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Review

Potential psychological & neural mechanisms in binge eating disorder: Implications for treatment

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HIGHLIGHTS

- Psychological and neural mechanisms in Binge Eating Disorder (BED) are unknown.
- Distinguishing BED from co-morbid obesity is key to examining this psychopathology.
- Emotion reactivity, food cue reactivity, and craving are candidate processes in BED.
- Deficits in cognitive control may further exacerbate emotion/craving dysregulation.
- Targeting these processes may improve treatments for BED.

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ABSTRACT

Binge Eating Disorder (BED) is a newly-established eating disorder diagnosis in the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). Although systematic research on BED is in its infancy and many studies feature small samples, several observations emerge. First, we review diagnostic, developmental, and socio-demographic features of BED. Next, although BED and obesity are linked and frequently co-occur, we review data suggesting that BED is a distinct phenotype. Importantly, we take a mechanism-focused approach and propose four psychological processes with neurobiological bases that may uniquely differentiate BED from obesity: emotion reactivity, food-cue reactivity, food craving, and cognitive control. Further, we propose that interactions between impairments in cognitive control and increased emotional reactivity, food-cue reactivity, and craving may underlie emotion dysregulation and promote binge eating. Consistently, neuroimaging studies point towards neural alterations in the response to rewards and to food specifically, and suggest preliminary links between impaired cognitive-control-related neural activity and binge eating. However, additional systematic work is required in this area. We conclude with a detailed review of treatment approaches to BED; specifically, we suggest that psychological and pharmacological treatments that target core mechanisms – including cognitive control and emotion/craving dysregulation – may be particularly effective.

1. Binge eating disorder – introduction

Excessive eating behavior has been observed throughout history, from Roman banquets to yearly holiday feasts (Donahue, 2003; Klesges, Klem, & Bene, 1989). In contrast, binge eating is associated not only with excessive eating, but also with loss of control over eating and eating-related psychopathology. Importantly, frequent binge eating is a core diagnostic feature of Binge Eating Disorder (BED), as well as other forms of eating pathology (e.g., Bulimia Nervosa). Although BED is a newly-included diagnosis in DSM-5 (APA, 2013), recent studies indicate that BED is now the most prevalent eating disorder diagnosis, with up to 3.5% of adults affected in their lifetime (the rate is higher for females than males; Hudson, Coit, Lalonde, & Pope, 2012; Hudson, Hiripi, Pope, & Kessler, 2007; Kessler et al., 2013; Smink, van Hoeken, & Hoek, 2012; Spitzer et al., 1992; Sysko et al., 2012). As such, work investigating mechanisms and potential treatment targets for BED is especially timely and important. Indeed, the National Institute of Mental Health (NIMH) prioritizes research that identifies and targets underlying mechanisms involved in mental disorders, in line with a "rational therapeutic approach" (National Institute of Mental Health, 2017). Here, we propose four psychological processes with neurobiological bases that may underlie BED, and therefore may be important targets for treatment.

In this paper, we will first review diagnostic features and socio-demographic features of BED. Next, we will discuss comorbidity

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between obesity and BED, and suggest that BED is likely a distinct phenotype from obesity. Further, we will review four psychological processes that may be involved in BED, as well as their neural substrates: emotion reactivity, food cue reactivity, food craving, and cognitive control (including the regulation of emotion and craving). These processes may interact to cause loss of control over eating, and therefore are essential to understanding BED. Finally, we will discuss how some existing treatments for BED do, or do not, target these specific processes. Overall, we argue that treatment outcomes could be improved through the advancement of mechanism-focused treatment.

2. Diagnostic features of binge eating disorder

BED was first introduced as a provisional eating disorder diagnosis in the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994). At that time, two main eating disorders were recognized: Anorexia Nervosa (AN) and Bulimia Nervosa (BN). Thus, those individuals who experienced clinically-significant eating disturbances, and who did not meet the specific diagnostic criteria of AN or BN (including individuals with BED-like symptoms), were diagnosed with an Eating Disorder Not Otherwise Specified (ED-NOS; APA, 1994; Striegel-Moore & Franko, 2008). Importantly, a BED-like syndrome was already one of the six suggested presentations within ED-NOS, and the most prevalent of them (APA, 1994; Striegel-Moore & Franko, 2008). Its designation as a provisional diagnosis spurred a period of increased scientific interest, after which BED was subsequently recognized as a formal eating disorder diagnosis in the 5th edition of the DSM (DSM-5; APA, 2013).

An episode of binge eating is characterized by (a) eating an amount of food that is definitely larger than what most people would eat under similar circumstances, in a discrete period of time (e.g., two hours), coupled with (b) a sense of lack of control over eating during the binge episode (i.e., the feeling that one cannot control what or how much is being eaten; (APA, 2013). In turn, BED is diagnosed by: (A) the presence of recurrent binge eating episodes; (B) the episodes are associated with three or more of the following: (1) eating much more rapidly than normal, (2) eating until feeling uncomfortably full, (3) eating a large amount of food when not feeling physically hungry, (4) eating alone because of feeling embarrassed by how much one is eating, and/or (5) feeling disgusted with oneself, depressed, or very guilty afterwards; (C) marked distress regarding binge eating; (D) binge eating occurs at least once a week for three months, on average; and (E) binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in Bulimia Nervosa, and does not occur exclusively during the course of Bulimia Nervosa or Anorexia Nervosa (APA, 2013). That is, individuals with BED do not engage in "purging behaviors" to reduce weight gain such as vomiting, taking laxatives or diuretics, excessive exercising, fasting, etc. (as in BN, or in the binge/purge subtype of AN).

Diagnosing BED requires differential diagnosis of other forms of psychopathology (e.g., major depressive disorder, bipolar disorder), other eating disorders (e.g., BN), and obesity (APA, 2013, p. 352–353; see p. 9 below for further discussion). Importantly, because BED is frequently co-morbid with obesity, attention to binge size and loss of control are especially important to diagnosis (Cooper & Fairburn, 1987; Fairburn & Cooper, 1993; Wilfley, Schwartz, Spurell, & Fairburn, 2000). For instance, it can be challenging to determine whether a reported (potential) binge episode includes a sufficient quantity of food to be considered an "objective binge episode" versus a "subjective binge episode." That is, an "objective binge episode" – the type defined in DSM-5 – is when a patient experiences loss of control over eating and consumes an objectively-large amount of food. Conversely, a "subjective binge episode" occurs when a patient experiences loss of control over eating but does not consume an objectively-large amount of food (Cooper & Fairburn, 1987; Fairburn & Cooper, 1993). Thus, in a subjective binge episode, the quantity consumed would not be objectively large, but is nevertheless perceived by the patient as large and as accompanied by a loss of control. For clinicians, it is important to determine whether the quantity of food consumed is larger than what most people would eat, in order to separate objective from subjective binge episodes, because only the former are diagnostic.

