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Emotion Regulation Strategies in Binge Eating Disorder: Rumination, Distress Tolerance, and Expectancies for Eating

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EMOTION REGULATION STRATEGIES IN BINGE EATING DISORDER:
RUMINATION, DISTRESS TOLERANCE, AND EXPECTANCIES FOR EATING

A Dissertation Presented

by

Lilya Sitnikov

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of

The University of Vermont

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for the Degree of Doctor of Philosophy
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Accepted by the Faculty of the Graduate College, The University of Vermont, in partial fulfillment of the requirements for the degree of Doctor of Philosophy, specializing in Psychology.

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ABSTRACT

Binge Eating Disorder (BED) is characterized by recurrent episodes of binge eating without the use of compensatory behaviors. Functional accounts of BED propose that negative affect is an antecedent to binge eating because binge eating serves to alleviate negative affect. However, previous studies investigating the association between negative affect and binge eating have yielded inconsistent findings, perhaps due to individual vulnerability factors that moderate the effects of negative affect on binge eating behavior. As one candidate, the current study investigated emotion regulation strategies that may be implicated in the maintenance of binge eating in BED, particularly under conditions of negative affect: brooding rumination, distress tolerance, and mood-related expectancies for eating. These emotion regulation strategies were: a) compared in 38 women with BED vs. 36 non-eating disordered female controls, b) examined in relation to markers of current binge eating severity among BED women, and c) used as predictors of caloric intake and urge to eat in response to a personally-relevant dysphoric mood induction upon presentation of snack foods in a “taste task.” Results revealed that women with BED endorsed higher brooding rumination, more positive expectancies that eating serves to ameliorate negative affect, and lower distress tolerance than controls. Among women with BED, higher brooding rumination was associated with greater binge eating severity, and stronger expectancies that eating reduces negative affect were associated with more frequent binge eating episodes and greater urge to eat in response to depression. Surprisingly, better distress tolerance was associated with more frequent binge eating episodes. Women with BED consumed more calories and reported greater loss of control as well as a greater sense of guilt in response to the taste task relative to control participants. Contrary to hypothesis, there were no direct or indirect effects of any of the three emotion regulation strategies on change in urge to eat or calories consumed on the taste task following sad mood induction in BED women. In controls, better distress tolerance and stronger expectancies that eating alleviates negative affect were associated with decreased caloric intake on the taste task after mood induction. Overall, these findings highlight the importance of considering trans-diagnostic processes in BED as well as the need to identify other theoretically-relevant factors that contribute to the cognitive and behavioral features of BED. Limitations and directions for future studies are discussed.

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INTRODUCTION

Binge Eating Disorder (BED) is a serious and debilitating mental health problem with significant social costs. Core features of the disorder are binge eating episodes, defined as consuming an objectively large amount of food in a short period of time (i.e., behavioral disturbance) while experiencing a loss of control over eating (i.e., cognitive/affective component). According to estimates from the National Comorbidity Survey Replication, an estimated 3.5% of American women engage in recurrent binge eating (Hudson, Hiripib, Pope, & Kessler, 2007). Rates of binge eating are particularly high among undergraduate women, with prevalence estimates ranging between 32% (Keel, Baxter, Heatherton, & Joiner, 2007) and 48% (Striegel-Moore, Silberstein, Grunberg, & Rodin, 1990). Recurrent binge eating is considered a major health concern as it adversely affects psychological functioning, is associated with lower quality of life, and predisposes individuals to the morbidity and mortality associated with obesity (Johnson, Spitzer, & Williams, 2001).

Although a number of factors, such as personality characteristics, physiological states, and cognitive styles have been conceptually linked to BED, research is needed to identify which vulnerability factors reliably discriminate BED from obesity and other eating disorders and are uniquely implicated in the etiology, maintenance, and course of BED. Studies are also needed to elucidate how putative risk factors contribute to features of BED. Theoretical accounts of eating pathology and empirical investigations have attempted to link negative affect and binge eating. Binge eating may function to

alleviate/avoid/escape negative affect and thinking by shifting attention to eating and is, therefore, negatively reinforced. For instance, the affect regulation model of binge eating (Cooper, Wells & Todd, 2004; Heatherton & Baumeister, 1991) conceptualizes binge eating as an avoidant coping strategy aimed at temporarily escaping negative affect and reducing aversive self-awareness. In contrast, the spiral model of eating disorder development (Heatherton & Polivy, 1992) proposes that negative affect results from the decrements in self-esteem experienced after repeated failures to successfully restrain eating in chronic dieters. According to this model, negative feelings associated with recurrent dietary failure lead to binge eating, followed by renewed attempts at dietary restraint, leading to a “spiral” of behaviors that characterize Bulimia Nervosa (BN).

These models may explain recurrent binge eating among individuals with BN, yet their applicability to individuals with BED remains unknown. Empirical studies using BED samples have yielded inconsistent findings, with some studies reporting an *increase* in negative affect following binge eating episodes in BED. Laboratory studies examining the number of calories consumed by individuals with BED following a negative mood induction have also yielded contradictory findings. Thus, it is possible that binge eating may help regulate negative affect for some individuals with BED, whereas binge eating may serve a different function for others.

To understand the functional relationship between binge eating and negative affect, it is necessary to consider processes that may impact the duration and intensity of negative affect as well as perceived ability to withstand and effectively manage negative

emotions. Cognitive and affective processes may prolong negative affect and/or precipitate the use of escape-based coping strategies, such as binge eating, to ameliorate affective and cognitive states perceived as intolerable, unacceptable, or unmanageable. Consequently, the current study will investigate individual differences in facets of emotion regulation in relation to BED.

It is proposed that cognitive-affective factors, such as brooding rumination (i.e., the tendency to repetitively focus on the negative consequences of one's mood) and low distress tolerance (i.e., inability to tolerate negative affect) may a) characterize persons with BED and serve to maintain binge eating, b) are associated with severity of binge eating and propensity to engage in emotional eating, and c) help identify individuals with BED who are most likely to respond to negative affect with increased caloric consumption. Brooding rumination and emotion regulation may be related to binge eating and may precipitate binge eating in persons with BED experiencing dysphoric mood by increasing reactivity to or prolonging and exacerbating transient negative affect. Thus, for persons with BED, binge eating may serve to escape or distract from thoughts and emotions that are experienced as aversive. This relation may be particularly salient for individuals who expect eating to regulate negative affect.

The current study aimed to elucidate the role of affective (i.e., distress tolerance and perceived ability to use effective distress regulation strategies) and cognitive (i.e., brooding rumination and negative reinforcement expectancies for eating) processes in BED by examining whether low distress tolerance, positive eating expectancies, and

high levels of brooding rumination a) characterize individuals with BED relative to non-eating disordered individuals and b) are associated with binge eating severity. It is also proposed that brooding rumination, distress tolerance, and positive eating expectancies will predict urge to eat and caloric intake in response to a dysphoric mood induction in BED participants relative to control participants. In particular, we expected brooding rumination to affect urge to eat and caloric intake both directly and indirectly.

Specifically, we predicted that greater frequency of brooding rumination would predict stronger reactivity to sad mood induction, which would in turn account for significant variability in caloric intake in BED women (Figure 1). Moreover, it was proposed that, for BED-diagnosed participants, negative reinforcement eating expectancies would moderate the association between ruminative brooding (as well as the association between distress tolerance) and urge to eat and calories consumed after a dysphoric mood induction (Figure 1). That is, the combination of greater negative reinforcement eating expectancies and brooding rumination/distress tolerance would promote binge eating behaviors in individuals with BED who experience transient dysphoric mood. Eating expectancies are the proposed moderator because they may shape the behavioral expression of underlying emotion regulation difficulties and promote the use of food to regulate mood. It is proposed that negative reinforcement eating expectancies may place an individual with BED who is characterized by high levels of brooding rumination and low distress tolerance at increased risk for a binge eating episode in response to dysphoric mood.

The following sections first describe the syndrome of BED, highlighting differences between BED and other eating disorders. Subsequently, prior research on negative affect, including relevant theoretical accounts and empirical findings, will be reviewed. Limitations of the extant literature and potential contributions of the proposed study will be highlighted.

Binge Eating Disorder (BED)

Key Characteristics and DSM-5 Criteria

BED was introduced in the fourth edition of the DSM (DSM-IV; American Psychiatric Association, 1994) as a provisional diagnostic category in need of further study within the spectrum of eating disorders not otherwise specified (EDNOS). BED was formally included as a diagnostic category in the 5th edition of the DSM (American Psychiatric Association, 2013). Binge eating is the hallmark of BED, defined as consuming an objectively large amount of food in a short period of time while experiencing a loss of control over eating. The DSM-5 criteria also specify that individuals must experience at least three of the following behavioral indicators of impaired control: (1) eating rapidly; (2) eating until uncomfortably full; (3) eating large amounts of food when not hungry; (4) eating alone due to embarrassment; and (5) feeling depressed, disgusted, or guilty after overeating. Individuals must experience significant distress and abstain from compensatory weight-control behaviors (e.g., self-induced vomiting, laxative use, excessive exercise, fasting, etc.) characteristic of Bulimia Nervosa (BN) and the Bingeing-Purging subtype of Anorexia Nervosa (AN). DSM-5 BED

criteria, which were followed in the current study, specify that binge eating must occur, on average, at least once a week for three months.

Epidemiology

Because the diagnostic category of BED first appeared in the DSM-IV in the “Disorders for Future Study” section, very few epidemiological studies of BED have been conducted, and many of those that have been conducted are limited by methodology (for a review see Striegel-Moore & Franko, 2003). One early field trial using self-report questionnaires as a measure of BED found point prevalence estimates of approximately 2.5% for women and 1.1% for men (Spitzer et al., 1992). A recent study conducted by Grucza, Przybeck, and Cloninger (2007) found a 6.6% prevalence in a community sample including both men and women. These rates likely overestimate the true prevalence as research suggests that use of self-report questionnaires yields false-positives for eating disorder diagnoses (Fairburn & Beglin, 1990). Two studies conducted in Australia using community samples assessed by interview reported BED point-prevalence of 3.3% among women and 0.8% among men (Kinzl, Traweger, Trefalt, Mangweth, & Biebl, 1999a; Kinzl, Traweger, Trefalt, Mangweth, & Biebl, 1999b) and a combined rate of 1% (Hay, 1998). Using interview-based diagnostic classification data from the World Health Organization World Mental Health Survey Initiative, Kessler and colleagues (2013) estimated the lifetime prevalence of BED to be 1.9% (range of .2 to 4.7%) across the 14 countries included in the survey. In the United States, approximately 8.5-million people are estimated to have BED (Hudson, Hiripi, Pope, & Kessler 2007),

with lifetime prevalence of 2.6% and 12-month prevalence of 1.2%. The lifetime prevalence of BED in community samples is estimated to be between 2-5% (e.g., de-Zwaan, 1997; Dingemans et al., 2002). However, rates of BED may be as high as 20 to 40% among clinical samples of individuals enrolled in weight loss programs (Marcus, 1995; Spitzer et al., 1993).

In terms of demographic correlates of BED, preliminary research on sex differences is mixed. Some research suggests that no significant differences exist between men and women on rates of BED in an exclusively Caucasian sample (Streigel-Moore & Franko, 2003) or in a large community-representative sample (Grucza et al., 2007) whereas a different study found a higher prevalence of BED in women than in men by a ratio of 3:2 (Marcus, 1995). Data from the National Comorbidity Survey Replication (Hudson et al., 2007) revealed higher rates of BED in women (3.5% in women versus 2.0% in men) and greater lifetime prevalence of sub-threshold binge eating in men (1.9 versus 0.6%). Additionally, preliminary research suggests no differences between ethnic groups in rates of BED (Grucza et al., 2007; Spitzer et al., 1992), although research in this area is extremely limited and almost exclusively compares Black and White individuals (Streigel-Moore & Franko, 2003). Grucza et al. (2007) found no differences in BED prevalence between individuals age 45 or below and those above age 45, between married and non-married individuals, or between individuals with a college degree and those without. The average age of onset for BED is late adolescence or early 20s

(Fairburn, 1995; Hudson et al.), and the average duration is 8.1 years (Hudson et al., 2006).

Binge eating severity in BED is positively correlated with psychological distress, interpersonal problems, depression, and low self-esteem (e.g., Telch & Agras, 1994; Yanovski, Nelson, Dubbert, & Spitzer, 1993). In the National Comorbidity Survey Replication (Hudson et al. 2007), 62.8% of persons diagnosed with BED reported at least some impairment in one of the domains assessed, with 18.% reporting severe impairment in at least one domain (i.e., work, social functioning). BED is frequently comorbid with other Axis I and Axis II disorders. Individuals diagnosed with BED also have significantly higher lifetime rates of panic disorder, personality disorders, and BN relative to individuals without BED. Hudson and colleagues reported that 78.9% of those with BED and 63.6% with sub-threshold BED met criteria for at least one additional Axis I disorder (based DSM-IV criteria). Grilo, White, and Masheb (2009) reported that 73.8% of patients with BED had at least one additional lifetime psychiatric disorder and 43.1% had at least one current psychiatric disorder. Mood (54.2%), anxiety (37.1%), and substance use (24.8%) disorders were the most common lifetime diagnoses. For current comorbidity, mood (26.0%) and anxiety (24.5%) disorders were most common. BED patients with current comorbidity also had significantly higher levels of current eating disorder psychopathology and negative affect and lower self-esteem relative to patients with BED with either lifetime (non-current) or no psychiatric histories. Wifely and colleagues (2000) reported that patients with BED commonly had comorbid personality

disorders: obsessive-compulsive personality disorder (14%), avoidant personality disorder (12%), and borderline personality disorder (9%).

BED and Other Eating Disorders

Recurrent binge eating is the core behavioral feature of both BN and BED. Two recent reviews of the available literature on BED concluded that BED is distinct from other eating disorders and obesity (Holm-Denoma et al., 2007; Wonderlich, Gordon, Mitchell, Crosby, & Engel; 2009). For example, Holm-Denoma and colleagues (2007) reported that individuals with BED differ from individuals with BN in clinical presentation, patterns of comorbidity, demographic features (e.g., age, gender distribution, BMI), and response to treatment. Laboratory studies and naturalistic observations reliably demonstrate that individuals with BN and BED differ in terms of patterns of binge eating and binge characteristics (e.g., calorie intake, types of binge food, levels of restraint; Walsh & Boudreau, 2003). For example, individuals with BN consume more calories during a binge than individuals with BED, but they exhibit greater dietary restraint during nonbinge meals (Wilfley, Schwartz, Spurrell, & Fairburn, 2000). In contrast, persons with BED demonstrate a pattern of chaotic eating and general overeating (Wilfley, Bishop, Wilson, & Agras, 2007). Interestingly, during binges, individuals with BN eat primarily carbohydrate-rich foods, such as sweets and desserts, whereas individuals with BED eat a full range of foods, with a higher percentage of calories from fat (Castonguay, Eldredge, & Agras, 1995). The conceptual distinction between BN and BED was also supported in latent class analyses and taxometric studies,

which conclude that both disorders are distinct categories independent from other eating disorders and from normality (e.g., Crow, Agras, Halmi, Mitchell, & Kraemer, 2002; Williamson et al., 2002).

Negative Affect

Conceptual Overview

Theoretical accounts and empirical studies consistently link binge eating and aversive mood states. Negative affect, broadly defined as general subjective distress, is an umbrella term for a range of unpleasant mood states, including irritability, sadness, anxiety, and anger (Watson & Clark, 1984). Individuals demonstrate stability in their propensity to experience negative affect over time (Watson & Walker, 1996). Individual differences in this mood-dispositional dimension influence risk for developing a variety of psychiatric conditions, including eating disorders (Clark, Watson, & Mineka, 1994; Leon, Fulkerson, Perry, Keel, & Klump, 1999). A review of risk and maintenance factors for eating pathology concluded that negative affect is a risk factor for disordered eating and a causal maintenance factor for bulimic symptoms, particularly binge eating (Stice, 2002). Measelle, Stice, & Hogansen (2006) also demonstrated that depressive symptoms predicted disordered eating over a 4-year period among adolescent girls, but the reverse was not true. Similarly, in trying to tease apart the relation between dieting and binge eating, Goldschmidt, Wall, Loth, Le Grange, Neumark-Stainzer (2012) found that depressive symptoms and low self-esteem predicted onset of binge eating beyond the effects of dieting in a large population-based sample over 5 year follow-up periods

spanning from early adolescents to middle young adulthood. Importantly, Burton, Stice, Bearman, & Rohde (2007) reported that an intervention aimed to specifically target depressive symptoms in women with eating disorders yielded clinically significant reductions in binge eating and purging. Difficulties regulating negative affect and individual characteristics that potentiate the experience of negative affect in response to stressful events or that limit an individual's ability to use adaptive mood-regulation strategies may serve to maintain binge eating among individuals with BED.

Theoretical Accounts of Binge Eating

Affect regulation models of binge eating posit that binge eating provides comfort, distraction, and/or relief from aversive affective states (Castonguay et al., 1995; Cooper et al., 2005; Heatherton & Baumeister, 1991; Fox & Power, 2009, Stice & Agras, 1998). Because functional accounts of binge eating were developed to explain binge eating in the context of bulimic pathology, they were developed to account for binge eating that alternates with restraint and/or compensatory behaviors and may or may not generalize to binge eating in BED. Thus, theoretical accounts of binge eating consider culturally shaped appearance concerns, body dissatisfaction, and escalating dieting attempts as key pathways to both negative affect and binge eating (Stice, 1994; 2001). The spiral model of eating disorders (Polivy & Herman, 1993) posits that excessive dieting (dietary restraint) and cognitive restraint (preoccupation with control over eating) will eventually lead to disinhibited eating and violation of dietary rules. Small violations of dietary rules are perceived as inconsistent with one's goals and are attributed to stable, internal, and

global causes. Such attributions result in negative mood, which further disinhibits control over eating, leading to binge eating. Once binge eating occurs, compensatory behaviors (e.g., fasting, purging, etc.) serve to “make-up” for calories consumed during a binge. Renewed attempts at dieting maintain the spiral of behaviors characteristics of BN (Grillo & Shiffman, 1994; Polivy & Herman, 1993).

However, the impact of dieting on binge eating problems has primarily been examined using individuals with BN or community samples, with the relevance of dieting history and dietary restraint in the etiology of BED extrapolated. Differences between BN and BED suggest that this extrapolation may not be appropriate. Individuals diagnosed with BED demonstrate lower dietary restraint between binge eating episodes than persons with BN (Castonguay et al., 1995). Moreover, a significant history of repeated dieting attempts precedes binge eating for only a subgroup of individuals with BED (Grilo & Masheb, 2000). Howard and Porzelius (1999) reviewed retrospective and prospective studies on the role of dieting in BED and concluded that a significant number (i.e., 38% to 54%) of individuals with BED reported that binge eating began before dieting. Across studies, individuals with BED reported a decrease in binge eating during the most restrictive period of the diet and an increase in binge eating during less restrictive phases of weight loss treatment. It is possible that negative affect that is not directly linked to dieting or to striving to meet appearance-related standards also triggers binge eating among individuals with BED (Castonguay et al., 1995).

The “escape” from awareness model (Heatherton & Baumeister, 1991) provides a theoretical framework for considering the functional relationship between negative affect and binge eating in the absence of compensatory behaviors and, therefore, may generalize to BED. Binge eating is conceptualized as an avoidant coping strategy that is aimed at escaping aversive and negative affective states. Aversive self-awareness (i.e., awareness of one’s own negative feelings and reflection on others’ perceived negative feelings towards the self) follows when shortcomings in valued domains are interpreted as personal failures and are attributed to aspects of the self rather than to external factors. In turn, aversive self-awareness results in negative affective states. To reduce negative affect, individuals are motivated to engage in behaviors aimed at reducing aversive self-awareness. A narrow focus of attention on immediate and concrete environmental stimuli diverts attention away from self-evaluations, comparisons against expectations, and contemplations about the implications of one’s actions; thus, attention is directed away from internal experiences (i.e., emotions and cognitions) perceived as aversive, thus temporarily reducing negative affect. However, because meaningful thought processes are suspended, this cognitive narrowing also results in behavioral disinhibition (e.g., control over eating is abandoned because the meaning and consequences of one’s actions are not considered). A study by Blackburn, Johnston, Blampied, Popp, and Kallen (2006) tested the applicability of the escape model to binge eating in a non-clinical sample of 129 women who self-identified as dieters. The findings supported the hypothesized relationships between the variables predicted in the theory. Moreover, although dieting

was included in the original model, the findings did not support the importance of dietary restraint as a precursor to binge eating. In contrast to escape theory, the study found that dietary restraint was not significantly associated with binge eating. It may be, therefore, that the processes explicated in the escape model generalize to individuals with BED who do not engage in chronic dietary restraint. Two similar models of bulimic symptoms were proposed by Cooper et al. (2004) and Waller, Kennerley, and Ohanian (2007). These cognitive-affective models conceptualize binge eating as an affect regulation strategy when external events trigger negative self-beliefs/schemas, which in turn facilitate negative emotions. Binge eating allows the individual to disengage from negative affect and is particularly likely to occur among individuals who hold positive beliefs about the efficacy of eating in reducing distress. The authors further elaborate that compensatory behaviors are driven by the cognitive dissonance created by the discrepancy between positive beliefs about impact of eating on mood and negative beliefs about the effect of eating on weight/shape. A somewhat different functional account was offered by Wonderlich, Mitchell, Peterson, and Crow (2001). According to their Integrated Cognitive-Affective (ICAT) model, a person with BN experiences a discrepancy between the 'ideal' (i.e., thin) and 'perceived' (i.e., not thin enough) self, which creates depression/anxiety. Binge eating in the context of this model functions to reduce awareness of the self and associated negative emotions. Consistent with this model, Wonderlich et al. (2008) found that negative mood states mediated relations between bulimic status and binge eating. Likewise, Dunkley and Grilo (2007) reported that the

relation between self-criticism and binge eating was partially mediated by low self-esteem and depressive symptoms in women with BED.

Across affect regulation models, different precursors/pathways are thought to precipitate aversive mood states. However, negative affect is consistently conceptualized as an antecedent and reinforcing condition for binge eating whereby binge eating functions to alleviate negative affect and is negatively reinforced. Thus, whether negative affect is driven by body dissatisfaction (Stice, 1994), guilt and shame associated with repeated failures to successfully restrain eating (Heatherton & Polivy, 1992), stressful life events, or other potentiating factors; across affect regulation models, binge eating is rooted in attempts to regulate aversive emotional states (Cooper et al., 2004; Waller et al., 2007).

Research Findings

A number of correlational, retrospective, prospective, and experimental studies have examined the association between negative affect and binge eating. Overall, studies link both trait-level depressive symptoms and state-level acute negative mood to binge eating. More severe binge eating is related to higher levels of depressive symptoms and self-rated distress (Henderson & Huon, 2002; Stice et al., 2000; Telch & Agras, 1994). Depressive symptoms also account for significant variability in emotional overeating and eating psychopathology (Gianini, White, & Masheb, 2013). Among individuals with BED, the most prevalent comorbid lifetime diagnosis is Major Depressive Disorder (Grilo et al., 2009), with a positive association between depression severity and binge

eating severity (Telch & Agras, 1994). Retrospectively, aversive emotional states, including feeling depressed, upset, hopeless, and stressed, are identified as the most frequent antecedents for binge eating episodes (Binford, Pederson Mussell, Peterson, Crow, & Mitchell, 2004; Stickney, Miltenberger, & Wolff, 1999). Moreover, because women with BED may perceive stress as more intense and emotionally disruptive than women without a history of eating disorder (Crowther et al., 2001), greater perceived psychological stress and negative affect may precipitate binge eating episodes (Freeman & Gil, 2004).

