explorative study in patients with anorexia nervosa and bulimia nervosa. *Frontiers in Psychology, 9*, 2122. doi: 10.3389/fpsyg.2018.02122

Petrovich, G. D., Ross, C. A., Gallagher, M., & Holland P. C. (2007). Learned contextual cue potentiates eating in rats. *Physiology & Behavior, 90*(2-3), 362-367. doi: 10.1016/j.physbeh.2006.09.031

Polivy, J., Herman, C., & Pallak, M. S. (1985). Dieting and binging: A causal analysis. *American Psychologist, 40*(2), 193-201. doi: 10.1037/0003-066x.40.2.193

Polivy, J., Zeitlin, S. B., Herman, C. P., & Beal, A. N. (1994). Food restriction and binge eating: A study of former prisoners of war. *Journal of Abnormal Psychology, 103*(2), 409-411. doi: 10.1037/0021-843x.103.2.409

Quiles Marcos, Y., Quiles Sebastián, M. J., Pamies Aubalat, L., Botella Ausina, J., & Treasure, J. (2012). Peer and family influence in eating disorders: A meta-analysis. *European Psychiatry, 28*(4), 199-206. doi: 10.1016/j.eurpsy.2012.03.005

Rieger, E., Van Buren, D. J., Bishop, M., Tanofsky-Kraff, M., Welch, R., & Wilfley, D. E. (2010). An eating disorder-specific model of interpersonal psychotherapy (IPT-ED): Causal pathways and treatment implications. *Clinical Psychology Review, 30*(4), 400-410. doi: 10.1016/j.cpr.2010.02.001

Robison, J. (2005). Health at every size: Toward a new paradigm of weight and health. *Medscape General Medicine, 7*(3), 13.

Russel, S. L., Haynos, A. F., Crow, S. J., & Fruzzetti, A. E. (2017). An experimental analysis of the affect regulation model of binge eating. *Appetite, 110*. 44-50. doi: 10.1016/j.appet.2016.12.007

Saper, C. B., & Lowell, B. B. (2014). The hypothalamus. *Current Biology, 24*(23), R1111-R1116. doi: 10.1016/j.cub.2014.10.023

Singh, D. (1993). Adaptive significance of female physical attractiveness: Role of waist-to-hip ratio. *Journal of Personality and Social Psychology, 65*(2), 293-307. doi: 10.1037/0022-3514.65.2.293

Smink, F. R., Hoeken, R., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports, 14*(4), 406-414. doi: 10.1007/s11920-012-0282-y

Sobik, L., Hutchison, K., & Craighead, L. (2005). Cue-elicited craving for food: A fresh approach to the study of binge eating. *Appetite, 44*(3), 253-261. doi: 10.1016/j.appet.2004.12.001

Södersten, P., Bergh, C., Leon, M., Brodin, U., & Zandian, M. (2017). Cognitive behavior therapy for eating disorders versus normalization of eating behavior. *Physiology & Behavior, 174*, 178-190. doi: 10.1016/j.physbeh.2017.03.016

Speakman, J. (2007). A nonadaptive scenario explaining the genetic predisposition to obesity: The "Predation Release" hypothesis. *Cell Metabolism, 6*(1), 5-12. doi: 10.1016/j.cmet.2007.06.004

Speakman, J. R., Levitsky, D. A., Allison, D. B., Bray, M. S., De Castro, J. M., Clegg, D. J., Clapham, J. C., Dulloo, A. G., Gruer, L., Haw, S., Hebebrand, J., Hetherington, M. M., Higgs, S., Jebb, S. A., Loos, R. J., Luckman, S., Luke, A., Mohammed-Ali, V., O'Rahilly, S.,. . . Westerterp-Plantenga, M. S. (2011). Set points, settling points and some alternative models: Theoretical options to understand how genes and environments combine to regulate body adiposity. *Disease Models & Mechanisms, 4*(6), 733-745. doi: 10.1242/dmm.008698

Stice, E., Mari, C. N., Shaw, H., & Jaconis, M. (2010). An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. *Journal of Abnormal Psychology, 118*(3), 587-597. doi: 10.1037/a0016481

Striegel-Moore, R. H., Cachelin, F. M., Dohm, F. A., Pike, K. M., Wilfley, D. E., & Fairburn, C. G. (2001). Comparison of binge eating disorder and bulimia nervosa in a community sample. *International Journal of Eating Disorders, 29*(2), 157–165. doi: 10.1002/1098-108x(200103)29:2<157::aid-eat1005>3.0.co;2-8

Telch, C. F., Agras, W. S., & Linehan, M. M. (2001). Dialectical behavior therapy for binge eating disorder. *Journal of Consulting and Clinical Psychology, 69*(6), 1061-1065. doi: 10.1037//0022-006X.69.6.1061

Tylka, T. L., Annunziato, R. A., Burgard, D., Daníelsdóttir, S., Shuman, E., Davis, C., & Calogero, R. M. (2014). The weight-inclusive versus weight-normative approach to health: Evaluating the evidence for prioritizing well-being over weight loss. *Journal of Obesity, 2014*, 1-18. doi: 10.1155/2014/983495

Tyng, C. M., Amin, H. U., Saad, M. N., & Malik, A. S. (2017). The influences of emotion on learning and memory. *Frontiers in Psychology*, 8. doi: 10.3389/fpsyg.2017.01454

Vocks, S., Herpertz, S., Rosenberger, C., Senf, W., & Gizewski, E. R. (2011). Effects of gustatory stimulation on brain activity during hunger and satiety in females with restricting-type anorexia nervosa: An fMRI study. *Journal of Psychiatric Research, 45*(3), 395-403. doi: 10.1016/j.jpsychires.2010.07.012

Wilson, G. T., Wilfley, D. E., Agras, W. S., & Bryson, S. W. (2010). Psychological treatments of binge eating disorder. *Archives of General Psychiatry, 67*(1), 94-101. doi: 10.1001/archgenpsychiatry.2009.170

Wilfley, D. E., Welch, R. R., Stein, R. I., Spurrell, E. B., Cohen, L. R., Saelens, B. E., Dounchis, J. Z., Frank, M. A., Wiseman, C. V., & Matt, G. E. (2002). A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge eating disorder. *Archives of General Psychiatry, 59*(8), 713-721. doi: 10.1001/archpsyc.59.8.713

Zhang, Q., Li, H., & Guo, F. (2011). Amygdala, an important regulator for food intake. *Frontiers in Biology, 6*(1), 82-85. doi: 10.1007/s11515-011-0950-z

Zunker, C., Peterson, C. B., Crosby, R. D., Cao, L., Engel, S. G., Mitchell, J. E., & Wonderlich, S. A. (2011). Ecological momentary assessment of bulimia nervosa: Does dietary restriction predict binge eating? *Behaviour Research and Therapy, 49*(10), 714-717, doi: 10.1016/j.brat.2011.06.006.