However, the "objective largeness" of an eating episode can be challenging to determine. For instance, because binge eating is often associated with disgust, guilt, and shame, patients' perception and reporting of food quantity may be biased. Further, determining the largeness of a potential binge episode currently relies on subjective clinician judgment (Wolfe, Baker, Smith, & Kelly-Weeder, 2009). Portion size can be used to help determine binge size (e.g. 5 bananas, 3 candy bars, a family-sized bag of chips), as well as considering the context of the eating (e.g., holiday meals, celebrations; Cooper & Fairburn, 1987; Fairburn & Cooper, 1993). Some studies have quantified the caloric consumption involved in binge episodes, although there is no widely-accepted diagnostic cutoff. For instance, estimates of binge size in BED range from 1500 to 3000 calories and estimates in BN range from 3000 to 4500 cal based on test meals and self-report, with some significant variability (for review, Wolfe et al., 2009). Consistently, some studies use caloric thresholds to define a binge episode (e.g., > 1000 cal; Forney, Bodell, Haedt-Matt, & Keel, 2016; Kaye et al., 2004; Keel, Wolfe, Liddle, De Young, & Jimerson, 2007; for review see Wolfe et al., 2009). However, it remains unknown whether considering "objective largeness" based on portion size or caloric thresholds would have greater diagnostic reliability and validity, or whether it is clinically meaningful.

Future work could consider whether "objective largeness" determinations may also be influenced by individual differences. For instance, "largeness" may vary with patient body mass index (BMI), defined as weight divided by the square of height, expressed in kilograms/meters^2 (overweight is defined as $25 < BMI < 30$, and obesity is defined as $BMI \geq 30$). Specifically, because recommended portion quantity increases with BMI, a seemingly large amount of food for a lean individual may be appropriate for an obese individual. Consistently, it is known that BMI correlates with binge size in BED (Guss, Kissileff, Devlin, Zimmerli, & Walsh, 2002) and that individuals with higher BMI report higher thresholds for "normal" food consumption (Arikian et al., 2012). Thus, "largeness" determinations could incorporate the expected portion quantity for each specific patient – given their BMI – and compare this with the amount of food consumed in a reported binge episode; however, such individual comparisons have not yet been tested.

Furthermore, "loss of control" reports can also be influenced by the intensity of disgust, guilt, and shame that an individual experiences following consumption of a large amount of food (Cooper & Fairburn, 1987; Fairburn & Cooper, 1993; Wilfley et al., 2000). For instance, an individual may judge food consumption as "out of control" because they regret consuming it in retrospect. Such a judgment is common in individuals with obesity even in the absence of eating pathology or binge eating behavior (APA, 2013; Rø, Reas, & Rosenvinge, 2012). To determine whether an eating episode is a binge, clinicians can clarify that what is meant by "loss of control" is the inability to stop eating behavior, as opposed to a reflection about the appropriateness of their behavior in retrospect (i.e., guilt/shame; Sanftner, Barlow, Marschall, & Tangney, 1995). Clinicians can also use metaphors to increase clarity about loss of control (e.g., a ball rolling on a flat road that will eventually stop vs. a ball rolling down a hill that you are unlikely to be able to catch), or use more easily accessible language (e.g., "feel helpless to control," "go out of your way to get food," "ignore an interruption to keep eating," "keep eating even though you thought you should stop"; Blomquist et al., 2014; Cooper & Fairburn, 1987; Fairburn & Cooper, 1993; Wolfe et al., 2009). Relatedly, several studies have suggested that the severity of loss of control over eating is related to more severe eating-related psychopathology in BED, although this is not diagnostic (Colles, Dixon, & O'Brien, 2008; Grilo, Masheb, Wilson, Gueorguieva, & White, 2011; Johnson, Roberson-Nay, Rohan, & Torgrud, 2003; Latner

& Clyne, 2008; Latner, Hildebrandt, Rosewall, Chisholm, & Hayashi, 2011; Sonneville et al., 2013).

Severity for BED is specified by the number of binge eating episodes per week, such that "mild" is associated with 1–3 episodes per week, "moderate" with 4–7 episodes, "severe" with 8–13, and "extreme severity" with 14 or more episodes per week. Severity may also be increased (e.g., from moderate to severe) to reflect functional impairment or additional symptoms, including emotional dysregulation (APA, 2013). However, there is mixed evidence for the clinical validity of these severity markers in BED. Specifically, some studies found differences in treatment outcome and clinical presentation across severity levels (Dakanalis, Riva, Serino, Colmenga, & Clerici, 2017; Gianini et al., 2017), but others did not (Nakai et al., 2017). Further, some data suggests that BMI may be an important severity indicator. Indeed, there may be differences in the clinical utility of severity ratings in obese and non-obese patients with BED, such that those with lower BMI engage in more unhealthy weight control behaviors (Carrard, Van der Linden, & Golay, 2012; Goldschmidt et al., 2011) and those with higher BMI report more concerns about weight and more frequent binge eating (Dingemans & van Furth, 2012).

3. Developmental and socio-demographic features of BED

Because BED is a new diagnosis in DSM-5, only a few studies have examined its developmental course. The extant data suggest that BED begins to emerge in adolescence, but most cases emerge in early adulthood (Hudson et al., 2007; Stice, Marti, & Rohde, 2013; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). For example, in a prospective cohort of young women, lifetime prevalence assessed at age 20 was higher for BED (3%) compared to all other eating disorders, with 0.8% for AN and 2.6% for BN; further, age of onset for BED was 18–20, approximately 1–2 years later than AN and BN (Stice et al., 2013). Additionally, although BED typically has a higher remission rate than AN or BN, it can persist past middle age (Guerdjikova, O'Melia, Mori, McCoy, & McElroy, 2012). However, future work with large longitudinal samples is necessary to examine the development of BED at earlier ages and its course across the lifespan.

Additionally, recent work has examined socio-demographic features of BED, including its prevalence across race, ethnicity, and gender/sex. Unlike AN and BN, which are predominantly found in White women, BED has a much smaller disparity across gender/sex and racial/ethnic groups. Indeed, BED is at least as common in African American (AA) and Hispanic individuals as in White, non-Hispanic individuals (APA, 2013; Marques et al., 2011). Further, BED affects approximately 3.5% of women and 2% of men (APA, 2013; Hudson et al., 2007), whereas the ratio is more skewed in AN and BN (AN: 0.9% women, 0.3% men; BN: 1.5% women, 0.5% men; Hudson et al., 2007).

These socio-demographic factors may influence clinical presentation. For instance, in treatment-seeking individuals with BED, BMI and binge eating frequency may be higher in AA individuals; however, the onset of dieting, binge eating, and obesity may be earlier in White individuals (Franko et al., 2012; Lydecker & Grilo, 2016). This suggests that BED could have a faster developmental trajectory in AA individuals (Lydecker & Grilo, 2016). Gender may also influence clinical presentation. For example, females with BED are more likely to seek treatment than males (Kessler et al., 2013). Further, across 11 treatment trials, males reported lower global Eating Disorder Examination (EDE) scores (and lower shape, weight, and eating concerns) but reported similar levels of dietary restraint and objective binge episodes, compared to females (Shingelton, Thompson-Brenner, Thompson, Pratt, & Franko, 2015). At post-treatment, males with lower shape/weight concerns achieved remission after shorter treatments, whereas men with high shape/weight concerns and women were more likely to achieve remission only after longer treatments (Shingelton et al., 2015). However, it is important to note that much of this work is preliminary, and larger replication studies are needed to establish the consistency of these effects.