Longitudinal Studies. Longitudinal research on binge eating risk factors largely supports the association between negative affect and binge eating. The broad dimension of negative affect has been found to predict binge eating onset in community adolescent girls (Stice, Killen, Hayward, & Taylor, 1998) and future increases in bulimic behaviors among college-aged women (Cooley & Toray, 2001), although a study of community adult women did not find a significant association between self-reported negative affect and binge eating onset (Vogeltanz-Holm et al., 2000). Further, when examining facets of negative affect, Stice, Presnell, and Spangler (2002) found that elevated depressive symptoms and low self-esteem, but not anxiety and anger, predicted onset of binge eating among asymptomatic adolescent girls, suggesting that binge eating may function to alleviate feelings of depression and inadequacy rather than anxiety and anger. Spoor and colleagues (2006) found that depressive symptoms predicted later development of binge eating among adolescent girls. Whereas bulimic symptoms in general have been found to

predict depressive symptoms and onset of depression in adolescent girls (Stice & Bearman, 2001; Stice, Burton, and Shaw, 2004), Spoor and colleagues did not find an association between initial binge eating and future depressive symptoms. Similarly, Measelle et al. (2006) reported that depressive symptoms predicted disordered eating but that disordered eating did not prospectively predict depression severity in adolescent girls. Some longitudinal research findings suggest that the relation between binge eating and depression is bidirectional. In examining the temporal relationship among overeating, binge eating, and depressive symptoms in a large sample of adolescents and young women (N = 6,578 at the final follow-up point), Skinner, Haines, Austin, and Field (2012) found that a) binge eating and overeating were predictive of depressive symptom onset during the next two years; and b) depressive symptoms predicted onset of overeating and binge eating initiation. Likewise, Presnell, Stice, Siedel, and Madeley (2009) reported that symptoms of depression predicted increased levels of bulimic symptoms and that bulimic symptoms predicted higher levels of depression in a community sample of 496 adolescent girls over an 8-year follow-up period. Overall, these findings support affect regulation models of binge eating, but also suggest that binge eating may increase depression, such that the relationship between binge eating and depression may be mediated by guilt/shame following binge eating episodes.

Experimental studies. Overall, across a variety of experimental paradigms, most laboratory studies show that individuals with BED consume more calories than weight matched individuals without BED, particularly when presented with a variety of

foods/dishes (e.g., Goldfein et al., 1993; Raymond, Bartholome, Lee, Peterson, & Raatz, 2007). Experimental studies have used negative affect inductions versus neutral or positive mood inductions to evaluate the effects of acute negative mood on subsequent caloric consumption. Among individuals without an eating disorder, some studies found that a negative mood induction did not result in increased caloric consumption as compared to a neutral mood induction (Heatherton, Herman, & Polivy, 1991; Steere & Cooper, 1993; Telch & Agras, 1996a), whereas other studies found that a negative versus neutral mood induction resulted in increased caloric intake (Cools, Schotte, & McNally, 1992; Schotte, Cools, & McNally, 1990). Telch and Agras (1996a) explored the relationship between negative affect and BED by comparing 15 women with BED to 15 women without BED on caloric consumption at a multi-item buffet following a negative mood induction. Although BED participants consumed significantly more calories than control participants after the mood induction, BED participants did not differ in the calories they consumed at the post-negative mood induction meal as compared to a control meal in the absence of a mood induction. Agras and Telch (1998) assigned BED participants to a neutral or negative mood induction, with the latter entailing asking participants to imagine a situation that resulted in negative thoughts or feelings. Although no significant differences in caloric intake were observed between the two mood induction conditions, participants in the negative mood induction were more likely to label their episode a binge, describing the eating episode as excessive and reporting loss of control, relative to participants in the neutral mood induction (47% versus 13% of

participants, respectively). The authors concluded that negative affect may influence BED participants' interpretations of their eating. Conversely, Chua, Touyz, and Hill (2004) found support for mood-induced overeating in obese women meeting criteria for BED based on a self-report measure. Regardless of their level of dietary restraint, participants in the negative mood induction condition (i.e., viewing a sad film) ate significantly more chocolate than participants in the neutral mood condition. Similarly, Svaldi, Caffier, and Tuschen-Caffier (2010) reported that urge to binge increased in participants with BED following a sadness-inducing film clip but remained unchanged in control participants. Negative affect may also change BED patients' behavior during an eating episode, adding to the subjective perception of loss of control. Schulz and Laessle (2012) measured both caloric intake and eating behavior in obese patients with and without BED following a psychosocial stress induction. Although caloric intake did not differ between the two groups, the authors found that BED patients showed a faster initial eating rate and less deceleration in eating at the end of the meal compared to the non-stress condition. In contrast, control participants had a slower initial eating rate and faster deceleration in the stress than in the non-stress condition. Thus, stress and presumably negative affect, resulted in greater motivation to eat and slower activation of inhibitory response in BED patients.

Overeating may be associated with negative affect because it serves, albeit temporarily, to repair negative mood. Munsch, Michael, Biedert, Meyer, and Margraf (2008) examined whether acute negative mood and unbalanced nutrition style,

characterized as high fat, low-carbohydrate intake, triggers binge eating in persons with BED. The authors did not find support for the hypothesized synergetic effect of diet and mood on subsequent caloric intake. Neither negative mood induction nor unbalanced nutritional style was associated with increased caloric intake during the test meal. However, participants who underwent a negative mood induction reported a decrease in negative affect and tension following food intake. When participants were median-split based on scores on a measure of depressive symptoms, within the high depression group, participants who were characterized by higher levels of depression experienced the greatest negative mood shift and ate the most, whereas participants with lower levels of depression experienced the smallest mood shift and ate the least. In contrast, in the low depression group, based on median split, degree of mood change was unrelated to number of calories consumed.

The observations that initial mood prior to a negative mood induction is restored back to baseline following a taste task and that depressive symptoms predict subsequent caloric intake were supported by Dingemas, Martijn, Jansen, and van Furth (2009a). Among participants with BED, severity of depressive symptoms predicted caloric intake independent of mood induction condition (i.e., instructed to suppress emotions or react naturally following an upsetting film). Individuals with BED who had moderate-to-severe depressive symptoms consumed more calories than individuals who had no or mild depressive symptoms. Within the moderate-to-severely depressed group, highest caloric intake was observed among participants who reported more negative mood change during

the sad film and among those who reported more positive mood change during the taste task. These findings were replicated by Dingemans, Martijn, van Furth, and Jansen (2009b). The authors proposed that depression depletes coping resources, such that BED individuals with moderate-to-severe depressive symptoms may not be able to cope effectively with transient mood changes. Thus, in persons with BED, negative affect may not be a direct trigger for overeating and binge eating episodes. However, binge eating may function to regulate and cope with negative affect, particularly among individuals who have reduced capacity to use more adaptive emotion regulation skills. In contrast, Agras and Telch (1998) found that depressive symptoms were not associated with caloric intake after a mood induction in BED. Overall, research findings suggest that individuals with BED consume more calories in a laboratory setting than weight-matched individuals without BED. However, experimental studies examining the causal relationship between acute negative mood, depressive symptoms, and binge eating have yielded inconclusive findings. Individual differences in emotion regulation strategies may explain inconsistent findings. Thus, there is a need to delineate factors that exacerbate or prolong negative affect, interfere with adaptive coping in response to negative affect, and/or promote the use of food in regulating acute negative affect in BED.

Ecological Momentary Assessment Studies. Studies using ecological momentary assessment (EMA) methods to assess concurrent binge-related experiences have attempted to delineate the course of negative mood in relation to binge eating. Overall, negative mood states tend to precede binge eating episodes among subclinical

binge eaters as well as individuals with BN and BED (Davis, Freeman, & Garner, 1988; Greeno, Wing, & Shiffman, 2000; Haedt-Matt & Keel, 2011; Munsch, Meyer, Quartier, & Wilhelm, 2012). Among women meeting criteria for BN, binge days are characterized by dysphoric mood and higher stress levels. BN participants report increased negative affect, anger, and stress prior to binge eating episodes, with rapid recovery in mood following binge/purge episodes, suggesting that these behaviors are negatively reinforced as they provide relief from negative affect, particularly dysphoria (Crosby et al., 2009; Smyth et al., 2007). In part, binge eating is thought to ameliorate negative affect through narrowing of attention towards immediate stimuli in the environment, such as food. Shift in awareness from negative self-perception to palatable food items impedes higher level cognitive functioning and inhibition in behavior, resulting in loss of control overeating and a dissociative state. Accordingly, Engelberg, Steiger, Gauvin, and Wonderlich (2007) demonstrated that among women with prominent bulimic symptoms, higher negative affect and dissociation ratings were associated with greater likelihood of classifying an eating episode as a “binge.” Likewise, among obese adults, days characterized by high or increasing negative affect were associated with more frequent overeating and binge eating episodes than days characterized by low negative affect (Berg et al., 2014).

EMA studies examining reinforcing properties of binge eating in BED have yielded mixed findings. One study with subclinical binge eaters found less pleasantness prior to than during a binge eating episode, with pleasantness decreasing following the binge (Deaver, Miltenberger, Smyth, Meidinger, & Crosby, 2003). Relative to controls,

subclinical binge eaters reported higher levels of negative affect before binge eating episodes. Munsch and colleagues (2012) investigated daily courses of mood and tension relative to occurrence of binge episodes in women with BED. The study found that negative mood and tension ratings were significantly higher and that positive mood ratings were significantly lower on binge days and prior to the first daily binge. However, ratings for negative mood remained fairly low overall and positive mood appeared to increase immediately post-binge. Of note, depressed individuals experienced less of an increase in tension before the first daily binge, suggesting that individuals with co-morbid depressive symptoms may be more sensitive to minor fluctuations in mood. In addition, ratings of mood 30 min before a binge eating episode followed a different pattern, with slight improvement in mood 30 minutes before the binge followed by an abrupt deterioration just before the binge. Consistent with the escape theory of binge eating (Heatherton & Baumeister, 1991), Munsch et al. concluded that binge eating reflects a reactive and sudden breakdown in impulse control that is associated with a tendency to experience shifting affective states and a drive to distract from affective states (Anestis et al., 2010). Following binge eating episodes, Munsch et al. also observed a slower and less pronounced reduction in negative mood in women with BED relative to controls. Other EMA studies reported more negative mood during and after binge eating in community women meeting criteria for BED (Hilbert & Tuschen-Caffier, 2007), treatment seeking women with BED (Stein, Kenardy, Wiseman, Dounchis, & Arnow, 2007), and subclinical binge eaters (Wegner et al., 2002). Hilbert and Tuschen-Caffier

examined the course of mood in a community sample of women with BED or BN. For individuals with BN or BED, mood prior to binge eating was significantly more negative than mood at random assessment or mood prior to episodes of regular eating (i.e., eating episodes classified by participants as non-binge meals and snacks). However, for both eating disorder groups, negative mood increased following binge eating episodes compared to mood before and during binge eating.

Although the aforementioned studies seemingly challenge mood regulation accounts of binge eating, methodological aspects of the studies limit the conclusions that can be drawn. The studies used a measure of global negative affect, without considering different emotional facets of this construct. For example, binge eating may function to ameliorate sad mood and increase pleasantness, but lead to increased anxiety. The relief may also be brief and fleeting, such that individuals experience relief only while they are in the active phase of the binge eating episode. For example, Macht and Mueller (2007) found that the positive effect of chocolate on experimentally-induced sad mood in normal weight community participants lasted only 3 minutes. Moreover, temporary amelioration of negative affect may be a particularly reinforcing property of binge eating for those individuals with BED who experience depressive symptoms or demonstrate mood regulation difficulties. It is also possible that loss of control over eating is a feature of BED that is independent from actual amount eaten and/or affective states (Pollert et al., 2013).

The aforementioned findings and theoretical accounts highlight the need to a) study binge eating in the absence of compensatory behaviors, b) specify the circumstances under which negative affect may lead to overeating among persons with BED, and c) distinguish between types of negative affect. Binge eating may provide relief from aversive emotions or induce feelings of dysphoria (i.e., the later among individuals attempting to restrict their caloric intake, including chronic dieters and individuals with BN). Binge eating in the context of BED may provide comfort and distraction from negative affect, particularly feelings of depression; conversely, alternating periods of restraint/purging and binge eating as in BN may decrease self-efficacy and increase feelings of depression. Experimental findings also suggest that only some individuals with BED will respond to negative affect by increasing caloric consumption and binge eating. Consequently, individual characteristics that heighten negative emotions, prolong negative emotional states, and/or account for individual differences in the tendency to respond to negative affect with binge eating need to be elucidated in BED.

Difficulties with Emotion Regulation and BED

Conceptual Overview

Although affect regulation models of binge eating link negative affect and mood, these models are a) not specific to BED and b) do not specify why persons with BED engage in binge eating and do not engage in compensatory behaviors, restriction, and/or self-harm to regulate mood. These models also do not specify whether the relation

between intensity of negative affect and binge eating episodes is further moderated by difficulties with emotion regulation, such as the perception of negative affect as intolerable (Anestis et al., 2007).

As reviewed above, research on eating behaviors in individuals with BED reveals that affective shifts in response to a negative mood induction precede overeating in some, but not all, individuals with BED (Agras & Telch, 1998; Chua et al., 2004). In experimental studies, BED patients reporting higher levels of depressive symptoms experienced a greater negative shift in mood over the course of mood induction (e.g., Dingmans et al., 2009). In turn, the degree of mood reactivity to a negative mood induction was positively associated with caloric consumption. Moreover, although binge eating is thought to ameliorate negative mood (e.g., Heatherton & Baumeister, 1991), the majority of studies assessing mood subsequent to binge eating found that mood deteriorates following binge eating episodes (e.g., Hilbert & Tuschen-Caffier, 2007; Stein et al., 2007). What accounts for the discrepant findings reported in the literature? In addition to previously described methodological limitations, individual differences in ability to manage emotions, including inability to withstand emotions perceived as aversive and deployment of cognitive emotion regulation strategies (e.g., brooding rumination) may account for observed differences in binge eating in response to negative affect among persons with BED. Moreover, individual differences in ability to manage emotions combined with learned expectancies about the effects of eating on mood may

characterize individuals with BED who engage in binge eating in response to transient negative affect.

Management of Emotions

Emotion regulation, as conceptualized by Gratz and Roemer (2004) is a multidimensional construct consisting of intertwining processes, including, a) recognition of emotions, b) acceptance of emotions, c) ability to modulate behavioral response and act in accordance with desired goals when experiencing negative emotions, and d) ability to flexibly use context appropriate emotion regulation strategies in order to achieve stated goals.

Difficulties in regulating emotions have been functionally linked to a variety of problem behaviors, such as substance abuse (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996) and self-harm behavior (Gratz, 2003). Functionally, problem behaviors, including binge eating, may serve an affect regulatory function among individuals who demonstrate deficits in adaptive management of emotions. Consistent with Linehan's (1993) model of borderline personality disorder, problematic behaviors characteristic of this disorder, such as substance use and self-harm behaviors, serve to regulate intense emotions that are experienced as aversive, intolerable, and all consuming. Wisner and Telch (1999) extended Linehan's (1993) emotion regulation model to binge eating behavior, proposing that binge eating is an affect regulation strategy that is deployed in the absence of more adaptive emotion regulation skills and/or when attempts to regulate emotions fail. When an emotion is experienced as intolerable and/or unacceptable

(Corstorphine, 2006), individuals who lack adaptive and effective strategies to modulate emotions might resort to binge eating.

Overall, persons with eating disorders, including those with BED, report significantly higher levels of emotion intensity, lower acceptance of emotions, less emotional awareness and clarity, and increased use of dysfunctional emotion regulation strategies relative to control participants (Harrison et al., 2009; Svaldi, Tuschen-Caffier, & Ehring, 2012). Research findings regarding emotion modulation in BED are mixed. Relative to other ED diagnostic categories, persons with BED actually report higher capacity to use adaptive emotion regulation strategies and display higher levels of emotional clarity and acceptance (Svaldi et al.). However, relative to weight-matched controls, binge eaters appear to have fewer available strategies to modulate emotions. Svaldi et al. (2010) found that women with BED dispositionally suppressed emotions more, re-appraised emotions less, and had higher alexithymia scores than weight-matched controls. Moreover, the authors reported that experimental instructions to suppress emotions following a sad mood induction led to increased urge to binge and a decrease in parasympathetic activation in BED women. Studies also find that emotion dysregulation is associated with binge eating and eating to cope with negative affect. Whiteside et al. (2007) reported that among students, binge eating was associated with self-perceived difficulty in changing negative moods and greater difficulty identifying and making sense of emotions. Likewise, Gianini et al (2013) demonstrated that limited access to emotion regulation strategies and lack of emotion clarity, as indexed by subscales of the

Difficulties with Emotion Regulation Scale, predicted emotional overeating after accounting for depression severity in a clinical sample of treatment-seeking individuals with BED.

Components of Emotion Regulation

Past studies have examined different aspects of emotion regulation in relation to binge eating. In particular, both brooding rumination and inability to tolerate and effectively cope with aversive emotional states have been hypothesized to play a role in the maintenance of binge eating. The subsequent sections will review the empirical and theoretical literature on the association between negative affect, emotion regulation, and binge eating. Consistent with the overall aims of this study, the review will focus on two factors (i.e., rumination and distress tolerance) that may contribute to maintenance of binge eating behaviors in persons with BED.

Rumination. Rumination is a maladaptive cognitive emotion regulation strategy that has been associated with the development and maintenance of different types of psychopathology, including depression (Aldao & Nolen-Hoeksema, 2010; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), binge drinking (Nolen-Hoeksema & Harrell, 2002), and eating pathology (e.g., Nolen-Hoeksema & Aldao, 2010). In the response styles theory (Nolen-Hoeksema, 1987), rumination is conceptualized as a pervasive tendency to repetitively and passively think about symptoms, causes, and consequences of distress. That is, rumination entails directing attentional resources towards negative affect (e.g., sadness) and its consequences (Kross, Ayduk, & Mischel, 2005; Watkins,

2008; 2009). Rumination has been most widely studied in the context of depression. Because rumination essentially results in re-experiencing events that are perceived to be related to depression, research findings suggest that rumination maintains, exacerbates, and prolongs negative affect and depression (Rusting & Nolen-Hoeksema, 1998; Thomsen, 2006; Webb, Miles, Sheeran, 2012), possibly via impairing adaptive problem-solving (Lyubomirsky, Tucker, Caldwell, & Berg, 1999), eroding social support, enhancing negative thinking, interfering with instrumental behavior, and limiting sources of positive reinforcement in the environment. For example, under rumination instructions, dysphoric participants appraise their problems as overwhelming and unsolvable (Lyubomirsky et al., 1999) and fail to come-up with effective solutions relative to dysphoric participants under distraction instructions. There is also some indication of a bidirectional, circular relationship between negative affect and rumination. Using experience sampling methodology, Moberly and Watkins (2008) found that individuals who had reported high (low) rumination at one experience signal were likely to report higher (lower) negative affect at a subsequent experience signal.

The tendency to ruminate, as assessed by the Response Styles Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991), is relatively stable. Individual differences in ruminative tendencies, particularly brooding rumination (i.e., the tendency to think about the negative consequences of one's mood; Watkins, 2009), may predispose some individuals to use maladaptive coping, including binge eating, to reduce acute negative affect. Ruminative processes both exacerbate negative affect, particularly dysphoric

mood, and interfere with adaptive problem-solving, such that individuals with BED may use food to escape brooding, self-focused, and aversive thoughts and to reduce emotional reactivity (e.g., escalation in negative affect) to stress (Aldao & Nolen-Hoeksema, 2010). Thus binge eating in BED may be maintained via a trade-off process, whereas emotions preceding a binge are experienced as more aversive than the guilt and shame that follow a binge (Kenardy, Arnow, & Agras, 1996).

Rumination and emotion management. Cognitive and behavioral emotion regulation strategies can be used to manage and alter emotions (Gross, 1998). Selby, Anestis, and Joiner (2008) proposed that behavioral dysregulation (e.g., binge eating) results from failed attempts to regulate emotions using maladaptive cognitive emotion regulation strategies, particularly rumination. Advancing the construct of emotional cascades, the authors proposed a positive feedback loop between negative affect and rumination whereby rumination is a cognitive emotion regulation strategy that intensifies negative affect, the rumination-induced increase in negative affect leads to a further increase in rumination, and so on. In turn, binge eating and other forms of behavioral dysregulation serve to down-regulate intense emotions by focusing attention on concrete stimuli in the environment, such as chewing palatable food. Although initially developed to explain extreme forms of behavioral dysregulation seen among individuals with Borderline Personality Disorder, including binge eating, the emotional cascades model provides a framework for examining the association between negative affect and binge eating in persons with BED and is consistent with Heatherton and Baumeister's (1991)

affect regulation model of binge eating and emotion regulation conceptualizations of problem behaviors articulated by Linehan (1993) and targeted by Dialectical Behavior Therapy (DBT).

Selby and colleagues (2008; 2009) hypothesize that salient distractions with sensory effects are needed to disrupt an emotional cascade because other more adaptive behaviors may not be sufficiently engaging to distract someone who is ruminating intensely. In testing the emotional cascades model, Selby et al. (2008) found that rumination mediated the association between initial negative affect in response to an emotion-eliciting event and subsequent behavioral dysregulation in a sample of college students. Rumination and deficits in adaptive emotion regulation strategies mediated the association between current symptoms of anxiety and depression and behavioral dysregulation. Moreover, participants reported more frequent binge eating as well as other forms of behavioral dysregulation during times characterized by high rumination. Thus, emotion regulation difficulties, such as the use of maladaptive cognitive emotion regulation strategies, like rumination, may help to explain the complex relationship between negative affect and binge eating in persons with BED. High levels of brooding rumination may place individuals with BED at increased risk for experiencing increased and prolonged negative affect following a mood-evoking event. With respect to binge eating, this may be particularly problematic for those individuals with BED who have developed a greater expectancy that food will ameliorate negative affect.

Rumination and binge eating. A number of studies have linked rumination and disordered eating. Results from a recent meta-analysis by Aldao, Nolen-Hoeksema, and Schweizer (2010) indicate that eating disorder symptoms are associated with maladaptive cognitive emotion regulation strategies, including rumination. Using an experience sampling approach in a sample of obese adolescents, Kubiak, Vogele, Siering, Schiel, and Weber (2008) demonstrated that ruminative thinking in response to daily hassles was associated with an increase in individuals' desire to eat. Similarly, Aldao and Nolen-Hoeksema (2010) found that rumination was significantly related to eating pathology in a sample of college students. Although in both studies rumination had a stronger relationship to anxiety and depression than to eating pathology, there was a unique relationship between rumination and eating disorder symptoms after controlling for anxiety and depression. Notably, the eating pathology outcome variable did not differentiate between behavioral components of eating disorders (i.e., restrictive behaviors characteristic of anorexia versus binge-purge cycles present in BN versus binge eating in the absence of compensatory measures, which is the hallmark of BED). A study of community adolescent females found that baseline RSQ rumination predicted increases in eating disorder symptoms and the onset of bulimia nervosa over a 4-year period (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Moreover, ruminative coping increased risk for onset of binge eating, but was only marginally related to onset of compensatory behaviors. Similarly, Rawal, Park, and Williams (2010) found that preoccupation with eating, weight, and shape concerns was associated with rumination,

particularly brooding, in college students. The study also reported higher levels of brooding rumination in a group of Anorexia Nervosa patients relative to controls. Taken together, these studies suggest that ruminative processes may serve to maintain eating disorder features.

Accordingly, binge eating may a) primarily be an emotion regulation strategy used to reduce negative affect (Polivy & Herman, 2002), b) serve to escape aversive self-awareness associated with brooding and regulate escalating negative mood that results from ruminative processes (Selby et al., 2008), and/or c) occur when a confluence of individual tendencies precludes the use of more effective emotion regulation strategies and promotes the use of eating to regulate mood. For example, Aldao and Nolen-Hoeksema (2010) proposed that a confluence of maladaptive cognitive emotion regulation strategies, such as rumination and/or avoidance of internal states, combined with deficits in using effective strategies (e.g., reappraisal) may lead to particularly rapid escalation of distressing thoughts and emotions.