4. Obesity and BED

In the United States, more than two-thirds of the population is overweight or obese (Greenberg, 2013), more than one-third is obese (Flegal, Kruszon-Moran, Carroll, Fryar, & Ogden, 2016; Ogden, Carroll, Fryar, & Flegal, 2015), and rates of obesity continue to rise (Flegal, Carroll, Kit, & Ogden, 2012). Unfortunately, psychological treatments for obesity are largely ineffective in the long-term. Indeed, weight loss is often < 5–10% of body weight, which can improve health indicators in the short-term (Franz et al., 2007; Grilo & Masheb, 2005; Wadden et al., 2011). However, 95% of individuals who do lose weight, re-gain it within a year (Weiss, Galuska, Khan, Gillespie, & Serdula, 2007; Wing & Phelan, 2005). Notably, surgical treatments such as bariatric surgery may lead to greater initial weight loss than behavioral treatments (e.g., Courcoulas et al., 2015; Martins et al., 2011). However, surgical treatments also have mixed long-term outcomes. Indeed, 10–37% of patients regain a significant portion of lost weight after bariatric surgery (Cooper, Simmons, Webb, Burns, & Kushner, 2015; Karmali et al., 2013; Yanos, Saules, Schuh, & Sogg, 2015). It is possible that these treatments have limited long term efficacy at least in part because they do not adequately target specific psychological mechanisms associated with obesity. This is concerning because obesity is the second leading cause of preventable disease and death in the United States (Center for Disease Control, 2013), with elevated BMI leading to ~2.8 million deaths each year worldwide (World Health Organization, 2010).

Individuals with BED tend to have higher BMI than individuals without eating pathology (de Zwaan, 2001; Hudson et al., 2007; Hudson et al., 2012), likely in part because they engage in food consumption without subsequent compensatory behaviors (e.g., unlike individuals with BN who do engage in compensatory behavior). Further, BED is associated with increased risk for obesity and related health complications (Bulik & Reichborn-Kjennerud, 2003; Bulik, Sullivan, & Kendler, 2002; Hudson et al., 2010; Johnson, Spitzer, & Williams, 2001). Specifically, recent estimates suggest that compared to individuals without eating pathology, individuals with BED are more than twice as likely to be obese: 41.7% in BED vs. 15.8%, with an additional 31.8% of BED patients in the overweight range (R. C. Kessler et al., 2013). Further, BED is especially prevalent in individuals seeking weight loss treatment (Grucza, Przybeck, & Cloninger, 2007; Nicdao, Hong, & Takeuchi, 2007). Thus, obesity and BED are often considered to be closely-related conditions (Devlin, 2007; Hudson et al., 2006; Leehr et al., 2015; Smith & Robbins, 2013; Telch & Agras, 1994).

However, there are important differences between obesity and BED. For example, although some obese individuals (OB) report occasional binge eating (e.g., Matos et al., 2002), BED is considered a clinically-diagnosed psychiatric condition that includes frequent binge-eating episodes accompanied by perceived loss of control, unlike obesity, which is not clinically-defined in DSM-5 and is considered a physical condition (APA, 2013; Marcus & Wildes, 2009). Additionally, compared to OB, some studies suggest that obese individuals with BED (OB-BED) eat more calories in regular meals (Guss, Kissileff, Walsh, & Devlin, 1994; Yanovski et al., 1992) and eat larger amounts of food ad libitum when prompted to lose control (Walsh, 2011; Walsh & Boudreau, 2003), demonstrating behavioral differences in eating patterns. However, two ecological momentary assessment studies reported mixed findings about the frequency of loss of control eating among OB-BED compared to OB. Specifically, one study found a greater frequency (but similar caloric consumption per episode) in OB-BED compared to OB (Greeno, Wing, & Shiffman, 2000), and another did not (Le Grange, Gorin, Catley, & Stone, 2001).

Further, although > 66% of the United States population is overweight or obese, only 2–3.5% meet criteria for BED (Hudson et al., 2007), and individuals with BED are at a greater risk of major medical morbidity (Bulik et al., 2002; Bulik & Reichborn-Kjennerud, 2003;

Hudson et al., 2010) and comorbid psychopathology (Bulik et al., 2002; Grilo et al., 2008; Peterson, Latendresse, Bartholome, Warren, & Raymond, 2012; Striegel-Moore & Franko, 2008; Telch & Agras, 1994). In addition, early genetics studies suggest that BED aggregates separately in families (Hudson et al., 2006; Kessler, Hutson, Herman, & Potenza, 2016). Thus, it has been proposed that BED represents a distinct phenotype within the obesity spectrum, with a distinct neurobiological basis (Carnell, Gibson, Benson, Ochner, & Geliebter, 2012; Devlin, 2007; Kessler et al., 2016; Leehr et al., 2015; Schag, Schonleber, Teufel, Zipfel, & Giel, 2013; Wang et al., 2011). Importantly, although some studies on BED use other eating disorders as controls, much research on BED compares OB-BED to OB as a control group. As such, these studies can identify mechanisms that underlie BED above and beyond obesity. Below, we identify several such domains of functioning that may underlie BED: emotion reactivity, food cue reactivity, food craving, and cognitive control. We specifically propose that these domains can be important underlying mechanisms of BED and thus can serve as treatment targets.

5. Emotion reactivity and dysregulation in BED

"Emotion reactivity" typically refers to the intensity and duration with which emotions are experienced in response to stimuli (Nock, Wedig, Holmberg, & Hooley, 2008), is related to "emotional vulnerability" (Linehan, 1993; Nock et al., 2008), and has been linked to psychopathology (Gross & Jazaieri, 2014). Importantly, emotion reactivity has been proposed as a unique mechanism involved in eating-related psychopathology, which may differentiate between obesity and BED (Leehr et al., 2015). For example, an early study with OB-BED patients reported a significant association between binge eating severity, distress, and depressive symptoms, and proposed that this may account for a previously-reported relationship between obesity and psychopathology (Telch & Agras, 1994). Since then, multiple studies have suggested that individuals with BED may display different – and greater – responses to psychological and physiological stress (Klatzkin, Gaffney, Cyrus, Bigus, & Brownley, 2015), greater depression and anxiety symptoms (Grilo et al., 2008), and more frequent and severe daily negative affect than OB, overweight, or lean controls (e.g., Zeeck, Stelzer, Linster, Joos, & Hartmann, 2011). Further, binge-eating symptoms were shown to correlate with depressive symptoms and emotional overeating (Masheb & Grilo, 2006). In several studies with multiple eating disorder groups, patients (including with BED) reported greater emotional intensity, lower emotional awareness, and lower acceptance of emotion compared to healthy controls (e.g., Brockmeyer et al., 2014; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012).

Indeed, negative affect is associated with more severe eating pathology (Gianini, White, & Masheb, 2013; Grilo & White, 2011; Whiteside et al., 2007), and is thought to play a role in the development and maintenance of all eating disorders (Hilbert et al., 2007; Stice, 2002; Stice, Presnell, & Spangler, 2002). Consistently, a recent review of 18 experimental studies suggested that negative emotion serves as a trigger for binge eating in BED, but not in obese individuals without BED (Leehr et al., 2015). Further, prior work has suggested that negative affect may have a causal impact on binge eating (Chua, Touyz, & Hill, 2004; Greeno et al., 2000; Haedt-Matt & Keel, 2011; Masheb & Grilo, 2006; Stein et al., 2007; Zeeck et al., 2011). For instance, several studies have shown that negative affect precedes binge episodes (e.g., Greeno et al., 2000; Le Grange et al., 2001; Munsch, Meyer, Quartier, & Wilhelm, 2012). Accordingly, a meta-analysis of 36 ecological momentary assessment studies found that negative affect was greater preceding binge episodes, with a large effect size (Haedt-Matt & Keel, 2011). This may be in part due to stress increasing the reward value of food; indeed, under conditions of stress, the reinforcement value of food, desire to binge eat, and cortisol levels increase – specifically in binge eaters (Gluck, Geliebter, Hung, & Yahav, 2004; Gluck, Geliebter, & Lorence, 2004; Goldfield, Adamo, Rutherford, & Legg, 2008). Thus, heightened emotional reactivity may be linked to overeating and binge eating in individuals with BED more than in individuals with obesity.

In contrast, "emotion regulation" encompasses the set of processes that are activated by the goal to modulate one's emotional experience (Gross & Jazaieri, 2014), is a form of cognitive control (Tabibnia et al., 2011), and is related to general psychological wellbeing (Gross & John, 2003). Deficits in emotion regulation might include either emotion-regulation failures (i.e., not engaging in regulation when it would be helpful) or emotion misregulation (i.e., using ineffective emotion regulation; Gross & Jazaieri, 2014). Such emotion regulation deficits are considered a core feature of all eating disorders (e.g., Brockmeyer et al., 2014; Danner, Sternheim, & Evers, 2014), and may play an important role in BED. For example, in one study, individuals with BED reported poorer emotion regulation compared to healthy controls, including lower use of adaptive reappraisal and higher use of maladaptive suppression strategies (Svaldi et al., 2012). These data have been replicated in treatment-seeking individuals with BED, in which emotional suppression was further related to depression symptoms (Danner et al., 2014). Other studies have also linked BED with difficulties in emotion regulation, compared to both normal weight and overweight controls (e.g., Brockmeyer et al., 2014). Ultimately, the experience and/or appearance of unregulated emotions – hereinafter "emotion dysregulation" – may result from an interaction between greater emotional reactivity and deficits in emotion regulation. Thus, emotion dysregulation – as an umbrella term – constitutes a core feature of BED, above and beyond obesity.

6. BED, food cue reactivity, and craving

A century of empirical animal work beginning with Ivan Pavlov has demonstrated that cues experimentally paired with food (e.g., the sound of a bell) evoke strong conditioned responses, including salivation (Pavlov, 1927), ghrelin secretion (Sugino et al., 2004), and dopamine neuron firing (Roitman, Stuber, Phillips, Wightman, & Carelli, 2004; Schultz, Dayan, & Montague, 1997). In humans, similar conditioned responses to food cues have been documented, including increased salivation, heart rate, gastric activity, and neural activity in the ventral striatum, amygdala, insula, and orbitofrontal cortex (see Boswell & Kober, 2016 for review). Furthermore, exposure to food cues also strongly and reliably produces the conscious experience of *cue-induced craving* (Cornell, Rodin, & Weingarten, 1989; Fedoroff, Polivy, & Herman, 1997, 2003; Gendall, Joyce, & Sullivan, 1997; Ng & Davis, 2013; Weingarten & Elston, 1990). Craving is a diagnostic criterion for Substance Use Disorders in DSM-5, where it is defined as "a strong desire to use" (APA, 2013). Consistently, food craving is commonly defined as "a strong desire to eat" (Hill, 2007; Weingarten & Elston, 1990; White, Whisenhant, Williamson, Greenway, & Netemeyer, 2002). We recently conducted a quantitative meta-analysis on this topic and reported that food cue reactivity and craving increase eating and prospectively predicts weight gain consistently, reliably, and significantly, with medium-to-large effect sizes (Boswell & Kober, 2016). The results suggest that variance in food cue reactivity and food craving account for up to 26% of the variance in eating and weight gain, more than any other single known factor.

The link between food cue reactivity, craving, eating, and weight gain is important for understanding BED, because obese individuals experience more food cue reactivity and craving compared to lean controls in both behavioral (Davis, Strachan, & Berkson, 2004; Ferriday & Brunstrom, 2011; Graham, Hoover, Ceballos, & Komogortsev, 2011; Ng & Davis, 2013; Ouwehand & Papies, 2010; Zoon, He, de Wijk, de Graaf, & Boesveldt, 2014) and functional magnetic resonance imaging (fMRI) studies (Carnell et al., 2012; Frankort et al., 2012; Giuliani, Mann, Tomiyama, & Berkman, 2014; Lawrence, Hinton, Parkinson, & Lawrence, 2012; Pursey et al., 2014; Silvers et al., 2014). However, this relationship is especially strong in individuals who are obese with BED (Greeno et al., 2000; Jarosz, Dobal, Wilson, & Schram, 2007; Ng &

Davis, 2013; Schienle, Schafer, Hermann, & Vaitl, 2009; Sobik, Hutchison, & Craighead, 2005; Walsh & Boudreau, 2003).

Indeed, although individuals with obesity and BED both share greater food cue reactivity and craving than lean controls, individuals with BED have been shown to be especially reactive to cues, as well as to their effects on eating and weight. For example, individuals with BED report experiencing stronger cravings than overweight (Carnell et al., 2012; Ng & Davis, 2013; Zeeck et al., 2011) and lean individuals (Wolz et al., 2017). Importantly, an early theoretical model posited that craving is a causal factor in binge eating (Jansen, 1998); consistently, craving is shown to precipitate overeating and binge eating in BED (Greeno et al., 2000; Ng & Davis, 2013), is associated with binge eating (Chao, Grilo, & Sinha, 2016), and can interfere with weight loss (Jakubowicz, Froy, Wainstein, & Boaz, 2012; Meule, Lutz, Vogele, & Kubler, 2012). Thus, food craving was recently proposed as a mediator between measures of problematic eating and binge eating episodes, and between measures of problematic eating and BMI (Joyner, Gearhardt, & White, 2015). Therefore, food cue reactivity and craving may affect both OB and BED/OB-BED individuals, but individuals with BED may demonstrate more impairment in this domain.

7. BED and cognitive control

In the prior two sections, we argued that emotion reactivity/dysregulation, food cue reactivity, and food craving may all be particularly important for our understanding of BED. However, emotion dysregulation and impairments in the regulation of craving in BED may simply reflect a broader deficit in cognitive control in this population. Cognitive control can be conceptualized as the ability to regulate thought and action in accordance with internal goals (Miller & Cohen, 2001), and is thought to rely on prefrontal regions, including dorsolateral and ventrolateral prefrontal cortex (dlPFC and vlPFC, together lPFC; Braver, 2012; Buhle et al., 2014; Miller & Cohen, 2001). Many models of obesity and BED emphasize impaired cognitive control in their etiology and maintenance, including impaired regulation of craving (Balodis, Grilo, & Potenza, 2015; Jansen, Havermans, & Nederkoorn, 2011; Kessler et al., 2016; van den Akker, Stewart, Antoniou, Palmberg, & Jansen, 2014; Volkow & Baler, 2015; Volkow, Wang, Fowler, Tomasi, & Baler, 2012; Volkow, Wang, Tomasi, & Baler, 2013; Ziauddeen, Alonso-Alonso, Hill, Kelley, & Kahan, 2015). Consistently, cognitive control deficits have been observed in individuals with obesity (for reviews, see Coppin, Nolan-Poupart, Jones-Gotman, & Small, 2014; Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016; Prickett, Brennan, & Stolwyk, 2015).