Nevertheless, it is not known whether binge eating itself is an attempt to regulate negative emotions and escape aversive self-awareness or is a secondary escapist emotion regulation strategy that is deployed when cognitive regulation of affect fails. Conceptual models and extant empirical findings implicate rumination in maintenance of binge eating, yet the role of rumination in BED requires further clarification. Based on the extant literature, it is plausible that a) individuals with BED would be expected to endorse higher levels of ruminative brooding than non-eating disordered individuals; b)

levels of brooding rumination would be positively associated with changes in intensity and duration of experimentally-induced state dysphoric mood; and c) because individuals who ruminate may be less able to use adaptive problem-solving and experience more intense dysphoric mood for longer periods of time, BED persons with higher levels of brooding rumination would be more motivated to deploy escape-based coping strategies, such as binge eating. That is, rumination may intensify and prolong dysphoric mood and/or interfere with adaptive use of other coping skills. Persons with BED and high brooding rumination may experience elevated intensity of negative affect following a life event that evokes 'normal' feelings of dysphoria and sadness. In turn, increased negative affect may precipitate abandonment of control over eating. It is also possible that greater tendency to ruminate will be directly associated with binge eating tendency by 'blocking' individual's access to other coping skills. Individual differences in learned expectancies about the effects of eating on mood may promote the use of binge eating to down regulate intense dysphoric mood and aversive self-awareness.

Distress Tolerance. A construct relevant to the study of individual differences in proclivity to binge eat in response to negative affect is distress tolerance. Distress tolerance is a multifaceted construct that is a component of emotion regulation; it refers to the perceived capacity to withstand aversive states, including emotions and/or physical discomfort (Leyro, Zvolensky, & Bernstein, 2010; Zvolensky & Otto, 2007). This global construct incorporates lower order, domain-specific dimensions of distress (e.g., aversive emotional states versus physical sensations; Bernstein, Zvolensky, Vujanovic, & Moos

2009). The predisposition to appraise aversive states as tolerable or intolerable and the ability to withstand distress has been indexed via self-report measures as well as behavioral and biological challenge tasks designed to measure the duration of time an individual can tolerate exposure to a specific form of distress, such as physical distress, physiological arousal, and aversive psychological states (Leyro et al., 2009). Distress tolerance has been linked to a range of problematic behaviors and psychological disorders, including smoking, substance use and dependence, borderline personality disorder, and anxiety disorders (e.g., Leyro et al., 2009). For example, fear of physical symptoms of anxiety and the potential consequences of those symptoms has been linked to BN symptom severity (Anestis, Holm-Denoma, Gordon, Schmidt, & Joiner, 2008).

Affective processes that impact the experience and evaluation of affective states are of particular relevance to the study of functional relations between BED and negative affect. Particularly, factors that influence individuals' experience and evaluation of negative emotions, such as dysphoria. Overall, an individual's capacity to tolerate negative emotional states (i.e., affective distress tolerance) consists of a number of interrelated component process, including a) perceived ability to tolerate negative emotions and accept emotional situations, b) deployment of emotion regulation strategies, and c) the degree to which negative attention depletes attentional resources and interferes with adaptive functioning (Simons & Gaher, 2005).

Low affective distress tolerance, as operationalized via the Distress Tolerance Scale (DTS; Simons & Gaher, 2005), has been concurrently and prospectively linked to a

variety of problem behaviors, including alcohol and substance use in response to life stressors among HIV-positive patients (O’Cleirigh, Ironson, & Smits, 2007), endorsement of alcohol use to reduce negative mood (i.e., coping motive) among young adult current drinkers (Howell, Leyro, Hogan, Buckner, & Zvolensky, 2010), and coping motive for marijuana and alcohol use among adults (Simons & Gaher, 2005; Zvolensky et al., 2009). Similarly, Buckner and colleagues (2007) found that DTS scores concurrently mediated the association between depressive symptoms and alcohol and marijuana use problems in a sample of undergraduate students.

Poor affective distress tolerance may drive individuals to attempt to ‘down-regulate’ emotions experienced as aversive, including dysphonic mood, via a variety of maladaptive behaviors (e.g., Linehan 1993). Trafton and Gifford (2011) proposed a functional process model linking distress tolerance processes with behavioral responding. From this perspective, high distress tolerance entails not responding to immediate opportunities to reduce distress. Therefore, behaviors that are functionally linked to low distress tolerance are maintained by negative reinforcement or reward learning (i.e., reduction in distress). The model further proposes that the development, maintenance, and expression of (low) distress tolerance is shaped by behavioral consequences and is a form of context-specific learning. The model may have particular relevance to eating behavior in BED because persons with BED and low distress tolerance may have learned that an aversive emotional state is a cue for eating. For instance, Ricca and colleagues (2009) found that persons meeting criteria for BED scored highest on a measure

assessing eating in response to aversive emotions relative to persons with subthreshold BED and obese control participants.

Consistent with theoretical accounts of binge eating (e.g., Heatherton & Baumister, 1991), individuals with BED and low distress tolerance may appraise negative affect as intolerable and expect, based on past experiences, that eating will serve to regulate mood. Specifically, a situation or an emotional state that is experienced by a person with BED as aversive and is perceived as exceeding his/her resources may precipitate overeating. Indeed, extant empirical studies indicate that persons who engage in binge eating behavior relative to controls a) are more likely to perceive situations as stressful (Hansel & Wittrock, 1997), b) engage in higher rates of emotion-focused coping (Bittinger & Smith, 2003), c) report greater urges to binge during periods of stress (Pendleton et al., 2001), and d) consume more calories on days characterized by high levels of stress (Crowther, Sanftner, Bonifazi, & Shepherd, 2001). Such findings suggest that the combination of low distress tolerance and dysphoric mood following a mood-evoking event may cause a person with BED to respond to negative mood via binge eating. Conversely, negative affective states may be less relevant as antecedents to binge eating episodes for individuals who demonstrate greater ability to withstand aversive emotional states.

A limited number of studies have examined the association between distress tolerance and disordered eating behaviors. Indirect support is provided by studies examining the concurrent association between distress tolerance and bulimic symptoms.

For instance, college students reporting low levels of distress tolerance and high impulsivity were most likely to report relatively high levels of bulimic symptoms in response to negative affect (Anestis, Selby, Fink, Joiner, 2007). Corstorphine, Mountford, Tomlinson, Waller, and Meyer (2007) found that eating-disordered women were significantly more likely than women with no history of an eating disorder to endorse use of emotional avoidance, a component of maladaptive affective distress tolerance.

Although limited, the literature reviewed suggests that low distress tolerance may characterize individuals with BED and be implicated in the etiology of the disorder. Kenardy et al (1996) reported that relative to weight matched controls patients with BED reported greater distress and lower tolerance of negative affective states. Because individuals who are low in distress tolerance appraise negative emotions as aversive, they are more likely to seek immediate relief from negative affect (Simons & Gaher, 2005). That is, low distress tolerance may provide opportunities to link eating and regulation of negative affect, such that individuals develop expectancies about the positive effects of food on mood. In turn, negative reinforcement eating expectancies may further promote eating in response to distress. Over time, binge eating may become a habitual response to negative affect that is perceived as intolerable. Individual differences in distress tolerance may also serve to maintain BED and be associated with frequency and severity of binge eating. BED individuals who are lower in distress tolerance may be more likely to appraise transient dysphoric mood as intolerable and resort to escape-based coping strategies than BED-diagnosed individuals with a greater ability to tolerate distress. In

BED, a lower ability to tolerate distress combined with greater negative reinforcement expectancies for eating is expected to be particularly toxic.

Expectancies about the Effects of Eating on Mood

Individuals who struggle to effectively and adaptively manage negative emotions may attempt to regulate emotions, particularly negative affect, via eating (e.g., Heatherton & Baumister, 1991; Dingemans et al., 2009). Yet, why do some individuals with emotion regulation difficulties engage in binge eating whereas others do not? Consistent with expectancy theory, Smith, Simmons, Flory, Agnes, and Hill (2007) propose that expectancies for the consequences of eating are a “summary of one’s learning history and are the cognitive mechanisms by which prior learning leads to subsequent behavior (p. 188). Individuals with BED may have learned vicariously, through past experiences or mass media, to expect eating to be reinforcing (i.e., eating will improve negative mood or will increase positive affect; e.g., Desmet & Schifferstein, 2007; Macht, 2008; Macht & Simmons, 2000).

Indeed, eating may provide a particularly salient distraction from aversive internal states as, at least initially, the flavors, textures, and smells of palatable foods consumed during a binge are perceived as enjoyable. That is, eating may provide temporary relief from negative affect, reduce distress, and be perceived as a rewarding experience. Heatherton and Baumister (1991) propose that eating cascades into a binge because “cognitive narrowing” on food- and eating-relevant stimuli leads to disinhibition of eating and loss of control. Individuals become “absorbed” by the sensory experiences

associated with eating and fail to consider the consequences of their behavior (i.e., the ensuing feelings of guilt and shame) or pay attention to internal satiety cues.

Paradoxically, a post-binge increase in negative affect may also strengthen negative reinforcement eating expectancies in persons with BED. Specifically, individuals with BED may continue eating while in the midst of a binge because they learned through past experiences that an end of a binge eating episode signals an increase in aversive self-awareness, a corresponding increase in negative affect, and cessation of pleasure. That is, during a binge eating episode, the consequences of eating cessation are perceived as aversive. Thus, several pathways are potentially implicated in the development of reinforcement expectancies from eating in persons with BED. In turn, the expectancy that eating will reduce negative mood may itself promote the use of food to regulate negative affective states.

Individuals with symptoms of BN have negative reinforcement expectancies for eating (e.g., Hohlstein, Smith, & Atlas, 1998). For instance, Hayaki (2009) reported that negative reinforcement expectancies for eating predicted unique variance in BN symptoms among college students. Several studies have also investigated the association between eating expectancies and binge eating behavior. Smith and colleagues (2007) found that negative reinforcement eating expectancies predicted the onset of binge eating in a sample of adolescents girls. Negative reinforcement eating expectancies were also prospectively related to binge eating severity in a sample of middle school girls (Combs, Smith, and Simmons, 2011). Consistent with the idea the binge eating itself influences

subsequent expectancies, binge eating behavior predicted the trajectory of eating expectations. Higher initial binge eating behavior characterized those girls whose eating expectancies started low and increased over time, relative to those who endorsed continuously low expectations (Combs et al.; Smith et al.). Consistent with past findings (e.g., Fischer, Anderson, & Smith, 2004; Fischer, Smith, Anderson, & Flory, 2003), Moreover, Fischer and Smith (2008) demonstrated that the association between emotion regulation (i.e., individual differences in tendency to respond to negative affect with impulsive action) and binge eating was moderated by eating expectancies. Undergraduate women who scored high on a measure of action urgency and had positive expectations for the effects of eating reported the most binge days. Thus, eating expectancies is a mechanism through which individual differences in emotion regulation may lead to the development and maintenance of BED.

Limitations of Extant Literature

In summary, the current study added to the extant literature in several important ways. First, studies of individuals meeting diagnostic criteria for BED and employing an experimental design are needed to demonstrate a causal relationship between putative antecedents, such as negative affect and binge eating. Second, although components of emotion regulation have been hypothesized to have a role in BED, these associations have not been examined empirically. Thus, it is currently unknown whether individuals with BED are characterized by a ruminative response style, low ability to withstand psychological distress, perceived inability to effectively manage psychological distress,

and positive expectancies about the effects of eating on mood relative to non-eating disordered participants. It is also unknown whether these factors are related to binge eating severity or emotional eating in this population. Thus, the current study will address important gaps in the literature. Third, past studies investigating the relationship between negative affect and BED have yielded inconsistent findings. Methodological limitations of past investigations should be considered. It is necessary to investigate whether different types of negative affect are differentially related to subsequent caloric consumption. The majority of studies reviewed have used film clips to induce negative affect, yet it remains unclear whether participants were reporting increases in anxiety, depression, or another emotion. Moreover, film clips are not expected to elicit a personally-relevant aversive negative state. It is not known whether participants with BED will increase caloric consumption when the affect induced is dysphoric mood, in general, and/or when the stimulus used evokes a personally-relevant dysphoric mood. As Fox and Power (2009) note, ‘the literature on the role of sadness in eating disorders has not been researched at all,’ (p. 251). Although BED is distinct from other eating disorders in terms of demographic characteristics, clinical features, and migration between diagnostic categories (more common for AN and BN; Holm-Denoma et al., 2007), functional theories of BED have been largely based on extant theories of BN. Theoretical accounts of BED have also been informed by clinical samples of women with BN, examined bulimic behaviors without differentiating between binge eating and purging, and/or studied college students with sub-clinical levels of binge eating.

Potential Implications of Current Study

The current study adds to the extant literature on affective and cognitive vulnerability factors for BED and addresses several important gaps in the literature. First, two meta- analyses have specifically called for studies that examine novel risk factors for eating disorders (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Stice, 2002). Consistent with Stice's (2002) recommendations, the current study first establishes an association between the proposed vulnerability factors (i.e., brooding rumination, perceived ability to withstand and manage emotional distress, and eating expectancies) and indices of binge eating pathology. The current study aims to demonstrate that the aforementioned vulnerability factors uniquely characterize individuals with BED relative to controls and are related to severity of binge eating pathology and use of food to regulate emotions.

The current study also uses moderation analyses and an experimental procedure (i.e., dysphoric mood induction) to explore how the proposed vulnerability factors work together to perpetuate BED. Specifically, the current study examines whether individual differences in brooding rumination and distress tolerance interact with negative reinforcement eating expectancies to perpetuate binge eating in response to a dysphoric mood in individuals with BED. To the best of my knowledge, no studies have examined the associations between distress tolerance, rumination, negative affect, and indices of problematic eating behavior among individuals meeting criteria for BED. Moreover, the current study adds to the literature by examining the hypothesized relationship between

putative diatheses for binge eating and a proximal antecedent (i.e., negative affect following a stressor).

The current study also addresses important gaps in the literature by examining proposed vulnerability factors in a community sample of individuals meeting diagnostic criteria for BED. Past studies have primarily used persons with BN, student samples and/or “chronic dieters.” However, studies demonstrate that BED is distinct from other eating disorders and overweight/obesity. In addition, this study a) uses a mood-induction procedure that has been reliably shown to elicit self-relevant dysphoric mood as opposed to global negative affect and b) includes several indicators of binge eating behavior, including urge to eat, perceived loss of control over eating, and actual caloric consumption.

Clinically, studies examining individual vulnerability factors may help identify individuals who are most likely to engage in binge eating in response to experiencing negative affect following stressful life events. Such studies may help specify under which conditions researchers and clinicians can expect individuals meeting diagnostic criteria for BED to respond to negative mood with disinhibited eating. Moreover, studies examining underlying cognitive-affective and emotional vulnerability processes in BED may inform the development of efficacious intervention strategies, optimally designed to target maintaining mechanisms.

Study Aims and Hypotheses

The current study aimed to elucidate the role of affective and cognitive processes in BED.

Aim 1

To examine group differences in proposed cognitive and affective vulnerability processes between individuals with BED and non-eating disordered control participants

Hypothesis 1. Participants meeting diagnostic criteria for BED would score higher on measures of ruminative brooding, distress tolerance (i.e., demonstrate lower distress tolerance), and would perceive themselves as having fewer strategies to cope with negative mood relative to non-eating disordered controls.

Hypothesis 2. Participants meeting diagnostic criteria for BED would endorse a greater urge to eat to cope with negative affect and would report a more positive expectancy that food will serve to regulate negative affect.

Aim 2

To examine the relationships between cognitive and affective processes and binge eating severity among BED individuals.

Hypothesis 3. Within the BED group, brooding rumination scores, distress tolerance, and negative reinforcement expectancies for eating would each contribute to the prediction of binge eating severity, frequency of binge eating, and urge to eat in response to negative emotions.

Aim 3

To replicate and extend past findings on the association between experimentally-induced sad mood and caloric consumption, urge to eat, and perceived loss of control.

Hypothesis 4. Consistent with past findings (e.g., Agras & Telch, 1996; Agras & Telch, 1998), participants meeting criteria for BED would a) report greater increase in urge to eat from pre- to post- mood induction and b) consume more calories and report greater loss of control over eating during the taste task.

Hypothesis 5. It was expected that BED participants who endorsed a greater increase in sad affect following the dysphoric mood induction would report a greater increase in urge to eat, consume more calories, and would report greater loss of control over eating than BED participants demonstrating a smaller increase in negative affect in response to the mood induction. Degree of change in sad mood from pre- to post-mood induction would not be associated with subsequent urge to eat, caloric intake, or loss of control for control participants.

Aim 4

To elucidate the relationship between negative affect and binge eating behavior by specifying the circumstances under which dysphoric mood has an effect on binge eating behavior.

Hypothesis 6a. Participants with BED who scored higher on a self-report measure of brooding rumination would experience a greater increase in sad affect

following the dysphoric mood induction than control participants or participants scoring lower on a self-report measure of brooding rumination.

Hypothesis 6b. Participants with BED who scored higher on a self-report measure of brooding rumination would eat more calories during the taste task and would report greater increase in urge to eat from pre- to post-mood induction than BED participants scoring lower on a self-report measure of brooding rumination or control participants.

Hypothesis 7. Brooding rumination and negative reinforcement eating expectancies would each uniquely predict pre- to post-mood induction change in urge to eat and caloric intake following dysphoric mood induction for BED-diagnosed participants. It was expected that both brooding rumination and negative reinforcement eating expectancies would be positively associated with change in urge to eat and with caloric intake. Neither brooding rumination nor negative reinforcement expectancies were expected to predict change in urge to eat or caloric intake in control participants.

Hypothesis 8. A brooding rumination by eating expectancies interaction would qualify the main effects of each predictor. Within the BED group, participants with higher ruminative brooding and greater negative reinforcement eating expectancies would consume the most calories and show the largest increase in urge to eat following the dysphoric mood induction. The interaction of brooding rumination by eating expectancies was not expected to be significant for the control group.

Hypothesis 9. Distress tolerance and negative reinforcement eating expectancies would each uniquely predict pre- to post-mood induction change in urge to eat and caloric intake following the dysphoric mood induction for BED-diagnosed participants. It was expected that both low distress tolerance and negative reinforcement eating expectancies would be positively associated with change in urge to eat and with caloric intake. Neither distress tolerance nor negative reinforcement expectancies were expected to predict change in urge to eat or caloric intake in control participants.

Hypothesis 10. A distress tolerance by eating expectancies interaction would qualify the main effects of each predictor. Within the BED group, participants with lower distress tolerance and greater negative reinforcement eating expectancies would consume the most calories and show the largest increase in urge to eat following the dysphoric mood induction. The interaction of distress tolerance by eating expectancies was not expected to be significant for the control group.

RESEARCH DESIGN AND METHOD

Participants

Participants in this study were 38 females with BED and 36 female control participants, aged 18 or older. Inclusion criteria for the BED group were a primary diagnosis of BED or sub-threshold BED (i.e., an average of one binge eating episode per week over the previous 24 weeks as opposed to two or more episodes per week in BED) as assessed by the Structured Clinical Interview for DSM-IV Axis I Disorders-Non-Patient Version (SCID-NP; First, Spitzer, Gibbon, & Williams, 2002). Women with sub-threshold BED were included in the BED group because what was regarded as sub-threshold BED was subsumed under the diagnostic category of BED in DSM-V. DSM-V criteria specify that binge eating must occur at least once per week for 3 months. Individuals with sub-threshold BED do not differ significantly from full-syndrome BED in terms of weight and shape concerns, dietary restraint, psychiatric distress, and treatment seeking (Striegel-Moore & Dohm, 2000), suggesting that the distinction between these groups may be arbitrary. Exclusion criteria for the BED group were history of Bulimia Nervosa (BN) or Anorexia Nervosa (AN). Exclusion criteria for the control group were a current or past diagnosis of BED, BN, AN, or Eating Disorder Not Otherwise Specified (ED-NOS) as assessed by the SCID-NP. Participants in both groups were excluded from the study based on a) acute and serious suicidal intent (assessed by using the SCID-NP suicide questions in the Mood Disorders Module, b) psychotic spectrum symptoms or disorders, c) current substance abuse or dependence, d) current

Major Depressive Episode, e) current Manic or Hypomanic Episode, e) the presence of medical conditions that may affect eating, including postmenopausal or perimenopausal (e.g., hot flashes) and pregnancy, and f) use of medications that were known to affect eating (e.g., appetite suppressants). Regarding (f), participants who were on a stable dose of antidepressant medication for at least one month were included in the study.

Comorbidity was generally allowed in the study to increase generalizability, except for those conditions specified in a-d, which were excluded because it is anticipated that they would interfere with data collection. Major Depression was excluded because it is ethically problematic to put a clinically depressed individual through a dysphoric mood induction.

Recruitment and Compensation

Females between the ages of 18 to 65 were recruited from a) the community via flyers posted in local businesses, Internet websites, and community boards and b) undergraduate psychology courses at the University of Vermont. Because awareness of study objectives may bias participants' behavior (i.e., influence their eating behavior during the taste task), advertisements for a study of "personal beliefs and taste perceptions in women" sought females between the ages of 18 to 65 who experienced "episodes of overeating." The advertisements also stated that women who do not experience overeating episodes are also being sought for the study in order "to compare women with and without overeating episodes on taste perceptions." Because BED and obesity are frequently comorbid, an attempt was made to match the two groups on Body

Mass Index (BMI). This was done to ensure that study results are attributed to BED and not to comorbid overweight/obesity. An attempt was also made to match participants on age.

Eligible community participants who completed the entire study were compensated a total of \$35. To reduce attrition, qualified participants were compensated based on their involvement; they received \$10 dollars at the completion of the first session and \$25 for participation in the second visit. Participants who were recruited from undergraduate psychology courses at the University of Vermont offered were offered either financial compensation or course credit in exchange for their participation.

Measures

Screening and Diagnostic Assessment

Questionnaire on Eating and Weight Patterns (QEWP). The QEWP (Spitzer et al., 1992) was used as an initial screening measure, such that questions from the QEWP will be incorporated into the phone screening process. The QEWP is a self-report measure assessing DSM-IV criteria for eating disorders (e.g., AN, BN, BED; Spitzer et al., 1992). A short 8-item version of the QEWP was used in the current study to identify potential participants among responders to the study advertisements. The short-version contains eight diagnostic items assessing presence/absence of a) binge eating (e.g., “During the past six months, did you often eat within any two-hour period of time what most people would regard as an unusually large amount of food?”), b) loss of control (e.g., “When you ate this way, did you often feel you couldn’t stop eating or control what

or how much you were eating?”), c) behavioral indicators regarding loss of control (e.g., eating more rapidly than usual, eating until uncomfortable full, eating alone, etc.), and d) distress (e.g., “How upset were you by overeating...”). The QEWP also assess frequency of binge episodes.

Previous studies have demonstrated that the short version of the QEWP possesses good concurrent and discriminate validity; the 8-item version is sufficient to identify binge eaters (Stice, et al, 2001) and reliably discriminates between nonclinical and clinical binge eaters (Nangle, Johnson, Carr-Nangle, & Engler, 1994; Stice et al., 2001). Consistent with proposed DSM-V criteria for BED, respondents were invited to the in-person SCID to determine eligibility for the BED study group if they a) described binge eating episodes characterized by loss of control, b) endorsed at least three behavioral indicators, c) reported abstaining from compensatory behaviors (e.g., self-induced vomiting, laxatives use, excessive exercise), and d) reported an average of at least one binge per week over the past 3 months. Individuals who did not meet criteria for the BED study group and who did not fulfill other exclusion criteria were invited to the SCID to determine eligibility for the control group.

Structured Clinical Interview for DSM-IV Axis I Disorders–Non Patient Version (SCID-NP). The SCID-NP (First et al., 2002) is a structured diagnostic interview that assesses DSM-IV criteria for Axis I disorders. The PI, who is trained and experienced with SCID administration, or another clinical graduate student with SCID training and experience administered the full SCID-NP. Extant studies indicate that the

SCID-NP is a reliable (Zanarini & Frankenburg, 2001; Zanarini et al., 2000) and valid (Shear et al., 2000) diagnostic instrument. The SCID-NP was used to confirm a diagnosis of BED for the BED study group, to assess for the presence of diagnostic exclusion criteria for both groups, and to document comorbid conditions. A structured clinical interview was needed to confirm a diagnosis of BED because self-report measures lack sufficient specificity to distinguish between subjective and objective binge eating episodes (Fairburn & Beglin, 1994). According to DSM-IV-TR criteria, the latter were needed for a diagnosis of BED.