However, individuals with BED may experience more severe deficits than OB. Although some inconsistencies have been reported, those with BED performed worse than OB without BED on executive function batteries (Schag et al., 2013), including inhibitory control, attention, and cognitive flexibility (Boeka & Lokken, 2011; Duchesne et al., 2010; Mobbs, Iglesias, Golay, & Van der Linden, 2011; Svaldi, Naumann, Trentowska, & Schmitz, 2014; for reviews, see Balodis et al., 2015; Kessler et al., 2016, but c.f. Lavagnino et al., 2016; Wu, Hartmann, Skunde, Herzog, & Friederich, 2013). Individuals with BED were also shown to perform poorly on decision-making tasks (e.g., Danner, Ouwehand, van Haastert, Hornsveld, & de Ridder, 2012; Manwaring, Green, Myerson, Strube, & Wilfley, 2011). Further, recent work found that individuals with BED perform worse on a food-cue-based go/no-go task (Hege et al., 2015) as compared to OB controls, suggesting that any impairments in cognitive control may be amplified by food-cue-specific reactivity or in a food context. Indeed, impairment in cognitive control over food-related stimuli may specifically differentiate individuals with OB-BED from OB (for review, see Schag et al., 2013; Wu et al., 2016).

One specific form of cognitive control may be especially important for BED: the ability to regulate emotions. As noted above, negative affect is associated with binge episodes in BED (Chua et al., 2004; Greeno et al., 2000; Haedt-Matt & Keel, 2011; Leehr et al., 2015; Masheb & Grilo, 2006; Munsch et al., 2012; Stein et al., 2007). Thus, increased ability to effectively regulate emotion may be particularly protective. For example, cognitive reappraisal is an emotion regulation strategy that is part of cognitive restructuring in CBT (S. G. Hofmann, Asmundson, & Beck, 2013) and that involves changing the trajectory of a response by reinterpreting the meaning of the stimulus (Gross, 2015). Reappraisal can reduce the strength of negative emotional responses, including reduction in clinically-meaningful symptoms (Denny, Inhoff, Zerubavel, Davachi, & Ochsner, 2015; Denny & Ochsner, 2014; Gross, 2015; Ochsner & Gross, 2008) and emotion-related brain activity (Buhle et al., 2014). However, there have not yet been studies specifically testing this in BED.

Moreover, BED may also be associated with specific deficits in the regulation of craving. As noted above, craving is a potent predictor of eating, and may precipitate binge episodes in BED (Chao et al., 2016; Greeno et al., 2000; Jansen, 1998; Ng & Davis, 2013). Accordingly, the ability to regulate craving may be protective, and may further be impaired in BED. For example, using cognitive strategies such as thinking about the long-term consequence of consuming unhealthy foods can reduce self-reported craving, as well as alter its neural substrates (Giuliani et al., 2014; Giuliani, Calcott, & Berkman, 2013; Kober et al., 2010; Kober, Kross, Mischel, Hart, & Ochsner, 2010; Lopez, Onyemekwu, Hart, Ochsner, & Kober, 2015; Naqvi et al., 2015; Silvers et al., 2014; Yokum & Stice, 2013). Also, we recently found that using cognitive reappraisal strategies can also alter economic valuation of food, and that training in such strategies can change food choices and reduce caloric consumption (Boswell & Kober, 2017). Together, these findings are consistent with the idea that affective reactivity (including craving and emotion) is malleable (i.e., can be reduced over time) and can be regulated using cognitive control. Thus, although this has not yet been tested directly, it is likely that BED is associated not only with increased craving, but also with deficits in the regulation of craving, and with cognitive control more broadly.

8. Neurobiological features of BED

The data described in the prior sections suggest a role for emotion reactivity/dysregulation, food cue reactivity, food craving, and cognitive control in BED; below we discuss the emerging neuroimaging literature on these processes. Importantly, this section should be interpreted with caution, as research on the neurobiological aspects of BED is just beginning, only a handful of neuroimaging studies have been published to date, and many of them focus on reward-related constructs and include relatively small sample sizes.

For example, in a small study using the Monetary Incentive Delay Task (MID; Knutson, Adams, Fong, & Hommer, 2001), we showed that treatment-seeking obese individuals with BED exhibited lower activity in the ventral striatum (VS) during the anticipation of reward compared to lean and OB individuals without BED (Balodis, Kober et al., 2013). VS is a region implicated in reward (Floresco, 2015; Haber & Knutson, 2010; Knutson & Greer, 2008), and relative hypoactivation may suggest impaired reward processing. Follow-up analyses from the same sample further linked hypoactivation in VS to continued binge eating after treatment – that is, those individuals who continued binge eating after treatment exhibited lower activity in the VS compared to those who did not binge eat after treatment (Balodis et al., 2014). Together, these findings implicate VS hypoactivation during reward anticipation in BED, and in BED symptom severity. However, whether these findings would replicate in a larger sample or have predictive implications remains to be seen. Furthermore, differences are difficult to interpret since the stimuli were not food-related – and it has been strongly argued that it is important to focus on food-related stimuli and behavior when studying eating-related conditions (Walsh, 2011; Walsh & Boudreau, 2003).

Consistently, several studies have directly examined neural activity in BED using food cue exposure or food-related stimuli. For example, in

a study using electroencephalography (EEG) and chocolate cues, individuals who binge eat (with BED or BN) exhibited stronger event-related potentials to chocolate compared to lean controls (Wolz et al., 2017). In a similar EEG study, those with BED again exhibited larger event-related potentials compared to overweight controls in response to high-calorie food cues (Svaldi, Tuschen-Caffier, Peyk, & Blechert, 2010). In a small study using single photon emission computerized tomography (SPECT), participants viewed food images (Karhunen et al., 2000). Results showed that individuals with OB-BED vs. OB exhibited increased cerebral blood flow in the left hemisphere (Karhunen et al., 2000), including in the orbitofrontal cortex (OFC), which is involved in response to food-related stimuli (Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001). In another small study using positron emission tomography (PET), OB-BED exhibited increased dopamine release to food stimuli following methylphenidate (Wang et al., 2011). A small fMRI study reported that individuals with BED exhibited stronger activations in the VS in response to food images compared to healthy controls (Lee, Namkoong, & Jung, 2017). Another fMRI study reported greater reactivity to food stimuli in the orbitofrontal cortex in OB-BED compared to OB (Schienle et al., 2009). One fMRI study in obese individuals with “moderate binge symptoms” reported increased neural response to food taste (in VS, amygdala, and insula) that correlated with binge episodes (Filbey, Myers, & Dewitt, 2012). A study that combined fMRI and classification methods also implicated the VS in BED during food cue exposure; activity in VS accurately identified and separated individuals with BED from overweight control participants (Weygandt, Schaefer, Schienle, & Haynes, 2012).

While these findings from studies using food-related stimuli point towards specific alterations in the neural response to reward and food cues in BED — including differences between OB-BED and OB individuals, and perhaps a specific role for VS — more systematic work is required in larger samples. This may be especially true when considering the role of cognitive control in reducing emotion reactivity, food cue reactivity, and craving. Indeed, the one small fMRI study on this topic reported that OB-BED individuals exhibited lower recruitment of brain regions associated with inhibitory control, compared to OB individuals (Balodis, Molina et al., 2013). Consistently, another study using magnetoencephalography (MEG) reported that individuals with BED compared to overweight and obese individuals without BED showed reduced recruitment of the prefrontal control network during a food-based go/no-go task (Hege et al., 2015).

In sum, prior neuroimaging studies have focused primarily on reward-related processing, and have not yet directly explored the interactions between emotion, food cue reactivity, food craving, and cognitive control, which are prime candidate mechanisms in BED. Based on the work reviewed in prior sections, we propose that there may be interacting effects of stimulus-type (food vs. emotion vs. non-affective), balance of reactivity vs. regulation, and group (OB-BED vs. OB vs. lean). Specifically, we expect OB-BED and OB groups to demonstrate heightened food cue reactivity compared to lean controls, and expect OB-BED to show more emotion reactivity than OB and lean controls. Additionally, prior work has not examined whether potential cognitive control deficits might be specific to emotion vs. food-related stimuli. Exploring putative interactions between stimulus type and group is especially important, given that cognitive control deficits have been reported in BED (reviewed above; Balodis et al., 2015; Danner et al., 2012; Hege et al., 2015; Kessler et al., 2016; Svaldi et al., 2014), but it is yet unclear whether such a deficit is general or specific. Ultimately, uncovering differences in these processes across OB-BED, OB, and lean controls can help target treatments more specifically.

9. Implications for treatment of BED

There are several empirically-supported treatments for BED, including psychological and pharmacological treatments; however, they are not very effective (see below) and much remains to be learned about specific treatment components and their mechanisms of action (Craighead & Agras, 1991; Kazdin, 2008; McElroy, Guerdjikova, Mori, Munoz, & Keck, 2015; Pennesi & Wade, 2016; Vall & Wade, 2015). Moreover, there is currently a lacuna of literature examining: (1) how specific aspects of existing treatments work, (2) which variables might serve as predictors of treatment outcomes, and (3) who might benefit from a particular treatment approach (e.g. Pennesi & Wade, 2016; Vall & Wade, 2015). Consistent with the NIMH mechanism-focused strategic priorities, such knowledge could be used to refine existing treatments, and/or to develop more effective and targeted treatments, including those targeting specific impairments in BED. Specifically, if emotional reactivity, food cue reactivity, food craving, and cognitive control are important to the etiology and maintenance of BED, then altering them would be a potent treatment target. However, although such processes are sometimes addressed in BED treatments, they are rarely specifically evaluated, trained, or targeted.

9.1. Psychological treatments

Psychological treatments for BED typically instruct patients to learn and apply behavioral and/or cognitive strategies to alter their patterns of food consumption, with the goal of reducing binge-eating behavior. Although existing treatments contain components that influence emotion reactivity, food cue reactivity, craving, and cognitive control, they do not always specifically target or evaluate these processes. Although existing treatments contain components that influence emotion reactivity, food cue reactivity, craving, and cognitive control, they do not always specifically target or evaluate these processes. Here we primarily focus on cognitive behavioral therapy (CBT) and behavioral weight loss (BWL), which are considered gold-standard treatments for binge eating and obesity respectively, with the most robust and long-standing evidence base (Peat et al., 2017; Powell, Calvin, & Calvin, 2007). We also briefly review several other approaches with demonstrated preliminary efficacy for BED (e.g., third-wave approaches, interpersonal therapy; Peat et al., 2017).

CBT for BED (Butler, Chapman, Forman, & Beck, 2006; Fairburn, 2013) includes (a) behavioral techniques to alter food consumption in order to avoid food cue reactivity and craving (e.g., pre-planning regular meals), as well as (b) ongoing monitoring and problem-solving of eating-related behaviors (e.g., food diaries), and (c) strategies to regulate emotion and craving, such as cognitively changing thoughts and feelings (via cognitive restructuring; Hofmann et al., 2013; e.g., reappraisal, a form of cognitive control; Gross, 2015). In people who are OB-BED, a secondary goal for psychological treatments is reducing weight. BWL is a standard treatment for obesity, which is sometimes used for BED. BWL includes (a) psychoeducation with information about the nutritional density of foods (Brownell, 2004; Butryn, Webb, & Wadden, 2011; Grilo et al., 2011), which in turn facilitates patients' ability to implement (b) strategies like situation selection and situation modification. For example, BWL encourages patients to avoid food cue reactivity and craving for unhealthy, calorically-dense, nutrient-poor foods (e.g., by buying only healthy foods; Brownell, 2004, p. 107). In addition, BWL encourages (c) careful monitoring and problem solving of food consumption and exercise patterns (Brownell, 2004; Hartmann-Boyce et al., 2014; Wing, 2004). Importantly, BWL focuses exclusively on behavioral techniques, whereas CBT also includes cognitive skills, such as cognitive reappraisal for emotion regulation.

Although CBT and BWL are both gold-standard treatments, they have significant room for improvement in terms of efficacy. For example, CBT has mixed outcomes for reducing binge eating (Butler et al., 2006; Fairburn, 2013; Hay, 2013; Iacovino, Gredysa, Altman, & Wilfley, 2012; Vocks et al., 2010; Wilson & Zandberg, 2012) and is associated with a 20–60% relapse rate (for review, see Brown & Keel, 2012; Vocks et al., 2010). Similarly – although BWL treatment is effective for individuals who comply with its guidelines – overall it results in only 5–10% weight loss. This can improve health indicators but rarely

reverses obesity itself (Wadden et al., 2011). Furthermore, such weight losses are challenging to maintain (Franz et al., 2007; Grilo & Masheb, 2005; Wadden et al., 2011), and most individuals re-gain lost weight within 1–2 years (Weiss et al., 2007; Wing & Phelan, 2005).

Interestingly, recent work has directly compared CBT and BWL for BED and Obesity, in order to test the boundary conditions of their efficacy. For instance, one clinical trial tested BWL vs. CBT vs. control treatment for BED, and found that CBT had significantly higher binge eating remission rates, and that neither BWL nor CBT resulted in significant weight reduction (Grilo & Masheb, 2005). However, CBT more effectively reduced affective and cognitive components of BED, such as eating concern and low self-esteem (Grilo & Masheb, 2005). A follow-up trial investigated sequential BWL, CBT, and CBT + BWL treatments for BED (Grilo et al., 2011). They found that CBT produced the greatest reduction in binge eating overall, although BWL also led to reductions in binge eating. Furthermore, they found that although BWL produced the greatest initial weight reduction, binge-eating remission (via CBT) was most strongly associated with longer-term BMI reduction (Agras, Telch, Arnow, Eldredge, & Marnell, 1997; Grilo et al., 2011). These findings suggest that CBT is more effective than BWL at reducing binge-eating behavior, whereas BWL may be effective for reducing weight in the short-term, although weight loss is challenging to sustain if binge-eating continues in the long-term. Indeed, this difference in efficacy between CBT and BWL was recently confirmed by a recent review and meta-analysis (Peat et al., 2017).

What might be the components that differentiate these treatments? The behavioral techniques included in these treatments are often based in situation selection and modification (Gross, 2014). Importantly, situation selection and modification do not rely on cognitive control as much as reappraisal-based cognitive restructuring strategies (Gross, 1998), and may thus be easier to implement. Specifically, situation selection and modification involve choosing or altering one's physical environment so as to alter its emotional impact, including pre-planned avoidance of food cues (Butryn et al., 2011; Poelman et al., 2015; Spiegler & Guevremont, 1993; Weingarten, 1985). This is thought to be effective because it reduces exposure to food cues, which might otherwise lead to reactivity, craving, and eating. Indeed, it has been recently suggested that such antecedent-focused skills may be effective because they reduce the likelihood of temptations that may require the use of limited cognitive control resources (e.g., Duckworth, Gendler, & Gross, 2016). Consistently, ecological momentary assessment (EMA) studies have shown that individuals who successfully resist temptation often do so by avoiding the temptations altogether; successful self-controllers report fewer encounters with tempting stimuli, suggesting that they are not necessarily better at resisting temptation (cognitive control) but instead may be better at avoiding temptation (Hofman, Baumeister, Forstter, & Vohs, 2012; Hofmann, Adriaanse, Vohs, & Baumeister, 2014). Thus, BWL – which relies on such behavioral strategies – may be simpler to implement in the short term, which might explain its efficacy for short-term weight loss.