The Eating Disorder Examination (EDE-Q). The EDE-Q (Fairburn & Beglin, 1994) is a 38-item self-report measure assessing the presence and severity of eating pathology. The EDE-Q consists of four subscales, a) dietary restraint, b) eating concerns, b) weight concerns, and d) shape concerns. For the four subscales, items are rated on a 7-point Likert scale ranging either from 0 (*no days*) to 6 (*every day*) or from 0 (*not at all*) to 6 (*markedly*). The EDE-Q was derived from the Eating Disorder Examination, 12th edition interview (EDE; Fairburn & Cooper, 1993) such that each item of the EDE-Q is directly taken from a corresponding item of the EDE interview (Mond, Hay, Rodgers, Owen, & Beumont, 2004). Studies assessing the relationship between the EDE-Q and EDE interview version have found that the two measures are highly convergent in terms of assessing important features of eating disorders (e.g., Fairburn & Beglin, 1994; Goldfein, Devlin, & Kamenetz, 2005; Grilo, Masheb, & Wilson, 2001; Sysko, Walsh, & Fairburn, 2005). The EDE-Q also provides frequency data on the key behavioral features

of eating disorders in terms of the number of days out of the past 28 days that the behavior has occurred.

Estimates for test- retest reliabilities for the four subscales are .81 for Restraint, .94 for Shape Concern, .92 for Weight Concern, and .87 for Eating Concern. Cronbach's alphas for the four scales range from .78 (Eating Concern) to .93 (Shape Concern; Luce & Crowther, 1999). The EDQ was used to examine differences between persons with BED and controls in terms of eating pathology relevant variables. The internal consistency of EDE-Q dietary restraint total score ($\alpha = .73$), eating concerns total score ($\alpha = .86$), weight concerns total score ($\alpha = .90$), and shape concerns total score ($\alpha = .94$) in the current sample were good to excellent.

Demographic Variables. Participants completed a demographics form. Variables of interest included: a) marital status, b) age, c) educational level, c) current medications, d) assessment of health-relevant behaviors (e.g., dieting, exercise, caffeine and alcohol intake), e) and current and past mental health treatment.

Self-Report Assessments of BED Features and Eating Motives

Binge Eating Scale (BES). Binge eating severity was assessed via the BES (Gormally, Daston, & Radin, 1982). This 16-item scale measures both the behavioral features of binge eating as well as the feelings and cognitions associated with binge eating. Participants are asked to respond to such statements as "How often do you binge?" and "Which best describes your feelings during a binge?" The BES has good

test-retest reliability and discriminate validity (Marcus, West, & Hopkins, 1988). Internal consistency of the BES total score in the current sample was excellent ($\alpha = .95$).

Eating Expectancies Inventory (EEI). The EEI (Hohlstein, Smith, & Atlas, 1998) is 34-item self-report measure assessing learned reinforcement expectancies about the consequences of eating. Although the scale consists of five subscales, only one subscale was employed in the current study. The “Eating helps manage negative affect subscale (EEI-NA)” consists of 18 items. The subscale includes such items as, “Eating helps me forget or block out negative feelings like depression, loneliness and fear.” The scale possesses adequate concurrent and discriminate validity, accurately differentiating between participants with AN and BN participants and between both groups and psychiatric and healthy controls. Internal consistency for the EEI-NA subscale is .94 (Hohlstein et al., 1998). Internal consistency of the EEI-NA subscale in the current sample was excellent ($\alpha = .94$).

Emotional Eating Scale (EES). The EES (Arnow, Kenardy & Agras, 1995;) assess the extent to which 25 different emotions are associated with urge to cope by eating. Participants rate their urge to eat in response to each emotion on a 5-point scale ranging from “1 = no desire to eat” to “5= overwhelming urge to eat.” The EES yields three subscale scores based upon the mean of items reflecting the urge to eat in response to anger/frustration, anxiety, and depression. The scale has good construct and discriminate validity as well as adequate reliability (Arnow et al., 1995). In the current

study, items assessing urge to eat in response to depression were used (EES-D). Internal consistency of the EES-D subscale in the current study was $\alpha = .85$.

Covariates

Body Mass Index (BMI). BMI was calculated to ensure that the groups were comparable with regard to height and weight. BMI ($\text{BMI} = \text{kg}/\text{m}^2$) rather than weight in pounds was used because it accounts for variations in weight due to height. During the initial assessment, each participant was instructed to remove their shoes and jewelry and was weighed on a digital scale. Next, each participant was instructed to remove her shoes and stand up straight against the wall where height was measured via a wall mounted height rod. The correlation between BMI calculated based on self-reported height and weight and BMI calculated based on laboratory measured height and weight was .96 in Control participants and .95 in BED group.

Beck Depression Inventory-Second Edition (BDI-II). The BDI-II is a 21-item self-report questionnaire that measures affective, cognitive, and physical symptoms of depression (Beck, Steer, & Brown, 1996). Items are rated on a 4-point scale ranging from 0 (symptom not endorsed) to 3 (symptom present at maximum severity). Beck and colleagues (1996) found that the BDI-II has good convergent validity, internal consistency, and test-retest reliability. When comparing psychometric properties between the original Beck Depression Inventory and the BDI-II, the BDI-II had stronger factorial validity than the earlier version (Dozois, Dobson, & Ahnberg, 1998). The BDI-II was

used to assess depressive symptomatology, and, as appropriate, was included as a covariate. Cronbach alpha for BDI-II scores in the current study was .93

Cognitive and Affective Vulnerability Factors

Ruminative Response Scale (RRS). Rumination was assessed via the RRS (Nolen-Hoeksema & Morrow, 1991). The RRS consists of 22 items on a 4-point Likert scale assessing the frequency with which individuals engage in self-reflection and dwell on the symptoms, causes, and consequences of mood. The RRS has sound psychometric properties, including good concurrent validity, test-retest-reliability, and internal consistency (see Nolen-Hoeksema, Wisco, Lyubomirsky, 2008 for a review). Results of a recent investigation by Treynor, Gonzalez, and Nolen-Hoeksema (2007) suggest that the RRS contains two subscales (i.e., brooding and self-reflection) reflecting two different facets of rumination. Each subscale is composed of 5 items. Whereas self-reflection reflects more adaptive attempts to understand the reasons for one's mood, brooding rumination is the maladaptive tendency to dwell on one's negative mood and its consequences. For the purposes of this study, the sub-scale of brooding-rumination (RRS-B) was used, as brooding rumination is thought to reflect a more "toxic" form of rumination (Nolen-Hoeksema et al., 2008). The internal consistency of the RRS-B scale ($\alpha = .85$) in the current sample was excellent.

The Distress Tolerance Scale (DTS). The DTS (Simons & Gaher, 2005;) is a 15-item measure of difficulty in tolerating affective states. Respondents are asked to rate on a 5-point scale (1= "Strongly agree" to 5 = "Strongly disagree") the extent to which they

agree with statements such as “I can’t handle feeling distressed or upset” and “When I feel distressed or upset, I must do something about it immediately.” In addition to providing an overall measure of distress tolerance, the scale assesses four facets of distress tolerance: a) perceived ability to handle distress, b) subjective appraisal of distress, c) attention absorbed by negative emotions, and d) regulation of distress. Data suggest that the scale possess good internal consistency and discriminate validity and evidences expected correlations with other measures of affect (e.g., see Leyro et al., 2010 for a review; Simons & Gaher, 2005). DTS total scores were used as a global distress tolerance measure. Internal consistency for DTS total scores in the current sample was ($\alpha = .93$), suggesting excellent internal consistency.

Difficulties with Emotion Regulation Scale (DERS). Emotion management difficulties were assessed via the DERS (Gratz & Roemer, 2004). The DERS is a 36-item self-report measure. Respondents rate on a 5-point Likert scale the frequency with which various aspects of emotion dysregulation apply to them. The scale consists of 6 empirically-derived subscales mapping onto different components of emotion regulation, including a) nonacceptance of emotions, b) difficulties engaging in goal-directed behavior when experiencing negative emotions, c) impulse control difficulties when experiencing negative emotions, d) lack of emotional clarity, e) limited access to emotion regulation strategies, and f) lack of emotional awareness. Findings from the initial validation study suggest that the scale possess acceptable temporal stability and internal consistency (as indexed by Cronbach’s alpha) for both DERS total and individual

subscale scores (Salters, Roemer, Tull, Rucker, & Mennin, 2006). Gratz and Roemer (2004) reported Cronbach's alphas of .93 and .88 for DERS total score and access to emotion regulation strategies, respectively. Internal consistency for the DERS total scores in the current sample was ($\alpha = .96$), suggesting high internal consistency.

Assessment of Mood, Distress and Eating

Visual Analogue Scale (VAS). Consistent with past research in this area (e.g., Chua et al., 2004; Dingemans et al., 2009), current subjective mood state were assessed via six adjectives (sad, tense, tired, happy, angry, and anxious) immediately before and after the mood induction and after the taste task. To indicate their current subjective mood state, participants were asked to complete a visual analogue scale (VAS) for each of the five adjectives by drawing a vertical mark along a 100-mm line with the anchors of “not at all” and “extremely.” Two additional scales asked participants to rate their current state of hunger and the strength of their urge to eat (i.e., from “not at all” to “extremely”).

Subjective Units of Distress Scale (SUDS). The SUDS (Wolpe, 1958) is a self-report visual analogue scale, where participants indicate along a 100-mm line the degree of distress they are experiencing from “not at all distressed” to “very much distressed.” The SUDS were administered pre- and post-mood induction and post-taste task.

Taste Test Questionnaire (TTQ). Consistent with past studies (e.g., Chua et al., 2004; Dingemans et al., 2009), participants were asked to rate each food item on how sweet, salty, pleasant tasting, and pleasant smelling it was and on how much they liked it using a 100-mm VAS with the anchors of “not at all” and “extremely.”

Food Intake Questionnaire (FIQ). Aspects of food intake were also assessed on a separate 100 mm VAS. Loss of control over eating was assessed by the item “Did you have control over the amount of food you ate?” and rated on a scale ranging from “completely not” to “completely.” Perceived amount of food eaten (“How much did you eat?”) was scored on a VAS ranging from “not a lot” to “a lot.” Guilt about amount eaten was assessed by the item “How guilty did you feel after eating” (0 = no guilt at all and 100 = extremely guilty), and perceived rate of eating was assessed by asking participants “how rapidly did you eat” (0 = “not fast at all” and 100 = “extremely fast”). Finally, whether participants would label their eating as a binge was assessed with a yes/no question (“Did you feel as though you experienced a binge eating episode?”).

Assessment of Awareness and Suspicion

Participants completed a funneled debriefing form adapted from Bargh and Chartrand (2000). The form was used to assess whether participants were aware of study objectives and to determine whether awareness of study objectives and study characteristics affected their behavior.

Procedures

Prior to data collection, approval for all study procedures was obtained from the Institutional Review Board at the University of Vermont.

Screening and Questionnaire Session (First Study Visit)

Persons responding to the study advertisements were first be screened for eligibility for either study group (i.e., BED or control group) via a short phone screen, including the QEWP. Respondents deemed eligible for either study group were invited to the laboratory for a screening visit in order to provide informed consent and determine final eligibility status. At this visit, participants first completed written informed consent. Participants were informed that the study assesses “the effects of personal beliefs on taste perceptions in women with and without overeating episodes” and participated in a complete diagnostic interview (full Structured Clinical Interview for DSM-IV Axis I Disorders; SCID-NP; First et al., 2002). For participants who met exclusionary psychiatric diagnoses the SCID-NP was stopped as soon as an exclusionary diagnosis was determined, and the participant was informed of her ineligibility and given a referral list.

If eligible for either study group, the participant completed a battery of questionnaires and her height and weight were obtained and recorded. At the end of the first visit, qualified participants were compensated for their time (i.e., monetary compensation or course credit) and scheduled for a second laboratory visit within 2 weeks of the first study visit.

Laboratory Session (Second Study Visit)

Prior to the second laboratory visit, each participant was reminded to refrain from eating 2 ½ hours prior to the scheduled visit. Upon her arrival, each participant was asked

whether she adhered to these instructions. Upon her arrival each participant was given instructions for the mood induction and the taste task. Specifically, sad mood induction and taste task were conducted in adjacent laboratory rooms. Participants were instructed to proceed to the taste task immediately after completing the sad mood-induction and remain in that room until experimenter's return (i.e., 15 minutes, although participants were not informed of this time frame). Participants could not bring personal items into the room as these may serve as distractors. In addition, written instructions were placed in each room.

Before the mood induction, each participant completed the VAS scales for mood, provided a SUDS rating, and indicated her perceived hunger level and urge to eat (Time 1). Immediately following the mood induction, the participant again completed the VAS scales for mood, distress, hunger level, and urge to eat (Time 2). Subsequently, the participant was asked to participate in a 15-minute taste task. The participant was presented with three kinds of foods, representative of food items commonly consumed during a binge by persons with BED (e.g., Castonguayet al., 1995). She was instructed to taste all three food items and, after tasting all food items, to fill out the Taste Test Questionnaire. She was told that she should feel free to eat as much as she likes or thinks was necessary to evaluate each food. She was also informed that she was the last participant of the day, so she should help herself to as much food as she would like. During the taste task, each participant was left alone for 15 minutes in the room to minimize uncomfortable and negative feelings that might arise if someone were to see

her eat. After the taste task, the participant was again asked to complete the mood, distress, hunger level, and urge to eat VAS scales (Time 3). Next, participants were presented with the Food Intake Questionnaire. To ascertain whether participants suspected or were aware of the purpose of the study, each participant also completed the funneled debriefing form. At the end of the study, participants were fully debriefed of the real purpose of the study, compensated for their time, and if needed provided with referrals.

Mood Induction. To induce sad mood, participants were asked to write down on a piece of paper “a memory that makes you sad.” After writing, participants were instructed to continue thinking of the memory and to “try to get into a sad mood” for 10 minutes. The participant was alone in the room listening to the orchestral introduction to *Russia Under the Mongolian Yoke* by Prokofiev, recorded at half-speed (Martin, 1990). Studies by Segal et al. (1999) and Gemar, Segal, Sagrati, and Kennedy (2001) showed that this procedure is effective in inducing a transient dysphoric mood state that lasts for 10-15 minutes. This procedure was also personally-relevant in that it used a self-selected sad memory. These instructions were provided both verbally and in written form as a reminder.

Taste Task and Food Intake. Instructions for the taste task were provided during orientation to visit 2. Written instructions were presented as well. During the taste task, each participant was asked to take a seat behind a desk with three pre-weighed bowls of food: chocolate candies, potato chips, and cookies. Consistent with procedures used by

Dingemans et al.(2009) after the tasting, participants were asked to rate the three kinds of foods for palatability, intentions to eat, taste and smell on the Taste Task Questionnaire (TTQ). Participants were asked to fill out the questionnaire in order to ensure that they actually consumed each food item. However, data from the TTQ was not analyzed. The total amount of food remaining was measured afterwards and the total caloric intake was calculated. The taste task employed and the instructions provided to the participants were consistent with those employed in other studies assessing the impact of a mood induction on subsequent caloric consumption (e.g., Dingemans et al., 2009).

DATA ANALYTICAL STRATEGIES

Overview of Data Analysis

All analyses were conducted using PAS-W 22.0 statistical software. To address the a priori hypotheses, t-tests, one-way AN(C)OVA, repeated measures AN(C)OVA, and hierarchical multiple regression procedures were conducted as appropriate. Prior to data analyses, all study data was screened for missing data and accuracy of data entry was verified. Univariate descriptive statistics were computed to ensure that variables and their distributions met assumptions of proposed univariate and multivariate analyses. For regression analyses involving interaction terms, all continuous variables were mean centered prior to their inclusion in the model. In addition, because the current study was not adequately powered to detect statistically significant 3-way interactions, 3-way interactions among predictors are deemed exploratory. Accordingly, analyses involving the Group \times DTS/RRS \times EEI-NA interaction term were conducted to explore whether the interactive impact of emotion regulation strategies varied by group. Analyses were repeated within each study group (i.e., DTS/RRS \times EEI-NA) even in the absence of a significant 3-way interaction.

Because BDI-II and/or BMI may influence reactivity to sad mood induction and/or change in urge to eat in response to sad mood induction or taste task, we aimed to evaluate whether a repeated measures ANCOVA or ANOVA framework should be used. To this end, both group differences and the relation between potential covariates (i.e., BMI and BDI-II scores) and outcomes of interest (i.e., sad mood ratings and urge to eat

ratings on the VAS were examined) were examined. To assess whether BDI-II scores and/or BMI should be used as control variables in regression analyses we examined zero-order correlations among BDI-II total scores/BMI, outcomes of interest, and other predictors. VIF and tolerance values within regression models were also examined to assess for multicollinearity.

Participant Characteristics

ANOVA, independent group t-tests, and chi-square procedures were used to assess whether BED-diagnosed participants significantly differed from control participants in baseline ratings of mood, age, years of education, income, marital status, BMI, and BDI-II total scores, scores on questionnaires measuring eating-related pathology and health-relevant behaviors. Distribution of comorbid psychiatric conditions in each group was also examined. Zero-order correlation coefficients were computed to examine bivariate relations among study variables with each study group.

Changes in Mood Across the Study

To establish that all participants became significantly more sad after the mood induction, a 2 (BED; Control) \times 3 (baseline, post-mood induction, post-taste test) repeated measures ANOVA was used to examine changes in sad mood (VAS score) across the study. An ANOVA versus an ANCOVA framework was selected based on preliminary analyses that examined group differences in putative covariates (i.e., BDI-II and BMI) as well as patterns of correlation among these variables and VAS scores within each study group. Similar procedures were followed for anger, anxiety, tired, tense, and

happy VAS scales and for SUDS ratings. This served as a manipulation check and to identify significant group differences in patterns of mood across the study.

Aim 1: Hypotheses 1 and 2: Examine Group Differences in Proposed Cognitive and Affective Vulnerability Processes

A series of t-tests was conducted to test whether participants with BED, on average, scored higher on self-report measures of ruminative brooding on the RRS-B, demonstrated lower distress tolerance on the DTS, and perceived themselves as having fewer strategies to cope with negative mood on the DERS (Hypothesis 1). For the second set of analyses (Hypothesis 2), a series of t-tests tested whether BED-diagnosed participants endorsed greater urge to eat to cope with negative emotions on the EES and reported greater expectancy that food will serve to regulate negative affect on the EEI. Group differences in these variables were also examined within an ANCOVA framework, with BDI-II scores included as a covariate.

Aim 2: Hypothesis 3: Individual Differences in Proposed Cognitive and Affective Vulnerability Processes and Binge Eating Severity in Persons with BED

Using only BED-diagnosed participants, three separate hierarchical linear regression analyses were conducted on the following dependent variables: BES scores, EES scores, and the number of binge eating episodes in the past 28 days as indicated on the on the EDE-Q. When appropriate, BDI-II scores were controlled for in the first step. Predictor variables, including DTS total score, RRS-Brooding, and EEI-NA were entered as a block in the second step to examine the variance in the outcome accounted for by

each predictor. Although, a-priori, we proposed to include DERS scores in the model, preliminary analyses revealed that DERS total scores were highly correlated with DTS scores, leading to suppression. Thus, DERS total scores were not included in the multivariate model although relations between DERS total scores and outcomes of interest were examined at the univariate level via zero-order correlations.

Aim 3. Experimentally Induced Sad Mood and Caloric Consumption, Urge to Eat, and Perceived Loss of Control

Hypothesis 4: Urge to Eat, Caloric Consumption, and Perceived Loss of Control Following Mood Induction. A repeated measures 2 (BED, control) \times 3 (baseline, post-mood induction, post taste-task) ANOVA was used to examine the effects of mood induction on urge to eat as rated on the VAS. T-tests were conducted to assess whether BED-diagnosed participants consumed more calories and experienced greater sense of loss of control during the taste task relative to control participants. To assess potential impact of BMI and BDI-II on results, analyses were re-run using an ANCOVA framework, with BDI-II and BMI in the model. Follow-up analyses used independent samples t-tests to compare BED and control participants on perceived rate of eating and amount eaten as well as post-taste task guilt ratings. Because there was only one type of mood induction in this study, findings were not attributed to effects of mood on food intake; instead, results examined the hypothesis that participants with BED consumed more calories than control participants under conditions of dysphoric mood in a laboratory setting.

Hypothesis 5: Mood Reactivity and Binge Eating. To assess whether degree of change in sad mood in response to the dysphoric mood induction predicted subsequent caloric intake, change in urge to eat, and loss of control, three separate hierarchical regression analyses were conducted, one for each of these three outcome variables. Group (BED, control) and sad mood change scores (Time 1 to Time 2) were the main predictors. Consistent with other work in this area (Dingemans et al., 2009), a mood change score was calculated by subtracting the VAS sad mood score before the mood induction (Time 1) from the VAS sad mood score obtained after the mood induction (Time 2). BDI-II scores were entered in the first step. The two predictors were entered in the second step, and an interaction term (Group \times VAS sad mood change score) was entered in the final step. To address power concerns analyses were also repeated without BDI-II scores in the model. Exploratory analyses within each patient status group were also conducted. Specifically, controlling for the effects of BDI-II, impact of VAS sad mood change score on caloric intake, perceived control over eating, and change in urge to eat was examined.

Aim 4. To Elucidate the Relationship between Sad Mood and Binge Eating Behavior by Examining the Role of Cognitive and Affective Vulnerabilities

Hypotheses 6-8: Brooding Rumination. It was proposed that combined results from analyses associated with Hypothesis 5 and 6 would provide initial support for the proposed relation among rumination, negative affect, and binge eating. Specifically, it was proposed that levels of rumination would predict increase in sadness (hypothesis 6a). In turn, degree of change in sad mood was proposed to predict caloric intake and change

in urge to eat (hypothesis 5). To test whether ruminative-brooding predicted reactivity to a dysphoric mood induction (Hypothesis 6a), the first regression equation included Group and Rumination as predictors in the first step and an interaction (Group \times RRS-B score) in the second step. The outcome variable was change in sadness in response to the mood induction (VAS change score). We also performed this analysis with BDI-II total scores accounted for in the first step. Caloric intake and VAS change in urge to eat score were also regressed on RRS-B total scores, Group, and the RRS-B \times Group interaction to assess direct effects of rumination on caloric intake and change in urge to eat from pre- to post-sad mood induction (Hypothesis 6b).

The next set of analyses assessed whether brooding rumination and negative reinforcement eating expectancies directly predicted change in urge to eat and caloric intake following a dysphoric mood induction in BED and control participants (Hypothesis 7). For both change in urge to eat (VAS change score) and actual caloric intake, identical hierarchical linear models were computed. Specifically, predictors (i.e., brooding rumination on the RRS-B, eating expectancies on the EEI-NA, and Group) were entered in the first step, followed by the interaction terms of EEI-NA \times RRS-B, RRS-B \times Group, and EEI-NA \times Group in the second step. The EEI-NA \times RRS-B \times Group was entered in the third step.

Finally, we examined unique and interactive effects of brooding rumination and negative reinforcement eating expectancies on caloric intake and change in urge to eat within each study group (Hypothesis 8). For both outcomes, hierarchical linear models

were computed with RRS-B total scores and EEI-NA total scores entered in the first step and the EEI-NA \times RRS-B interaction entered in the second step.

Hypothesis 9-10: Distress Tolerance. The analyses used to address hypotheses 7 and 8 for brooding rumination (as described above) were repeated using distress tolerance as the main predictor. Specifically, the first set of analyses used a hierarchical regression approach to examine impact of DTS total scores, EEI-NA total scores, and their interaction on group differences in caloric intake and change in urge to eat following dysphoric mood induction. For both outcomes, Group, DTS total scores and EEI-NA were entered in the first step, followed by DTS \times EEI-NA, DTS \times Group, and EEI-NA \times Group in the second step, and the DTS \times EEI-NA \times Group in the third step. The second set of analyses used a hierarchical linear modeling approach to assess the unique and interactive effects of DTS and EEI-NA total scores within each study group. In each study group, for both outcomes, DTS and EEI-NA total scores were entered in the first step followed by DTS \times EEI-NA interaction term in the second step.

RESULTS

Preliminary Analyses

Although variables were generally normally distributed when collapsing across groups (i.e., BED and control groups), variables assessing Eating Disorder psychopathology as well as BDI-II, RRS-B, DTS, and EEI-NA were negatively skewed within BED participants, reflecting that the majority of respondents with BED had higher (vs. lower) scores on these measures. BED participants were expected to score relatively high on these measures, consistent with theoretical models of BED and affect-regulation models of binge eating. Because transformation would hinder interpretation of the analysis, results with untransformed variables are reported. However, a logarithmic transformation was conducted for study variables that were significantly skewed and analyses were conducted with both the untransformed and transformed variables for comparison, yielding identical results (Tabachnick & Fidell, 2007). See Table 2 for descriptive information on study variables.

Participant Characteristics

Of the 102 participants who signed the informed consent form, 77 female participants met study eligibility criteria and were enrolled in the study (BED $n = 39$; Control $n = 38$). A participant in the BED group subsequently reported purging and, therefore, her data was not included in current analyses. The final sample for data analysis included 76 participants (38 BED, 38 controls) with complete data available at

visit 1 and for 74/76 participants (97.4%) at visit 2. Specifically, two control participants did not return for visit 2.

Descriptive information for baseline demographic characteristics and study outcome variables, along with statistics comparing the BED and control groups, are presented in Table 1 and Table 2, respectively. Paired samples *t*-tests were conducted to examine differences between BED and Control participants on demographic variables. In cases where, according to the Levene's Test, the equality of variances assumption for the *t*-test was violated (i.e., variability in scores in the two groups was not the same), results with equal variance not assumed are reported. Overall, of the majority of the 76 participants included in the study, ($n = 67$, 88.2%) self-identified as Caucasian/White. The mean age of participants was 24.7 years ($SD = 10.2$; range = 18-65) and most ($n = 60$, 78.9%) were single. There were no differences between the BED and control groups in marital status, age, and years of education (see Table 1). On average, participants' BMIs were in the overweight range ($M = 25.9$, $SD = 4.8$). Although the study aimed to control for BMI, on average, participants in the BED group had significantly higher BMIs ($M = 27.0$, $SD = 5.4$) than participants in the control group ($M = 24.4$, $SD = 10.3$).

In terms of health relevant behaviors, 81.6% ($n = 62$) of participants reported exercising on a regular basis and 28.9% ($n = 22$) endorsed following a special diet or trying to lose weight, with BED participants more likely to endorse dieting efforts than control participants (22.4% versus 6.6%, respectively), $X^2(1, N = 76) = 9.21$, $p < .01$. Fifty percent of participants acknowledged drinking alcohol on a regular basis and 56.6%

($n = 43$) of participants reported using birth control methods. Only three participants self-identified as cigarette smokers.

Participants with BED were more likely than control participants to meet diagnostic criteria for another Axis I disorder $X^2(1, N = 76) = 9.77, p < .01$. The most common concurrent diagnosis among BED participants was Generalized Anxiety Disorder ($n = 4$). Accordingly, participants with BED (15.8%) were also more likely to be currently receiving mental health treatment than control participants (5.3%), $X^2(1, N = 76) = 5.07, p < .05$. Although a current diagnosis of major depression was an exclusion criterion, 10 participants met criteria for lifetime Major Depressive Disorder. In terms of current depressive symptoms, participants with BED had significantly higher BDI-II scores than control participants. Participants with BED also scored higher than controls on a questionnaire measuring eating-related pathology, the EDQ, and on its weight, shape, eating concerns, and dietary restraint subscales. Participants with BED scored higher on EES-D than control participants, endorsing a greater tendency to eat to cope with depressed mood. As expected, participants with BED had significantly higher BES scores, endorsing symptoms consistent with a diagnosis of binge eating disorder¹.

¹ Of note, when BDI-II scores were entered as covariates in analyses examining group differences in binge eating severity as indexed by the BES, a complex pattern emerged. Higher BDI-II scores were associated with more severe binge eating symptoms, regardless of group membership. However, within the control group, higher depressive symptoms were associated with lower binge eating severity relative to the BED

Zero-Order Correlations among Study Variables

In order to examine univariate relations among study variables, zero-order correlations were computed among study variables separately for each patient group. (See Table 3). Neither BMI nor BDI-II total scores were significantly correlated with caloric intake, pre- to post-mood induction change in sad mood, or pre- to post-induction change in urge to eat in either group. However, among control participants only, BDI-II scores were correlated with perceived loss of control ratings ($r = -.58, p < .01$), post taste-task sadness rating ($r = .33, p = .05$), perceived amount eaten ($r = .35, p < .05$), and post-task guilt ratings ($r = .69, p < .01$). In BED women, BDI-II scores were only positively correlated with post-taste task guilt ratings on the VAS ($r = .37, p < .05$). Although BED participants, on average, had higher BMIs, BMI was not significantly correlated with any outcome of interest in either study group. Thus, BMI was not included in regression analyses. Because BDI-II scores were positively and significantly associated with BES total scores among both participants with BED and control participants, BDI-II total scores were included in analyses focusing on BES total scores as the outcome measure.

Among control group women only, BDI-II scores were also significantly and positively correlated with EES-D scores. In line with this result, among control women only, number of binge eating days was positively associated with EES-D total scores and

group, lending further credence to the hypothesis that binge eating may serve to escape depressive symptoms among individuals with BED.

BES total scores were positively correlated with EEI-NA. Of interest, among control women, perceived loss of control over eating during the taste task was correlated with BES scores ($r = -.58, p < .01$), Pre- to Post-Taste Task change in sad mood ($r = -.35, p < .01$), Pre- to Post-Induction change in urge to eat ($r = -.34, p < .01$), and perceived quantity of food eaten ($r = -.46, p < .01$). In contrast, among women with BED, perceived loss of control over eating was only correlated with actual amount of calories eaten ($r = -.58, p < .01$) and perceived amount eaten. Of note, a greater increase in sad mood in response to the sad mood induction was associated with lower caloric intake in control women ($r = -.45, p < .01$).

At the univariate level, among women with BED, BES total scores were positively correlated with RRS-B total scores and were not significantly associated with DTS total scores or EEI-NA total scores. In contrast, among control women, BES total scores were positively associated with EEI-NA total scores and negatively correlated with DTS total scores (i.e., better distress tolerance was associated with lower BES total scores). Among BED women, brooding rumination was also positively correlated with EEI-NA scores, such that greater tendency to brood was associated with higher negative reinforcement eating expectancies. In both groups RRS-B total scores were negatively correlated with DTS total scores and DTS total scores were negatively correlated with EES-D total scores. That is, lower distress tolerance was associated with greater tendency to brood and reported tendency to eat to cope with sad mood. In control women, distress tolerance was also negatively associated with EEI-NA (i.e., better distress tolerance was

associated with lower EEI-NA). In both groups, negative reinforcement eating expectancies were positively correlated with EES-D scores (Table 3).

In control women only, reported guilt following the taste task was correlated with RRS-B total scores ($r = .52, p < .01$) and with EEI-NA scores ($r = .41, p < .01$). In BED women, reported guilt after the taste task was correlated with perceived rate of eating ($r = .60, p < .01$), EES-D scores ($r = .33, p < .05$), and perceived loss of control over eating ($r = -.56, p < .01$). The correlation among reported guilt and brooding rumination scores approached significance ($r = .31, p = .06$). In control women, guilt ratings were correlated with perceived loss of control ($r = -.49, p < .01$), EES-D scores ($r = .41, p < .01$), and RRS-B total scores ($r = .52, p < .01$). In BED women, perceived rate of eating was negatively correlated with perceived control over eating ($r = -.58, p < .01$) and positively correlated with perceived guilt ($r = .60, p < .01$), total calories eaten ($r = .50, p < .01$), and DTS total scores ($r = -.37, p < .01$). In control women, only the correlation between perceived rate of eating and degree of sad mood change following sad mood induction ($r = -.32, p = .06$) approached significance. In BED women only, there was a trend towards a correlation between post-induction sad mood rating RRS-B total scores ($r = .31, p = .06$).

Manipulation Check for Mood Induction: Changes in Mood across the Dysphoric Mood Induction and Taste Task

Sad mood ratings on the VAS by patient status group are shown in Table 4. Given that both independence of the covariate and treatment effect (i.e., group) and

homogeneity of regression slopes assumptions underlying ANCOVA were not met for BDI-II scores and BMI, a repeated measures ANOVA framework was used for all repeated measure analyses. To examine if the mood manipulation had the intended effect, a mixed design repeated measure analysis of variance (ANOVA) was conducted utilizing two factors: Group as a between-subjects factor (i.e., BED, Control) and Time as a within-subject factor (Time 1:pre-mood induction, Time 2: immediately after sad mood induction and before the taste task, Time 3: immediately after taste task) predicting the dependent variable of sad mood on the VAS sad-mood scale. The Huynh-Feldt correction was used when, according to Mauchly's test, the assumption of sphericity for the repeated measures design was violated. As expected, there was a main effect of Time. Across groups, there was a significant change in mood over the course of the laboratory session ($F(2, 144) = 125.04, p < .01$). Follow-up analyses revealed that within each group, there was an increase in dysphoric mood from pre- to post- mood induction and a decrease in dysphoric mood from post-mood induction to post-taste task (Figure 2). In contrast to our prediction, we did not observe a main effect for Group ($F(1, 72) = 1.32, p > .05$). Ratings of sad mood were, on average, comparable across groups. The interaction of Group \times Time approached significance ($F(2, 140) = 1.86, p = .09$). Specifically, post-mood induction, participants with BED appeared to demonstrate greater increase in sad mood. Because greater reactivity to sad mood induction among BED participants may reflect greater depression severity in this group, we examined the pattern of correlations among BDI-II scores and VAS sad mood ratings within each study group at pre- and

post-mood induction and post taste task. Among control participants only, the correlation among BDI-II scores and both pre-induction and post-taste task ratings of mood approached significance ($r = .28, p = .09$ and $r = .33, p = .05$, respectively). However, among BED participants, at no point were BDI-II associated with sad mood ratings. Overall, these findings suggest that greater depressive symptoms predict reactivity to sad mood and lesser improvement in mood following taste task only among control participants.

Ratings of anger, anxiety, tiredness, subjective distress, and happiness were also examined. Across groups, there was a main effect of Time on ratings of tiredness ($F(2, 144) = 4.27, p < .05$), subjective distress ($F(2, 144) = 9.53, p < 0.01$), anger ($F(1.59, 111.25) = 3.58, p < 0.05$), and happiness ($F(1.78, 124.88) = 25.11, p < .01$, again using the Huynh-Feldt correction). Follow-up analyses revealed that, for both groups, subjective ratings of tiredness, distress, and anger increased from pre- to post- mood induction and decreased from post-mood induction to post-taste task, whereas subjective ratings of happiness decreased from pre- to post-mood induction and increased from post-mood induction to post-taste task. For subjective distress ratings, the interaction of Group \times Time was also significant ($F(2, 144) = 3.53, p < 0.05$). Although both groups reported comparable levels of subjective distress pre-mood induction, BED participants demonstrated greater increase in distress from pre- to post-mood induction and remained more distressed relative to controls. As expected, we did not find statistically significant within or between group differences on anxiety ($F(2, 144) = 2.31, p = 0.10$).

Study Results by Hypothesis

A summary of the study hypotheses, the statistical methods used to test each hypothesis, and a descriptive summary of results are presented in Table 5.

Group Differences in Proposed Cognitive and Affective Vulnerabilities (Hypotheses 1 and 2)

A series of t-tests was conducted to evaluate group differences in putative cognitive and affective vulnerabilities to BED. Results are presented in Table 2. As expected, BED participants had significantly higher RRS brooding rumination scores and endorsed greater difficulty with emotion regulation as indexed by higher DERS scores. As predicted, BED participants also had lower distress tolerance relative to control participants and endorsed more positive expectancies that food serves to regulate negative affect.

Individual Differences in Proposed Cognitive and Affective Vulnerability Processes and Binge Eating in Persons with BED (Hypothesis 3)

Using only BED-diagnosed participants, three separate hierarchical linear regression analyses were conducted on the following dependent variables: BES scores, EES-D scores, and the number of binge eating episodes in the past 28 days as reported on the on the EDE-Q. Because BDI-II scores were positively and significantly correlated with BES total scores, BDI-II total scores were entered in the first step when specifying a model for this outcome. Predictor variables, including DTS total score, RRS-B total scores, and EEI-NA total scores were entered as a block to examine the variance in the

outcome accounted for by each predictor. For BES total scores, in step 1, BDI-II scores accounted for 28.6% of variance in BES scores ($F(1, 35) = 14.44, p < 0.01$). After controlling for BDI-II scores, cognitive and affective vulnerabilities explained additional 16.7% of variability in BES total scores ($F(4, 32) = 3.37, p < 0.01$). However, of the predictors examined in the second step, only RRS-B total scores was positively and significantly associated with BES scores ($b = .40, t(33) = 2.47, p < .05$). For EES-D total scores, BDI-II scores were again entered in Step 1 and accounted for 8.0% of variability in EES-D scores ($F(1, 36) = 3.14, p = 0.08$). Processes related to emotion regulation explained additional 28.7% of variation in EES-D scores. Of the predictors examined in the second block, only EEI-NA emerged as a significant predictor of EES-D total scores ($b = 1.35, t(34) = 2.87, p < .01$). Because BDI-II scores were not correlated with number of binge eating days in the past 28 days, BDI-II scores were not included in the model pertaining to this outcome. The overall model with RRS-B, DTS, and EEI-NA entered simultaneously, accounted for 21.8% of variability in frequency of binge eating ($F(3, 34) = 3.17, p < 0.05$). In predicting number of binge eating days, DTS scores and EEI-NA scores emerged as significant predictors ($b = 3.76, t(34) = 2.51, p < .01$ and $b = 1.84, t(34) = 2.05, p = .05$, respectively). Interestingly, for BED women, better (i.e., higher) distress tolerances was associated with increased frequency of binge eating days over the past 28 days. Consistent with current study's hypothesis, a greater negative reinforcement eating expectancy was associated with higher binge eating frequency.

Group Differences in Urge to eat, Perceived loss of control, and Caloric Consumption Following Mood Induction (Hypothesis 4)

A repeated measures 2 (BED, control) \times 3 (baseline, post-mood induction, post taste-task) ANOVA was used to examine the effects of mood induction on urge to eat as rated on the VAS. There was a main effect for Group, such that, collapsing across time points, participants with BED reported greater urge to eat than controls ($F(1, 72) = 16.89, p < .01$, partial eta squared .19). The effect of time was not significant, suggesting that, collapsing across groups, urge to eat did not change from pre- to post- mood induction, to post-taste task. Although, and in contrast to study hypothesis, the Group \times Time interaction was not significant, visual display of participants' reported urge to eat suggested that urge to eat decreased somewhat from pre- to post- mood induction for control participants, whereas urge to eat increased for BED group participants. (See Figure 3).

T-tests were used to assess group differences in caloric intake and perceived loss of control. BDI-II scores and BMI were not included as covariates because neither was significantly associated with these outcome variables². During the taste task, BED

² Supplementary analyses with BDI-II and BMI in the model were also performed and yielded similar results. An ANCOVA framework controlling for the effects of depressive symptoms (BDI-II) and BMI was used to assess group differences in caloric intake and perceived loss of control. During the taste task, BED participants consumed, on average, 169.11 more calories than control participants ($F(1, 73) = 7.37, p < .01$). As expected, control participants rated their perceived control over the amount eaten as significantly

participants consumed, on average, 222 more calories than control participants. As expected, control participants rated their perceived control over the amount eaten as significantly higher than BED participants. Following the taste task, on average, control participants rated their perceived control over amount eaten as 81.57 ($SD = 15.05$) on a 100mm VAS (0 = no control and 100 = complete control) versus 53.05 ($SD = 29.94$) for BED participants. A larger portion of participants with BED endorsed having experienced a binge episode during the taste task relative to controls, 14/38(36.8%) in BED group versus 2/38(5.3%) in the control group ($X^2(1, N = 76) = 12.08, p < .01$).

In terms of participants' subjective experience as assessed with the VAS scales, participants with BED reported eating faster (mean differences of 21.81mm on the VAS, $t(74) = 3.72, p < .01$) and consuming a greater amount of food (mean difference of 12.58mm on the VAS, $t(57.41) = 2.30, p < .05$) than control participants. Women with BED also reported feeling more guilt after the taste task relative to control women (mean difference of 30.23mm on the VAS, $t(63.71) = 0.08, p < .01$, equal variance not assumed).

higher than BED participants ($F(1, 73) = 13.66, p < .01$). Following the taste task, on average, control participants rated their perceived control over amount eaten as 80.01 ($SD = 4.60$) on a 100mm VAS (0 = no control and 100 = complete control) versus 54.34 ($SD = 4.37$) for BED participants.

Mood Reactivity and Binge Eating (Hypothesis 5).

To assess whether degree of change in sad mood in response to the dysphoric mood induction predicts subsequent caloric intake, change in urge to eat, and/or loss of control separate hierarchical regression models were conducted, with caloric intake, loss of control rating, and change in urge to eat as the outcome variables, respectively. For all models, BDI-II scores were entered in the first step to control for potential effects of depressive symptoms on reactivity to the sad mood induction³, followed by Group (BED, Control) and sad mood change scores [Time 1 (before mood induction) to Time 2 (after mood induction)] in Step 2. An interaction term (Group \times VAS sad mood change score) was entered in the final step.

In predicting caloric intake during the taste task, the overall model with Group, VAS sad mood change score, and BDI-II scores was significant ($F(3, 68) = 5.79, p < .01$), accounting for 22.9% of variability in caloric intake during the taste task. In contrast to study hypothesis, only Group predicted caloric intake during the taste task ($\beta = .36, t(72) = 2.79, p < .01$). Neither degree of sad mood change ($\beta = -.02, t(72) = -.22, p = .82$) nor the interaction of Group \times VAS sad mood change score ($\beta = .39, t(72) = 1.61, p = .10$) were significantly associated with caloric intake. BDI-II scores also did not account for variability in caloric intake. A similar pattern of results was observed for perceived control over eating. The overall model with Group, VAS sad mood change

³ Analyses were repeated without BDI-II scores in the model, yielding similar results.

score, and BDI-II scores was significant ($F(3, 68) = 8.65, p < .01$), accounting for 24.2% of variability in perceived control over eating during the taste task. In contrast to study hypothesis, only Group predicted ratings of control on the VAS ($\beta = -.46, t(72) = -3.61, p < .01$). Neither degree of sad mood change ($\beta = -.08, t(72) = -.77, p = .44$) nor the interaction of Group \times VAS sad mood change score ($\beta = -.19, t(72) = -.78, p = .43$) were significantly associated with perceived control over the amount eaten. For change in urge to eat from pre- to post- mood induction, none of the factors examined were significantly associated with change in urge to eat ($p > .05$ for all zero-order correlation coefficients). The overall model with BDI-II score, Group, VAS sad mood change score, and Group \times VAS interaction did not account for significant variability in change in urge to eat ($F(4, 68) = .85, p = .50$).

Using a hierarchical regression framework, analyses within each patient status group were conducted. Specifically, controlling for the effects of BDI-II, impact of VAS sad mood change score on caloric intake, perceived control over eating, and change in urge to eat was examined. Surprisingly, the majority of significant findings pertained to control participants rather than to women with BED. In predicting total caloric intake for the control group, adding VAS mood change score to the model, increased variance accounted for by 10.3%, which approached statistical significance ($F(1, 32) = 1.60, p = .06$). Consistent with findings reported by Loxton, Dawe, and Cahill (2011), greater VAS sad mood change (i.e., worsening mood) was associated with lower caloric intake for control group participants ($\beta = -.32, t(32) = -1.92, p = .06$). VAS sad mood change score

was not associated with caloric intake for BED women. In addition, for control participants only, BDI-II scores were significantly and negatively associated with perceived control over amount eaten ($r = -.58, p < .01$). This effect was maintained in the full model, with a significant partial effect for BDI-II scores ($\beta = -.59, t(32) = -4.08, p < .01$). In contrast, for BED participants, change in VAS sad mood scores were weakly and negatively associated with perceived control over eating ($r = -.15, p < .05$). However, this effect was not maintained in the full model. No significant predictors were identified for change in urge to eat for either group.

Direct and Indirect Effects of Brooding Rumination (Hypotheses 6-8)

A series of analyses were conducted to evaluate both direct and indirect effects of rumination on participants' eating behavior and subjective ratings. A series of hierarchical linear regression models were computed to assess impact of brooding rumination on change in sad mood from pre-to post sad mood induction (Hypothesis 6a) as well as total caloric intake and change in urge to eat from pre- to post-sad mood induction. To test whether ruminative-brooding directly predicts reactivity to a dysphoric mood induction and whether this effect varies by group, Group and⁴ Rumination were entered as predictors in the first step and an interaction (Group \times RRS-B total scores) in

⁴ Of note, the analysis was redone with BDI-II total scores in the model, yielding similar results to those reported in text. However, we chose to present results without BDI-II total scores in the model because BDI-II scores did not co-vary with the outcome of interest (i.e., VAS sad mood change score), but were significantly, positively, and highly correlated with RRS-B total scores.

the second step. For all models, the overall regression model was not significant (with $p > 0.05$ for all steps). Thus, RRS-B total scores, Group, and/or the Group \times RRS-B total scores interaction did not account for significant variability in pre- to post-mood induction change in VAS sad mood score. Of note, results of univariate analyses showed that, in the entire sample, total caloric intake differed by group and was positively associated with RSS-B total scores ($r = .28, p < .05$). Next we assessed direct effect of rumination on caloric intake (Hypothesis 6b). Although the overall model predicting caloric intake from RSS-B total scores and group was significant ($F(3, 69) = 6.86, p < .01$) and accounted for 23% of variance in caloric intake, RSS-B was not a significant predictor of caloric intake ($\beta = -.06, t(72) = -.37, p = .71$). Adding the Group \times RSS-B interaction term did not significantly increase the variance accounted for ($F(1, 68) = 8.65, p = .65$). Regarding change in urge to eat, urge to eat change score was only weakly correlated with BDI-II scores ($r = .19, p = .05$) and was not significantly associated with RRS-B total score ($r = .11, p = .24$). For all three sequential hierarchical models, the overall regression model was not significant (with $p > 0.05$ for all steps). Group, RRS-B total scores, and/or Group \times RRS-B did not account for significant variability in VAS urge to eat change score. Thus, although brooding rumination correlated with caloric intake, neither RRS-B score nor RSS-B \times Group interaction predicted changes in SAD mood, change in urge to eat, or actual caloric consumption in the context of hierarchical linear models.

The next set of analyses evaluated the unique and interactive effects of brooding rumination and negative reinforcement eating expectancies on actual caloric intake (see Table 5) and changes in urge to eat (see Table 6). For both outcome variables, identical linear regression models were constructed hierarchically. Group, RRS-B total scores, and EEI-NA total scores were entered as a block in the first step. The interaction terms of EEI-NA \times RRS-B, RRS-B \times Group, and EEI-NA \times Group were included in the second step, followed by an exploratory 3-way interaction EEI-NA \times RRS-B \times Group in the third step (Hypothesis 8). Again, for caloric intake, the overall model was only significant in the first step. Specifically, only group accounted for significant variability in caloric intake. None of the predictors examined accounted for variability in change in urge to eat from pre- to post- mood induction.

Finally, we conducted a series of analyses (Hypotheses 7- 8) looking at unique and interactive effects of brooding rumination and negative reinforcement eating expectancies on caloric intake (see Table 7) and change in urge to eat (see Table 8) within each study group. For each study group, RRS-B total scores and EEI-NA were entered in the first step followed by RRS-B \times EEI-NA interaction in the second step. Neither RRS-B, EEI-NA, nor their interactive effects were predictive of caloric intake in either patient status group ($p > .05$ for all steps, see Table 7). These variables also did not predict VAS change score in urge to eat in either study group ($p > .05$ for all steps, see Table 8). Overall, the predictors examined in this set of analyses did not explain between

or within group variability in both actual caloric intake or subjective change in urge to eat from pre- to post- mood induction.