However, environmental factors may interfere with long-term reliance upon situation selection and modification strategies. Indeed, unhealthy calorically-dense food and associated food cues (e.g., most food advertisements) have become ubiquitous, in what some have termed a “toxic food environment”; Brownell & Horgen, 2004; Wadden, Brownell, & Foster, 2002). For instance, following the instructions of BWL, one might maintain a home that is well-stocked with healthy options (Brownell, 2004, p. 107), but still occasionally encounter food advertisements, drive past McDonald's, or need to resist the temptation to snack on the homemade cookies a colleague brought to work. In this way, situation modification strategies may only be effective in a controllable environment and in the short-term, which can be especially challenging for individuals attempting to lose weight or maintain weight loss.

In contrast, cognitive strategies in CBT are typically based in cognitive change. As thoroughly investigated by emotion regulation theorists, cognitive change occurs after emotions and craving have already been consciously experienced (Gross, 2014). These strategies, especially cognitive reappraisal, are applied later in the emotion generation process, and thus require cognitive control. Indeed, cognitive reappraisal is thought to rely on a range of cognitive abilities, including working memory and set-shifting (McRae, Jacobs, Ray, John, & Gross, 2012; Ochsner & Gross, 2005). Consistently, our neuroimaging meta-analysis has demonstrated that the use of cognitive reappraisal to regulate emotions involves the recruitment of prefrontal brain regions that are often associated with cognitive control (Buhle et al., 2014). Use of such strategies may therefore be cognitively costly (Botvinick & Braver, 2015; Heatherton & Wagner, 2011), but also necessary, given the ubiquity of unhealthy food options and food cues – especially as an adjunct to less-costly behavioral strategies. In CBT for eating disorders, such skills are taught to specifically target craving, as well as cognitive over-evaluation of dieting and weight/shape that may increase temptation to binge (Fairburn, 2013; Fairburn, Cooper, Shafran, & Wilson, 2008; Garner, Vitousek, & Pike, 1997; Wilson, Fairburn, & Agras, 1997). Thus, CBT-based strategies such as cognitive reappraisal may serve as important, and necessary, backup in inevitable situations filled with temptation, where situation selection and modification are not possible. The inclusion of such strategies in CBT may therefore explain its greater efficacy in the long term, compared to BWL.

Ultimately, treatments for BED could be improved by evaluating and targeting core processes involved in BED, including emotion reactivity, food cue reactivity, craving, and cognitive control. For example, videogames or computer-based training that includes practice in cognitive control, may offer mechanism-focused skills learning that can complement existing treatment approaches (Juarascio, Manasse, Espel, Kerrigan, & Forman, 2015). However, there is little evidence of cognitive control training generalizing to other domains (e.g., Melby-Lervåg & Hulme, 2013). In contrast, explicit training in cognitive reappraisal may enhance cognitive control, specifically in response to emotional and appetitive stimuli (Denny et al., 2015; Denny & Ochsner, 2014; Stice et al., 2015). Furthermore, training in the regulation of craving improves food choices and reduces unhealthy food consumption, which could improve treatment outcomes (Boswell & Kober, In Revision). This type of mechanism-specific training has the distinct advantage of not requiring generalization when applied to obesity and eating-related disorders.

Indeed, enhancing cognitive reappraisal training specifically during CBT treatment has been shown to improve outcomes for depression (Berking et al., 2008; Berking, Ebert, Cuijpers, & Hofmann, 2013), but this has not yet been investigated for other psychological disorders, including eating disorders. Although CBT includes cognitive reappraisal techniques, thus far there has been no work examining the effects of cognitive training in regulation of craving or regulation of emotion on eating behavior in BED. Further, no work has compared whether cognitive reappraisal, behavioral strategies, other strategies, or a combination of these approaches is more effective at reducing weight or binge eating. Future work should examine whether evaluation of and training in cognitive reappraisal and other cognitive control skills might improve treatment outcomes for BED.

Interestingly, newly investigated “third-wave” acceptance- and mindfulness-based treatments for BED provide an alternative approach to addressing emotion and craving. These treatments teach the acceptance of strong affective experiences; this might specifically include observing, describing, and choosing to not act upon feelings of distress or craving. These treatments include mindfulness-based therapies (MBTs; Katterman, Kleinman, Hood, Nackers, & Corsica, 2014; Kristeller, Wolever, & Sheets, 2014; Wanden-Berghe, Sanz-Valero, & Wanden-Berghe, 2011) and dialectical behavior therapy (DBT) for BED (Chen, Matthews, Allen, Kuo, & Linehan, 2008; Telch, Agras, & Linehan, 2001; Wisiniewski & Kelly, 2003). Such approaches have been shown to bolster long-term weight loss and remission rates for eating disorders and obesity (Forman et al., 2013; Forman & Butryn, 2015;

Forman, Butryn, Manasse, & Bradley, 2015; Katterman et al., 2014; Kristeller et al., 2014; O'Reilly, Cook, Spruijt-Metz, & Black, 2014; Wanden-Berghe et al., 2011). Furthermore, recent brain imaging work has found that unlike cognitive reappraisal, acceptance-based strategies may not rely on recruitment of prefrontal brain regions associated with cognitive control (Kober, Brewer, Tuit, & Sinha, 2017; Kober, Buhle, Weber, Ochsner, & Wager, in revisions; Westbrook et al., 2013). As such, acceptance-based treatments could be particularly effective for individuals who experience deficits in cognitive control, as has been observed in BED. Thus, individuals who are unable to learn cognitive control strategies (e.g., reappraisal) may uniquely benefit from mindfulness-based approaches. Future work should compare acceptance-based and reappraisal-based strategies for regulating negative affect and craving, as well as compare these treatments in obesity and OB-BED.

In addition, alternative approaches such as interpersonal therapy (IPT) have demonstrated preliminary efficacy for BED (McElroy, Guerdjikova, Mori, Munoz et al., 2015; Peat et al., 2017). Indeed, IPT and CBT demonstrated comparable efficacy in some studies, despite employing different techniques and clinical targets (McElroy, Guerdjikova, Mori, Munoz et al., 2015; Peat et al., 2017). Instead of addressing emotions, craving, or eating behavior directly, IPT focuses on problem resolution in four domains: grief, interpersonal role disputes, role transitions, and interpersonal deficits. A small set of clinical trials have shown that IPT and CBT similarly reduce binge eating, including at 1-year follow-up (Wilfley et al., 2002), 2-year follow-up (Wilson, Wilfley, Agras, & Bryson, 2010), and 4-year follow-up (Hilbert et al., 2012). However, surprisingly little work has examined the mechanisms of change in IPT. Some have suggested that IPT may operate by enhancing social support, decreasing interpersonal stressors, facilitating emotional processing, and improving interpersonal skills (for review, see Lipsitz & Markowitz, 2013). These processes may decrease negative emotion and increase emotion regulation indirectly. For instance, social support may enhance emotion regulation, emotional processing may reduce emotional reactivity, and reductions in interpersonal stress and improved interpersonal skills may reduce the frequency of negative affect. In the context of BED, reductions in emotional reactivity and stress may reduce the likelihood that negative affect will precipitate binge eating, while increases in emotion regulation may also enhance the ability to regulate craving. However, future work is necessary to directly test these potential mechanisms of change and their effects on reducing binge eating.