Distress tolerance (hypotheses 9-10)

The analyses used to address hypotheses 7 and 8 for brooding rumination (as described above) were repeated using distress tolerance as the main predictor. With patient status group in the model, neither DTS total scores nor did EEI-NA total scores accounted for significant variability in total caloric intake (see Table 9)⁵. In the context of a hierarchical model, the groups did not differ in the effect of EEI-NA or DTS total scores on caloric intake. The *t* test for the DTS × EEI-NA interaction term was also not significant. The 3-way interaction of Group × EEI-NA × DTS total scores was also not significant.

In terms of urge to eat, there was no main effect for Group, DTS total scores, or EEI-NA total scores ($p > .05$ for all steps, see Table 10). In step 2, with main effects and 2-way interaction terms in the model, the interaction of EEI-NA total and DTS total scores was a significant predictor VAS change in urge to eat score ($\beta = .32$, $t(72) = 2.09$, $p < 0.05$). However, because the overall model was not significant (i.e., adding interaction terms in step 2 did not increase variability accounted for in VAS urge to eat change

⁵ Post-hoc, as with rumination, these analyses were also done with just Group and DTS scores and just Group and EEI-NA scores in the model, such that Group and DTS/EEI-NA were entered in step 1 and the interaction of Group x EEI-NA/DTS was entered in step 2. There were no significant findings for either caloric intake or VAS change in urge to eat score ($p > .05$ for all models).

score), we did not probe the interaction. The 3-way interaction of EEI-NA \times DTS \times Group was not significant.

A series of analyses (Hypothesis 10) examining unique and interactive effects of distress tolerance and negative reinforcement eating expectancies on caloric intake (see Table 11) and change in urge to eat (see Table 12) within each study group were conducted. For each patient status group, DTS total scores and EEI-NA were entered in the first step followed by DTS \times EEI-NA interaction in the second step. Among control group participants, both DTS total scores and EEI-NA total scores emerged as significant predictors of caloric intake. Specially, among control participants, better distress tolerance was associated with decreased caloric intake on the taste task. Interestingly, greater endorsement of negative reinforcement eating expectancy was also associated with lower caloric intake on the taste task. The 2-way interaction was not a significant predictor of caloric intake (Table 11). Neither EEI-NA, DTS, nor their interaction emerged as significant predictors of caloric intake in BED participants.

In terms of change in urge to eat, no significant predictors for VAS change in urge to eat scores were identified for either control group or BED diagnosed participants ($p > .05$ for all steps, Table 12). However, observed relations were in the expected direction. In both groups, higher DTS total scores were associated with decreased pre- to post-mood induction change in urge to eat whereas greater negative reinforcement eating expectancies were associated with increased pre- to post-mood induction change in urge

to eat. In other words, better distress tolerance was associated with a lower urge to eat in response to the dysphoric mood induction.

DISCUSSION

The primary aim of the current study was to understand cognitive and affective mechanisms, particularly emotion regulation strategies that may explain binge eating in women with Binge Eating Disorder (BED). Three emotion regulation strategies were examined as candidate vulnerability constructs that may be implicated in the maintenance of binge eating in BED, particularly under conditions of negative affect: brooding rumination, expectancies that eating will alleviate negative affect, and distress tolerance. We examined whether lower distress tolerance, stronger expectations that eating serves to alleviate negative affect, and greater tendency to engage in brooding rumination a) characterize women with BED relative to control participants, b) impact frequency and severity of binge eating and eating to cope with depressed mood in BED women, and c) impact whether women with BED respond to transient sad mood with disinhibited eating in the lab.

Participants included 38 women meeting criteria for BED and 36 control women with no recent history of disordered eating. Both groups completed self-report measures assessing these emotion regulation strategies as well as measures assessing disordered eating and problematic eating tendencies. All women participated in a 10-minute sad mood induction, involving reflection on a personally-relevant sad memory while listening to music suggestive of dysphoric mood, followed by a 15-minute “taste task” that was designed to assess impact of the sad mood induction on subsequent caloric intake when snack foods were presented.

Cognitive and Affective Vulnerabilities for Binge Eating (Hypotheses 1 and 2)

We tested the hypothesis that women with BED evidence more frequent brooding rumination (brooding subscale of the Ruminative Response Scale; RRS-B scores), have lower distress tolerance (Distress Tolerance Scale; DTS scores) and greater difficulty with emotion regulation (Difficulties with Emotion Regulation Scale; DERS scores), endorse greater expectancies that eating will improve mood (negative affect subscale of the Eating Expectancies Inventory; EEI-NA scores), and have a greater urge to eat to cope with negative affect (depression subscale of the Emotional Eating Scale; EES-D) relative to controls. As expected, relative to controls, BED participants scored higher on brooding rumination, reported lower distress tolerance, endorsed greater difficulty regulating emotions, and displayed greater negative reinforcement eating expectancies (EEI-NA). Consistent with past findings (e.g., Masheb, 2009), women with BED were also more depressed than control participants, as indexed by Beck Depression Inventory-Second Edition (BDI-II) scores and reported greater urge to eat to cope with depressive symptoms (EES-D scores).

Individual Differences in Proposed Cognitive and Affective Vulnerability Processes and Binge Eating in Persons with BED (Hypothesis 3)

Among women with BED, the study examined whether brooding rumination (RRS-B scores), distress tolerance (DTS scores), and expectancies that eating will alleviate negative affect (EEI-NA scores) were associated with binge eating severity, number of binge eating days in the past month, and urge to eat to cope with negative

affect, after controlling for current depressive symptoms (BDI-II scores). Consistent with past research (Henderson & Huon, 2002; Stice et al., 2000; Telch & Agras, 1994), among women with BED, depressive symptoms were positively associated with binge eating severity, justifying the decision to consider depression as a covariate. Of the predictors examined in multivariate analyses, frequency of brooding rumination predicted binge eating severity among women with BED. This finding is correlational and, therefore, the direction of the relationship and the mechanism(s) underlying it are not known.

Rumination may lead to binge eating indirectly by exacerbating symptoms of depression or directly by interfering with adaptive coping. Individuals with BED may use food to escape brooding, self-focused, and aversive thoughts and/or to manage symptoms of depression (Aldao & Nolen-Hoeksema, 2010). As has been previously discussed, brooding rumination may further interfere with problem solving and use of effective coping strategies in persons with BED, particularly depressed BED patients. Alternatively, more severe binge eating may fuel rumination, such that patients with more frequent or extreme binge eating episodes may have more “to brood about.” Moreover, whereas the current study specifically focused on rumination about sadness, it is possible that patients with BED who engage in more frequent rumination also ruminate about other negative affective states (e.g., anxiety, disgust, frustration, guilt). These effects may be especially relevant in the case of repetitively thinking about the negative consequences of binge eating, including the impact of this behavior on weight/shape and self-attributions of being “out of control.” Accordingly, greater tendency to engage in

rumination as a cognitive control strategy may result in a greater sense of loss of control and greater perceived guilt, symptoms that are captured on our measure of binge severity, the Binge Eating Scale. In fact, brooding rumination frequency was associated with more guilt following the taste task in both the BED and control groups.

Consistent with the escape model of binge eating, a stronger expectancy that eating will improve mood was associated with eating to cope with depression and more frequent binge eating episodes in women with BED. These findings are in line with results reported by De Young, Zender, & Anderson (2014), who found that negative reinforcement eating expectancies prospectively predicted frequency of binge eating episodes in a mixed sample of ED patients. Surprisingly, in women with BED, better distress tolerance was associated with more frequent binge eating. Although this is the opposite of what was predicted, it could be interpreted as consistent with the escape model of binge eating. Women with BED may effectively tolerate distress in other (i.e., non-eating) life domains by approaching tasks that are challenging or unpleasant, with binge eating serving as a strategy for ‘replenishing’ coping resources depleted by life demands, rumination, and depressive symptoms. Alternatively, the consumption of palatable food, at least during the binge eating episode, is perceived as pleasant and rewarding (Parylak, Koob, & Zorrilla, 2011). Thus, eating may provide an ‘escape’ to a guilty pleasure, with the high distress tolerant BED patient using food more frequently to reward herself for coping with distress in other life domains.

Reactivity to Sad Mood Induction and Subsequent Caloric Intake (Hypotheses 4 and 5)

The current study tested whether a personally-relevant sad mood induction differentially affects women with BED versus control participants, such that women with BED consume more calories and report a greater increase in urge to eat following the mood induction as well as a greater loss of control and more guilt during the taste task relative to control participants. The sad mood induction had the intended effect, with all participants responding with a self-reported increase in sad mood. Consistent with past findings (e.g., Ng & Davis, 2013; Telch & Agras, 1996), women with BED consumed more calories and reported greater loss of control as well as a greater sense of guilt in response to the taste task relative to control participants. In contrast to study hypothesis, degree of change in sad mood in response to dysphoric mood induction did not predict subsequent caloric intake, change in urge to eat, or perceived loss of control over eating among BED participants. Although results for the BED group were surprising and in contrast to those reported by Chua et al. (2004) and Svaldi et al. (2010), they were in line with findings reported by Munsch et al. (2008) and Telch and Agras (1996, 1998) and are discussed in detail below. Interestingly, among control participants, a larger increase in sad mood following the mood induction was associated with decreased caloric intake. This finding was not surprising as it has been well documented that transient negative affect decreases appetite in non-eating disordered individuals, possibly via shifting attention away from eating cues.

There are several reasons why the degree of experimentally-induced sad mood did not affect outcomes in the BED group. First, it is possible that increased caloric intake during the taste task in BED reflects disinhibition of eating in the presence of food cues rather than reactivity to experimentally induced sad mood. Specifically, the effect of sad mood on caloric intake or perceptual features of binge eating may be “trumped” by the presence of food cues and their learned association with disinhibited eating. Therefore, food stimuli previously associated with overeating (i.e., the sight, smell, and taste of typical binge foods such as the cookies, chocolates, and/or chips presented in this taste task) may be sufficiently salient to promote overeating regardless of momentary sad affect. For instance, Todd, Winterbauer, and Bouton (2012) suggested that return to a context in which food-seeking has been reinforced leads to return of food-seeking behavior in sated rats. This work demonstrated that mere removal from a context associated with extinction of eating behavior was sufficient to re-activate the inhibited response of overeating (Todd et al., 2012).

Second, Dingemans et al. (2007, 2009) found that depression severity predicted both reactivity to sad mood induction and subsequent caloric intake. The authors reported that currently depressed BED patients, as indexed by moderate to severe depression levels on the BDI-II, were most affected by the sad mood induction and in turn consumed most calories on the taste task.

To assess whether baseline depressive symptoms on the BDI-II differentially predicted caloric intake and/or impact of reactivity to sad mood induction on caloric

intake, we conducted a series of post-hoc analyses using a hierarchical linear regression approach. Although results for caloric intake were not statistically significant ($p = .36$), likely due to small sample size, they are clinically meaningful. Depression severity did not impact caloric intake in control participants. Control women who scored -1 or +1 SD above mean BDI-II scores for the entire sample ($M = 8.81$, $SD = 8.80$), consumed on average 345.29 and 319.69 calories, respectively. In contrast, BED participants who scored +1 SD above the mean BDI-II (i.e., BDI-II scores > 17.61) consumed, on average, 586.68 calories relative to 475.09 calories among BED participants who scored at or -1 SD below the mean BDI-II. However, the majority of control participants endorsed minimal depressive symptoms on the BDI-II (see Table 2). Therefore, the BED group was subsequently divided, based on BDI-II norms (Beck et al., 1996), into a moderately to severely depressed group (BDI-II score = 20 or higher) and a non- to mildly depressed group (BDI-II score between 0 and 19). On average, “depressed” BED participants consumed 180.44 more calories than “not-depressed” BED women ($M_s = 684.54$ and 504.10, respectively). Thus, although BED women, in general, consumed more calories than controls on the taste task, BED women with more severe depressive symptoms, as indexed by the BDI-II, ate, on average, more calories than BED women with minimal depressive symptoms.

Further, post-hoc, we examined the interaction between VAS sad mood change score and BDI-II scores in terms of caloric intake among BED participants. As evident in Figure 3, highest caloric intake was observed among BED participants who were both

more reactive to the sad mood induction and endorsed greater depressive symptoms on the BDI-II (i.e., greater depressive symptoms relative to less depressed BED participants). Again, although the overall model was not statistically significant ($p = .10$), adding the interaction of VAS sad mood change \times BDI-II in the second step, explained an additional 9.4% of variability in caloric intake ($p = .06$). Although not statistically significant, the interactive effects of momentary sad mood state and general depression severity over the past 2 weeks are likely important for understanding processes driving binge eating in persons with BED. Consistent with past findings (Dingemans et al. 2009), it is possible that individuals with BED who endorse higher depressive symptoms at baseline are most vulnerable to binge eat in response to a transient increase in sad mood.

Third, if affective states are conceptualized as contexts for overeating, momentary negative affect alone (regardless of its intensity) and/or in conjunction with food cues may be sufficient to trigger habitual behavior (i.e., overeating) in BED (Loxton, Dawe, & Cahill, 2011). These results are in line with the observation that persons with BED tend to binge eat with a moderate increase in negative affect (Hilbert & Tuschen-Caffier, 2007; Munsch et al., 2012). Because the mood induction was effective in inducing sad mood, it is possible that BED participants ate more than controls because everyone got ‘sufficiently sad’ for disinhibition of eating to occur in those with BED, particularly in the context of appetitive food cues. Importantly, BED participants varied in their responses to the mood induction, with Visual Analogue Scale (VAS) change in sad mood scores ranging from 7 to 79, and the majority of participants showing at least a 20 point

increase from pre- to post- mood induction. Future studies should include a neutral and/or positive mood induction control condition.

Finally, Agras and Telch (1996; 1998) suggested that changes in negative affect may more robustly impact key cognitive/perceptual features of BED than caloric intake. However, zero-order correlations among study variables suggested that changes in sad mood from pre- to post-induction or from post-induction to post-taste task were not associated with the cognitive feature of loss of control, guilt or the perceptual feature of perceived amount of food eaten among women with BED. Consistent with Pollert et al. (2007), although BED participants experienced greater loss of control over eating as well as a greater sense of guilt than controls, these subjective features of BED appeared unrelated to actual caloric intake and/or current affective state.

Direct and Indirect effects of Brooding Rumination (Hypothesis 6)

The current study tested the hypothesis that a greater tendency to engage in brooding rumination in BED participants would have both direct and indirect effects on eating behaviors during the taste task. [i.e., a) trait rumination directly predicting caloric intake/change in urge to eat (Hypothesis 6b) and b) trait rumination predicting reactivity to sad mood induction (Hypothesis 6a), with greater degree of sad mood change subsequently predicting greater caloric intake/change in urge to eat/loss of control (Hypothesis 5)]. Although underpowered, these analyses represent a first step towards testing this moderated-mediation (i.e., the expectation that VAS change would mediate the effects of rumination on calories consumed uniquely in BED women). We did not

find support for this hypothesis. In both BED and control women, brooding rumination (RRS-B) scores were positively correlated with depressive symptoms on the BDI-II. However, a greater tendency to brood was not associated with greater mood reactivity to experimental mood induction nor was it associated with caloric intake or change in urge to eat. Individuals with BED may ruminate about an increase in dysphoric affect following, rather than preceding, binge eating episodes. Momentary assessments of ruminative behaviors and mood over the course of a binge would be needed to elucidate the temporal sequence. It is also possible that proclivity towards brooding rumination is more broadly related to degree of psychopathology/depressive symptoms in women with BED rather than to binge eating specifically. In turn, depressive symptoms contribute to greater disinhibition of eating and increased caloric consumption. Thus, future studies should use a prospective design to assess whether the relation between trait-brooding rumination and binge eating behaviors is partially or fully mediated by depressive symptoms or vice-versa (i.e., whether the relation between depressive symptoms and binge eating is mediated by rumination). Future studies should also distinguish between the content and process of rumination. Rumination, as operationalized in the current study, referred to a pervasive tendency to think about the causes and consequences of sad mood. However, some researchers have suggested that women with eating pathology are more likely to pervasively reflect on food, body image, and/or weight-related concerns (Cooper et al., 2005; Fairburn, 2003; Rawal, Parks, & Williams, 2010). Finally, it is also possible that women with BED binge eat in the context of negative affect out of habit,

regardless of momentary ruminative responses. That is, binge eating may function to alleviate negative affect, such that binge eating behavior becomes a habit-like response to negative affect over time.

Cognitive Affective Vulnerability Processes and Caloric Intake/Urge to Eat

Following Mood Induction (Hypotheses 7-10)

The current study tested the hypothesis that brooding rumination and distress tolerance would interact with negative reinforcement eating expectancies in the prediction of caloric intake during the taste task and change in urge to eat following mood induction among BED participants. Neither brooding rumination (RRS-B), expectancies for eating to alleviate negative affect (EEI-NA), nor their interactive effects predicted caloric intake or urge to eat in either patient status group (Hypotheses 7-8). Neither distress tolerance (DTS scores), expectancies that eating relieves negative affect (EEI-NA scores), nor their interaction accounted for caloric intake or urge to eat in BED participants (Hypotheses 9-10). However, in control group women, both DTS scores and EEI-NA scores, but not their interaction, emerged as significant predictors of caloric intake. Specially, among control participants, better distress tolerance and stronger expectancies that eating will alleviate negative affect were each associated with decreased caloric intake on the taste task.

Conceptualization of Findings

Brooding Rumination and Reactivity to Sad Mood Induction

The general pattern of null findings with respect to brooding rumination was surprising, particularly regarding its non-significant main effect on change in sad mood from pre- to post-dysphoric mood induction, and requires some consideration. A review of the extant literature on rumination revealed that past studies used experimental designs to demonstrate detrimental effects of rumination on sad mood; that is, participants were instructed to ruminate or distract following a sad mood induction and dysphoric mood was more intense and lasted longer in the rumination relative to the distraction condition (e.g., Lyubomirsky et al., 1999). To our knowledge no studies assessed effects of broader, trait-like tendency to rumination as operationalized by the RRS-B on reactivity to sad mood induction. Thus, it is possible that explicit instructions to ruminate must be given for the ruminative tendency to “come online” during an experimental task. Moreover, because brooding rumination is the tendency to think about the causes and consequences of sad mood, it is possible that brooding is associated with reactive distress, rather than an increase in dysphoric mood, following mood induction. Conway, Csank, Holm, and Blake (2000) reported that rumination on sadness, a concept theoretically similar to brooding rumination, predicted subjective distress following mood induction. In turn, consistent with the “trade-off theory,” those who experience their affective state following the mood induction as particularly aversive and distressing may be most likely to engage in disinhibited eating (Kenardy et al., 1996).

In addition, during the mood induction in the current study, participants were asked to record a personally relevant sad memory and to feel sad mood evoked by this memory. It can be argued that participants were essentially ruminating during the mood induction, thereby creating a ceiling effect. The mood induction procedure might have forced even those low in dispositional rumination to ruminate and experience rumination's effects on sad mood to a comparable extent as those with a naturally high tendency to brood. A similar argument was made by Trask and Sigmon (1999) for the Velten Mood Induction Procedure (VMIP; Velten, 1968). During the VMIP, participants read 60 self-referent statements typical of thoughts experienced in depression and are asked to feel what each statement portrays. The authors argue that this task is essentially a rumination task. A general tendency to ruminate as assessed by the RRS-B may not account for variability in intensity of sad mood change above and beyond what is induced by the task itself. It is conceivable instead that stronger tendency to ruminate as assessed by the RRS-B prolongs sad mood and predicts longer *duration* of sad mood and slower return to baseline versus *intensity* of sad-mood, particularly in the context of the experimental dysphoric mood induction used in the current study. It is also possible that the nature of the task precluded participants from ruminating about the causes or consequences of sad mood, as is explicit in an instructed rumination task. That is, participants were instructed to think about a sad memory and therefore knew why they were feeling sad. Broadening out beyond transient dysphoria, future studies should assess whether participants' distress in response to a dysphoric mood induction (as evident in

participant's observable behavior, psychophysiological responses, and/or subjective units of distress ratings) is predicted by ruminative response style and is, in turn, associated with disinhibited eating during the taste task and/or greater urge to eat. Future studies should also examine whether degree of sad mood change and/or distress following dysphoric mood induction interact with negative reinforcement eating expectancies and/or low distress tolerance. It is possible that individuals who experience a greater increase in sad mood and/or are sad for longer engage in problematic eating behaviors if they are unable to tolerate distress and/or have the expectancy that eating will improve mood.

A related construct that may predict how individuals respond to dysphoric mood is experiential avoidance (i.e., the tendency to attempt to alter the type, length, or occurrence of private experiences such as negative thoughts, feelings, memories, or somatic sensations; Blackledge & Hayes, 2001). Gird and Zettle (2009) reported that individuals high in experiential avoidance, as indexed by the Acceptance and Action Questionnaire, reported higher subjective distress following a dysphoric mood induction relative to individuals lower in experiential avoidance.

Negative Reinforcement Eating Expectancies versus Positive Reinforcement Eating Expectancies

Although participants with BED consumed more calories overall than control women during the taste task, greater negative reinforcement eating expectancies did not predict greater caloric consumption in BED. It is possible that the laboratory is not an

appropriate setting for assessing impact of learned expectancies on use of food to ameliorate affect. In the context of a laboratory experiment, eating may not provide sufficient escape from aversive thoughts and feelings (Heatherton et al., 1991). It is also possible that negative reinforcement eating expectancies are related to initiation of eating in the context of negative affect and frequency of binge eating episodes rather than actual caloric intake during an episode. In the current study, greater negative reinforcement eating expectancies was associated with tendency to eat to cope with depressed mood (EES-D) in women with BED and correlated at the univariate level with binge eating severity. Prior studies found that negative reinforcement eating expectancies predicted emotional eating (i.e., eating when feeling upset), and, in turn, emotional eating predicted binge eating onset (e.g., Stice et al., 2002).

Importantly, the current study did not examine other types of eating expectancies; for example, that eating is pleasurable and/or rewarding. Binge eating may be a ‘guilty pleasure’ that provides an escape from stress (Parylak et al., 2011). A recent study (Hennegan, Loxton, & Mattar, 2013) examined cognitive pathways linking reinforcement (reward and punishment) sensitivities and the tendency to overeat in response to appetitive and emotional cues in a large sample of undergraduate women. Whereas expectancies that eating alleviates negative affect/boredom mediated the effects of both reward and punishment sensitivity on emotional eating, the expectancy that eating is pleasurable and rewarding mediated the effects of reward sensitivity on external eating. Thus, in the presence of food cues, such as in the current study, responses of BED

women to the taste task may be better explained by expectancies that eating is pleasurable and rewarding vs. alleviates negative affect.

Maintenance Model vs. Etiological Model of Binge Eating

The current investigation was essentially a study of vulnerabilities that may be related to BED maintenance because it included women with diagnosed BED. The emotion regulation strategies studied here (i.e., brooding, distress tolerance, and expectations that eating alleviates negative affect) may be more related to the initial onset of binge eating in those who go on to develop clinical BED. Women who later develop BED may initially demonstrate a greater tendency to ruminate or have lower distress tolerance and stronger learned expectancies that food will improve mood may be more likely to a) experience significant depressive symptoms and/or b) resort to eating to “escape” aversive mood states, particularly when other coping resources having been depleted by comorbid depression (Dingemans et al., 2009b), c) resort to eating to self-soothe/reward and/or d) use eating in an effort to prevent onset of anticipated aversive mood states. Overtime, these tendencies may become automatic and reflect habitual responding in the presence of food cues rather than transient sad mood states, although for some women, sad mood may remain the context in which this disinhibition of eating is more likely to occur (e.g., Loxton et al., 2011). Consistent with this hypothesis, studies find that women with BED consume more calories than control participants in a laboratory setting whereas studies comparing BED and control individuals on the effects of mood state on subsequent caloric consumption in the laboratory have been equivocal

(e.g., Agras & Telch, 1996; 1998). Regarding binge eating maintenance over time in BED, sad mood/depressive symptoms may become the “context” in which the “habit” of binge eating occurs, particularly when food cues are present.