9.2. Pharmacological treatments

Although overall less effective than psychological treatments, several pharmacological treatments have been tested for BED. A recent meta-analysis estimated that pharmacological treatments for BED reduce binge eating with a small-to-medium effect size (Vocks et al., 2010). However, these treatments do not reduce cognitive components of BED, such as shape or weight concern (McElroy, Guerdjikova, Mori, & Keck, 2015; Reas & Grilo, 2008, 2014; Vocks et al., 2010). Further, many of the pharmacotherapy trials for BED report strong placebo response rates and adverse side effects, and have been tested over relatively short durations (e.g., average length of psychological treatment trials: 16 weeks vs. medication trials: 12 weeks; Brownley et al., 2016). Crucially, pharmacotherapies do not improve BED outcomes when combined with CBT, and there are little data on long-term outcomes of medications (Grilo, Masheb, & Wilson, 2005; Reas & Grilo, 2008). Thus, although some pharmacotherapies have shown small-to-moderate efficacy for treating BED in the short term, they are not considered gold-standard treatments.

One reason for the limited efficacy of pharmacotherapies may be that their development was not mechanism-driven and they do not target BED-specific psychological or neural mechanisms. Instead, early trials merely tested pharmacotherapies that had been efficacious for related conditions, such as BN and obesity. For example, an early medication tested for BED is fluoxetine, a selective serotonin reuptake inhibitor (SSRI) that was previously shown to be effective for (and is still used to treat) depression and other psychological disorders, including BN. Although some clinical trials of fluoxetine found a reduction in binge eating behavior (e.g., Arnold et al., 2002), others found no reduction in binge eating or weight, accompanied by a high placebo response rate (Grilo et al., 2005; Ricca et al., 2001). These studies had a relatively short duration (6–16 weeks of treatment), and no randomized controlled maintenance study for an SSRI has yet been conducted.

A second medication tested for BED is topiramate, an anti-epileptic agent with a broad mechanism of action (including activating $GABA_A$ and AMPA/NMDA glutamate receptors) that has been previously prescribed for weight loss, before it was tested with BED. Topiramate effectively reduced binge eating and weight in several trials (Appolinario, Fontenelle, Papelbaum, Bueno, & Coutinho, 2002; McElroy et al., 2003), but its use is accompanied by especially high rates of dropout and adverse effects. A third group of medications tested for BED was previously used for obesity, such as sibutramine (a selective norepinephrine reuptake inhibitor, now off the market) and orlistat (a lipase inhibitor; Golay et al., 2005; Grilo & White, 2013). These medications have not been shown to reduce binge eating frequency during 16–24 weeks of treatment, and have a very modest effect on weight (0–3 kg; Golay et al., 2005; Grilo & White, 2013).

Indeed, only a few of the pharmacological treatments that have been tested have a proposed mechanism that is related to BED symptomatology, such as food cue reactivity, craving, or cognitive control. Still, these pharmacotherapies were not developed with any BED-related mechanisms in mind, and were only tested on BED after showing efficacy for other conditions with similar hypothesized mechanisms. For instance, buproprion is a norepinephrine-dopamine reuptake inhibitor that has been found to reduce cravings for nicotine (Brody et al., 2004) and food (Jain et al., 2002). As such, it has been prescribed as a smoking cessation aid (P. Wu, Wilson, Dimoulas, & Mills, 2006) and as a treatment for obesity (Greenway et al., 2010; Vetter, Faulconbridge, Webb, & Wadden, 2010). However, when tested for BED, 8 weeks of treatment with buproprion did not lead to weight loss or to a change in binge eating, food craving, eating disorder symptomatology, or depressive symptoms (White & Grilo, 2013).

In comparison, lisdexamfetamine (LDX) is a d-amphetamine prodrug that typically serves as a treatment for attention deficit hyperactivity disorder (ADHD; Cunill, Castells, Tobias, & Capella, 2015). LDX is thought to enhance cognitive control, which could help individuals with BED resist binge eating. In two relatively large trials for LDX, there were reductions in binge eating episodes compared to placebo over 11 weeks of treatment (McElroy, Hudson, Ferriera-Cornwall et al., 2015; McElroy, Hudson, Mitchell et al., 2015), suggesting potential promise for this treatment. Indeed, although not approved for weight loss, LDX was the first pharmacological treatment to receive regulatory approval for the treatment of BED, and large-scale clinical trials are ongoing. However, LDX is a Schedule II drug due to its high abuse potential, suggesting that caution is necessary. For instance, a recent large-scale trial found that, after a 12-week LDX treatment that reduced binge eating episodes, continued LDX treatment over 26-weeks had a lower relapse rate compared to placebo (Hudson, McElroy, Ferriera-Cornwall, Radewonuk, & Gasior, 2017). This suggests that there may be some long-term efficacy of continued LDX treatment, but also a tradeoff between the potential for relapse and the potential for abuse.

Overall, there is some evidence that pharmacotherapies may reduce binge eating. However, this literature has several major limitations, including that many medications were tested with short treatment duration, and almost no data exists regarding long-term medication effects or long-term outcomes. Further, few pharmacotherapies are targeted towards specific mechanisms involved in BED. Even LDX, the first pharmacotherapy to receive regulatory approval, has not been shown to improve cognitive control, regulation of emotion, or

regulation of craving in BED. Additionally, to our knowledge, no pharmacotherapies have been tested for individuals who do not respond to BWL or CBT, which may be an important group of individuals to target with additional treatment. Ultimately, psychological treatments continue to provide the most effective outcomes for this condition, and future work is necessary to examine when, how, and for whom pharmacotherapies might improve treatment outcomes. It is possible that the most effective treatments – either pharmacological or psychological – will be those that directly target mechanisms that are central to BED.

10. Conclusions

Research on BED is in its infancy, commensurate with its status as a new eating disorder diagnosis in DSM-5. In this paper, we argued that BED and obesity are linked but distinct conditions, and that BED is uniquely associated with greater negative affect, emotional reactivity and dysregulation, altered affective and neural responses to food cues, and cognitive control deficits, above and beyond obesity. Further, food craving may be one specific affective process that is increased in this group, which may relate causally to increased binge eating behavior, along with deficits in the regulation of craving. Neuroimaging studies suggest that neural responses to food – and to rewards more broadly – may be altered, and uniquely relate to binge eating symptoms. However, many of the studies reviewed herein were small and preliminary, and findings would need to be replicated with larger samples.

Additionally, we reviewed existing psychological and pharmacological treatment approaches for BED; specifically, we suggested that targeting cognitive control over emotional reactivity, food cue reactivity, and craving may be core processes of these treatments that underlie differences in their efficacy. Finally, we proposed avenues for future research and treatment development based on targeting mechanisms that may be specifically impaired in BED as opposed to obesity. Specifically, we hope that future studies would directly investigate the interaction between affective reactivity and cognitive control in BED, by testing the regulation of negative emotion and craving. This may be useful for improving our understanding of the etiology of BED, and for improving treatment outcomes.

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Conflict of interest

The authors declare no conflict of interest.

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