Personality Traits Relevant to Binge Eating

What other variables (if not brooding rumination, distress tolerance, and expectations that eating alleviates negative affect) may explain variance in caloric intake and urgency to eat among BED women under conditions of acute negative affect. It has been proposed that binge eating in BED is driven by disinhibition of eating in the presence of food cues due to impulsivity. Specifically, binge eating is an impulsive behavior that is particularly likely to occur in the presence of food cues and other contexts previously associated with eating, including sad mood.

Research findings support the importance of impulsivity in binge eating (Fischer, Smith, & Anderson, 2003; Racine, Culbert, Larson, & Klump, 2009). Although impulsivity is a multifaceted construct, reward sensitivity (i.e., hypersensitivity for appetitive or rewarding stimuli, particularly disorder-specific food cues) and negative action urgency (i.e., the tendency to engage in rash action in response to aversive affect) appear to be particularly relevant to BED. Schag et al. (2013) used eye-tracking technology to demonstrate that women with BED displayed both greater reward sensitivity as measured by time spent gazing at food versus neutral stimuli and had greater difficulty looking away from food stimuli (i.e., inhibiting rash-spontaneous behavior towards food stimuli) relative to weight-matched and normal weight controls.

Research has shown that relative to weight-matched controls, BED patients are characterized by hyper-responsiveness to reward stimuli, particularly food cues, as evidenced by sustained stimulation of the reward system (e.g., Davis and Carter, 2009; Schienle, Schafer, Hermann, Vaitl, 2009), substantiating the importance of dopaminergic mechanisms in etiology of BED (Davis et al., 2008; 2011; 2012). Similarly, negative action urgency, is a genetically transmitted risk factor that contributes to both binge eating and emotional eating (Anestis, Smith, Fink, & Joiner, 2009; Claes et al., 2005; Fischer & Smith, 2008; Racine et al., 2013) and a prospective risk factor for binge eating in both middle school- and college-aged samples (Fischer, Peterson, & McCarthy, 2013; Pearson, Combs, Zapolski, & Smith, 2012). Negative action urgency may confer risk for binge eating by interfering with deliberate control over eating. Thus, persons with BED may have greater sensitivity to reward stimuli, particularly food cues, and difficulty inhibiting rash behavior in the presence of these cues.

Racine and colleagues (2013) demonstrated that genetic factors contributed to both negative urgency and trait levels of negative affect, which in turn accounted for unique and significant variability in emotional eating and binge eating. These results suggest that individuals most at risk for binge eating experience high levels of negative affect, are prone to depression, and display a tendency towards emotion-based rash action. A tendency to respond to negative affect with rash action may be a stronger predictor of binge eating development than low distress tolerance and/or brooding, particularly when combined with learning experiences relative to amelioration of

negative affect via eating. Further, because trait levels of negative affect explained unique variability in binge eating (see Schag, Schonleber, Teufel, Zipfel, & Giel, 2013 for a review), higher brooding and lower distress tolerance scores may reflect higher levels of negative affectivity and confer vulnerability to binge eating development either directly or through depressive symptomatology. To assess whether action urgency plays a role in BED maintenance, particularly in the context of transient sad mood, future studies should test direct and indirect effects (i.e., as mediated by depressive symptoms and/or moderated by increase in dysphoric mood) on both perceptual features of BED, such as perceived amount eaten and loss of control as well as actual caloric intake following an experimental dysphoric mood induction.

Eating Behavior in the Laboratory Setting

Although the methodology employed in the current study increased internal validity, the ecological validity and generalizability of participants' behavior while in the laboratory must be considered. In the current study, higher binge eating severity was associated with emotion regulation difficulties, particularly brooding rumination. However, difficulties with emotion regulation and/or negative reinforcement eating expectancies did not predict participants' eating behavior during the "taste task." Although we found that BED participants consumed more calories than controls on the taste task, it is possible that BED participants, particularly those with a more frequent/severe pattern of binge eating, inhibited their eating in the laboratory setting. Eating alone due to embarrassment of overeating is a diagnostic criteria for BED. It is

possible that participants with BED did not feel comfortable enough to eat as “much as they wanted.” Thus, it would have been informative to assess whether participants with BED engaged in disinhibited and/or binge eating upon leaving the laboratory.

Specifically, did exposure to food cues in the laboratory “trigger” naturalistic binge eating in this population? A qualitative study and/or a study using EMA technology could empirically test this question. EMA procedures may be particularly useful, as they are more ecologically valid given that they occur in the natural environment rather than in a laboratory setting

Relative to other studies, participants in the current study were presented with relatively small amounts of each food (e.g., ~ 150-200g of each food item). Rolls, Morris, and Roe (2002) demonstrated that BED participants ate significantly more when offered the largest portion (1000g) than when offered the smallest portion (500g). Whereas the current study found a difference between BED and control women in caloric intake, studies that presented a single-item or a small portion did not (Gosnell et al., 2001; Lattimore, 2000; Schulz & Laessle, 2012). It is possible the difference would have been larger and/or that the predictors examined would have shown significant effects on caloric intake if a larger amount was presented. We also did not instruct participants to “binge eat” or “to eat as much as you can” (Samuels et al., 2009). In fact, participants were told to eat as much as “needed” to assess taste perceptions of food items presented. Thus, we may have failed to give BED participants “permission” to engage in binge eating and/or did not provide appropriate cues for binge eating.

Although participants' suspiciousness of study aims was assessed, it is possible that participants suspected that amount of food eaten would be measured. The majority of participants did not voice concerns about measurement of food intake. In response to the question "What do you think this experiment was trying to study?" on the funnel debriefing form, many participants wrote that the study aimed to investigate the relationship between "sad mood and eating behaviors." Likewise, some participants were aware that the two tasks (i.e., sad mood induction and taste task) were related. No participant explicitly stated that the taste task was intended to measure caloric intake. However, impact of participants' awareness/suspicion on behavior cannot be ruled out. Participants also noted that the length of time they spent in the taste task room impacted their behavior. Several participants in the BED group responded that they would have lost control over eating and would have experienced a binge eating episode if they had to spend more time alone presented with taste task foods. It is important to note that hunger levels and food preferences were accounted for in the current study. Specifically, all participants were instructed to abstain from eating 2 hours prior to the taste task study visit and all but one participant stated that they followed this instruction. Participants also provided favorable ratings to food items presented during the taste task, with all foods rated as at least 65 on the VAS likeability scale ("0 = did not like at all" and "100 = extremely liked it").

It is also possible that daily affect (i.e., stable negative mood and/or increase in negative affect throughout the day) is more robustly associated with binge eating than

momentary increase in sad mood. EMA studies consistently find that negative affect, particularly guilt, increase in the hours leading to a binge eating episode (e.g. Berg et al., 2014). Likewise, in the current study, depressive symptoms were associated with caloric intake in BED women. Thus, the relation between affective-shifts, cognitive-affective emotion regulation processes, and binge eating may be best captured using EMA technology.

Limitations and Future Directions

As a major limitation, our sample consisted of a relatively homogenous group of relatively young and educated Caucasian women. Although we aimed to recruit community participants, many of our participants were undergraduate students who completed the study for course credit. Thus, generalizability of findings to community women with longer duration BED, men with BED, older individuals, and more diverse samples (across race, ethnicity, and SES) is questionable. It is also worth noting that the sample size was relatively small. Therefore, significant findings should be replicated and, conversely, null findings are subject to Type 2 error and should be evaluated in a larger, more definitive study. Although we made an effort to match participants on BMI, participants with BED had significantly higher BMI's than controls. BMI did not appear to correlate with outcomes of interest; however, greater effort should be made to include overweight and obese control participants in future comparisons with BED patients.

Although the experimental design facilitated “in vivo” assessment of the impact of sad mood on caloric intake, future research regarding the etiology of BED would

benefit from prospective, longitudinal studies that examine how potential risk factors and their interactions relate to development and maintenance of binge eating in BED. As previously noted, constructs examined in the current study may be more relevant to onset of binge eating versus maintenance. In addition, because the current study used self-report measures, current findings reflect participants deliberate responses on scales and are likely influenced by their perceptions of factors contributing to binge eating behavior. For example, binge eating in BED might in fact reflect a habitual response pattern, but BED patients may perceive their behavior as being driven by negative emotional states. Further, a control condition involving neutral or positive mood induction would provide additional support for the specific role of negative affect in the proposed theoretical model. Alternatively, a within-subjects design with different mood induction conditions may provide a powerful illustration of the proposed relationships.

Conclusions and Clinical Implications

The current investigation compared women with BED and healthy controls with no history of eating disorders in terms of brooding rumination on the RRS-B, distress tolerance on the DTS, and expectancies that eating will improve negative affect on the EEI-NA. Results revealed that women with BED reported higher brooding rumination and stronger expectancies that eating will alleviate negative affect and lower distress tolerance than controls. Among women with BED, brooding rumination was positively associated with binge eating severity, and stronger negative reinforcement expectancies for eating and less distress tolerance were associated with greater urgency to eat in

response to depression and more frequent binge eating episodes. Degree of reactivity to sad mood induction was not associated with subsequent caloric intake, perceived loss of control, or pre- to post-dysphoric mood induction change in urge to eat in BED. There was no support for direct or indirect effects of brooding rumination on change in urge to eat over the mood induction or total caloric intake on the taste task. We also did not find support for the effects of distress tolerance and negative reinforcement expectancies for eating on these outcomes.

Regarding the clinical implications of these results, difficulties with emotion regulation strategies and depressive symptoms should be assessed in persons with BED and may represent important treatment targets. BED patients may benefit from learning more adaptive emotion regulation strategies, given that brooding rumination was associated with binge eating severity, and both negative reinforcement expectancies and low distress tolerance were associated with emotional eating in persons with BED. Indeed, recent studies have tested the efficacy of interventions aimed at helping individuals with BED reduce emotional overeating by developing more adaptive emotion regulations skills (Robinson & Safer, 2012; Safer, Robinson, & Jo, 2010; Telch, Agras, & Linehan, 2001). Although cognitive-affective emotion regulation processes were not directly linked to eating behavior during an experimental task, results support the idea that women with BED consume more calories and experience greater loss of control in the presence of binge foods relative to weight-matched controls. Greater caloric consumption and loss of control have been linked to increased food-related reward

sensitivity and rash spontaneous behavior in response to food cues. Thus, helping BED patients learn more adaptive emotion regulation and distress tolerance skills, including identifying and accessing alternative rewards (i.e., self-sooth skills, building mastery experiences, etc.) and managing action urgency via distraction or consideration of pros and cons of engaging in an impulsive behavior might be particularly powerful interventions.

Recent randomized controlled studies support the efficacy of dialectical behavior therapy for BED (DBT-BED; Safer et al., 2010) in reducing binge severity. DBT-BED focuses on mindfulness, emotion regulation, distress tolerance, and interpersonal effectiveness skills. However, because Safer and colleagues did not find that treatment effects were maintained at 3- 6- or 12-month follow-up assessments, additional methods to sustain abstinence from binge eating after DBT-BED should be considered. Because new learning related to eating appears to be context-specific and susceptible to renewal effects, exposure with response prevention should occur in variety of contexts, including a variety of affective states, to increase the generalizability of extinction effects (e.g., inhibition of binge eating in response to contextual antecedents; Bouton, 2004; Bouton, 2011; Todd et al., 2012). Thus, in this patient population, cognitive-behavioral interventions should incorporate exposure to food cues (i.e., typical binge foods) with response prevention in a variety of contexts to promote inhibitory learning.

In summary, results suggest that factors other than brooding rumination, distress tolerance, and negative reinforcement eating expectancies may account for increased

caloric intake observed among BED diagnosed participants during an experimental taste task. Possible candidate constructs include impulsivity, sensitivity to food cues, experiential avoidance, and severity of depressive symptoms. Brooding rumination, low distress tolerance, and negative reinforcement eating expectancies may be more relevant to BED onset via their role in increasing vulnerability to emotional eating versus maintenance and/or reactivity to shifts in affective states outside the laboratory. Specifically, constructs examined in the current study may have contributed to initial BED development and/or depressive symptoms, but not serve as proximal antecedents for episodes of binge eating. Greater caloric intake observed among BED participants in the laboratory setting may reflect a habitual response pattern to food cues and/or be associated with reactive distress to a negative mood induction rather than degree of sad mood change per se. It is also possible that facets of emotion dysregulation are best conceptualized as consequences of binge eating that are predictive of worse outcomes and/or greater psychopathology in this patient population. Given emerging research findings on heightened reward sensitivity and altered dopaminergic activity, particularly in response to food cues, longitudinal studies that prospectively examine the relation between facets of emotion regulation, behavioral/cognitive features of BED, and relevant genetic/neurobiological/behavioral indicators are needed to fully understand cognitive, affective, and behavioral components of binge eating in BED.

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Table 1. Participant Demographic Variables and Baseline Characteristics

	All (<i>N</i> = 76)	BED (<i>n</i> = 38)	Controls (<i>n</i> = 38)	Statistics
Age, Mean (SD)	24.66 (10.24)	24.89 (10.30)	24.42 (10.32)	$t(74) = .20$, $p = .84$, Cohen's $d = .05$
BMI, Mean (SD)	25.9 (4.8)	27.04 (5.44)	24.74 (3.80)	$t(66.14) = 2.14$, $p = .04$, Cohen's $d = .53$
Ethnicity, Number (%)				
Hispanic/Latino	1 (1.3%)	0	1 (2.6%)	$X^2(1, N = 76)$ $= 1.01, p = .31$
Non Hispanic	75 (98.7%)	38 (100%)	37 (97.4%)	
Race, Number (%)				
Caucasian	67 (88.2%)	33 (86.8%)	34 (89.5%)	$X^2(1, N = 76)$ $= .13, p = .72$
African American	1 (1.3%)	0	1 (2.6%)	
Asian	7 (9.2%)	4 (10.5%)	3 (7.9%)	
Native American	0 (0%)	0	0	
Other	1 (1.3%)	1 (2.6%)	0	
Marital Status, Number (%)				
Single	60 (78.9%)	30 (78.9)	30 (39.5)	$X^2(1, N = 76)$ $= .11, p = .74$
Engaged	1 (1.3)	1 (2.6)	0	
Married	2 (2.6%)	2 (5.3)	0	
Living Together	8 (10.5%)	3 (7.9%)	5 (13.2%)	
Divorced	5 (6.6%)	2 (5.3%)	3 (7.9%)	
Education (%)				
High School Degree	18 (23.7%)	9 (23.7%)	9 (23.7%)	$X^2(1, N = 76)$ $= .24, p = .62$
Currently in College	34 (44.7%)	18 (47.4%)	16 (42.1%)	
Graduated from a 4-yr College	13 (17.1%)	5 (13.2%)	8 (21.1%)	
Graduated with a Master's Degree	7 (9.2%)	5 (13.2%)	2 (5.3%)	
Graduated with a Ph.D./MD/JD Degree	4 (5.3%)	1 (2.6%)	3 (7.9%)	

Notes. BMI = Body Mass Index as calculated based on weight and height measurements.

Chi-Square is compares proportion of Caucasian versus all others in each group.

Chi-Square compares single/divorced versus engaged/married/living together marital status. Chi-Square compares High School Degree/Some College versus College Graduate/Graduate Education.

Table 2. Study Outcome Variables in the BED and Control Groups

	BED (n = 38)	Control (n = 36)		
Variable	Mean (SD)	Mean (SD)	t (df)	Cohen's <i>d</i>
BES	23.45 (7.77)	4.39 (4.35)	13.19 (58.08)**	3.46
BDI-II	13.82 (9.18)	3.68 (4.99)	5.96 (57.41)**	1.57
EES-D	12.74 (3.75)	6.68 (4.06)	6.75 (74)**	1.57
DTS	3.21 (.80)	3.97 (.71)	-4.38 (74)**	-1.02
EEI-NA	4.09 (1.20)	2.42 (1.04)	6.47 (74)**	1.50
EDQ-R	1.71 (1.20)	.82 (1.10)	3.38 (74)**	.78
EDQ-S	3.34 (1.68)	1.08 (1.03)	7.07 (61.40)**	1.80
EDQ-W	3.07 (1.64)	.93 (.92)	7.04 (58.04)**	1.85
EDQ-E	2.21 (1.27)	.17 (.26)	9.69 (39.38)**	3.09
EDQ Global Score	2.58 (1.31)	.75 (.69)	7.65 (56.22)**	2.04
EDQ Binge Days	9.87 (7.77)	.72 (1.97)	7.03 (41.72)**	2.18
DERS	83.32 (23.97)	63.46 (17.50)	4.11 (67.74)**	1.00
RRS-B	6.21 (3.73)	2.58 (2.46)	5.01 (64.03)**	1.25
Loss of Control	53.05 (29.94)	81.57 (15.05)	-5.13 (55.22)**	-1.38
Amount Eaten	51.10 (26.06)	38.53 (20.53)	2.30 (72)*	.54
Speed of Eating	57.38 (37.47)	35.57 (22.62)	3.71 (72)**	.87
Total Calories Eaten	561.08 (269.87)	338.72 (138.86)	4.49 (55.95)**	1.20
Guilt	47.30 (30.55)	17.07 (19.74)	5.08 (63.71)**	1.27

Notes. BES = Binge Eating Scale. BDI-II = Beck Depression Inventory, Second Edition. EES-D = Emotional Eating Scale – Depression Subscale. DTS = Distress Tolerance Scale, higher scores reflect greater ability to tolerate distress. EEI-NA = Eating Expectancy Inventory – Negative Reinforcement Expectancy Subscale. EDQ = Eating Disorders Examination Questionnaire. DERS = Difficulties with Emotion Regulation Scale. RRS-B = Ruminative Response Style Questionnaire – Brooding Ruminations. Loss of Control, Amount Eaten, and Speed of Eating were assessed via visual analogue scale. For Loss of Control, higher scores reflect greater sense of control over eating. In cases where, according to the Levene's Test, the equality of variances assumption for the t-test was violated (i.e., variability in scores in the two groups was not the same), results with equal variance not assumed are reported. * $p < .05$ ** $p < 0.01$

Table 3. Correlations Among Predictors and Outcome Variables in the BED and Control Groups

Group		1	2	3	4	5	6	7	8	9	10	11	12	13	14
Control	1.BMI	1.00	.08	.24	.21	-.07	-.02	-.07	.27	.12	-.39*	.15	.03	.06	-.19
	2.BDI-II	.08	1.00	-.05	.50**	.45**	.30	.06	-.04	.19	.56**	-.51**	.64**	-	.05
	3.Cal	.24	-.05	1.00	-.22	-.09	.01	-.32	.29	.18	-.07	-.17	-.20	-.06	.41*
	4.BES	.21	.50**	-.22	1.00	.30	.03	.12	.00	.27	.09	-.29	.54**	-	.35*
	5.EES-D	-.07	.45**	-.09	.30	1.00	.36*	-.01	.02	.11	.32	-.39*	.68**	-.37*	-.01
	6.BE Days	-.02	.30	.01	.03	.36*	1.00	-.13	-.14	-.15	.22	-.30	.45**	-.01	-.25
	7.T1 to T2 Sad	-.07	.06	-.32	.12	-.01	-.13	1.00	-.70**	-.08	-.17	.12	.26	.25	-.32
	8.T2 to T3 Sad	.27	-.04	.29	.00	.02	-.14	-.70**	1.00	.34*	.07	-.15	-.18	-.35*	.29
	9.T2 to T3 Urge to Eat	.12	.19	.18	.27	.11	-.15	-.08	.34*	1.00	-.06	-.09	.12	-.34*	.23
	10.RRS-B	-.39*	.56**	-.07	.09	.32	.22	-.17	.07	-.06	1.00	-.54**	.19	.11	.07
	11.DTS	.15	-.51**	-.17	-.29	-.39*	-.30	.12	-.15	-.09	-.54**	1.00	-	.01	.17
	12.EEI-NA	.03	.64**	-.20	.54**	.68**	.45**	.26	-.18	.12	.19	-.51**	1.00	-.24	.28
	13.LOC	.06	-.58**	-.06	-.58**	-.37*	-.01	.25	-.35*	-.34*	.11	.01	-.24	1.00	-
	14.Amo unt Eaten	-.19	.05	.41*	.35*	-.01	-.25	-.32	.29	.23	.07	.17	.28	-.46**	1.00
BED	1.BMI	1.00	.27	.20	-.04	.09	.06	.10	.16	.13	.16	.14	.16	.25	-.13
	2.BDI-II	.27	1.00	.21	.53**	.28	.21	.02	-.13	.10	.56**	-.50**	.18	-.10	.28
	3.Cal	.20	.21	1.00	0.16	.08	-.13	.14	.19	-.04	.08	-.04	-.11	-.54**	.66**
	4.BES	-.04	.53**	.16	1.00	.22	.53**	.14	-.27	.05	.60**	-.20	.32	.06	.22
	5.EES-D	.09	.28	.08	.22	1.00	.03	-.06	-.22	.04	.40*	-.36*	.54**	-.01	.25
	6.BE Days	.06	.21	-.13	.53**	.03	1.00	-.16	-.02	-.08	.14	.30	.27	-.15	-.04
	7.T1 to T2 Sad VAS	.10	.02	.14	.14	-.06	-.16	1.00	-.58**	.01	.18	-.12	.07	.14	-.20
	8.T2 to T3 Sad VAS	.16	-.13	.19	-.27	-.22	-.02	-.58**	1.00	-.29	-.30	.14	-.27	-.08	-.07
	9.T2 to T3 Urge to Eat	.13	.10	-.04	.05	.04	-.08	.01	-.29	1.00	.12	-.08	.10	.04	.24
	10.RRS-B	.16	.56**	.08	.60**	.40*	.14	.18	-.30	.12	1.00	-.37*	.35*	-.06	.13
	11.DTS	.14	-.50**	-.04	-.20	-.36*	.30	-.12	.14	-.08	-.37*	1.00	-.25	-.06	.20
	12.EEI-NA	.16	.18	-.11	.32	.54**	.27	.07	-.27	.10	.35*	-.25	1.00	-.21	.18
	13. LOC	.25	-.10	-.54**	.06	-.01	-.15	.14	-.08	.04	-.06	-.06	-.21	1.00	-
	14.Amount Eaten	-.13	.28	.66**	.22	.25	-.04	-.20	-.07	.24	.13	.20	.18	-.45**	1.00

Notes. *Correlation is significant at the 0.05 level; **Correlation is significant at the 0.01 level. Cal = Total calories eaten. BE = Number of binge days in the past 28 days. T1 to T2 = change in VAS score from pre to post- mood induction. T2 to T3 = change in VAS score from post-induction to post-taste task. LOC = Perceived loss of control rating on the VAS. For other measures abbreviations see Table 1.

Table 4. Changes in VAS Scores Across the Mood Induction and Taste Task in the BED and Control Groups

	Group													
	Control (n = 36)						BED (n = 38)							
	Time			Time			Time			Time				
	Pre-Induction	Post-Induction	Post-Taste Task	Pre-Induction	Post-Induction	Post-Taste Task	Pre-Induction	Post-Induction	Post-Taste Task	Pre-Induction	Post-Induction	Post-Taste Task		
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Sad Mood	14.42	(17.89)	45.15	(24.56)	18.75	(17.27)	13.21	(17.47)	51.39	(19.81)	26.86	(21.16)	26.86	(21.16)
Tension	17.32	(18.70)	22.94	(21.44)	17.76	(19.23)	23.86	(21.13)	35.75	(25.88)	27.14	(21.82)	27.14	(21.82)
Urge to Eat	26.19	(23.08)	23.22	(20.52)	22.14	(21.43)	38.59	(21.26)	42.34	(22.08)	41.50	(24.94)	41.50	(24.94)
Happiness	68.82	(17.88)	43.46	(16.16)	58.78	(15.04)	61.30	(22.06)	39.36	(19.25)	48.97	(18.31)	48.97	(18.31)
Shame	7.87	(13.66)	15.69	(19.79)	11.23	(14.23)	11.63	(15.87)	18.18	(22.73)	36.47	(27.83)	36.47	(27.83)
Anger	9.46	(12.29)	18.81	(20.97)	8.86	(12.69)	6.36	(7.74)	25.18	(24.89)	12.30	(14.90)	12.30	(14.90)
Anxiety	21.40	(22.48)	25.19	(21.64)	19.18	(23.29)	25.42	(23.99)	28.50	(24.16)	25.28	(22.78)	25.28	(22.78)
Subjective Distress	11.72	(16.26)	29.76	(22.33)	11.44	(16.26)	15.46	(17.57)	28.37	(22.06)	22.95	(21.49)	22.95	(21.49)
Hunger	33.51	(22.84)	25.08	(21.41)	21.97	(19.08)	41.01	(20.48)	43.16	(22.75)	27.03	(21.91)	27.03	(21.91)
Guilt	7.96	(12.82)	17.70	(21.05)	12.57	(16.44)	10.51	(14.51)	25.93	(23.68)	34.09	(25.95)	34.09	(25.95)

Note. VAS = visual analogue scale.

Table 5. Summary of Study Hypotheses, Statistical Tests, and Results

Hypothesis	Statistical Methods Used	Results Summary
1. BED participants will score higher on measures of ruminative brooding (RRS-B), distress tolerance (i.e., demonstrate lower distress tolerance, DTS), and will perceive themselves as having fewer strategies to cope with negative mood (DERS) relative to controls.	T-tests/or ANCOVA framework controlling for effects of depressive symptoms (BDI-II scores).	BED vs. control: - Higher frequency of brooding rumination (RRS-B) - Lower Distress tolerance (DTS) - Greater difficulties with emotion regulation (DERS)
2. BED participants will endorse a greater urge to eat to cope with negative affect (EES) and report a more positive expectancy that food will serve to regulate negative affect (EEI-NA).	T-tests/or ANCOVA framework controlling for effects of depressive symptoms (BDI-II scores).	BED vs. control: - Greater urge to eat to cope with negative affect (EES-D) - More positive expectancies that food will improve mood (EEI-NA scores)
3. Among BED participants, significant variability in BES scores, EES scores, and the number of binge eating episodes in the past 28 days, will be accounted for by DTS total score, RRS-Brooding, and EEI-NA.	Linear regression or hierarchical linear regression analyses controlling for BDI-II scores.	Among BED participants: - Sig. variability in binge eating severity (BES scores) accounted by depressive symptoms (BDI-II scores; step 1) and brooding rumination (RRS-B scores; step 2). - Sig. variability in eating to cope with depressed mood (EES-D scores) accounted by negative reinforcement eating expectancies (EEI-NA scores).
4. BED participants will report greater increase in urge to eat over the mood induction, consume more calories during the taste task, and report greater loss of control than control participants after eating.	Urge to Eat: A repeated measures 2 (BED, control) \times 3 (baseline, post-mood induction, post-taste task) ANOVA on urge to eat. Independent samples t-tests: Caloric intake and perceived loss of control.	Urge to Eat: - Collapsing across time points, BED > controls on urge to eat. - No Time main effect. - Time \times Group <i>ns</i> , but visual display suggests that urge to eat increased in BED group. Caloric Intake: - BED > controls - Perceived Control Over amount Eaten - Control > BED
5. Among BED participants only, greater increase in sad affect following the dysphoric mood induction will be associated with greater increase in urge to eat, caloric intake, and loss of control over eating during the taste task.	Hierarchical regression analyses, with caloric intake, degree of change in urge to eat, and loss of control rating as the outcome variables. Controlling for BDI-II scores (Step 1), Group (BED, control) and sad mood change scores (Time 1 to Time 2) as predictors in step 2. Group \times VAS sad mood change in step 3.	BED group vs. control: - Degree of mood change did not account for group differences in caloric intake, perceived control over eating, and change in urge to eat Among BED participants: - Change in sad mood from pre- to post-mood induction was not predictive of caloric intake or change in urge to eat - Increase in sad mood associated with decreased sense of control over eating, but effect not

<p>9. Distress tolerance (DTS) and negative reinforcement eating expectancies (EEI-NA) will each uniquely predict pre- to post-mood induction change in urge to eat and caloric intake following dysphoric mood induction for BED-diagnosed participants.</p>	<p>Linear hierarchical regression models:</p> <ul style="list-style-type: none"> - Caloric intake and urge to eat regressed on DTS, EEI-NA, and Group - Step 1: DTS, EEI-NA, Group - Step 2: EEI-NA × DTS, DTS × Group, and EEI-NA × Group in the second step. - Step 3: The EEI-NA × DTS × Group <p>Within group analyses:</p> <ul style="list-style-type: none"> - Within each study group caloric intake and urge to eat regressed on DTS and EEI-NA - Hypothesis 9: Step 1: DTS & EEI-NA - Hypothesis 10: Step 2: EEI-NA × DTS 	<p>Change in urge to eat from pre- to post-induction</p> <ul style="list-style-type: none"> - DTS total scores, EEI-NA, Group, and/or the their interactive effects did not explain significant variance <p>Caloric Intake during taste task</p> <ul style="list-style-type: none"> - Among control group participants, both DTS total scores and EEI-NA total scores predicted caloric intake. - Better distress tolerance → decreased caloric intake on the taste task. - Greater endorsement of negative reinforcement eating expectancy → decreased caloric intake <p>BED group: Neither EEI-NA, DTS, nor their interaction predicted caloric intake.</p>
<p>10. DTS × EEI-NA interaction will qualify the main effects of each predictor. Within the BED group, lower DTS and higher EEI-NA scores will be associated with greater caloric intake and greater increase in urge to eat</p>		

Notes. BES = Binge Eating Scale. BDI-II = Beck Depression Inventory, Second Edition. EES-D = Emotional Eating Scale – Depression Subscale.

DTS = Distress Tolerance Scale, higher scores reflect greater ability to tolerate distress.

EEI-NA = Eating Expectancy Inventory – Negative Reinforcement Expectancy Subscale.

EDQ = Eating Disorders Examination Questionnaire.

DERS = Difficulties with Emotion Regulation Scale. RRS-B = Ruminative Response Style Questionnaire – Brooding Ruminations. VAS = visual analogue scale.

Table 6. Ruminative Response Style and Negative Reinforcement Eating Expectancies Predicting Total Caloric Intake

Variable	Model 1			Model 2			Model 3		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	328.21	42.08	7.79**	317.69	54.11**	5.87	316.04	54.83	5.76**
Group	246.13	65.91	3.73**	253.50	70.29**	3.61	254.93	70.98	3.59**
RRS-B	7.05	8.75	.80	1.93	18.45	.10	-.86	21.67	-.04
EEL-NA	-31.41	23.83	-1.31	-20.59	38.70	-.53	-26.66	44.98	-.59
Group × EEL-NA				-20.48	54.98	-.37	-16.28	57.50	-.28
Group × RRS-B				5.24	23.00	.23	7.31	24.40	.30
EEL-NA × RRS-B				3.61	7.27	.50	-.21	15.94	-.01
Group × EEL-NA × RRS-B							4.84	17.94	.27
<i>R</i> ²		.23			.01			0.0	
<i>F</i> for change in <i>R</i> ²		7.13**			.17			.7	

Note. *N* = 74. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

p* < .05. *p* < .01.

Table 7. Ruminative Response Style and Negative Reinforcement Eating Expectancies
 Predicting Change in Urge to Eat from Pre- to Post Induction

Variable	Model 1			Model 2			Model 3		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	-1.4	3.61	-.39	-2.97	4.57	-.65	-3.25	4.62	-.70
Group	3.57	5.66	.63	5.30	5.94	.89	5.55	5.98	.92
RRS-B	.07	.75	.10	-1.59	1.56	-1.02	-2.07	1.79	-1.16
EEI-NA	1.75	2.05	.86	1.52	3.27	.46	.46	3.79	.12
Group × EEI-NA				1.07	4.64	.23	1.80	4.85	.37
Group × RRS-B				2.66	1.94	1.37	3.022	2.06	1.47
EEI-NA × RRS-B				-.89	.61	-1.44	-1.55	1.34	-1.15
Group × EEI-NA × RRS-B							.84	1.51	.56
<i>R</i> ²		.04			.08			.43	
<i>F</i> for change in <i>R</i> ²		1.10			.93			.31	

Note. *N* = 74. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

p* < .05. *p* < .01.

Table 8. Rumination and Negative Reinforcement Eating Expectancies Predicting

Total Caloric Intake by Patient Status Group

Control Participants (<i>n</i> = 36)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	316.13	34.30	9.21**	316.13	35.11	9.00**
RRS-B	-.71	11.21	-.06	-.86	13.62	-.06
EEI-NA	-26.32	23.45	-1.12	-26.66	28.80	-.92
EEI-NA × RRS-B				-.21	10.21	-.02
<i>R</i> ²		.04			0	
<i>F</i> for change in <i>R</i> ²		.72			0	

BED Participants (<i>n</i> = 38)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	571.98	55.52	10.30**	570.97	56.21	10.16**
RRS-B	9.72	12.87	.75	6.44	14.92	.43
EEI-NA	-34.44	40.02	-.86	-42.94	44.67	-.96
EEI-NA × RRS-B				4.63	10.28	.45
<i>R</i> ²		.03			.01	
<i>F</i> for change in <i>R</i> ²		.49			.20	

Note. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

p* < .05. *p* < .01.

Table 9. Rumination and Negative Reinforcement Eating Expectancies Predicting

Change in Urge to Eat by Patient Status Group

Control Participants ($n = 36$)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	-2.59	4.92	-.52	-3.25	4.95	-.66
RRS-B	-.94	1.61	-.58	-2.07	1.92	-1.08
EEL-NA	2.93	3.37	.87	.46	4.06	.11
EEL-NA × RRS-B				-1.55	1.43	-1.08
R^2		.03			.03	
F for change in R^2		.43			1.16	

BED Participants ($n = 38$)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	2.14	3.53	.61	2.30	3.53	.61
RRS-B	.45	.82	.54	.95	3.53	1.01
EEL-NA	.96	2.55	.38	2.26	2.80	.81
EEL-NA × RRS-B				-.71	.64	-1.10
R^2		.02			.03	
F for change in R^2		.33			1.21	

Note. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

* $p < .05$. ** $p < .01$.

Table 10. Distress Tolerance and Negative Reinforcement Eating Expectancies Predicting Total Caloric Intake

Variable	Model 1			Model 2			Model 3		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	326.85	40.79	8.01**	323.06	47.81	6.76**	335.26	47.91	7.00**
Group	248.09	63.44	3.91**	251.78	65.24	3.86**	234.71	65.4	3.59**
DTS	-41.51	24.17	-1.47	-78.08	61.67	-1.26	-68.52	61.29	-1.12
EEL-NA	-35.53	35.87	-1.15	-52.83	42.49	-1.24	-62.60	42.47	-1.47
Group × EEL-NA				24.74	52.86	.47	31.64	52.46	.60
Group × DTS				62.38	87.85	.71	128.16	96.29	1.33
EEL-NA × DTS				-6.58	29.59	-.22	26.37	35.91	.73
Group × EEL-NA ×							-98.15	61.97	-1.58
<i>R</i> ²		.24			.01			.03	
<i>F</i> for change in <i>R</i> ²		7.42**			.19			2.51	

Note. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

p* < .05. *p* < .01.

Table 11. Distress Tolerance and Negative Reinforcement Eating Expectancies

Predicting Change in Urge to Eat

Variable	Model 1				Model 2				Model 3			
	<i>B</i>	<i>SEB</i>	<i>t</i>		<i>B</i>	<i>SEB</i>	<i>t</i>		<i>B</i>	<i>SEB</i>	<i>t</i>	
Constant	-1.29	3.52	-.37	.88	.88	4.0	.22	.74	.74	4.09	.18	
Group	3.34	5.47	.61	1.75	5.48	5.48	.32	1.96	5.59	5.59	.35	
DTS	-1.13	3.09	-.36	.81	5.17	5.17	.16	.70	5.23	5.23	.13	
EEI-NA	1.53	2.08	.74	.54	3.56	3.56	.15	.66	3.63	3.63	.19	
Group × EEI-NA				.922	4.43	4.43	.20	.84	4.48	4.48	.19	
Group × DTS				-8.04	7.37	7.37	-1.09	-8.85	8.22	8.22	-1.08	
EEI-NA × DTS				5.18	2.48	2.48	2.09*	4.78	3.07	3.07	1.56	
Group × EEI-NA × DTS								1.2	5.29	5.29	.23	
<i>R</i> ²		.05				.06			.00			
<i>F</i> for change in <i>R</i> ²		1.14				1.46			.052			

Note. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

p* < .05. *p* < .01.

Table 12. Distress Tolerance and Negative Reinforcement Eating Expectancies Predicting Total Caloric Intake by Patient Status Group

Control Participants ($n = 36$)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	325.50	27.94	11.65**	335.26	28.87	11.61
EEI-NA	-54.79	24.96	-2.19*	-62.60	25.60	-2.45*
DTS	-76.17	36.67	-2.08*	-68.52	36.94	-1.85
EEI-NA \times DTS				26.37	21.64	1.22
R^2		.15			.04	
<i>F</i> for change in R^2		2.97*			1.49	
BED Participants ($n = 38$)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	575.33	56.33	10.21**	569.97	56.32	10.12**
EEI-NA	-27.80	38.98	-.71	-30.96	38.94	-.79
DTS	-23.30	58.31	-.40	59.64	93.92	.63
EEI-NA \times DTS				-71.78	63.87	-1.12
R^2		.02			.03	
<i>F</i> for change in R^2		.28			1.26	

Note. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

* $p < .05$. ** $p < .01$.

Table 13. Distress Tolerance and Negative Reinforcement Eating Expectancies Predicting Change in Urge to Eat by Patient Status Group

Control Participants ($n = 36$)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	-1.03	4.29	-.24	.74	4.40	.17
EEI-NA	2.07	3.83	.54	.66	3.89	.17
DTS	-.69	5.62	-.12	.70	5.61	.12
EEI-NA \times DTS				4.78	3.28	1.45
R^2		.02			.06	
<i>F</i> for change in R^2		.27			2.11	
BED Participants ($n = 38$)						
Variable	Model 1			Model 2		
	<i>B</i>	<i>SE B</i>	<i>t</i>	<i>B</i>	<i>SE B</i>	<i>t</i>
Constant	2.25	3.57	.63	2.70	3.52	.45
EEI-NA	1.23	2.47	.50	1.50	2.44	.54
DTS	-1.25	3.70	-.34	-8.15	5.88	.14
EEI-NA \times DTS				5.97	4.00	.14
R^2		.01			.06	
<i>F</i> for change in R^2		.23			2.23	

Note. See Table 2 for measures abbreviations. All continuous predictors were centered at the mean.

* $p < .05$. ** $p < .01$.

Figure 1. Cognitive and Affective Processes Impacting Binge-Eating Relevant Behaviors in Response to Dysphoric Mood Induction.

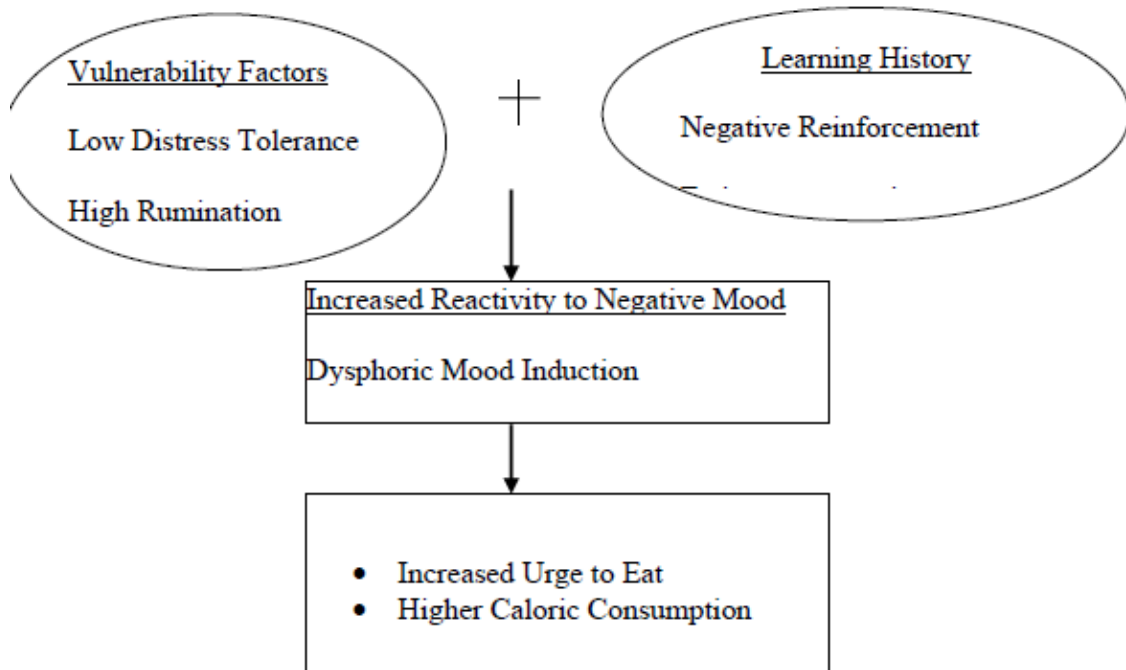


Figure 2. Changes in Sad Mood VAS Ratings across the Mood Induction and Taste Task in BED and Control Participants

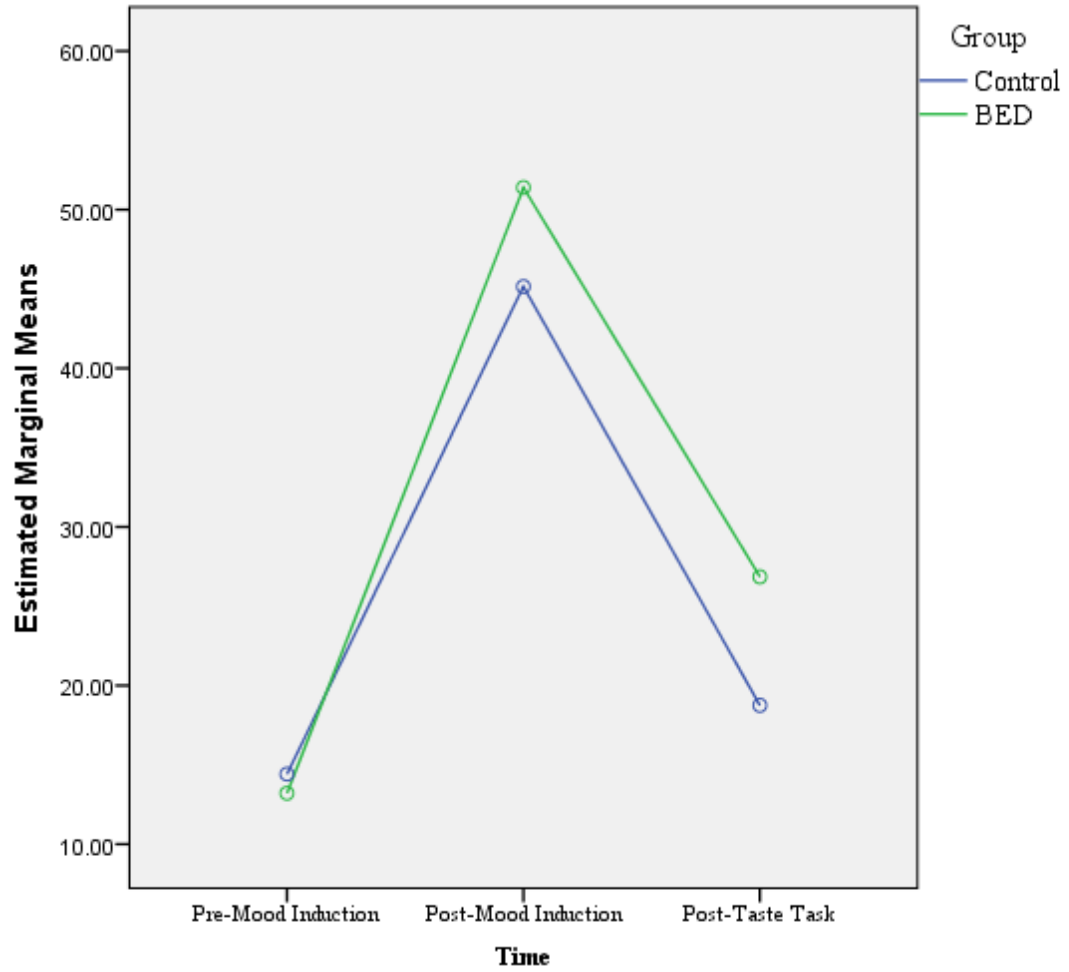


Figure 3. Changes in Urge to Eat VAS Ratings across the Mood Induction and Taste Task in BED and Control Participants

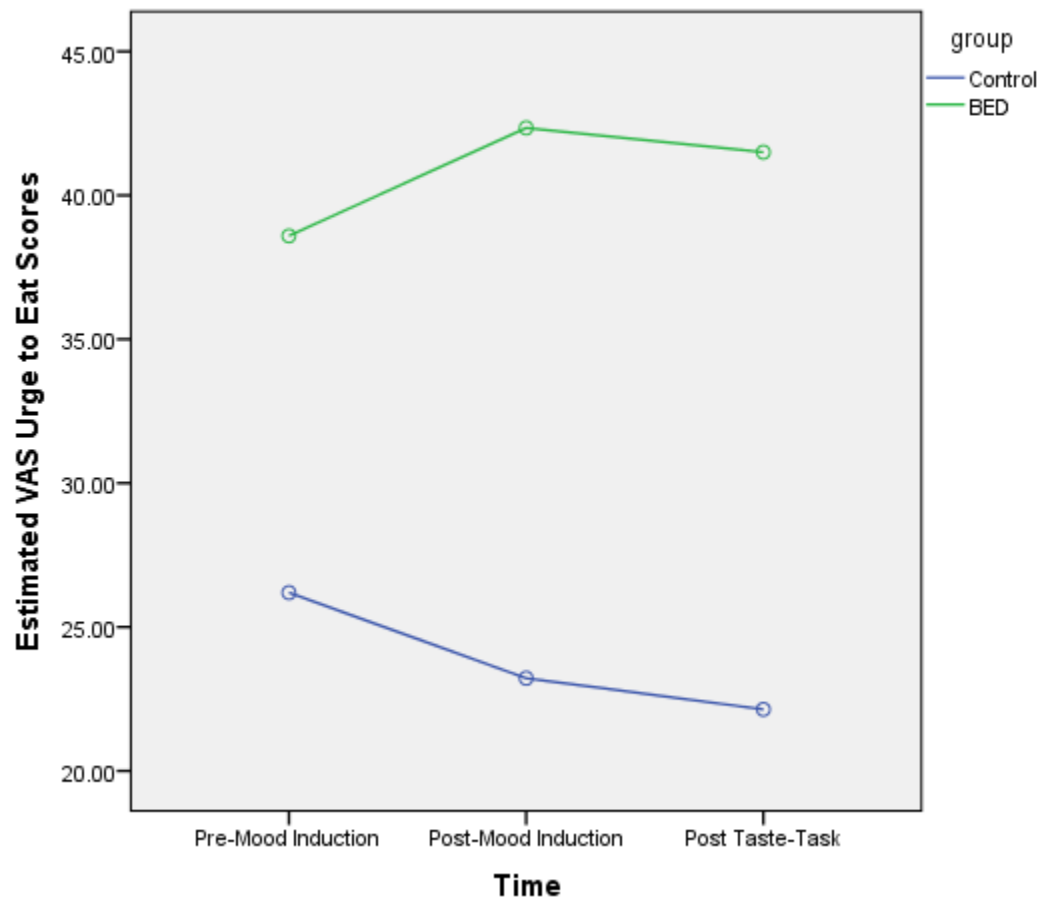


Figure 4. Changes in Sad Mood VAS Ratings and BDI-II Scores Predicting Caloric Intake in BED Participants.

